

Commentary on: Sauvageau A, Bourgault A, Racette S. Cerebral traumatism with a playground rocking toy mimicking shaken baby syndrome. *J Forensic Sci* 2008;53(2):479–82

Sir,

Sauvageau et al. open their case report: “Shaken baby syndrome, one of the most deadly and devastating forms of child abuse, is caused by violent shaking” (1). Then citing five references that pre-date the recent experimental literature that gravely undermine the validity of the shaken baby hypotheses, they state, “The SBS refers to the severe brain injury caused by the violent shaking of an infant” (1). They continue: “The classic signs of shaking-induced damage (the “classic triad”) include subdural hemorrhage, brain swelling, and retinal hemorrhages....The classic lesions of SBS are caused by an acceleration-deceleration mechanism. The child is seized by the chest or shoulder and violently shaken for an average of 5–15 sec, the head being shipped back and forth in the anterior posterior direction...(this) generates repetitive movement of the brain within the skull and tearing of bridging veins” (1). This definition is not vague and it does not include impact. It is unambiguously asserted that shaking alone, pure shaking without impact, is the cause of the “triad” and that this “SBS classic triad is not compatible with a minor trauma history such as simple falls from a couch or from parent’s arms, or a story of a bumped head during baby carrying” (1). The author does concede that “motor-vehicle accidents and falls from great height are considered possible mimicker of SBS” (1).

I would point out to Sauvageau et al. that both the 2001 Technical Report from the American Academy of Pediatrics (AAP) (2) and the Position Paper of Mary Case of 2001 (3), on which they relied for their information, were allowed to expire by the AAP and the National Association of Medical Examiners (NAME) and were not renewed in the face of the newly published experimental research. This new research found that:

1. Shaking-induced vitreous traction did not cause primary retinal hemorrhage, schisis, or folds, in an appropriate animal model subjected to $>100,000 \text{ rad/sec}^2$ of rotational acceleration (4), which is 40 times greater than what the biomechanical research indicates would be the upper limit of what could be realistically achieved in an abusive shaking (5) and more than 70 times greater than what Carole Jenny of the AAP’s Committee on Child Abuse and Neglect (COCAN) was able to generate with the shaking of a more advanced anthropomorphic model weighing $<8 \text{ lbs}$ in 2005 (6). If primary vitreous traction from shaking does not cause primary eye findings, then eye findings observed in infants with encephalopathy must be secondary to other factors, such as increased intra-cranial pressure, increased intra-vascular pressure, hypoxia, or coagulopathy, none of which are specific for blunt force trauma. A brief summary of this recent literature has been posted on the AAP’s website as a P3R attached to the current Clinical Report from the AAP’s Section on Child Abuse and Neglect (7).
2. Primary brain injury was not demonstrated in motor vehicle accident reconstructions involving infants <6 months of age which produced resultant Gs of acceleration that greatly exceed what has been predicted to be achievable with an abusive shaking (8). Football players have also been shown to routinely sustain rotational head accelerations and resultant Gs of head acceleration that greatly exceed what has been predicted to be

achievable with an abusive shaking and do so without brain injury (9). If shaking cannot produce head accelerations even approaching the levels that human subjects have been shown to tolerate without brain injury, then the assertion that abusive shaking causes primary shear injury in the brain is severely undermined and such an assertion should no longer be accepted until valid experimental evidence can establish its validity. Other mechanisms must be responsible for brain swelling and injury when there is no history or evidence of impact. That the vast majority of autopsies in these cases of alleged abuse demonstrate only evidence of hypoxic/ischemic encephalopathy, further undermines the hypothesis that primary parenchymal shearing injury occurs in an alleged abusive shaking as a brain injuring mechanism.

3. Infants have sustained (8) and football players routinely sustain (9) accelerations substantially exceeding accelerations predicted to be achievable with an abusive shaking without incurring symptomatic subdural bleeding (or brain injury). In addition, the thin films of subdural bleeding that have been asserted as typical of SBS by the expired Case 2001 Position Paper (3), have recently been demonstrated on MRI in 46% of normal newborn infants (10). It must also be pointed out that retinal hemorrhages are present in a significant percentage of normal vaginally delivered newborns as well. Clearly, these newborn infants were not shaken and clearly these findings were the result of some other mechanisms that do not involve shaking or impact injury. One of the proposed mechanisms for these documented findings is “increased pressure during the labor process may augment the intracranial venous pressures” (10). Increased intracranial venous pressures can result from a numbers of medical and traumatic conditions.

In the light of this new research, the AAP has deemed it necessary to re-evaluate the validity of the shaken baby hypothesis and has given the task to members of the AAP Committee on Child Abuse and Neglect (11). These committee members have been provided with brief summaries of this newly published research with cited references (7,12). A new Clinical Report specifically addressing the significant new research of the last 5 years is due out in the Fall of 2008 (11).

