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Serial Radiography in the Infant Shaken Impact Syndrome

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Abstract
Certain CT and/or MRI abnormalities have been used medicolegally to time intracranial injuries from the infant shaken impact syndrome (ISIS). For example, parenchymal hypodensities on CT scans are said to arise only after 6–48 h have elapsed postinjury, and the presence of chronic or mixed subdural hematomas suggests injury that occured 1–4 weeks prior. However, these statements are based largely upon inference from data obtained in other conditions such as ischemic anoxic injury and chronic subdural hemorrhage in adults. Direct evidence about the evolution of intracranial injuries in infants with ISIS is sparse, and the radiographic changes following ISIS have never been systematically studied on serial imaging studies. One hundred-seventeen serial CT and MRI scans obtained from 33 infants with ISIS were reviewed retrospectively. The exact scan dates and times were obtained directly from the scans. Acute subdural hemorrhage was the most common intracranial abnormality and was present in 27 (81%) of the 33 infants. Other intracranial abnormalities included chronic subdural collections, subarachnoid hemorrhage, epidural hematomas, parenchymal hypodensities, edema and contusions, and atrophy and encephalomalacia. In 15 of the 33 infants, the injury could be timed with reasonable certainty, and the evolution of the radiographic changes followed over time. Six of the 15 infants had evidence of prior cranial trauma such as chronic subdural collections (5 infants) or mild atrophy (1 infant). Of the remaining 9 infants, parenchymal abnormalities such as hypodensities, edema and contusion appeared in virtually all of the initial scans performed approximately 3 h following the report of injury. One 'chronic' subdural collection was absent on the first scan performed 2.75 h postinjury, but appeared on a second scan performed 17 h later, suggesting that some 'chronic' subdural fluid collections may arise much sooner than previously thought. These findings challenge some of the current dogma about the timing of radiographic changes following abuse and are important in timing the alleged abuse for legal purposes.

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Introduction

A number of intracranial injuries (both extra- and intra-axial) are well known to occur in association with the infant shaken impact syndrome (ISIS), and their radiographic appearances on computerized tomographic (CT) and magnetic resonance imaging (MRI) studies have been well described [1–27]. Certain radiographic findings such as posterior perifalcine and/or tentorial subdural hemorrhage, or the presence of mixed density subdural hemorrhage in an infant, constitute highly suspicious markers for abuse [11, 17, 21, 22, 25–29].

The timing of such injuries is of obvious medicolegal importance in that it gives a legal framework for the timing of the alleged abuse, and may identify a likely perpetrator [14]. For example, the available literature suggests that the presence of parenchymal hypodensities on the initial CT scan implies abuse which occurred at least 6–48 h prior [26, 30]. The presence of mixed density subdural hematomas suggests multiple episodes of abuse, and the presence of chronic subdural hematomas implies abuse which occurred 1–4 weeks prior [5, 8, 12, 22, 30, 31].

However, these statements are based largely upon observations made in other disorders. For example, the suggestion that parenchymal hypodensities in ISIS take 6–48 h to appear is derived largely from the evolution of radiographic changes in ischemic-anoxic injuries [4, 11, 26]. The suggestion that chronic subdural hematomas require 1–4 weeks to evolve is based largely upon the evolution of subdural and intraparenchymal hemorrhage in adults and animals [8, 14, 26, 32]. The pathophysiological mechanisms underlying ISIS are very different from those underlying either ischemic-anoxic injury or chronic subdural hemorrhage in adults. To date, the timing of radiographic changes on CT and MRI scans in ISIS has never been systematically studied. We therefore examined the evolution of radiographic changes on serial imaging studies in children with ISIS.

Materials and Methods

The Children's Hospital of Buffalo (CHOB) is a tertiary referral center for pediatric head trauma for an eight county region of Western New York. The medical records and serial radiographs of all infants with ISIS treated at CHOB between November 1992 and May 1997 were retrospectively reviewed. The clinical history was determined by systematically examining all information in the chart including ambulance and Emergency Department records, admission and consult notes, social workers' notes, New York State Child Protective Service records, and discharge summaries. Whenever possible, the exact time at which the injury was reported to have occurred was established through (1) direct statements in the medical chart; (2) the time of calls made to Emergency Medical Services, and/or (3) the time at which the ambulance received a call or arrived on the scene.

