

**COMPLICATIONS OF
ACCIDENTS AND ILLNESS
MASQUERADING AS ABUSE**

PRESENTED BY

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The telephone call signaling a new case brings a positive balance to the office in many ways. But when the telephone call is about an allegation of physical child abuse, sometimes resulting in the death of a child, the positive is quickly replaced by doubts, revulsion and the fear that you just might prevail for a guilty client. And then again you just might lose for an innocent client if you don't know what you're doing. Numerous accidental injuries and illnesses mimic child abuse. If you can't effectively present them, an innocent person will go to jail.

Cases of alleged child abuse are strange creatures indeed. Autopsy and injury photographs are exponentially worse with children than with adults, the younger the more gut-wrenching. The prosecutor's expert witnesses will always be 100% certain that the injuries were caused by abuse. They are equally certain the injuries could not have been the result of any other cause. Sometimes the physicians know better: Sometimes they just don't know. Sometimes they are ignorant of the subtleties of a differential diagnosis.

The clients are a mental wreck. They have just lost a child to an accident or illness, what will turn out to be a blood problem, an inherited metabolic disorder, or maybe to abuse at the hands of another. They now find themselves to be the targets of a police investigation. Many feel guilty because they wonder if there isn't something they could have done to prevent the child's death, a natural feeling for a loving parent, teacher, school nurse, babysitter. If the child's injuries were not fatal, then the child and brothers/sisters are likely to be snatched up and put in foster care. Ensuing publicity only adds to the mounting horror show. Good people who believe in the system and trust the police to help them now find that entire frame of reference destroyed. They come to you

as their only source of help. To be able to free an innocent client, you must know the anatomy of these child abuse/death cases, particularly the subtleties that others within the system have glossed over.

ASSEMBLE A TEAM

Once you've gotten a preliminary idea of what you're dealing with, the first thing to do is assemble a team with a view to destroy the validity of the prosecution evidence, and build defense evidence that will set your client free. Your goal may be to keep clients from being charged, to have all charges dismissed, or to obtain a not guilty verdict. To do so you will need help from multiple disciplines, each addressing a different portion of the case.

1. Assign a Team Member to deal with the client's emotional needs.

Someone on the team must deal with the emotional needs of the client.

Assign someone in your office to be responsible for talking with the client about loss and pain, anger and fear. Many of us have had cases fall apart because the client falls apart. A busy lawyer will be so focused on the motion practice or investigation that s/he misses important clues that all is not well emotionally with the client. (I once missed big clues that my client had "lost it." It was not until he had his hands around my throat that I thought "maybe" something was wrong.) So, you must assign someone to monitor the client's emotional well being. It must be someone with good communication skills. Within the office, a secretary, paralegal, or receptionist who will take the time, stop, look and listen, will do well. A minister, close family member, business colleague, or simply a friend needs to be recruited to perform this function when the office isn't open. The client may need professional help to keep together emotionally, and if they need that help

get it as early as the need is detected. Be careful. At least in Nevada, psychiatrists can testify against their patients to admissions (of murder) the client made in the course of psychiatric treatment. State vs. Woods, 101 Nev. 128, 696 P.2d 464 (1985) Further, all states now have laws requiring all of the medical profession to report child abuse. See, NRS 432.255. The Catholic Church is considering abandoning the priest/parishioner privilege when child abuse is confessed. The husband-wife privilege in many states, including Nevada, can be invoked only by the testifying spouse. NRS 49.295. That means that if wife turns on husband, or husband turns on wife, you're in big trouble. The spouse's can decide to testify against your client. **Anyone** who talks to the client about case related issues **must** be legally placed under the umbrella of the attorney/client privilege, however that is accomplished in your jurisdiction. You cannot rely on any other privilege to safeguard confidential communications. Consider the use of a contract, similar to those used in multi defendant cases, to assure that the person communicating with the client agrees to be part of the defense team and to be bound by the attorney/client privilege.

2. An Investigator.

In the best of all worlds, you hire a private investigator with unlimited funds from the client's trust account. In the real world, money will be very tight and must be saved for those physicians you can't get to donate their time. You still need an investigator. Find someone to go with you during interviews, like your secretary or paralegal. College students studying a law related field, psychology, or speech communication, will welcome the opportunity to work for you – free. If it's too late in the semester for the student to enroll in an internship course, they can still receive college

credit for the experience through an independent study (which will focus on a non privileged aspect of work on your case) next semester. If you have any friends left after accepting “that child abuse case,” recruit a friend with good communication instincts. You can teach the rest easily.

Whether to document the interview literally, or from investigator field notes, or not at all, is a strategic decision. You might want to keep this in mind: it is my experience that in child abuse/death cases allegiances often change. A person who is 100% behind the client turns against him. A witness who was 100% against the client, becomes your best ally. It is certainly a complicating factor when trying to decide how to best document investigative interviews.

3. A Transcriber.

You will receive tape recorded statements of witnesses (and all too often your client) through discovery. Mostly they are not transcribed, and only the inculpatory facts seem to make their way into the police report. Get them transcribed. If the statements are transcribed, you need a transcriber to listen closely to the tapes. My transcribers routinely find an incredible amount of exculpatory evidence from those portions of the transcript labeled “unintelligible.” In many transcriptions, people’s names get mixed up, so that what was said by “X” is attributed to “Y.” What is transcribed as a “yes” (uh huh) is clearly a “no” (uh uh) when your transcriber watches the video and sees the witness shake her head no.

Most transcribers omit many important speech cues, cues that need to be documented in the transcript. For example, a child who giggles while describing how her father broke her femur conveys a far different message from the child who cries, or who

has a catch in her breath describing the same even. A pause before an answer can change the context of the words surrounding the pause. False starts in speech (change a word in midstream, starting a sentence over) may be important too. Speeding up or slowing down speech rate can add meaning to (or subtract meaning from) a conversation. Train your transcriber to include these important points in the transcription. During a trial the jury may hear the audio tape, but during deliberations days or weeks later, the jury may rely on the transcript. If so, you certainly want the transcript to convey **all** of the exculpatory material within the conversation, verbal and nonverbal.

4. Medical Consultants.

When a child's death or injury/illness is attributed to a client, mastering the medical evidence will be of paramount importance. It has often been my experience that I have no idea which of the medical facts are truly inculpatory and which of the medical facts are truly exculpatory until I achieve a fairly high level of competence with a given area of medicine (since my husband's a physician, that's not too tough for me – if Steve wants dinner, that is!) Sometimes evidence I feel is very damaging to the client, turns out to be the strongest defense material, and vice versa. Very few prosecutors or police officers delve much below the surface of the medical evidence. If there is any hope of convincing a prosecutor to not file criminal charges, or to dismiss those filed, you must have enough mastery of the discipline to not only understand, but to teach the medicine to the prosecution. When the prosecutors come to understand the medical evidence and that they are going to lose the case, you win. You will need members of the medical profession to teach you, to teach the prosecutors, and to teach jurors. The question is, how do you find someone who will help?

There is a dichotomy in physicians in terms of the likelihood that the physician will be open minded in cases of child abuse. Certain sub-specialists tend to be more prosecution oriented, the pediatricians, and county coroner – type pathologists. Generally the physician, nurse, etc... who views herself as a protector of children, will be useless to you, at least initially. The sub-specialists who treat adults as well as children tend to take a more scientific look and are therefore more objective. So, if the injuries are to the head, look for a neurosurgeon. If there are broken bones, look for an orthopedic surgeon or a radiologist. If there is damage to the eye, look for an ophthalmologist. If you suspect bleeding complications, look for an hematologist.

The same dichotomy is true for scientific publications. Scientific publications by the “child advocates” about child abuse show a distinct prosecution bias. If, however, you break down the allegation against your client to its medical parts and research each independently, you will see a very different picture. For example, a camp counselor says that the child fell from a tree. The child dies and the head at autopsy shows signs of trauma, is very swollen, and there is diffuse bleeding throughout various parts of the body. We know the coroner’s cause of death already. Now, independently research head trauma, edema, and diffuse bleeding. You will find that it is much more likely that the child died from DIC (a well documented hematological phenomenon discussed under the “Illnesses” section herein) related to accidental trauma, such as a fall from a tree, than from multiple intentionally inflicted blows to the head.

Money is always a problem, especially where a consulting physician wants \$400.00/hour (with an 8 hour minimum) to help you. Physicians who come to believe in a client’s innocence may well donate their fees and pay their own travel

expenses, or at least reduce their fees significantly. Treating physicians may testify, or may have to testify, with very little payment because they are in fact witnesses just like anyone else. Occasionally some of the treating physicians believe that the child had an accident or an illness, and are indignant that someone has been charged with a crime. If that is the case, you need to look no further. Where money is tight, an honest conversation with the doctor about money can produce some surprising and creative solutions. In one case, a frank conversation convinced the physician to require only that the client donate \$1,000.00 to an organization dedicated to child abuse prevention. At debriefing, jurors said that the donation made them disposed to believe both the physician and the client.

Make sure that all of the physicians you consult understand that they are initially just **consultants** to help you understand the medical evidence, and **not** expert witnesses. Place them under the protective attorney/client privilege. This avoids making someone an expert witness before you know that they can be of assistance. Once the consultation does become a witness, then make sure you meticulously follow the court's discovery order to avoid a risk of not being able to use the physician.

The Internet is an unending resource for physicians who may help. Go to the National Library of Medicine, a branch of the National Institute of Health, www.nih.gov. Wind your way through the website until you find the search engine. Enter any combination of key words such as child abuse – head injury – lucid interval – retinal hemorrhage – spiral fracture – metabolic acidemia. You will be able to review an abstract of every medical article in the world for your subject matter. The National Library of Medicine is changing its format to allow more of the articles to be read and

printed online at no cost. From the articles, you find your consultants/experts. If the physicians are far from you, call them. They will know the helpful people in your geographic area. You'll probably have to call/write and leave many messages before a completed telephone call. Don't get discouraged.

UNDERSTANDING THE ANATOMY

While there are an endless variety of types of child abuse accusations, most fall into one of five areas, with some overlapping – injuries to the head, the eye, broken bones, soft tissue injuries, and bleeding. Prosecutors sometimes call the injuries Shaken Baby Syndrome or Battered Child Syndrome (either acute or chronic.) To prove that your client did not shake a baby to death or batter the child, you have to understand the “syndromes” and then be able to show an alternate mechanism of illness or injury to the child. (At the end of these materials I have enclosed a list of medical texts and journal articles I frequently consult. Some are prosecution oriented, some defense oriented. Not all are cited in these materials.)

