Revisiting Neuroimaging of Abusive Head Trauma in Infants and Young Children

Kevin Li-Chun Hsieh[1,2]
Robert A. Zimmerman[3]
Hung Wen Kao[4]
Cheng-Yu Chen[1, 2, 5]

[1]Department of Medical Imaging, Taipei Medical University Hospital, No. 252, Wu Hsing St, Taipei City 110, Taiwan, ROC. Address correspondence to C. Y. Chen (sandy0932@gmail.com).

[2]Imaging Research Center, College of Medicine, Taipei Medical University, Taipei City, Taiwan, ROC.

[3]Department of Radiology, The Children Hospital of Philadelphia, 34th Street and Civic Center Blvd, Philadelphia, PA.

[4]Department of Radiology, Tri-Service General Hospital, Taipei City, Taiwan, ROC .

[5]School of Medicine, National Defense Medical Center, Taipei City, Taiwan, ROC.

OBJECTIVE. The purpose of this article is to use a mechanism-based approach to review the neuroimaging findings of abusive head trauma to infants. Advanced neuroimaging provides insights into not only the underlying mechanisms of craniocerebral injuries but also the long-term prognosis of brain injury for children on whom these injuries have been inflicted.

CONCLUSION. Knowledge of the traumatic mechanisms, the key neuroimaging findings, and the implications of functional imaging findings should help radiologists characterize the underlying causes of the injuries inflicted, thereby facilitating effective treatment.

The prevalence of abusive head trauma remains high with nearly 690,000 substantiated reports each year [1]. Because of the morbidity and mortality due to injuries to the CNS, the focus has continued to be on the heads of abused children. In the United States each year, approximately 1640 children die of nonaccidental injury, and at least 10% of cases of mental retardation and cerebral palsy result from child abuse [1, 2]. As of 2000, child abuse was the most common cause of serious head injury among children younger than 1 year [3]. The risk factors for inflicted traumatic brain injury include age younger than 1 year and mother young and unmarried with a low educational level, low socioeconomic status, disability, behavior or emotional problems, and multiple births [1, 4]. The mother is the most common perpetrator, followed by parent (i.e., the perpetrator was identified as the parent, but the sex was not specified), father, and male relatives [1].

Abusive head trauma (AHT) involves different mechanisms of forces, including shaking and impact injuries, direct blows to the head, compression, strangulation, penetrating injuries, smothering, and suffocating [5, 6]. There is often a combination of cranial and skeletal injuries to a young child without a history of overt trauma or to a child with a history of trivial trauma inconsistent with the degree of the CNS injuries. The most common presentations are seizures, encephalopathy, retinal hemorrhages, and altered level of consciousness without external evidence of head injury [7]. These findings are nonspecific and can be misleading. Investigating them is time-consuming, resulting in delays in diagnosis and treatment. Although abused children may be reported as having accidental falls at home, these falls are rarely associated with severe intracranial injury [8–10]. Having no history of trauma reported to treating physicians is the most predictive historical feature that a child with head trauma has been abused [11]. Therefore, radiologists are frequently called to court to testify about imaging findings [12].

The general mortality rate of AHT is greater than 15% [4]. Only one third of abused children completely recover, and as many as one half of survivors have cognitive or other neurologic deficits [13]. A number of psychiatric and physical disorders are associated with child abuse [14–18] and have been related to alteration of epigenetic regulation of several disease-related genes [19, 20].

The protective mechanisms of the brain that guard against injury mature with age. Infants are particularly vulnerable to nonaccidental trauma, such as shaking, because of the size of the head in relation to the rest of the body (almost one tenth of the body mass), the sizable subarachnoid spaces, and ineffective head support from weak neck muscles [21]. The infant brain is soft owing to immature myelination and small axonal size [13]. Furthermore, the infant skull is thin, soft, and easily deformed and readily transmits impact to the deeper brain structures.

Neuroimaging Features That Differentiate Abusive From Nonabusive Head Trauma

Although AHT and nonabusive head trauma have many common neuroradiologic findings, some features are helpful in differentiating one from the other. In a meta-analysis of 21 studies [22], hemorrhages that were subdural, multiple, in the convexity, interhemispheric, or in the posterior fossa were significantly associated with AHT. In addition, hypoxic-ischemic injury and cerebral edema were significantly associated with AHT, but focal parenchymal injury and subarachnoid hemorrhage (SAH) were not discriminatory features. In a systematic review of 24 studies [23], subdural hematoma, cerebral ischemia, retinal hemorrhage, skull fracture, and intracranial injury were found to be significantly associated with AHT. In contrast, epidural hematoma, scalp swelling, and isolated skull fracture were significantly associated with nonabusive head trauma. In a pooled analysis, Maguire et al. [24] found that retinal hemorrhages and rib fractures were the most discriminating findings of AHT in children younger than 3 years with intracranial injuries and any of the other clinical features, including apnea, seizure, and head or neck bruising. A positive correlation was found between the predictive value of AHT and number of clinical features; the finding implied a combination of multiple mechanisms involved in AHT.

The radiologic findings of AHT may vary in different types of injury, such as blunt impact, shaking with or without impact, strangling, stabbing, and poisoning. We review the common and uncommon neuroimaging patterns of CNS injuries in the categories of different traumatic mechanisms of child abuse.

Injury Mechanisms and Related Neuroimaging Features

Blunt Impact

Blunt impact is a dynamic force delivered at a point. It varies by type and degree of assault, such as being hit by a hard object, being thrown out of a window and hitting the ground, or being struck against a wall [25]. This type of injury usually leads to an immediate impact effect that includes subgaleal hematoma, skull fracture, brain contusion, and epidural or subdural hemorrhages (Fig. 1A).

Skull fractures are found in as many as one third of children with AHT [26]. Nonaccidental skull fractures may be linear or depressed, mainly in the parietal or occipi-

tal bones. The fractures can be stellate (eggshell fractures), bilateral, and multiple and can cross the sutures. Growing fractures are frequent in infants [27] (Fig. 1B and 1C). In children with inflicted injuries, fractures of the long bones and ribs are more frequent than skull fractures [25]. Therefore, careful examination of the long bones and ribs with a high-quality radiologic skeletal series survey is mandatory to search for recent or old fracture in suspicious cases [28, 29].

The force resulting in skull fracture tends to cause focal underlying brain injuries, such as hemorrhagic contusion and subdural hematoma. The subdural hematoma is along the interhemispheric fissure and over the convexities. Extradural and intracerebral hemorrhages are relatively uncommon and may be overlooked when the hematomas are small. Susceptibility-weighted imaging has shown promise compared with CT and conventional MRI, depicting additional hemorrhagic lesions 30% of the time in cases of pediatric traumatic brain injury [30] (Fig. 2B). In AHT, the number and volume of hemorrhagic lesions on susceptibility-weighted images have been proved to correlate with Glasgow coma scale score, need for surgical intervention, length of hospital stay, length of intubation, and intellectual function [31]. The imaging findings in the initial stages of AHT may be important for treatment and prognosis.

Intracranial hemorrhages due to blunt impact are often accompanied by brain edema and white matter laceration, which are important causes of increased intracranial pressure in the acute stage. Late sequelae of contusion injury include gray and white matter necrosis, encephalomalacia, ventricular dilation, shrinkage of the cerebral hemispheres, and subdural collections. In children with mild traumatic brain injury, trivial changes in regional brain volume can be detected with whole-brain parcellation analysis [32].

