11 Violence and Abuse: Battered Child

Antonina Argo, Giuseppe Lo Re, Elvira Ventura Spagnolo, Alberto Calandra, Marija Čaplinskiené, Agata Crapanzano, Antonio Pinto, and Sergio Salerno

Child abuse is an important social and medical problem which represents a major cause of morbidity and mortality among children. Battered Child Abuse (BCA) is a comprehensive term to indicate classical features at first described by Ambroise Tardieu (1818–1879), a French forensic pathologist, in 1860 in a series of 32 cases of cruelty to children, resulting to death in 21 cases [1].

In 1946, John Caffey (1895–1978) published his first paper where he described six infants with multiple fractures in the long bones, who additionally had chronic subdural haematoma and no history of injury [2]. He recommended that unexplained fractures of the long bones warranted investigations for subdural haematoma (SDH). In 1962, Kempe and co-workers published their study on 'The battered-child syndrome', first real recognition of child abuse as a disease and of the responsibility physicians held for its diagnosis and prevention [3–5]. Finally, Kempe et al. provided radiographic clues to whether trauma was accidental or non-accidental [6]. Worthy of noting, this article was considered one of the best paediatric research articles in the last 150 years, as Kempe et al. established that physicians have a special responsibility to children—a responsibility to help keep them safe, sometimes even from their own parents [1]. During decades scientific interest as reflected by online search of the MEDLINE database yields relevant data about the development of child maltreatment awareness by health care professionals since the article by Kempe et al. was published in 1962. In 1963, the keyword *child abuse* was added to the MEDLINE system [7]. Following keywords were first assigned to battered-child article by the National Library of Medicine (*wounds and injuries*, *child*, *child welfare*, and *infant*). Twelve articles were categorized under this keyword, in 2006 almost 600 articles were listed in MEDLINE under the keyword *child abuse* and 1989 in 2016. Not surprisingly, the marked increase in knowledge about child maltreatment has led to the development of a new paediatric subspecialty, child abuse paediatrics [8–10]. In 2009, the American Board of Pediatrics will administer the first examination for board certification in this subspecialty, legacy related. A significant result of Kempe's battered-child syndrome article has been the raising of paediatricians [11–13] who are dedicated to diagnosing, treating, and preventing child abuse

A. Argo · E. Ventura Spagnolo
Department for Health Promotion, Maternal and Child Care, University of Palermo, Palermo, Italy
e-mail: antonella.argo@unipa.it

G. Lo Re · A. Calandra · A. Crapanzano · S. Salerno
Department of Radiology, Policlinic Hospital, Palermo, Italy
e-mail: sergio.salerno@unipa.it

M. Čaplinskiené (✉)
State Forensic Medicine Service, Vilnius, Lithuania
e-mail: marija.caplinskiene@vtmt.lt

A. Pinto
Ospedale Caldarelli, Naples, Italy

G. Lo Re et al. (eds.), *Radiology in Forensic Medicine*,
https://doi.org/10.1007/978-3-319-96737-0_11

and neglect. Over the years, diagnosis of child abuse becomes more sophisticated within methods such as biomechanics [14], proteomics [15], biochemistry [16] and genetics [17] also in perspective of forensic molecular approach as epigenetic modifications [18, 19].

The 'child abuse syndrome' (also known as the 'battered baby' or 'non-accidental injury in childhood') is a clinical condition in young children who have received serious physical abuse and is a frequent cause of permanent injury or death. The syndrome should be considered in any child exhibiting evidence of fracture of any bone, subdural haematoma, failure to thrive, soft tissue swellings or skin bruising, in any child who dies suddenly, or where the degree and type of injury is at variance with the history given regarding the occurrence of the trauma [20, 21] (Fig. 11.1). After hundreds of researches and contribution of both clinical and forensic interest in decades, as emphasized (Knight's 2016) the BCA happen when an infant or child suffers repetitive physical injuries inflicted by a parent or guardian, in circumstances that exclude accident.

11.1 Fatal Physical Abuse of Children Epidemiology

Child maltreatment—the physical, sexual, mental abuse and/or neglect of children younger than 18 years—exists in every society. It is common in the WHO European Region [22] and globally, often occurring with other negative experiences (mental illness, drug or alcohol problem, prison, witnessing intimate partner, domestic violence, parental separation). While severe child maltreatment may come to [23] the attention of child protection agencies, more hidden forms that progress over many years also exist [24].

