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Abuse to the pediatric central nervous system.

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Abuse to the Pediatric Central Nervous System

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

—John Caffey

Large series of infants and children with head injuries of diverse cause (1, 2), have shown that a disconcertingly large number of the injuries occur under the age of 2 years. These series were compiled before the advent of computed tomography (CT) and magnetic resonance (MR) imaging, so the true nature of possible intrinsic central nervous system (CNS) damage was often not identified, other than at postmortem or at operation.

The primary work toward the identification and understanding of child abuse was by Caffey (3), followed by Kemple et al, and Silverman (4, 5) who coined the term "battered child." Caffey (6, 7) together with Guthkelch (8) described the prevalence and pathogenesis of CNS damage due to the whiplash-shaken infant syndrome. Understanding of the neuroimaging of the abused child was consolidated in comprehensive reports from diverse sources (9–12), with specific contributions of CT imaging (13–15), ultrasound, and now MR (16, 17).

It has been established that at least 1.5 million children are abused in the United States alone, one-half probably as a result of direct violent abuse. At least 3000 deaths occur, most commonly from head injuries (9). At least 10% of

children with mental retardation and cerebral palsy are presumed to have been damaged by abuse (9).

This report will review skull fractures; intracerebral hematomas, particularly the unusual parafalcial and intrafalcial hematomas; ischemic changes; and the "white cerebellum" sign, where the normal cerebellum appears bright on CT, relative to darker, globally ischemic cerebral hemispheres. The "white cerebellum" is an early sign, and sequel, of extracranial nonaccidental hypoxia.

Skull Fractures

In a child of any age, the presence of a linear skull fracture does not indicate any *increased* likelihood of significant intracranial injury (2); contrarily, its absence does not lessen the likelihood of intracranial damage. However, the presence of a depressed fracture from a seemingly mild injury, of a fracture that crosses the midline, or of a fracture of the occiput without a suitable cause raises suspicion of nonaccidental trauma.

In Merten's study of physically abused children with clinical evidence of a head injury, two-thirds had a skull fracture; 45% of the abused children with significant intracranial damage had a fracture, but, significantly, 55% did not (18). A fracture was very rarely seen in those children with evidence of brain damage due to extracranial nonaccidental causes of hypoxia, such as suffocation. Notwithstanding, the combination of skull fracture and evidence of child abuse significantly increases the likelihood of some intracranial injury. CT scans of potentially abused children should routinely include an additional set of wide window "bone" images of the head to facilitate detection of skull fractures (Fig. 1). It is our belief that it is also beneficial to obtain a preliminary

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Fig. 1. Skull fracture with acute-on-chronic subdural hematoma. Unenhanced axial CT demonstrates bilateral chronic frontal subdural hematomas and a large left chronic occipital hematoma with a fluid level of acute blood in the chronic hematoma. Soft-tissue swelling overlies a comminuted fracture with mild depression of the inner table.



set of standard skull radiographs, although, unfortunately, use of skull radiographs is declining in "modern" practice.

Intracranial Hematomas

Posttraumatic extradural, subdural, and intracerebral hematomas are common in violent child abuse (2, 12, 18). Such hematomas often coexist. Intraventricular hemorrhage and often subtle shear hemorrhages of the brain indicate severe "commotio cerebri." Severe violent shaking is probably responsible for both. In adults, the shape of the hematomas is nearly stereotypical and distinctive. In children, however, subdural and extradural hematomas may both be huge and may appear quite similar, no matter how the brain is imaged. Subdural hematomas may be biconcave and extradural hematomas convex. The specific shape of the hematoma probably depends on the rapidity of bleeding (often from a torn sinus); the variable malleability of the adjacent brain and vault; vault thinness; and the presence of sutures. A central nidus of low density or low signal intensity within an obvious clot may signify active bleeding.

CT and MR (19–21) confirms Caffey's (6) and Guthkelch's (8) proposal that interhemispheric (parafalcial) subdural hematomas (Fig. 2) result from violent, to-and-fro shaking of the pediatric head, with tearing of the bridging veins, commonly in the young child. Careful study of this abnormal force and its sequelae (22) indicates that sudden deceleration forces play a significant part whether the deceleration results from impact

against a hard or a soft object. The interhemispheric subdural hematomas may be as thick as 15 to 20 mm. Acceleration/deceleration trauma may also cause infrequent intrafalcial hematomas, thin, quill-like collections of blood (Fig. 3), due to fiber movement or micro tears within the relatively vascular pediatric falx (23, 24).

