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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 hematoma.

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 $\pm$ 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 funduscopic 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 ictal 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 funduscopic 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	Lucid 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	1.1/3.5	No	5–10 minutes	Bilateral multilayered	Yes +IHF	Limited	Occipital fracture with subdural/subarachnoid hemorrhage progressing to cerebral edema and herniation	Yes
5[a]	DC 9312060661	23 mos	F	Platform	0.70/2.3	Yes	10 minutes	Bilateral, NOS	Yes	Yes	Acute subdural hematoma	Yes
6	DC 9451016513	26 mos	M	Swing	0.9–1.8/3.0–6.0	Yes	No	Bilateral multilayered	Yes +IHF	Yes	Subdural hematoma with associated cerebral edema	Yes
7[a]	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 9253024577	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 960517HCC5175	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

[a]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 noncaretaker 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 (⅜-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/mm^3, 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 "contracoup" 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 fracture, 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 premortem, 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 to1.8 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 II/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 nonyielding 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 1.75×10^5 rad/s^2 (61). A body in motion with an angular acceleration of 1.75×10^5 rad/s^2 has a tangential acceleration of 17,500 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 examination 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 funduscopic 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 looses 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:

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

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:

$$M = I\alpha \qquad \omega = v^t/r \qquad \alpha = a^t/r$$

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:

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

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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Addendum

This death was reported to and investigated by the CPSC after submission of the manuscript.

Case 19. A 7 year old boy was playing at a school playground, hanging by his hands from the gymnastic rings of a wooden play structure with his feet approximately 30 inches above the ground. Another child grabbed him by the legs, forcing him to let go, and he fell face-first onto 7 inches of wood mulch, which had been placed on the playground surface the day before. The fall was witnessed by several other children and adults. He seemed uninjured and continued playing. However, 10-15 minutes later he complained of a headache and went to the school nurse's office to lay down. He had a seizure and lost consciousness while in the office, was transported to a local hospital, then transferred to a tertiary care children's hospital. A CT scan indicated acute occipito-parietal subdural hemorrhage, extending along the tentorium and posterior interhemispheric fissure. The hematoma was emergently evacuated, but he had a cardiopulmonary arrest in the operating room and could not be resuscitated. A postmortem examination indicated residual subdural hemorrhage at the base of the skull. He had no impact injury in his scalp, consistent with the history of a face-first fall, and had no identifiable facial lacerations or abrasions. The neck and cervical spinal cord examination were normal. The eyes were not examined.

John Plunkett, M.D.
September 12, 2000

Is gene therapy ready for HIV/Ebola virus-derived viral vectors?

In 1999, an investigation into the death of Jesse Gelsinger, who died while particpating in a gene theapy trial, severely criticised James Wilson, director of the Institute of Gene Therapy at the University of Pennsylvania (Philadelphia, PA, USA). The controversy is revisited this month with the publication of a paper by Wilson's laboratory reporting the development of an HIV-based viral vector that carries envelope proteins from the Ebola virus.

The group suggest that the new vector, EboZ, which efficiently transduces intact airway epithelium in vitro and in vivo, may form the basis of an effective gene therapy for cystic fibrosis. "At a time when gene therapists, the FDA, and many others in the field are struggling for restoration of public confidence in gene therapy, one might question the approach of creating a 'strange bug' instead of optimising the known viral or even nonviral transfer technologies", says Wolfgang Walther (Max-Delbrück-Center for Molecular Medicine, Berlin, Germany).

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Using Ebola envelope proteins

Wilson's team created vectors that incorporated various viral envelope proteins and showed that a vector containing envelope proteins from the Zaire strain of Ebola virus was the most effective transducer of cultured apical airway cells in culture. Further in-vitro experiments on excised sections of healthy human trachea demonstrated transduction of tracheal epithelial cells by the EboZ vector. This was followed up by in-vivo experiments in which the vector was introduced into the tracheas of immunocompetent young mice—the animals had high-levels of vector expression by day 28, that persisted until at least day 63 (*Nat Biotech* 2001; **19:** 225–30).

"The EboZ vector construct serves as a research tool and provides the means to ask if there is a single epitope in the Ebola virus envelope that is critical for binding the receptors on a respiratory epithelial cell", stresses Nelson A Wivel, deputy director of the Institute of Gene Therapy in Philadelphia. "The idea of employing Ebola envelopes to achieve transduction of airway epithelium—the natural target of Ebola infection—is intriguing", agrees Walther, but he warns that safety concerns are under-represented in the study. "At least one experiment should have investigated whether cell types other than epithelial cells can be infected by the new vector", he says. A scenario of efficient but unwanted infection of other tissues could rule out use of the vector for human gene therapy, he adds.

Gaetano Romano (Thomas Jefferson University, Philadelphia, PA, USA) also warns that a major drawback of HIV-based vectors is the seroconversion to HIV. He also points out that insertion of the viral vector into the genome of human cells and possible recombination between retroviridae-based vectors and human endogenous retroviruses need to be considered. Insertional mutagenesis could be avoided by engineering self-inactivating vectors but Romano notes that "the transfer vector used by Wilson belongs to the early generations of HIV-based vectors, which are not self-inactivating." "Obviously, many more studies need to be done and the question of a clinical trial is very remote in our thinking at this juncture", says Wivel.

Kathryn Senior

Accident or murder in children?

In 1998, there were several well-publicised trials of child carers who were accused of killing children in their care by shaking them. Experts for the prosecution gave evidence that there were features of the fatal event, and physical signs in the children, that were diagnostic of inflicted injury, although the evidence underlying their assertions was slight. Many observers, including *The Lancet* (1998; 352: 335), expressed concern at this deficiency.

John Plunkett from the Regina Medical Center, Hastings MN, USA, examined the records of the United States Consumer Products Safety Commission between January, 1988, and June, 1999, to find the records of children who died after short falls (0·6–3 m) from playground equipment (*Am J Forensic Med Pathol* 2001; 22: 1–12). 18 children were identified, aged 12 months to 13 years. Legal investigations concluded that death was accidental in all cases. A non-caretaker witnessed 12 of the accidents, and in the 13th the fall was videotaped by the child's grandmother.

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Kevin Harrison/Medipics

Is retinal haemorrhage diagnostic?

"Many physicians believe that . . . a lucid interval does not exist in an ultimately fatal paediatric head injury", says Plunkett, yet 12 of the 18 children who died had a lucid interval lasting from 5 min to 48 h. Four of the six children whose fundi were examined had bilateral retinal haemorrhages, which contradicts the assumption, "that retinal haemorrhage is highly suggestive, if not diagnostic, of inflicted trauma".

The author's conclusion that, "a history by the caretaker that the child may have fallen cannot be dismissed", is likely to echo through courtrooms for many years to come. He is more forthright in person about the issue: "I am genuinely distressed at what medicine has done in the arena of child abuse. Even a cursory understanding of the biomechanics of brain trauma would have predicted the results I was fortunately able to document . . . It [the publication] has already been 'trashed' by many paediatricians and ophthalmologists, and the journal was not mailed until last Friday [Feb 23]! It has even been suggested that the videotape of the fatal short-distance fall was fabricated."

John Bignall

The American Journal of Forensic Medicine and Pathology 22(3):332–336, 2001.

