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FOREWORD

The best feature of this innovative approach to the evaluation of children who may be victims of abusive head trauma (AHT) is the thoughtful approach to features such as cerebral edema, several forms of intracranial bleeding, ophthalmologic concerns, and other important features while sifting through, sometimes extensive, differential diagnoses. The inclusion of discussions of findings like birth trauma, genetic and metabolic disorders, short falls, and other possible diagnoses (although usually extremely rare) that will be considered in some children is another excellent feature of this 2-volume pocket atlas.

A variety of images, either photographs or radiologic studies, are presented to inform the thinking of clinicians who often need quick access to information in order to perform a thorough evaluation in the shortest time possible. If a patient is stable, this book also allows for a quick, but in-depth review of results after an episode of AHT or one of the other diagnoses that must be considered in most cases.

The Pediatric Abusive Head Trauma 2-volume set also allows medical students and residents to have a concise reference to augment what is usually only a brief exposure to this important diagnosis. Brain injury is a serious and all-too-frequent cause of morbidity and mortality in children. This diagnosis will often be challenged in court, as perpetrators of the injury are frequently prosecuted. Consequently, not only is the material in the Atlas important for guiding the diagnostic evaluation but also serves as a reference for both the general pediatrician and the subspecialist who may present their opinions in a court case.

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Foreword

Law enforcement faces many challenges during the investigation and prosecution of child abuse. Nonetheless, we as professionals seek justice through a search for the truth, found in the evidence obtained during an investigation and through interviews with witnesses and potential perpetrators. With abusive head trauma (AHT), the leading cause of injury and death in young children, being armed with a working knowledge of pediatric medicine and forensic methods enhances our initial response, evidence collection, and interviewing efforts. In addition, an investigator’s ability to document a victim’s presentation, symptoms, and medical history and incorporate them during a suspect’s initial interview may yield critical information for other multidisciplinary professionals who are often involved in AHT cases.

The various chapters in the Pediatric Abusive Head Trauma series address topics relevant to law enforcement involved in child abuse investigations. In particular, the chapter on Biomechanics will increase one’s understanding of the various types of head injuries and the effects of motion and force. This chapter expands on two areas significant during the early stages of an investigation: differentiating between abusive and accidental injuries and how/why the presentation of head trauma in children can be quite different from head injuries in adults. Familiarity with these areas allows law enforcement to gather a comprehensive history of an event and corroborate a caregiver’s statements or challenge a possible perpetrator’s initial account. The case studies, which will likely sound familiar to many experienced investigators, illustrate the most common social and environmental factors of AHT cases and provides investigative context for the medical findings in these cases. The section on medical mimics clarifies and demystifies the various conditions which can appear similar to traumatic head injuries. Since accurate and detailed timelines can be linchpins in these cases, understanding the differentiations between AHT and diseases or preexisting birth/accidental trauma can be critical to successful prosecution. Finally, the inclusion of color photographs, illustrations, and diagrams are beneficial, especially for nonmedical professionals, in that they visually demonstrate the associations between anatomy, biomechanics, injury presentation, and interpretation of tests results.

Successful child abuse investigations and prosecutions often depend upon law enforcement, social programs, and the medical community sharing information and valuable techniques. The significance of this collaboration is never more apparent than in cases of AHT, due to the frequency of its occurrence and the complexities surrounding diagnosis and mechanism. By providing a comprehensive resource
to improve recognition and responses to AHT, this 2-volume pocket atlas will be of particular assistance to law enforcement professionals who seek to integrate important medical aspects into their child abuse and child homicide investigations.

Joy Lynn E. Shelton, BA

\[1\text{Ms. Shelton, a Crime Analyst with the Federal Bureau of Investigation's (FBI), Behavioral Analysis Unit III - Crimes Against Children, has 15 years of experience in the investigation and analysis of violent crimes against children and has coauthored numerous articles on the topic of child homicide. This Foreword is being provided in an unofficial capacity and is not an endorsement by the FBI.}\]
FOREWORD

Two weeks into my career as child abuse prosecutor in a rural community, I was asked to try a termination of parental rights case. Suddenly, I was enmeshed in myriad legal and medical issues involving neglect, failure to thrive, excessive discipline, and more. I quickly realized that law school had not prepared me for any of this. Even more alarming, I quickly learned that most of my colleagues, including law enforcement officers, social workers, doctors, nurses, and psychologists were similarly inadequately trained in many aspects of child maltreatment.

The problem of inadequate training in child maltreatment is particularly pronounced in small, rural communities where, by necessity, every practitioner is a general practitioner. In the prosecutor’s office where I worked, we didn’t have “divisions” or “sections,” we had me and my boss. Accordingly, we handled every crime in our county from speeding to murder. As a result, it was difficult to specialize in any particular area.

In order to survive, much less excel in cases of child abuse, I learned the value of books. In particular, I relied on concrete, practical treatises that helped me understand myriad aspects of child maltreatment and that would assist me in explaining complex issues to jurors and judges. Our rural community eventually gained national recognition for our work in addressing child abuse, and it was our reliance on quality, practical publications that made all the difference in the world.