True coronal CT and MR will best identify both interhemispheric and falcial hematomas. These will suggest the possibility of nonaccidental cranial abuse. Discovery of subdural hematomas of differing maturity, including superimposition of acute upon chronic bleeds (Figs. 1, 4, and 5) will help to confirm this diagnosis. Clinical or imaging detection of retinal hemorrhages, especially bilateral retinal hemorrhages, is very important to diagnosis, since these are strongly linked with shaking or asphyxiation and may even be seen on CT if large (Fig. 6). Intravenous contrast-enhanced CT is often necessary to confirm chronicity by differing attenuation or intensity of its contents and by the presence of an inner membrane delimiting the hematoma. The subdural membrane may be seen as soon as 7 days after trauma.

MR is especially sensitive and specific as to the varying ages of subdural hematomas (Fig. 5). Use of MR increases the probability of detecting posterior fossa subdural hematomas that are commoner in an abused and shaken infant (Fig. 7) than in routine trauma victims. MR may show an inner line of low signal intensity that likely represents either an inner rim of hemosiderin and calcification within the chronic membrane (which can be seen on CT as early as 2 weeks) or within

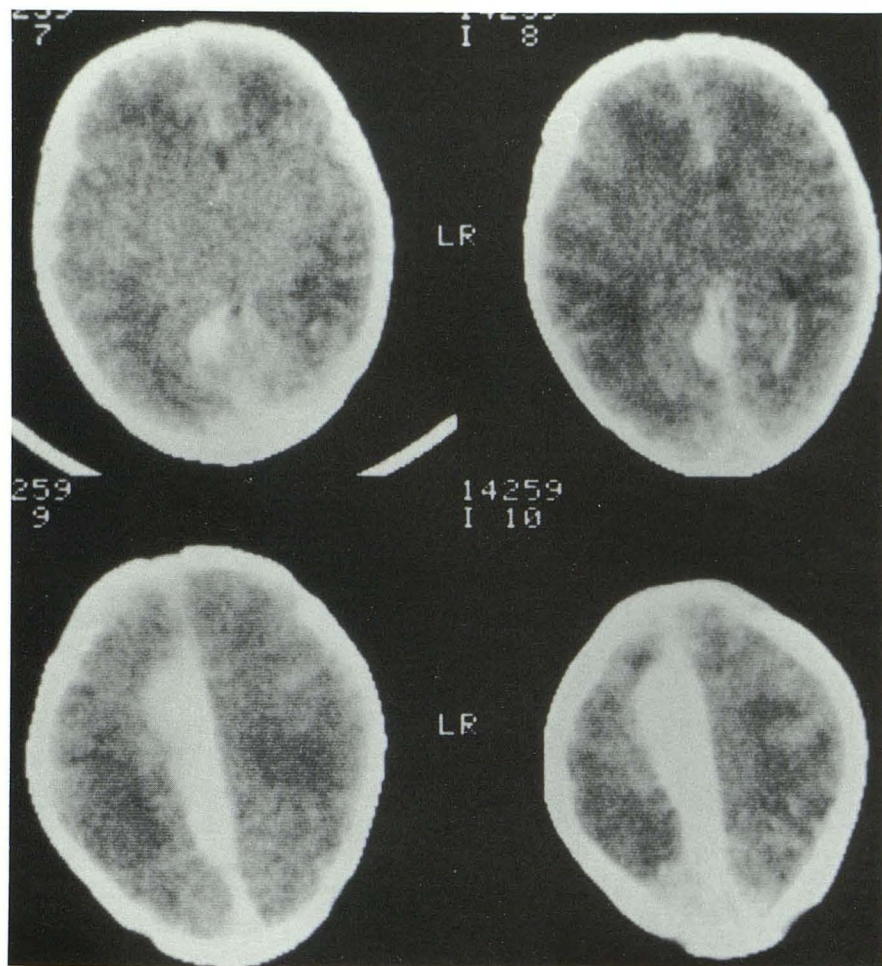


Fig. 2. Parafalcine subdural hematoma. An extensive right-sided parafalcine parietal subdural hematoma in a shaken infant with a right-to-left midline shift.