Assessments of child abuse involve the interaction of multiple disciplines, including medicine, social work, law enforcement, and the judicial system [25, 26]. This interdisciplinary approach, which is facilitated by Child Advocacy Centers or similar multidisciplinary models, can be challenging because of differing definitions of child abuse, expectations regarding information that can be determined during the medical evaluation, or interpretations of findings [22, 27].

Fig. 11.1 Clinical and forensic constellation related to Battered child syndrome (authors' observation). Male patient, 6 years old at Emergency Department observation, presenting multiple skin bruises (periorbital and on the back), adult bite mark on genital area

Severe abuse can lead to homicide [28–34]. While homicide rates for children aged under 15 in the Region appear low at about 850 deaths per year, many child deaths are not investigated and the numbers may be much higher [35–37]. National statistics on child abuse in the USA show that in 2013 approximately 679,000 children were victims of maltreatment, and approximately 1520 of them died. Children in the first year of their life had the highest rate of victimization of 23.1 per 1000 children in the national population of the same age. Of the children who experienced maltreatment or abuse, 18% suffered physical abuse. Although figures vary yearly, approximately 700,000 cases of child abuse and neglect are reported annually in the United States, of which 117,772 are physical abuse, as documented by the Department of Health and Human Services in 2015 [23, 24].

Child maltreatment is considered an important public health issue in the European Region. Within data from European Report on Preventing Child Maltreatment, 2013, child maltreatment leads to the premature death of 852 children under 15 years in the European Region every year. In that document it is also properly observed that 'not all deaths from maltreatment are properly recorded and this figure is likely to be an underestimate'. Deaths are the tip of the iceberg, as it is estimated that for every death, there are between 150 and 2400 substantiated cases of physical abuse [38]. The number of children suffering from maltreatment whose plight goes unrecognized is likely to be very much higher and may only come to light through population surveys [39]. Global estimates state that prevalence ranges from 4 to 47% for moderate-to-severe physical abuse, 15 to 48% for emotional and 20% for sexual abuse in girls and 5 to 10% in boys [40], suggesting that tens of millions of children in the Region suffer different forms of maltreatment. Differences also exist within countries and child death rates are several times higher in disadvantaged populations than wealthier communities; this is also true for hospital admissions, with children from deprived neighbourhoods more likely to be admitted for assaults. Deprivation exposes children to more risk factors for abuse: these can grow over time, increasing the likelihood of violence and neglect [41].

Trauma is the most common cause of death in childhood, and inflicted head injury is the most common cause of traumatic death in infancy [40]. The physician may be asked to render a legal opinion as to whether medical findings indicate abuse [42]; many published reports on medical findings indicative of abuse are based on observational data—primarily from case series—and on clinical judgment [43]. In addition to a medical evaluation to guide treatment, findings that do not require therapy but that support an inflicted cause must also be documented [44]. The legal mandate for physicians to report suspected child abuse requires a reasonable suspicion of abuse, which is sometimes a difficult criterion to meet because of uncertainty regarding the diagnosis [45], particularly when the physician is also a paediatrician caretaker of family, possibly the physician may want to be more certain of the diagnosis [46]. Sometimes the history and/or examination findings facilitate a prompt accurate diagnosis of assault, but this is an uncommon scenario [47–50]. More commonly, the suspicion of child abuse arises after a doctor is told an uncommon story about how an injury occurred or the doctor discovers an injury frequently attributed to assault [51–53]. Suspicion can arise when the pattern of injury seems discordant with the alleged mechanism, especially after consideration of injury biomechanics. Discordance is only one of many factors [30, 54–56] that raise concern about child abuse but it is an important consideration when evaluating children's fractures [57, 58].

Once child abuse is suspected, forensic practitioners must remain open to the possibility that the history provided may be truthful, fabricated, deliberately misleading or incomplete. A careful search for other evidence of injury, such as patterned bruising [59, 60] from fingertip pressure, wounds of different ages, cigarette burns and signs of neglect must form part of the child's clinical examination [61]. Object of protection of such vulnerable persons as a minor must be an absolute imperative.

11.2 Clinical Points on Physical Abuse of Children

The diagnosis of child abuse is often not just a simple diagnosis but requires knowledge from different medical disciplines (paediatrics, neurology, ophthalmology, dermatology, surgery, forensic medicine, toxicology) to reveal a solid diagnostic basis taking into account all differential diagnoses of accidental trauma or confounding diseases [62–64] (Table 11.1). The diagnosis of child abuse may have a number of legal consequences [65]. To avoid legal consequences against the treating physicians—in cases of unreported suspected child abuse as well as in the event of reported but not proven child abuse—the diagnosis has to be confirmed and validated [66, 67].