ANATOMY AND PHYSIOLOGY

Before launching into a discussion of illnesses, accidents and intentionally inflicted injuries, we all must have at least a basic knowledge of the anatomical structures of an infant or child. This includes the anatomy of the scalp, skull, tissues between the skull and brain, the brain itself and the vascular and nerve structures of the head, as well as the anatomy of the eye, neck and upper spine.

The discussion on anatomy is not meant to be complete. Dr. Gray will most certainly roll over in his grave knowing that I have omitted many structures which

are not particularly important to the discussion at hand. Gray and Goss, Gray's Anatomy, 28th ed., Lea & Febiger, 1966. But it will give you a good start.

1. The Skull and Brain

At birth, a child's skull is pliable, so that it may pass through the birth canal. The skull itself is made up of seven different plates and the base of the skull. Gray's Anatomy at 161. These plates come together at sutures lines. These sutures have not yet fused in an infant, another accommodation for birth. Thus, there are open areas on the skull known as the fontanelles. The seven skull plates are still grossly or microscopically separated from one another. (We call the fontanelles "soft spots" or in Spanish "las molleras," the most well known being the one right on top of the skull.) An infant's skull itself is thinner than an adults or older child's. Gray's Anatomy at 161 – 162. A baby's brain is also very soft for several reasons. At birth, nerves that form much of the baby's brain are not well myelinated. That fatty tissue, myelin, which wraps around the nerves like a steal belted radial, are not well developed. Indeed, it is completely absent for some nerve processes, or axons, also called white matter, of the brain. The unmyelinated tissues are much softer than the well mylenated white matter of the older child/adult. The very young brain also has a much higher water content. The higher water content softens the brain's texture. Gray's Anatomy at 806- 809.

We all know that an infant's head wobbles and flops around for the baby's first two or three months, and thereafter becomes progressively steadier. In response we take great care to support the baby's head. There are several reasons for the floppy head. An infant's skull is disproportionately large compared to the rest of the body in infancy, while the face is small and in proportion. At birth the child's head weighs about 15% of

its body weight; an adult's brain weight is about 2-3% of its body weight. The bony parts of the base of the skull (which help prevent easy rotational movement of the brain within the skull) are absent in a baby, allowing the brain to rotate within the skull with relative ease. The weak neck muscles (the superficial and lateral cervical, suprahyoid and infrahyoid, the anterior and lateral vertebral muscle groups) allow the entire head to move about without much resistance. Gray's Anatomy at 394-399, 404. All of these factors make a baby's head much more vulnerable to injury, and makes the injuries more serious than in an older child or in an adult. E. Gilles, *Abusive Head Injury in Children: A Review*, 20(2) West. St. Univ. L.R. 335,340 (1993)

The skull and contents are covered by the scalp which is made up of skin, fibroadipose tissue (under the skin,) galea aponeurotica, loose subaponeurotic tissue (under the galea,) and the periosteum (the layer of tissue closest to the bone.) Gray's Anatomy at 49. The scalp can be pulled or sheared away from the skull creating bleeding, known as a subgaleal hemorrhage. A blow to the head can cause a subgaleal hemorrhage, as can vigorous hair pulling. H. Hamlin, *Subgaleal Hematomas Caused by Hair-Pull*, 204 JAMA 129 (1968)

Surrounding the brain are two layers of connective tissue. The outer layer is the tough dura mater (the dura.) The inner layer is made up of the pia mater (the pia) and the arachnoid mater (the arachnoid.) Together the connective tissue is referred to as the meninges. In the space between the pia and the arachnoid floats the cerebral spinal fluid, which also bathes all of the cranial nerves including the optic nerve. The cerebral fluid leaves the brain at the base and follows the entire course of the spine, also bathing all of the spinal nerves. *Id.* Thus, bleeding which has made its way into the cerebral

spinal fluid in the head may be detected by a spinal tap at the cervical (neck) spine and occasionally even in the lumbar (lower back) spine, on the optic nerve. Gray's Anatomy, at 886 – 887, 912. Proportionately, the ventricles and subarachnoid spaces of a baby's brain contain more cerebrospinal fluid than an adults. The added fluid allows the baby's brain to shift faster and farther during shaking, which increases the damage. Chiocca, *Shaken Baby Syndrome: A Nursing Perspective*, Vol. 21, No. 1 Pediatric Nursing (1995)

Hemorrhages between the skull and the brain are defined by their location vis a vis the meninges. Thus, an epidural hemorrhage lies between the scalp and the dura mater; an arachnoid hematoma between the dura mater and the arachnoid; and, a subdural hemorrhage between the dura and the brain itself.

The brain itself is divided into two hemispheres, the left and the right. Between the hemispheres in a depression which runs along the midline of the brain from the middle of the forehead to the area near the middle of the base of the skull. Gray's Anatomy at 795. Blood is supplied to the brain by two pairs of arteries, the internal carotids in front and vertebral arteries in the back. Segments of cerebral veins cross the left and right hemispheres of the brain within the meninges and are called bridging veins. Gray's Anatomy at 690. Nerve fibers, known as the corpus callosum, also cross or bridge both hemispheres of the brain. Gray's Anatomy at 792, 851. The optic nerve, which is the second cranial nerve, leaves the front of the brain directly behind the eyes and enters the eye from the back of each eye. These veins and nerve fibers which cross both hemispheres of the brain, as well as the optic nerve, are particularly sensitive to damage by rapid acceleration/deceleration of the head. This violent motion causes the blood vessels to tear and the nerves to stretch/tear at the location of the bridging, because there

is less underlying brain tissue for support, and because the nerves lack much of the protection myelin coating. (See, T.A. Gennarelli & L.E. Thibault, *Biomechanics of Acute Subdural Hematoma*, 22 J. Trauma 680 (1982) for an excellent discussion of the subdural injuries from the perspective of the discipline of physics.)

2. The Eye.

The bulbs which we call eyes are attached to the skull by various muscles and connect to the brain via the optic nerve. Gray's Anatomy at 912. The optic nerve is surrounded by the protective optic nerve sheath which contains dura and arachnoid, the arachnoid being connected to the subarachnoid spaces of the brain. Id. Gilles at 365. The size and shape of the eye is maintained by the sclera, a tough, inelastic membrane which encapsulates the inner structures of the eye. The inside of the eye is composed of several structures, which from front to back are the cornea, the anterior chamber, the iris, the lens, and the large vitreous. The retina is a thin and delicate nervous membrane which begins at the back of the back of the eye in continuity with the optic nerve, and then wraps around much of the outer circumference of the vitreous body, about three quarters of the way up to the lens. The retina itself is divided into ten microscopic layers. Gray's Anatomy at 1051, 1052.

The retinal layers are fed by an extensive network of capillary veins and arteries. The capillaries are the smallest of the blood vessels, and are only one cell circumference, or said another way, the vessels are so small/narrow that they only allow one blood cell at a time to pass through. This capillary bed is alternately fed and drained by the central retinal artery and vein. Both the central retinal artery and vein run for part of their course through the center of the optic nerve. The central retinal vein then drains

into the cavernous sinus or the superior ophthalmic vein. B. Kaur & D. Taylor, *Retinal Hemorrhages*, 65 Archives Dis. Children 1369 (1990.) Because of the relatively closed nature of the eye, the retinal circulation has limited ability to adapt or respond to changes in pressure within the eye. Gray's Anatomy, Id. The capillary walls within the eye are so fragile that an increase in ocular pressure will cause the walls to break, or leak out a small amount of blood. This leakage produces a pinprick sized hemorrhage, called a petechiae, in the optic nerve or in the eye.

SHAKEN BABY SYNDROME (SBS) AKA "SHAKE AND SLAM" OR "SHAKE AND IMPACT": WHAT "THEY" SAY

*Guard well your baby's precious head
Shake, jerk and slap it never,
Lest you bruise his brain and twist his mind,
And whiplash him dead, forever. John Caffey, 1974*

When a child is violently shaken the head accelerates rapidly forward or backward, decelerates, stops, and accelerates rapidly in the opposite direction. The unique physiology of a baby or young child, described above, allow for the certain of a certain cluster of injuries now known as Shaken Baby Syndrome. Giles, E., *Abusive Head Injury in Children; A Review*, 20(2) WestStULRev 334 (1993) Caffey, J., *The Whiplash Shaken Infant Syndrome: Manual Shaking by the Extremities with Whiplash Induced Intracranial and Intraocular Bleeding Linked with Residual Permanent Damage and Mental Retardation*, 54(4) Pediatrics 396 (1974); Caffey, J., *On the Theory and Practice of Shaking Infants*, 124 AJDC 161 (1972) The injuries typically involve bleeding inside the head (subdural and/or subarachnoid hemorrhages,) a swollen brain (cerebral edema) pinprick sized hemorrhages (petechiae) of the eye and optic nerve, retinal hemorrhages, skull fractures (shake and slam injuries,) bulging fontalles and

increased space between skull sutures in response to high intracranial pressure, as well as white matters (of the brain) tears. The diagnosis of Shaken Baby Syndrome is made when the physician sees a collection of symptoms, **none** of which **alone** is conclusive evidence that the baby has been violently shaken. E. Chiocca, *Shaken Baby Syndrome: A Nursing Perspective*, 21 (1) Pediatric Nursing 48 (1995.) The difficulty lies in separating out accidental trauma from inflicted trauma, and illnesses from trauma.

As the shaken child's head whiplashes back and forth, blood vessels surrounding the brain tear, causing a subdural and/or subarachnoid hemorrhages. The bridging vessels, discussed above, are particularly vulnerable to tearing as they are not as well cushioned as others. Various nerves and nerve fibers in and around the brain also stretch and tear, causing independent neurological damage. If the nerves are torn, the neurological damage is irreversible. J. Hume, *Diffuse Brain Damage of immediate Impact Type: Its Relationship to Primary Brain-Stem Damage in Head Injury*, 100 Brain 489, 500 (1977); Chiocca at 49.

PETECHIAE IN THE EYES/OPTIC NERVE OF SHAKEN BABIES

Petechiae (pinprick sized hemorrhages) to the eye and the optic nerve has been coined a hallmark of Shaken Baby Syndrome. A.C. Duhaime et al., *Head Injury in Very Young Children: Mechanisms, Injury Types and Ophthalmologic Findings in 100 Hospitalized Patients Younger than 2 Years of Age*, Pediatrics 179 (1) Regular bruises (purpurae) and big bruises (hematomae) are caused by trauma – hitting the body and breaking blood vessels. Petechiae are different. They are caused by venous pressure. (Remember that arteries pump blood from the heart and veins, return blood to the heart.)

Something presses on the vein and does not allow the blood to flow back to the heart. The blood backs up in the blood veins, like cars in rush hour traffic.