Shaking Injury

Shaking injury is caused by forceful shaking of an infant's head by grasping the shoulders or chest in an impulse that changes the momentum of the force by acceleration or deceleration [33]. The asynchronous to-and-fro movements of both cerebral hemispheres at different speeds along the sides of the falx cerebri are further aggravated by the large size of the head and the weakness of the neck muscles. The shearing force on the retina and cerebral veins and the high intracranial venous pressure resulting from

chest wall compression are thought to lead to retinal hemorrhage and bleeding into the potential subdural space. In addition to the linear force, the angular or rotational shearing force, especially when prolonged shears are applied, is likely to cause diffuse axonal injury (DAI), which frequently involves the parasagittal white matter [34] (Fig. 3A). Moreover, shaking without impact may not generate enough force to cause the typical brain damage of shaken baby syndrome [21]. Classic shaking-impact trauma to the head encompasses a scalp bruise, depressed skull fracture, and brain contusions (Fig. 2). Shaking-impact injury may accompany fractures of the humeral shafts and the ribs at the costovertebral junction owing to grasping by the shoulders, upper arms, or rib cage [21].

After traumatic injury, cerebral autoregulation and ionic homeostasis can be lost, and leakage of fluid from damaged blood vessels and increased cerebral blood flow from direct disturbance of vasomotor control may result in subsequent brain edema. In an animal model of head impact injury, raised intracranial pressure caused by extensive breakdown of the blood-brain barrier and albumin extravasation plays an important role in cerebral edema [35]. The dysregulated cerebral region is also at high risk of ischemic or hyperemic injury. The immature infant brain is more vulnerable to brain edema, though the underlying mechanism is not fully understood [36, 37].

Acute cerebral edema can sometimes be recognized at CT within the first 12 hours after injury but is less well visualized at MRI owing to the high water content of the unmyelinated white matter during infancy. At CT, the major findings are decreased attenuation of cerebral gray and white matter, resulting in low attenuation of the involved brain region with loss of gray-white matter differentiation. The thalamus, brainstem, and cerebellum may be selectively spared during the initial phase of ischemia, probably because the posterior circulation is preserved by autoregulation. The low-attenuation cortical mantle and relatively dense thalamus and cerebellum seen at CT are known as the reversal sign [38].

Infants sustaining shaking injury typically have retinal hemorrhages and subdural and diffuse cerebral edema [24]. This type of injury can present without external evidence of injury, such as a skull fracture, scalp bruise, or soft-tissue swelling, making a clinical diagnosis challenging.

Subdural hematomas are uncommon as a result of nonabusive head trauma in the first 2 years of life but are the most frequent imaging findings in infants with AHT [9]. The hematomas occur most often in the posterior interhemispheric region and tend to extend over or under the cerebral hemispheres [39] (Fig. 3B). CT shows thin layers of subdural hematoma, which may be obscured by the beam-hardening effect, commonly in the posterior cranial fossa. In addition, small subacute and chronic subdural hematomas are difficult to detect owing to similar attenuation between the hematomas and the CSF or the brain. Unlike CT, MRI is sensitive to the degraded RBCs and can be used to estimate the age of a subdural hematoma [40]. In general, the evolution of subdural hematoma is similar to that of intracerebral hematoma; acute hematomas are mildly hypointense on T1-weighted images and hypointense on T2-weighted images. Early subacute hematomas are hyperintense on T1-weighted images and hypointense on T2-weighted images. In late subacute subdural hematomas, the T2 relaxation time is prolonged owing to RBC lysis; the result is high signal intensity on both T1- and T2-weighted images (Fig. 4A). In the chronic stage, the signal intensity of a hematoma is less than in the acute phase but remains higher than that of CSF on FLAIR images because its protein content is greater than that of CSF (Fig. 4B).

The signal-intensity changes of subdural hematomas can be different from those of intracerebral hematomas. For example, the hemosiderin deposition in chronic parenchymal hematomas may not be observed in chronic subdural hematomas because of the paucity of phagocytosis in the subdural space [41]. Subdural hematomas may resolve slower than parenchymal hematomas because brain tissue contains high concentrations of tissue thromboplastin, which accelerates the resolution of a hematoma [42]. In addition, the signal intensity of subdural hematoma also varies with the size and location of the hematoma, rate of hemoglobin degradation, and mixed components of hematoma and CSF [43, 44]. Owing to the complex signal characteristics of subdural hematoma, particularly in patients with repeated trauma, caution should be exercised when timing the traumatic event solely on the basis of MRI findings.

In a 2012 meta-analysis [23] AHT was found to be not significantly associated with SAH and DAI [23]. A 2011 systematic review

[22], however, had shown a significant association between AHT and SAH. The meta-analysis was limited by the considerable heterogeneity of the studies included, and the systematic review did not include DAI in the analysis. In the neuroimaging literature, DAI and SAH are frequently reported to be related to AHT [45–48]. In shaking injury, SAH commonly results from tears of the small vessels in the pia and arachnoid mater and appears in the interhemispheric fissure or high convexity. CT and T2-weighted FLAIR MRI both are sensitive for SAH [49].

DAI is an important sequela of severe acceleration-deceleration brain shear injuries. In a classic study, Duhaime et al. [50] found DAI in pathologic examinations of both infants who died and a shaking-impact model in subhuman primates. The findings highlighted the importance of impact as part of the pathogenesis of DAI in shaking injuries. Shear injury often involves the subcortical white matter, corpus callosum, brainstem, and internal capsule. Gradient-echo sequences and susceptibility-weighted imaging are the techniques of choice for detecting small white matter hemorrhages. Diffusion-weighted MRI (DWI) together with apparent diffusion coefficient mapping can be used to detect nonhemorrhagic axonal injury [51, 52] (Fig. 5A). DWI is sensitive in the detection of early DAI lesions and for showing a greater extent of injury to the cerebral white matter than does conventional T2-weighted imaging [51, 53, 54]. The pathogenesis of restricted water diffusion in DAI is not entirely understood. Factors such as ischemia and propagated injury from oxidants or free radicals to the differentiating oligodendrocytes in cerebral white matter have been suggested as possible causes of DAI [55, 56].

Retinal hemorrhage has been strongly associated with AHT [22, 23]. It is believed to be associated with the firm vitreous in infants and is caused by a sudden increase in intracranial venous pressure due to violent chest compression. Hemorrhage may also occur in the subretinal, preretinal, and intraretinal spaces and sometimes fills the vitreous. On MR images, susceptibility-sensitive imaging techniques, such as gradient-recalled echo and susceptibility-weighted imaging, can delineate the nodular foci of low signal intensity in the posterior aspect of the globe along the retina with relatively high sensitivity [57, 58]. Although retinal hemorrhage was considered one of the classic presentations of AHT with high prevalence (54–100%) [59],

the finding is controversial and can be associated with various other diseases [60].

Diffusion-tensor imaging can delineate subtle white matter alterations in nonabusive head trauma patients with DAI [61]. Arfanakis et al. [62] reported reduced fractional anisotropy values in the corpus callosum and internal capsule in mild brain injury. Bazarian et al. [63] also found changes in fractional anisotropy and mean diffusivity in patients subjected to multiple subconcussive blows to the head. The value of diffusion-tensor imaging in AHT must be further investigated. DAI may also disrupt brain networks by damaging white matter tracts, a finding that can be revealed with resting-state functional MRI [64, 65].

MR spectroscopy can reveal metabolite derangement in injured brain tissue that is not seen on conventional MR images. In patients with shaking injury, low ratios of *N*-acetyl aspartate (NAA) to creatine and of NAA to choline and the presence of lactate peaks in the gray matter may appear without structural signal-intensity abnormalities. These findings are associated with poor outcome in the care of neonates with brain injury [66] (Fig. 5B).

