

Confessed abuse versus witnessed accidents in infants: comparison of clinical, radiological, and ophthalmological data in corroborated cases

Matthieu Vinchon · Sabine de Foort-Dhellemmes ·
Marie Desurmont · Isabelle Delestret

Received: 30 October 2009 / Published online: 28 November 2009
© Springer-Verlag 2009

Abstract

Background The diagnosis of inflicted head injury (IHI) or accidental trauma (AT) in infants is based on clinical, radiological, and/or ophthalmological findings such as subdural hematoma (SDH), encephalopathy, retinal hemorrhage (RH), and signs of impact. As a consequence, the diagnostic value of these criteria is difficult to assess because of a circularity bias.

Purpose In order to assess the predictive value of these diagnostic criteria avoiding circularity, we studied prospectively corroborated cases of head injuries in infants, comparing IHI with AT.

Material and methods We selected 45 cases of confessed IHI and 39 cases of AT having occurred in public places. Patients were systematically evaluated clinically, with computed tomography scanner and ophthalmological evaluation by a trained ophthalmologist. RH was rated as absent, mild, and severe, according to the depth and extent of the RH.

Results Brain ischemia was found in 26.7% of IHI; the most prominent elements in favor of IHI were SDH, severe

RH, and absence of signs of impact; the predictive diagnostic values of these three features were 0.685, 0.961, and 0.830, respectively; however, only severe RH in the absence of ocular impact was specific of IHI. When all three features were combined, the specificity was 100%, but the sensitivity was only 24.4%.

Conclusion Our study confirms the high diagnostic value of RH, SDH, and signs of impact for the differential diagnosis between AT and IHI. The evaluation of head injuries in infants requires a high level of awareness and thorough and systematic examination by a trained multidisciplinary team.

Keywords Subdural hematoma · Infant · Inflicted head injury · Shaken baby syndrome · Accidental trauma

Introduction

The differential diagnosis between inflicted head injury (IHI) and accidental trauma (AT) in infants is of major legal importance and should be based on medical evidence. However, this evidence is difficult to gather: In IHI, the principal witness is generally the perpetrator and the history of trauma is generally absent or insincere, and AT mostly occurs at home in the absence of independent witness. Several series have dealt with corroborated AT [25, 35, 40] or IHI [1, 30], but the clinical and radiological data were not precisely described, and comparison between AT and IHI is lacking in the medical literature.

The hallmarks of shaken baby syndrome (SBS) are subdural hematomas (SDH), encephalopathy, and retinal hemorrhage (RH) [10, 20]. Since in most series the diagnosis of IHI is based on the presence of these features, evaluation of the diagnostic value of these criteria is flawed

M. Vinchon · I. Delestret
Department of Pediatric Neurosurgery, Lille University Hospital,
Lille, France

S. de Foort-Dhellemmes
Department of Neuroophthalmology, Lille University Hospital,
Lille, France

M. Desurmont
Department of Forensic Medicine, Lille University Hospital,
Lille, France

M. Vinchon (✉)
Service de Neurochirurgie Pédiatrique, CHRU de Lille,
59 037 Lille Cedex, France
e-mail: m-vinchon@chru-lille.fr

by a bias of circularity [3, 8, 20, 29, 33, 39]. Other biases relate to the age range of the patients: In series mixing infants with older children, IHI and SDH were more common in infants while AT and impact-related lesions were prevalent in juvenile [7, 27]. In previous studies limited to head injuries in infants, we found that the pathophysiology of the SDH was age specific rather than cause specific [35, 38]. As a result, the real diagnostic value of SDH for the diagnosis of SBS is debated. Evaluation of the diagnostic value of RH is also problematic; RH have been reported in rare cases of AT; however, fundoscopy is performed mainly in cases of suspected abuse [2, 8, 15]. In addition, many studies give scanty details regarding RH; however, the latter one is of paramount importance for the diagnosis of IHI [20].

In order to provide evidence-based characterization of clinical, ophthalmological, and radiological lesions found in IHI and AT, we studied cases with undisputed AT or IHI from our prospective database of head injuries in infants. Feldman et al. earlier proposed to classify infantile head traumas along a spectrum, from certain abuse (confessed) to certain accident (witnessed), through highly probable abuse, probable abuse, undetermined, probable accident, and highly probable accident [8]. We selected both extremes of this spectrum in order to compare the clinical, ophthalmological, and radiological features of IHI and AT and their outcome. The purposes of our study were to provide reliable elements for the differential diagnosis between AT and IHI and to test the diagnostic value of these features.

Materials and methods

We started in 2001 a prospective register collecting medical information on all traumatic head injuries in infants. All children under the age of 24 months referred alive to the emergency room, the pediatric intensive care unit (ICU), or our neurosurgical department were included. Our institution is the sole facility for the management of pediatric neurosurgical cases for a four million population area. The methodology and the first 150 cases of this series were published earlier [38]. We selected cases of corroborated IHI and AT; cases of obstetrical trauma were excluded. Corroborated AT was defined as an accident having occurred in a public space in front of independent witnesses. Corroborated IHI was defined as abuse confessed by the perpetrator; information on the confession was obtained by a forensic pediatrician (MD) from judicial sources during expertise or after the judicial hearings were made public.