Letters to the Editor

Fatal Pediatric Head Injuries Caused by Short-Distance Falls

To the Editor:

The case series drawn from Consumer Product Safety Commission (CPSC) databases (1) is the largest series of reasonably well-described deaths in childhood resulting from falls of less than 10 feet. Only a few other cases have been this well reported (2,3). For this reason, therefore, it is unfortunate that not one computed tomography scan or autopsy photograph was included to amplify or elucidate any of the reported findings. Similarly, the lack of detailed neuropathologic descriptions limits the reader's ability to define mechanism of injury. For example, four of the seven cases with skull fracture had fractures involving the occipital bone (cases 4, 13, 17, and 18). In three of these four (cases 4, 13, and 18), acute subdural hematoma (ASDH) was part of the reported findings. Fractures in the occipital bone have the potential for laceration of the sagittal or transverse sinus. This would cause a large mass-effect, subdural hematoma arising as a contact injury. This should be contrasted with ASDH, which results from the rupture of bridging veins and which may happen focally due to contact forces or more widely due to inertial forces.

The series establishes the extreme rarity of death from head injury in young children who fall short distances. The CPSC reports that over 200,000 children are seen in emergency departments each year because of injuries incurred while using playground equipment. Sixty percent (approximately 120,000) of these are the result of falls (4). Over the 11.5-year period covered in Plunkett's report, more than 1.3 million such emergency department visits would be expected. However, only 18 deaths from head injury were identified. This yields a rate of 1.3 deaths per 100,000 such falls. In fact, since we may presume that most children who fall from playground equipment are not brought to an emergency department, the fatality rate must be considerably lower than this. By anyone's standards, such deaths are very rare events. Included in this small series are two cases (cases 7 and 16) in which natural causes (thrombocytopenia–absent radii syndrome, intracranial A-V malformation) may have increased susceptibility to fatal head injury. If these two cases are excluded, the fatality rate drops to 1.16 per 100,000 children brought to an emergency department after a playground equipment fall.

The series also establishes that skull fracture is much less commonly associated with fatal head injury in children than in adults. Eleven (61%) of the 18 children described by Plunkett had no skull fracture (cases 2, 3, 5–7, 9, 11, 12, 14–16), despite falls of 0.6 to 2.4 meters (approximately 2–8 feet). This finding is in agreement with the few reports of fatal falls from short distances that include enough pathologic description to ensure their reasonable validity. In the three cases described by Claydon (2) and Reiber (3), none of the children had skull fractures.

The findings in this series once again cast serious doubt on the validity of the two papers by Weber (5,6) cited in this report. If Weber's conclusions were valid, all of these children should have had skull fractures. A recent paper (7) establishes the ability of the infant skull to deform without fracture and, in fact, indicates that such transient deformation may actually contribute to the severity of a head injury associated with impact.

The article states that all of the 18 cases reported here reached expected biomechanical thresholds for traumatic brain injury in adults, but no such analysis is provided in even one case. Without such a description, there is no way to evaluate the validity of the analysis.

It is extremely interesting that 6 of the 18 cases (cases 1, 3, 6, 16–18) involved falls from swings. It is unfortunate that Plunkett did not take the opportunity to discuss the physics of swing injuries. With regard to linear deceleration at impact, the distance from the ground at the moment the child falls from the swing is inconsequential. The determining factor in the terminal velocity prior to impact will be the maximum height attained during the swing cycle. As the swing descends through the arc, increasing amounts of the potential energy are transformed into kinetic energy and therefore into linear velocity. Thus, the energy available at impact, and

hence the terminal velocity, is equivalent to a fall from the maximum height of the arc. In fact, the nadir of the swing cycle is associated with the highest angular velocity. When a child falls from a swing, he begins to rotate around his own center of gravity, rather than around the support beam, which may be as much as 8 feet from the swing seat. This leads to an immediate and marked decrease in the moment of inertia. Preservation of angular momentum, then, will cause the child's angular velocity to increase. If the child has fallen from the swing at a point low in the descending arc, when angular velocity is higher to begin with, this further increase in angular velocity may lead to very high angular decelerations at impact. Since angular acceleration and velocity are highly correlated with severity of diffuse brain injury (8,9), a fall from the lower ⅓ of the swing cycle may be more injurious than a fall from the apex of the arc. It is significant that five (cases 1, 3, 6, 17, and 18) of the six children who fell from swings were immediately unconscious, and the remaining child (case 16) was "groggy." This is consistent with diffuse brain injury arising from inertial forces. Only one child (case 10) who was not playing on a swing appears to have lost consciousness initially. This fall was not observed, and no conclusions about mechanism of injury can be made.

Twelve (67%) of the 18 cases in this series had a lucid interval (cases 2, 4, 5, 7–9, 11–16). However, in eight of these cases, the lucid interval was less than 1 hour (cases 2, 4, 5, 7, 8, 11, 12, and 16). One child (case 14) had a lucid interval of 2 days. This is by far the longest lucid interval ever described in a child. There is no precedent either in this series or in the papers cited (10,11) for a child who appears well and suddenly deteriorates many days or even weeks after apparently minor head trauma, a claim that is sometimes made in court.

The frequency of lucid intervals in this series correlates well with the infrequency of evidence of traumatic axonal injury on computed tomography scan or autopsy; it was recognized only in case 6 (5.6%), who fell from a swing. This is again in contrast with findings in children who have died of abusive head trauma (12,13) where traumatic axonal injury is common, particularly in the lower brainstem and upper spinal cord. With such findings, one would expect immediate loss of consciousness to be the rule rather than the exception.

The discussion of the literature concerning retinal hemorrhage and head injury in childhood is inadequate and misleading. The literature concerning the rarity of retinal hemorrhages in severe accidental head injury in childhood is extensive (14–16). These studies ably demonstrate that fewer than 2% of children with significant accidental head injuries have retinal hemorrhages when examined under optimal conditions by a pediatric ophthalmologist (3 of 209 in the combined series). In addition, the description of at least one of the retinal hemorrhages reported by Plunkett is probably erroneous. The report indicates that, in case 16, the hemorrhages extended to the periphery. Since the author stated elsewhere that none of the children had formal eye exams, it must be assumed that the fundus was examined by direct, rather than indirect, ophthalmoscopy. The periphery can be examined only by indirect ophthalmoscopy. If the physician responsible for describing the retinal hemorrhages was unaware of this, the validity of the rest of the description, in this case at least, is open to serious question. The author correctly identifies traumatic retinoschisis as a finding highly correlated with abusive head trauma. This finding was not described in any of the patients in the series. The paper cites the recommendation that children with serious head injury be examined by indirect ophthalmoscopy (17). Such examination would have clarified the extent and nature of retinal injury in this unusual group of patients.

The cited paper by Greenes and Schutzman (18) is misrepresented. This study of young children who were admitted to the hospital after detection of intracranial injuries revealed 19 children who had no neurologic symptoms, 7 of whom had subdural hematomas. However, Plunkett failed to indicate that 18 (95%) of the 19 children had a large scalp hematoma and skull fracture, injuries that are contact in origin. The visible scalp hematoma would have provided evidence to their caretakers that an injury had occurred. None of the 19 children deteriorated in hospital, although 1 had a post-traumatic seizure, which was well controlled with anticonvulsant therapy. All were discharged in good condition. Two-week follow-up of all cases revealed no subsequent deterioration.

