Check for updates

Shaken Infants Die of Neck Trauma, Not of Brain Trauma

Evan W. Matshes MD FRCPC, Rhian M. Evans PhD, J. Keith Pinckard MD PhD, Jeffrey T. Joseph MD PhD, Emma O. Lew MD

ABSTRACT

Shaken baby syndrome (SBS) is routinely diagnosed on the basis of a classic triad of autopsy findings, namely retinal hemorrhage, subdural hemorrhage, and anoxic encephalopathy. However, ongoing controversy exists regarding the specificity and potential causes of these signs, and hence their reliability as de facto markers of SBS, or of non-accidental head injury, where no external signs of trauma are evident.

We investigated the deaths of 35 infants and young children, which fell into two broad groups: those with suspected hyperflexion/extension neck injuries, and those without. At autopsy, the entire cervical spinal column (spinal cord, vertebrae, intervertebral discs, neurovascular structures and adjacent soft tissues) was removed, formalin-fixed, decalcified, dissected, and microscopically evaluated.

Of the 12 cases in which hyperflexion/extension was either suspected or confirmed, all had evidence of either bilateral or unilateral hemorrhages within or surrounding the C3, C4, and/or C5 cervical spinal nerve roots.

We provide evidence that hyperflexion/extension forces as experienced by shaken and impacted infants and young children lead to injury of the cervical spinal nerve roots that innervate the diaphragm, with resulting asphyxia and hypoxic brain injury. Therefore, we propose that trauma to the third through fifth cervical spinal nerve roots induced by hyperflexion/extension of the neck is the cause of the anoxic encephalopathy of the classic SBS triad, and is therefore not only a more specific indicator of hyperflexion/extension injury than subdural hemorrhage alone, but is the mechanism of injury in these cases.

KEYWORDS: Forensic pathology, Neuropathology, Shaken baby syndrome, Brain injuries

Evan Matshes, MD FRCPC is Assistant Chief Medical Examiner for the Province of Alberta (Calgary, Canada).
Author affiliations: Academic Forensic Pathology Incorporated, Calgary, AB (RE), Southwestern Institute of Forensic Sciences, Dallas, TX (KP), University of Calgary and Calgary Laboratory Services - Division of Neuropathology, Calgary, Alberta (JJ), Miami-Dade County Medical Examiner Department, Miami, FL (EL).
Contact Dr. Matshes at: matshes@academicfp.com.
Acad Forensic Pathol
2011 1 (1): 82-91
https://doi.org/10.23907/2011.009

© 2017
Academic Forensic Pathology International

INTRODUCTION

Shaken baby syndrome was first described by Guthkelch in 1971 (1) , and later by John Caffey (2, 3), who used the term "whiplash shaken infant syndrome" to describe the cause of subdural hemorrhage (SDH) in infants with no external signs of head trauma. Subsequently coined as Shaken Baby Syndrome (SBS), the condition has gained such prominence in the public consciousness that the US Senate has in recent years unanimously passed a resolution declaring the third week in April "National Shaken Baby Syndrome Awareness Week."

However, in the forensic and legal communities, there is ongoing controversy over the definition, diagnosis, and even the very existence of SBS. SBS is commonly characterized by a combination of three clinico-radiologic or autopsy signs – retinal hemorrhage, SDH (usually a thin film of hemorrhage rather than a space-occupying lesion) and anoxic encephalopathy (AE) – the "triad" (4). These injuries are postulated to be the direct result of the acceleration/deceleration forces to which the infant skull and brain would be subjected during shaking. Although the significance of the triad should be taken in the context of patient history before abuse is considered, sev-

eral high profile cases illustrate that the dogma of the triad as pathognomonic of abuse persists, despite a growing body of evidence questioning its specificity. In particular, the causative mechanism of SDH in infants has been the subject of significant debate (5-7).

SDH was first described in the context of abuse by Guthkelch (1), who noted that infants' "relatively large head and puny neck muscles" make them particularly vulnerable to acceleration/deceleration forces. Caffey (2) went on to describe cases of "whiplash shaking" leading to bilateral SDH as the cause of death, with both Guthkelch and Caffey noting torn dural bridging veins in several of the cases they reported. Support for the hypothesis of dural bridging vein rupture by acute acceleration/deceleration forces as the source of SDH came from Yamashima and Friede in 1984 (8). The encephalopathy component of the triad has commonly been assumed to occur either as a result of the subdural bleeding (9, 10), or as a direct consequence of mechanical injury (11). This theory remained largely unchallenged until Geddes et al (12-14) put forward what came to be known as the "unified hypothesis". They observed that severe axonal damage is rare in infants, and proposed that hypoxia, its associated encephalopathy, and increased central venous pressure, rather than bridging vein rupture, are responsible for the SDH and retinal hemorrhages in these cases. This caused considerable controversy (15, 16), and the credibility of the hypothesis was tested during the Goldsmith Review when Dr. Geddes, under cross-examination as an expert witness, conceded that the theory was intended to stimulate academic debate rather than be taken as fact (17, 18).

In both the UK (Goldsmith Review, 2006) and Canada (Goudge Inquiry, 2008) (18), large scale judicial reviews have resulted in a significant number of SBS cases being revisited and/or overturned. These recommendations for review were based on both committees' conclusions that, in light of growing scientific evidence, the presence of the triad alone can not and should not be automatically diagnostic of inflicted trauma.

We present evidence that infant head trauma, by shaking and/or by blunt impact, causes hyperflexion/extension injury to the cervical nerve roots, thereby disrupting innervation of the diaphragm and leading to asphyxia and AE. SDH occurs either as a result of the AE or as a direct result of the blunt trauma. We further suggest that in infants and young children, nerve root damage at levels C3 through C5 is a more specific marker of hyperflexion/extension injuries sustained due to shaking and other forms of indirect neck trauma that accompany head impact.