In the light of this new research, the American Academy of Ophthalmology (AAO) could no longer sustain its previously held positions on eye findings and allegations of SBS, and in June of 2007, the AAO took down its Shaken Baby Resource Website for extensive revisions. Likewise, those responsible for this website have been provided with a summary of this new research with cited references (7).

The AAP has assigned the task of reevaluating the role, if any, of eye findings in unexpected infant deaths in view of the new experimental research challenging the vitreous traction hypothesis. This task has been assigned to its pediatric ophthalmology members (11). The lead pediatric ophthalmologist of this effort has also been made aware of the new research with cited references (7, 12).

Without valid experimental evidence, it can no longer be assumed that the unproven vitreous traction hypothesis is valid. This Clinical Report is due in the Spring of 2009.

Considering all of these emerging developments, I fail to see how Sauvageau can continue to argue that the Shaking Baby Syndrome (impulse-loaded rotational acceleration/deceleration without impact) as a valid primary cause of the entire TRIAD or even a

valid primary cause for any single feature of the TRIAD in a previously normal infant. Although Sauvageau et al. evidently believe in SBS, I have encountered very few pediatricians testifying in forensic cases over the last 2 years, still willing to state such a belief under oath. Increasingly they seek to disassociate themselves from the very term “shaken baby syndrome” and are unwilling to allow the unproven shaking mechanism to stand on its own merit. I suspect Sauvageau et al. may be the vocal representatives of a dwindling minority of physicians still willing to testify that pure shaking is a valid primary cause of the three components of the “triad.”

We will have to await the AAP's new Clinical Reports in the Fall of 2008 and Spring of 2009 to see if the shaken baby hypothesis is exposed as a flawed hypothesis or if its validity can be confirmed with quality evidence-based medical science to counter the growing body of evidence against it. If the shaken baby hypothesis is indeed flawed, then this fundamentally flawed hypothesis has been instrumental in the convictions of thousands of parents and caregivers and in the destruction of thousands of family units at an enormous social cost. If so, then let us hope that the AAP has the integrity and good conscience to actively undertake the righting of more than three decades of a grave injustice.

References

1. Sauvageau A, Bourgault A, Racette S. Cerebral traumatism with a play-ground rocking toy mimicking shaken baby syndrome. *J Forensic Sci* 2008;53(2):479–82.
2. American Academy of Pediatrics: Committee on Child Abuse and Neglect. Shaken baby syndrome: rotational cranial injuries-technical report. *Pediatrics* 2001;108(1):206–10.
3. Case ME, Graham MA, Handy TC, Jetzen JM, Monteleone JA. National Association of Medical Examiners Ad Hoc Committee on Shaken Baby Syndrome. Position paper on fatal abusive head injuries in infants and young children. *Am J Forensic Med Pathol* 2001;22(2):112–22.
4. Binenbaum G, Forbes BJ, Raghupathi R, Judkins A, Rorke L, Margulies SS. An animal model to study retinal hemorrhages in nonimpact brain injury. *JAAPOS* 2007;11:84–5.
5. Prange MT, Coats B, Duhaime AD, Margulies SS. Anthropomorphic simulations of falls, shakes, and inflicted impacts in infants. *J Neurosurg* 2003;99:143–50.
6. Commonwealth v Ann Power, 2005. Report to the Middlesex County District Attorney's Office Cambridge Massachusetts by Carole Jenny dated December 29, 2005.
7. Galaznik JG. Eye findings and allegations of shaking and nonaccidental injuries. August 8, 2007. P3R attached to Kellogg ND; Committee on Child Abuse and Neglect. Evaluation of suspected child physical abuse. (Clinical Report). *Pediatrics*. 2007. Available at: <http://www.pediatrics.org/cgi/eletter/119/6/1232>, Accessed on July 13, 2008.
8. Klinich KD, Hubert GM, Schneider LW. Estimating infant head injury criteria and impact response using crash reconstruction and finite element modeling. *Stapp Car Crash J* 2002;46:1–30.
9. Funk JR, Duma SM. Biomechanical risk estimates for mild traumatic brain injury. *Annu Proc Assoc Adv Automot Med* 2007;51:343–61.
10. Rooks VJ, Eaton JP, Ruess L, Peterman GW, Keck-Wherley J, Pedersen RC. Prevalence and evolution of intracranial hemorrhage in asymptomatic term infants. *AJNR Am J Neuroradiol* 2008;29:1082–89.
11. SCAN American Academy of Pediatrics Newsletter of the Section on Child Abuse and Neglect. *Am Acad Pediatr Newslett* 2007;19(4):9.
12. Galaznik J. Shaken baby syndrome. *Dev Med Child Neurol* 2008;50:317–20.

John Galaznik,^{1,*} M.D., FAAP

¹P.O. Box 342, Northport, AL 35476

E-mail: jgalaznik1@aol.com

*Statement on conflict of interest: I have given expert testimony in civil and criminal proceedings on cases involving allegations of physical abuse of infants.