This contrasts sharply with the dismal outcome noted in follow-up of infants who have suffered abusive head trauma. In Bonnier et al.'s study (19), 13 children diagnosed with abusive head trauma with a presumptive mechanism of shaking were followed for 5 to 13 years. Of these children, one died in the acute period, five were quadriplegic, blind and severely retarded, and one had delayed onset of epilepsy; all of these were diagnosed within the first year after injury. These children continued to be profoundly impaired during the follow-up period, and one suffered delayed death. At autopsy of this child, 10 separate tears were noted in the brainstem and cerebral hemispheres. Five of the six children

who were felt to be "normal" in the first year after injury had been identified as having significant disability by school age: two were hemiparetic; five were mentally retarded; and three had severe behavioral disorders. Only one still appeared to be normal. The extremely high morbidity and mortality are not unexpected with severe diffuse brain injuries caused by high inertial loads.

Plunkett's series adds to the knowledge concerning a rare but real event, childhood death from a short fall. However, it would be wrong and dangerous to draw conclusions from these deaths, which primarily resulted from serious contact forces, and apply those conclusions to a very different group of infants who die from shaking and/or shaking associated with impact, who have injuries primarily resulting from inertial forces. The differences between these two populations are well documented in the literature and are apparent in the findings in this report.

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To the Editor:

This communication is intended to address the biomechanical issues contained in the letter to the Editor by Betty Spivack, M.D., that critiqued the article by John Plunkett entitled, "Fatal Pediatric Head Injuries Caused by Short Distance Falls," which appeared in the recent issue of the American Journal of Forensic Medicine and Pathology. Its concepts are closely related to the work that she presented at the Third Shaken Baby Syndrome Symposium, with particular reference to the section labeled, "The biomechanics of Childhood Falls." The latter dissemination is either incorrect, incomplete, or out of context. Spivack's attempt to simplify this phenomenon fails because it both omits critical physical factors and misrepresents the motion. In particular, one issue emerging from both documents concerns the velocity of impact in falls from a swing, and another, on which this concept is predicated, utilizes the conservation of energy during the motion of the swing. Both representations are manifestly incorrect.

The motion of a rider falling off a moving swing is complex because of the possible independent movements of the jointed components of the body that may be produced voluntarily or involuntarily during both the phases while on the swing and during a subsequent free fall. In the final analysis, what matters with respect to the generation of trauma is the condition of impact with the ground and not what transpires during the fall. This depends on the velocity, direction, and constitution of the initial point of contact on the rider's body, the angular motion of the element of which this point is a part at that instant, the configuration of the rider at the time, the weights of the various components of the child, and the mechanical characteristics of the striking surfaces.

To discuss this issue in a proper context of mechanics, it is first necessary to define possible types of motions as well as the physical characteristics of swings and their riders. For simplicity, it will be assumed that the motion can be represented by a cross-section of the rider restricted to a single plane, a two-dimensional model called plane mo-

tion. Translation in this plane consists of a movement in which each point of the object under consideration travels the same path and has the same velocity and acceleration at a given instant of time. Rotation here occurs about an axis or a set of axes that are always perpendicular to the reference plane. In pure rotation, the velocity of any point of an object is equal to the instantaneous rotational (or angular) velocity multiplied by the perpendicular distance to the axis of rotation. In general, plane motion consists of a combination of translation and rotation. It is most frequently described by the translational motion of the center of gravity of the system and the rotation of its components about this position. This will entail an analysis of the motion of the independent parts of the body subject to the constraint that motion at the pin is the same for both joined segments. Here, the motion is most conveniently depicted with reference to the rider's center of gravity, whose path must be traced.

All swings hang by chains or similar suspensions attached to a set of bearings on the support structure at equal height from the ground. Most swings are arranged so that the seat is always perpendicular to the suspension, but in some cases there are bearings attaching the chain to the seat that permit either partial or full rotation with respect to the chain; in the last instance, the seat is maintained in a horizontal position. The fundamental motion of the seat and rider in these circumstances are totally different. In the first case, the system executes rotation about the axis through the two fixed suspension points, absent any twisting of the chains (which may very well occur, at least at certain times, and will produce three-dimensional motion). When the seat is permitted to rotate to some extent or even completely, the rider's movement is different. If the seat remains horizontal at all times, the motion of seat and rider is curvilinear translation. These and subsequent concepts may be found in any elementary text on dynamics, such as in (1) or (2).

All analyses of movement, other than the theory of relativity, which is irrelevant here, are based on Newton's second law of motion for an object treated as a geometric point (called a particle), but endowed with the property of mass, the quantity that resists motion in translation. Mass, in turn, is the weight of an object divided by the acceleration of gravity. Newton's second law states that force equals the product of mass and acceleration for a particle. All descriptions of causes of motion for bodies that do not qualify as a particle are derived from this law. Extension to rigid and deformable bodies, and to systems of connected bodies, can become very complicated. A particle, by definition, cannot execute rotation unless attached to another object, such as a string.

Consider first the conservation of energy, which, in part, underlies Spivack's hypothesis that the velocity of a child striking the ground in a fall from a swing depends only on the maximum height that the system has attained. Conservation of energy requires the sum of the kinetic energy (the energy of motion) and the potential energy (the height above a given reference plane) to be a constant. This implies the absence of external forces that do work on the system; but in a swing this is not the case. The rider "pumps" with his legs and body to make the seat rise to greater heights. This feeds energy into the system, drawn from the internal energy of the rider that the person can apply. Secondly, this "conservation" ignores the presence of both bearing and air friction; the former is substantial and, as is well known, soon brings a swing to rest if energy is not supplied either by the rider or a person on the ground pushing the swing. Any energy supplied by the rider can, in practice, never be instantaneously equal to the energy lost by friction.

For a system of completely rigid objects, whose configuration remains invariant, executing pure rotation about a fixed axis and ignoring the loss of energy because of friction permits the calculation of the velocity of any point of the system by equating the loss of energy of position to the gain of energy in motion. However, as already indicated, the rider is definitely not a rigid object (and perhaps the configuration of chain and seat may also not be regarded as totally fixed). In that case, the analysis of the motion of the system or its components becomes much more complex. It is first necessary to define the simplest possible model that will correctly define the movement of the system and its parts, particularly if the rider should fall from the swing.

It is not permissible to describe the motion of the rider as that of a mass point located at his or her center of gravity (a particle to which Newton's fundamental law applies without extension) because the dimensions of the rider are comparable to the distance traversed by the swing (for example, the earth in its circuit around the sun may be regarded as a point mass because its diameter, about 3,960 miles, is minuscule in comparison with the millions of miles traveled in one pass). The portion of Spivack's treatment dealing with the dependence of the velocity of impact solely upon the height of fall hints at such a point model, presumably the center of mass; she does not specify to which location on the rider her velocity refers, although it should designate the impact position. The rider cannot be considered a single rigid body, such as a match stick, because of

the ability to move the head and neck, upper and lower arms and legs, hands and feet, and even the torso, relative to the seat.

Thus, the simplest conceivable model appropriate for an analysis of the motion of a swing rider and a subsequent fall is a two-dimensional representation of plane motion, as described above, for a segmented object pinned at the joints, which minimally should include the head and neck junction, the shoulder and elbow joints, the hips and knees, and possibly the ankles and wrists. The analysis of such segmented motion has been established for some time and is detailed in (3), (4), and (5), but is too complicated and tedious to be described here, although it is not particularly difficult. It requires a knowledge of the weights and dimensions of the body segments. However, the actual motion is indeterminate here because of the need to precisely describe the configuration of the rider at the time of separation from the seat, the possible effects of a push of the swing during this event, and, most importantly, the unknown voluntary and/or involuntary movement of the child during the fall. Finally, the position of the center of gravity to the rider, which may be constantly changing at all times prior to impact, must be traced, preferably relative to some fixed point in space, although it can be referred to some anatomical feature of the rider.