MATERIALS AND METHODS

Thirty-five non-sequential infants and young children were autopsied in three different Medical Examiner jurisdictions. Cervical spines (including the skull base and proximal thoracic spine) were removed following the technique of Matshes and Joseph (19). After removal of the brain, the anterior, posterior, and lateral soft tissue compartments of the neck were dissected, the carotid arteries were lifted from the carotid sheaths, and the cervical spine was separated from the body. Each spine was then fixed in 10% formalin for at least two weeks, and then decalcified in 18% formic acid solution for at least 10 days. Following decalcification, each spine was serially sectioned in 3 to 5 millimeter-thick slices, and histologically examined on at least 8 levels. These levels always included at least C3, C4 and C5, one section proximal to C3, and one section distal to C5. The tissue blocks were then processed as per routine histologic methods. Three consecutive levels were then cut on a microtome; the first two levels were stained with hematoxylin and eosin and the third was stained with an iron stain (Perl's Prussian blue).

RESULTS

Thirty-five autopsies were performed on infants and young children ranging from newborn to 36-months-of-age (median age 3.5 months; see Table 1). Of these, 12 were either confirmed or suspected by history and circumstances to have been subjected to hyperextension and hyperflexion forces, either as a result of accident or homicide. In the remaining 23 cases, the largest group of deaths were asphyxial (co-sleeping/overlay; 11), followed by sudden unexplained deaths (SUID; 8), 2 cases of blunt abdominal trauma, 1 drowning, and 1 death attributed to the septic complications of pneumonia.

Figure 1 provides a schematic of the areas under examination, while Images 1 and 2 compare the macroscopic and microscopic presentation of vertebrae assessed as normal (Images 1A and 1B) and those with evidence of nerve root damage (Images 2A and 2B).

Of the 12 hyperflexion/extension cases, all showed either unilateral or bilateral intraneural and/or perineural hemorrhage involving the nerve roots at spinal levels C3 to C5. Of the 23 cases without suspected hyperflexion/extension, only 1 showed any sign of nerve damage ($p<0.001$; case 14 described below). In this series, the sensitivity and specificity of cervical spinal nerve root damage for hyperflexion/extension injury were 100% and 96%, respectively. The positive and nega-

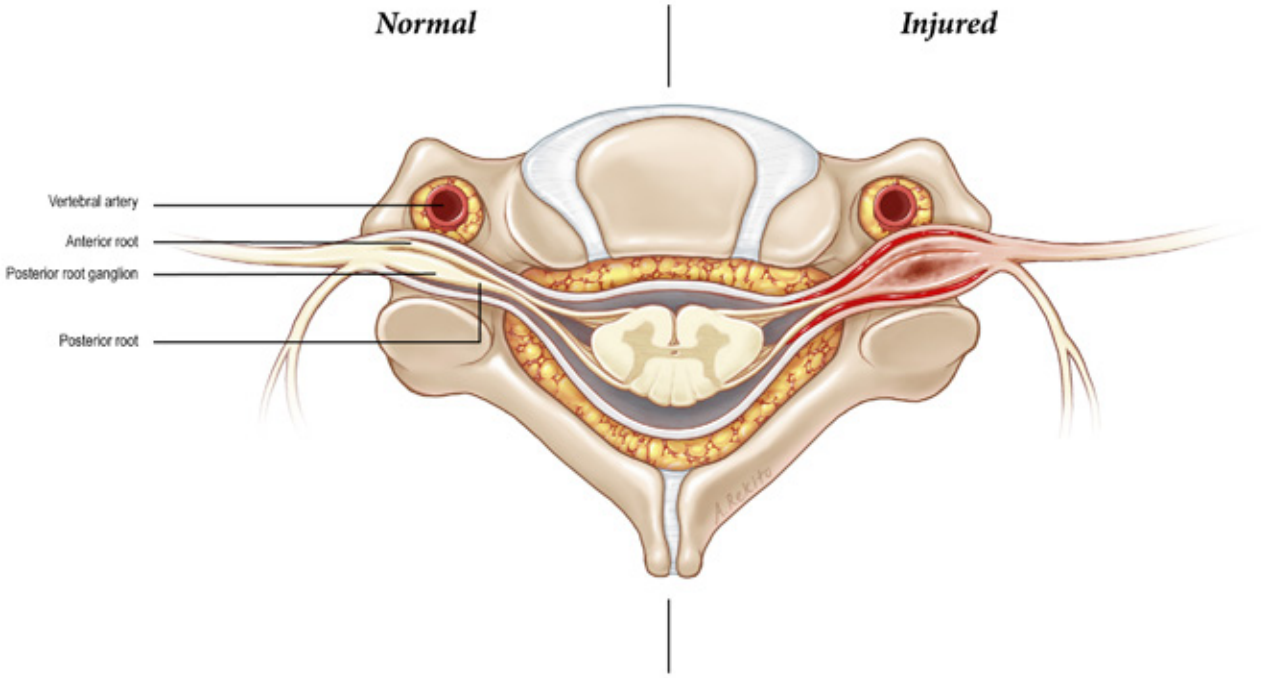

FIGURE 1: Illustrative axial section of the cervical spine, showing the spinal cord and nerve roots. The left side shows normal anatomy, and the right side shows perineural and intraneural hemorrhage.