The legal mandate for physicians to report *suspected* child abuse requires a reasonable suspicion of abuse [68], which is sometimes a difficult instance to meet because of uncertainty regarding the diagnosis, particularly when the physician has a relationship with the family, in which case the physician may want to be more certain of the diagnosis. Following key points and assessment for Suspected Physical Abuse of a Child (Table 11.1) are to be carefully considered in diagnostic approach [7]:

Table 11.1 Assessment for suspected physical abuse of a child

Step 1:	*Obtain a careful history of the alleged circumstances surrounding the injury* Were there witnesses to the event? Who was present with the child when the event occurred? Can the alleged event account for the injuries? Is the child's developmental level consistent with the proposed mechanism of injury? What was done when the event occurred or the child became symptomatic? Was there a delay in seeking medical attention?
Step 2:	*Perform a complete examination with the child fully unclothed* Document the overall clinical status of the child Document the presence of any bruises, burns, or other cutaneous findings Document the presence of intraoral lesions by carefully checking each frenulum for injury Document the presence of findings such as subconjunctival haemorrhages Photograph the findings or request that law enforcement obtain photographs
Step 3:	*Initiate a diagnostic workup on the basis of the findings and clinical condition of the child. The acuteness of the child's condition and the need for medical intervention may determine the order in which diagnostic studies are obtained* Perform CT or MRI of the head Perform CT of the abdomen with contrast enhancement if abdominal injuries are suspected Obtain complete blood count, assess bask (metabolic profile, perform coagulation studies, and measure hepatic and pancreatic enzymes) Perform a full skeletal survey Perform a funduscopic examination with photographs
Step 4:	*Manage any acute medical problem*
Step 5:	*Notify child protective services as mandated in the state. Notification of law enforcement is also mandated in some jurisdictions*
Step 6:	*Hospitalize the child if needed*
Step 7:	*Have hospital personnel or a child protective services social worker perform an extensive social evaluation*
Step 8:	*Consider an additional forensic workup if indicated or requested or refer the case to a paediatrician, team, or centre that specializes in child abuse cases. Additional tests that might be performed include:* A radionuclide scan to look for occult or acute fractures A repeat skeletal survey in 2 weeks Evaluation for blood dyscrasia Evaluation for osteogenesis imperfecta Evaluation of other medical problems as suggested by the differential diagnosis of the findings

List of the recommended steps that should be taken to assess cases of suspected child abuse

- Physically abused children, particularly infants, may present with non-specific symptoms and signs, such as vomiting or apnoea; the possibility of abusive head trauma requires consideration in such cases.
- Physical findings, such as bruising of the face, neck, or torso, or intraoral lesions, such as torn frenula, in infants who are not yet ambulatory should arouse suspicion of inflicted trauma.
- The evaluation of infants and young children for suspected inflicted trauma should include a complete physical examination of the child, with particular attention to the skin, oral cavity [69], and abdomen; imaging of the brain; an examination for retinal haemorrhages; a skeletal survey; and measurement of hepatic and pancreatic enzymes [70–72].
- Physicians are mandated to report to child protective services cases in which they have a reasonable suspicion of child abuse [73].

Detecting fragile bones is a part of clinical assessment, difficult to determine, particularly in children. Neither X-ray images nor bone densitometry scans provide a clinically useful measure of bone strength [57]. Bones have two main structural components: mineral and protein. Abnormality of bone mineralization in children is called osteomalacia or rickets, most commonly related to vitamin D deficiency but there are multiple congenital and acquired causes of rickets. Abnormality of the protein component of bones can also be due to a large number of congenital or acquired conditions and present either as part of a generalized bone disorder effecting multiple bones or joints or incidentally as a localized deformity [74]. The bones of children with reduced bone strength can fracture as a result of lesser forces than children with normal bones (Table 11.2).

Blood tests that should be performed to evaluate bone metabolism include blood levels of calcium, phosphate, alkaline phosphatase, urea, electrolytes, creatinine and vitamin D [75, 76]. There is an ongoing debate regarding the association between vitamin D levels [77], bone mineral density and bone strength at different ages [78] and the AAP have recently recommended that breast-fed infants receive vitamin D [79].