The smallest blood vein is a capillary, a vein so small it is only one blood cell wide. If there is too much pressure on the capillary, the blood cells leak out in very small amounts causing a bruise the size of a pin prick – a petechiae. Hematology at 1256.

Prosecutors will argue that petechiae, especially along the optic nerve and the retina, are commonly found in shaken babies. There are several hypothesized mechanisms for this – increase intracranial pressure, tearing of bridging veins with the optic nerve sheath, and hydraulic pressure. Gilles at 372. As the pressure on the brain increases, the pressure may be transmitted to the subarachnoid space with in the optic nerve. The bridging veins themselves may tear, or shear off, which in turn puts pressure of the retinal vessels, causing the capillaries to leak, resulting in petechiae. Hydraulic pressures from the acceleration/deceleration forces may also account for larger retinal hemorrhages. *Id.*

In reality, any number of non abuse mechanisms, including accidents and illnesses, can cause the bleeding, and they are explored later.

ACCIDENTAL TRAUMA THAT MIMICS SHAKEN BABY

Babies may be accidentally injured in a number of ways that mimic an intentional battering. The younger the baby, the harder you should look to birth injuries as the true cause.

TRAUMA AT BIRTH

As a baby's large head passes through the birth canal, the soft bones of the skull move and mold to allow the birth. As the baby is pushed out naturally by the mother, or pulled out artificially by the obstetrician and attending instruments, the head can be injured. Up to 1/3 of all babies sustain subgaleal, epidural, subdural, subarachnoid and intracranial hemorrhages during birth. These hemorrhages leave hematomas, or blood clots. K. Shapiro & L.P. Smith, Jr., *Special Considerations for the Pediatric Age Group In Head Injury*, P. Cooper ed., 3d ed. 1993. R.H. Hovind, *Traumatic Birth Injuries in Head Injuries in the Newborn and Infant*, A.J. Raimondi et al., eds., 1986.

The hematomas calcify and persist in the brain. They will show up on subsequent brain imaging studies as "old bleeds." Anecdotally, physicians will tell you that the old hematomas can spontaneously rebleed for up to two years. Sometimes the children have been in the hospital during a rebleed, ruling out child abuse and anything other than the most minor of trauma as the cause of the rebleed. Get the mother and the child's labor and delivery and neonatal medical records to see if the hematoma could have been birth related and therefore a widely seen birth complication.

Obtain and carefully review ALL of the child's medical records from the prenatal period on. A minor subdural hematoma, caused accidentally or intentionally, can mimic the flu. So, you are looking in the medical records for a doctor visit for something that looks like a virus, infection, like the flu – symptoms of fever, malaise, nausea, headache. If you find records that the child was seen in the emergency room for "flu" a week before your client appeared on scene, it may well be that you can establish that the bleeding event happened before your client even knew the child.

It is easy to confuse the spontaneous rebleed of a hemorrhage suffered at birth with child abuse, or the flu with a minor subdural bleed. CT scans or MRI's show the old bleed, and a spontaneous rebleeding should be suspected if a new hematoma overlies the old. If the child died, at autopsy any competent pathologist will try to determine the age of the hematoma.

Infants also suffer skull fractures during birth. They are almost always linear and not depressed. The fractures occur whether the child was delivered vaginally, with the use of forceps, or by cesarean section. K. Shapiro & L.P. Smith, Jr., *Special Considerations for the Pediatric Age Group*, in Head Injury (P. Cooper ed., 3d ed. 1993.)

Retinal hemorrhages occur in up to 40% of newborn babies, due to the pressure of child birth. The statistics differ for premature deliveries, and those by cesarean section. W. Smith, et al., *Magnetic Resonance Imaging Evaluation of Neonates with Retinal Hemorrhages*, 89 Pediatrics 332 (1); J. Schenker & G. Gombos, *Retinal Hemorrhages in the Newborn*, 49 Am J Ophthalmology 1005 (1966) The birth related hemorrhages occur for the same mechanical reason as do shaking related hemorrhages, pressure. One cannot therefore make a diagnosis of SBS in a young infant based upon retinal hemorrhages. Most (but not all) resolve within a week or so, which means that birth trauma as the cause for retinal hemorrhage is a consideration only in the very young infant. F. Sezen, *Retinal Hemorrhages in Newborn Infants*, 55 Brit J. Ophthalmology 248 (1970)

ACCIDENTAL TRAUMA: SHORT DISTANCE FALLS

Physicians have long said that short distance accidental falls simply do

not result in death. Chadwick, D., Chin S. Salerno C.; et al., *Deaths from Falls in Children: How far is Fatal? J Trauma* 1991;31:1353-5. Accidental short distance falls they say do not cause retinal hemorrhages. Rather, retinal hemorrhages are said to be virtually diagnostic of inflicted trauma. Amaya M. Bechtel K. Blau SD et al. *Shaken Baby Syndrome and the Death of Matthew Esppen*. (November 11, 1997.) (Available at www.silcon.com/&thlscim:ptave/shaken.htm.) Lucid intervals after an accidental lethal fall were formerly considered nigh impossible. William KY Bank DE. Sena M. Chadwick DL. *Restricting the Time of Injury in Fatal Inflicted Head Injury. Child Abuse Negl.* 1997;21:929-40. Only a brave few suggested taking a more cautious approach to making a diagnosis of inflicted trauma. Hall JR. Reyes HM. Horvat M. et al. *The Morality of Childhood Falls. J Trauma* 1989;29:1273-5. Rieber GD *Fatal Falls in Childhood: How far must children fall to Sustain Fatal Head Injury: Report of Cases and Review of the Literature. Am. J. Forensic Med Pathol* 1993;14:201-7. Root I. *Head Injuries from Short Distance Falls. Am J Forensick Med Pathol* 1995;16:154-7. This minority view was soundly criticized because the childrens' falls in the studies happened at home and were witnessed only by the caretakers – the often targets for abusers. Lyons TJ. Oates RK. *Falling out of Bed: A Relatively Benign Occurrence. Pediatrics* 1993;92:125-7. Swalwell C. *Head Injuries From Short Distance Falls. Am J Forensic Med Pathol* 1993;14:171-2.

Dr. John Plunkett set out to begin to resolve the disagreement on these issues. He searched the United States Consumer Product Safety Commission, the National Injury Information Clearinghouse, and the National Electronic Injury

Information Clearinghouse for head injuries to children caused by playground equipment.

He was particularly interested in:

1) falls witnessed by noncaretakers; 2) short distance playground falls; 3) falls that resulted in the child's death; 4) falls where the child had a lucid intervals after the accident; and 5) retinal hemorrhages after an accident. John Plunkett, M.D., *Fatal Pediatric Head Injuries Caused by Short Distance Falls*.

Dr. Plunkett's statistical review turned up eighteen short distance fall playground head injury fatalities. Twelve of the eighteen falls were witnessed by a noncaretaker. Four of the six who were examined for retinal hemorrhages, had bilateral retinal hemorrhages. Twelve had lucid intervals after the fall and before death. Plunkett, *Fatal Pediatric Head Injuries Caused by Short Distance Falls*, at 3-7.

To say that he has taken "heat" for his study, is an understatement.

Most of the children in the Plunkett study were diagnosed with malignant cerebral edema. Plunkett Article at pages 3-7. Malignant cerebral edema, or in slang terms, a "malignant brain" happens like this.

A child has a head injury. There is some bleeding in the head. Once bleeding begins in the closed confines of the cranial cavity, a rather complicated, but predictable chain of events can be set in motion.

First, the intracranial bleeding itself begins to take up more and more of the space usually occupied by the brain. The bleeding takes the form of a subdural and/or subarachnoid hemorrhage which causes a hematoma. The hematoma alone may cause brain damage by squeezing the brain into too small a space, depriving the brain of oxygen (ischemia.)

Second, the hematoma puts pressure on whichever cerebral vessels which have torn. This tamponades, or stops by pressure, the bleeding, much as if one had tightly pressed a bandage on a cut finger. If there is enough pressure at this point, blood flow may be cut off causing ischemia (lack of oxygen) which leads to tissue necrosis (death.)

Third, the body senses that the fluid balance in the area of the bleeding has been upset. The abnormal collection of clotting blood in the cranium is too thick for the body's liking.

Fourth, the body in response to the fluid imbalance, oozes through osmosis, a diluted watery solution to the affected area.

Fifth, the presence of additional watery fluid in the cranial cavity puts additional pressure on the brain, causing increased ischemia and necrosis. This cycle causes more swelling within the brain (edema,) less space, and the cycle begins all over again. At any point in this cycle, cerebral edema (a malignant brain) may occur, causing necrosis and then death. With a little necrosis, some of the brain dies and there is irreversible brain damage; with more necrosis there is death. E. Aldrich et al., *Diffuse Brain Swelling in Severely Head-Injured Children*, 76 J. Neurosurgery 450 (1); A.C. Duhaime et al., *The Shaken Baby Syndrome: A Clinical, Pathological and Biomechanical Study*, 66 J. Neurosurgery 409 (1987)

ACCIDENTAL TRAUMA: INFANT WALKERS/STAIRCASE FALLS

A child falling down a flight of stairs (particularly in an infant walker or similar device) can be seriously hurt, and occasionally the injuries may be fatal. In walker injuries, the bottom half of the child's body is held in, and somewhat protected by

the walker. The arc of the fall, its velocity, and multiple impacts, tumbling of the walker, can result in very serious injury to the child. M.D. Partington et al., *Head Injury and the Use of Baby Walkers: A Continuing Problem*, 20 *Annals Emergency Medicine* 652 (1991); S. Wellman & J.A., Paulson, *Baby Walker-Related Injuries*, 23 *Clinical Pediatrics* 98 (1984) Comb the staircase for corroborating physical evidence of the fall. Are there paint transfers from the wall? Are there walker marks on the wall? Are there traces of blood along the staircase? They will corroborate accidental trauma.

ILLNESSES AND COMPLICATIONS THAT MIMIC RETINAL HEMORRHAGES IN SHAKEN BABIES

Retinal hemorrhages are found as a complication to leukemia, infection, bleeding disorders, hemophilia, diabetes, sickle cell anemia, high altitude sickness, congenital malformations, carbon monoxide poisoning, anemia, papilledema (increased cerebral pressure) and trauma (accidental or inflicted.) Hoffman, Benz, Shattil, Fure & Cohen, Hematology, Basis Principles and Practice, Churchill Livingstone, NY, NY; 1991 @ 1485-1490; 1724, 207-208. Spontaneous subarachnoid hemorrhages, which continue into the optic nerve arachnoid space, do occur in infants, though many authors claim they are exceedingly rare. Gilles at page 371. Many authors swear that resuscitative efforts cannot cause ocular hemorrhages, but at least two studies document the presence of retinal hemorrhages with CPR being the most likely case. C. Bacon et al., *Extensive Retinal Hemorrhages in an Infancy – An Innocent Cause*, 1 *Brit. J Med* 281 (1978); V. Weedn et al., *Retinal Hemorrhages in an Infant After Cardiopulmonary Resuscitation*, *Am. J. Forensic Med Pathology* 79 (1990) Accidental trauma has also been shown to cause retinal hemorrhages in children. Plunkett, *Fatal Pediatric Head Injuries Caused by Short Distance Falls*. How often they occur is still an open question.