Information was gathered regarding the mechanism of trauma, and the presence of risk factors such as perinatal

illness (diseases affecting the prenatal period, traumatic or premature birth, neonatal diseases requiring hospitalization), presence of a step parent, and familial dysfunction (history of violence, substance abuse, neglect, and/or psychiatric disorder). The delay from trauma to admission, first computed tomography (CT) scan, and occurrence of intracranial hypertension was calculated based on the time of trauma. The timing of AT is generally precise and unequivocal. Regarding IHI, precise timing is considered less reliable; because several reports have established that symptoms of IHI generally occur immediately after assault [1, 30, 31], we used the time of clinical onset of symptoms reported on admission as an approximation for the time of abuse.

Clinical findings, including neurological symptoms, signs of impact, peripheral lesions, and life-threatening events were collected. All patients had at least one head CT scan. Radiological data included the delay of the CT relative to clinical onset, the presence of skull fracture, extradural, meningeal bleeding, the presence of hyperdense and/or hypodense lesions, ischemic lesions, and the maximal thickness of pericerebral collections on axial view. For statistical analysis, scalp swelling, skull fracture, extradural hematoma, and focal brain contusion were pooled together and labeled as “radiological signs of impact”.

Fundoscopy was performed systematically by an ophthalmologist for all cases of trauma, and in case of RH, repeated controls and seriated pictures were made by a trained neuro-ophthalmologist. The RH were rated as reported earlier by Vinchon [37] and illustrated in Fig. 1. RH were either absent, mild (preretinal: superficial, splinter, or flame-shaped; or intraretinal, dot, or blot-shaped), moderate (preretinal, pearl-shaped with a diameter less than twice the papilla), or severe (large preretinal, associated with other types, diffuse with or without retinoschisis).

The evaluation of cases suspect of IHI also included a complete clinical examination by a pediatrician (ID), extensive biological evaluation to rule out clotting deficiency, and a skeletal survey using X-rays and/or isotopic bone scan. Whenever the possibility of child abuse or neglect arose, the multidisciplinary team (neurosurgeon, pediatrician, forensic doctor, psychologist, and social worker) met and discussed whether the case should be referred to justice, to social network follow-up, or not be regarded as suspicious. Following the French legislation, cases reported to the judiciary were then investigated by the city attorney with hearing of all adults involved in the child's care at the time of trauma.

The clinical outcome was evaluated using the Glasgow Outcome Scale (GOS), with 1—meaning normal life, 2—mild sequels, 3—severe sequels, 4—vegetative state, and 5—dead. Whenever a child with SDH presented with



Fig. 1 Grading of RH in infants. RH were either absent (not illustrated), mild (preretinal: superficial, splinter, or flame-shaped; or intraretinal, dot or blot-shaped; **a**) moderate (preretinal, pearl-shaped

with a diameter less than twice the papilla; **b**), or severe (large preretinal, associated with other types, diffuse with or without retinoschisis; **c**)

intracranial hypertension not relieved by one or more subdural puncture, a subduroperitoneal shunt was inserted following a protocol described previously [34]. We calculated the delay to onset of intracranial hypertension as the time between trauma and either the first subdural puncture or subdural drainage.

Statistics

Continuous variables were tested using Student's *t* test; binary variables were tested using the chi-square test. For the variables relevant to the differential diagnosis between AT and IHI, the positive and negative predictive values were calculated and tested using the chi-square test for significance.

Results

From May 2001 to February 2009, we collected 412 cases of head injuries caused by AT or IHI in infants; 124 (30.4%) were considered IHI, and 288 (69.8%) were considered AT. Among these 412, 39 AT (13.5) and 45 IHI (36.3%) were corroborated. These 84 patients constitute the clinical material of the present study.

The mean age was significantly younger in the IHI group. A male predominance was found in both groups: The male/female ratio was 1.81 in the IHI group, compared with 1.44 in the AT group; the difference was not significant. Perinatal illness was more common in IHI, although not significantly; family dysfunction was significantly more prevalent in IHI. The mechanism of trauma in the IHI group was SBS in 30 cases and beaten baby syndrome in 15. The AT group breaks down as follows: passenger in a car (19), defenestration (five), baby in a carriage hit by a vehicle (three), fall from arms (three), from a supermarket trolley (two), on a sidewalk (two), from a

hospital bed (two), from a seat (one), and with a walker in stairs (one). The delay from trauma to admission was shorter in the AT group but without reaching statistical significance. The delay between trauma and initial CT scanner, however, was significantly shorter in the AT group. Patients with IHI presented significantly more often with seizures, coma, raised intracranial pressure, and neurological deficit. Scalp swelling was found in a minority of children with IHI; a notable exception was fatal IHI, seven of the ten children who died of IHI having scalp swelling, and four of these having skull fracture; the fifth case of IHI with skull fracture survived with major sequelae. AT was associated in most cases with swelling of the scalp. Systemic failure considered life threatening was also more common in AT, without reaching statistical significance, while peripheral fractures were significantly more common in IHI. The constitution of the series, medical past history, clinical presentation, and delay to presentation and to first CT scanner are summarized in Table 1.