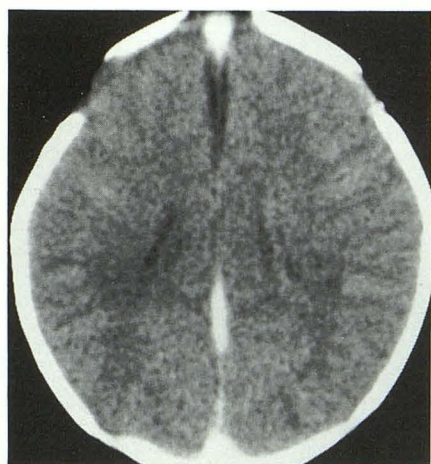


Fig. 3. Intrafalcine hematoma. In this abused infant, the hematoma is intrafalcine, ie, situated within the leaves of the falx posteroinferiorly.

the dura itself, delimiting an extradural hematoma.

Shear hemorrhages, often difficult to see by CT (25, 26), are more easily detected by MR, and indicate a significant rotatory or whiplash head

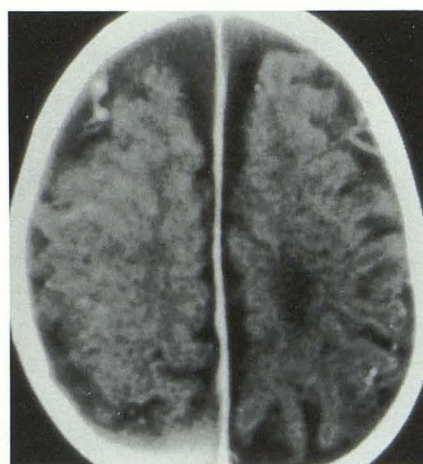


Fig. 4. Acute on chronic subdural hematoma. Nonenhanced axial CT demonstrates bilateral chronic subdural hematomas with acute blood posteriorly on the right and a small amount anteriorly on the right. Note a dense irregular falx, probably smeared with acute blood.

motion. They are often seen at gray/white interfaces or within large white matter tracts, like the corpus callosum and forceps. Shear hemorrhages

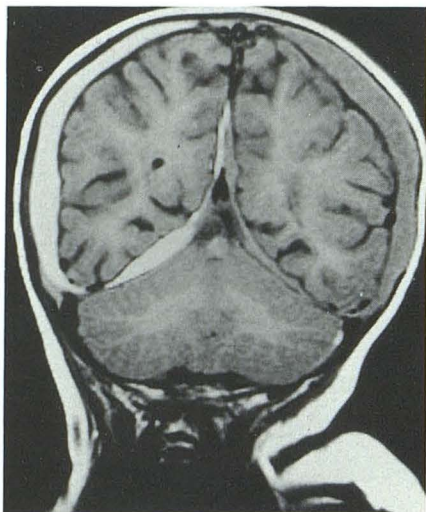


Fig. 5. Acute and chronic subdural hematomas. Coronal T1-weighted MR demonstrates bilateral acute and chronic circumferential, posteriorly placed subdural hematomas.

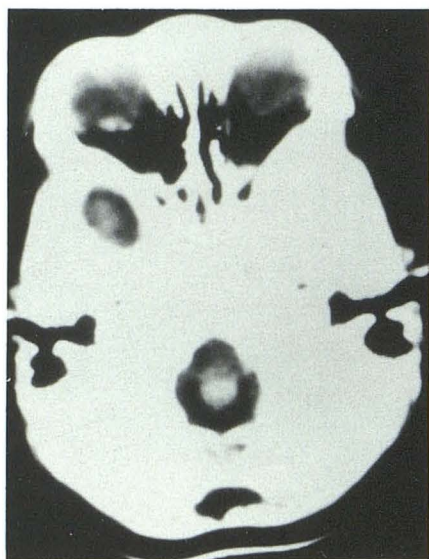


Fig. 6. Retinal hemorrhage. Unenhanced CT shows a right-sided retinal hemorrhage in a severely shaken child.

indicate an ominous prognosis of probable significant cerebral dysfunction. Tearing of ependymal veins with intraventricular hemorrhage carries a similarly grim prognosis.