The date and time of each CT or MRI scan, as well as the time between the alleged injury and the scan, were recorded. All scans were reviewed by two of the authors (M.S.D. and J.B.). For each scan, all intracranial injuries were documented. In particular, the presence or absence of the following injuries was assessed: (1) skull fractures; (2) epidural hemorrhage; (3) acute subdural hemorrhage (defined as hyperdense extra-axial collections on CT, without a gyral pattern, overlying the cerebral convexities, adjacent to the falx cerebri, or along the tentorium); (4) chronic or mixed subdural hemorrhage; (5) subarachnoid hemorrhage (defined as hyperdense extra-axial collections on CT, with a gyral pattern, overlying the cerebral convexities, adjacent to the falx cerebri, or along the tentorium; or hyperdensities within the basal cisterns or sylvian fissures); (6) parenchymal hypodensities on CT; (7) the 'reversal sign' (in which the densities of the deep nuclei and the overlying cerebral hemispheres are reversed); (8) brain edema (defined as loss of gray-white matter differentiation on CT); (9) parenchymal contusion (defined as focal punctate parenchymal hyperdensities on CT); (10) atrophy (defined as a diffuse, global loss of brain substance with sulcal widening and ventricular enlargement), and (11) encephalomalacia (defined as focal loss of brain substance). For each of these abnormalities, we documented both the number of the scan (first, second, etc.) on which the abnormality *first* appeared, and the time interval between the scan and the time at which the injury was reported.

Results

Thirty-three infants with ISIS were identified during the study period, yielding an average of 7.2 infants with ISIS per year. The average age of the cohort was 6.7 months (with a range of 1–26 months). Twenty-four (73%) of the infants were boys. With respect to the alleged mechanism of injury (as provided in the chart), the baby fell in 7 cases (21%), was dropped in 3 (9%), and was struck in 1 (3%). In the remaining 22 cases, no history of trauma was provided. Apnea and/or seizures occurred in 22 (67%) of the 33 infants, and retinal hemorrhages were documented in 23 (92%) of the 25 infants in whom this was recorded. The overall mortality rate was 21% (7 of 33 infants).

One hundred and seventeen MRI and CT scans from the 33 infants were evaluated; skull X-rays (whenever available) were also evaluated for skull fractures which might not have been visible on the CT scans. All infants had undergone at least one scan; 5 infants had undergone only 1 scan, 4 infants had undergone 2 scans, 3 infants had undergone 3 scans, 10 infants had undergone 4 scans, and 11 infants had undergone 5 or more scans. One hundred and ten of the studies were CT scans; MRI scans

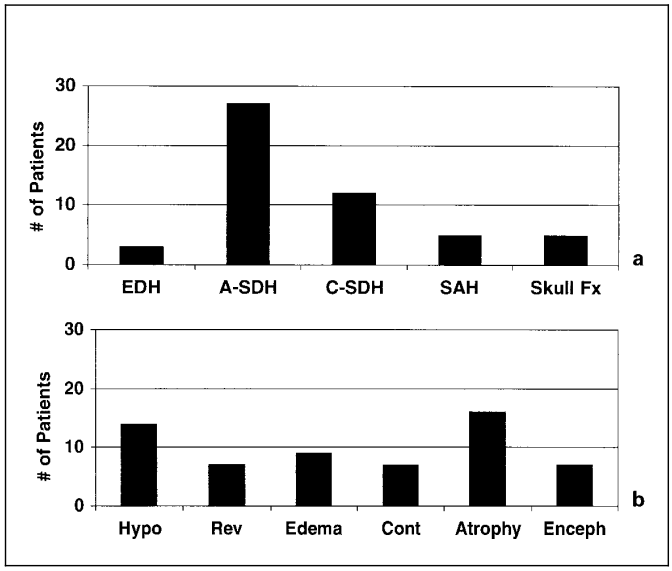

Fig. 1. Distribution and frequency of intracranial abnormalities among 33 infants with ISIS. **a** Extra-axial abnormalities. EDH: Epidural hematoma; A-SDH: acute subdural hematoma; C-SDH: chronic subdural hematoma; SAH: subarachnoid hemorrhage; Skull Fx: skull fracture. **b** Intraparenchymal abnormalities. Hypo: Hypodensities, Rev: reversal sign, Cont: contusion, Enceph: encephalomalacia.

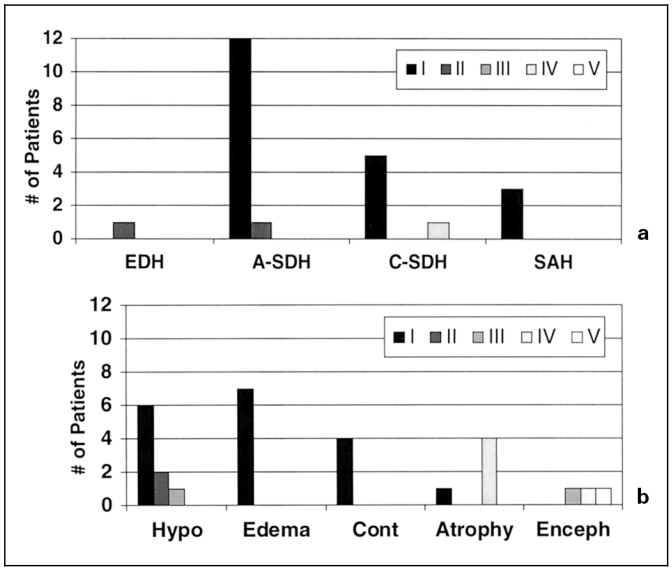

Fig. 2. Distribution and frequency of intracranial abnormalities among 15 infants with ISIS in whom the time of alleged abuse could be pinpointed. **a** Extra-axial abnormalities. **b** Intraparenchymal abnormalities. For abbreviations, see legend for figure 1.

were performed in only 7 infants – 2 as second studies, 4 as third studies, and 1 as the fourth study.