Whiplash-Shaking Injury at Craniovertebral Junction

In addition to damaging the cerebrum, whiplash-shaking injury can damage the lower brainstem and upper cervical cord [36], contributing to apnea and hypoxia. Violent shaking may lead to substantial cervical spinal injury with subdural and epidural hematomas and contusions of the spinal cord at the cervicomedullary junction [67]. Ligamentous ruptures at the craniovertebral junction are not rare in patients with this type of injury (Fig. 6).

Strangulation Injury

Manual strangulation can lead to two types of craniocerebral trauma. One is hypoxic-ischemic encephalopathy (HIE) secondary to compromise of the blood flow from the common carotid artery. The other is subdural hematoma secondary to shearing of bridging veins while the infant is being grasped by the neck and shaken. HIE is usually confined to the territories of the internal carotid artery. DW images show largely unilateral white matter injury after extensive cortical infarction [47]. This is caused by the inherent anatomic vulnerability of the carotid arteries to extrinsic compression where the arteries lie between the transverse processes of the cervical vertebrae and the ster-

nocleidomastoid muscles. Bimanual assault can produce bilateral injuries (Fig. 7), a first sign of strangling.

Although not pathognomonic, AHT with strangulation should be suspected when a young child presents with HIE [68]. Careful inspection of CT or MR images for traces of intracranial hemorrhage, such as subdural hematoma, is important for correct diagnosis (Fig. 8). In addition to vascular compression, reactive vasospasm adjacent to hemorrhagic lesions, cervicomedullary injuries, and apnea can also lead to HIE in strangulation injury [69–71]. Because cerebral hypoperfusion caused by strangling is transient, the infarction is usually not associated with arterial stenosis or occlusion. Hemorrhagic laminar necrosis may occur in strangulation survivors and usually appears 7–10 days after the initial hypoxic injury [45] (Figs. 7B and 7C).

Whole-brain arterial spin-labeled perfusion MRI has been successfully used to evaluate neonatal brains [72, 73]. Because it does not carry the risk of contrast administration, this noninvasive technique may play a role in the assessment of ischemic injury inflicted on a young brain.

Stabbing Injury

Stabbing injury to the brain is a neurosurgical emergency with a high risk of death from intracranial bleeding and subsequent infection [74, 75]. Potential complications include intracerebral hematoma, posttraumatic aneurysm, carotid-cavernous fistula, arterial occlusion, venous thrombosis, and CSF leakage [76]. The severity and extent of the injury should be confirmed with neuroimaging. Because MRI is contradicted in the presence of a metallic foreign body, CT is the modality of choice in this circumstance (Fig. 9). Most patients need a craniotomy because direct extraction carries risk of bleeding [75].

Fetal Abuse

Fetal abuse includes physical assaults on an unborn fetus and neglect or failure to protect the fetus from toxic substances introduced by the maternal route, such as alcohol, nicotine, and drugs [77]. Direct fetal injury involves a blow to the abdomen of a pregnant woman. This type of assault can be partially cushioned by the maternal tissue and amniotic fluid, and thus the fetus is protected. When fetal injury occurs, the most frequent finding is intracranial hemorrhage [78, 79] (Fig. 10).

Fetal alcohol syndrome, the most serious adverse consequence of prenatal alcohol exposure, is fairly frequent with an estimated prevalence rate ranging from 0.5 to 7.0 cases per 1000 births in the United States [80]. Alcohol disrupts developmental processes through multiple sites of action, harming the developing embryo and fetus [81]. Presentations of fetal alcohol syndrome include characteristic facial abnormalities, such as smooth philtrum, thin vermillion border, and short palpebral fissures; prenatal and postnatal growth deficits; and CNS abnormalities, such as neurologic, behavioral, and structural anomalies [81].

Proposed Imaging Protocols

Jaspan et al. [82] proposed a neuroimaging protocol for cases of high clinical suspicion of AHT. Cranial CT should be performed on the day of presentation. On days 1–2, skull radiography and cranial ultrasound are suggested. On days 3–4, MRI should be performed if the child has neurologic symptoms in the presence of initially normal or equivocal first CT findings. In addition to conventional T1- and T2-weighted and FLAIR sequences, the MRI protocol should include T2*-weighted gradient-echo or susceptibility-weighted imaging to detect hemorrhage. DWI is suggested for evaluating HIE. Major arterial trauma is rarely encountered and can be evaluated with MR angiography. Other advanced imaging techniques may be considered optional and be used on a case-by-case basis.

Conclusion

With knowledge of the mechanisms of AHT and the related neuroimaging findings in young children, radiologists continue to play a pivotal role not only in diagnosis but also in facilitating effective treatments and improving patient outcome.

References

1. U.S. Department of Health and Human Services, Administration for Children and Families, Administration on Children, Youth and Families, Children's Bureau. *Child maltreatment 2012*. www.acf.hhs.gov/sites/default/files/cb/cm2012.pdf. Accessed January 8, 2015
2. Minns RA, Brown JK, eds. *Shaking and other non-accidental head injuries in children*. London, UK: Mac Keith Press, 2005:154–184
3. Barlow KM, Minns RA. Annual incidence of shaken impact syndrome in young children. *Lancet* 2000; 356:1571–1572
4. Keenan HT, Runyan DK, Marshall SW, Nocera MA, Merten DF, Sinal SH. A population-based study of inflicted traumatic brain injury in young children. *JAMA* 2003; 290:621–626
5. Merten DF, Osborne DR, Radkowski MA, Leonidas JC. Craniocerebral trauma in the child abuse syndrome: radiological observations. *Pediatr Radiol* 1984; 14:272–277
6. Cohen RA, Kaufman RA, Myers PA, Towbin RB. Cranial computed tomography in the abused child with head injury. *AJR* 1986; 146:97–102
7. Alexander R, Sato Y, Smith W, Bennett T. Incidence of impact trauma with cranial injuries ascribed to shaking. *Am J Dis Child* 1990; 144:724–726
8. Williams RA. Injuries in infants and small children resulting from witnessed and corroborated free falls. *J Trauma* 1991; 31:1350–1352
9. Duhaime AC, Alario AJ, Lewander WJ, et al. Head injury in very young children: mechanisms, injury types, and ophthalmologic findings in 100 hospitalized patients younger than 2 years of age. *Pediatrics* 1992; 90:179–185
10. Thomas AG, Hegde SV, Dineen RA, Jaspan T. Patterns of accidental craniocerebral injury occurring in early childhood. *Arch Dis Child* 2013; 98:787–792
11. Hettler J, Greenes DS. Can the initial history predict whether a child with a head injury has been abused? *Pediatrics* 2003; 111:602–607
12. Royal College of Radiology and Paediatrics and Child Health. *Standards for radiological investigations of suspected non-accidental injury*. London, UK: Royal College of Radiology and Paediatrics and Child Health, 2008
13. Case ME, Graham MA, Handy TC, Jentzen JM, Monteleone JA. Position paper on fatal abusive head injuries in infants and young children. *Am J Forensic Med Pathol* 2001; 22:112–122
14. Felitti VJ, Anda RF, Nordenberg D, et al. Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults: the Adverse Childhood Experiences (ACE) study. *Am J Prev Med* 1998; 14:245–258
15. Dong M, Giles WH, Felitti VJ, et al. Insights into causal pathways for ischemic heart disease: adverse childhood experiences study. *Circulation* 2004; 110:1761–1766
16. Anda RF, Brown DW, Dube SR, Bremner JD, Felitti VJ, Giles WH. Adverse childhood experiences and chronic obstructive pulmonary disease in adults. *Am J Prev Med* 2008; 34:396–403
17. Dube SR, Fairweather D, Pearson WS, Felitti VJ, Anda RF, Croft JB. Cumulative childhood stress and autoimmune diseases in adults. *Psychosom Med* 2009; 71:243–250
18. Brown DW, Anda RF, Felitti VJ, et al. Adverse childhood experiences are associated with the risk of lung cancer: a prospective cohort study. *BMC Public Health* 2010; 10:20
19. Yang BZ, Zhang H, Ge W, et al. Child abuse and epigenetic mechanisms of disease risk. *Am J Prev Med* 2013; 44:101–107