SDH was the main diagnosis in the majority of children with IHI but was also found in a large minority of AT. By contrast, impact-related lesions (linear fractures, depressed fractures, extradural hematomas, and focal brain contusion) were found almost exclusively in AT; the difference was highly statistically significant. All cases of trivial fall (from arms or from hospital beds) resulted in minor lesions (skull fractures in two, mild subarachnoid hemorrhage in three), with spontaneous and quick recovery. The main diagnoses in AT and IHI are compared in Fig. 2. SDH was present on CT scanner in a majority of patients but significantly more often in IHI. The density of the lesions was mixed (hypo- and hyperdense) in the majority of patients in both groups, significantly more often in IHI. Brain edema/ischemic brain damage was almost specific of IHI. Fundoscopic data were available in 44 cases of IHI (97.8%) and 35 cases of AT (89.8%), respectively. While severe RH were found in the

Table 1 Clinical presentation in the IHI and AT groups

	Inflicted	Accident	<i>p</i>
<i>N</i>	45	39	
M/F	29/16 (1.81)	23/16 (1.44)	0.24
Perinatal illness	22 (48.9%)	11 (28.2%)	0.055
Socio-psy	20 (44.4%)	2 (5.1%)	<0.001
Stepparent	5	1	NS
Age (months)			
Mean	3.8	8.1	<0.001
Median	3.2	5.5	
Extremes	0.8–18.3	0–23.9	
Delay to referral (hours)			
Mean	57.0	21.2	0.09
Median	12.0	3.5	
Extremes	0–646	0–396	
Delay to CT (days)			
Mean	2.3	0.5	0.026
Median	0.8	0.1	
Extremes	0–27	0–3.7	
Clinical features			
Seizures	31 (68.9%)	5 (12.8%)	<0.001
Somnolence	18 (60.0%)	12 (30.8%)	0.38
Coma	20 (44.4%)	9 (23.1%)	0.04
Deficit	20 (44.4%)	8 (20.5%)	0.02
Raised intracranial pressure	30 (66.7%)	9 (23.1%)	<0.001
Swelling	1 (2.2%)	30 (76.9%)	<0.001
Impact on head	17 (37.8%)	34 (87.2%)	<0.001
Other impact	11 (24.4%)	6 (15.4%)	0.30
Peripheral fracture	12 (26.7%)	2 (5.1%)	0.008
Transfusion	10 (22.2%)	10 (25.6%)	0.71
Life threat	3 (6.7%)	8 (20.5%)	0.060

majority of cases of IHI (56.8%), RH were found in only six cases of AT: mild, flame-shaped in five, and severe in only one patient, who had facial trauma suggesting impact on the globe. Overall, severe RH in the absence of direct

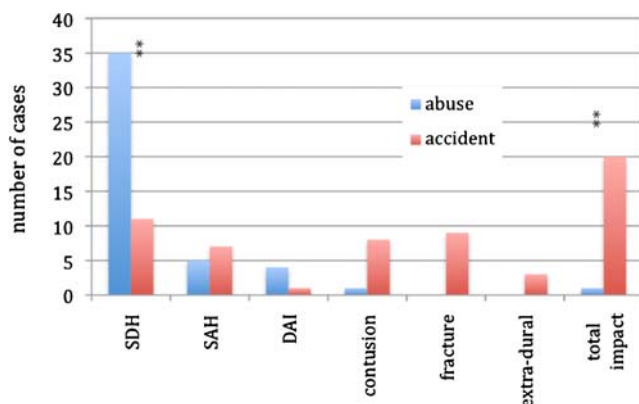


Fig. 2 Main diagnosis in the AT and IHI groups. The “total impact” subgroup represents the sum of cases with contusion, fracture, or extradural hematomas. ** $p < 0.01$

impact appeared specific of IHI. The proportions of the different grades of RH in AT and in IHI are compared in Fig. 3. Radiological and ophthalmological features are summarized in Table 2.

Thirty-two patients developed a SDH requiring drainage, significantly more often in IHI than in AT; the delay between trauma and intracranial hypertension was longer in AT, but the difference was not significant. The mean duration of assisted ventilation and overall stay in ICU was longer in IHI; the overall hospital stay was also significantly longer in IHI, which was caused at least in part by the legal procedure. Twelve children died as a consequence of the trauma, significantly more in IHI (ten cases, 22.2%) than in AT (two cases, 5.1%). Eleven of the 12 children who eventually died were comatose on presentation. Both fatal AT were high-velocity traffic accidents with systemic failure. At last control, the number of patients with normal overall status (GOS 1) was significantly higher in AT (Fig. 4). Regarding neurological sequels, only epilepsy was significantly more common in