The overall frequency of intracranial injuries, as documented on any radiographic study, is provided in figure 1. Only 5 of the 33 infants (15%) had skull fractures (documented by either skull X-rays or CT scans). Acute subdural hemorrhage was the most common intracranial injury, and was present in 27 (81%) of the 33 infants. Among 43 acute subdural hematomas in 27 infants (several infants had subdural hematomas involving more than one area), the perifalcine and/or tentorial regions were involved in 28 (65%) whereas convexity hemorrhage occurred in only 15 (35%).

The clinical documentation in 15 (45%) of the 33 infants was sufficient to pinpoint the time at which the injury was reported. Among these infants, there was either a specific and documented time at which an injury was reported to have occurred, or there was an apneic spell, seizure, abrupt coma, or other significant and immediate event that prompted a call for emergeny medical services. The earliest appearance of various intracranial abnormalities on imaging studies among these 15 patients is listed in figure 2. Twelve of 13 acute subdural hemorrhages, 6 (67%) of 9 hypodensities, and all cases of edema and contusions were present on the initial scans. In contrast, only 1 (12.5%) of 8 cases of atrophy or encephalomalacia was present on the initial scans.

Table 1 lists the average elapsed time between the reporting of the injury and the earliest appearance of these abnormalities on imaging studies. For example, among the 12 acute subdural hemorrhages which were present on the initial scans, the average elapsed time between the reporting of the injury and the first scan was only 2.5 h. Among the hypodensities, edema, and contusions which were present on the first scan, the average elapsed time between the reporting of the injury and the initial scan was 3.2 h, 3.0 h, and 2.4 h, respectively.

Among the 15 infants in this group, 5 had either a chronic or mixed subdural hemorrhage, and 1 infant had mild atrophy on the initial scan, suggesting the possibility of prior or repeated abuse. These 6 infants were therefore excluded, and the remaining 9 infants were again analyzed separately. Figure 3 shows the earliest appearance of the various abnormalities, and table 2 the average times between the reported injury and the scans for this group of 9 infants. The pattern is similar; acute subdural hemorrhage, hypodensities, edema and contusions appear with overwhelming frequency on the first scan after the injury,

Table 1. Timing of the first appearance of various abnormalities on imaging studies among the 15 infants in whom the timing of the abuse could be pinpointed

	I	II	III	IV	V
EDH		18.4 (1)			
A-SDH	2.7 (12)	18.4 (1)			
C-SDH	2.7 (5)			12 d (1)	
SAH	3.9 (3)				
Hypodensity	3.2 (6)	14.6 (2)	67.5 (1)		
Edema	3.0 (7)				
Contusion	2.4 (4)				
Atrophy	4.7 (1)			26 d (4)	
Encephalomalacia			15 d (1)	7 d (1)	827 d (1)

The numbers in parentheses represent the number of patients in whom the abnormality was first detected on the imaging study (represented in Roman numerals at the top). The numbers indicate the average times (in hours unless otherwise specified) after the injury that these scans were performed. For abbreviations, see legend for figure 1.

Table 2. Timing of the first appearance of various abnormalities on imaging studies among the 9 infants in whom the timing of the abuse could be pinpointed; excludes the 6 infants in whom chronic subdural hemorrhage or atropy were seen on the first scan

	I	II	III	IV	V
EDH		18.4 (1)			
A-SDH	2.4 (7)	18.4 (1)			
C-SDH				12 d (1)	
SAH	3.9 (3)				
Hypodensity	3.2 (5)	18.4 (1)	67.5 (1)		
Edema	3.2 (5)				
Contusion	3.0 (4)				
Atrophy				26 d (4)	
Encephalomalacia				7 d (1)	827 d (1)

The numbers in parentheses represent the number of patients in whom the abnormality was first detected on the imaging study (represented in Roman numerals at the top). The numbers indicate the average times (in hours unless otherwise specified) after the injury that these scans were performed. For abbreviations, see legend for figure 1.

with average times from the reported injury of 2.4–3.2 h. In contrast, the earliest appearance of atrophy or encephalomalacia was on the fourth scan after injury, with average times from the reported injury of 7–26 days.