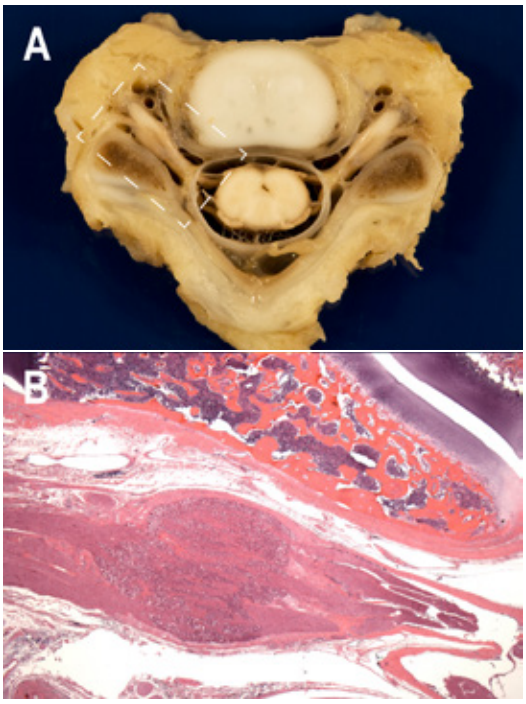

IMAGE 1: Macroscopic (A) and microscopic (B) examples of cervical spine sections. The examples in this figure are normal, with no evidence of injury, and were removed from an infant not suspected to have been subjected to hyperflexion/extension forces. The photomicrograph of an H&E-stained section was taken with a 10X objective, and features the proximal-most segment of a cervical spinal nerve root. The rootlets and spinal dura mater are visible in the bottom right quadrant of the image. A dash-line white box outlines the region of the specimen featured in the photomicrograph.

tive predictive values were 92% and 100%. For each case, sections proximal to C3 and distal to C5 were also examined. Approximately 50% of cases had injury at C2 (5 of 12 cases) and/or C6 (6 of 12 cases), with only 4 of 12 having damage more distally, at C7, indicating that the focus of the insult is spinal levels C3 through C5. Stainable iron was not detected in any case. The C3 through C5 regions of the cervical spinal cords were atraumatic in all cases.

There was also a relationship in our series between nerve root damage and AE. Of the 13 cases with nerve root damage, 9 (69%) also showed signs of AE at autopsy. In contrast, of the 22 cases without nerve root damage, only 4 (18%) showed any evidence of AE ($p<0.01$).

The morphologic changes of AE are dependent upon post-incident survival. Of our total series, the 13 cases with AE had a mean post-incident survival of 2.7 (± 0.9) days, while those without AE had a mean post-incident survival of just 0.01 (± 0.01) days ($p<0.001$). Of note, only 1 non-AE case survived post-incident for 4 hours.

SDH was evident in all 12 of the hyperflexion/extension cases (Table 1), and 2 of the remaining 23 ($p<0.001$) (one of which was case 14).

CASE STUDIES

CASE 14 was a 3-month-old male infant who was under the care of his biologic father when he was found unconscious. The father could not offer a reliable account of the infant's pre-arrest condition. Emergency medical services arrived and commenced CPR. Following successful restitution of a perfusing rhythm at the hospital, the infant was diagnosed with SBS based on the presence of AE and retinal hemorrhages. He died one week later. Extensive liquefaction of the brain (respirator brain) was found at autopsy. Fragments of subdural blood clot were seen grossly. Small, punctate retinal hemorrhages were along the posterior pole of one eye. Microscopically, unilateral intraneural hemorrhages were in the C3 and C4 nerve roots, and unilateral perineural hemorrhages were around the C3 nerve root. The clinical and pathological information available was not sufficient to support a diagnosis of inflicted head trauma; the cause and manner of death were ruled undetermined.

CASE 32 was a 3-month-old female fraternal twin under the care of her biologic mother. The baby had been feverish and 'grunty' for several days, prompting a visit to her primary care physician who diagnosed a mild 'respiratory illness'. Two days later the baby deteriorated and mom noticed blue lips. Emergency medical services found the infant apneic and pulseless. Resuscitative efforts successfully restored a perfusing rhythm and the baby was admitted to the intensive care unit. Over the course of the five-hour admission, features of shock with probable sepsis were manifested; the baby had a sudden cardiac arrest and resuscitative efforts were unsuccessful.

Autopsy demonstrated an ill-looking baby girl with features of shock and sepsis. The lungs had patchy hemorrhagic atelectasis and the upper respiratory tract had focally erythematous mucosa. A thin liquid, focally gelatinous SDH was over both cerebral convexities. The brain had macro- and microscopic features of AE. Retinal hemorrhages were focal. The cervical spinal nerve roots were atraumatic.

Microscopically, the lungs showed extensive angio- and bronchial-invasive candida species. The remaining fraternal twin was healthy and uninjured, but the biologic mom was 'unwell' and had features of possible immunosuppressive illness.

The cause of death was ruled septic complications of fungal pneumonia, and the manner of death was ruled natural.

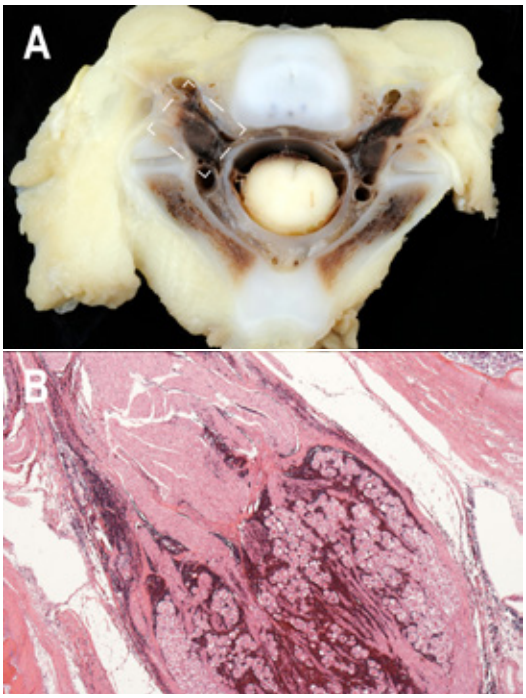

IMAGE 2: Macroscopic (A) and microscopic (B) examples of cervical spine sections removed from an infant subjected to hyper-flexion/extension forces, in which the perpetrator admitted to shaking without admission of, or evidence for impact. Hemorrhage is clearly visible bilaterally within the ganglia and nerve roots. The photomicrograph of an H&E-stained section was taken with a 20X objective, and demonstrates blood dissecting throughout the ganglia and the more distal segment of the nerve root. A dash-line white box outlines the region of the specimen featured in the photomicrograph.