At the instant of separation, the path of the center of mass of the rider will be a straight line perpendicular to the chains, with a constant linear velocity v_1 at an angle θ in the absence of gravity or a push from the swing, neglecting air resistance. All other points of the object will similarly retain the speed that they had just before departure at this same angle, resulting in a further rotation of the rider. However, gravitational attraction of all points of the person will contribute an additional vertical velocity component proportional to their distance of free fall, which must be added vectorially, never algebraically, to the velocity of any element of the rider because of the initial swing motion and subsequent deliberate or reflex action. The latter relative motion during the fall cannot be predicted; it can only be determined by photographic or other observation of the event. Thus, at impact, the total velocity of the center of mass, if its location remains constant within the configuration, depends on v_1, θ, and the velocity v_2, the latter being proportional to the square root of the height of fall at this point. Corresponding arguments apply to the total velocity of the contact point with the ground, whose location may be approximated from damage considerations. Its velocity component, because of swing and body actions, will practically never be in the vertical direction, so that its total value must also be determined vectorially and predicated on the configuration at impact. Hence, Spivack's contention that impact velocity does not depend on where a rider leaves the swing, only on total height attained, is invalid. Of course, the analysis presented here is a slightly simplified version of what may actually occur, because three-dimensional motions may be involved and an unknown amount of energy is pumped into the system by the controlled or involuntary independent motions of the rider. However, although extremely elementary, this representation at least depicts the action in a manner that is mechanically correct.

It should be mentioned that, if peak force experienced during the impact is a correlater of injury, the shorter the duration of contact (until the force is reduced to zero), the higher this peak force will be for a given set of conditions. This fact documents the obvious need for the most resilient possible surface under a swing, to decelerate a falling body more gradually and, hence, reduce the potential for injury. The analysis of the contact phenomenon is another story.

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Letters to the Editor

Retinal Hemorrhages: Evidence of Abuse or Abuse of Evidence?

To the Editor:

Sir, I would like to congratulate Dr. Plunkett and the *American Journal of Forensic Medicine and Pathology* for publishing his recent valuable contribution regarding infant head injury/shaken baby syndrome (1). No reasonable professional of any discipline wants to see malicious child homicide go undetected and unpunished. However, it is equally abhorrent that over-zealous investigation, refusal to study alternative explanations, or narrow-minded pursuit of blame for an infant's death can destroy the lives of the accused and their family, creating supplementary victims (2). This is particularly salient when the accused and the families of the accused are recently bereaved relatives of the deceased child. We have an inescapable duty to get this right! (3)

To date, published literature strongly supports the hypothesis that *only severe* traumatic forces cause the constellation of subdural hemorrhage, parenchymal brain injury, and retinal hemorrhage (4,5). However, in scientific terms, this is merely a *hypothesis* based on intrinsically imperfect epidemiology in the form of a case series, with case ascertainment and selection bias, and the formation of personal opinion. Experimental tests of the hypothesis via biomechanical or animal models have failed to confirm the necessity for severe force (6,7). However, these models are rightly criticized for not accurately reproducing the human state (2). Similarly, extrapolations from severe accidents, such as automobile accidents, while seemingly supporting the hypothesis (2,8), are themselves also imperfect biomechanical models of shaken baby/shaken impact syndrome. Biologic variation in responses is rarely considered (2) and professionals with voluminous experience working in this field will repeatedly encounter cases that just don't comfortably fit the mold. There may be little other suspicion of abuse, few other features of physical trauma, and minimal unequivocal evidence of traumatic brain injury despite the incongruous necessity for severe trauma. In addition, some adults consistently offer explanations involving minimal trauma or show bewilderment about the cause of the child's condition. Lawyers also candidly say that, while convinced of the guilt of some of their clients, they are convinced of the innocence of others. Nevertheless, the weight of best available medical and scientific knowledge is against them. Despite these difficulties, we are resolutely stuck with only *one* widely accepted hypothesis - a scenario very unusual in biologic systems in which multiple routes to a common end are normal.

Medical witnesses often refuse to challenge the weight of professional opinion, no matter how light, and prosecutors are only too willing to seize upon this convenience. Convictions add to the dogma that severe force is necessary and strengthens the self-fulfilling prophecy by adding "gray cases" to the spectrum of "proven black cases". Repeated "learned" publication of this dogmatic-received wisdom supports the potential myth, inexperienced professionals consume the hypothesis as *established fact*, and it becomes sacrilegious to challenge the hypothesis via contradictory observations or by offering alternative hypotheses (2). In this field more than others, we are more readily prone to alter the facts to fit the hypothesis rather than alter the hypothesis to fit the facts. For example, if the accused admits to severely harming the child, we tend to believe because this fits our hypothesis, but if the accused offers an alternative apparently innocuous explanation we discount the explanation because it doesn't fit the hypothesis (2). Published cases purporting to demonstrate less traumatic causes (9–12) are attacked for their anecdotal data and for overlooking the real, more sinister explanation (5). This "illogical inconsistency" overlooks scientific process and forgets that the requirement for severe shaking forces is no more than a *favored* but *unproven* hypothesis (13).

The main cause of bias and hindrance to progress is the fact that very few of these cases are verified by eyewitnesses (4). Dr. Plunkett's case series is very important because it involves verification of events by eyewitnesses and because the data was collected via an independent non-prosecutorial agency concerned with child safety and not conviction at all costs of a parent, guardian, or other child caregiver (1). The careful observations collated by Dr. Plunkett confirm that there are cases of innocent minor trauma that may mimic those of shaken baby syndrome. Dr. Plunkett employs sound biomechanical insight to challenge the accepted hypothesis

and to propose alternatives. His hypotheses may prove to be correct or wrong and are potentially as impotent as any others that have challenged the prevailing dogma. However, Plunkett's series is no weaker than those series that support the traditional hypothesis. Like other emerging publications questioning the causes and mechanisms of infantile brain injury (14,15), its most important function is to reopen debate, help us consider the status of our beliefs, and force us to seek the truth. In my own field of involvement, the pathologic interpretation of retinal hemorrhages, I am forced to note that Plunkett reports retinal hemorrhages in his series. This directly challenges the widely held view that retinal hemorrhages are "evidence of abuse" (5,16). To prevent retinal hemorrhages from being unwittingly abused as evidence, Dr. Plunkett's important paper must stimulate fresh thought and investigation into the spectrum of causes and mechanisms of infantile retinal hemorrhages (17).

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Sudden Infant Death Syndrome

To the Editor:

I read with great interest the Letter to the Editor entitled, *SIDS Doesn't Exist*, by Drs. Sawaguchi and Nishida, which appeared in the June 2001 issue of the American Journal of Forensic Medicine and Pathology (1). I am a forensic pathologist who has, over the years, often stressed to students the true meaning of the term "SIDS". Sudden infant death syndrome is by definition a sudden natural death of an apparently healthy infant in whom no detectable cause of death can be found. This diagnosis is made only after all reasonable efforts to find the cause of death have been exhausted. It is, thus, not a wastebasket diagnosis and it does not mean that there is no cause of death, only that none can be determined by the forensic protocols and procedures used. It remains true that, in general, we recognize only that which is visible. It should therefore be clear that SIDS is not a cause of death. While it means that a cause of death is undetectable, it also implies that the death is a natural one.