Table 11.2 Conditions that may affect bone strength in children

Abnormalities of mineralization
• Vitamin D deficiency
• Renal disease
• Hypoparathyroidism
• Vitamin D resistant rickets
• Resorption due to disuse
• Osteopaenia of prematurity
• Hypophosphatasia
Abnormalities of protein formation
• Osteogenesis imperfecta
• Congenital bone dysplasias
• Scurvy
• Osteopetrosis
• Menkes disease tumours
• Neuroblastoma
• Leukaemia
• Langerhans cell histiocytosis
Infections
• Osteomyelitis
• Syphilis
Drugs
• Methotrexate
• Vitamin A toxicity
• Prostaglandin E
Aetiology not known
• Infantile cortical hyperostosis

Second-line blood tests include parathyroid hormone levels and urine metabolic screening. Tests for metabolic disease, osteogenesis imperfecta, copper deficiency, syphilis and scurvy might be considered when there are suggestive clinical or radiological features [80]. Additional tests for rare disorders should be considered when additional information related to family history (such as family members with a history of abnormal bones) and the child's particular circumstances (such as abnormal diet, clinical findings and concurrent illness) warrant further consideration.

Bone densitometry tests are not recommended as a useful tool in the routine forensic investigation of bone injury in childhood because these tests have controversial reference ranges in children. In addition, the tests have such low sensitivity and specificity for reduced bone strength that they are of limited use in forensic evaluation of injury.

In circumstances when the diagnosis of osteogenesis imperfecta or other rare metabolic conditions are being considered, advice from an expert in metabolic and genetic conditions should be obtained [81, 82]. The diagnosis of osteogenesis imperfecta is usually a clinical one. Specific tests of collagen synthesis and the genes that code for type 1 collagen (COL1A1 and COL1A2) might be recommended when the diagnosis of osteogenesis imperfecta is being seriously considered [83]. In literature report of a case of OI type I misdiagnosed as child abuse in which treatment was successful despite a tardive diagnosis are still present [84, 85].

11.2.1 Child Fatalities Related to Physical Abuse and Head Trauma

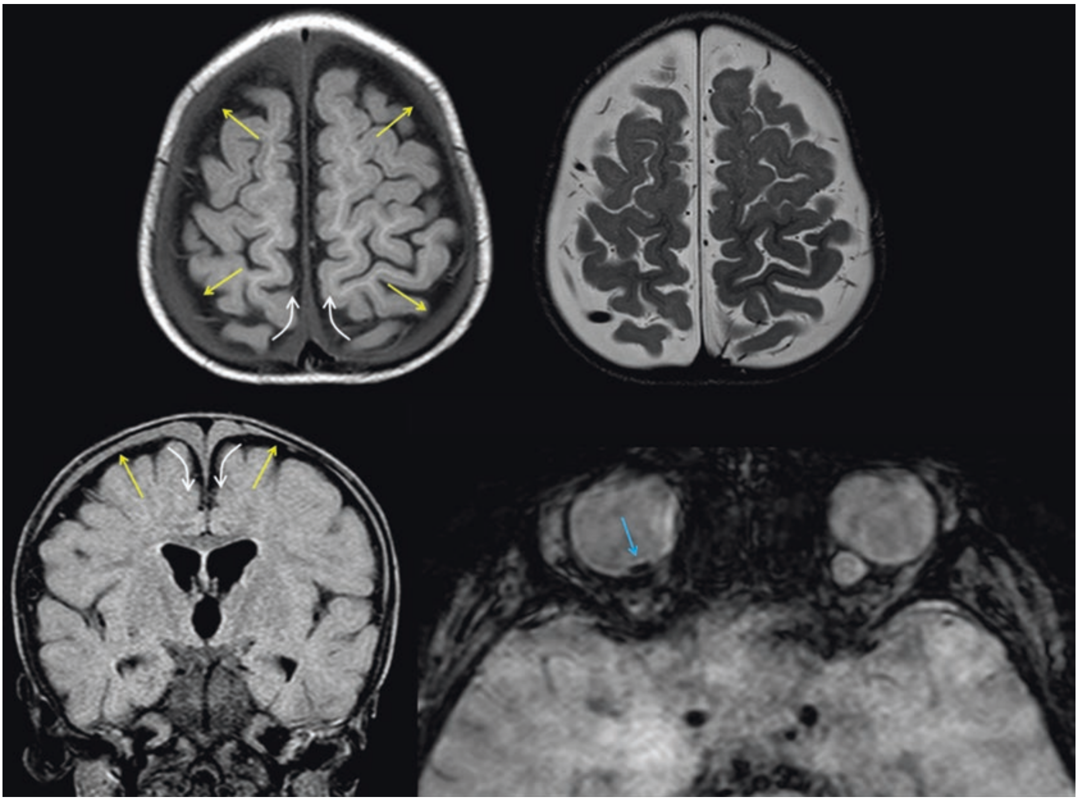
Deaths due to child abuse can occur as a consequence of intracranial or extra cranial injuries [86]. Overall, head injuries are the leading cause of death in abused young children [87], and extensive research has been published describing the epidemiology, patterns and mechanisms of injury associated with paediatric abusive head trauma [88–95]. Uncertainties continue to surround determinations of abusive head trauma [96, 97]; the dispute about the aetiology of these injuries relies both with the advances of forensic knowledge about this matter (way and manner) [98–100] and duty against the law.