Ten of 12 of the former also had an impact site. While the concurrent presentation of an impact site complicates interpretation of the data in the context of SDH, significant blows to an unsupported head could result in severe hyperflexion/extension of the cervical spine with subsequent nerve root injury. One infant (case 32) found to have a thin SDH in the context of profound fungal sepsis had atraumatic nerve roots. The presence of SDH in the context of cervical nerve root damage is discussed below.

Eyes were examined in only 9 of 12 hyperflexion/extension cases, and 4 of 23 of the other cases (including the seven cases with suspected or admitted shaking; data not shown). Of the hyperflexion/extension cases examined, 8 of 9 had evidence of retinal hemorrhage, as did 2 of 4 other cases where the eyes were examined.

Together, these data support the development of AE as a consequence of survival after a primary asphyxial event – an event that can be attributed to cervical spinal trauma with injury to the nerve roots innervating the diaphragm.

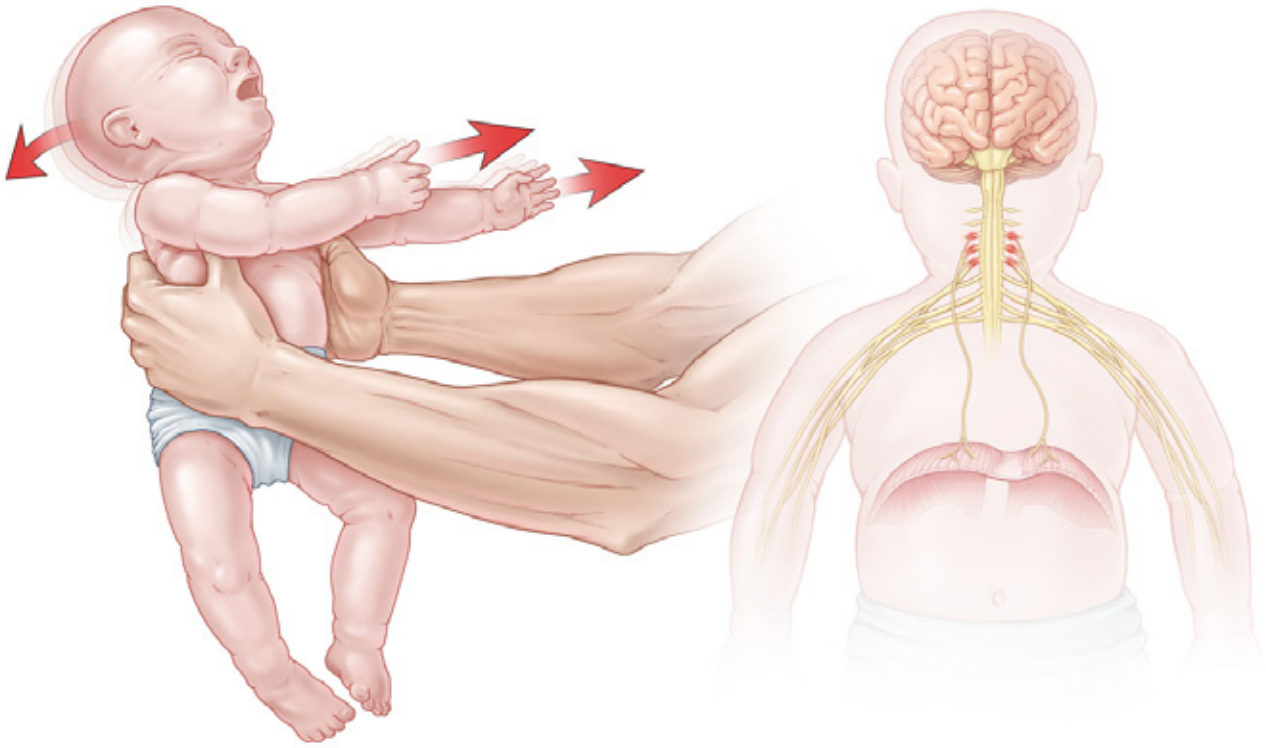

FIGURE 2: Illustration of the forces an infant is subjected to when shaken. A - The size and weight of the head combined with the lack of developed neck musculature is expected to lead to alternating hyperflexion and hyperextension of the cervical spine. B – Cervical spine anatomy, showing levels C3, C4 and C5 highlighted, with nerve roots innervating the diaphragm.

DISCUSSION

Our data provide a mechanism to explain how isolated shaking (without impact) might result in AE with subsequent death, and provide evidence for a causative mechanism. We suggest that the hyperflexion and hyperextension of the cervical spine occurring from either shaking, severe blunt impact, or both (Figure 2) results in physical injury of the nerve roots at C3, C4 and/or C5 that disrupts innervation of the diaphragm and severely compromises respiration. Thus, the primary insult to the brain is anoxic, rather than mechanical.

Support for shaking that leads to cervical spine damage comes from a number of studies. The initial reports by Guthkelch and Caffey (1, 2) both focused on "whiplash" stresses and pointed out the size and weight of the infant head combined with lack of cervical musculature (in the absence of external signs of neck injury) concentrated on the effects of such forces on the brain rather than on the spinal cord. Caffey's whiplash hypothesis was based on biomechanical studies that used a rhesus monkey model (20, 21). The cervical musculature in rhesus monkeys is comparatively well developed and the head is relatively smaller than the head of human infants. In Ommaya's studies, nearly half the monkeys showed signs of cervical spinal injury. Bohn (22) showed that infants and children may suffer damage to the cervical spinal cord resulting in apnea or cardiorespiratory arrest, without displaying external or radiological signs of bony injury to the area. It has been suggested (23, 24) that the occurrence of cervical spinal cord injuries in child abuse cases is underreported, and potentially easy to overlook in patients presenting with concomitant head injury and no external signs of spinal injury. Further biomechanical investigation (25) has demonstrated that cervical spinal cord injury can occur at much lower velocities and accelerations than are typically reported for SBS. Indeed, numerous studies (26-29) found evidence for damage to the cervical spinal cord in SBS cases. Our data unequivocally support these findings, with clear evidence of bleeding in or around the nerve roots at C3, 4 and 5 in all 12 cases where hyperflexion/extension was suspected or confirmed (Table 1). The one case that had nerve root injury without historical features to support hyperflexion/extension (case 14) was classified as undetermined in cause and manner because of insufficient total case data in this objectively suspicious case.