As I travel around the country working with frontline child protection professionals from every state, I repeatedly hear the need for publications of value to practitioners. Accordingly, I am excited about the publication of the Pediatric Abusive Head Trauma series.

In the field of child protection, there is no area more complex than abusive head trauma nor any area that generates as much controversy. Unfortunately, many child protection professionals are poorly equipped to recognize actual indicators of abusive head trauma, as opposed to symptoms that merely mimic abuse. Equally concerning, some defense experts prey on the naïveté of child protection professionals, judges, and jurors and have assisted some child abusers in escaping justice.

Through a concrete, comprehensive analysis of all aspects of abusive head trauma, this book will be of immeasurable assistance to the field. Through understandable prose and invaluable charts, photographs, and pertinent citations, this book will advance our field by helping frontline professionals properly and thoroughly assess cases.
I am grateful to the thousands of child protection professionals who labor long hours on behalf of children in need. It is for you and the children you serve that this book was written. If the text that follows helps even one of you assist a single child, this writing will not have been in vain.

To that end, I invite you to turn the pages of this book and to turn a new chapter in the history of child protection.

**Victor I. Vieth**  
Executive Director,  
National Child Protection Training Center  
A Program of Gundersen Health System
Preface

Abusive head trauma (AHT) is among the most severe manifestations of child physical abuse, and it is a major cause of morbidity and mortality. Yet, even though it has been documented since the very first descriptions of child maltreatment—by Tardieu in the 19th century, 1 Caffey, 2 Silverman, 3 and Kempe 4 in the 20th—it’s clinical recognition is often delayed. Its pathogenic mechanism continues to be a source of debate, and it has many potential mimics.

Earlier clinical recognition of AHT and many topics of contention regarding its mechanisms, pathogenesis, and evolution over time could be eliminated by a thorough exposition of the facts. This book will, therefore, describe and illustrate multiple aspects of AHT. It is constructed to serve as a reference for medical, social service, law enforcement, investigative, and legal professionals regarding recognition, diagnosis, and treatment of traumatic injuries and medical mimics.

We hope that this 2-volume pocket atlas will provide readers with a broad perspective and clarify many of the points in question about this important topic.

Lori Frasier, MD, FAAP
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REVIEWS

The Pediatric Abusive Head Trauma 2-volume set is a comprehensive review of the leading cause of physical abuse deaths in the United States today. It offers an in-depth evaluation of victim clinical presentation, usual physical examination, laboratory, and neuroradiologic findings, and outcomes. It also explores reasonable differential diagnoses and provides illustrative cases. This is an outstanding reference for child abuse pediatricians and for those professionals such as pediatric neurologists, child development specialists, and generalists who care for AHT survivors often afflicted with complex medical problems.

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This text contains concise and up-to-date discussions of facets and controversial aspects of abusive head trauma (AHT). As an example, Dr. Spivack briefly and in a very understandable manner discusses the basics of AHT biomechanics and injury evolution. Chapters on injury types discuss the basic background, causation, and treatment of AHT injuries while also documenting related conditions and possible abuse mimics. Dr. Fingarsen’s chapter on hypoxic-ischemic injuries nicely discusses causes and the radiologic and clinical evolution of these injuries, accompanied by prototypic images. Real injuries, such as venous sinus thrombosis, which are often within the legal venue, inappropriately, proposed as causing head injury findings as an alternative to AHT are succinctly summarized by Dr. Siffermann, while noting the lack of evidence that sinus thrombosis causes subdural hemorrhage.

For a quick reference to multiple aspects of AHT, this book deserves a place on your bookshelf.

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Having been involved in child protection investigations and prosecutions for over 15 years, I can say that every serious unexplained injury of a child presents challenges to medical professionals, law enforcement, CPS, and courts. It is important that professionals in each of these domains have access to the most reliable information with which to meet these challenges.

Most lawyers and judges, and certainly most jurors, do not come into
the courtroom with a particularly strong background in medical science. To an untrained person, the line between good science and junk science can be difficult to discern. To be effective, attorneys must be familiar enough with the science underlying medical evidence to be able to elicit the medical testimony in a way that is understandable and helpful to the listener.

Child protection investigations and litigation typically proceed at the accelerated pace necessitated by the critical need to protect children who are at risk of harm, and it is of the utmost importance for all involved to get it right. Betting it wrong means either leaving children in danger or, perhaps needlessly, disrupting custody and relationship between parent and child.

This book provides a valuable resource for Child Protective Services investigation staff and attorneys. Concise and authoritative, this book will assist the nonmedical professional in formulating the questions and avenues of inquiry that will help to get at the truth of situations which can involve complex fact patterns, conflicting witness statements, ostensibly unwitnessed events, and defense claims which mirror defense theories easily available on the Internet.

Zev Kianovsky
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Pediatric Abusive Head Trauma is an excellent resource that provides both superb visual depictions and discussions of traumatic head injuries. Case illustrations, detailed annotations, and a current reference lists make each chapter a treasure trove for clinicians, particularly for those who educate students, residents, and fellows. Multiple medical conditions that could be confused with abusive head trauma, such as birth trauma, disorders of coagulation, and genetic disorders, are included, which makes this 2-volume atlas an invaluable reference to use in exploring alternative hypotheses in both clinical care and courtroom settings.