The names applied to the syndromes of Inflicted Head Injury in infancy reflect the evolving and sometimes controversial [101] understanding of the actions necessary to cause the types of injuries seen [11, 102–105], such as shaking an infant held by the arms or trunk or forcefully striking an infant's head against a surface [40, 106, 107]. A special focus concerns the triads of Subdural Haematoma, Retinal haemorrhage, brain injury patterns and Shaking syndrome [99, 108–114], with a more recent caveat of the possibility that uncommon [115], or silent 'pathological' [112, 116–126] causes determining retinal haemorrhages.

During inflicted head injury, a distinct type of trauma occurs causing more global brain injury with hypoxic-ischaemic brain injury (HII) and more severe retinal haemorrhages [127–130]. HII is not a necessary factor for severe retinal haemorrhage to develop from inflicted trauma [131].

Understanding of paediatric abusive head trauma has evolved over the last five decades. In 1962 issue of Kemp identified intracranial haemorrhage in young children as a hallmark sign in many cases. In 1971, Guthkelch suggest shaking as a form of abusive injury, reporting on 23 children (22 < 18 months of age) presenting with various combinations of subdural haemorrhage, fractures, parenchymal brain injury, and retinal haemorrhages [113]. Shortly thereafter, Caffey coined the term Whiplash-Shaken infant syndrome [1]. Both Authors noted a frequent absence of external signs of trauma and suggested the role of torn bridging vessels in the brain as the cause of the intracranial haemorrhage [3, 103, 104]. Using autopsy evidence and a dummy model, Duhaime and colleagues in 1987 suggested that blunt impact trauma may be a prerequisite to generate sufficient deceleration forces for the characteristic injuries to occur [132]; however, consistency across perpetrator confessions suggests that shaking alone is sufficient to cause such injuries, and actual injury threshold levels for infant brains have yet to be established [133]. There are currently multiple hypothesized factors in the pathogenesis of brain pathology and retinal haemorrhage in abusive head trauma [134–136], including deceleration and sheering injury, hypoxic-ischaemic injury (from decreased perfusion or apnoea), blunt impact, neck flexion-extension, and raised intracranial or venous pressures [137]. However, the relative importance of these factors cannot be determined precisely based on the published data [138–140].

The availability of diffusion-weighted magnetic resonance imaging (DW-MRI) and non-invasive vascular imaging techniques now make it possible to evaluate the role of hypoxic-ischaemic injury (HII) in traumatic paediatric head injuries [141, 142]. DW-MRI enables identification of acute cellular injury and cytotoxic oedema, which in the context of head trauma may result from hypoxic-ischaemic injury, direct traumatic injury, or both. Brain tissue damage causes shifting of water molecules from extracellular to intracellular compartments, which can be identified as reduced diffusion of water on DW-MRI, in comparison to undamaged areas. Such changes can be identified early, hours or days before changes in the appearance of tissue on T2-weighted sequences. DW-MRI (versus T1 or T2) is particularly helpful in infants, whose brains have a high water content and immature myelination. Biousse and colleagues reported a high incidence of possible HII in a cohort of infants with presumed abusive head trauma. In 2007, Ichord and colleagues demonstrated a relationship between HII and inflicted trauma using DW-MRI in a cohort of children with both accidental and inflicted head injuries [143].



(**a**) Axial T1, (**b**) axial T2 and (**c**) coronal flair MR images of a 12-month boy affected by bilateral subdural chronic haematoma (arrows) with involvement of the interhemispheric fissure (curved arrows). (**d**) SWI MR image demonstrating retinal haemorrhage (arrow). Courtesy of Dr. Andrea Rossi Giannina Gaslini Children's Hospital, Genoa

It has been clearly established that both the presence and increasing severity of retinal haemorrhages are highly associated with abusive versus accidental injury in children presenting with traumatic intracranial haemorrhage [144–146]; however, the mechanisms underlying retinal haemorrhages are still not clearly established, and there is limited information in the literature addressing HII as it relates to ocular findings in the setting of paediatric head trauma [147, 148].