The nerve roots at spinal levels C3, 4 and 5 innervate the diaphragm – "C 3, 4, 5, keeps the dia-

phragm alive" (commonly cited medical school mnemonic). In infants (obligate diaphragm breathers), damage to these nerve roots could induce a period of diaphragmatic compromise with concomitant respiratory paralysis. The predicted neuropathological outcome would be hypoxic-ischemic damage to the brain with subsequent AE. Again, our data support this hypothesis, with AE being reported in 8 of the 12 cases (66%) showing nerve root damage. It is noteworthy that of the 4 cases with nerve root damage but without AE, 3 did not survive the initial incident, and one survived only 4 hours, thus the absence of AE is not surprising. In contrast, the 8 cases with AE survived between 12 hours and 6 days post-injury.

Encephalopathy observed as part of the triad was previously assumed to occur as a result of either SDH or, more likely, acute and diffuse axonal injury from the same shearing forces thought to rupture dural bridging veins. Case views diffuse axonal injury as an inevitable feature of SBS (11, 30, 31), and Gleckman (32) identified axonal injury in 5 out 7 SBS infants examined. However, other groups found that most abusive head trauma cases did not show signs of diffuse axonal injury, but rather, of widespread hypoxic injury (12, 33), leading some to conclude that it is altered perfusion rather than mechanical trauma that leads to encephalopathy (27). These findings were supported by radiological evidence (29, 34, 35). It has also been suggested that under some circumstances, particularly where patients are resuscitated and/or ventilated, axonal injury can occur secondarily to hypoxic-ischemic damage (36). Talbert (37) goes so far as to assert that SBS, as currently defined, cannot be due to shaking if there are no traumatic axonal injuries, but concedes that the described condition is consistent with an episode of apnea. There is evidence that both SDH and retinal hemorrhage can be induced by apnea following paroxysmal coughing (38, 39). Our data support Talbert's conclusion that apnea is an important factor in the described syndrome. We propose that apnea can occur directly as a result of shaking through diaphragmatic paralysis.

One of the contentious issues surrounding shaken baby syndrome is whether shaking forces alone, without impact, are sufficient to cause the injuries. Animal models and dummies of varying designs suggest that shaking is insufficient to produce either axonal injury or SDH (40-43). In fact, adult volunteers shaking dummies were physically unable to replicate the forces thought to be required to cause SDH by shaking alone, assuming of course that the necessary forces predicted by the models are accurate. The picture is further complicated by pure shaking cases being relatively rare – as in our series, shaken infants often present with a concurrent impact. Recently, research using a finite element model of an infant brain purported to demonstrate that anteroposterior shaking could lead to predictable patterns of "brain matter motion" with subsequent focal axonal injury (44). However, this does not address the inability of most pathologists to demonstrate traumatic axonal injuries in cases of actual infant injury and death.

Although the effect of whiplash forces on the cervical nerve roots has been largely overlooked, with investigators concentrating on the effects of such forces in the brain, it seems reasonable that any infant shaken violently enough to induce SDH would be expected to also suffer neck trauma (12, 25). Opponents of SBS have often based their arguments on the absence of neck injuries (45). We contend that up until now, 'neck injuries' have not been seen, not because they were not present, but rather because the appropriate anatomical structures were not dissected. Unlike other authors who also undertook detailed studies of infants in an effort to understand the nature of inflicted head trauma (46), our definition of the 'neck' was not limited to the spinal cord. Instead, we evaluated the entire cervical spine including the osseous and neurovascular structures, and the associated deep neck soft tissues. Of note, injuries to the cervical nerve roots are not visible with routine anterior and posterior neck dissection, nor with standard removal of the spinal cord (Figure 3). One group of authors did

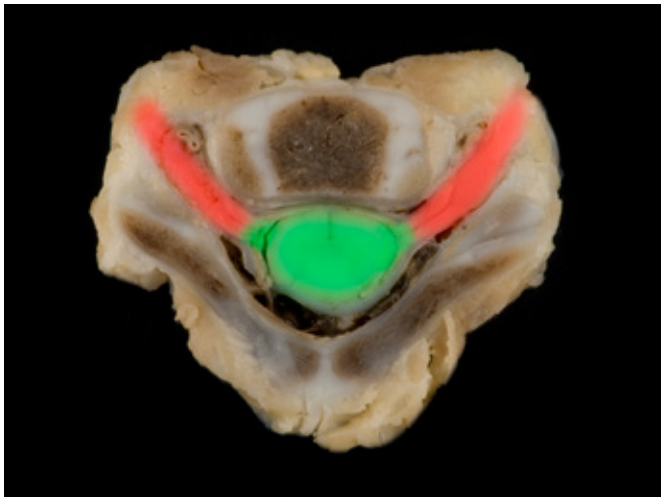

FIGURE 3: This schematic illustrates those neurologic structures accessible with routine anterior or posterior spinal cord removal (green), and those which can only be examined through the technique utilized in this study (red). Note that only the proximal rootlets are examined through routine dissection techniques (removal of the spinal cord only).

thorough neuropathologic studies of injured and uninjured infants (47), including resections of the cervical spine, but did not evaluate the spine and nerve roots in a fashion that would have facilitated as thorough an evaluation of the nerve roots. Despite this, they concluded that "the process of death [in the Shaken Baby Syndrome] is primarily caused by a shaking trauma that triggers a hypoxic–ischemic process as the final and deadly event." Their research did not detect, and therefore did not appreciate the role of the cervical spinal nerve root trauma in causing diaphragmatic paralysis and thus anoxia. By implicating nerve root trauma with subsequent paralysis of the diaphragm as the proximate cause of the anoxic brain injury in these cases, we close a previously unexplained loop in the understanding of shaking-related trauma.