J. Elder et al., *Retinal Hemorrhage in Accident Head Trauma in Childhood*, 27 J.

Pediatric Child Health 286 (1991) Not surprisingly, bungee jumpers too suffer retinal hemorrhages. David DB, Mears T. Quinlan MP *Ocular Complications Associated with Bungee Jumping*. Ophthalmol 1994;78:234-5. Jain BK, Talbot EM. *Bungee Jumping and Intraocular Hemorrhage*. BR J Ophthalmol 1994;78:236-7. Thus it is not at all a “sure bet” that retinal hemorrhages are caused by inflicted trauma. Edlow JA, Caplan LR. *Avoiding Pitfalls in the Diagnosis of Subarachnoid Hemorrhage*. N. Engl J Med 2000;342:29-36. Look for the cause of the bleeding by considering all possibilities. Eliminate them one by one until you are left with just a few choices, some of which will be accident, illness, or hematology complications.

ACCIDENTAL TRAUMA: BUNKBED/SWING/ “MONKEY BAR” FALLS

Children falling off the top bunk, from a swing, or “monkey bars,” most often escape serious injury, but that is not always the case. A child may fall off of a top bunk while asleep or be pushed/thrown during play. Infants tumble out of baby swings. Children fall on the playground. Children taken to the emergency room for such falls often present with head and facial injuries. In one study, one of the children sustained a fractured skull and subdural hematoma from such a fall. Selbst et al., *Bunk Bed Injuries*, 144 AJDC 721 (1990)

Memorize Dr. Plunkett’s paper on deaths from falls on the playground.

He gives an excellent discussion of the physics of the mechanism of accidental injury that refutes what other authors say (that short distance falls cannot cause death.) His statistical review of playground accidents proves that short distance accidental falls do

produce enough force to cause serious injury and death. They do. Plunkett Article at pages 7-11. They also look much like shaking injuries.

WALK, TALK AND DIE or TALK AND DETERIORATE/DIE "TADD" – THE LUCID INTERVAL AND ACCIDENTAL TRAUMA

Consider for a moment the possibility that the child was beaten or violently shaken and that the battering caused the observed injuries. Assume that your client did not abuse the child, but that the child was left in another's care. When the client came home the baby seemed fine for an interval of time. Or, assume that the child fell from the "monkey bars" at school and cried but seemed fine. Hours later, at home, the child slowly (or quickly) became lethargic, less responsive, unconscious, and eventually died. Perhaps the child was resuscitated and recovered. If your client was with the child when the symptoms appeared, brought the child to the emergency room, and had no explanation for the injuries, the client is the prime suspect (scratch that – client is the ONLY suspect.) Prosecutors (and some pathologists) will chant their mantra that "these are forceful injuries like those seen in motor vehicle accidents. Children all lose consciousness immediately and never regain consciousness. Thus, your client is a liar and guilty. Not so.

The walk, talk, and die phenomenon or TADD, has long been associated with adult head injuries. Rockswold GL. Leonard RP. Nagib MG. *Analysis of Management in Thirty-three Closed Head Injury Patients Who "Talked and Deteriorated."* Neurosurgery 1987;21:51-5. Just a few reports suggested that walk, talk and die occurred with children as well. A. Bruce et al., *Diffuse Cerebral Swelling Following Head Injuries in Children: The Syndrome of "Malignant Brain Edema,"* 54 J. Neurosurgery 170 (1981) Snoek JW, Minderhoud JM Wilmink JT. *Delayed*

Deterioration Following Mild Head Injury In Children. Brain 1984:107:15-36. Dr Plunkett found that twelve of the eighteen children in his study had a lucid interval (they walked and talked/cried) before death. (Plunkett article at pages 3, 8, 9) Clinical histories of children with head trauma in the Bruce article showed that 37% of the group had a clear history of a lucid period following trauma (they walk and talk and tragically die.) Bruce article at 176. In many cases where the walk, talk and die phenomenon has been documented, deaths were attributable to causes other than immediate impact injury. J. Adams, et al., *Diffuse Brain Damage of Immediate Impact Type: Its Relationship to Primary Brain-Stem Damage in Head Injury*, 100 Brain 489 (1977)

Almost all of the literature published on the Shaken Baby Syndrome says that with SBS there can be no lucid interval, no walk, talk and die phenomenon. However, the time interval between the shaking and the onset of symptoms had never been scrutinized until Dr. Plunkett's review of accident data bases. There still is simply not enough detailed clinical histories available to determine how often children have a lucid interval between the battering and the onset of symptoms. M. Nashelsky, *The Time Interval Between Lethal Infant Shaking and Onset of Symptoms: A Review of the Shaken Baby Literature*, 16(2) A.J. Forensic Med. & Path. 154 (1995) It is certainly higher than previous estimates.

Where your client's defense relies on the walk, talk and die phenomenon, it will be critical to make sure, if there is intracranial bleeding, that a great deal of care is taken to accurately determine the type and age of each hematoma. Munro & Merritt, *Surgical Pathology of Subdural Hematoma*, 35 Arch. Neuro. & Psychiat. 64 (1936) You

may be able to prove that the bleeding began before or after your client cared for the child, notwithstanding the prosecutor's view to the contrary.

DISSEMINATED INTRAVASCULAR COAGULOPATHY, DIC A COMPLICATION THAT MIMICS CHILD ABUSE

DIC is a bleeding disorder. It is most often secondary to an underlying medical problem, either illness or trauma. The disorders which cause DIC fall into three main categories. The first is a disorder which causes the release of procoagulant substances, such as certain snake bites, certain cancers and head trauma. The second is contact between blood and an injured or abnormal surface, such as extensive burns, infections, heat stroke, and organ grafts. The third is the creation of procoagulant active substances within the blood. This occurs where red and white blood cells themselves are damaged, by leukemia, blood transfusions, gram-negative infections or hemolytic anemia, and traumas. Hematology, Chapter 13, 1394-1405; Cecil, Textbook of Medicine, 17th Edition, 914 1031, 1035, 1053-1055 (1985)

For our purposes it is enough to know that accidental head trauma does cause DIC. In children, up to 25% of all patients with head trauma show signs of DIC.

To see if the child showed signs of DIC, get ALL of the child's medical records, from EMT, to ambulance ride, hospital number one, helicopter ambulance, and hospital number two. Look for laboratory blood coagulation studies. There are several lab tests -- platelet count, prothrombin time (PT) and partial thromboplastin time (PTT, PLT or PLT-CT) fibrinogen (FIB) and fibrinogen split products (FSP.) Are those tests abnormal? If so, the child may have DIC. Hematology at 1396-1397. In the physician and nurses' notes, see if there is any documentation that needle stick sites, or IV sites bled or oozed inordinantly, or that minute trauma, such as rubbing an arm, leg, head or

chest caused a significant bruise. Do wounds continue to bleed after suturing? It may be that in the emergency room physicians found that the child had only one bruise, let's say on the forehead. Three hours later, after being air ambulated to the ICU of a larger hospital, the child had multiple bruises, and bleeding to several organs, caused not by the initial trauma, but by DIC. However the presentation, **DIC can mimic child abuse.**

As a complicating factor in head trauma, DIC biochemically works like this. The brain is rich in thromboplastin (clot activating chemicals.) The head trauma can cause the massive release of the thromboplastin substances, which can initiate blood clotting. Once all of the blood's clotting factors are exhausted, the blood vessels begin to bleed. (After clotting factors are all used up, at autopsy, or via CAT scan, you may not see blood clots, just bleeding.) The bleeding which results from DIC may be any type of bleeding. It may be frank hemorrhage, ecchymotic (large bruises) purpuric (small bruises) or petechial (pinprick sized bruises.) Any of these types of bleeding may be throughout the entire body, or localized to any area or combination of areas of the body. DIC is often severe enough to cause death, but in a milder form can be reversed with treatment, or can be self reversing where the instigating factor either ceases to exist or is corrected by giving the patient blood products.

A single blow to the head, with relatively minor cerebral trauma, if complicated by DIC, can be confused with inflicted head trauma, the kind caused by severe battering or Shaken Baby Syndrome. How does that happen? A single blow to the head, or minor head trauma causes the DIC. The DIC causes more serious bleeding within the cranium, which causes cerebral edema, which causes ischemia, which causes necrosis, which causes death. The "DIC" child's head may present as soft, massively

swollen and extensively bruised inside and outside of the skull. The question then becomes, did inflicted trauma and severe bleeding cause the DIC? Or, did minor trauma, or a single accidental blow cause the DIC? Sometimes through the careful construction of a time line which correlates symptoms with blood clotting studies, you can prove that it was the DIC which caused the great majority of the damage and not the trauma.

Where DIC is involved, and was diagnosed, there will probably be disagreement among treating physicians as to the initial cause of the DIC – accident or abuse. If the DIC went undiagnosed, but you are able to prove it in hindsight, then you are likely to see the same difference of opinion among doctors, with the child advocates on one side, and subspecialists on the other.

ILLNESSES THAT MIMIC CHILD ABUSE

There are a surprising number of illnesses that mimic battering and the Shaken Baby Syndrome. All are uncommon. All have led to innocent people being accused of child abuse.

1. Glutaric Acidemia, GA-1.

GA-1 is an inherited disease, and is autosomal recessive genetically. That means that both parents must pass the gene for GA-1 to the child for the child to have the disease. Carriers of the disease will show no symptoms. Although rare, the disease is more common among those of Amish heritage.

Children with GA-1 are unable to process certain amino acids and thus build up abnormal levels of glutaric acid in their bodies. When the acid reaches a toxic level, the children become lethargic, vomit, have fevers, seizures, collect fluid beneath the dura, and have retinal bleeding. They look like shaken children. Eventually, if

untreated, these children lapse into a coma and die. There is a relatively inexpensive test to identify GA-1 which you may want to consider. Nelson et al., Textbook on Pediatrics, Twelfth Edition, at 433 (1983)

2. Methylmalonic Acidemia.

Methylmalonic acid, like GA-1 is an enzyme which the normal child can break down and metabolize. When infants cannot metabolize the enzyme, a toxic level of methylmalonic acid builds up. Children with this disorder often present as failure to thrive infants, often with vomiting. Many children have repeated episodes of metabolic acidosis. There are many variants of methylmalonic acidemia, most of which are related to a deficiency of vitamin B12.

