Journal of Forensic and Legal Medicine 39 (2016) 46–49

Contents lists available at ScienceDirect

Journal of Forensic and Legal Medicine

journal homepage: www.elsevier.com/locate/jflm

Letter to the Editor

A response to Mimics of child abuse: Can choking explain abusive head trauma? [35 (2015) 33–37]

Keywords:
Shaken baby syndrome
Choking
Abusive head trauma
Retinal hemorrhages
Subdural bleeding
Hypoxic brain injury

A B S T R A C T

In the recently published article in this journal, "Mimics of Child Abuse: Can Choking Explain Abusive Head Trauma?",[1] the author chose to revisit a discussion prompted by a case report from 5 years ago which was inappropriate in his opinion. He went further to suggest that bringing an unvalidated mechanism of injury into the legal setting "obstructs justice", is a "further victimization of the child", and is a "travesty of justice".[1] Given the "Shaken Baby Syndrome: Rotational Cranial Injuries" has always been only an unvalidated hypothesis lacking experimental confirmation, the exploring of alternative injury mechanisms should be entirely appropriate. In 2010, the post publication discussion ended with a challenge to the American Academy of Pediatrics Committee on Child Abuse and Neglect (AAP COCAN) to either support the pure shaking mechanism with quality EBMS or eliminate any positive support for it from any official policy statement until the exact nature of each injury that pure abusive shaking has the potential to cause is clearly defined and supported with quality experimental research.[4] Since this is an area of acknowledged controversy by the AAP, it is appropriate to examine the evidence based experimental literature that has emerged over the last five years that is relevant to the abusive shaking hypothesis and the hypothesis of any primary brain-lethal hypoxic event leading to the findings of retinal hemorrhages, extra-axial bleeding, and brain injury when an infant presents to medical attention after an Acute/Apparent Life Threatening Event. In that light, this review was undertaken.

In the recently published article in this journal

"Mimics of Child Abuse: Can Choking Explain Abusive Head Trauma?"[1]

Dr. Edwards chose to revisit a discussion prompted by a case report from 5 years ago, entitled,

"Infant acute life-threatening event – Dysphagic choking versus nonaccidental injury",[2] which in his opinion was inappropriate. At the time it was originally published in 2010, it generated a "response by Dr. Greeley"[3] and "our response to Dr. Greeley".[4] In this published dialog, we made it clear that our purpose was not to re-try an old case, but stated that: "Single case reports are the lowest level of EBMS with the possible exception of opinion statements … Single case reports do not serve to establish absolute proof, change the entire concept of medicine … single case reports generally serve to bring previously unrecognized possibilities to public knowledge and promote dissemination of something potentially new … Our case is a single case report to promote thought and discussion/consideration."[4] In 2015, Dr. Edwards went further to suggest that bringing an unvalidated mechanism into the legal setting "obstructs justice", "further victimization of the child", and is a "travesty of justice".[1]

We would remind Dr. Edwards that while it is true that in 2001, the AAP COCAN presented "Shaken Baby Syndrome/Rotational Cranial Injuries" as settled science "no longer a diagnosis of exclusion" and guided pediatricians to a "presumption" of rotational cranial injuries from abusive shaking injury whenever the "constellation" of retinal hemorrhage and SDH/SAH and brain injury were encountered,[5] it has always been only a hypothesis lacking any experimental validation.

By May 1, 2009, this lack of validation that the levels of rotational acceleration/deceleration that would be predicted in an abusive shaking could actually cause primary brain injury, primary subdural hemorrhage, or primary retinal hemorrhages, forced the AAP COCAN to acknowledge that this unvalidated hypothesis of an injury mechanism, which had been presented as settled science up to 2009, was controversial to the point that legal challenges to the very term "Shaken Baby Syndrome" had become a distraction. "Legal challenges to the term "shaken baby syndrome" can distract from the more important questions of accountability of the perpetrator and/or the safety of the victim."[6] Furthermore the AAP COCAN 2009 Policy statement recommended dropping the term from any medical diagnosis and medical communications and re-defined the "constellation of findings", replacing retinal hemorrhages with spinal injury. "Pediatricians should use the term "abusive head trauma" rather than a term

DOI of original article: http://dx.doi.org/10.1016/j.jflm.2015.06.012.

http://dx.doi.org/10.1016/j.jflm.2016.01.006
1752-928X/

that implies a single injury mechanism, such as shaken baby syndrome, in their diagnosis and medical communications."[6] "However, for medical purposes, the American Academy of Pediatrics recommends adoption of the term "abusive head trauma" as the diagnosis used in the medical chart to describe the constellation of cerebral, spinal, and cranial injuries that result from inflicted head injury to infants and young children."[6] Furthermore, by 2010, Dr. Robert Block, president pro tem of the AAP at the National SBS Meeting in Atlanta, stated:

"Only people who are not active physicians working with children, naïve journalists, and professors with a biased agenda would propose that only three signs and symptoms support a diagnosis."

He went on to assert that,

"Valid Causation: Must Be Able to Demonstrate:

— Can Cause *[a particular finding]*
— Did Cause *[in this particular case]*

We closed our response in 2010 with a challenge to the AAP COCAN to "either support the pure shaking mechanism with quality EBMS or eliminate any positive support for it from any official policy statement until it can be definitively validated and the exact nature of each injury that it has the "potential to cause" is clearly defined and supported.".[4]

In that light, the question in 2015 is, not whether our article and response in 2010 was appropriate and worthy of publication, but rather: Has the recent literature been able to "demonstrate" that rotational cranial injury from abusive shaking is in fact a valid primary cause of each of the three findings - i.e. primary RH, primary SDH/SAH, and primary diffuse traumatic brain injury — that had been taken as a constellation of findings justifying a "presumption" of abusive shaking?