How do we account for the finding of SDH, if indeed it is a secondary neuropathological event rather than a result of blunt trauma? One would expect that rupture of a bridging vein would result in larger, more often unilateral, space occupying SDH, rather than the classically described thin, bilateral, non-space occupying hemorrhages identified in shaking and impact head trauma cases. Squier and Mack instead implicate the intradural vascular plexus as a likely source of subdural collections (7). These valveless vessels are far more extensive in infants than in older individuals (6). A combination of increased intravascular pressure and hypoxic damage to the endothelium of meningeal vessels may lead to extravasation of blood into the subdural space.

Our work is a refinement of the Geddes hypothesis, which indicated SDH as a result of rapid changes in central venous and intracranial pressures (13). One of the objections to this theory (15, 16) was the lack of evidence for SDH in cases such as drowning or strangulation, in which rapid changes in pressure would be expected. The mechanism proposed here addresses some of those concerns. In the examples given in Richards' work (16), victims would rarely be expected to survive long enough for SDH induced by AE to develop. This may provide an explanation for findings of SDH in some non-trauma cases, as well as in SBS cases presenting without impact. Our data support this, with only 4 of 35 cases showing evidence of SDH in the absence of AE; in each of these 4 cases, blunt head trauma had occurred and an impact site was present.

CONCLUSION

We submit that cervical nerve root damage is the primary injury in hyperflexion/extension whiplash forces on the infant neck. Cervical nerve root damage disrupts innervation of the diaphragm and results in asphyxia, which if survived, will progress to AE. SDH may result directly from impact trauma, or be secondary to complications of anoxia. We therefore conclude that whiplash forces, either as pure shaking events, or with concurrent impact, can be fatal. Our clinicopathologic evidence explains how infants die from shaking.

Table 1: Thirty-five cases of infant and young child death (newborn to 36-months-of-age). Cases 1 through 12 had either confirmed or suspected hyperflexion/extension, while the remainder of the table describes cases without suspected hyperflexion/extension.

#	Age*	COD (MOD)	Circumstances	Impact site	Hyper-Flexion/ Extension	Post-Injury Survival	C3	C4	C5	AE	SDH
1	5	Blunt head trauma (H)	Abused	Y	Y -suspected	6 days	Uni pn	Uni pn in	Uni pn	Y	Y
2	1	Blunt trauma (H)	Abused	Y	Y -suspected	0.5 days	Bil pn	Bil pn, uni in	Uni pn	Y	Y
3	36	Blunt trauma (H)	Abused	Y	Y - suspected	4 hours	Pn	Pn	pn	N	Y
4	7	Blunt trauma (A)	Unrestrained passenger in MVC	Y	Y	1 day	Bil pn, in	Bil pn, in	Bil pn, in	Y	Y
5	12	Blunt trauma (A)	Pedestrian struck by car	Y	Y	0	Uni pn	Uni pn	Uni pn	N	Y
6	2	Blunt head trauma (H)	Abused	N	Y - admitted	3 days	Bil pn, in	Bil pn, in	Bil pn, in	Y	Y
7	12	Blunt head trauma (H)	Abused	Y	Y - suspected	3 days	Bil in	Bil in	Bil pn, uni in	Y	Y
8	0	Blunt trauma (A)	Unrestrained passenger in MVC	Y	Y	0	Bil pn	Bil pn	Uni in	N	Y
9	16	Blunt trauma (A)	Unrestrained passenger in MVC	Y	Y	0	Bil pn	Bil pn	bil pn	N	Y

Table 1: Continued

#	Age*	COD (MOD)	Circumstances	Impact site	Hyper-Flexion/ Extension	Post-Injury Survival	C3	C4	C5	AE	SDH
10	7	Blunt trauma (A)	Unrestrained passenger in MVC	Y	Y	1 day	Bil pn, in	Bil pn, in	Bil pn, uni in	Y	Y
11	0.5	Blunt head trauma (H)	Abused	Y	Y -suspected	3 days	Bil pn, in	Bil pn, in	Bil pn, uni in	Y	Y
12	4	Blunt head trauma (H)	Abused	N	Y- admitted	0.5 days	Bil pn, in	Bil pn, in	Bil pn, uni in	Y	Y
13	3	Sudden Unexplained (U)	History unknown due to inconsistent caregiver accounts	N	Unknown	3 days	-	-	-	Y	N
14	3	Undetermined (U)	History unknown due to inconsistent caregiver accounts	N	Unknown	7 days	Uni pn uni in	Uni in	-	Y	Y
15	12	Drowning (A)	Fell into backyard pool while unsupervised	N	N	12 days	-	-	-	Y	N
16	2.5	Sudden Unexplained (U)	Co-sleeping; parent denied overlay	N	N	0	-	-	-	Y	N
17	1.5	Asphyxia (A)	Cosleeping	N	N	0	-	-	-	N	N
18	0.75	Asphyxia (A)	Overlay	N	N	0	-	-	-	N	N
19	6	Asphyxia (A)	Positional asphyxia	N	N	0	-	-	-	N	N
20	1	Sudden Unexplained (U)	Sudden death while sleeping; scene, circumstance and all studies negative	N	N	0	-	-	-	N	N
21	2	Sudden Unexplained (U)	Co-sleeping; parent denied overlay	N	N	0	-	-	-	N	N
22	2	Asphyxia (A)	Wedging	N	N	0	-	-	-	N	N
23	2	Sudden Unexplained (U)	Unsafe sleep environment	N	N	0	-	-	-	N	N
24	6	Asphyxia (A)	Smothering	N	N	0	-	-	-	N	N
25	4	Sudden Unexplained (U)	Unsafe sleep environment	N	N	0	-	-	-	N	N
26	4	Asphyxia (A)	Suffocation	N	N	0	-	-	-	N	N
27	2	Sudden Unexplained (U)	Co-sleeping; parent denied overlay	N	N	0	-	-	-	N	N
28	4	Asphyxia (A)	Overlay	N	N	0	-	-	-	N	N
29	3.5	Asphyxia (A)	Cosleeping	N	N	0	-	-	-	N	N
30	13	Blunt Abdominal Trauma (H)	Abuse	Y	N	Unknown	-	-	-	N	N
31	1	Asphyxia (A)	Overlay	N	N	0	-	-	-	N	N
32	3	Sequelae of pneumonia (N)	Recent respiratory illness, features of shock and sepsis during ICU admission	N	N	5 hours	-	-	-	Y	Y
33	7	Blunt Abdominal Trauma (H)	Abuse	N	N	Unknown	-	-	-	N	N
34	5	Asphyxia (A)	Wedging	N	N	0	-	-	-	N	N
35	4	Asphyxia (A)	Overlay	N	N	N	-	-	-	N	N