As genetic screening for inherited metabolic disorders increases, more children are being diagnosed with disorder, some of whom respond to treatment with vitamin B12, and some of whom do not. Nelson, Pediatrics, at page 434.

3. Schonlein-Henoch Syndrome.

Characteristics of Schonlein-Henoch Syndrome are a rash, with or without soft tissue swelling. With kidney involvement, the child will present with hemolytic-uremic syndrome, which is associated with purpura (midsized bruising) secondary to thrombocytopenia. The diagnosis of Schonlein-Henoch Syndrome is not an easy one and may require biopsy of the purpura. The biopsy will reveal the characteristic leukocytoclastic vasculitis, deposits of IGA, and fibrin in the vessels and surrounding tissue.

These children present with bruising without an explanation, and that puts caregivers at risk for an accusation of battering the child.

4. Vascular Hemophilia (Von Willebrand Disease)

This inherited disease is exceedingly complex and characterized by decreased levels of (clotting) Factor VIII. Children with Von Willebrand disease have nosebleeds, bleeding from gums, oozing cuts, and increased bleeding time after surgery. When these children get an infection, or are exposed to a toxin, they easily develop ITP (idiopathic thrombocytopenia) with pinprick sized hemorrhages, or TTP (thrombocytopenia purpura,) with small bruises as a manifestation. Neslon, Pediatrics at 1246, 1520. If the disease, especially in a mild form, has gone undiagnosed, these children will also have bruising that cannot be accounted for. Caretakers are then charged with child abuse.

5. ITP and TTP as a Complication of a Virus Infection.

A child has a viral or infectious disease (flu) and seems to recover. Days to a couple of weeks later the child has pinprick – sized bruises (ITP) or medium bruises/clots (TTP.) In one family I know the two year old had the flu and developed ITP. She almost died. Fifteen years later her sister had a bad cold and developed blood clots in both kidneys (TTP.) The older girl survived but needed a kidney transplant.

This bleeding and bruising can look very much like injuries from abuse. A bone marrow is diagnostic, but if doctors believe the child has been abused, they may choose not to do a bone marrow test. Hematology 207-208; 1485-1490.

CONCLUSION

This paper only scratches the surface of alternatives to the diagnosis of abuse. It is meant to give you enough of an understanding of most of the accidents and illnesses that masquerade as child abuse. When a shaken baby or battered child case comes your

way, you will have a starting point for much hard/tedious work. Don't forget to follow up on the Internet in the National Library of Medicine.

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Fatal Pediatric Head Injuries Caused by Short-Distance Falls

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Physicians disagree on several issues regarding head injury in infants and children, including the potential lethality of a short-distance fall, a lucid interval in an ultimately fatal head injury, and the specificity of retinal hemorrhage for inflicted trauma. There is scant objective evidence to resolve these questions, and more information is needed. The objective of this study was to determine whether there are witnessed or investigated fatal short-distance falls that were concluded to be accidental. The author reviewed the January 1, 1988 through June 30, 1999 United States Consumer Product Safety Commission database for head injury associated with the use of playground equipment. The author obtained and reviewed the primary source data (hospital and emergency medical services' records, law enforcement reports, and coroner or medical examiner records) for all fatalities involving a fall.

The results revealed 18 fall-related head injury fatalities in the database. The youngest child was 12 months old, the oldest 13 years. The falls were from 0.6 to 3 meters (2-10 feet). A noncaretaker witnessed 12 of the 18, and 12 had a lucid interval. Four of the six children in whom funduscopic examination was documented in the medical record had bilateral retinal hemorrhage. The author concludes that an infant or child may suffer a fatal head injury from a fall of less than 3 meters (10 feet). The injury may be associated with a lucid interval and bilateral retinal hemorrhage.

Key Words: Child abuse—Head injury—Lucid interval—Retinal hemorrhage—Subdural hematomas.

Many physicians believe that a simple fall cannot cause serious injury or death (1-9), that a lucid interval does not exist in an ultimately fatal pediatric head injury (7-13), and that retinal hemorrhage is highly suggestive if not diagnostic for inflicted trauma (7,12,14-21). However, several have questioned these conclusions or urged caution when interpreting head injury in a child (15,22-28). This controversy exists because most infant injuries occur in the home (29,30), and if there is history of a fall, it is usually not witnessed or is seen only by the caretaker. Objective data are needed to resolve this dispute. It would be helpful if there were a database of fatal falls that were witnessed or wherein medical and law enforcement investigation unequivocally concluded that the death was an accident.

The United States Consumer Product Safety Commission (CPSC) National Injury Information Clearinghouse uses four computerized data sources (31). The National Electronic Injury Surveillance System (NEISS) file collects current injury data associated with 15,000 categories of consumer products from 101 U.S. hospital emergency departments, including 9 pediatric hospitals. The file is a probability sample and is used to estimate the number and types of consumer product-related injuries each year (32). The Death Certificate (DC) file is a demographic summary created by information provided to the CPSC by selected U.S. State Health Departments. The Injury/Potential Injury Incident (IR) file contains summaries, indexed by consumer product, of reports to the CPSC from consumers, medical examiners and coroners (Medical Examiner and Coroner Alert Project [MECAP]), and newspaper accounts of product-related incidents discovered by local or regional CPSC staff (33). The In-Depth Investigations (AI) file contains summaries of investigations performed by CPSC staff based on reports received from the NEISS, DC, or IR files (34). The AI files provide details about the incident from victim and witness interviews, accident reconstruction, and review of law enforce-

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ment, health care facility, and coroner or medical examiner records (if a death occurred).

METHODS

I reviewed the CPSC, DC, IR, and AI files for all head and neck injuries involving playground equipment recorded by the CPSC from January 1, 1988 through June 30, 1999. There are 323 entries in the playground equipment IR file, 262 in the AI file, 47 in the DC file, and more than 75,000 in the NEISS file. All deaths in the NEISS file generated an IR or AI file. If the file indicated that a death had occurred from a fall, I obtained and reviewed each original source record from law enforcement, hospitals, emergency medical services (EMS), and coroner or medical examiner offices except for one autopsy report. However, I discussed the autopsy findings with the pathologist in this case.

RESULTS

There are 114 deaths in the Clearinghouse database, 18 of which were due to head injury from a fall. The following deaths were excluded from this study: those that involved equipment that broke or collapsed, striking a person on the head or neck (41); those in which a person became entangled in the equipment and suffocated or was strangled (45); those that involved equipment or incidents other than playground (6 [including a 13.7-meter fall from a homemade Ferris wheel and a 3-meter fall from a cyclone fence adjacent to a playground]); and falls in which the death was caused exclusively by neck (carotid vessel, airway, or cervical spinal cord) injury (4).

The falls were from horizontal ladders (4), swings (7), stationary platforms (3), a ladder attached to a slide, a "see-saw", a slide, and a retaining wall. Thirteen occurred on a school or public playground, and five occurred at home. The database is not limited to infants and children, but a 13-year-old was the oldest fatality (range, 12 months-13 years; mean, 5.2 years; median, 4.5 years). The distance of the fall, defined as the distance of the closest body part from the ground at the beginning of the fall, could be determined from CPSC or law enforcement reconstruction and actual measurement in 10 cases and was 0.6 to 3.0 meters (mean, 1.3 ± 0.77 ; median, 0.9). The distance could not be accurately determined in the seven fatalities involving swings and one of the falls from a horizontal ladder, and may have been from as little as 0.6 meters to as much as 2.4 meters. The maximum height for a fall from a swing was assumed to

be the highest point of the arc. Twelve of the 18 falls were witnessed by a noncaretaker or were videotaped; 12 of the children had a lucid interval (5 minutes-48 hours); and 4 of the 6 in whom fundoscopic examination was performed had bilateral retinal hemorrhage (Table 1).

CASES

Case 1

This 12-month-old was seated on a porch swing between her mother and father when the chain on her mother's side broke and all three fell sideways and backwards 1.5 to 1.8 meters (5-6 feet) onto decorative rocks in front of the porch. The mother fell first, then the child, then her father. It is not known if her father landed on top of her or if she struck only the ground. She was unconscious immediately. EMS was called; she was taken to a local hospital; and was icteric and had decerebrate posturing in the emergency room. She was intubated, hyperventilated, and treated with mannitol. A computed tomography (CT) scan indicated a subgaleal hematoma at the vertex of the skull, a comminuted fracture of the vault, parafalcine subdural hemorrhage, and right parietal subarachnoid hemorrhage. There was also acute cerebral edema with effacement of the right frontal horn and compression of the basal cisterns. She had a cardiopulmonary arrest while the CT scan was being done and could not be resuscitated.

Case 2

A 14-month-old was on a backyard "see-saw" and was being held in place by his grandmother. The grandmother said that she was distracted for a moment and he fell backward, striking the grass-covered ground 0.6 meters (22.5 inches) below the plastic seat. He was conscious but crying, and she carried him into the house. Within 10 to 15 minutes he became lethargic and limp, vomited, and was taken to the local hospital by EMS personnel. He was unconscious but purposefully moving all extremities when evaluated, and results of fundoscopic examination were normal. A CT scan indicated an occipital subgaleal hematoma, left-sided cerebral edema with complete obliteration of the left frontal horn, and small punctate hemorrhages in the left frontal lobe. There was no fracture or subdural hematoma. He was treated with mannitol; his level of consciousness rapidly improved, and he was extubated. However, approximately 7 hours after admission he began to have difficulty breathing, both pupils suddenly dilated, and he was rein-