Ischemia and Infarction

The early edema phase of post-abuse ischemia/infarction is little understood and less well identified by imaging techniques. The accumulation of abnormal fluid, cellular biochemical changes (27–29), and hypoxic damage may cause either potentially reversible ischemia or

nonreversible infarction. However, present imaging studies are unable to distinguish between these two.

Local edema (with or without associated intracerebral hematomas) appears to be reversible, unless it causes a significant brain shift, occlusion of cerebral vessels, and a resultant vicious circle of herniation and progressive vascular compromise (23, 24).

Irreversible ischemia leading to infarction appears to arise from such a vicious circle or from extracranial nonaccidental abuse resulting in hypoxia, cerebral ischemia, and global infarction. The specific patterns of ischemia/infarction are variable. Hypoxia may lead to an initial selective ischemia of the basal ganglia (Fig. 8) or to cortical/subcortical ischemia with decreased prominence of the gray/white matter interface on CT (30–32). Paradoxically, ischemia may selectively spare the basal ganglia (Fig. 9), or cause relatively increased attenuation of cerebral white matter (15), the so-called "reversal sign." This reversal sign is less well identified by MR in the acute phase. The most reliable and certain sign of acute ischemia is a relative sparing of the cerebellum and the brain stem from the effects of hypoxia, probably due to the primitive "diving" reflex, leading to the preservation of the cerebellum. Because the cerebellum is preserved, it has a much greater (normal) attenuation on CT, relative to the subtler reduced attenuation of the ischemic cerebral hemispheres. This is the *white cerebellum sign* (Fig. 10). In our experience this sign commonly indicates an extracerebral cause of cerebral hypoxia, induced by such unfortunate

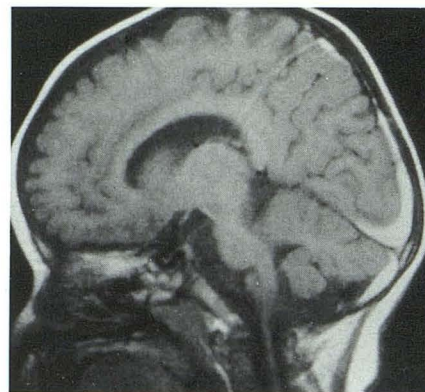


Fig. 7. Posterior fossa subdural hematomas. Sagittal T1-weighted MR demonstrates posteriorly placed subdural hematomas in the posterior fossa and surrounding the occipital pole. This appearance is a characteristic of abuse in the young child and is probably due to marked deceleration forces.