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Biomechanics of Abusive Head Trauma

Betty Spivack, MD

Common Presentations
Clinicians are regularly tasked with assessing whether patients’ histories are compatible with their cranial injuries. Biomechanics is the application of the principles of motion and force to biological tissues. Injury biomechanics is a subspecialty of biomechanics. Clinicians should apply biomechanical principles as clinicians synthesize historical and injury information and formulate medical assessments and treatment plans. This chapter will focus on examining and understanding the injuries typically seen in cases of abusive head trauma (AHT) and distinguishing those injuries from injuries typical of common childhood accidents.

Features
The features or material properties of biological tissues are one of the factors used to determine whether injuries will likely occur and the spectrum and severity of those injuries, following an injury event. Head injuries concern the components that make up the cranium and its contents. Important components include the skull, meninges, intracranial and extracranial blood vessels, cortex, white matter, brain stem, ventricles, and cerebro-spinal articulation at the atlas and axis. As these components have various chemical and structural makeups, their features or material properties differ. Certain features stand out as factors of great importance.

— Thicker structures of homogenous material are stronger and more resistant to failure. Fractures of the thin-walled parietal bones are more common than fractures of the thick occipital bone.

— When a force or load is distributed over a small area, it is more likely to cause a failure in the material than if it is evenly distributed over a wider area. Poking a pencil through a thin parietal
bone is more likely to cause a fracture than a short fall, although parietal fractures from such falls do occur in toddlers and young children.

— Many biologic tissues behave elastically, i.e., they have a temporary deformation when exposed to relatively low forces for as long as the force is applied, and resume their original shape once the force is removed (Figures 1). This is truer of immature tissues, including pediatric bone, compared to adult bone. A young child falling on the side of his or her head may have sufficient elastic rebound of the parietal bone to cause tearing of an artery or vein without overlying fracture. This occurs in 13-55% of children requiring surgical evacuation of their EDH.1-4

— Biologic tissues are rarely plastic. Plastic substances may be permanently deformed without rupture. When a plastic substance is exposed to a force, it will not deform until a yield point is reached. Beyond the yield point, the degree of permanent deformation is dependent upon the length of time the force is applied (Figure 2).

— Many biologic tissues have elastoplastic properties. Such substances respond elastically until reaching their yield point (point beyond which elastic recovery will not occur), and then behave in a quasi-plastic manner. Because they are not truly plastic, they will rupture as they reach their ultimate strength (point at which material will be susceptible to injury) (Figure 3). An example of this phenomenon involves the skull of infants and toddlers. The thin parietal or temporal bones may act elastically with relatively low forces, but then if further force is exerted, a “ping-pong” fracture, rather than the true depressed skull fracture seen in older children or adults, may occur.

— Few, if any, biologic tissues are truly viscous, but many demonstrate viscoelastic properties. Viscous substances respond to forces differently depending on the rate at which the force is delivered. Viscous materials are stiff (nonresponsive) to forces delivered rapidly but very malleable and compliant to forces delivered slowly and steadily. Velocity, not acceleration, is the critical threshold in events of short duration for viscoelastic materials, including the brain. This principle is important when examining head injuries in young children. Viscoelastic materials also demonstrate continuing permanent deformation when forces are applied in a prolonged manner.
Scaling is also an important factor in inclination to injury (injury threshold). Volume, mass, and weight increase as a cubic function while surface area increases as a function of a square when objects proportionally increase in size. For this reason, loads (or forces) exert more pressure on the surrounding surface area with a proportionate increase in an object’s size. A relative protective effect is present in infant humans. The brain volume of an infant human is small compared to its surface area; therefore, it is more resistant to shearing injuries; however, it is not as resistant as pure geometric scaling would suggest.

*Figure 1-a.* Viscoelastic substances are relatively rigid when forces are rapidly applied. Low strain rates lead to greater deformation with less stress. (Adapted from Cochran GVB. A Primer of Orthopaedic Biomechanics. New York, NY: Churchill Livingstone; 1982.)

*Figure 1-b.* Viscoelastic substances develop a rapid, elastic response to applied loads but then experience a slow, continuing deformation or “creep” until the load is discontinued. The elastic deformation is rapidly reversible, the creep may slowly dissipate after cessation of the load. (Adapted from Cochran GVB. A Primer of Orthopaedic Biomechanics. New York, NY: Churchill Livingstone; 1982.)
Translational (crush or linear) acceleration and rotational (angular) acceleration produce different patterns of injury. Crush injuries and linear acceleration injuries both require impact; however, crush injuries are associated with long durations, low velocities, and high masses impacting the victim. Linear acceleration injuries occur when acceleration or deceleration occurs in a straight-line motion. Angular acceleration injuries occur when there is sufficient acceleration around an axis to cause shearing damage to one or more tissues within the body. Unlike crush and linear acceleration, impact need not be made during angular acceleration of the head for substantial head injury to occur.
SUBTYPES: BIOMECHANICAL BASIS OF TRAUMATIC BRAIN INJURY

PRIMARY AND SECONDARY INJURIES

Biomechanically, head injuries can be classified in relation to the time the traumatic event occurred. This classification has 2 groups: primary and secondary head injuries. Primary head injuries are those injuries caused directly by the traumatic event. Secondary head injuries are complications arising from changes in neurophysiology, increased intracranial pressure, and other problems after the event. This is not to say all head injuries recognized immediately are primary, and those recognized after minutes or hours are secondary. An epidural hematoma is a primary head injury whether it is recognized immediately or not. The herniation or near-herniation it may cause if left unrecognized is a secondary injury. Biomechanics best describe the causation of primary injuries while pathophysiology helps explain secondary injuries.