I would like to note that the late Dr. Milton Helpern, my mentor, did in fact object to the use of the term "SIDS" because it could mistakenly convey the meaning of a medical condition, which it is not. For many years, he and his staff used the term "sudden unexplained death of infant," as can clearly be seen from the old records of the New York City Office of the Chief Medical Examiner. I believe that this is, in fact, a more accurate description of this phenomenon. The inherent danger of using the term "SIDS" can be seen in the case of a mentally deranged woman in Philadelphia who, at age 70, confessed to smothering eight of her young children decades ago. With no clear evidence to show otherwise, doctors and investigators had reluctantly attributed the deaths of these eight children—none of whom lived longer than 14 months—to sudden infant death syndrome (2). Thus, the danger of the ill-considered use of the term "SIDS" is clear.

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Fatal Pediatric Head Injuries Caused by Short Distance Falls

To the Editor:

Dear Editor,

I read with interest the recent article "Fatal Pediatric Head Injuries Caused by Short-Distance Falls" (1)

The paper reports a retrospective chart review of 18 children who died from alleged accidental head injury. For six patients, data was available regarding an eye examination. Four of these patients had retinal hemorrhages (RH). The author uses these cases to suggest that perhaps RH must be interpreted with caution when evaluating a child for possible Shaken Baby Syndrome (SBS).

Unfortunately, "None of the children in this study had a formal retinal evaluation" and not enough details were given about the nature of the RH for us to make any determination about whether the findings are consistent with currently available published literature. One child had "extensive bilateral retinal and preretinal hemorrhage". Another had "bilateral retinal hemorrhage". A third had "extensive bilateral retinal, vitreous hemorrhage" in one eye and papilledema. The fourth child with RH was 10 years old and therefore not very applicable to the SBS age range. This child had "extensive bilateral confluent and stellate posterior, peripheral preretinal and subhyaloid hemorrhage". No details are given regarding post mortem microscopic examinations in any case.

If no child had a "formal" retinal examination (which I assume means ophthalmology consultation) then how are all the distinctions made about the types of RH? In our study of non-ophthalmologist retinal examinations (unpublished data) in a large cohort of SBS victims, not once did a non-ophthalmologist even attempt to make such distinctions. What does "extensive" mean? In my experience, what a non-ophthalmologist calls extensive might very well be well within the accepted posterior pole hemorrhages, which may be moderate in number (to me), and quite acceptable following severe accidental life-threatening head injury. What does "stellate" mean? I have never seen that word used to describe RH by an ophthalmologist or non-ophthalmologist. I cannot believe a non-ophthalmologist could distinguish between subhyaloid and preretinal hemorrhage. This is sometimes even hard for the ophthalmologist to do. And clearly, peripheral RH can only be seen with formal retinal examination using the indirect ophthalmoscope.

Interestingly, the incidence figures make perfect sense. To get these four cases, the author needed to search literally tens of thousands of records. In fact, one might even say that this article is proving that RH is even more rare than the literature already suggests. Posterior pole RH is seen in up to 3% of children who sustain severe accidental life-threatening head injury. In these cases, the history alone is almost always sufficient to differentiate the situation from SBS. Certainly, these are not falls out of the arms of adults or off of beds and sofas. In the current article, two of the cases with RH occurred on falls from swings. Even the author acknowledges that the height of swing falls "could not be accurately determined". In addition, with a child in motion, the velocity of the impact might be expected to be even higher than that from the height alone. Clearly, this is different than a "short-distance fall". The child with papilledema may have had RH secondary to the papilledema alone: a completely non-specific finding. Papilledema is uncommon in SBS.

So maybe this article reports an *extremely rare* circumstance where multiple factors coincide to allow RH (maybe even "extensive" RH) to occur following accidental trauma that is less than that which is usually needed (e.g. a motor vehicle accident) but still beyond common minor household falls. On the other hand, the paper may be completely inaccurate and misinterpreted if the eye examinations were not confirmed by an ophthalmologist.

Other than birth, SBS is by far and away the most common cause of RH in the first three years of life. Careful description of the number, types, and distribution of RH is essential in determining the specificity of the fundus picture. Much has gone into understanding RH in SBS and other conditions. Contrary to the author's suggestion that there is "scant objective evidence" to address this issue, the literature is replete with useful data. One review (2) has over 200 references. Rigorous application of the scientific method rather than incomplete anecdotal retrospective reports, is our best tool towards finding the truth.

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Author's Response to Drs. Spivack and Levin

To the Editor:

I thank Drs. Spivack (1) and Levin for their letters and have only a few comments.

The initial manuscript was almost 50% longer than the final version. The published version, at more than 6000 words, was still very long by any standard. It is difficult to include every detail for every case in a series this large and still make the article readable. Ideally, this study would have been prospective and would have involved every death investigation jurisdiction in the United States, with a detailed protocol for evaluating and documenting each death. However, this could not be done, in part because there has been no impetus to do so since these types of injuries and deaths "cannot and do not occur".

The series cannot be used to establish either the commonality or the rarity of death from a given event for the reasons stated in the "Limitations" and the "Conclusions"(2).

Weber's papers (3-4) are actual scientific studies based on a given mechanical input (a gravitational fall of 82 cm to a variety of surfaces) and a measured or observed output (skull fracture) under controlled conditions. My observations should not be used to either confirm or deny the validity or applicability of his observation.

The mechanism (with few exceptions) for an ultimately fatal brain injury in an infant or child is not multifocal traumatic axonal injury (DAI), but focal traumatic axonal injury with secondary anoxia and/or cerebral edema (5-8) or an acute subdural hematoma. The focal injury may be a contact-induced cortical contusion, cortical laceration from a skull fracture, or superficial brainstem contusion caused either by hyperextension or by movement of the brainstem through the foramen magnum (that may occur secondary to mass movement of the brain caused by deformation of the skull during impact) (9). One would not predict nor does one usually find DAI in circumstances where the mechanism is a short-distance fall or a "slam". (The physics of a short-distance fall and a "slam" are identical).

There is not a single reference to "shaken baby syndrome" or "SBS" in the text of the article. Dr. Levin states that I "suggest perhaps that RH must be interpreted with caution when evaluating a child for possible shaken baby syndrome (SBS)". My actual "conclusion" regarding retinal hemorrhage is, "The characteristics of the bleeding cannot be used to determine the ultimate cause."

The eye examinations were performed by a Board-certified neurosurgeon with 25 years of experience (case #16) and by Board-certified pediatric intensivists with 5–15 years of experience (cases #4, 5 and 6). I discussed the medical record entries with each of these physicians. The descriptions in the article are theirs, not mine. If someone believes that these physicians were not qualified to make the observations and conclusions that they did, then so be it.

Although this study should not be used to determine the incidence of retinal hemorrhage associated with head impact from a fall (for the reasons stated in the "limitations"), it is not true that "tens of thousands" of cases were searched to identify these four. The correct number is *18*, and of these, only *six* had fundoscopic examination performed.

There is no evidence that "shaking" can cause retinal hemorrhage or that the mechanics of an impact from a "slam" are somehow different from those of a "fall". The conclusion that they are different is tautology, not science. If someone has a theory for a cause of retinal hemorrhage different from those that I discuss in the study, then please do the appropriate experiments and prove it. Reiterating the hypothesis and stating that it is "widely and generally accepted, published," and authenticated by "vast clinical experience" does not make it true.