20. Beach SR, Brody GH, Todorov AA, Gunter TD, Philibert RA. Methylation at *SLC6A4* is linked to family history of child abuse: an examination of the Iowa adoptee sample. *Am J Med Genet B Neuropsychiatr Genet* 2010; 153B:710-713

21. Bandak FA. Shaken baby syndrome: a biomechanics analysis of injury mechanisms. *Forensic Sci Int* 2005; 151:71–79

22. Kemp AM, Jaspan T, Griffiths J, et al. Neuroimaging: what neuroradiological features distinguish abusive from non-abusive head trauma? A systematic review. *Arch Dis Child* 2011; 96:1103–1112

23. Piteau SJ, Ward MG, Barrowman NJ, Plint AC. Clinical and radiographic characteristics associated with abusive and nonabusive head trauma: a systematic review. *Pediatrics* 2012; 130:315–323

24. Maguire SA, Kemp AM, Lumb RC, Farewell DM. Estimating the probability of abusive head trauma: a pooled analysis. *Pediatrics* 2011; 128:e550–e564

25. Kraus JF, Fife D, Cox P, Ramstein K, Conroy C. Incidence, severity, and external causes of pediatric brain injury. *Am J Dis Child* 1986; 140:687–693

26. Arnholz D, Hymel KP, Hay TC, Jenny C. Bilateral pediatric skull fractures: accident or abuse? *J Trauma* 1998; 45:172–174

27. Rinehart GC, Pittman T. Growing skull fractures: strategies for repair and reconstruction. *J Craniofac Surg* 1998; 9:65–72

28. Hornor G. Physical abuse: recognition and reporting. *J Pediatr Health Care* 2005; 19:4–11

29. Nimkin K, Kleinman PK. Imaging of child abuse. *Radiol Clin North Am* 2001; 39:843–864

30. Beauchamp MH, Ditchfield M, Babl FE, et al. Detecting traumatic brain lesions in children: CT versus MRI versus susceptibility weighted imaging (SWI). *J Neurotrauma* 2011; 28:915–927

31. Beauchamp MH, Beare R, Ditchfield M, et al. Susceptibility weighted imaging and its relationship to outcome after pediatric traumatic brain injury. *Cortex* 2013; 49:591–598

32. Zhou Y, Kierans A, Kenul D, et al. Mild traumatic brain injury: longitudinal regional brain volume changes. *Radiology* 2013; 267:880–890

33. Gerber P, Coffman K. Nonaccidental head trauma in infants. *Childs Nerv Syst* 2007; 23:499–507

34. Hymel KP, Partington MD, Winston KR. Abusive head trauma? A biomechanics-based approach. *Child Maltreat* 1998; 3:116–128

35. Byard RW, Bhatia KD, Reilly PL, Vink R. How rapidly does cerebral swelling follow trauma? Observations using an animal model and possible implications in infancy. *Leg Med (Tokyo)* 2009; 11(suppl 1):S128–S131

36. Hadley MN, Sonntag VK, Rekate HL, Murphy A. The infant whiplash-shake injury syndrome: a clinical and pathological study. *Neurosurgery* 1989; 24:536–540

37. Kinney HC, Armstrong DD. Perinatal neuropa-

thology. In: Graham DI, Lantos PL, eds. *Greenfield's neuropathology* 7th ed. London, UK: Arnold, 2002:519–606

38. Han BK, Towbin RB, De Courten-Myers G, McLaurin RL, Ball WS Jr. Reversal sign on CT: effect of anoxic/ischemic cerebral injury in children. *AJR* 1990; 154:361–368

39. Ewing-Cobbs L, Prasad M, Kramer L, et al. Acute neuroradiologic findings in young children with inflicted or noninflicted traumatic brain injury. *Childs Nerv Syst* 2000; 16:25–33; discussion, 34

40. Datta S, Stoodley N, Jayawant S, Renowden S, Kemp A. Neuroradiological aspects of subdural haemorrhages. *Arch Dis Child* 2005; 90:947–951

41. Chen CY, Zimmerman RA, Rorke LB. Neuroimaging in child abuse: a mechanism-based approach. *Neuroradiology* 1999; 41:711–722

42. Vezina G. Assessment of the nature and age of subdural collections in nonaccidental head injury with CT and MRI. *Pediatr Radiol* 2009; 39:586–590

43. Zouros A, Bhargava R, Hoskinson M, Aronyk KE. Further characterization of traumatic subdural collections of infancy: report of five cases. *J Neurosurg* 2004; 100:512–518

44. Duhem R, Vinchon M, Tonnelle V, Soto-Ares G, Leclerc X. Main temporal aspects of the MRI signal of subdural hematomas and practical contribution to dating head injury. *Neurochirurgie* 2006; 52:93–104

45. Reece RM, Sege R. Childhood head injuries: accidental or inflicted? *Arch Pediatr Adolesc Med* 2000; 154:11–15

46. Rajaram S, Batty R, Rittey CD, Griffiths PD, Connolly DJ. Neuroimaging in non-accidental head injury in children: an important element of assessment. *Postgrad Med J* 2011; 87:355–361

47. Ashwal S, Wycliffe ND, Holshouser BA. Advanced neuroimaging in children with nonaccidental trauma. *Dev Neurosci* 2010; 32:343–360

48. Matschke J, Herrmann B, Sperhake J, Korber F, Bajanowski T, Glatzel M. Shaken baby syndrome: a common variant of non-accidental head injury in infants. *Dtsch Arztebl Int* 2009; 106:211–217

49. Noguchi K, Ogawa T, Inugami A, et al. Acute subarachnoid hemorrhage: MR imaging with fluid-attenuated inversion recovery pulse sequences. *Radiology* 1995; 196:773–777

50. Duhaime AC, Gennarelli TA, Thibault LE, Bruce DA, Margulies SS, Wiser R. The shaken baby syndrome: a clinical, pathological, and biomechanical study. *J Neurosurg* 1987; 66:409–415

51. Chan YL, Chu WC, Wong GW, Yeung DK. Diffusion-weighted MRI in shaken baby syndrome. *Pediatr Radiol* 2003; 33:574–577

52. Liu AY, Maldjian JA, Bagley LJ, Sinson GP, Grossman RI. Traumatic brain injury: diffusion-weighted MR imaging findings. *AJNR* 1999; 20:1636–1641

53. Biousse V, Suh DY, Newman NJ, Davis PC, Mapstone T, Lambert SR. Diffusion-weighted mag-

netic resonance imaging in shaken baby syndrome. *Am J Ophthalmol* 2002; 133:249–255

54. Dan B, Damry N, Fonteyne C, Jissendi P, Ziereisen F, Christophe C. Repeated diffusion-weighted magnetic resonance imaging in infantile non-haemorrhagic, non-accidental brain injury. *Dev Med Child Neurol* 2008; 50:78–80

