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Traumatic Spinal Cord Injury: Accidental Versus Nonaccidental Injury

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A 21-month-old boy with steroid-dependent asthma presented to the emergency room with Glascow Coma Score (GCS) 3 and retinal hemorrhages. He was found to have subdural and subarachnoid hemorrhage on computed tomography plus findings of hypoxic-ischemic encephalopathy (HIE). The caretaker history was thought to be inconsistent with the clinical and imaging features, and the patient was diagnosed with nonaccidental injury (NAI) and "shaken baby syndrome." The autopsy revealed a cranial impact site and fatal injury to the cervicomedullary junction. Biomechanical analysis provided further objective support that, although NAI could not be ruled out, the injuries could result from an accidental fall as consistently described by the caretaker.
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Nonaccidental injury (NAI) or "shaken baby syndrome" (SBS) is a diagnosis that is often considered in infants presenting with an acute life-threatening event. Emergency physicians, family practitioners, and pediatricians are often the first to evaluate a child in this situation. Pediatric neurologists and neuroradiologists are often consulted in such cases. It has been previously accepted that in the absence of a history of significant trauma (ie, motor vehicle accident or 2-story fall), the "triad" of (1) infant encephalopathy, (2) subdural hemorrhage (SDH) or subarachnoid hemorrhage (SAH), and (3) retinal hemorrhages (RHs) is diagnostic of NAI/SBS based on a rotational acceleration-deceleration trauma mechanism. This empirical formula has been challenged by evidence-based medical and legal standards.[1-12]

We present a case of a toddler with a household fall scenario resulting in "spinal cord injury without radiographic abnormality" ("SCIWORA") identified at autopsy.[13] The case was initially labeled as NAI/SBS.

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Case Report

Clinical Course

A 21-month-old boy was brought to the emergency room with a GCS of 3. He reportedly fell onto a tiled floor from a standing position on a kitchen chair while eating. The caretaker saw the child standing in the chair and then turned away. He heard but did not see the actual fall and then found the child limp on the floor making gasping sounds. He attempted to clear food chunks from the child's mouth and then carried the child to a hospital 10 minutes away. On arrival at the ER, the patient was apneic and pulseless, with fixed and dilated pupils. Cardiopulmonary resuscitation (CPR) with chest compressions was applied for approximately 10 minutes to establish a heart rate. During CPR, copious amounts of milk and mucus were noted in the mouth. The child was intubated and placed on a ventilator. No evidence of traumatic injury was identified on physical examination. He was then transported to a pediatric intensive care unit (PICU) at another facility. Laboratory analysis showed a coagulopathy that was treated with fresh frozen plasma. In the PICU, food particles were suctioned from the lungs and nasopharynx. The patient displayed decortication with occasional gasps but soon became completely unresponsive. He was never sedated. An intracranial pressure monitor recorded an initial pressure of 46 mm Hg with subsequent pressures ranging from 40 to 70. The initial ophthalmologic examination was done in the PICU and revealed bilateral RHs with perimacular folds. Brain death was subsequently documented, and vital support was withdrawn. The patient died 44 hours after the fall.

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Imaging Evaluation

The initial computed tomography (CT) scan obtained within 2 hours of the fall showed findings of early diffuse cerebral edema, SDH, and SAH, including a prominent left parietal focus of SAH and a focal right frontal SDH (Fig 1). A much smaller SAH/SDH were also present along the convexities, falx (ie, interhemispheric), and tentorium. There was no evidence of brain hemorrhage. No scalp or skull abnormalities were identified, although 1 observer could not rule out a "healed" skull fracture along the sutures in the parieto-occipital region. A CT scan performed 5 hours later showed progression of the cerebral edema and no change in the SAH or SDH (Fig 2). A CT scan of the cervical spine was negative (Fig 3). Magnetic resonance imaging was recommended but never obtained. A skeletal survey showed anterior wedge-like vertebral body deformities from T5 through T12 and inferior L2. Some widening of the cranial sutures was present, but no fractures were confirmed on the plain radiographs. The remainder of the survey was negative. A postmortem CT scan of the entire spine (Fig 4), confirmed the vertebral deformities.