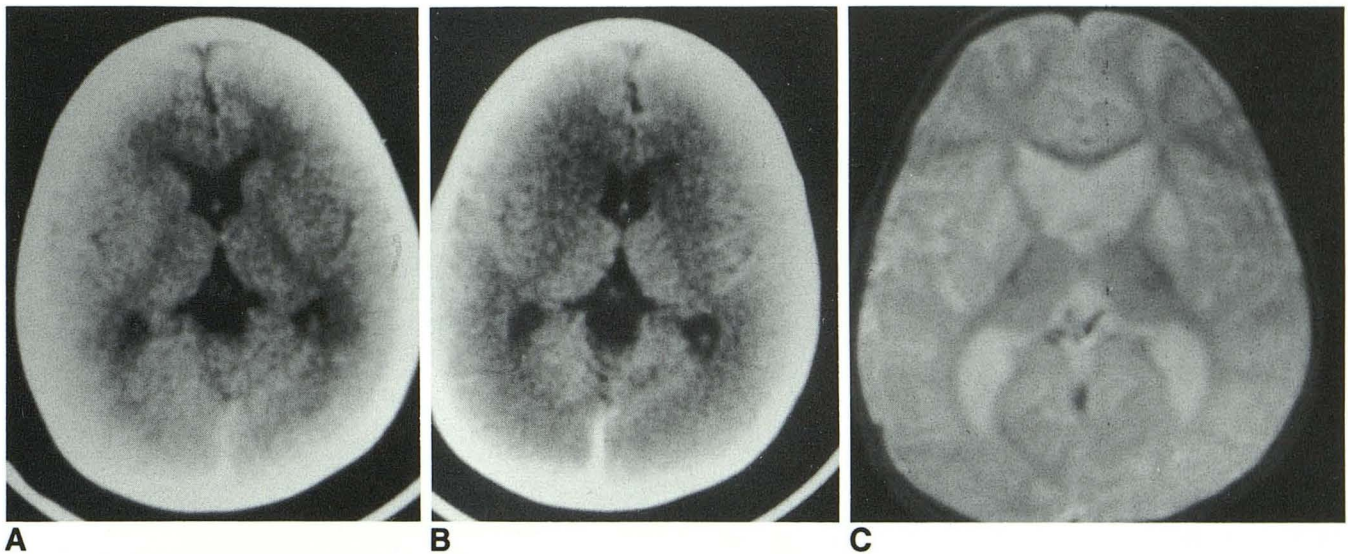


Fig. 8. Nonaccidental extracranial asphyxia.

A, Initial unenhanced axial CT in a child that was smothered. Lack of definition between gray and white matter superficially and at the basal ganglia is a suspicious warning sign in a child with diminished consciousness 2 hours after "falling off the couch."

B, Unenhanced CT performed 36 hours later shows classical ischemia of the basal ganglia and hypodensity of the white matter, with relative sparing of the thalami.

C, Proton density-weighted MR performed 1 day after the second CT demonstrates ischemia of the caudate nucleus and globus pallidus bilaterally. The white matter changes are less obvious.



Fig. 9. Cerebral ischemia with basal ganglia sparing. Unenhanced CT shows severe edema in both hemispheres with poor cortical gray/white matter interface, starkly preserving the basal ganglia, thalami, and cerebellum.

circumstances as smothering, strangulation (30), or pulmonary aspiration by abdominal contents due to trunk compression. Regrettably this subtle imaging sign has often been the only indication of suspicious injury in patients subsequently identified as having suffered abuse. This sign is obviously not pathognomonic for abuse, but includes other clinically well-recognized causes of

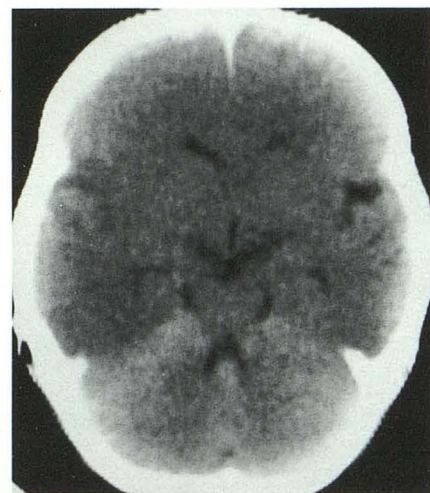


Fig. 10. The white cerebellum sign in a deeply unconscious child with severe chest compression and probable strangling. Unenhanced CT within 6 hours of the assault demonstrates the white cerebellum. The greater density of the cerebellum than of the cerebrum indicates that the cerebellum is relatively normal compared to the ischemic cerebral hemispheres. This is a valuable sign of early, still subtle cerebral ischemia.

hypoxia, be they drowning, circulatory collapse of any cause, smoke inhalation (Fig. 11), among others.

Focal infarction may be associated with subsequent hemorrhage, not directly due to the trauma. Generalized lobar or hemispheric ische-

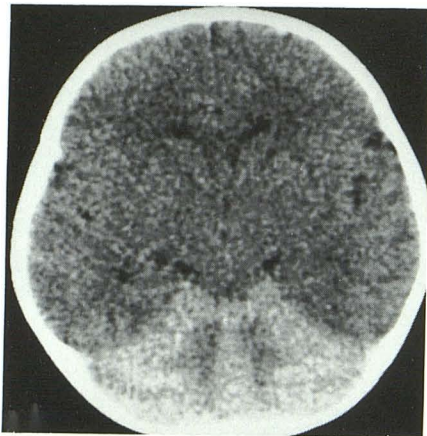


Fig. 11. White cerebellum sign in smoke inhalation. Unenhanced CT shows a markedly white cerebellum in a nontraumatized child with severe smoke inhalation and asphyxia, who subsequently died, probably due to brain-stem infarction.