55. Shannon P, Smith CR, Deck J, Ang LC, Ho M, Becker L. Axonal injury and the neuropathology of shaken baby syndrome. *Acta Neuropathol* 1998; 95:625–631

56. Volpe JJ. Brain injury in the premature infant: neuropathology, clinical aspects, pathogenesis, and prevention. *Clin Perinatol* 1997; 24:567–587

57. Altinok D, Saleem S, Zhang Z, Markman L, Smith W. MR imaging findings of retinal hemorrhage in a case of nonaccidental trauma. *Pediatr Radiol* 2009; 39:290–292

58. Zuccoli G, Panigrahy A, Haldipur A, et al. Susceptibility weighted imaging depicts retinal hemorrhages in abusive head trauma. *Neuroradiology* 2013; 55:889–893

59. Aryan HE, Ghosheh FR, Jandial R, Levy ML. Retinal hemorrhage and pediatric brain injury: etiology and review of the literature. *J Clin Neurosci* 2005; 12:624–631

60. Graham DL, Gennarelli TA, McIntosh TK. Trauma. In: Graham DI, Lantos PL, eds. *Greenfield's neuropathology,* 7th ed. London, UK: Arnold, 2002:823–898

61. Shenton ME, Hamoda HM, Schneiderman JS, et al. A review of magnetic resonance imaging and diffusion tensor imaging findings in mild traumatic brain injury. *Brain Imaging Behav* 2012; 6:137–192

62. Arfanakis K, Haughton VM, Carew JD, Rogers BP, Dempsey RJ, Meyerand ME. Diffusion tensor MR imaging in diffuse axonal injury. *AJNR* 2002; 23:794–802

63. Bazarian JJ, Zhu T, Blyth B, Borrino A, Zhong J. Subject-specific changes in brain white matter on diffusion tensor imaging after sports-related concussion. *Magn Reson Imaging* 2012; 30:171–180

64. Pandit AS, Expert P, Lambiotte R, et al. Traumatic brain injury impairs small-world topology. *Neurology* 2013; 80:1826–1833

65. Palacios EM, Sala-Llonch R, Junque C, et al. Resting-state functional magnetic resonance imaging activity and connectivity and cognitive outcome in traumatic brain injury. *JAMA Neurol* 2013; 70:845–851

66. Ashwal S, Holshouser BA, Shu SK, et al. Predictive value of proton magnetic resonance spectroscopy in pediatric closed head injury. *Pediatr Neurol* 2000; 23:114–125

67. Geddes JF, Vowles GH, Hackshaw AK, Nickols CD, Scott IS, Whitwell HL. Neuropathology of inflicted head injury in children. Part II. Microscopic brain injury in infants. *Brain* 2001; 124:1299–1306

68. Grant PE, Yu D. Acute injury to the immature brain with hypoxia with or without hypoperfusion. *Radiol Clin North Am* 2006; 44:63–77

69. Kemp AM, Stoodley N, Cobley C, Coles L, Kemp KW. Apnoea and brain swelling in non-accidental head injury. *Arch Dis Child* 2003; 88:472–476, discussion, 472–476

70. Pierce MC, Bertocci GE, Berger R, Vogeley E. Injury biomechanics for aiding in the diagnosis of abusive head trauma. *Neurosurg Clin N Am* 2002; 13:155–168

71. Zimmerman RA, Bilaniuk LT, Farina L. Non-accidental brain trauma in infants: diffusion imaging, contributions to understanding the injury process. *J Neuroradiol* 2007; 34:109–114

72. Miranda MJ, Olofsson K, Sidaros K. Noninvasive measurements of regional cerebral perfusion in pre-term and term neonates by magnetic resonance arterial spin labeling. *Pediatr Res* 2006; 60:359–363

73. Wang Z, Fernandez-Seara M, Alsop DC, et al. Assessment of functional development in normal infant brain using arterial spin labeled perfusion MRI. *Neuroimage* 2008; 39:973–978

74. James G, Blakeley CJ, Hashemi K, Channing K, Duff M. A case of self-inflicted craniocerebral penetrating injury. *Emerg Med J* 2006; 23:e32

75. Large M, Babidge N, Nielssen O. Intracranial self-stabbing. *Am J Forensic Med Pathol* 2012; 33:13–18

76. du Trevou MD, van Dellen JR Penetrating stab wounds to the brain: the timing of angiography in patients presenting with the weapon already removed. *Neurosurgery* 1992; 31:905–911; discussion, 911–912

77. Condon JT. The spectrum of fetal abuse in pregnant women. *J Nerv Ment Dis* 1986; 174:509–516

78. Lane PL. Traumatic fetal deaths. *J Emerg Med* 1989; 7:433–435

79. Pearlman MD, Tintinalli JE, Lorenz RP. Blunt trauma during pregnancy. *N Engl J Med* 1990; 323:1609–1613

80. May PA, Gossage JP, Kalberg WO, et al. Prevalence and epidemiologic characteristics of FASD from various research methods with an emphasis on recent in-school studies. *Dev Disabil Res Rev* 2009; 15:176–192

81. Warren KR, Hewitt BG, Thomas JD. Fetal alcohol spectrum disorders: research challenges and opportunities. *Alcohol Res Health* 2011; 34:4–14