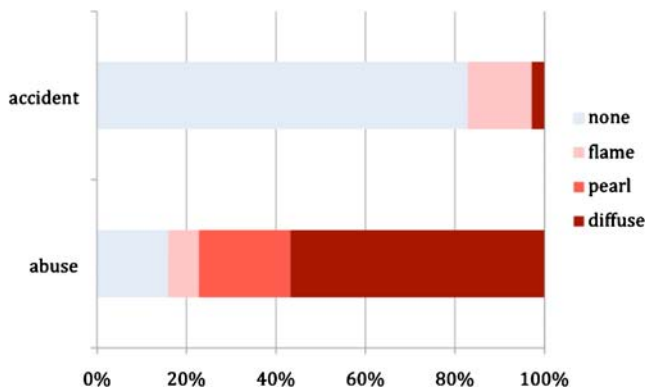


Fig. 3 Retinal hemorrhage in the AT and IHI groups. Although most cases of abuse were associated with severe hemorrhage, seven had no hemorrhage, and three had only mild hemorrhages. Conversely, mild RH were found in five cases of AT, and one had severe hemorrhage caused by direct facial impact. We conclude that severe retinal hemorrhage is pathognomonic of IHI but admits a number of false negatives

IHI. However, the mean age at last control was less than 2 years in 55 patients, and we assume that with a longer follow-up, the differences between the two groups will become more pronounced. Data on immediate and subsequent outcome, duration of hospital stay, occurrence of raised ICP, and requirement for subduroperitoneal drainage are summarized in Table 3.

The sensitivity, specificity, positive predictive value, and negative predictive value of brain ischemia, SDH, severe retinal hemorrhage, and signs of impact, relative to the diagnosis of IHI, are summarized in Table 4. Comparison of

Table 2 Radiological and ophthalmological findings in the IHI and AT groups

	Inflicted	Accident	<i>p</i>
CT findings			
Subdural collection	37 (82.2%)	17 (43.6%)	<0.001
Mixed density image	39 (90.7%)	17 (53.1%)	<0.001
Brain ischemia	12 (26.7%)	1 (3.2%)	0.0023
Extradural	1 (2.4%)	5 (13.9%)	NS
Contusion	7 (15.6%)	10 (25.6%)	0.25
Fracture	5 (11.4%)	26 (66.7%)	<0.001
Total impact	10 (22.2%)	26 (66.7%)	<0.001
Retinal hemorrhage			
None	7 (15.9%)	29 (82.9%)	<0.001
Mild	3 (6.8%)	5 (14.3%) ^a	NS
Moderate	9 (20.5%)	0	NS
Severe	25 (56.8%)	1 (impact)	<0.001

^a Percents represent the ratio of the number of features on the total number of available data; data may be missing, producing different percents for the same absolute value

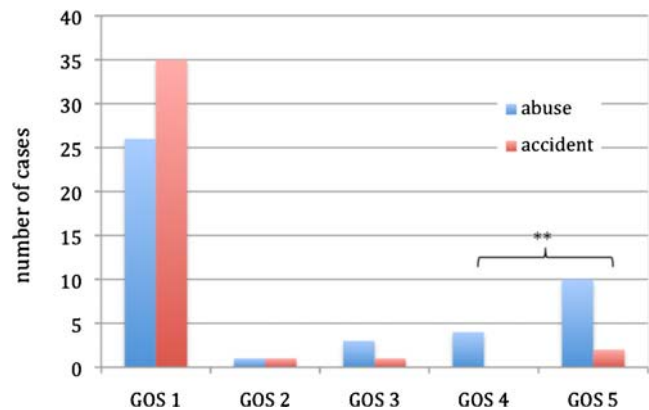


Fig. 4 Clinical outcome in the AT and IHI groups, according to the Glasgow Outcome Score (1 normal life, 5 dead). The number of cases with severe morbidity (GOS 4) or mortality was very significantly higher in the IHI group (** $p=0.002$)

the corroborated series with uncorroborated cases shows that fatal IHI was probably overrepresented in the corroborated group (ten of 45 versus three of 77 in the uncorroborated group, NS). The majority of cases of corroborated AT were traffic accidents, generally more violent than trivial household accidents; however, three cases of uncorroborated trauma were fatal (two falls with walker, one fall in the stairs without walker).

Table 3 Early and later evolution in the IHI and AT groups

	Inflicted	Accident	<i>p</i>
Delay to raised ICP (days)			
Mean	4.9	7.0	0.441
Median	1.6	4.5	
Subdural drainage	25	7	<0.001
Mean duration (days)			
Ventilated	3.3	1.4	0.056
In ICU	4.3	1.9	0.042
In hospital	16.1	9.0	0.0002
Last control			
Follow-up (months)	17.3	19.0	0.76
Age (months)	20.9	27.1	0.23
Developmental delay	8 (23.5%)	2 (5.4%)	NS
Motor delay	9 (26.5%)	4 (10.8%)	0.218
Deficit	7 (20.6%)	2 (5.4%)	NS
Epilepsy	10 (29.4%)	1 (2.7%)	0.008
Behavior	5 (14.7%)	1 (2.7%)	NS
Vision	9 (26.5%)	3 (8.1%)	0.108
GOS 1	26 (57.8%)	35 (89.7%)	0.001
GOS 2	1	1	NS
GOS 3	3	1	NS
GOS 4	4	0	NS
GOS 5	10 (22.2%)	2 (5.1%)	0.026