Autopsy Findings by the Medical Examiner

Head and Brain

A focus of hemorrhage was present in the left posterior parietal scalp (ie, impact site). Microscopy showed acute hemorrhage with acute vital reaction within the fibrous connective tissue of the galea in that region. Brain weight was 1,270 g with the spinal cord attached. No skull fracture was shown. The brain was extremely swollen with generalized flattening and ablation of the normal gyral pattern. Histologically, a diffuse axonal injury pattern consistent with HIE was present. There were no gross traumatic brain parenchymal injuries (eg, no contusion or shear lesions) or any histological evidence of traumatic axonal injury (TAI) observed microscopically on beta-amyloid precursor protein (*B*-APP) immunoperoxidase stains. There were bilateral, holohemispheric, thin-layer SDHs with no mass effect.

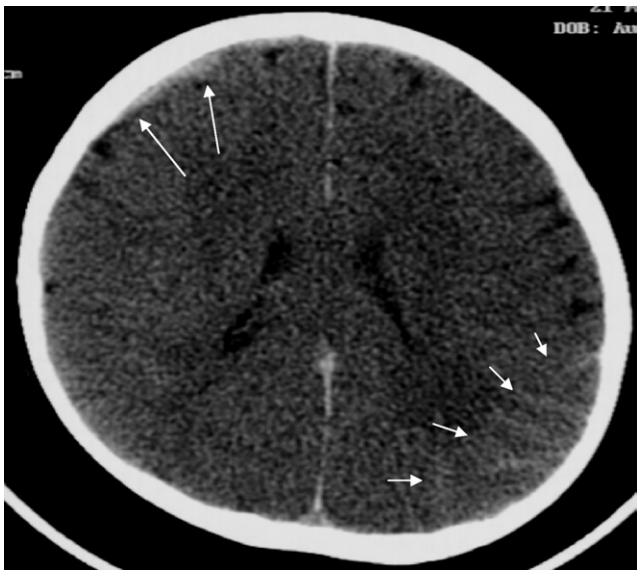

Figure 1 A nonenhanced CT image of the brain preformed within 2 hours of the fall shows decreased differentiation of gray/white matter representing edema.

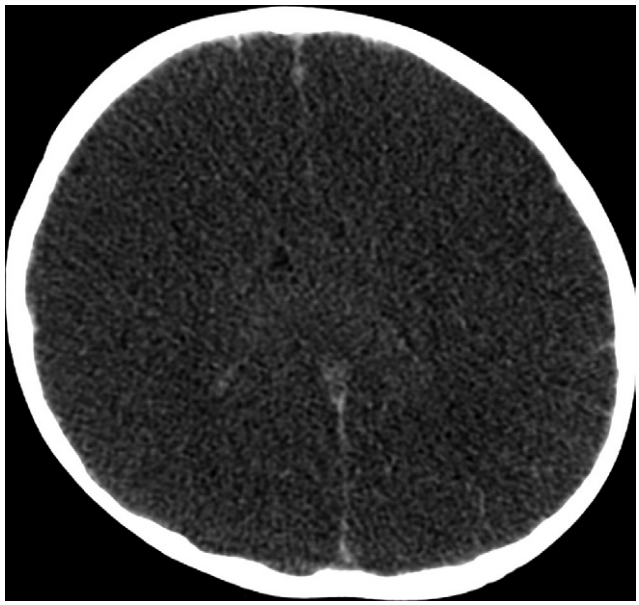

Figure 2 A CT scan performed 5 hours after the initial CT scan shows marked progression of cerebral edema with complete loss of gray/white matter differentiation, obliteration of sulci, and near complete obliteration of the ventricular system. A SAH is seen within compressed sulci in the left parietal lobe.