Finally, serial scans from all infants with an acute subdural hemorrhage on the first imaging study were analyzed separately to determine the evolution of acute to chronic subdural hemorrhage. Among the 33 infants, 21 had acute subdural hemorrhage, without a chronic or mixed component, on the initial scan. Five of the 21 infants died soon after admission. Two of the 5 underwent an evacuation of the acute subdural hematoma but died shortly thereafter because of their initial parenchymal injuries, and the remaining 3 infants died with smaller, nonoperative subdural collections and widespread parenchymal injuries. Of the 16 surviving infants, in none was the acute subdural hemorrhage large enough to require surgical intervention initially. Eleven (69%) of these 16 subdural collections eventually resolved without sequelae. Of the remaining 5 acute subdural hemorrhages, 1 rebled acutely on the third scan performed 7 days after admission and required evacuation, 1 had evolved into a chronic collection on the second scan performed 3 days after presentation, 2 on the third scan performed 7 and 11 days after presentation respectively, and 1 on the fourth scan performed 12 days after presentation. Unfortunately, in only 8 of the 21 patients with acute subdural hemorrhage on the first scan could the injury be accurately

pinpointed; 5 of the 8 died acutely of their injuries. Of the 3 remaining infants, the acute subdural hemorrhage eventually disappeared in two, and evolved into a chronic subdural collection on the fourth scan performed 12 days after the reporting of injury in the remaining infant.

One case deserves special mention. This 1-month-old infant, in whom the exact time of the injury could not be determined, allegedly was found by her mother on the morning of admission with a right hip deformity but without any history of trauma. An ambulance was summoned at 13.00 h, and the child was taken to the hospital. Retinal hemorrhages were noted on admission, and the initial CT scan performed at 15.45 h (2 h and 45 min after the initial call for an ambulance) showed only acute interhemispheric blood and a small amount of extra-axial fluid which was interpreted as being within normal limits for a 1-month-old infant (fig. 4a). A CT scan performed the next morning at 08.23 h (approximately 19.5 h after the ambulance call and less than 17 h after the initial scan) showed new bilateral frontal and anterior temporal, low density subdural collections (fig. 4b) reminiscent of 'chronic' subdural hematomas.

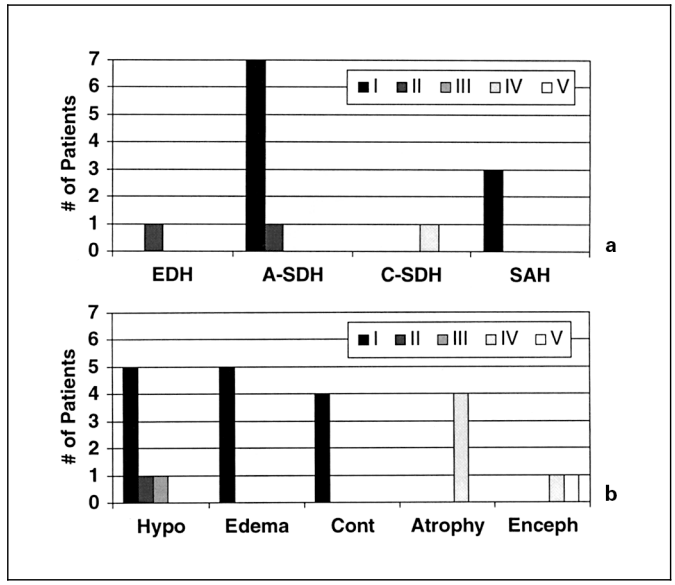

Fig. 3. Distribution and frequency of intracranial abnormalities among 9 infants with ISIS in whom the time of alleged abuse could be pinpointed; excludes the 6 infants in whom chronic subdural hemorrhage or atrophy was seen on the first scan. **a** Extra-axial abnormalities. **b** Intraparenchymal abnormalities. For abbreviations, see legend for figure 1.