Table 4 Diagnostic value of brain ischemia, subdural hematoma, retinal hemorrhage, and absence of signs of impact, for the diagnosis of child abuse

	Sensitivity	Specificity	Positive predictive value	Negative predictive value	Chi-square (<i>p</i>)
Brain ischemia	0.267	0.971	0.921	0.505	<0.01
SDH	0.822	0.552	0.685	0.724	<0.001
Severe RH	0.556	0.974	0.961	0.655	<0.001
Absence of scalp swelling	0.978	0.769	0.830	0.968	<0.001
SDH and diffuse RH and no scalp swelling	0.244	1	1	0.534	<0.001

Note that the absence of signs of impact has roughly the same diagnostic value for IHI as has severe retinal hemorrhage. These figures show that the sensitivity, specificity, and positive and negative predictive values of these three features are uneven but that the specificity and positive predictive value of the three combined was 100% (at the cost of a loss of sensitivity)

Discussion

The diagnosis of IHI is based on clinical, radiological, and ophthalmological criteria. The differential diagnosis between AT and IHI is a frequent dilemma in medical and legal practice: “the dire consequences of either false-positive or false-negative diagnosis intensify the need to establish accurate diagnostic criteria” [27]. The aim of our study was to evaluate the diagnostic value of these criteria. It was therefore necessary to dispose of another diagnostic criterion as a gold standard in order to avoid circularity; we chose to select cases of witnessed AT and confessed IHI.

Reliability of data

Our study was intended to select data as reliable as possible. We considered that accidents occurring in public spaces or confession of abuse by the perpetrator give the best possible assurances for the diagnosis of AT and IHI, respectively.

Details of abuse as reported by abusers may be unreliable, however, because they seek to underestimate the violence of abuse and the repetition of trauma and often lack precision in timing [19, 29]. Regarding the notion of abuse itself, we think the information on confessions obtained from the judiciary can be trusted. In IHI, the absence of immediate, spontaneous confession, the longer delay to referral, and the context of guilt confirm that shaking was abusive, not playful. We have limited data regarding details of the confession; however, the dating generally appeared consistent with the time of clinical onset as initially reported when the child was admitted. We have few data on the perpetrator and the mechanism of abuse, as detailed in other studies [1, 30, 31]; instead, our focus was on the clinical, radiological, and ophthalmological data in IHI versus AT.

Accidental traumas occurring in private spaces cannot escape a measure of doubt over possible concealed abuse [40]. In a nonverbal child, everything depends on the value

of the witnesses, who should be independent and reliable: In the cases reported by Gardner and by Piatt, the witnesses (the 5-year-old brother of the victim and the parents of the victim, respectively) cannot be regarded as such [9, 23]. On the contrary, traumas occurring in public spaces are generally seen by independent witnesses; traffic accidents are investigated by the police and insurance and considered to be definitely accidental [27]. Exceptional trivial AT occurring in hospital (fall from a bed) also belong to the corroborated category [22], as do exceptional cases caught on video [25].

Analyzing data in categories defined by these same data represents a bias of circularity. In many series studied by radiologists, ophthalmologists, pathologists, and examiners, the criterion for IHI was that the cases had been declared abusive by a team of clinicians [10, 12, 20, 33, 39]. These authors ignored the fact that the IHI team had based their diagnosis on the same data, which are the subject of their study. In studies conducted by clinicians, the criterion for IHI is generally that the lesions are considered not explained by the mechanism of the alleged trauma [3, 5, 8, 16, 38]. The basis on which these lesions are considered incompatible with the trauma may be subjective, and these studies are thus not immune of circularity either [29]. In some studies, a laudable effort was made to avoid the circularity bias, by artificially setting aside RH from the criteria of IHI [8, 24]. This method appears unrealistic in retrospective studies and could be dangerous in prospective studies by underestimating IHI. How can the circularity ring be broken? We think that our study, with prospective inclusion of all cases of head injuries, systematic ophthalmological evaluation, and selection of cases based on nonmedical data, allows the evaluation of the medical data in IHI and AT with reasonable certainty, even though the selection of corroborated cases introduces other biases.