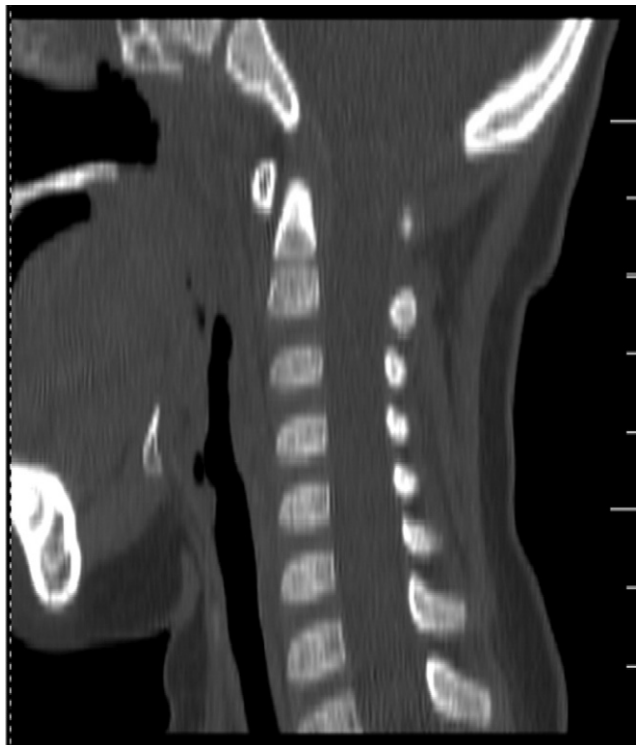

Figure 3 Sagittal reconstructed CT image of the cervical spine shows normal alignment and no fractures.

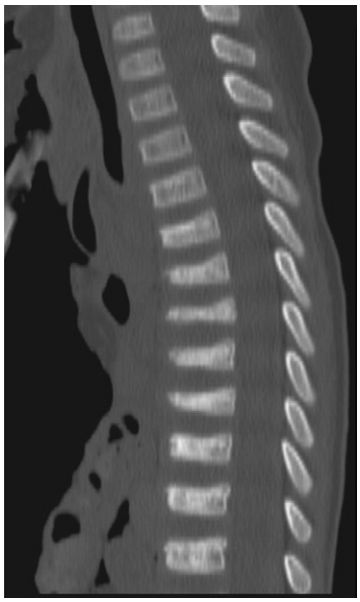

Figure 4 Sagittal reformation of the thoracic spine from a postmortem CT scan shows multilevel anterior wedge compression fractures of varying degrees.

Neck and Spinal Cord

Focal soft-tissue hemorrhages were present in relation to the right posterior neck and shoulder regions as well as the posterolateral transverse processes of the atlas and axis and at the C1-C2 intervertebral junction. No vertebral artery abnormality was noted. The dura appeared tense and filled with blood-stained fluid. Sagittal sections at the cervicomedullary junction showed partial transection and disruption of the central cord immediately distal to the inferior medullary olives. The tissue appeared elongated and physically separated suggesting axial tension of the cord (Fig 5). The cellular response consisted of round and polymorphonuclear cells with acute, focal hemorrhage. Findings of ischemic neuronal degeneration were most prominent in, and adjacent to, the dorsal and ventral neurons and consisted of cytoplasmic swelling, degranulization, loss of fine detail, and nuclear pynknosis.

Eyes

On gross examination, the pigmented layer of the retina was focally separated from the choroid. RHs were present primarily in the ganglion cell layer anteriorly but extended posteriorly. An optic nerve sheath hemorrhage was also present bilaterally. There was no mention of perimacular folds.

Vertebral Column

The vertebral bodies from T2 through L3 to 4 were removed en bloc. A hemorrhage was seen in the anterior and lateral perivertebral fibroconnective tissues. Microscopic sections showed acute hemorrhage with fibrin deposition replacing normal marrow of all vertebral bodies. Multiple areas of disrupted cancellous bone with acute-phase osteogenic granulation tissue were especially prominent T7 through T10. No callus or osteoblast activity was present.