I hope that my study encourages us to re-examine our concepts regarding traumatic brain injury (TBI) and the relative importance of inertial or impulsive loading (whiplash) and contact. Dr. Caffey's "theory," accepted for almost 30 years, taught in medical schools, approved as an ICDA-9 "codable disease" and testified to as "truth" in court, is based on a misinterpretation of early pioneering experiments performed for the automotive and space industry. Ommaya (10) published a landmark study in 1968 showing that TBI could be produced in rhesus monkeys by acceleration of the head alone (with the midneck as a fulcrum) and no contact. However, the level of acceleration he used to cause these injuries was 10,000–100,000 r/s^2, with the lower limit being the *concussion* threshold. (Ten thousand r/s^2 at a radius of 6 inches is 5,000 f/s^2 or 156 G's). Caffey called Ommaya after his (Caffey's) 1972 article (11) was published and discussed it with him (12). Ommaya told him that he (Caffey) was misinterpreting his (Ommaya's) studies, but Caffey either didn't understand or forgot to tell us. This misinterpretation is repeated in Caffey's 1974 article (13). And here we are today. A WWII paratrooper aphorism concerning chute-deployment failure says it best: "It is not the fall that kills you. It's when you hit the ground."

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Death Resulting from Asthma Associated with Sertraline "Overdose"

To the Editor:

In their report, Carson et al. (1) speculate that the asthma-related death of a young woman might have been caused by serotonin syndrome, in turn caused by a sertraline overdose.

In support of an overdose, they state that the blood level of sertraline, 620 ng/ml, was "very high." However, they do not mention the analytical method used or the sampling site of the blood, nor do they use relevant postmortem reference levels.

To determine whether a postmortem drug concentration is high, reference data are needed. The authors mention a steady-state concentration range of 30–200 ng/ml in persons receiving "therapeutic dosage regimens," but they do not give a reference. Although this is approximately the range that Gupta and Dziurdy (2) report in their clinical study, such data are usually not suitable for comparison with postmortem levels, particularly because of postmortem drug redistribution (3). In addition, even postmortem reference data may be unsuitable if the sampling site is unknown or if the procedures and methods are not specified.

In a compilation of postmortem analytical results, Druid and Holmgren (4) have tried to overcome some of these obstacles for several drugs. First, only femoral blood levels are used. Second, levels are provided both for lethal intoxications and for carefully selected cases in which the cause of death with certainty was not related to intoxication, and in which the victims were not incapacitated by drugs immediately before death. Third, as to the fatal intoxications, levels are given separately for cases related to intoxication with one substance only and for cases in which other substances or conditions may have contributed to death.

In a recent report, the same strategy was used to evaluate additional drugs, including some of the newer antidepressants (5). A total of 211 cases in which sertraline was quantified in femoral blood were reviewed. There were no sertraline-only deaths. In cases in which sertraline intoxication in combination with other factors was considered to have caused the death, the median concentration was 2.2 (10th and 90th percentiles 1.3 and 3.6) μg/g blood (n = 11), in comparison with 0.1 (0.1 and 0.4) μg/g blood (n = 61) in nonpoisoning deaths. The lack of sertraline-only deaths in this Swedish report may also suggest a low toxicity in comparison with other selective serotonin reuptake inhibitor drugs when the prescription data are taken into account (6).

Although the reported concentration of 0.62 μg/ml exceeds the median level in the "controls" of this report, it is still lower than the concentrations found in the cases in which sertraline was considered to have been a contributory cause of death. It is therefore hardly justified to say that the level is "very high." Moreover, the origin of the blood sample is not stated. If the blood sample was collected from the heart, it is even more likely that the concentration actually was not much elevated ante mortem, given that leakage and redistribution of the drug from other tissues with high concentrations could have occurred. In a recent study by Goeringer et al. (7), based at least partly on peripheral blood, the authors concluded that sertraline appeared to be a primary contributory factor to the death at levels above 1.5 mg/L, thus also significantly higher than the reported level of 0.62 mg/L.

The authors are apparently unaware that they do provide one clue in support of a *recent* intake: the parent drug–to-metabolite ratio, which is 1.9. This figure is higher than the median (0.6) reported by Druid and Holmgren (5) for controls, but it does not necessarily confirm a *large* intake. The ratio might also be influenced by various pharmacokinetic and pharmacogenetic factors.

In conclusion, the suggestion that the asthma attack was precipitated by a sertraline overdose leading to very high sertraline concentrations thus remains speculative.

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Letters to the Editor

Chronic Lead Poisoning: Induced Psychosis in an Adult?

To the Editor:

I recently had a case of a 35-year-old healthy white man with no history of physical or mental illness who, for 1 1/2 years, had been sanding the paint off a 125-year-old ranch house. He worked both indoors and outdoors, rarely using a mask. Two months before his death, he experienced chest pain and tingling in his fingertips. His physician ruled out cardiac problems and prescribed alprazolam, thinking his symptoms were secondary to stress. Three days later, he became extremely anxious, restless, delusional, and paranoid, and was unable to sleep.

One month before death, he attempted suicide with his automobile but survived. During his hospitalization, he was anemic (hemoglobin of 12.1 g/dL) and had 1+ proteinuria and 3+ hematuria. He became very depressed and was started on an antidepressant. A month later, he committed suicide by way of an intraoral gunshot wound. The only unexpected finding at autopsy was a blood lead level of 51.3 μg/dL. The last exposure to the ranch house was 8 weeks before death.

A brief review of the literature suggests that it is possible that the decedent's psychiatric disturbances were caused by chronic lead poisoning. The primary routes of lead absorption are ingestion and inhalation. Homes built before 1940 used lead-based paints on both the exterior and interior surfaces. A tiny flake from an old house can contain up to 100 mg of lead (1). Accumulation and toxicity occur if more than 0.5 mg/day is absorbed. Chronic lead poisoning is more common than acute lead poisoning and produces a variety of signs and symptoms (2).

Lead interferes with the synthesis of heme, and consequently a hypochromic normocytic anemia develops along with a compensatory reticulocytosis and basophilic stippling. Gastrointestinal symptoms are constipation, abdominal pain ("lead colic"), anorexia, weight loss, vomiting, and metallic taste. There is damage to the proximal tubules of the kidney, with subsequent proteinuria, hematuria, and oliguria. Thus, the anemia, proteinuria, and hematuria during this individual's hospitalization for attempted suicide are consistent with hematopoietic and renal injury caused by chronic lead exposure.

Relative to the nervous system, patients with chronic lead poisoning manifest encephalopathy (including irritability, fatigue, restlessness, and insomnia followed by delirium, convulsions, and coma) as well as peripheral neuropathy with tingling in the hands and feet, and wrist and foot drop. Baker et al. performed a prospective study of lead neurotoxicity in foundry workers and referents in 1983, finding that there was an increased rate of depression, confusion, anger, fatigue, and tension in workers with blood lead levels over 40 μg/dL (3).

Several factors support the possibility that the decedent in this case had chronic lead poisoning. The blood level at autopsy was 51.3 μg/dL. A blood lead level above 5–10 μg/dL indicates exposure, and levels above 40 μg/dL are considered toxic. One's risk of encephalopathy is great with levels above 80 μg/dL, and it is considered an emergency with levels above 100 μg/dL (2). Death was 8 weeks after his last exposure to the lead, and given the fact that the t½ of lead in blood is 1 to 3 months, his lead level at the onset of his psychiatric symptoms was probably above 100 μg/dL.

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Fatal Pediatric Head Injuries Caused by Short Distance Falls

To the Editor:

In the recent article by Plunkett entitled "Fatal pediatric head injuries caused by short distance falls" (1) he states, based on a literature review, "many physicians believe that a simple fall cannot cause serious injury or

death, that a lucid interval does not exist in an ultimately fatal pediatric head injury, and that retinal hemorrhage is highly suggestive if not diagnostic for inflicted trauma." Our review of the literature referenced by Plunkett shows his opening statements to be an inaccurate and misleading summation of the conclusions drawn by the authors of the work cited.