82. Jaspan T, Griffiths PD, McConachie NS, Punt JA. Neuroimaging for non-accidental head injury in childhood: a proposed protocol. *Clin Radiol* 2003; 58:44–53
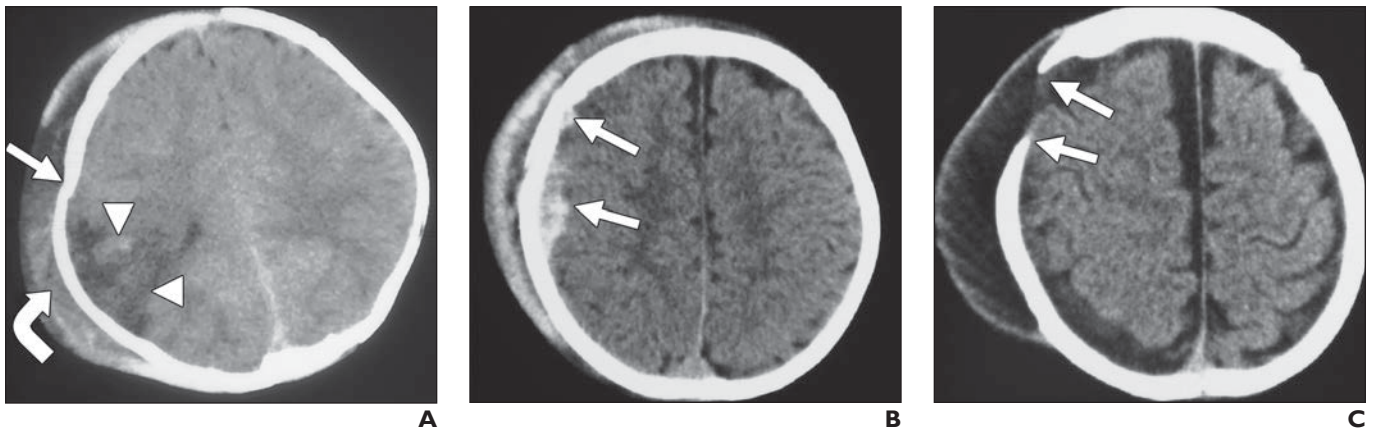
Fig. 1—Impact type of nonaccidental trauma.
A, 6-month-old boy. Axial CT scan shows right frontal depressed skull fracture (*straight arrow*) along with frontoparietal lobe contusion (*arrowheads*). Subgaleal hematoma is also present (*curved arrow*).
B, 5-month-old girl subjected to blunt impact over right frontal region. Axial CT image shows scalp swelling and cortical contusion hemorrhages (*arrows*). Linear diastatic fracture in right coronal suture was identified at bone window setting (not shown).
C, Same infant as in **B.** Axial CT image 3 months after initial injury shows fracture (*arrows*) has grown due to expansion of leptomeningeal cyst through fracture gap into subgaleal region.
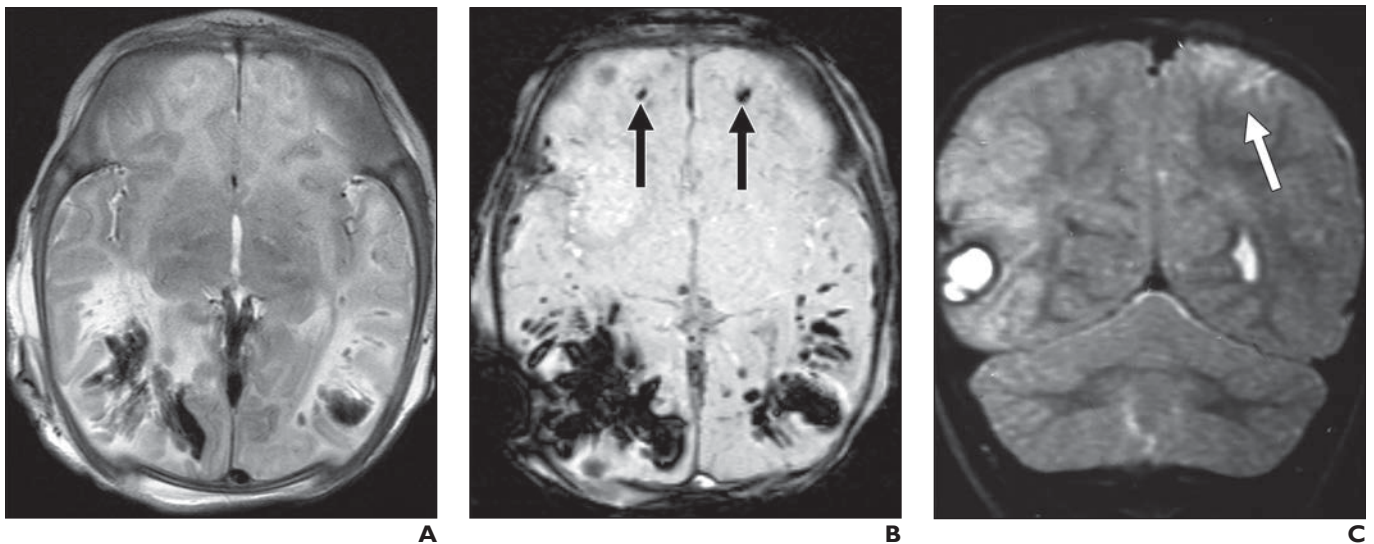

Fig. 2—11-month-old boy with shaking-impact injury.
A, Axial T2-weighted MR image shows edema and intracranial hemorrhages of both temporooccipital lobes.
B, Susceptibility-weighted MR image shows additional bilateral hemorrhagic foci (*arrows*) in frontal lobes; finding is consistent with diffuse axonal injury.
C, Coronal T2-weighted MR image shows cortical edema (*arrow*) over contralateral high convexity of frontal lobe.
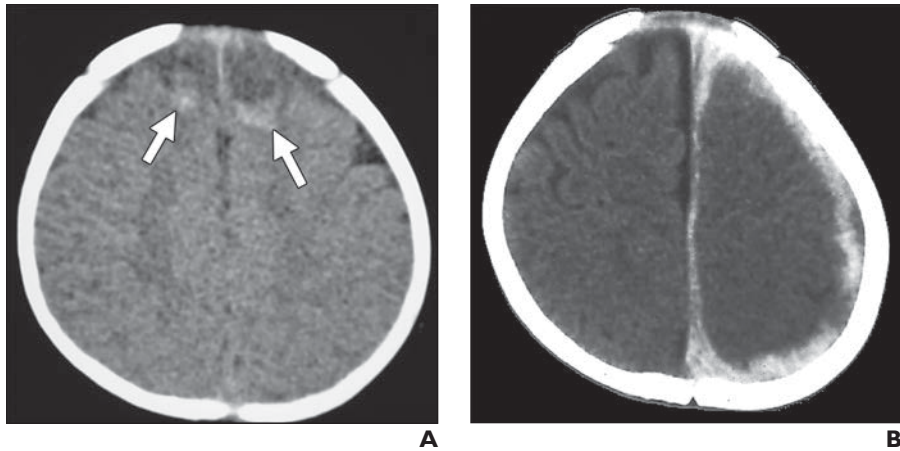
Fig. 3—CT of shaking injury.
A, 4-month-old girl subjected to violent shaking insult. CT scan shows typical parasagittal hemorrhages (*arrows*) in both superior medial frontal lobes as result of shearing injury.
B, 11-month-old boy. CT scan shows acute subdural hematomas, mainly over frontoparietal convexities and along interhemispheric fissure.
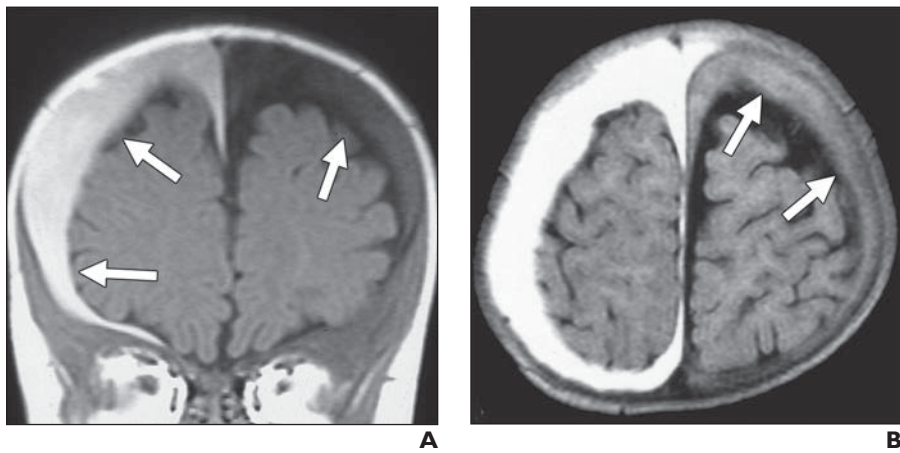