Selection bias derives from the pattern of accrual of basically any study. In autopsy series, the trauma is a priori more violent than in survivors, and more severe lesions should be expected. The high prevalence of impact lesions

in SBS (the shaken impact theory) [4] or of brain ischemia [10] could thus be an overestimation due to a selection bias in autopsy series. Our study does not escape a selection bias, as attests the higher mortality rate in corroborated compared to noncorroborated IHI. Also, fatal AT in corroborated and noncorroborated cases resulted from different mechanisms, with different lesion patterns. The higher mortality and morbidity in IHI, compared to AT, can likewise be artificially raised by a selection bias. In the literature, the mortality rate in IHI varied between 27% and 61% according to series [18], which suggests that the threshold for the diagnosis of IHI was highly variable. What raises concern, however, is that if a trauma is all the more suspect of abuse that its consequences are dire, conversely, mild cases of IHI are likely underdiagnosed. Cases of apparently isolated SDH have been eventually diagnosed as IHI after a thorough investigation [21]. Undiagnosed IHI will possibly recur, and several studies have shown that recurrent IHI bears a poor prognosis [13, 14, 34]. This emphasizes the need for a high level of awareness for IHI in all cases of head injuries in infant and for an adequate and thorough evaluation.

The age of included patients introduces another bias because the lesion pattern depends on the age of the patients as well as on the mechanism. In series including children of all ages, Ewing–Cobbs noted that the age was different in the AT and IHI groups [6], which muddles the evaluation of the different traumatic patterns in AT and IHI. Although the present study included only infants (less than 24 months), the mean age was still significantly lower in IHI, which may account partly for a higher proportion of SDH in this group. However, the median age was only 5.5 months in the AT group, which corresponds to the peak prevalence of SDH [34]. Our opinion is that the lesser prevalence of SDH in the AT group relates to the higher proportion of patients with purely extradural, impact-related lesions, rather than to the age bias.

Differential diagnosis between AT and IHI

The diagnosis of SBS is classically based on the triad—SDH, RH, and encephalopathy [11, 22, 29]—while other authors refer to another triad: SDH, RH, and absence of signs of impact [26]. Overall, our study confirms the diagnostic value of all of these latter features for the diagnosis of IHI.

In our study, the clinical manifestations of encephalopathy were often minimal, and brain ischemia was detected on CT scanner in only 26.7% of patients with IHI. Magnetic resonance imaging has a much higher yield than CT for brain lesions but has some disadvantages (impractical in young children, low sensitivity for blood in early studies), and we do not use it routinely for mild cases [36]. CT

scanner is the most commonly used diagnostic tool for head injuries in infants [33]. We think that encephalopathy is more a marker of severity than a mainstay of the diagnosis of IHI.

Impact lesions are prominent in AT, in particular traffic accidents, characterized by extensive lesions of the skull and its covering, which may be life threatening on account of blood loss and coagulopathy [32]. In case of SDH, the presence of signs of impact are considered important diagnostic features in favor of AT [28, 35]. Since a negative finding is of no value unless the absent feature is systematically and prospectively recorded, we looked systematically for clinical and/or radiological signs of impact in all cases of infantile head trauma. In a previous study, we found signs of impact in a minority of cases of IHI [38]. It appears from the present study that this negative finding was a very significant element of the differential diagnosis between AT and IHI, with the same diagnostic value as severe RH; when combined with SDH and severe RH, the predictive value regarding IHI was 100%, confirming the value of the “Ontario triad” [26]. These findings are in apparent contradiction with the “shaken impact” theory, which states that shaking alone does not provide the required energy to produce diffuse brain damage [4]. As discussed above, we think that a selection bias may explain the higher incidence of impact lesions in fatal IHI [4, 10]. Reciprocally, IHI has been underdiagnosed in the past, and many minor cases (without impact) may have been overlooked [14]. The prevalence of signs of impact in IHI thus varies between 100% and 33% according to the series [16, 18, 26], reflecting the investigators’ personal opinion, the intensity of the pursuit of evidence of impact, and the zeal for declaring cases to the judiciary. In our series, we found that seven of ten cases of fatal IHI had signs of impact, and four had a skull fracture. We conclude that shaken impact baby syndrome represents a subgroup of IHI with a worse prognosis but that SBS without impact can also be fatal, as shown earlier [1, 18]. Whatsoever, we do not think that the absence of signs of impact lessens in any measure the perpetrator’s responsibility in the child’s illness.

SDH is a cardinal feature in IHI, occurring in the majority of cases [10, 16, 21]. However, subdural collections are also found in infants after AT [35], meningitis, craniotomy, and overdrainage by shunts. Their pathogenesis should be viewed as a common final pathway of many different processes affecting the meninges at an age of special hydrodynamic fragility. The lower prevalence of SDH in AT likely reflects the higher rate of impact-related, purely extradural lesions in this group. SDH alone thus cannot be considered indicative of IHI, but we found that its association with severe RH and absence of signs of impact is virtual certainty of abuse. Classically, SDH in

SBS are mixed-density collections, which has long been considered a sign of rebleeding indicating repeated assault [6]; however, we found that mixed density SDH are also found in AT [35], mixed density indicating sedimentation rather than rebleeding [33]. Our present study confirms these earlier findings.

The high value of RH for the diagnosis of IHI is well established [20, 24]; in corroborated series, RH were noted in 75–100% of cases [18]. In our study, 97.8% of cases of IHI and 89.9% of cases of AT underwent fundoscopy, and RH were noted in 84.1% and 17.1% of these, respectively. The main problems with RH are thus false positives (RH in AT) and false negatives (absence of RH in IHI). In a series of corroborated IHI, Starling et al. found no RH in 18% of SBS [31]. As said above, the value of negative findings depends on how intensely they are looked for. Serial imaging of the retina shows that severe RH can resolve over a period of a week (unpublished data); it may also be reminded that RH is not a feature of the beaten baby syndrome.