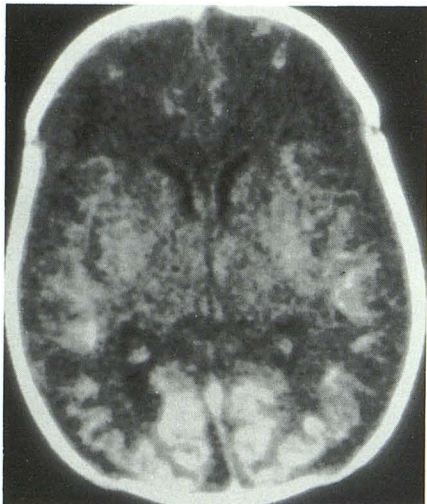


Fig. 12. Global ischemia with laminar infarction. Unenhanced CT demonstrates global cerebral ischemia with diffuse parieto-temporal occipital gray matter laminar infarction with hemorrhage and minor preservation of the basal ganglia perfusion.

mia may result in laminar infarction within the gray matter vascular watershed (Fig. 12), and result in calcification that may be strangely evanescent, disappearing after a variable time.

MR will best confirm basal ganglia damage (Fig. 8) and also accurately demonstrate chronic sequelae, varying from periventricular leukomalacia in the young to focal or general leukomalacia (Fig. 13).

Conclusion

A child, particularly an infant, who presents with overt (or absent) signs of abuse; peripheral fractures of different ages; subdural hematomas

of differing ages; a clinically puzzling or inappropriate loss of consciousness with a white cerebellum sign; and an enlarging head without hydrocephalus, should be suspected at least tentatively, of having received nonaccidental direct or indirect cranial and/or extracranial abuse. Skull radiographs, a chest radiograph to detect a fracture of the clavicle from chest or neck compression, a radionuclide bone scan to detect possible healing fractures, and a head CT should be performed initially. Whether or not a "white cerebellum sign" be present, a repeat CT should always be performed 5 days later if abuse is suspected. The two CTs and perhaps an MR, will confirm the possible presence of cerebral ischemia, especially if the diagnosis was not clear initially. Emerging infarction can also be correctly identified. MR will detect and identify subdural hematomas, especially those of differing ages (16) and accurately reveal laminar infarction with possible subsequent hemosiderin/calcification (23), as does CT.

Regardless of the social circumstances, rigorous but tactful inquiries should ensue as to whether abuse is a likelihood; rigorous for not only the immediate clinical management but to prevent infliction of future damage. *Nobody* within the day-to-day environment of the child is excluded. The well being of the unfortunate infant or child is paramount and becomes the responsibility of all who participate in his or her care. The neuroradiologist has an important part to

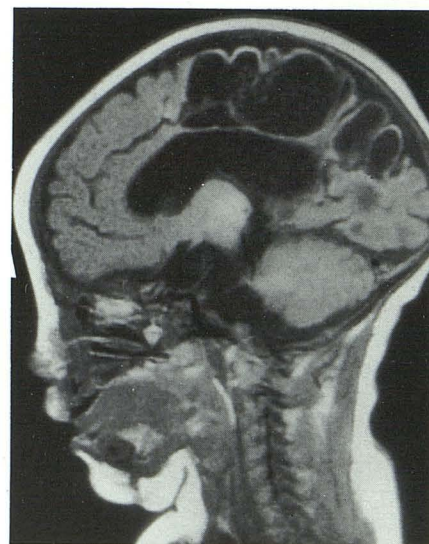


Fig. 13. Chronic sequelae of severe leukomalacia. Sagittal T1-weighted MR demonstrates extensive and severe leukomalacia of the parieto-occipital region following brain ischemia due to abuse. (Reproduced with permission from Radiological Society of North America and *Revista di Neuroradiologica*.)

play toward accurately identifying the morbid pathology, all the more because he or she is in the front line and is often the first clinical participant in this context.

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