Although the available evidence suggests that it is the sudden deceleration associated with the forceful striking of the head against a surface that is responsible for most, if not all, severe, inflicted brain injuries [149, 150]. Because the histories given when infants with such injuries present for medical attention are often vague or unreliable, the events must be inferred from knowledge of the causative forces in witnessed cases of accidental trauma and experimental models of injury [151].

Studies of the biomechanics of brain injury have established that forces applied to the head that result in a rotation of the brain about its centre of gravity cause diffuse brain injuries. A differential diagnosis that includes non-accidental as well as accidental causes of skull fractures in child deaths is always required in forensic practice [94, 149, 152, 153]. Accidental deaths due to severe trauma (i.e. motor vehicle collisions, vehicles striking pedestrian, skull fractures from heavy falling objects) usually result in extensive multiple injuries together with skull fractures and intracranial findings. In accidental cases such as these, the investigative history usually corroborates the manner of death, since it corresponds with the injuries found, and excludes child abuse [154–156]. Conversely, a fall from a short height (either unwitnessed or witnessed by a single caretaker) can result in skull fractures in children with a very similar pattern to the ones found in inflicted trauma. Confounding this diagnostic dilemma, the majority of head injuries in child abuse and accidental head trauma are often both explained by parents as accidental [157–159]. According to the literature, accidental skull fractures will rarely lead to serious or life-threatening intracranial injury. Further, skull fractures due to accidental falls are rarely seen in concert with simultaneous fractures in other skeleton segments (i.e. ribs or extremities) [160, 161]. The investigative history is a fundamental part of the diagnostic process, as an accidental skull fracture can nearly always be routinely absent [157, 159, 162].

Diastases of cranial sutures are more common than skull fractures and could be either a direct consequence of trauma or due to raised intracranial pressure from any cause, because suture diastases occurred both in cases with signs of impact to the head as well as no signs of impact [163–165]. Their frequency is significantly higher in infants than in toddlers, because of the progressive fusion of the sutures during growth. Extradural haemorrhages (EDH) is rarely seen in paediatric cases due to the tight adherence of the dura mater to the skull and because of the elasticity of the young skull and not always associated with skull fractures. EDH does not occur in subjects with no signs of an impact. EDHs are typically associated with accidental trauma but have been also described in abused children. EDH may occur with relatively minor trauma to the parietal or temporal skull if the vulnerable middle meningeal artery is torn, so it is frequently observed in accidental trauma (Fig. 11.2).

Intra-dural haemorrhages are caused by physical or physiologic damage to the dural capillary plexus that, according to some researchers, can lead to SDH [166]. SDH are observed both in the subjects with and in those without visible evidence of impact injury. Infants are more affected

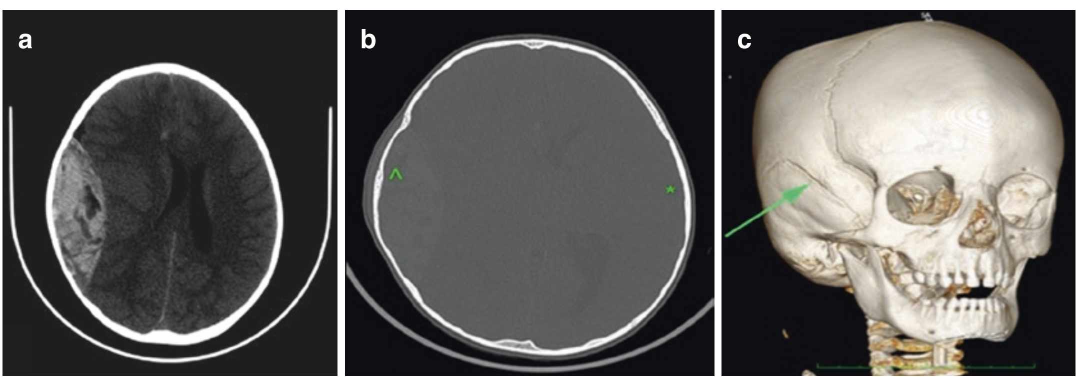


Fig. 11.2 Accidental head trauma in a 3-year-old boy. Fall from a short height (witnessed by caretakers and children) (Authors' observation). (**a**) Axial non-contrast CT scan shows a huge epidural right-side epidural haemorrhage, with inhomogeneous density of haematoma, due to different timing of haemorrhage; (**b**) axial CT scan slab-VR 3D reconstruction shows deformity of parietal and frontal bone along the squamosal (^) suture, compared to left side (∗); (**c**) 3D VR image clearly shows the fracture along the squamosal suture