FOCAL AND DIFFUSE INJURIES

The second classification of head injuries is the relation of the head injury to the type of force applied. This classification also has 2 groups: focal and diffuse injuries. Focal injuries are caused by contact or translational (linear) acceleration events primarily resulting in compression. Brains, even adult brains, are relatively resistant to compression. Animal studies have shown the immature brain is more resistant to compression than the mature brain.5 The injuries that result from contact or translational (linear) acceleration events include soft tissue injuries, skull fractures and deformations, epidural hematomas, contact subdural hematomas, and cortical contusions. The subdural hematomas resulting from contact or linear acceleration events are more likely to be mass lesions and are not associated with immediate loss of consciousness. Linear acceleration events occurring over a short duration have a different effect than those with a long duration. This is because of the viscoelastic properties of the skull and brain. They are stiffer when things occur quickly and yield more readily when things occur slowly. A short fall may have a high g-force associated with it, but because it is a short duration event, the likelihood of serious injury is low.

Crush injuries are focal injuries, but because of the great disparity of the mass of the crushing object and the long duration of contact, they may not appear as focal. Crush injuries have typical “blow-out” patterns at the point of initial contact with the crushing object, and then the flattening of the skull and brain contents progress from that point onward. A crush injury from a car will look different than a crush injury from a television set because of the great disparity in mass
Pediatric Abusive Head Trauma

between television set and car. Also, a television set may spontaneously roll off the infant or be removed by a caretaker, decreasing the time of exposure. Crush injuries differ from more common impact injuries in 2 ways. First, crush injuries are long duration events, measured in seconds rather than milliseconds, enhancing the likelihood of severe head injury. Secondly, the head is in a fixed position, and there is no possibility of elastic rebound to decrease the forces delivered to the skull and intracranial contents.

Diffuse injuries are caused by angular acceleration with or without associated impact. Unlike focal injuries, where compression is the principle manner of force, shearing injuries occur when rotational or angular acceleration is involved. If impact is present as well, the threshold of angular acceleration to cause a given degree of injury is decreased. The clinical hallmark of the diffuse injury is concussion. Concussion is a transitory event, leaving no visible result behind on neuroimaging but producing sufficient strain on cortical tissues to interrupt or alter consciousness to some extent, at least briefly. Subdural hematomas are usually small and multifocal with thin film; they are the most frequent visible intracranial sign of diffuse injury caused by angular acceleration, with or without associated impact. More severe degrees of angular acceleration will be associated with slit-like lacerations between the gray and white matter, punctate contusions within the white matter, lacerations within the corpus callosum or deeper within the basal ganglia and brain stem. As a group, these are termed “diffuse axonal injury” and are associated with prolonged coma or death. Animal studies have shown that immature brains are more susceptible to angular acceleration injuries than mature brains, and that this effect is enhanced when the acceleration occurs more than once.6,7

Diagnostic Significance:
Patterns of Pediatric Head Injury
Soft Tissue Injuries
Soft tissue injuries and skull fractures are contact injuries. Some soft tissue injuries may be easily recognized while others are less easily recognized. Scalp lacerations will bleed freely because of the vascularity of the scalp. Subgaleal hematomas may be readily visible even when they are small; they can be life-threatening when large due to volume of hemorrhage. Subgaleal hematomas are the result of shearing forces that separate the scalp from the surface of the skull. A frontal subgaleal hematoma, a frequent injury following a fall with forehead blunt impact in young school-aged children, may decompress by following facial fascial planes to cause periorbital
ecchymosis, or a “black eye.” Therefore, a black eye does not always mean a child was punched in the eye.

When children suffer fatal abusive head injuries, many of their scalp contusions are only visible when the scalp is reflected at autopsy. The absence of visible bruising in a living child does not mean there was no impact injury during an accidental or intentional case of head trauma.

**Skull Fractures**
An infant or toddler skull is thinner than an adolescent or adult skull. If there are forces concentrated on a very small surface area, fracture is more likely in the infant or toddler than in an adult. However, the higher degree of elasticity and the open sutures and fontanelles are protective factors for infants and toddlers falling from low heights when forces are distributed over larger surface areas. Simple parietal fractures are the most common type of skull fracture whether accidental or inflicted. It takes additional force to cross suture lines, or to cause stellate, or “star-burst,” fractures.