Fig. 4—MRI of shaking injury.
A, 4-month-old boy with subdural hematoma due to shaking injury. Coronal T1-weighted MR image shows bilateral subdural hematomas (*arrows*) in cerebral convexities in different stages. Right subdural hematoma is approximately in late subacute stage, and left is in chronic stage.
B, 4-month-old girl. Axial FLAIR image shows intermediate-signal-intensity chronic subdural hematoma (*arrows*) over high convexity of left hemisphere. Hyperintense right subdural hematoma is in subacute stage.
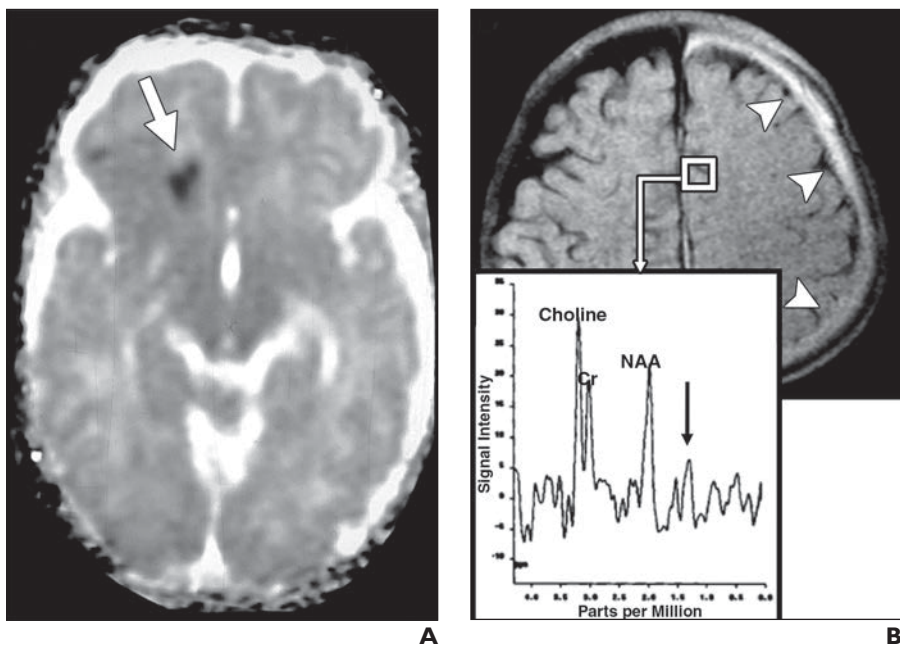

Fig. 5—Shaking-impact and diffuse axonal injury.
A, 8-month-old boy. Axial apparent diffusion coefficient image shows one spot of diffusion restriction (*arrow*), which appeared otherwise normal in other sequences (not shown).
B, 9-month-old girl with shaking injury. Voxel spectrum from multivoxel MR spectroscopic image (TE, 270 ms) of left frontal cortical region shows decrease in *N*-acetyl aspartate (NAA)-to-choline ratio compared with normal right frontal lobe spectrum (not shown) and presence of lactate peak (*arrow*) in frontal gray matter without abnormal signal intensity. Chronic subdural hematomas (*arrowheads*) are present in left cerebral convexity and along falx cerebri. Cr = creatine.
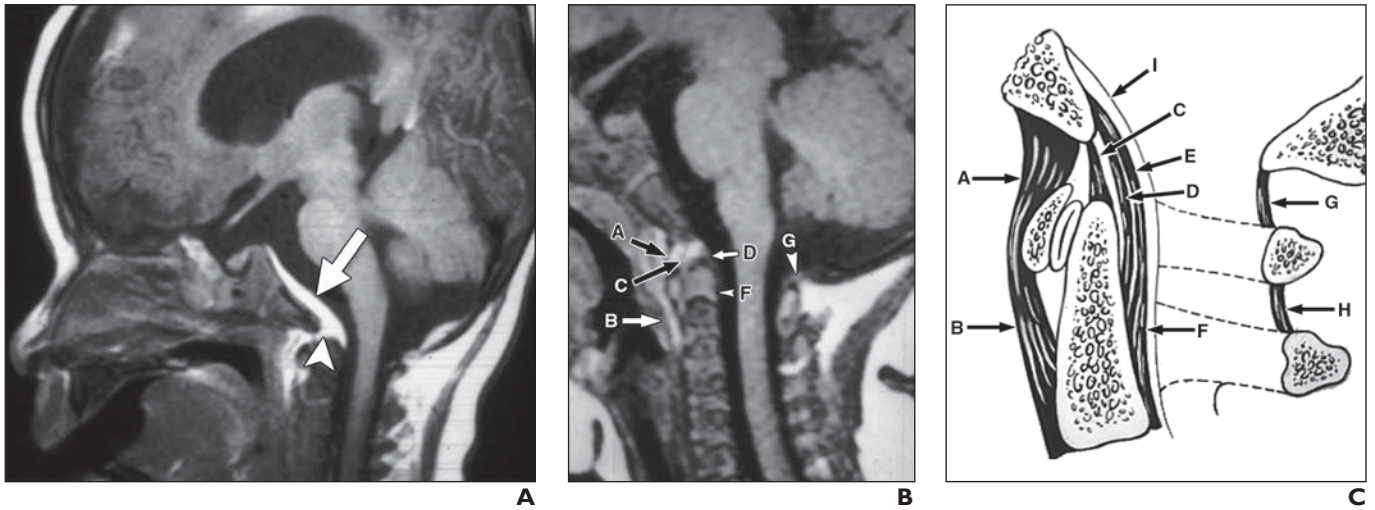
Fig. 6—3-month-old boy with craniovertebral junction injury.
A, Sagittal T1-weighted MR image shows, in addition to contusions of frontal and parietooccipital lobes, subdural hematoma along clivus and craniovertebral junction (*arrow*). Torn apical ligament with blood (*arrowhead*) is evident.
B, 1-year-old boy with normal ligamentous anatomy. Sagittal T1-weighted MR image shows ligamentous structures at craniovertebral junction. A = anterior atlantooccipital membrane, B = anterior longitudinal ligament, C = apical ligament, D = cruciate ligament, F = posterior longitudinal ligament, G = posterior atlantooccipital membrane.
C, Illustration shows craniovertebral ligaments. A = anterior atlantooccipital membrane, B = anterior longitudinal ligament, C = apical ligament, D = cruciate ligament, E = tectorial membrane, F = posterior longitudinal ligament, G = posterior atlantooccipital membrane, H = interspinous ligament, I = dura.
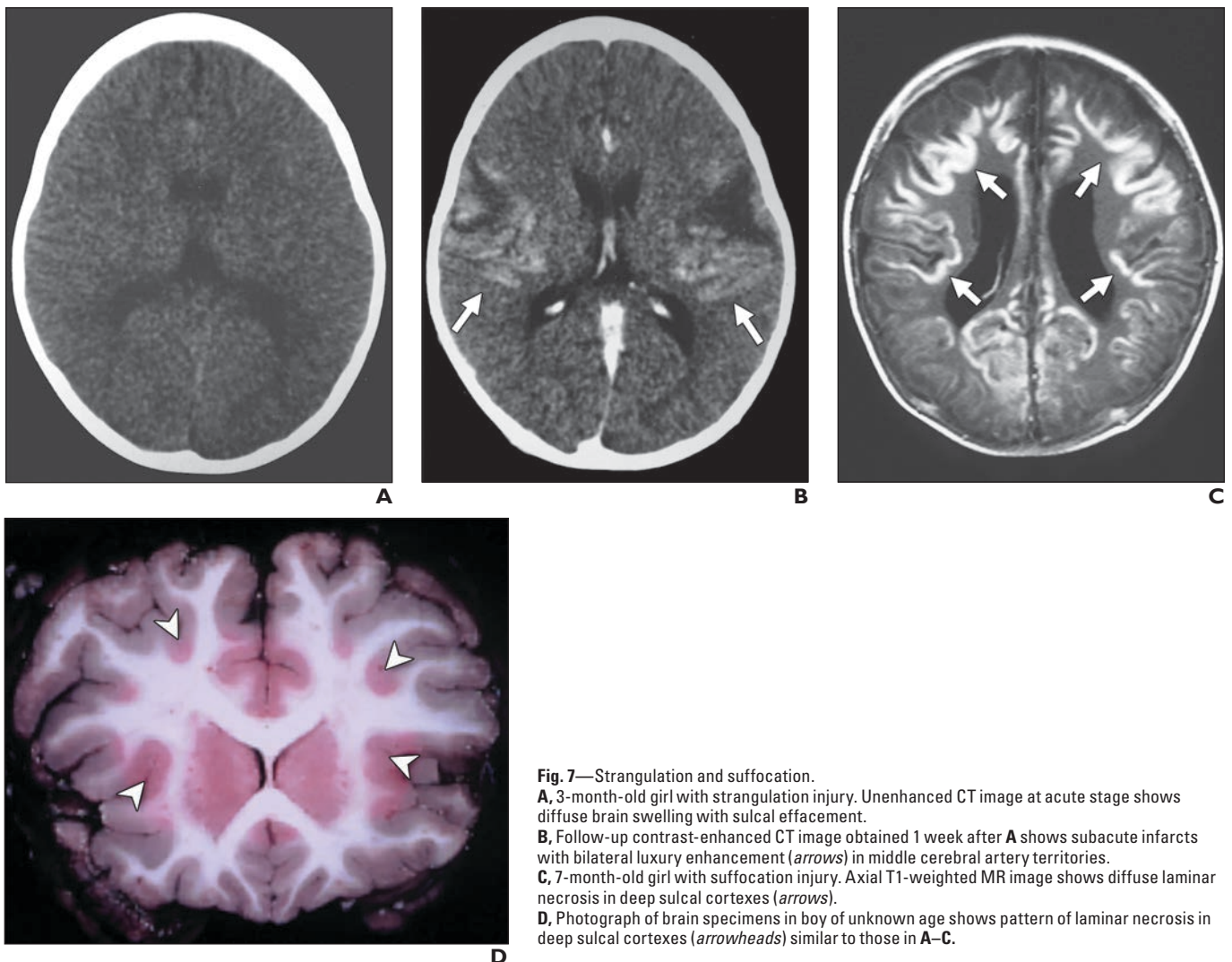