TABLE 1. Summary of cases

No.	CPSC No.	Age	Sex	Fall from	Distance M/F	Witnessed	Luck Interval	Retinal hemorrhage	Subdural hemorrhage	Autopsy	Cause of death	FP
1	DC 9108013330	12 mos	F	Swing	1.5-1.8/5.0-6.0	No	No	N/R	Yes +IHF	No	Complex calvarial fracture with edema and contusions	No
2	AI 890208HBC3088	14 mos	M	See-saw	0.6/2.0	No	10-15 minutes	No	No	No	Malignant cerebral edema with herniation	No
3	IR F910368A	17 mos	F	Swing	1.5-1.8/5.0-6.0	No	No	N/R	Yes +IHF	Yes	Acute subdural hematoma with secondary cerebral edema	Yes
4	AI 921001HCC2263	20 mos	F	Platform	1/3.5	No	5-10 minutes	Bilateral multilayered	Yes +IHF	Limited	Occipital fracture with subdural/subarachnoid hemorrhage progressing to cerebral edema and herniation	Yes
5*	DC 9312060661	23 mos	F	Platform	0.70/2.3	Yes	10 minutes	Bilateral, NOS	Yes	Yes	Acute subdural hematoma	Yes
6	DC 8451016513	28 mos	M	Swing	0.9-1.0/3.0-6.0	Yes	No	Bilateral multilayered	Yes +IHF	Yes	Subdural hematoma with associated cerebral edema	Yes
7*	AI 891215HcC2094	3 yrs	M	Platform	0.9/3.0	Yes	10 minutes	N/R	Yes	No	Acute cerebral edema with herniation	No
8	AI 910515HCC2182	3 yrs	F	Ladder	0.6/2.0	Yes	15 minutes	N/R	Yes (autopsy only)	Yes	Complex calvarial fracture, contusions, cerebral edema with herniation	Yes
9	DC 9253029577	4 yrs	M	Slide	2.1/7.0	Yes	3 hours	N/R	No	Yes	Epidural hematoma	Yes
10	AI 920710HWE4014	5 yrs	M	Horizontal ladder	2.1/7.0	No	No	N/R	Yes	No	Acute subdural hematoma with acute cerebral edema	Yes
11	AI 990517HCC5175	6 yrs	M	Swing	0.6-2.4/2.0-8.0	No	10 minutes	No	Yes +IHF	No	Acute subdural hematoma	Yes
12	AI 970324HCC3040	6 yrs	M	Horizontal ladder	3.0/10.0	Yes	45 minutes	N/R	No	No	Malignant cerebral edema with herniation	Yes
13	AI 881229HCC3070	6 yrs	F	Horizontal ladder	0.9/3.0	Yes	1+ hour	N/R	Yes +IHF	Yes	Subdural and subarachnoid hemorrhage, cerebral infarct, and edema	Yes
14	AI 930930HWE5025	7 yrs	M	Horizontal ladder	1.2-2.4/4.0-8.0	Yes	48 hours	N/R	No	Yes	Cerebral infarct secondary to carotid/vertebral artery thrombosis	Yes
15	AI 970409HCC1096	8 yrs	F	Retaining wall	0.9/3.0	Yes	12+ hours	N/R	Yes (autopsy only)	Yes	Acute subdural hematoma	Yes
16	AI 890621HCC3195	10 yrs	M	Swing	0.9-1.5/3.0-5.0	Yes	10 minutes	Bilateral multilayered	Yes	Yes	Acute subdural hematoma contiguous with an AV malformation	No
17	AI 920428HCC1671	12 yrs	F	Swing	0.9-1.8/3.0-6.0	Yes	No	N/R	No	Yes	Occipital fracture with extensive contra-coup contusions	Yes
18	AI 891016HCC1511	13 yrs	F	Swing	0.6-1.8/2.0-6.0	Yes	No	N/R	Yes +IHF	Yes	Occipital fracture, subdural hemorrhage, cerebral edema	Yes

*The original CT scan for case #7 and the soft tissue CT windows for case #5 could not be located and were unavailable for review.

CPSC, Consumer Products Safety Commission; AI, accident investigation; IR, incident report; DC, death certificate; M, male; F, female; Distance, the distance of the closest body part from the ground at the start of the fall (see text); M/F, meters/feet; Witnessed, witnessed by a noncarer/parent or videotaped; N/R, not recorded; IHF, including interhemispheric or falx; FP, forensic pathologist-directed death investigation system.

tubated. A second CT scan demonstrated progression of the left hemispheric edema despite medical management, and he was removed from life support 22 hours after admission.

Case 3

This 17-month-old had been placed in a baby carrier-type swing attached to an overhead tree limb at a daycare provider's home. A restraining bar held in place by a snap was across her waist. She was being pushed by the daycare provider to an estimated height of 1.5 to 1.8 meters (5-6 feet) when the snap came loose. The child fell from the swing on its downstroke, striking her back and head on the grassy surface. She was immediately unconscious and apneic but then started to breathe spontaneously. EMS took her to a pediatric hospital. A CT scan indicated a large left-sided subdural hematoma with extension to the interhemispheric fissure anteriorly and throughout the length of the falx. The hematoma was surgically evacuated, but she developed malignant cerebral edema and died the following day. A postmortem examination indicated symmetrical contusions on the buttock and midline posterior thorax, consistent with impact against a flat surface; a small residual left-sided subdural hematoma; cerebral edema with anoxic encephalopathy; and uncal and cerebellar tonsillar herniation. There were no cortical contusions.

Case 4

A 20-month-old was with other family members for a reunion at a public park. She was on the platform portion of a jungle gym when she fell from the side and struck her head on one of the support posts. The platform was 1.7 meters (67 inches) above the ground and 1.1 meters (42 inches) above the top of the support post that she struck. Only her father saw the actual fall, although there were a number of other people in the immediate area. She was initially conscious and talking, but within 5 to 10 minutes became comatose. She was taken to a nearby hospital, then transferred to a tertiary-care facility. A CT scan indicated a right occipital skull fracture with approximately 4-mm of depression and subarachnoid and subdural hemorrhage along the tentorium and posterior falx. Funduscopic examination indicated extensive bilateral retinal and preretinal hemorrhage. She died 2 days later because of uncontrollable increased intracranial pressure. A limited postmortem examination indicated an impact subgaleal hematoma overlying the fracture in the mid occiput.

Case 5

A 23-month-old was playing on a plastic gym set in the garage at her home with her older brother. She had climbed the attached ladder to the top rail above the platform and was straddling the rail, with her feet 0.70 meters (28 inches) above the floor. She lost her balance and fell headfirst onto a 1-cm (3/4-inch) thick piece of plush carpet remnant covering the concrete floor. She struck the carpet first with her outstretched hands, then with the right front side of her forehead, followed by her right shoulder. Her grandmother had been watching the children play and videotaped the fall. She cried after the fall but was alert and talking. Her grandmother walked/carried her into the kitchen, where her mother gave her a baby analgesic with some water, which she drank. However, approximately 5 minutes later she vomited and became stuporous. EMS personnel airlifted her to a tertiary-care university hospital. A CT scan indicated a large right-sided subdural hematoma with effacement of the right lateral ventricle and minimal subfalcine herniation. (The soft tissue windows for the scan could not be located and were unavailable for review.) The hematoma was immediately evacuated. She remained comatose postoperatively, developed cerebral edema with herniation, and was removed from life support 36 hours after the fall. Bilateral retinal hemorrhage, not further described, was documented in a funduscopic examination performed 24 hours after admission. A postmortem examination confirmed the right frontal scalp impact injury. There was a small residual right subdural hematoma, a right parietal lobe contusion (secondary to the surgical intervention), and cerebral edema with cerebellar tonsillar herniation.

Case 6

A 26-month-old was on a playground swing being pushed by a 13-year-old cousin when he fell backward 0.9 to 1.8 meters (3-6 feet), striking his head on hard-packed soil. The 13-year-old and several other children saw the fall. He was immediately unconscious and was taken to a local emergency room, then transferred to a pediatric hospital. A CT scan indicated acute cerebral edema and a small subdural hematoma adjacent to the anterior interhemispheric falx. A funduscopic examination performed 4 hours after admission indicated extensive bilateral retinal hemorrhage, vitreous hemorrhage in the left eye, and papilledema. He had a subsequent cardiopulmonary arrest and could not be resuscitated. A postmortem examination confirmed the retinal hemorrhage and indicated a right parietal scalp impact injury but no calvarial frac-

ture, a "film" of bilateral subdural hemorrhage, cerebral edema with herniation, and focal hemorrhage in the right posterior midbrain and pons.

Case 7

This 3-year-old with a history of TAR (thrombocytopenia-absent radius) syndrome was playing with other children on playground equipment at his school when he stepped through an opening in a platform. He fell 0.9 meters (3 feet) to the hard-packed ground, striking his face. A teacher witnessed the incident. He was initially conscious and able to walk. However, approximately 10 minutes later he had projectile vomiting and became comatose, was taken to a local hospital, and subsequently transferred to a pediatric hospital. A CT scan indicated a small subdural hematoma and diffuse cerebral edema with uncal herniation, according to the admission history and physical examination. (The original CT report and scan could not be located and were unavailable for review.) His platelet count was 24,000/mm³, and he was treated empirically with platelet transfusions, although he had no evidence for an expanding extra-axial mass. Resuscitation was discontinued in the emergency room.

Case 8

This 3-year-old was at a city park with an adult neighbor and four other children, ages 6 to 10. She was standing on the third step of a slide ladder 0.6 meters (22 inches) above the ground when she fell forward onto compact dirt, striking her head. The other children but not the adult saw the fall. She was crying but did not appear to be seriously injured, and the neighbor picked her up and brought her to her parents' home. Approximately 15 minutes later she began to vomit, and her mother called EMS. She was taken to a local emergency room, then transferred to a pediatric hospital. She was initially lethargic but responded to hyperventilation and mannitol; she began to open her eyes with stimulation and to spontaneously move all extremities and was extubated. However, she developed malignant cerebral edema on the second hospital day and was reintubated and hyperventilated but died the following day. A postmortem examination indicated a subgaleal hematoma at the vertex of the skull associated with a complex fracture involving the left frontal bone and bilateral temporal bones. There were small epidural and subdural hematomas (not identifiable on the CT scan), bilateral "contrecoup" contusions of the inferior surfaces of the frontal and temporal lobes, and marked cerebral edema with uncal herniation.

Case 9

A 4-year-old fell approximately 2.1 meters (7 feet) from a playground slide at a state park, landing on the dirt ground on his buttock, then falling to his left side, striking his head. There was no loss of consciousness, but his family took him to a local emergency facility, where an evaluation was normal. However, he began vomiting and complained of left neck and head pain approximately 3 hours later. He was taken to a second hospital, where a CT scan indicated a large left parietal epidural hematoma with a midline shift. He was transferred to a pediatric hospital and the hematoma was evacuated, but he developed malignant cerebral edema with right occipital and left parietal infarcts and was removed from the respirator 10 days later. A postmortem examination indicated a small residual epidural hematoma, marked cerebral edema, bilateral cerebellar tonsillar and uncal herniation, and hypoxic encephalopathy. There was no identifiable skull fracture.

Case 10

A 5-year-old was apparently walking across the horizontal ladder of a "monkey bar," part of an interconnecting system of homemade playground equipment in his front yard, when his mother looked out one of the windows and saw him laying face down on the ground and not moving. The horizontal ladder was 2.1 meters (7 feet) above compacted dirt. EMS were called, he was taken to a local hospital, and then transferred to a pediatric hospital. A CT scan indicated a right posterior temporal linear fracture with a small underlying epidural hematoma, a 5-mm thick acute subdural hematoma along the right temporal and parietal lobes, and marked right-sided edema with a 10-mm midline shift. He was hyperventilated and treated with mannitol, but the hematoma continued to enlarge and was surgically evacuated. However, he developed uncontrollable cerebral edema and was removed from life support 10 days after the fall.