by subdural haematoma than toddlers and older children, because the brain of infants has more space than the brain of older children to move around in the skull upon impact. It has been reported in the literature that subdural haemorrhages in cases of abusive paediatric head trauma are rarely massive [137, 167]. This seems to confirm that SDH is not typically a lesion producing increased intracranial pressure, but rather a marker of brain movement within the cranial cavity, which may be associated with some shearing brain injury (e.g. diffuse axonal injury). In fact, because the dura is firmly attached to the skull and the arachnoid to the cerebral cortex, most brain motion occurs across the potential subdural space. The thin-walled bridging veins are thus easily vulnerable to tearing. Therefore, in every paediatric autopsy it is extremely important for the pathologist to remove the brain personally or directly observe its removal when performed by a technician. Otherwise, a thin layer of blood from subdural bleeding could easily be missed as it will tend to slide quickly off the surface of the brain as the calvarium is removed. Subdural haemorrhages caused by accidental trauma are typically produced by severe force such as a motor vehicle accident, ejection from a motor vehicle, or a fall from a significant height. Accidental SDHs usually occur at the site of impact, are limited to the cerebral convexities, and are often isolated and associated with an overlying fracture.

11.2.2 Pathology Findings at Autopsy

Closed head injury in early infancy (5 months of age and younger) produces focal lesions, parenchymal laceration—also known as contusional tears—and diffuse astrocytic reaction [168–170].

The presence of skull fractures, diffuse axonal injury, and subdural haemorrhage suggest that abused children are subject to several forms of injury, direct trauma to the skull causing fractures; brain acceleration over a short arc producing subdural haematoma; and slower acceleration in shaking where axons are damaged [171]. This inertial effect may also produce acute subdural haemorrhage as the subdural veins are sensitive to shearing forces. Diffuse axonal injury was not usually seen in the brain stem. In the diffuse axonal injury described in adults axonal discontinuities are typically seen in the dorsolateral quadrant of the brain stem [172]. In all cases in which contusional tears had occurred, diffuse axonal injury was evident [141, 173]. Thus, the presence of a contusional tear was focal evidence of more diffuse damage. The experimental studies of axonal damage to subhuman primates, using angular acceleration, suggest that there is a slower component of the acceleration which produces the diffuse axonal injury, but it is impossible to identify the varieties of trauma used in the cases [174, 175] (Fig. 11.3).

Fig. 11.3 A 3-month-old infant with accidental head trauma, with clinical and circumstantial suggesting infant shaking syndrome; the child showed coma and apnoea at presentation to first aid of health professional and died after few minutes; at autopsy gross examination showed a thin layer of subarachnoidal haemorrage (∗) on cerebral convexities and left side (**a**) and dramatic brain swelling (**b**), with histology evidence of hypoxic ischaemic brain damage (**c**, H&E, 25×)

11.2.3 Child Abuse Imaging Protocol and Forensic

When bone trauma is suspected, the critical investigations are radiological and a classic aphorisms in the study of child abuse was stated by the forensic pathologists, Cameron, Johnson and Camps ('The skin and bones tell a story which the child is either too young or too frightened to tell'). The essence of forensic evaluation of bone trauma in children is being able to determine *whether* bone trauma has occurred, *when* bone trauma might have occurred and being able to determine the *likely mechanism* of the injury [56, 176, 177].

Forensic evaluation of bone injury in children requires a preliminary understanding of bone metabolism and growth, an understanding of injury biomechanics causing fractures and an appreciation of the power and limitations of radiological investigation to accurately detect the presence and estimate the time of bone injury.

The forensic opinion about the likely cause(s) of injury can then be compared to the offered explanation and their mutual compatibility analysed. To establish whether bone trauma has occurred, it is also essential to have an understanding of the normal structure of different bones and how they deform with application of different forces [178]. Infants and very young children have significantly different bone structure, metabolism, bone strength and reaction to mechanical trauma than older children, adolescents and adults [179]. In addition, there are a number of metabolic processes and congenital abnormalities that can affect the strength of children's bones and therefore their bones' susceptibility to injury when mechanical forces are encountered [180, 181]. Children's bones are softer, contain more cartilage than adults' bones, the growth plates are relatively fragile and the structure of the bone matrix alters with maturity.