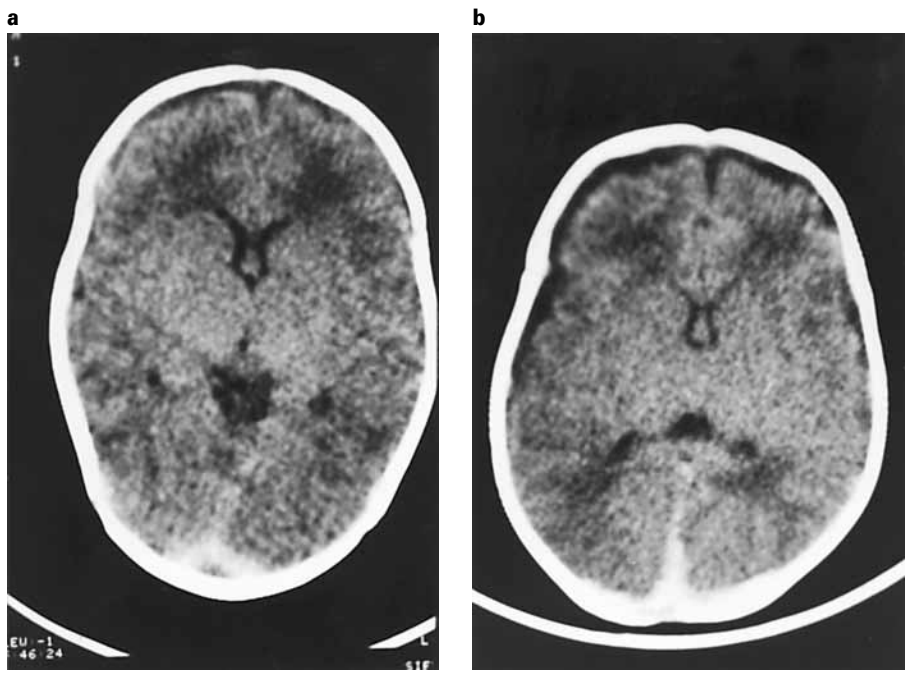

Fig. 4. Appearance of a 'chronic' subdural hematoma in an infant with ISIS first appearing 20 h after the alleged abuse. **a** CT scan performed 2.75 h after the reporting of the injury shows acute interhemispheric subdural hemorrhage, but no subdural blood over the convexities. Although there is a scant amount of low density extra-axial fluid over the frontal lobes which could, in retrospect, be interpreted as subdural blood, this was considered (by 4 independent reviewers examining the scan without knowing the results of the second scan) to be within normal limits for a 1-month-old child. **b** CT scan performed approximately 17 h later (19.75 h after the reporting of the injury) shows low density extra-axial collections overlying the right parietal and both frontal lobes. The collection extended over both temporal lobes as well (not shown). The interhemispheric blood is still visible posteriorly.

Discussion

The timing of changes in the clinical and radiographic appearance of various injuries following child abuse is critical from a medicolegal viewpoint, as it provides a legal framework for establishing the time of the alleged abuse. Moreover, these changes can sometimes identify a likely suspect, since the child may have been with only a single caregiver during the period in question. Although much has been written about the evolution of bone fractures and bruises in child abuse, comparatively little is known about serial changes on cranial imaging studies following the infant shaken impact syndrome.

Feldman et al. [8] studied 34 infants with ISIS, all having initial CT scans and 22 having additional scans. Seven (21%) of the 34 initial scans showed no intracranial abnormalities. In 4 of these infants, no further CT scans were performed, whereas in 3 infants, additional scans performed 1.5–5 days later showed evolving subdural hemorrhage, subdural and subarachnoid hemorrhage, and infarction respectively. The remaining 23 infants had abnormal initial scans; the evolution of their abnormalities on subsequent scans is not detailed.

Zimmerman et al. [25] reported the radiographic findings on the initial CT scan performed in 25 infants with ISIS; follow-up scans were evaluated in 18 (69%). Acute subdural hemorrhage was present on the initial scan in 17 (65%); of these, 15 (88%) were located in the interhemispheric fissure. Parenchymal abnormalities (contusion, hemorrhage, swelling, or infarction) were present in 18 (69%). Atrophy was reported in all patients on follow-up scans.

Sinal and Bell [22] studied 24 infants with ISIS, 22 of whom had initial CT scans. Only 3 of the initial CT scans were normal; chronic subdural collections were described in 4, and parenchymal hypodensities in 5 infants.

Giangiacomo et al. [9] studied 5 infants with serial scans. All had abnormal initial scans, 2 with acute and 2 with chronic subdural hemorrhage, and the fifth with parenchymal hypodensities. Subsequent CT scans showed progressive chronic subdural collections, new subdural hemorrhage, and progressive atrophy. However, 3 of the 5 patients were discharged between the first and subsequent scans, and repeated abuse was likely present.

In the most comprehensive study to date, Cohen et al. [4] examined CT scans in 37 infants with ISIS. Skull fractures were present on the initial scans in 11 infants (30%). Acute subdural hemorrhages were present in 5 infants, chronic subdural collections in 3, and mixed collections in 1. Twenty-seven infants (73%) had subarachnoid hemorrhage. Twenty-four infants (65%) had cerebral edema (18 focal and 6 diffuse); 3 had the reversal sign [10] in which the deep cerebral nuclei and cerebellum are more dense than the overlying cerebral hemispheres on CT scans. Follow-up examinations were performed in 24 patients between 2 weeks and 'several years' following the injury, and showed generalized atrophy in 6 patients, focal encephalomalacia in 17, and chronic subdural collections in 3.

In the aggregate, these studies suggest that both acute subdural hemorrhage and intracranial parenchymal injuries may be present on initial radiographic studies following ISIS. They suggest that chronic subdural collections may also be present on initial scans, which could be interpreted to mean the possibility of prior or repeated abuse. Unfortunately, in none of these studies was the time interval between the alleged injury and any of the radiological studies reported, nor were the follow-up scans examined in any systematic fashion. Therefore, the available literature does not allow an accurate determination as to when various radiographic abnormalities first become apparent.

Unfortunately, the paucity of data has not deterred many people from making rather dogmatic statements about the timing of intracranial radiographic findings in ISIS. For example, parenchymal hypodensities are said to arise only after at least 6–48 h have elapsed since the injury [26, 30], and chronic subdural collections are said to take 1–4 weeks to appear [5, 8, 12, 22, 30]. However, these statements are largely based upon observations in other conditions. In particular, the evolution of parenchymal hypodensities in ISIS is *presumed* to follow the time course of hypodensities after ischemic-anoxic injuries [4, 11, 26], and the evolution of acute to chronic subdural hematomas in ISIS is *presumed* to follow the time course of chronic subdural hematomas in elderly adults or animal models [8, 14, 26, 32].