Fig. 7—Strangulation and suffocation.
A, 3-month-old girl with strangulation injury. Unenhanced CT image at acute stage shows diffuse brain swelling with sulcal effacement.
B, Follow-up contrast-enhanced CT image obtained 1 week after **A** shows subacute infarcts with bilateral luxury enhancement (*arrows*) in middle cerebral artery territories.
C, 7-month-old girl with suffocation injury. Axial T1-weighted MR image shows diffuse laminar necrosis in deep sulcal cortexes (*arrows*).
D, Photograph of brain specimens in boy of unknown age shows pattern of laminar necrosis in deep sulcal cortexes (*arrowheads*) similar to those in **A–C.**
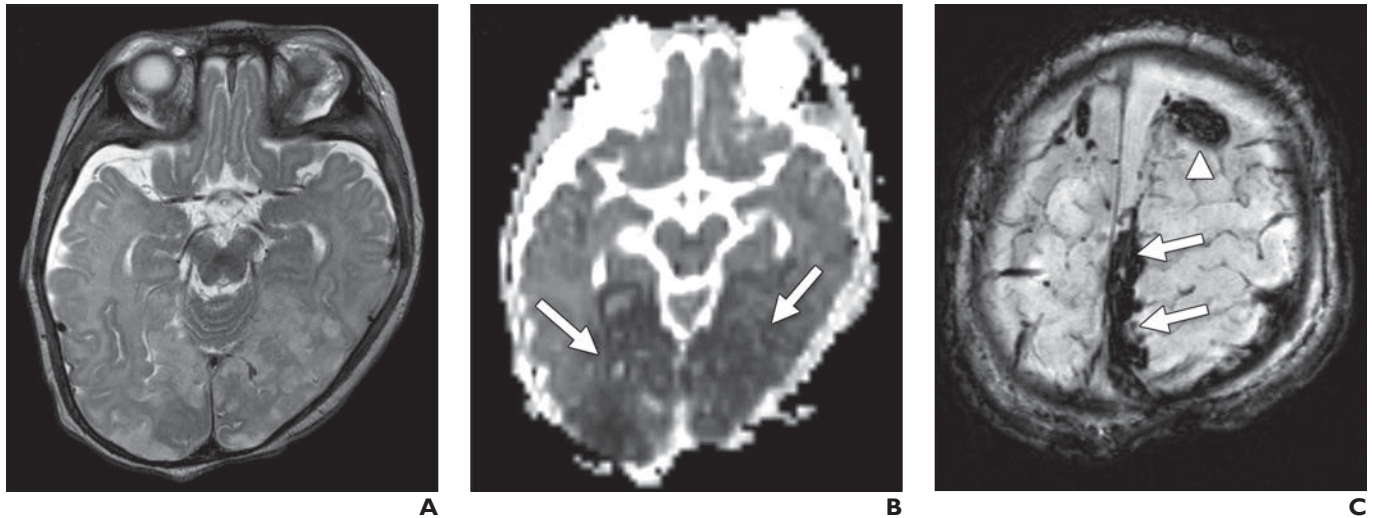
A   B   C

Fig. 8—2-month-old boy with shaking and strangulation injuries.
A, Axial T2-weighted MR image shows poorly defined injuries in unmyelinated brain.
B, Apparent diffusion coefficient map shows bilateral cytotoxic edema over posterior medial temporal and occipital lobes (*arrows*).
C, Susceptibility-weighted image shows subdural hematoma (*arrows*) and contusion hemorrhage (*arrowhead*).
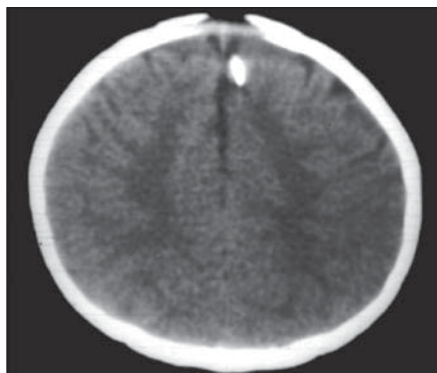

Fig. 9—6-month-old boy with stabbing injury through anterior fontanel. Axial CT scan shows retained paper clip in superior frontal interhemispheric cistern. Kidney-ureter-bladder radiograph (not shown) of mother, who had pica, showed numerous paper clips in stomach and intestines. (Reprinted with permission from Wang HS. A needle in an infant's brain. *Eur J Neurol* 1998; 5:517–518. Copyright 2003 by John Wiley and Sons)
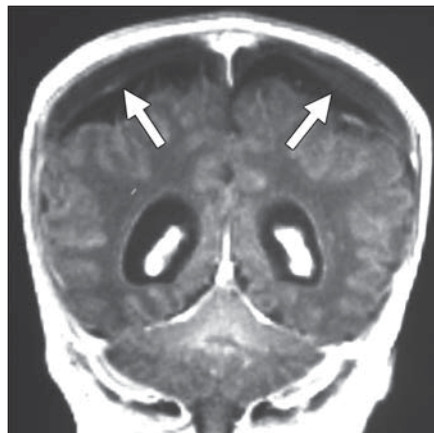

Fig. 10—12-day-old boy battered as fetus. Coronal contrast-enhanced T1-weighted MR image shows chronic bilateral subdural hematomas (*arrows*) in cerebral convexities.

FOR YOUR INFORMATION

This article is available for CME and Self-Assessment (SA-CME) credit that satisfies Part II requirements for maintenance of certification (MOC). To access the examination for this article, follow the prompts associated with the online version of the article.