RH have also been reported in corroborated AT, like traffic accidents, defenestration, or staircase accident [2, 5, 15, 35]. For these reasons, some authors have stated that the specificity of RH for IHI is “not supported by scientific evidence” [17]. Plunkett also reported RH in four of the six fatal AT who underwent fundoscopy, suggesting that the severity of trauma plays a role [25]; however, it is generally the absence of RH in major AT like traffic accidents that struck the authors.

In previous studies, we found that RH associated with AT were mild, while RH caused by IHI were severe [35, 38]. Other authors have shown that in IHI, the severity of RH was also correlated with the severity of brain damage [20]. In order to enhance the diagnostic value of RH and minimize false positives, it appeared important to establish a grading. We have proposed since 2004 a four-tier grading based on easily indentified features [37, 38]; however, it was difficult to validate the diagnostic value of this grading because of the circularity bias. The present study, basing the diagnosis of IHI on confession rather than medical features, eliminates this bias and confirms the very high predictive value of severe RH. Severe RH were found in the majority of cases of IHI (56.8%); only one case of AT had severe RH, was likely caused by craniofacial trauma, as published before [17]. We conclude that severe RH in the absence of facial trauma are specific of IHI.

Conclusions

The paucity of data gained from animal experiments is still a major handicap to our understanding of the natural history of traumatic intracranial lesions in infants. Advances rely

heavily on clinical data as a major source of information. However interpreting these data is delicate, especially in traumas which are either concealed or occur in private spaces. In order to be exploitable, studies must be calibrated, selective, and prospective. We think data gathered from corroborated cases of IHI and AT avoid the circularity bias and allow comparisons. Our study confirms the diagnostic value of the SDH, severe RH, and absence of signs of impact for the differential diagnosis between IHI and AT. The importance of the differential diagnosis between IHI and AT requires systematic evaluation by a trained multidisciplinary team.

References

1. Biron D, Shelton D (2005) Perpetrator accounts in infant abusive head trauma brought about by a shaking event. *Child Abuse Neglect* 29:1347–1358
2. Christian C, Taylor AA, Hertle RW, Duhaime AC (1999) Retinal hemorrhages caused by accidental household trauma. *J Pediatr* 135:125–127
3. Dashti SR, Decker DD, Razzaq A, Cohen AR (1999) Current patterns of inflicted head injury in children. *Pediatr Neurosurg* 31:302–306
4. Duhaime AC, Gennarelli TA, Thibault LE, Bruce DA, Margulies SS, Wiser R (1987) The shaken baby syndrome. *J Neurosurg* 66:409–415
5. Duhaime AC, Alario AJ, Lewander WJ, Schut L, Sutton LN, Seidl TS, Nudelman S, Budenz D, Hertle R, Tsiaras W, Loporchio S (1992) Head injury in very young children: mechanisms, injury types, and ophthalmologic findings in 100 hospitalized patients younger than 2 years of age. *Pediatrics* 90:179–185
6. Ewing-Cobbs L, Kramer L, Prasad M, Canales DN, Louis PT, Fletcher JM, Vollero H, Landry SH, Cheung K (1998) Neuroimaging, physical, and developmental findings after inflicted and noninflicted traumatic brain injury in young children. *Pediatrics* 102:300–307
7. Ewing-Cobbs L, Prasad M, Kramer L, Louis PT, Baumgartner J, Fletcher JM, Alpert B (2000) Acute neuroradiological findings in young children with inflicted or noninflicted traumatic brain injury. *Child's Nerv Syst* 16:25–34
8. Feldman KW, Bethel R, Shugerman P, Grossman DC, Grady MS, Ellenbogen RC (2001) The cause of infant and toddler subdural hemorrhage: a prospective study. *Pediatrics* 108:636–646
9. Gardner HB (2005) A witnessed short fall mimicking presumed shaken baby syndrome (inflicted childhood neurotrauma). *Pediatr Neurosurg* 43:433–435
10. Geddes JF, Hackshaw AK, Vowles GH, Whitwell HL (2001) Neuropathology of inflicted head injury in children. I. Patterns of brain damage. *Brain* 124:1290–1298
11. Geddes JF, Plunkett J (2004) The evidence base for shaken baby syndrome: we need to question the diagnostic criteria. *Brit Med J* 328:719–720
12. Harwood-Nash DC (1992) Abuse to the pediatric central nervous system. *Am J Neuroradiol* 13:569–575
13. Jayawant S, Rawlinson A, Gibbon F, Price J, Schulte J, Sharples P, Sibert JR, Kemp AM (1998) Subdural haemorrhages in infants: population based study. *Brit Med J* 317:1558–1561
14. Jenny C, Hymel KP, Ritzen A, Reinert SE, Hay TC (1999) Analysis of missed cases of abusive head trauma. *JAMA* 281:621–626