Biomechanical Analysis

A court-approved, biomechanical evaluation was performed including an investigation of the home setting where the injury reportedly occurred. A number of potential accidental and NAI (including SBS) scenarios were considered and analyzed primarily to address the thoracic spinal injuries and secondarily to address the cervical cord injury. The approach to the biomechanical analysis was to assume that the caretaker's history was truthful and accurate and to then apply the principle of mechanics to evaluate whether or not such a history could be consistent with the subsequent injuries. This approach was not intended to rule out other possibilities but simply to evaluate the history provided.

In that light, the caretaker consistently reported to all authorities that he had his back turned at the time of the incident but that the boy had been standing up on the seat of the chair. He then reported hearing a noise and turning to find the boy and the chair on the floor, with the chair lying on its back. Using the caretaker's consistent history as one scenario, along with the imaging and clinical findings, the child was assumed to have fallen, rotating with the chair until a point of separation (Fig 6). From that point, it was further assumed that he fell freely to strike the floor first with his head and then with his dorsal neck and a shoulder, again based on the imaging and autopsy findings. This "impact" scenario would

Figure 5 A histological specimen through the cervicomedullary junction shows complete disruption of the central cord elements (circle). (Color version of figure is available online.)

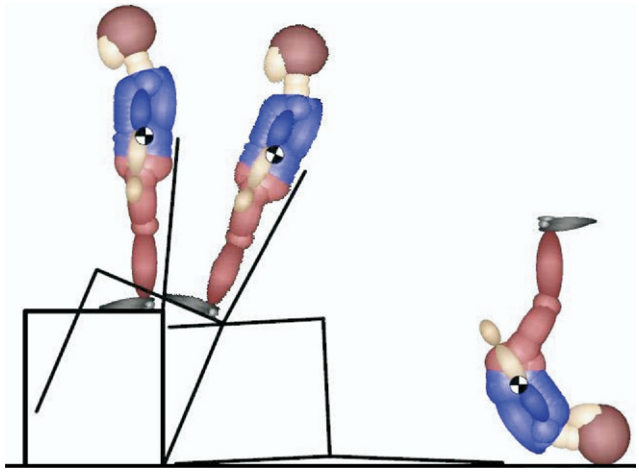

Figure 6 A schematic representation of the fall. (Color version of figure is available online.)

produce flexion and axial compression with the center of mass of his body trailing above. The initial motion of the child was assumed to have produced an initial rotation of the child and chair together as an inverted pendulum system about an axis at the base of the chair's rear legs. Once the chair reached its natural tipping angle, however, it was assumed to continue in its inverted pendulum motion while the child then fell freely to the floor.

Multiple anterior compression fractures of the thoracic spine, as reported in this case, are uncommon. The mechanism most consistent with this type of injury, however, would be severe flexion and/or axial compression of the spine. It is problematic, biomechanically, to conclude that such an injury can result from "SBS," particularly in a child of this age and size. Further evidence of head and shoulder impact suggests that the necessary loading of the spine may have been produced by forces applied to the head, neck, and/or shoulder. Thus, we chose to evaluate a scenario in which the boy somehow caused the chair to tip. He then rotated with the chair and ultimately fell in such a way that his head struck the floor first and quickly rotated to produce flexion in the neck and bring the shoulder/lower neck region into contact with the floor. The force acting through the shoulder area then acted to bring the remainder of the child's body mass to rest, resulting in flexion and axial compression of the middle and lower spine. There are clearly many variables associated with the chosen scenario, including the influence of body rotation and whether the child was rotating forward or backward, but there is no reason to believe the fall could not have occurred as described. Given the selected scenario, the next step was to evaluate whether or not the forces associated with the respective impacts to the head and shoulder/lower neck could have been severe enough to produce the injuries.