**Intracranial Primary Injuries**
Primary intracranial injuries include epidural hematomas; subdural hematomas; subarachnoid hemorrhages; cortical contusions and lacerations; diffuse or focal axonal injury; and injury to midbrain, basal ganglia, and brainstem. Some of these injuries are due only to contact or linear acceleration mechanisms, while others require angular acceleration and may be facilitated by impact.

Epidural hematomas require contact, even if there is no overlying fracture. Diffuse axonal injury is typically associated with angular acceleration, with or without impact. If impact is present, the threshold levels for angular acceleration are lower. Subdural hematomas may be caused by both contact/linear acceleration forces as well as by angular acceleration, though the type of subdural hematoma differs. A subdural hematoma caused by contact/linear acceleration force may produce a mass effect lesion, similar in its clinical course to an epidural hematoma and without initial loss of consciousness. Angular acceleration forces produce thin-film, or “skim,” multi-focal, small-volume subdural hematomas and are associated with an initial loss of consciousness. The hallmark of any injury caused by angular acceleration forces is immediate loss of consciousness.

**Clinical Injuries and Biomechanical Modeling**
Beginning in 1971, A.N. (or Norman) Guthkelch,8 closely followed by John Caffey,9 identified shaking as a mechanism causing head injury and death in infants and young children. In 1987, biomechanical testing based on the scaling principles determined
that shaking did not reach threshold limits for subdural hematoma or diffuse axonal injury.\textsuperscript{10} These thresholds had previously been based on adult animal models subjected to a single whiplash event. However, shaking is a violent, repetitive event.\textsuperscript{11} Since that time, test models have diversified. The use of "crash dummies" have yielded differing results, but in vivo studies of immature animals show that the scaling criteria were inaccurate. Not only were the immature animals more sensitive to angular acceleration than the mature animals, they became apneic and developed diffuse axonal injury at scaling levels where they were not expected to even lose consciousness.\textsuperscript{6} When immature animals of the same species were exposed to still lower levels of angular acceleration twice in quick succession, the degree of diffuse axonal injury was far greater than previously observed.\textsuperscript{7} In other words, there is a cumulative damaging effect following repeated head injury. More recent information still shows that immature animals exposed to moderate angular acceleration without impact demonstrated neurobehavioral deficits that were not demonstrated in similar animals exposed to mild acceleration.\textsuperscript{12} When the immature animals were exposed to a control of one episode of mild angular acceleration, or repeated accelerations either one day apart or one week apart, the animals dosed one day apart died significantly more frequently (43\%) and had impaired visual problem solving compared with instrumented shams. The authors concluded that "we have observed an increase in injury severity and mortality when the head rotations occur 24 hours apart compared to 7 days apart. These observations have important translation to infants subjected to inflicted head trauma."\textsuperscript{13}

Although traditional injury criteria are based on high energy single loads, recent animal and anthropometric dummy studies have increasingly focused on lower energy repetitive loads which are more characteristic of shaking trauma. Of additional interest, there are currently no biofidelity models of infants necks; however, there are histopathological findings in the medullo-cervical spinal region of a majority of victims of fatal AHT.\textsuperscript{14} If injury to the medullo-cervical region occurs during AHT with subsequent apnea and secondary brain injury, biomechanical thresholds for diffuse axonal injury would not have to be surpassed to explain the parenchymal injuries seen in AHT.

Biomechanical research on the etiology of extensive retinal injury, a unique feature of infant and toddler AHT, is also in its early stages. Finite element analysis involves computerized modelling of tissue stresses. A recent finite element analysis supports the accumulation of peak stresses at the ora serrata and macula, areas of the retina where hemorrhages and schisis respectively are characteristic of AHT.\textsuperscript{15}
Biomechanics of Abusive Head Trauma

**RELATED CONDITIONS AND POTENTIAL MIMICS: DIFFERENTIATING ABUSIVE FROM ACCIDENTAL HEAD TRAUMA**

The biomechanical principles reviewed here are not intended to act as the sole method of determining whether a head injury is abusive or not. Like any other branch of medicine, history, imaging studies, and laboratory data must be used to make a complete and accurate assessment. Unlike other branches of medicine, scene investigation data or postmortem data can also be used when assessing the injury.

The most common non–soft tissue, accidental head injury is an isolated parietal skull fracture. Of all epidural hematomas, 94% are accidental. Simple parietal skull fractures and some epidural hematomas may be inflicted. In isolation, imaging studies or biomechanical analysis cannot assist a clinician in the determination of inflicted injury. Clinical correlation and other injuries are required. Complex skull fractures require closer scrutiny. A short free fall is not generally enough to explain a complex fracture. A stellate fracture with a depressed center generally has a severe and focal impact as its source. While a stellate fracture can happen accidentally, it requires a proper history in order to rule out abuse. The lack of a clear history of injury that has an accidental mechanism should raise suspicion of abuse. Complex fractures crossing suture lines also require more force than is typically caused from short falls; however, increased height of the fall and other circumstances, such as features of the landing surface onto which the child fell, may increase the likelihood of such a fracture. Conversely, other factors about the landing surface may reduce the contact force and therefore the complexity of the fracture. If the surface is very resilient, ie, has the capacity to return to its original state after compressive stress, injury may be less likely (all else being equal) than a fall onto a rigid, unforgiving surface. For a resilient surface, consider a carpeted floor, or even better, a trampoline; rigid surfaces are things like steel, concrete, stone, and pavement. Meticulous attention to obtaining a history combined with scene investigation may be helpful in differentiating an abusive fracture from an unusual accidental injury.