Key: COD = cause of death, MOD = manner of death, MVC – motor vehicle collision, H = homicide, A = accidental, U = unknown, N = natural death. Y = yes, an impact site was observed at autopsy; N = no evidence of an impact site at autopsy. Columns C3 – C5 describe the extent of damage found at each spinal level: bil = bilateral, uni = unilateral, pn = perineural, in = intraneural, - = negative. AE = Anoxic encephalopathy; SDH = subdural hemorrhage. * Ages are represented in months.

ACKNOWLEDGEMENTS

The authors would like to thank the professional and support staff of the University of Texas Southwestern Medical Center at Dallas, Miami-Dade County Medical Examiner Department and the Office of the Chief Medical Examiner in Calgary. Talitha Thomas volunteered many hours in order to assist with the collection of data during the evaluation of cervical spines. High quality histologic preparations were produced by the Department of Neuropathology at the University of Texas Southwestern Medical Center at Dallas, by the neuropathology laboratory of Calgary Laboratory Services at Foothills Medical Center, and by Tissue Techniques Pathology Labs LLC in Dallas, Texas. Illustrations were prepared by Andrew Rekito (Rekito Visuals) with the financial support of Academic Forensic Pathology Incorporated and the University of Calgary (illustrations are copyright Academic Forensic Pathology Incorporated). Dr. Jeffrey Barnard, Dallas County Chief Medical Examiner, deserves special mention for his guidance and continuous support throughout this project.

REFERENCES

1) Guthkelch AN. Infantile subdural haematoma and its relationship to whiplash injuries. *Br Med J* 1971 May 22; 2(5759):430-1.
2) Caffey J. The whiplash shaken infant syndrome: manual shaking by the extremities with whiplash-induced intracranial and intraocular bleedings, linked with residual permanent brain damage and mental retardation. *Pediatrics* 1974 Oct; 54(4):396-403.
3) Caffey J. On the theory and practice of shaking infants. Its potential residual effects of permanent brain damage and mental retardation. *Am J Dis Child* 1972 Aug; 124(2):161-9.
4) Blumenthal I. Shaken baby syndrome. *Postgrad Med J* 2002 Dec; 78(926):732-5.
5) Squier W. Shaken baby syndrome: the quest for evidence. *Dev Med Child Neurol* 2008 Jan; 50(1):10-4.
6) Squier W, Lindberg E, Mack J, Darby S. Demonstration of fluid channels in human dura and their relationship to age and intradural bleeding. *Childs Nerv Syst* 2009 Aug; 25(8):925-31.
7) Squier W, Mack J. The neuropathology of infant subdural haemorrhage. *Forensic Sci Int* 2009 May 30; 187(1-3):6-13.
8) Yamashima T, Friede RL. Why do bridging veins rupture into the virtual subdural space? *J Neurol Neurosurg Psychiatry* 1984 Feb; 47(2):121-7.
9) Nooraudah AR, Mohd Sham K, Zahari N, Fauziah K. Non-accidental fatal head injury in small children--a clinico-pathological correlation. *Med J Malaysia* 2004 Jun; 59(2):160-5.
10) David TJ. Shaken baby (shaken impact) syndrome: non-accidental head injury in infancy. *J R Soc Med* 1999 Nov; 92(11):556-61.
11) Case ME. Abusive head injuries in infants and young children. *Leg Med (Tokyo)* 2007 Mar; 9(2):83-7.
12) Geddes JF, Hackshaw AK, Vowles GH, Nickols CD, Whitwell HL. Neuropathology of inflicted head injury in children. I. Patterns of brain damage. *Brain* 2001 Jul; 124(Pt 7):1290-8.
13) Geddes JF, Tasker RC, Hackshaw AK, Nickols CD, Adams GG, Whitwell HL, Scheimberg I. Dural haemorrhage in non-traumatic infant deaths: does it explain the bleeding in 'shaken baby syndrome'? *Neuropathol Appl Neurobiol* 2003 Feb; 29(1):14-22.
14) Geddes JF, Vowles GH, Hackshaw AK, Nickols CD, Scott IS, Whitwell HL. Neuropathology of inflicted head injury in children. II. Microscopic brain injury in infants. *Brain* 2001 Jul; 124(Pt 7):1299-306.
15) Punt J, Bonshek RE, Jaspan T, McConachie NS, Punt N, Ratcliffe JM. The 'unified hypothesis' of Geddes et al. is not supported by the data. *Pediatr Rehabil* 2004 Jul-Sep; 7(3):173-84.
16) Richards PG, Bertocci GE, Bonshek RE, Giangrande PL, Gregson RM, Jaspan T, Jenny C, Klein N, Lawler W, Peters M, Rorke-Adams LB, Vyas H, Wade A. Shaken baby syndrome. *Arch Dis Child* 2006 Mar; 91(3):205-6.
17) R v Harris and others. EWCA [2005] 1980.
18) Goudge ST. Inquiry into in pediatric forensic pathology in Ontario. Toronto 2008.
19) Matshes E, Joseph J. Pathologic evaluation of the cervical spine following neurosurgical or chiropractic interventions. *Journal of Forensic Sciences* 2010; In press.
20) Ommaya AK, Faas F, Yarnell P. Whiplash injury and brain damage: an experimental study. *JAMA* 1968 Apr 22; 204(4):285-9.
21) Ommaya AK, Yarnell P. Subdural haematoma after whiplash injury. *Lancet* 1969 Aug 2; 2(7614):237-9.
22) Bohn D, Armstrong D, Becker L, Humphreys R. Cervical spine injuries in children. *J Trauma* 1990 Apr; 30(4):463-9.
23) Feldman KW, Weinberger E, Milstein JM, Fligner CL. Cervical spine MRI in abused infants. *Child Abuse Negl* 1997 Feb; 21(2):199-205.
24) Ghatan S, Ellenbogen RG. Pediatric spine and spinal cord injury after inflicted trauma. *Neurosurg Clin N Am* 2002 Apr; 13(2):227-33.
25) Bandak FA. Shaken baby syndrome: a biomechanics analysis of injury mechanisms. *Forensic Sci Int* 2005 Jun 30; 151(1):71-9.
26) Hadley MN, Sonntag VK, Rekate HL, Murphy A. The infant whiplash-shake injury syndrome: a clinical and pathological study. *Neurosurgery* 1989 Apr; 24(4): 536-40.
27) Shannon P, Smith CR, Deck J, Ang LC, Ho M, Becker L. Axonal injury and the neuropathology of shaken baby syndrome. *Acta Neuropathol* 1998 Jun; 95(6):625-31.
28) Geddes JF, Whitwell HL. Inflicted head injury in infants. *Forensic Sci Int* 2004 Dec 16; 146(2-3):83-8.
29) Kemp AM, Stoodley N, Cobley C, Coles L, Kemp KW. Apnoea and brain swelling in non-accidental head injury. *Arch Dis Child* 2003 Jun; 88(6):472-6; discussion -6.
30) Case ME. Inflicted traumatic brain injury in infants and young children. *Brain Pathol* 2008 Oct; 18(4):571-82.
31) Case ME, Graham MA, Handy TC, Jentzen JM, Monteleone JA. Position paper on fatal abusive head injuries in infants and young children. *Am J Forensic Med Pathol* 2001 Jun; 22(2):112-22.