Here are a few examples. Williams' (2) concluded: "infants and small children are relatively resistant to injuries from free falls, and falls of less than 10 feet are unlikely to produce serious or life-threatening injury." Willman et al. (3) concluded: "unless an epidural hematoma is present, children who die from blunt head injuries probably do not experience lucid intervals." and Rao et al. (4) concluded: "if intraoccular hemorrhage is seen, the possibility of child abuse should be considered."

All of the authors recognize, as we do, that there are no absolutes in regard to head injuries in children and refrain from using Plunkett's absolutist vocabulary of "can not, does not, and is diagnostic for," especially when it concerns the issue of accidental versus inflicted trauma in children. As forensic pathologists we must address each case without prejudice, paying particular attention to anamnestic-anatomic disharmony, cognizant that the issue is seldom black or white, and admitting that impossible situations are few and far between.

Some of the limitations of the study have been cited within the text of the article but must be emphasized, with special attention to children under the age of 5. The biophysics of head injury in children from newborn to 60 months changes fundamentally, and most controversies involve children from 0 to 36 months of age. Six of the 18 falls were not witnessed, including 5 of the 10 falls of children 5 years of age or younger. In this study, no child less than 23 months of age had a witnessed fall. Seven of the 18 falls were not followed by a full autopsy, including 5 of the 10 falls of children 5 years of age or younger. Although physical and radiologic examinations are helpful in assessing injury, they cannot fully answer questions as to anamnestic-anatomic disharmony, occult injuries, or undiagnosed disease processes. Cases without autopsy results must be viewed with circumspection.

The evaluation of the history of a lucid interval in cases of lethal head injury is especially important in children under the age of 5, whose injuries are often unwitnessed. In this study, there are 4 witnessed cases of children under the age of 5 years (Cases 5, 6, 8, and 9), who were evaluated with autopsy and forensic pathologist directed-death investigation system examinations that deserve further discussion. The lucid intervals of the witnessed cases ranged from none to 3 hours. In the case with no lucid interval (Case 6), there was a lateral blunt impact injury of the head, focal hemorrhage in the right posterior midbrain and pons consistent with diffuse axonal injury, a small subdural hemorrhage, and cerebral edema with herniation after at least a 4-hour survival period. In two of the cases there were lucid intervals of a few minutes (Cases 5 and 8). In the first case, there was a blunt impact injury of the right side of the forehead with large subdural hematoma requiring evacuation. Cerebral edema and herniation developed after at least a 12-hour survival period. In the second case, there was a complex fracture of the frontal-temporal bones associated with cortical contusions, epidural and subdural hemorrhages, and the development of malignant cerebral edema with herniation on the second hospital day. The child died on the third hospital day. In the case with a lucid interval of 3 hours (Case 9), there was an epidural hematoma without skull fracture or blunt impact injury of the head, and the child died 10 days after admission to the hospital. In each case, the lucid intervals were consistent with the neuropathologic findings at the time of autopsy.

The height of the fall could be determined in only 10 of 18 cases. However, Plunkett's definition of the height of the fall as "the distance of the closest body part from the ground at the beginning of the fall" would mean that a fall from standing height would be interpreted as a height of zero! Obviously, the determination of height of the fall in assessing head injury cases would better be defined as "the distance of the head from the ground at the beginning of the fall," which would increase the distance determination in these cases.

The vast majority of children (14 of 18) had evidence of blunt impact to the head by radiologic evaluation, autopsy documentation, or witnessed reports that were consistent with the given scenarios. One case had no reports (Case 11). Of the three children with no evidence of blunt impact to the head (Cases 3, 9, and 15), two had large subdural hematomas, and the third had an epidural hematoma. This study suggests that lethal impacts to the head rarely leave no evidence of an impact. None of the children without reported head impact injury had retinal hemorrhages.

We agree with Plunkett that deformation and internal angular acceleration of the skull and brain caused by the impact produce injury. Although no specific mathematical analysis was applied to these cases, it also may be helpful to consider that in each of these cases the head was in motion preceding impact because of the nature of the accelerating and/or centrifugal forces of the playground equipment that the child was using (swings, seesaw, horizontal ladders) or because of a fall from losing balance. In falls from losing balance, the center of gravity becomes displaced with respect to the feet, and the force of gravity rotates the body in the direction of the displacement. In these rotational falls, the head's acceleration exceeds the acceleration because of gravity (5). Medical examiners frequently are presented with histories that are similar to the type of falls studied by

Lyons and Oates (6) rather than the histories that are presented in Plunkett's study.

This review of more than 75,633 entries of childhood falls associated with playground equipment in the databases of the U.S. Consumer Protection Agency during an 11½-year period has served to document the *extremely rare* occurrence of lethal head and neck injury in children in *unique scenarios*. By design, these databases are biased toward the collection of data regarding severely injured children, and the total number of entries in the databases cannot approximate the millions of childhood falls of short distances *not* reported to the Consumer Protection Agency during that same 11½-year period in association with playground equipment or otherwise. The study is limited in its evaluation of infants and children under the age of 5 because there are no witnessed cases in children under the age of 23 months. Evaluation of the only four witnessed cases of children under 5 years of age with findings documented by autopsy and forensic pathologist directed-death investigation system examinations showed that lucid intervals are consistent with neuropathologic findings at the time of autopsy. The determination of height of fall in this study is difficult to interpret because it does not reflect the height of the head at the time of the fall. There were no cases without blunt impact injury of the head that showed small subdural hemorrhages and retinal hemorrhages. Certainly a history given by a caregiver that a child may have fallen must be evaluated, but there is no substitution for a thorough postmortem examination, including medicolegal investigation and the interview of witnesses.

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Author's Response to Fatal Pediatric Head Injuries Caused by Short Distance Falls

To the Editor:

I thank Dr. Shaber and colleagues for their letter. I agree with many of their points regarding the limitations of the study. However, I have a few additional comments.

I do not think that my opening statement is "inaccurate and misleading" or unfairly characterizes the conclusions in the references that I cite. For example, Schaber et al. state that Williams (1) concludes: "Infants and small children are relatively resistant to injuries from free falls, and falls of less than 10 feet are unlikely to produce serious or life-threatening injury." This "conclusion" is from the abstract. The text of the article states: "That severe injuries and deaths from falls of 5 feet or less only occurred in the uncorroborated group leads one to suspect that many if not all of the injuries attributed to falls of low height represent child abuse." Likewise, when Willman et al. (2) conclude that "Unless an epidural hematoma is present, children who die from blunt head injuries probably do not experience lucid intervals" they also state: "The results of this study suggest that a fatal HI [head injury] that does not involve an epidural hemorrhage must have occurred after the last known time that the child exhibited normal behavior," "For an older child . . . the time of the injury event in a fatal HI without an epidural hemorrhage can be restricted to after a confirmed period of such normal behavior," and "Excepting cases involving epidural hematomas, the time of injury in a fatal head injury case can be restricted to after the last confirmed period of normal consciousness for the child". [I comment on the Willman et al. study in a letter published in 1998 (3).]