Focal injuries include soft tissue injuries, subdural hematomas, and cortical and white matter contusions and lacerations. Focal injuries are caused by contact and linear acceleration mechanisms, and are not usually characterized by immediate loss of consciousness. Nevertheless, such injuries may progress to severe secondary brain injuries and death. The subdural hematomas caused by these mechanisms are typically isolated, mass effect lesions of varying size.
Diffuse injuries are caused by angular acceleration mechanisms and are the result of shearing between cranial structures. These injuries include small, thin-film subdural hematomas; multifocal, gliding contusions; or small parenchymal lacerations at the gray-white interface. These lacerations are most common in infants under 6 months of age and can be characterized by petechial hemorrhages within the cerebral white matter, tears in the corpus callosum, and axonal injury that may extend to the midbrain and lower brainstem. The cardinal sign of diffuse primary brain injury is immediate loss of consciousness.

Severe brain injuries (both focal and diffuse) may occur as the result of both accidental and abusive processes. Once again, a detailed history and careful correlation with the injuries is necessary to differentiate between accidental and abusive etiologies. A common setting for severe traumatic brain injuries in children is that of a motor vehicle collision. A motor vehicle collision’s relatively long duration and potential for head on neck rotation, even when a safety belt is worn, is an optimal setting for a diffuse brain injury to occur, rendering a significantly brain-injured child unconscious at the scene. When a child involved in a motor vehicle collision presents to an emergency department, the staff know the mechanism of injury correlates with the child’s unconscious state and the diffuse brain injury on neuroimaging. This scenario is quite different from a child who presents to the emergency department with no history of a serious traumatic event, or a history of trivial head trauma, but has essentially the same neuroimaging characteristics and a similar or worse clinical course than the child injured in a motor vehicle collision. In summary, immediate loss of consciousness at the time of injury is characteristic of primary diffuse brain injury.

**Key Points**

— Biomechanics is a single tool for understanding clinical injury.

— A comprehensive history of an event is the best clinical tool for differentiating an accidental from an inflicted, nonaccidental injury.

— Not all focal head injuries are accidental.

— Not all diffuse head injuries are the result of abuse.

— Shaking may be associated with impact injury and often is, especially in fatal cases.

— Impact may occur without visible external injury or skull fracture.
REFERENCES


MEDICAL MIMICS OF ABUSIVE HEAD TRAUMA
Common Presentations

Injuries common to both birth trauma and abusive head trauma (AHT) include scalp hematoma, skull fracture, intracranial hemorrhage, and retinal hemorrhage. While much of birth-related injury is clinically asymptomatic, presentation varies depending on the severity of the injury.

Features

Intracranial Hemorrhage

Intracranial hemorrhage was initially believed to be relatively uncommon, but recent data shows it to be a common finding with parturition. In an extensive retrospective review of 583,340 single, non-breech, term, live births to nulliparous women, Towner et al found the incidence of intracranial hemorrhage to be uncommon (1 in 1900 deliveries). However, there were increased incidences of intracranial hemorrhage in infants subjected to abnormal labor (1 in 664 infants delivered by forceps, 1 in 860 infants delivered by vacuum extraction, and 1 in 907 infants delivered by cesarean section with a trial of labor). Recent studies have suggested the method of assisted delivery is more important in cranial birth injuries than the urgency of delivery or dysfunctional labor, and the presence of subdural hemorrhage is not necessarily indicative of excessive birth trauma.

While the Towner et al study reported a relatively small incidence of intracranial hemorrhage with birth, recent studies by Holden et al, Looney et al, and Rooks et al have reported much higher incidences of intracranial hemorrhage in asymptomatic term infants (26% to 50%). These most recent studies have found no statistically significant difference between the method of delivery or dysfunctional labor. Consequently, Holden et al, Looney et al, and Rooks et al have concluded that intracranial hemorrhage is a common result of parturition.

Intracranial hemorrhage is hypothesized to be secondary to tearing of bridging veins or tearing of the falx or tentorium during parturition. Multiple mechanisms for the tearing of these structures during birth
have been proposed. One suggested mechanism is that circumferential pressure from squeezing of the head in the birth canal results in overlapping of the sutures. This mechanical deformation then causes shearing of the bridging veins and consequent subdural hemorrhage.\textsuperscript{7} Other proposed mechanisms include hypoxia,\textsuperscript{8} abnormal labor,\textsuperscript{1} and difficult delivery.\textsuperscript{2} There are, however, limits to these proposed mechanisms. Specifically, subdural hemorrhages have been shown to occur in cesarean sections without trials of labor,\textsuperscript{6} and torn bridging vessels are not always observed at autopsy.\textsuperscript{9}

The most common location for intracranial hemorrhage in term, asymptomatic infants is the subdural compartment of the posterior cranium. While multiple studies have recognized the association of subarachnoid, cerebellar, intraventricular, germinal matrix, and intraparenchymal hemorrhage with parturition, subdural hemorrhages are by far the most common.\textsuperscript{1,3,5,6} Initially thought to be primarily infratentorial in location, recent literature demonstrates asymptomatic, birth-related subdural hemorrhages can be either supratentorial or infratentorial.\textsuperscript{6} Whether infratentorial or supratentorial, asymptomatic, birth-related subdural hemorrhages are primarily located in the posterior cranium.