32) Gleckman AM, Bell MD, Evans RJ, Smith TW. Diffuse axonal injury in infants with nonaccidental craniocerebral trauma: enhanced detection by beta-amyloid precursor protein immunohistochemical staining. *Arch Pathol Lab Med* 1999 Feb; 123(2):146-51.

33) Johnson DL, Boal D, Baule R. Role of apnea in nonaccidental head injury. *Pediatr Neurosurg* 1995; 23(6):305-10.

34) Stoodley N. Non-accidental head injury in children: gathering the evidence. *Lancet* 2002 Jul 27; 360(9329):271-2.

35) Parizel PM, Ceulemans B, Laridon A, Ozsarlak O, Van Goethem JW, Jorens PG. Cortical hypoxic-ischemic brain damage in shaken-baby (shaken impact) syndrome: value of diffusion-weighted MRI. *Pediatr Radiol* 2003 Dec; 33(12):868-71.

36) Dolinak D, Smith C, Graham DI. Global hypoxia per se is an unusual cause of axonal injury. *Acta Neuropathol* 2000 Nov; 100(5):553-60.

37) Talbert DG. Shaken baby syndrome: does it exist? *Med Hypotheses* 2009 Feb; 72(2):131-4.

38) Geddes JF, Talbert DG. Paroxysmal coughing, subdural and retinal bleeding: a computer modelling approach. *Neuropathol Appl Neurobiol* 2006 Dec; 32(6):625-34.

39) Talbert DG. Paroxysmal cough injury, vascular rupture and 'shaken baby syndrome'. *Med Hypotheses* 2005; 64(1):8-13.

40) Duhaime AC, Christian CW, Rorke LB, Zimmerman RA. Nonaccidental head injury in infants--the "shaken-baby syndrome". *N Engl J Med* 1998 Jun 18; 338(25):1822-9.

41) Duhaime AC, Gennarelli TA, Thibault LE, Bruce DA, Margulies SS, Wiser R. The shaken baby syndrome. A clinical, pathological, and biomechanical study. *J Neurosurg* 1987 Mar; 66(3):409-15.

42) Prange MT, Coats B, Duhaime AC, Margulies SS. Anthropomorphic simulations of falls, shakes, and inflicted impacts in infants. *J Neurosurg* 2003 Jul; 99(1):143-50.

43) Ommaya AK, Goldsmith W, Thibault L. Biomechanics and neuropathology of adult and paediatric head injury. *Br J Neurosurg* 2002 Jun; 16(3):220-42.

44) Couper Z, Albermani F. Mechanical response of infant brain to manually inflicted shaking. *Proc Inst Mech Eng H* 2010; 224(1):1-15.

45) Molina DK. Neck injuries and shaken baby syndrome. *Am J Forensic Med Pathol* 2009 Mar; 30(1):89-90.

46) Gill JR, Goldfeder LB, Armbrustmacher V, Coleman A, Mena H, Hirsch CS. Fatal head injury in children younger than 2 years in New York City and an overview of the shaken baby syndrome. *Arch Pathol Lab Med* 2009 Apr; 133(4):619-27.

47) Oehmichen M, Schleiss D, Pedal I, Saternus KS, Gerling I, Meissner C. Shaken baby syndrome: re-examination of diffuse axonal injury as cause of death. *Acta Neuropathol* 2008 Sep; 116(3):317-29.