This is the first study to systematically examine the timing of intracranial changes on serial radiographic studies in infants with ISIS. The Children's Hospital of Buffalo receives virtually all serious head injuries in young children from a geographic population of approximately 1.2 million people in an eight county region of Western New York. The present study likely includes most infants with ISIS treated in the region during this time (although it is possible that a few infants with minor injuries treated elsewhere, or infants with immediately fatal injuries, may have been excluded). The demographics of our study population, in terms of the gender of the infant, reported mechanism of injury, pattern of intracranial injury, and mortality rate, are similar to those of previous reports [15, 18, 25, 33, 34].

There are a number of limitations of such a study which must be made abundantly clear at the outset. First, this is a retrospective study and suffers from the potential selection bias of all such studies. Second, the history provided in cases of child abuse (particularly as given by the perpetrator, who is most often the only 'witness' to the injury) may not be accurate for a number of obvious reasons. Third, this study documents the time of *reporting* of the injury, which may be later than the time of the injury itself, particularly since the perpetrator may initially delay seeking medical care in the hope that the child may spontaneously improve [20]. However, Johnson et al. [35] suggest that the catastrophic and ictal clinical changes, including apnea, that we saw in many of the patients in this study are probably reliable indicators of the time of injury. Fourth, only 15 of the 33 infants had a single identifiable episode of injury which could be accurately timed, and in 6 of these 15 infants there was evidence (such as chronic subdural collections or atrophy) which suggested prior cranial injury. Even among the remaining 9 patients, 2 had old bruises suggesting prior physical trauma.

In addition, the intracranial injuries in our series may have been present earlier than they were documented, since the abnormalities could have arisen anytime between the injury and the first scan, or between any two subsequent scans. Moreover, the incidence of later abnormalities such as encephalomalacia and atrophy may be underreported since not all patients received later scans (particularly the 4th and 5th scans). Finally, although several studies have demonstrated the superiority of MRI over CT in ISIS [1, 5, 14, 21, 36], only 7 patients in this study underwent MRI scans. In each case, the MRI confirmed the findings on CT scan, and in only 1 case, which demonstrated a gyrus rectus infarct not seen on the CT scan, did the MRI disclose additional findings that were not already apparent on CT. Of course, the small numbers of MRI scans in this study precludes any meaningful interpretation of their value in the evaluation of ISIS.

Our study suggests that acute subdural hemorrhage is the most common intracranial abnormality in ISIS (occurring in 81% of infants), and is nearly always present on the first scan obtained an average of 2.4 h after the injury is reported. Perifalcine and tentorial hemorrhage in both ours and others' studies [12, 22, 25, 27] is more common in ISIS than is convexity hemorrhage. Subarachnoid hemorrhage is also most commonly present on initial scans performed an average of 3.9 h after injury. Subarachnoid hemorrhage was less common in our series (5/33 infants, 15%) compared with Cohen's study (73%); one possible reason for the discrepancy could be differences in the manner of defining subarachnoid and subdural hemorrhage between the two studies. We defined subdural hemorrhage as hyperdense extra-axial collections along the tentorium, adjacent to the falx cerebri, or overlying the cerebral hemispheres without a gyral pattern, and limited our definition of subarachnoid hemorrhage to blood within the basal cisterns, Sylvian fissures, or the cerebral convexities with a gyral pattern. A few of the acute subdural hemorrhages in our series that went on to become chronic or mixed subdural hemorrhages over time clearly developed two compartments – a subdural collection which evolved from the acute blood and which was more dense than cerebrospinal fluid, and a separate underlying, subarachnoid space which was isodense with cerebrospinal fluid (and usually accompanied by cerebral atrophy). We therefore feel justified in calling these subdural collections, although the possibility that these were partially (or even completely) subarachnoid cannot be absolutely excluded.

The evolution of acute to chronic subdural hemorrhage in this study was more difficult to analyze serially. Prior reports have suggested that chronic subdural hemorrhage on MRI scans in infants with ISIS appear approximately 30 days or more after the injury [31]. The majority (69%) of acute subdural hemorrhages among the surviving infants in our study resolved without sequelae; the remaining collections evolved from high density to either mixed or low density between 3 and 12 days following report of the injury. The fact that 13 of these collections occurred in children in whom we could not accurately pinpoint the exact time of the injury may be less relevant given the relatively long interval between presentation and the appearance of the chronic collections.