In contrast, subdural hemorrhages associated with AHT are primarily located along the cerebral convexities or interhemispheric fissure. However, the location of subdural hemorrhage formation alone is not specific enough to distinguish AHT from birth-related intracranial injury. Recent literature has noted asymptomatic, birth-related subdural hemorrhages along the interhemispheric fissure.\textsuperscript{5} Unlike some subdural hemorrhages seen in AHT, uncomplicated, birth-related subdural hemorrhages are not commonly seen along the frontal convexities.\textsuperscript{6}
Epidural hemorrhages are rare, birth-related intracranial injuries, most commonly associated with a traumatic parturition.\textsuperscript{10}

Subdural hemorrhages of varying ages, or chronic subdural hemorrhages, are rare in the neonate.\textsuperscript{10} From 1977 to 2005, only 6 radiologically confirmed cases of chronic subdural hemorrhages in neonates were reported.\textsuperscript{10} Of those 6 cases, intrauterine trauma, coagulopathy, vascular anomaly, and genetic syndromes have been identified as clear etiologies, with intrauterine trauma being the most common. In all cases, the neonates were immediately symptomatic and had correlative physical exam findings.\textsuperscript{10}

Whitby et al reported between 94\% to 100\% of birth-related subdural hemorrhages resolved by 1 month of age.\textsuperscript{3} In a similar study by Rooks et al, only 2 of 46 patients required follow-up imaging at 3 months of age; the follow-up images showed complete resolution of initial, birth-related subdural hemorrhages.\textsuperscript{6}

Most children with birth-related subdural hemorrhages have normal developmental examinations at 24 months of age. While neurologic sequelae and overall morbidity depend upon the extent of the subdural hemorrhage and its concurrent/consequent complications, current long-term data, while limited, reveals that most children have minimal developmental sequelae at 24 months of age.\textsuperscript{6} In the study by Rooks et al, 43 of 46 patients were followed for 24 months after their birth-related subdural hemorrhage. Of those patients, none had gross motor delay, 14\% had speech delay, and only 2\% were evaluated for autism-spectrum disorder.\textsuperscript{6}

Retinal Hemorrhages
Retinal hemorrhages are a relatively common finding with parturition, and are more commonly associated with vacuum-assisted deliveries. Reports of birth-related retinal hemorrhages range widely from 2\% to 50\%. However, most studies have placed the incidence of birth-related retinal hemorrhages at approximately 20\% to 35\%.\textsuperscript{11-16} While this wide variance in incidence is likely multifactorial, studies have indicated the incidence of birth-related retinal hemorrhages is dependent on the time of examination. The incidence can be as high as 45\% if the infant is examined in the first 36 hours of life, but it drops to approximately 11\% to 20\% by 72 hours.\textsuperscript{13,17-19}

The incidence of birth-related retinal hemorrhages varies with mode of delivery. Several studies have found a significantly increased incidence in vacuum-assisted deliveries, ranging from 46\% to 78\%.\textsuperscript{13,16,20,21} The data is equivocal regarding the use of forceps, with some studies finding an increased incidence, some finding a decreased incidence, and some finding no effect.\textsuperscript{16} The studies reflect a protective effect of cesarean section with regard to retinal hemorrhages.\textsuperscript{16}
Multiple maternal, prenatal, and perinatal factors have been studied to determine whether there is an increased risk or greater association with retinal hemorrhages. The data demonstrates that race, gender, birth weight, fetal head circumference, the appearance pulse grimace activity respiration (APGAR) scores, maternal toxemia, maternal cocaine use, and primiparity are either equivocal or unrelated to an increased risk for retinal hemorrhages. Maternal age has been associated with an increased risk for retinal hemorrhages in some studies; however, this issue is controversial, and the significance of a possible association has not been determined.

While there are many theories, the exact mechanism of injury for birth-related retinal hemorrhages is unknown. Some theories include increased intracranial pressure (ICP), direct birth or forceps trauma, ocular or cranial compression, increased intrathoracic pressure, increased prostaglandin release, asphyxia, hypertension, neck compression, and change from maternal to newborn circulation. In cases of shaken baby syndrome, the prevailing theory is that repetitive acceleration/deceleration forces induce shearing forces at the vitreoretinal interface, resulting in hemorrhages. However, for retinal hemorrhages associated with birth related trauma, mechanical effects on retinal vessels from direct compression of the globe during passage through the birth canal is likely. Additionally, maternal and fetal hemodynamic and the rheologic changes during labor and delivery may also participate in the genesis of retinal hemorrhaging, a theory consistent with the development of retinal hemorrhaging in babies born via cesarean section. Other factors such as elevation of ICP, hypoxia, anemia, and intracranial hemorrhage may contribute to the intraocular abnormalities, but many authors suggest acceleration/deceleration forces are key factors. This theory is supported by studies with correlative histopathologic findings and by recent studies utilizing optical coherence tomography, which has offered further insight into the pathophysiology of retinal hemorrhages.