Analysis of the chair provided a seat height of 43 cm (17 in) and a rearward tipping angle of 23°. The mass of the chair and its center of gravity (CG) were taken as equal to 6.8 kg (15 lb) and the height of the seat, respectively. The chair measurements, coupled with the height of the child (86 cm, 34 in), his mass (15.9 kg, 35 lb), and the position of his CG being at approximately 57% his height,[7] were used to determine the CG of the combined system (Appendix 1). Thus, the first phase of the fall was modeled with the child and chair combined as an inverted pendulum until the chair reached its natural tipping angle. Vertical and rotational velocities at this point were then used for the initial conditions of the subsequent free fall, resulting in a predicted impact velocity ranging from 3.7 to 4.0 month(s). Impulse momentum was then applied to determine the severity of a fully plastic impact, modeling the child as a single spring-mass system, with the flexing spine serving as the spring, and the mass defined as the measured mass of the child minus that of his head. Because there are limited data available to define an appropriate impact duration, a broad range of 50 to 100 milliseconds was considered. Thus, the peak impact force was estimated to range between 0.9 and 1.9 N. A free-body diagram of the flexed body was used to determine associated peak thoracic vertebral body forces ranging from 1.4 to 3.1 kiloNewtons (kN). Forces in the separately considered head impact were estimated to be between 3.4 and 3.7 kNs.

Discussion

NAI/SBS

In this case, the initial "diagnosis" of NAI/SBS was based on the heretofore classic "triad" of SDH, RH, and encephalopathy, along with a history presumed to be inconsistent with the injuries. Central nervous system findings that mimic NAI/SBS have been reported in accidental trauma and in a number of medical conditions.[14-21] The latter includes infection, coagulopathy, metabolic disorders, and others.[19-21] More recent reports also show that there is no specific pattern of intracranial hemorrhage that is diagnostic of NAI/SBS to include interhemispheric SDH and mixed-density SDH.[14-17] Furthermore, recent evidence-based medical reviews (and legal challenges) of the past NAI/SBS literature reveal that the vast majority of these publications failed to achieve quality of evidence ratings that would merit the use of the "triad" as a standard or guideline for proof of NAI/SBS.[1-8]

This case also shows the complexities involved in establishing the sequence of injuries given multiple findings. Although initial concern for NAI is important and must be reported, medical personnel must carefully correlate such findings with the history to establish a correct sequence of events, including predisposing factors.[19-21] The initiation of the criminal process before a complete and thorough child protection and medical evaluation can lead to a rush in judgment. The injuries in this particular case were attributed to SBS before the brain and spinal cord injuries were completely evaluated.[22] The father of the victim was charged with fatally shaking the child. After all the forensic evidence was considered, the ultimate verdict was acquittal.

Given the fact that the law requires physicians to report suspected NAI, there is the danger of assuming NAI in all cases of SDH and RH. As a result, further medical and imaging workup may not be pursued (eg, magnetic resonance

imaging of the brain and cervical spine). The American Academy of Pediatrics, as others, strongly endorse the use of magnetic resonance imaging in cases of suspected NAI.[17,20,21,23] In the absence of an apparent cause of diffuse cerebral edema (including HIE), cervical cord injury should be considered. After the initial CT scan, magnetic resonance imaging is the choice for delineating spinal, paraspinal, and intraspinal injury. A short tau inversion recovery (STIR) sequence should always be included because this technique provides the best sensitivity for these types of injury.[21]

SCIWORA

SCIWORA is not uncommon in toddlers and has been reported to occur after minor accidental trauma as well as in cases of alleged NAI.[13,24-29] Evidence of a spinal cord injury plus cranial, neck, and shoulder impact on the postmortem examination are the key findings in this case. The gross and histological findings, as well as the imaging findings, are entirely consistent with the caretaker history of a household fall as corroborated by the biomechanical evaluation. This is true for both the primary injury (ie, cord transection) and the secondary injury (ie, HIE) as reflected in the clinical course of the child.

The bony structures of the cervical spine in infants and younger children are not fully developed as compared with that of the adult. Such "immaturity" includes the horizontal nature of facet joints with flat morphology of uncinate processes, elastic paraspinal ligaments, and anterior wedge-like morphology of the vertebral bodies.[13] These factors account for the relative ease of vertebral subluxation with complete recovery of the bony elements to normal anatomical alignment. This predisposes the child to cervical cord injury in the absence of bony abnormalities. Instantaneous damage to the respiratory centers at the cervicomedullary junction correlated with the child's respiratory distress, and subsequent HIE lead to extensive edema and increased intracranial pressure (ICP).