Perhaps not surprisingly, chronic or mixed subdural collections are also relatively common on initial scans (5 of 15 infants), and suggest the possibility of prior or repeated abuse. In at least 1 of our cases, the 'chronic' subdural collection, while not identified on the initial scan performed within 2.75 h of the reporting of injury, was apparent on the second scan performed 17 h later. Had the first scan not been performed, this child would have been presumed to have had an earlier injury at least 1 week prior. Close examination of the initial scan shows a slight enlargement of the extra-axial spaces (fig. 4a) which could conceivably be interpreted as a tiny subdural collection. However, 4 independent examiners reviewing the initial scan without knowing the findings on the subsequent scan felt that both the amount and character of the extra-axial fluid on the initial scan was within normal limits for a 1-month-old infant. Although we cannot be cer-

tain that the collection was absent on the first scan, the significant change in the extra-axial collection within 17 h is distinctly unusual and cannot be accounted for by a simple lysis of red blood cells within a more acute subdural collection. An alternative possibility is that tears of the arachnoid or the parasagittal arachnoid granulations might allow CSF to leak into the subdural space, mix with the acute subdural blood, and produce mixed or hypodense subdural hemorrhage, or even a subdural hygroma that mimics chronic subdural hemorrhage [31, 37]. One final possibility is that the low-density extra-axial collection may actually represent 'hyperacute' blood [38] although this mechanism is very unlikely in our case.

Taken together, the evolution of acute to chronic subdural collections within 3–12 days in 4 of our cases, the appearance of a hypodense subdural collection within 20 h of reporting of the injury in an additional case, and the relatively frequent appearance of 'chronic' or mixed collections on initial scans in both ours and others' [4, 9, 12, 19, 21, 22] studies, suggests that 'chronic' or mixed subdural collections may not always require 1–4 weeks to develop. Our data suggest that hypodense extra-axial collections may appear on CT scans as early as 20 h, and certainly within 3–12 days following presentation. We suggest that the presence of such 'chronic' subdural fluid collections on initial CT scans be interpreted with caution. MRI may likely provide a better way to time subdural hemorrhage in ISIS [31]; we are currently evaluating prospectively the value of serial MRI scans in this disorder.

Parenchymal abnormalities are also obvious very early after injury. All but one of the infants with parenchymal injuries had some parenchymal changes on the initial scan. Five of 7 hypodensities and all cases of edema and contusions were present on the initial scan performed approximately 3 h after the injury was reported. Even among infants with the 'reversal sign' [10], 50% were evident on the initial scans performed on average 3.5 h after the injury was reported. These data strongly suggest that hypodensities may not take 6–48 h to develop, as has previously been suggested, and instead more commonly develop within a few hours of the reporting of the injury. Not unexpectedly, atrophy and encephalomalacia appear most commonly on later scans performed days after the injury. Predictably, these changes evolve from scan to scan, but are too variable to predict the timing of the injury with any certainty.

Summary and Conclusions

The results of this study should be interpreted with caution, although a few conclusions can be drawn. First and perhaps most importantly, one must be somewhat circumspect about making dogmatic medicolegal statements about the timing of ISIS based solely upon the appearance and timing of abnormalities on cranial imaging studies. Second, the presence of parenchymal hypodensities, or even the reversal sign, on the initial scan does not necessarily suggest that abuse occurred more than 6–24 h prior. Third, the presence of 'chronic' subdural collections on the initial scan may not necessarily imply repeated or prior abuse occurring 1 or more weeks prior, as they may evolve within 20 h, and certainly within 3–12 days of the injury. Further prospective studies are warranted, particularly in cases where the details of the abuse are known with as much certainty as possible, to further clarify the role of serial cranial radiography in ISIS.

References

1 Ball WS: Nonaccidental craniocerebral trauma (child abuse): MR imaging. Radiology 1989; 173:609–610.
2 Bernardi B, Zimmerman RA, Bilaniuk LT: Neuroradiologic evaluation of pediatric craniocerebral trauma. Top Magn Reson Imaging 1993;5:161–173.
3 Carter JE, McCormick AQ: Whiplash shaking syndrome: Retinal hemorrhages and computerized axial tomography of the brain. Child Abuse Negl 1983;7:279–286.
4 Cohen RA, Kaufman RA, Myers PA, Towbin RB: Cranial computed tomography in the abused child with head injury. Am J Radiol 1986;146:97–102.
5 Cox LA: The shaken baby syndrome: Diagnosis using CT and MRI. Radiol Technol 1996;67: 513–520.
6 Ellison PH, Tsai FY, Largent JA: Computed tomography in child abuse and cerebral contusion. Pediatrics 1978;62:151–154.
7 English PC, Grossman H: Radiology and the history of child abuse. Pediatr Ann 1983;12: 870–874.
8 Feldman KW: Evolution of the cranial computed tomography scan in child abuse. Child Abuse Negl 1995;19:307–314.
9 Giangiacomo J, Khan JA, Levine C, Thompson VM: Sequential cranial computed tomography in infants with retinal hemorrhages. Ophthalmology 1988;59:295–299.
10 Han BK, Towbin RB, De Courten-Myers D, McLaurin RL, Ball WS: Reversal sign on CT: Effect of anoxic/ischemic cerebral injury in children. Am J Neuroradiol 1989;10:1191–1198.
11 Harwood-Nash DC: Abuse to the pediatric central nervous system. Am J Neuroradiol 1992; 13:569–575.