Case 11

A 6-year-old was on a playground swing at a private lodge with his 14-year-old sister. His sister heard a "thump," turned around, and saw him on the grass-covered packed earth beneath the swing. The actual fall was not witnessed. The seat of the swing was 0.6 meters (2 feet) above the ground, and the fall distance could have been from as high as 2.4 meters (8 feet). He was initially conscious and talking but within 10 minutes became comatose and was taken to a local emergency room, then transferred to a tertiary-care hospital. A CT

scan indicated a large left frontoparietal subdural hematoma with extension into the anterior interhemispheric fissure and a significant midline shift with obliteration of the left lateral ventricle. There were no retinal hemorrhages. He was treated aggressively with dexamethasone and hyperventilation, but there was no surgical intervention. He died the following day.

Case 12

This 6-year-old was at school and was sitting on the top crossbar of a "monkey bar" approximately 3 meters (10 feet) above compacted clay soil when an unrelated noncaretaker adult saw him fall from the crossbar to the ground. He landed flat on his back and initially appeared to have the wind knocked out of him but was conscious and alert. He was taken to the school nurse who applied an ice pack to a contusion on the back of his head. He rested for approximately 30 minutes in the nurse's office and was being escorted back to class when he suddenly collapsed. EMS was called, and he was transported to a pediatric hospital. He was comatose on admission, the fundi could not be visualized, and a head CT scan was interpreted as normal. However, a CT scan performed the following morning approximately 20 hours after the fall indicated diffuse cerebral edema with effacement of the basilar cisterns and fourth ventricle. There was no identifiable subdural hemorrhage or calvarial fracture. He developed transtentorial herniation and died 48 hours after the fall.

Case 13

This 6-year-old was playing on a school playground with a 5th grade student/friend. She was hand-over-hand traversing the crossbar of a "monkey bar" 2.4 meters (7 feet 10 inches) above the ground with her feet approximately 1 meter (40 inches) above the surface. She attempted to slide down the pole when she reached the end of the crossbar but lost her grip and slid quickly to the ground, striking the compacted dirt first with her feet, then her buttock and back, and finally her head. The friend informed the school principal of the incident, but the child seemed fine and there was no intervention. She went to a relative's home for after-school care approximately 30 minutes after the fall, watched TV for a while, then complained of a headache and laid down for a nap. When her parents arrived at the home later that evening, 6 hours after the incident, they discovered that she was incoherent and "drooling." EMS transported her to a tertiary-care medical center. A CT scan indicated a right parieto-occipital skull frac-

ture, subdural and subarachnoid hemorrhage, and a right cerebral hemisphere infarct. The infarct included the posterior cerebral territory and was thought most consistent with thrombosis or dissection of a right carotid artery that had a persistent fetal origin of the posterior cerebral artery. She remained comatose and was removed from the respirator 6 days after admission. A postmortem examination indicated superficial abrasions and contusions over the scapula, a prominent right parietotemporal subgaleal hematoma, and a right parietal skull fracture. She had a 50-ml subdural hematoma and cerebral edema with global hypoxic or ischemic injury ("respirator brain"), but the carotid vessels were normal.

Case 14

A 7-year-old was on the playground during school hours playing on the horizontal ladder of a "monkey bar" when he slipped and fell 1.2 to 2.4 meters (4-8 feet). According to one witness, he struck his forehead on the bars of the vertical ladder; according to another eyewitness he struck the rubber pad covering of the asphalt ground. There are conflicting stories as to whether he had an initial loss of consciousness. However, he walked back to the school, and EMS was called because of the history of the fall. He was taken to a local hospital, where evaluation indicated a Glasgow coma score of 15 and a normal CT scan except for an occipital subgaleal hematoma. He was kept overnight for observation because of the possible loss of consciousness but was released the following day. He was doing homework at home 2 days after the fall when his grandmother noticed that he was stumbling and had slurred speech, and she took him back to the hospital. A second CT scan indicated a left carotid artery occlusion and left temporal and parietal lobe infarcts. The infarcts and subsequent edema progressed; he had brainstem herniation; and he was removed from life support 3 days later (5 days after the initial fall). A postmortem examination indicated ischemic infarcts of the left parietal, temporal, and occipital lobes, acute cerebral edema with herniation, and thrombosis of the left vertebral artery. Occlusion of the carotid artery, suspected pre-mortem, could not be confirmed.

Case 15

This 8-year-old was at a public playground near her home with several friends her age. She was hanging by her hands from the horizontal ladder of a "monkey bar" with her feet approximately 1.1 meters (3.5 feet) above the ground when she attempted to swing from the bars to a nearby 0.9-

meter (34-inch) retaining wall. She landed on the top of the wall but then lost her balance and fell to the ground, either to a hard-packed surface (one witness) or to a 5.1-cm (2-inch) thick resilient rubber mat (a second witness), striking her back and head. She initially cried and complained of a headache but continued playing, then later went home. Her mother said that she seemed normal and went to bed at her usual time. However, when her mother tried to awaken her at approximately 8:30 the following morning (12 hours after the fall) she complained of a headache and went back to sleep. She awoke at 11 a.m. and complained of a severe headache then became unresponsive and had a seizure. EMS took her to a nearby hospital, but she died in the emergency room. A postmortem examination indicated a right temporoparietal subdural hematoma, extending to the base of the brain in the middle and posterior fossae, with flattening of the gyri and narrowing of the sulci. (The presence or absence of herniation is not described in the autopsy report.) There was no calvarial fracture, and there was no identifiable injury in the scalp or galea.

Case 16

A 10-year-old was swinging on a swing at his school's playground during recess when the seat detached from the chain and he fell 0.9 to 1.5 meters (3-5 feet) to the asphalt surface, striking the back of his head. The other students but not the three adult playground supervisors saw him fall. He remained conscious although groggy and was carried to the school nurse's office, where an ice pack was placed on an occipital contusion. He suddenly lost consciousness approximately 10 minutes later, and EMS took him to a local hospital. He had decerebrate posturing when initially evaluated. Funduscopic examination indicated extensive bilateral confluent and stellate, posterior and peripheral preretinal and subhyaloid hemorrhage. A CT scan showed a large acute right frontoparietal subdural hematoma with transtentorial herniation. The hematoma was surgically removed, but he developed malignant cerebral edema and died 6 days later. A postmortem examination indicated a right parietal subarachnoid AV malformation, contiguous with a small amount of residual subdural hemorrhage, and cerebral edema with anoxic encephalopathy and herniation. There was no calvarial fracture.

Case 17

A 12-year-old was at a public playground with a sister and another friend and was standing on the seat of a swing when the swing began to twist. She

lost her balance and fell 0.9 to 1.8 meters (3-6 feet) to the asphalt surface, striking her posterior thorax and occipital scalp. She was immediately unconscious and was taken to a tertiary-care hospital emergency room, where she was pronounced dead. A postmortem examination indicated an occipital impact injury associated with an extensive comminuted occipital fracture extending into both middle cranial fossa and "contra-coup" contusions of both inferior frontal and temporal lobes.

Case 18

This 13-year-old was at a public playground with a friend. She was standing on the seat of a swing with her friend seated between her legs when she lost her grip and fell backwards 0.6 to 1.8 meters (2-6 feet), striking either a concrete retaining wall adjacent to the playground or a resilient 5.1-cm (2 inch) thick rubber mat covering the ground. She was immediately unconscious and was given emergency first aid by a physician who was nearby when the fall occurred. She was taken to a nearby hospital and was purposefully moving all extremities and had reactive pupils when initially evaluated. A CT scan indicated interhemispheric subdural hemorrhage and generalized cerebral edema, which progressed rapidly to brain death. A postmortem examination indicated a linear nondepressed midline occipital skull fracture, subdural hemorrhage extending to the occiput, contusion of the left cerebellar hemisphere, bifrontal "contra-coup" contusions, and cerebral edema.

DISCUSSION

General

Traumatic brain injury (TBI) is caused by a force resulting in either strain (deformation/unit length) or stress (force/original cross-sectional area) of the scalp, skull, and brain (35-37). The extent of injury depends not only on the level and duration of force but also on the specific mechanical and geometric properties of the cranial system under loading (38-40). Different parts of the skull and brain have distinct biophysical characteristics, and calculating deformation and stress is complex. However, an applied force causes the skull and brain to move, and acceleration, the time required to reach peak acceleration, and the duration of acceleration may be measured at specific locations (36,41). These kinematic parameters do not cause the actual brain damage but are useful for analyzing TBI because they are easy to quantify. Research in TBI using physical models and animal experiments has shown that a force resulting in angular acceleration pro-

duces primarily diffuse brain damage, whereas a force causing exclusively translational acceleration produces only focal brain damage (36). A fall from a countertop or table is often considered to be exclusively translational and therefore assumed incapable of producing serious injury (3,7-9). However, sudden impact deceleration *must* have an angular vector unless the force is applied only through the center of mass (COM), and deformation of the skull during impact *must* be accompanied by a volume change (cavitation) in the subdural "space" tangential to the applied force (41). The angular and deformation factors produce tensile strains on the surface veins and mechanical distortions of the brain during impact and may cause a subdural hematoma without deep white matter injury or even unconsciousness (42-44).

Many authors state that a fall from less than 3 meters (10 feet) is rarely if ever fatal, especially if the distance is less than 1.5 meters (5 feet) (1-6,8,9). The few studies concluding that a short-distance fall may be fatal (22-24,26,27) have been criticized because the fall was not witnessed or was seen only by the caretaker. However, isolated reports of observed fatal falls and biomechanical analysis using experimental animals, adult human volunteers, and models indicate the potential for serious head injury or death from as little as a 0.6-meter (2-foot) fall (48-52). There are limited experimental studies on infants (cadaver skull fracture) (53,54) and none on living subadult nonhuman primates, but the adult data have been extrapolated to youngsters and used to develop the Hybrid III/III and Child Restraint-Air Bag Interaction (CRABI) models (55) and to propose standards for playground equipment (56,63). We simply do not know either kinematic or nonkinematic limits in the pediatric population (57,58).

Each of the falls in this study exceeded established adult kinematic thresholds for traumatic brain injury (41,48-52). Casual analysis of the falls suggests that most were primarily translational. However, deformation and *internal* angular acceleration of the skull and brain *caused by the impact* produce the injury. What happens during the impact, not during the fall, determines the outcome.