Hypoxic-Ischemic Versus Traumatic Axonal Injury

Gross and microscopic examination showed the effects of severe HIE. There was no evidence of "primary" traumatic axonal injury (ie, TAI or shear injury) as an indicator of rotational acceleration-deceleration trauma to the brain. The primary injury (ie, TAI) was shown to occur only at the cervicomedullary junction. In cases of TAI (formerly "diffuse axonal injury"), distinctive discrete swellings of the the axons, known as axon bulbs, are observed microscopically on *B*-APP immunoperoxidase stains. These are focal or multifocal lesions and most often occur along deep gray/white matter junctions, the corpus callosum, and dorsal corticospinal tracts. They may also be associated with focal hemorrhage. HIE, whether primary or secondary, results in a diffuse pattern of axonal alteration. Furthermore, the histological appearance is different from that of TAI, forming a linear or streak pattern on *B*-APP staining.[30-32]

Impact Injury

Although there are no data available defining skull fracture thresholds (as an indicator of impact) for a 21-month-old, data reported for younger infants[33-36] and adults[37] suggest that the calculated head-impact forces are enough to result in fracture in at least some of the population. The absence of evidence of "significant" trauma to the scalp and skull may additionally be explained by the wide distribution of the force along the head, neck, and shoulders at the time of impact. In young children with impact injury, there may be no focal scalp injury or skull fracture on physical examination or by imaging. Therefore, the lack of such findings should not be interpreted as absence of impact injury. Additionally, fatal and otherwise significant intracranial injuries have been reported from accidental household or short falls resulting in the triad of SDH, RH, and encephalopathy.[38,39] The biomechanical literature suggests different thresholds for central nervous system injuries given various scenarios.[40-43] Neck and cervical spine tissues may have a lower threshold than brain for minimum forces required to produce traumatic injuries.[40]

SDH and SAH

There are a number of potential causes for the SDH/SAH in this case. These include impact trauma, coagulopathy, increased ICP, ischemic endothelial damage, and reperfusion. The focal left parietal SAH (Fig 1) correlates with the primary site of impact (ie, coupe injury), and the focal left frontal SDH may be consistent with contracoupe injury. Further hemorrhage may be related to the coagulopathy as supported by laboratory findings. This is a known phenomenon that may be initiated by tissue injury caused by trauma or hypoxia ischemia.[44,45] Once the capillary beds are open and leaking, further increases in ICP from brain edema and CPR may exacerbate this process. Geddes et al[32] suggest that additional factors such as venous and arterial hypertension (HTN) may exacerbate hemorrhage in the ischemic, swollen brain with increased ICP. They propose both increased oozing from hypoxic veins in the setting of venous HTN secondary to severe edema and increased hemorrhage from episodic or sustained arterial HTN (eg, with reperfusion) that may occur as a part of Cushing's triad or be neurogenic in origin. Additionally, choking, vomiting, or paroxysmal coughing (eg, pertussis) may also result in SDH and RH.[46-48] Furthermore, the distribution of SDH or SAH along the interhemispheric fissure is not pathognomonic for NAI, as previously reported, and has been shown to occur in cases of accidental trauma and HIE.[14,16,17,21]

RH

The initial funduscopic documentation of RH was not made until the child was in the PICU. Given the course of events to that point, the RH may be a result of multiple factors as described earlier regarding SDH and SAH. RH is a known manifestation of increased ICP. There is no single type or pattern of RH that is pathognomonic of NAI/SBS, and RH is reported in a number of other conditions.[15,16,49-57]

Thoracic Spinal Injury

The multiple thoracic compression fractures in this case are unusual in NAI (SBS) and require biomechanical assessment as well as consideration of patient risk factors. Based on studies of compression fracture in intact cadavers subjected to flexion and compression testing of both adult human[58,59] and pediatric baboon vertebral bodies,[60] the biomechanical analysis suggested the presence of sufficient force to cause the thoracic fractures, in addition to the cervical spinal cord injury in this case.