OR,

Has the recent literature supported the possibility that, if an infant were to experience a brain-lethal hypoxic event from an obstructive apnea or central apnea from any cause, then followed by successful cardiorespiratory resuscitation, could this infant present with a rapid onset of brain swelling, a hyperacute increase in the ICP, retinal hemorrhages, and thin films of subdural and subarachnoid blood?

To answer that question, one needs to examine the literature that has emerged since 2009.

FIRST one needs to look at the recent research into the potential for rotational cranial acceleration/deceleration from an abusive shaking to cause primary Traumatic Diffuse Axonal Injury (brain injury), primary retinal hemorrhages, and primary extra-axial bleeding.

The available biomechanical research with ATDs has indicated that the levels of rotational accel/decel that might be generated in the abusive shaking of an ~ 8lb infant and 17lb infant would be ~1434 rad/sec2 and 560 rad/sec2, respectively[7] and 480 rad/sec2 for a 24lb toddler.[8] Furthermore, the computer modeling data would indicate that any improvements in the neck design of the ATDs used in these experiments to make them more lifelike would only result in lower numbers.[9] While these levels of rotational accel/decel would not be predicted by the available animal research to be capable of causing concussion, retinal hemorrhages, and subdural/subarachnoid bleeding, the proponents of the abusive shaking hypothesis countered by saying that these are just "dummies" and "animals" — you can't shake babies and children. While it is true, you can't experimentally abusively shake babies and children, you can study infants and children at spontaneous play, and since 2009, there have been the publication of significant human infant/pediatric data. When one looks at this recent human experimental literature, one sees that infants and children at spontaneous play are "self-generating" repetitive cranial rotational accelerations/decelerations between 960 rad/sec2 and 1600 rad/sec2 and tolerating these levels of repetitive rotational accel/decel without clinically apparent brain injury, eye injury, or extra-axial bleeding.[10,11] Furthermore, young children engaged in contact sports are routinely tolerating up to 2200 rad/sec2 without signs of concussion and with some recorded events up to 7000 rad/sec2 without noted symptoms of concussion.[12]

In the area of retinal hemorrhages, animal studies specifically designed to test both the potential for massive levels of rotational acceleration/deceleration to 70—100 times what might be generated in an abusive shaking[13] and up to 5 total minutes abusively inflicted repetitive acceleration/deceleration,[14,15] have failed to produce grossly visible retinal hemorrhages that would be appreciated by an examining ophthalmologist. To be fair it should be pointed out that three of Finnie's 16 lambs died prior to planned sacrifice at 6 h. But given that these lambs, per protocol, were being monitored and supported with mechanical ventilation,[15] these deaths should not be attributed to any central apnea from brain, brainstem or cervical cord trauma. While the researchers did not specify a cause of these deaths, cardiac/circulatory failure from the chest trauma of 5 min of abusive shaking might be a likely explanation.

As for advances in our understanding of subdural and subarachnoid bleeding in these cases of alleged Rotational Cranial Injury from Abusive Shaking, prior to 2009, it was generally asserted that subdural bleeding was the result of trauma induced tensile stress failure of one or more bridging veins, and that the brain motion from the rotational accel/decel during an abusive shaking was adequate to result in such bridging vein tearing. However, with the experimental biomechanical data coupled with the human pediatric data indicating that the levels of cranial rotational accel/decel that were being spontaneously and repetitively self-generated while at play without observed clinical problems, the assertion that the 500—1500 rad/sec2 of cranial rotational deceleration that would be predicted in an abusive shaking would actually result in bridging vein tensile stress failure with subdural bleeding has not been supported.[16]

If the levels of rotational accel/decel that might be generated in an abusive shaking of an infant or toddler have not been "demonstrated" to be a valid "cause" (to echo Dr. Block) of primary RH, primary SDH/SAH, and primary diffuse traumatic cerebral injury, the question becomes, "What alternate pathophysiologies have emerged to account for these findings in infants and toddlers presenting to ERs and resuscitated from severe distress?"

In our 2010 article and response, we were raising for consideration the possibility that if an obstructive apneic event were prolonged to the point of resulting in a brain injuring hypoxic insult, followed by successful cardiorespiratory resuscitation, it could account for the TRIAD of findings of retinal hemorrhages, thin films of subdural/subarachnoid blood, and hypoxic brain injury.

In 2010, we were limited in our discussion to research that had been published, and we raised the possibilities of both primary injury mechanisms that might be transpiring during a choking event and cascading secondary pathophysiologies compounding and exacerbating any primary injuries. Each neuroimaging and each autopsy documented finding would not necessarily have to be limited to being a primary result, but could be a result of the cascade of secondary pathophysiologies set in motion by a hypoxic brain insult followed by successful cardiorespiratory resuscitation.

In the fatal cases of alleged rotational cranial injuries from abusive shaking, the neuropathologists generally report global hypoxic/ischemic encephalopathy. Hence, the immediate cause of the ALTE would most logically be a primary brain-lethal hypoxic event. A prolonged obstructive apnea would potentially be a possible cause of a prolonged apnea. Rotational cerebral injury, as in a