Retinal hemorrhages are most commonly dot, blot, or flame-shaped. Dot and blot hemorrhages represent ruptures in the walls of the capillaries of the deeper retinal layers. Flame-shaped hemorrhages are due to abnormalities in the retinal vessel walls of the superficial nerve fiber layer.

While birth-related retinal hemorrhages can be widespread, ie, extending to the ora serrata, and bilateral, there are characteristics that help distinguish them from the hemorrhages seen in AHT. First, the majority of birth-related retinal hemorrhages are confined to the posterior pole (zone 1). Second, the vast majority of birth-related retinal hemorrhages are intraretinal; in AHT, the vast majority
involves multiple layers of retina. Finally, traumatic retinoschisis, a condition that splits the retina, has never been reported in association with birth injury and is highly specific to AHT. Thus, extensive retinal hemorrhages in an otherwise healthy infant, without a history of significant accidental trauma, should raise suspicion of AHT.

Retinal hemorrhages cannot be reliably dated. Some data reveal there may be a 2- to 3-day delay in the development of vitreous hemorrhage. This data should not be used to rule out vitreous hemorrhage, occurring at the moment of abusive head injury. While dating the origin of the injury may be difficult, current data demonstrates that splinter-shaped or flame-shaped hemorrhages resolve by 1 to 2 weeks of age, and dot/blot hemorrhages resolve by 6 to 8 weeks. These estimates of resolution are conservative, as the literature demonstrates that the vast majority of birth-related hemorrhages—even when severe—resolve much sooner. Consequently, retinal hemorrhages outside 2 months of infant age are not believed to be birth-related.

The literature reveals that severe retinal hemorrhages and traumatic retinoschisis often resolve without long-term visual sequelae. More often, long-term visual impairment results from optic atrophy or intracranial complications of the occipital region.

**Subtypes**
Birth-related trauma is a condition without subtypes.

**Suggested Treatment**
Treatment is dictated by clinical presentation. The vast majority of birth-related skull fractures and intracranial hemorrhages naturally heal without complication. Neurosurgical consultation for evaluation and follow-up management is standard, but neurosurgical intervention is rarely required. Similarly, most birth-related retinal hemorrhages do not require surgical treatment. An ophthalmologist should monitor the patient until the condition resolves.

**Diagnostic Significance**
The most notable associated findings between birth trauma and AHT relate to subdural hemorrhages and retinal hemorrhages. In birth trauma, the subdural hemorrhages are usually acute and located in the posterior cranium. In AHT, the hemorrhages are, many times, either chronic or of varying age and located along the cerebral convexities or in the interhemispheric fissure. Birth-related retinal hemorrhages are almost always intraretinal and usually confined to the posterior pole. In AHT, the retinal hemorrhages are frequently multilayered, innumerable, and in all quadrants/zones of the retina. The finding of traumatic retinoschisis or macular folds is highly suggestive of significant inflicted trauma.
**RELATED CONDITIONS**
Birth trauma that mimics AHT is without related conditions.

**POTENTIAL MIMICS**
Conditions resulting from birth trauma, but mimicking AHT, include:

— Accidental trauma/obstetric complications
  *(Figures 1-2 through 1-4)*

— Hypoxic-ischemic encephalopathy

— Congenital malformations
  — Intracranial arteriovenous malformations (AVMs)

— Cerebral aneurysms
  — Osler-Weber-Rendu syndrome (hereditary hemorrhagic telangiectasia)

— Congenital hydrocephalus

— Superior sagittal sinus thrombosis

— Genetic/metabolic disorders
  — Glutaric acidemia type 1
  — Homocystinuria
  — Ehlers-Danlos syndrome
  — Marfan’s syndrome

— Hematologic disease/coagulopathy
  — Thrombocytopenia
  — Hemorrhagic disease of the newborn
  — Hemophilia A and B
  — Von Willebrand’s disease
  — Congenital dysfibrinogenemia
  — Hemophagocytic lymphohistiocytosis

— Infectious disease
  — Hemophilus influenzae meningitis
  — Streptococcus pneumoniae meningitis
  — Herpes encephalitis
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**Figure 1-2.** Sagittal T1 weighted MR image of the brain demonstrates a cephalohematoma over the vertex, related to vaginal delivery. (Courtesy of Kara G. Gill, MD.)

**Figure 1-3.** Parents may report palpation of a “lump” on a young infant’s head weeks after the birth. 3D reformatted head CT images of the skull demonstrate focal mild thickening of the high left parietal bone which is consistent with a healing cephalohematoma from birth related trauma. (Courtesy of Kara G. Gill, MD.)

**Figure 1-4.** AP radiograph of the clavicle in a 2-month-old male demonstrates an acute, mildly displaced fracture of the lateral right clavicle. The absence of healing callus argues against this fracture being related to birth trauma. (Courtesy of Kara G. Gill, MD.)

**REFERENCES**