12 Hymel KP, Rumack CM, Hay TC, Strain JD, Jenny C: Comparison of intracranial computed tomographic (CT) findings in pediatric abusive and accidental head trauma. Pediatr Radiol 1997;27:743–747.
13 Jaspan T, Narborough G, Punt JAG, Lowe J: Cerebral contusional tears as a marker of child abuse – detection by cranial sonography. Pediatr Radiol 1992;22:237–245.
14 Levin AV, Magnusson AR, Rafto SE, Zimmerman RA: Shaken baby syndrome diagnosed by magnetic resonance imaging. Pediatr Emerg Care 1989;5:181–186.
15 Ludwig S, Warman M: Shaken baby syndrome. A review of 20 cases. Ann Emerg Med 1984;13: 104–107.
16 Luerssen TG, Huang JC, McLone DG, Walker ML, Hahn YS, Eisenberg HM, Humphreys RP, Choux M: Retinal hemorrhages, seizures, and intracranial hemorrhages: Relationships and outcomes in children suffering traumatic brain injury; in Marlin AE (ed): Concepts in Pediatric Neurosurgery. Basel, Karger, 1991, vol 11, pp 87–94.
17 Merten DF, Osborne DRS: Craniocerebral trauma in the child abuse syndrome. Pediatr Ann 1983;12:882–887.
18 Merten DF, Radkowski MA, Leonodis JC: The abused child: A radiological reappraisal. Radiology 1983;146:377–381.
19 Merten DF, Osborne DRS, Radkowski MA, Leonidas JO: Craniocerebral trauma in the child abuse syndrome: Radiological observations. Pediatr Radiol 1984;14:272–277.
20 Neglect AAoPCoCAa: Shaken baby syndrome: Inflicted cerebral trauma. Pediatrics 1993;92: 872–875.
21 Sato Y, Yuh WTC, Smith WL, Alexander RC, Kao SCS, Ellerbroek CJ: Head injury in child abuse: Evaluation with MR imaging. Radiology 1989;173:653–657.
22 Sinal SH, Ball MR: Head trauma due to child abuse: Serial computerized tomography in diagnosis and management. South Med J 1987; 80:1505–1512.
23 Tsai FY, Zee C-S, Apthorp JS, Dixon GH: Computed tomography in child abuse head trauma. J Comput Tomogr 1980;4:277–286.
24 Zepp F, Brühl K, Zimmer B, Schumacher R: Battered child syndrome: Cerebral ultrasound and CT findings after vigorous shaking. Neuropediatrics 1992;23:188–191.
25 Zimmerman RA, Bilaniuk LT, Bruce D, Schut L, Uzzell B, Goldberg HI: Computed tomography of craniocerebral injury in the abused child. Radiology 1979;130:687–690.
26 Zimmerman RA, Bilaniuk LT: Pediatric head trauma. Neuroimaging Clin N Am 1994;4: 349–366.
27 Zimmerman RA, Bilaniuk LT, Bruce D, Uzzell B, Goldberg HI: Interhemispheric acute subdural hematoma: A computed tomographic manifestation of child abuse by shaking. Neuroradiology 1978;16:39–40.
28 Bruce DA, Zimmerman RA: Shaken impact syndrome. Pediatric Ann 1989;18:482–494.
29 Kleinman PK: Head Trauma; in Kleinman PK (ed): Diagnostic Imaging of Child Abuse. Baltimore, Williams & Wilkins, 1987, pp 159–199.
30 Brown JK, Minns RA: Non-accidental head injury, with particular reference to whiplash shaking injury and medico-legal aspects. Dev Med Child Neurol 1993;35:849–869.
31 Fobben ES, Grossman RI, Atlas SW, Hackney DB, Goldberg HI, Zimmerman RA, Bilaniuk LT: MR characteristics of subdural hematomas and hygromas at 1.5 T. Am J Neuroradiol 1989;10:687–693.
32 Barkovich AJ: Destructive brain disorders of childhood; in Barkovich AJ (ed): Pediatric Neuroimaging, ed 2. New York, Raven Press, 1995, pp 107–176.
33 Hahn YS, Raimondi AJ, McLone DG, Yamanouchi Y: Traumatic mechanisms of head injury in child abuse. Childs Brain 1983;10:229–241.
34 Starling SP, Holden JR, Jenny C: Abusive head trauma: The relationship of perpetrators to their victims. Pediatrics 1995;95:259–262.
35 Johnson DL, Boal D, Baule R: Role of apnea in nonaccidental head injury. Pediatr Neurosurg 1995;23:305–310.
36 Alexander RC, Schor DP, Smith WL: Magnetic resonance imaging of intracranial injuries from child abuse. J Pediatr 1985;109:975–979.
37 Aronyk KE: Post-traumatic hematomas; in Cheek WR (ed): Pediatric Neurosurgery: Surgery of the Developing Nervous System. Philadelphia, Saunders, 1994, pp 279–296.
38 Sargent S, Kennedy JG, Kaplan JA: 'Hyperacute' subdural hematoma: CT mimic of recurrent episodes of bleeding in the setting of child abuse. J Forensic Sci 1996;41:314–316.