Additionally, the patient had steroid-dependent asthma treated with daily beclomethasone dipropionate inhaler for a period of 8 months before the fall. During multiple visits to the emergency room for exacerbations before the reported fall, he also received additional doses of steroids in a form of prednisone. Two weeks before the fall, he received 20 mg of prednisone daily for 6 days. The influence of steroids in this case is uncertain. However, such high daily doses of oral steroids have been shown to significantly increase the risk of fractures.[61-63] Van Staa et al[61] also showed that high daily doses independent of duration of treatment or prior exposure put the patients at high risk for fractures. The patient also presented to the emergency room 3 months before the current incident after a fall down the stairs. He was evaluated clinically and released. No imaging was performed at that time. The postmortem histological examination of the vertebral column showed evidence of acute trauma. Additionally, an unusual distribution of diffuse microfractures was observed in all bones examined, supporting the possible effect of chronic steroid therapy on bone fragility. It was impossible to assess for bone density of the spine during necropsy because the bones were decalcified.

Conclusion

Physicians have an obligation to completely and timely evaluate suspected NAI, including its mimics. The imaging findings alone cannot distinguish NAI from AI or from the medical mimics. A complete and thorough medical evaluation, using evidence-based medicine principles, is necessary in parallel with the child-protection assessment. A multidisciplinary approach to this evaluation is also important, including the involvement of qualified specialists. Such an approach may be the difference between appropriate child protection versus the improper breakup of a family or a wrongful indictment and conviction.

Appendix

Equations Used in Biomechanical Analysis

- Angular velocity of combined chair-child system at end of inverted pendulum phase was determined as $\omega = \sqrt{2\left(g/L\right)(1-\cos\theta)}$, where L is height of combined system center of mass and θ is angle of rotation from vertical position (Fig A1).
- From conservation of energy, impact velocity $v = \sqrt{v_z^2 + 2gH}$ where v_z is the vertical component of linear velocity at the end of the inverted pendulum phase and H is the subsequent free-fall distance of the body CG to the floor.
- Using impulse momentum and assuming a triangular force pulse and plastic impact, peak force at impact $F = 2mv/\tau$, where m is the mass of the object, v is impact velocity, and τ is the duration of impact.
- Compression force on lumbar vertebrae, F_v, is determined by satisfying $\sum M = I\alpha$ about the ligament attachment point, as shown in the free-body diagram. Moment-arm distances were scaled from values used by Myklebust et al.[58]

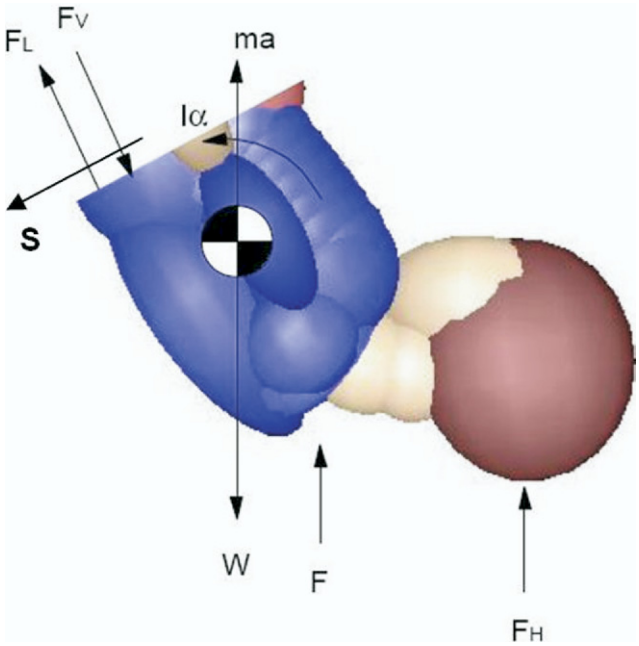

Figure A1 (See appendix). (Color version of figure is available online.)

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