15. Johnson DL, Braun D, Friendly D (1993) Accidental head trauma and retinal hemorrhage. *Neurosurgery* 33:231–234
16. King WJ, MacKay M, Sirnick A (2003) Shaken baby syndrome in Canada: clinical characteristics and outcomes of hospital cases. *Can Med Ass J* 168:155–159
17. Lantz PE, Sinal SH, Stanton CA, Weaver RG (2004) Perimacular retinal folds from childhood head trauma. *Brit Med J* 328:754–756
18. Leestma JE (2005) Case analysis of brain-injured admittedly shaken infants: 54 cases 1969–2001. *Am J Forensic Med Pathol* 26:199–212
19. Leestma JE (2006) “Shaken baby syndrome”: do confessions by alleged perpetrators validate the concept? *J Am Phys Surg* 11:14–16
20. Morad Y, Kim YM, Armstrong DC, Huyer D, Mian M, Levine AV (2002) Correlation between retinal abnormalities and intracranial abnormalities in the shaken baby syndrome. *Am J Ophthalmol* 134:354–359
21. Morris MW, Smith S, Cressman J, Ancheta J (2000) Evaluation of infants with subdural hematoma who lack external evidence of abuse. *Pediatrics* 105:549–552
22. Oehmichen M, Meissner C, Saternus KS (2005) Fall or shaken: traumatic brain injury in children caused by falls or abuse at home—a review on biomechanics and diagnosis. *Neuropediatrics* 36:240–245
23. Piatt JH (1999) A pitfall in the diagnosis of child abuse: external hydrocephalus, subdural hematoma, and retinal hemorrhages. *Neurosurg Focus* 7(4):e4. doi:10.3171/foc.1999.7.4.6
24. Pierre-Kahn V, Roche O, Dureau P, Uteza Y, Renier D, Pierre-Kahn A, Dufier JL (2003) Ophthalmologic findings in suspected child abuse victims with subdural hematomas. *Ophthalmology* 110:1718–1723
25. Plunkett J (2001) Fatal pediatric head injuries caused by short-distance falls. *Am J Forensic Med Pathol* 22:1–12
26. Pollanen MS, Smith CR, Chiasson DA, Cairns JT, Young J (2002) Fatal child abuse-maltreatment syndrome. A retrospective study in Ontario, Canada, 1990–1995. *Forensic Sci Int* 126:101–104
27. Reece RM, Sege R (2000) Childhood head injuries: accidental or inflicted? *Arch Pediatr Adolesc Med* 154:11–15
28. Shah M, Vavilala MS, Feldman KW, Hallam DK (2005) Motor vehicle crash brain injury in infants and toddlers: a suitable model for inflicted head injury? *Child Abuse Negl* 29:953–967
29. Squier W (2008) Shaken baby syndrome: the quest for evidence. *Dev Med Child Neurol* 50:10–14
30. Starling SP, Holden JR, Jenny C (1995) Abusive head trauma: the relationship of perpetrators to their victims. *Pediatrics* 95:259–262
31. Starling SP, Patel S, Burke BL, Sirotak AP, Stronks S, Rosquist P (2004) Analysis of perpetrator admissions to inflicted traumatic brain injury in children. *Arch Pediatr Adolesc Med* 158:454–458
32. Tibbs RE, Haines DE, Parent AD (1998) The child as a projectile. *Anat Rec* 253:167–175
33. Tung GA, Kumar M, Richardson RC, Jenny C, Brown WD (2006) Comparison of accidental and nonaccidental head injury in children on noncontrast computed tomography. *Pediatrics* 118:626–633
34. Vinchon M, Noulé N, Soto-Ares G, Dhellemmes P (2001) Subduroperitoneal drainage for subdural hematomas in infants: results in 244 cases. *J Neurosurg* 95:249–255
35. Vinchon M, Noizet O, Defoort-Dhellemmes S, Soto-Ares G, Dhellemmes P (2002) Infantile subdural hematomas due to traffic accidents. *Pediatr Neurosurg* 37:245–253
36. Vinchon M, Noulé N, Jissendi-Tchofo P, Soto-Ares G, Fourier C, Dhellemmes P (2004) Imaging of head injuries in infants: temporal correlates and implications for the diagnosis of child abuse. *J Neurosurg* 101(1 Suppl):44–52
37. Vinchon M (2004) Traumatic retinal hemorrhages. *Child’s Nerv Syst* 20:279
38. Vinchon M, Defoort-Dhellemmes S, Desurmont M, Dhellemmes P (2005) Accidental and nonaccidental head injuries in infants: a prospective study. *J Neurosurg (Pediatrics)* 4) 102:380–384
39. Wells RG, Vetter C, Laud P (2002) Intracranial hemorrhage in children younger than 3 years. *Arch Pediatr Adolesc Med* 156:252–257
40. Williams RA (1991) Injuries in infants and small children resulting from corroborated free falls. *J Trauma* 31:1350–1352