Shaber's assertion that "The biophysics of head injury and children from newborn to 60 months changes fundamentally" lacks an evidentiary basis. The brain, scalp, and skull of a newborn are *not* the same as the head and neck unit of an adult. Scaling commonly used for the automotive industry may *not* apply to the neonate or infant (4). The developmental anatomy and physiology are certainly different, but where is the cutoff, and where is the *biophysical* evidence to support it? There is only one published study of the biomechanical failure characteristics of the infant skull (5). There are only three published studies of experimentally produced infant skull fractures (6–8). There is one published reference concerning the concussion threshold in the pediatric age group (9). There are no other studies evaluating the failure thresholds or the differential biophysics of infant, toddler, or pediatric head injury, whether for a 3-month-old, 3-year-old, or 13-year-old.

I agree that "cases without autopsy results must be viewed with circumspection." It would have been invaluable if complete postmortem examinations and formal neuropathology evaluation, including microscopic examination, had been performed in all of the deaths. However, please note that four of the seven deaths in which an autopsy was not performed occurred in a jurisdiction directed by a board-certified forensic pathologist. (This is not a criticism; I am merely emphasizing that one can rarely anticipate all of the questions that may be asked in the future about a death that seems "routine" or "adequately and fully documented" today.)

I agree that the documentation of the height of the fall "would be better defined" as suggested. The definition I used allows an initial comparison of the cases in my study with those from other cited articles, none of which has *any* definition for "height of the fall" and wherein it must be assumed, correctly or incorrectly, that the "height" is the height of the object from which the person fell (10–14). However, none of the other studies describes the position of the body at the beginning of the fall, the initial velocity, the part of the body that first struck the ground, or the characteristics of the impact surface. These cited studies do not allow even a kinematic analysis, let alone a kinetic evaluation. Further, using "head above the ground" does not elucidate the mechanism for injury and only allows a calculation of the approximate impact velocity. For example, knowing the distance of the head above the ground does not allow one to evaluate momentum (mv), energy ($\frac{1}{2}mv^2$), force (ma), deformation and strain of the scalp, skull, or brain, etc.

Each of the falls in this study involved an external rotational component. None was purely "translational," and I discussed the mathematical constructs for analysis of the falls in the Appendix. However, whereas external angular motion during a fall may change the impact velocity from that calculated for simple translational motion (greater or less, depending on whether the vectors are additive or subtractive), angular motion does not significantly contribute to "inertial loading." What happens during the impact, not during the fall, determines the outcome.

Dr. Shaber states that "This review of more than 75,633 entries of childhood falls associated with playground equipment . . . document(s) the *extremely rare* occurrence of lethal head and neck injury . . . in *unique scenarios"* and "By design, these databases are biased toward collection of data regarding severely injured children." Her contention is a mischaracterization of the database (15–18). Certainly, the death certificate (DC) file is biased. However, only 4 of the 47 cases in the CPSC DC file involved a fall (Cases 1, 5, 6, and 9). The others involved events described in the first paragraph of the Results. The incident report (IR) file includes many entries in which there was *no* injury, only the potential for injury. The NEISS file (more than 75,000 entries) contains reports of emergency room visits in the United States hospitals participating during the study period, and includes all incidences, not simply falls (as I discussed in the Introduction, Methods, and Results). More than 98% of the entries in the NEISS file involved an injury not requiring hospitalization. It is important to know that deaths such as I reported do occur, and it is important to understand why they happen. Whether the events are common or rare is not.

Finally, I agree that there is "no substitution for a thorough postmortem examination including medicolegal investigation and interview of witnesses." However, I would add that this investigation must be founded upon the ability to critically evaluate the evidentiary bases for opinions and conclusions, and upon an accurate, complete, and current understanding of the biomechanics of head injury.

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Symptoms Following Head Injury

To the Editor:

A recent proposed position paper regarding pediatric nonaccidental abuse head injury would allege that we may depend on symptoms to appear immediately upon injury (1). I have a concern about that claim, which I will explain with the following case. CASE REPORT

A 13-month-old Hispanic girl was brought to the University of Wisconsin Hospital on the morning of September 18, 1999. The complaint was of vomiting that had lasted for 24 hours. She was described as irritable, sleepy, and vomiting. In our emergency room she was noted to have extensive bruises on the cheeks, chest, back, and arms; the mother attributed these to bites by a 3-year-old housemate. She was admitted and given intravenous fluids. She was sedated with pentothal followed by head computed tomography, which was negative. She was then admitted to the pediatric ward. The resident who saw her described her in the chart and in discussion as being fussy and clingy, but interactive and responsive. Because of the numerous bruises, the police were notified and took pictures.

At about 2:00 the following morning, a nurse coming in to care for the child noted that she had decreased respirations. It was then shown that she was unresponsive and had a right dilated unreactive pupil with a sluggish left pupil. She was taken emergently to the pediatric intensive care unit, where she was intubated and given mannitol. A subsequent computed tomography scan showed very poor differentiation of gray/white matter interface. A Codman catheter was placed and then replaced with a ventriculostomy tube after an intracranial pressure of 21 mm Hg was noted.

On the evening of the day after admission, a cerebral blood flow study showed no cerebral blood flow. She was pronounced brain dead.

In the interim, her mother had fled town and has not been found since.

An autopsy was done on September 20, 1999. This showed hemorrhage in the left optic nerve sheath and left retinal hemorrhages as well as marked cerebral edema and thin widespread subdural hemorrhage. Diffuse axon injury was demonstrated with amyloid precursor protein antibody.

My point is that the child did have some symptoms, but clearly the severe intracranial injury symptoms, which were confirmed on repeat computed tomography and autopsy, were delayed for several hours, during which time she was under our view and review in the hospital. Others have noted similar problems (2).

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Author's Response

To the Editor:

In response to Dr. Robert Huntington's letter in which he expresses concern about the Position Paper's position on interval from injury to symptoms in young children with abusive head injuries, I would make the following comments. Dr. Huntington describes a child who is noted to be "irritable, sleepy, and vomiting" on admission. The Position Paper states, "Symptoms demonstrated by these severely injured children include an immediate decrease in the level of consciousness (either lethargy or unconsciousness)." Lethargy is defined as a condition of drowsiness or indifference *(Dorland's Illustrated Medical Dictionary)*. The sleepiness in this child is the neurologic symptom that marks the time at which the injury to this child occurred. It indicates a decrease in the level of consciousness. After the child was in the hospital several hours, she showed signs of increased intracranial pressure (right dilated pupil and sluggish left pupil) and went on to brain death. Dr. Huntington remarks that the severe intracranial injury symptoms were delayed for several hours. The symptoms to which he is referring were not the initial symptoms of injury but those related to increased intracranial pressure. The brain injury that precipitated this course of events (diffuse axonal injury) occurred when the child first became neurologically symptomatic. Brain swelling followed the diffuse axonal injury and resulted in increasing intracranial pressure and eventually brain death. Certainly, the child's neurologic symptoms changed with time, reflecting this changing neuropathology. It is the initial neurologic change that marks the time of injury.

The article by Gilliland that Dr. Huntington mentions is not helpful in elucidating the interval from injury to symptoms. The cases reported in that article relied on histories from possibly biased witnesses (caregivers who

might have injured a child) and took at face value the time intervals provided in each case. Dr. Gilliland noted this problem in her article, stating, "It should be noted that in all of the cases where information was supplied by someone other than the perpetrator, the child was not normal during the interval."

The claim that a young child has been fine for hours after a fatal head injury was inflicted and then suddenly developed symptoms is a claim that has no support from legitimate or mainstream medicine. When a child has suffered a serious acceleration injury to the brain that will result in long-term neurologic impairment or cause death, the so-called lucid interval is a fiction. The change from "fine" to "not fine" may be lethargy or it may be unresponsiveness, but it is a neurologic change, and it occurs at the time of injury.

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