Subdural Hemorrhage

A "high strain" impact (short pulse duration and high rate for deceleration onset) typical for a fall is more likely to cause subdural hemorrhage than a "low strain" impact (long pulse duration and low rate for deceleration onset) that is typical of a motor vehicle accident (42,61). The duration of deceleration for a head-impact fall against a nonyield-

ing surface is usually less than 5 milliseconds (39,59-61). Experimentally, impact duration longer than 5 milliseconds will not cause a subdural hematoma unless the level of angular acceleration is above $1.75 \times 10^5 \text{ rad/s}^2$ (61). A body in motion with an angular acceleration of $1.75 \times 10^5 \text{ rad/s}^2$ has a tangential acceleration of $17,500 \text{ m/s}^2$ at 0.1 meters (the distance from the midneck axis of rotation to the midbrain COM in the Duhaime model). A human cannot produce this level of acceleration by impulse ("shake") loading (62).

An injury resulting in a subdural hematoma in an infant may be caused by an accidental fall (43,44,64). A recent report documented the findings in seven children seen in a pediatric hospital emergency room after an accidental fall of 0.6 to 1.5 meters who had subdural hemorrhage, no loss of consciousness, and no symptoms (44). The characteristics of the hemorrhage, especially extension into the posterior interhemispheric fissure, have been used to suggest if not confirm that the injury was nonaccidental (9,62,65-68). The hemorrhage extended into the posterior interhemispheric fissure in 5 of the 10 children in this study (in whom the blood was identifiable on CT or magnetic resonance scans and the scans were available for review) and along the anterior falx or anterior interhemispheric fissure in an additional 2 of the 10.

Lucid Interval

Disruption of the diencephalic and midbrain portions of the reticular activating system (RAS) causes unconsciousness (36,69,70). "Shearing" or "diffuse axonal" injury (DAI) is thought to be the primary biophysical mechanism for immediate traumatic unconsciousness (36,71). Axonal injury has been confirmed at autopsy in persons who had a brief loss of consciousness after a head injury and who later died from other causes, such as coronary artery disease (72). However, if unconsciousness is momentary or brief ("concussion") subsequent deterioration *must* be due to a mechanism other than DAI. Apnea and catecholamine release have been suggested as significant factors in the outcome following head injury (73,74). In addition, the centripetal theory of traumatic unconsciousness states that primary disruption of the RAS will not occur in isolation and that structural brainstem damage from inertial (impulse) or impact (contact) loading *must* be accompanied by evidence for cortical and subcortical damage (36). This theory has been validated by magnetic resonance imaging and CT scans in adults and children (75,76). Only one of the children in this study (case 6) had evidence for any component of DAI. This child had focal hemor-

rhage in the posterior midbrain and pons, thought by the pathologist to be primary, although there was no skull fracture, only "a film" of subdural hemorrhage, no tears in the corpus callosum, and no lacerations of the cerebral white matter (grossly or microscopically).

The usual cause for delayed deterioration in infants and children is cerebral edema, whereas in adults it is an expanding extra-axial hematoma (77). If the mechanism for delayed deterioration (except for an expanding extra-axial mass) is venospasm, cerebral edema may be the only morphologic marker. The "talk and die or deteriorate (TADD)" syndrome is well characterized in adults (78). Two reports in the pediatric literature discuss TADD, documenting 4 fatalities among 105 children who had a lucid interval after head injury and subsequently deteriorated (77,79). Many physicians believe that a lucid interval in an ultimately fatal pediatric head injury is extremely unlikely or does not occur unless there is an epidural hematoma (7,8,11). Twelve children in this study had a lucid interval. A noncaretaker witnessed 9 of these 12 falls. One child had an epidural hematoma.

Retinal Hemorrhage

The majority of published studies conclude that retinal hemorrhage, especially if bilateral and posterior or associated with retinoschisis, is highly suggestive of, if not diagnostic for, nonaccidental injury (9,14-21). Rarely, retinal hemorrhage has been associated with an accidental head injury, but in these cases the bleeding was unilateral (80). It is also stated that traumatic retinal hemorrhage may be the direct mechanical effect of violent shaking (15). However, retinal hemorrhage may be caused experimentally either by ligating the central retinal vein or its tributaries or by suddenly increasing intracranial pressure (81,82); retinoschisis is the result of breakthrough bleeding and venous stasis not "violent shaking" (15,83). Any sudden increase in intracranial pressure may cause retinal hemorrhage (84-87). Deformation of the skull coincident to an impact nonselectively increases intracranial pressure. Venospasm secondary to traumatic brain injury selectively increases venous pressure. Either mechanism may cause retinal hemorrhage irrespective of whether the trauma was accidental or inflicted. Further, retinal and optic nerve sheath hemorrhages associated with a ruptured vascular malformation are due to an increase in venous pressure not extension of blood along extravascular spaces (81-83,88). Dilated eye examination with an indirect ophthalmoscope is thought to be more sensitive for detecting retinal bleeding than routine ex-

amination and has been recommended as part of the evaluation of any pediatric patient with head trauma (89). None of the children in this study had a formal retinal evaluation, and only six had fundoscopic examination documented in the medical record. Four of the six had bilateral retinal hemorrhage.

Pre-existing Conditions

One of these children (case 16) had a subarachnoid AV malformation that contributed to development of the subdural hematoma, causing his death. One (case 7) had TAR syndrome (90), but his death was thought to be caused by malignant cerebral edema not an expanding extra-axial mass.

Cerebrovascular Thrombosis

Thrombosis or dissection of carotid or vertebral arteries as a cause of delayed deterioration after head or neck injuries is documented in both adults and children (91,92). Case 14 is the first report of a death due to traumatic cerebrovascular thrombosis in an infant or child. Internal carotid artery thrombosis was suggested radiographically in an additional death (case 13) but could not be confirmed at autopsy. However, this child died 6 days after admission to the hospital, and fibrinolysis may have removed any evidence for thrombosis at the time the autopsy was performed.

Limitations

1. Six of the 18 falls were not witnessed or were seen only by the adult caretaker, and it is possible that another person caused the nonobserved injuries.
2. The exact height of the fall could be determined in only 10 cases. The others (7 swing and 1 stationary platform) could have been from as little as 0.6 meters (2 feet) to as much as 2.4 meters (8 feet).
3. A minimum impact velocity sufficient to cause fatal brain injury cannot be inferred from this study. Likewise, the probability that an individual fall will have a fatal outcome cannot be stated because the database depends on voluntary reporting and contractual agreements with selected U.S. state agencies. The NEISS summaries for the study years estimated that there were more than 250 deaths due to head and neck injuries associated with playground equipment, but there are only 114 in the files. Further, this study does not include other nonplayground equipment-related fatal falls, witnessed or not witnessed, in the CPSC database (32).

CONCLUSIONS

1. Every fall is a complex event. There must be a biomechanical analysis for any incident in which the severity of the injury appears to be inconsistent with the history. The question is not "Can an infant or child be seriously injured or killed from a short-distance fall?" but rather "If a child falls (x) meters and strikes his or her head on a nonyielding surface, what will happen?"
2. Retinal hemorrhage may occur whenever intracranial-pressure exceeds venous pressure or whenever there is venous obstruction. The characteristic of the bleeding cannot be used to determine the ultimate cause.
3. Axonal damage is unlikely to be the mechanism for lethal injury in a low-velocity impact such as from a fall.
4. Cerebrovascular thrombosis or dissection must be considered in any injury with apparent delayed deterioration, and especially in one with a cerebral infarct or an unusual distribution for cerebral edema.
5. A fall from less than 3 meters (10 feet) in an infant or child may cause fatal head injury and may not cause immediate symptoms. The injury may be associated with bilateral retinal hemorrhage, and an associated subdural hematoma may extend into the interhemispheric fissure. A history by the caretaker that the child may have fallen cannot be dismissed.

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APPENDIX

Newtonian mechanics involving constant acceleration may be used to determine the impact velocity in a gravitational fall. However, constant acceleration formulas cannot be used to calculate the relations among velocity, acceleration, and distance traveled *during* an impact because the deceleration

is not uniform (45). This analysis requires awareness of the shape of the deceleration curve, knowledge of the mechanical properties and geometry of the cranial system, and comprehension of the stress and strain characteristics for the specific part of the skull and brain that strikes the ground. A purely translational fall requires that the body is rigid and that the external forces acting on the body pass only through the COM, i.e., there is no rotational component. A 1-meter-tall 3-year-old hanging by her knees from a horizontal ladder with the vertex of her skull 0.5 meters above hard-packed earth approximates this model. If she loses her grip and falls, striking the occipital scalp, her impact velocity is 3.1 m/second. An exclusively angular fall also requires that the body is rigid. In addition, the rotation must be about a fixed axis or a given point internal or external to the body, and the applied moment and the inertial moment must be at the identical point or axis. If this same child has a 0.5-meter COM and has a "matchstick" fall while standing on the ground, again striking her occiput, her angular velocity is 5.42 rad/second and tangential velocity 5.42 m/second at impact. The impact velocity is higher than predicted for an exclusively translational or external-axis angular fall when the applied moment and the inertial moment are at a different fixed point (slip and fall) or when the initial velocity is not zero (walking or running, then trip and fall), and the vectors are additive. However, the head, neck, limbs, and torso do not move uniformly during a fall because relative motion occurs with different velocities and accelerations for each component. Calculation of the impact velocity for an actual fall requires solutions of differential equations for each simultaneous translational and rotational motion (45). Further, inertial or impulse loading (whiplash) may cause head acceleration more than twice that of the midbody input force and may be important in a fall where the initial impact is to the feet, buttock, back, or shoulder, and the final impact is to the head (46,47).

The translational motion of a rigid body at constant gravitational acceleration (9.8 m/s^2) is calculated from:

$$F = ma \quad v^2 = 2as \quad v = at$$

where F = the sum of all forces acting on the body (newton), m = mass (kg), a = acceleration (m/s^2), v = velocity (m/s), s = distance (m), and t = time (s).

The angular motion of a rigid body about a fixed axis at a given point of the body under constant gravitational acceleration (9.8 m/s^2) is calculated from:

$$M = I\alpha \quad \omega = v/r \quad \alpha = a/r$$

where M = the applied moment about the COM or about the fixed point where the axis of rotation is located, I = the inertial moment about this same COM or fixed point, α = angular acceleration (rad/s^2), ω = angular velocity (rad/s), r = radius (m), v^t = tangential velocity (m/s), and a^t = tangential acceleration (m/s^2).

The angular velocity ω for a rigid body of length L rotating about a fixed point is calculated from:

$$\frac{1}{2}I_0\omega^2 = maL/2 \quad I_0 = (1/3) mL^2$$

where I_0 = the initial inertial moment, ω = angular velocity (rad/s), m = mass (kg), a = gravitational acceleration (9.8 m/s^2), and L = length.

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