Bone trauma in children can be difficult to detect with both clinical and radiological examination findings sometimes problematic to analyse. Errors in interpretation of findings can lead to incorrect forensic conclusions and subsequent erroneous action on the part of the state to protect vulnerable children. A misdiagnosis of child abuse can be as harmful as a missed diagnosis of child abuse. Studies attempted to compare magnetic resonance imaging (MRI) using limited sequence protocols to CT (computerized tomography) for evaluating paediatric head trauma. Although studies have some differences in sequences and population demographics, both conclude that the CT and MRI are comparable in terms of detecting acute intracranial haemorrhages. In fact, one institution claims to use MRI as the initial imaging exam in paediatric head trauma except 'if the wait for MRI is unacceptably long for appropriate patient care' [182], and moreover doesn't use ionizing radiation [183].

As it was said, if MRI could fulfil three conditions—be cheap, be available everywhere, with no or minimal wait time, and offer complete scans in very short times—every Emergency Room would have one and would use it almost exclusively for evaluating paediatric head trauma [184]. A full sequence MRI is more sensitive and specific than CT for everything in the head except for some bone lesions. Add to that the capability for diffusion imaging and diffusion tensor imaging for discovering new information and making diagnoses of brain lesions, not detectable on conventional MRI, and the user can appreciate why MRI may eventually become ubiquitous. Using MRI costs more and is more expensive to operate. It has physical limitations to access since the magnet is always on and is waiting to suck in the unwary. Also, the vast and ever growing array of possible MRI sequences causes longer and longer exam times. MRI imaging, furthermore, needs completely immobilization of patients and this condition is difficult to have in children, sometimes making necessary to anaesthetize young patients.

Some authors observe they are aware of and very sensitive to radiation exposure in children, particularly those who will need repeated studies using ionizing radiation. However, with the exception of the lens of the eye, the brain is pretty radiation insensitive, but is always necessary to consider the risk-benefit ratio of using CT imaging instead of MR one. If there is an acutely injured child it makes no sense to not perform a CT if that is what the situation demands. A properly performed head CT is highly unlikely to add much, if any, morbidity to a child's life, especially one with a critical head injury.

As with other areas of medicine, the diagnostic process builds upon the history provided and the clinical examination findings. The process of forensic evaluation of injury follows a standard pathway; this is no different for evaluation of suspected bone trauma than for any other injury that might have an inflicted cause.

Financial, political, jurisdictional and geographical considerations result in varying recommendations and practices throughout the world.

Recommendations from local health authorities, colleges and special societies guide good practice [185–187]. If access to facilities for specialist investigation is limited, alternative means of investigation need to be considered, as the following: it is also questionable the possibility to perform all investigations close to the child's home, to minimize the child's exposure to ionizing radiation and to investigate using recommended 'gold standard' tools. The long-term risks of ionizing radiation which may have a latent period of decades must be balanced against the short-term risk of further physical harm to the child. Obtaining consent for these imaging procedures should not be overlooked, but will sometimes come from a temporary Court-nominated child carer, instead of child's parents. Sometimes it is in a child's best interests for him/her and to assure physical protection to travel to a specialist centre or third level hospital, for investigation using techniques and facilities optimized for paediatric imaging. Decision-making must carefully consider the local national guidelines for health assessment of child protection. For children aged less than 2 years, consensus exists regarding the need for a high-quality radiographic skeletal survey as an essential part of the investigation of suspected non-accidental injury. Even if the injury appears to be localized to one body region, the whole skeleton is surveyed because these children are (usually) unable to give a reliable history, are vulnerable to injury because of their immature and relatively weak skeleton and their small size and the injuries may not be evident from clinical examination of the child. The recommended facilities, equipment, technique and protocols needed to obtain this high-quality skeletal survey vary slightly between countries and professional colleges, however, with each region recommending a standard protocol. The American College of radiology (ACR-SPR, Practice Parameter for the Performance and Interpretation of Skeletal Surveys in Children) recommends high-quality digital X-rays, with lowest possible radiation dose, and concurrent monitoring of the images by a radiologist (in case additional views are required in order to further define perceived abnormality) [12, 13]. A common error in acquisition of skeletal survey images by practitioners unfamiliar with accepted protocols is the failure to obtain coned views of the metaphyses and growth plates of the long bones (especially at the wrist, knees and ankles) and inadequate imaging of the ribs due to poor aeration of the lungs, patient movement, suboptimal exposure factors/image capture or poorly centred X-ray beam. A nuclear medicine scan is recommended as an additional 'first-line' investigation because the combination of nuclear medicine scan and radiographic skeletal survey increases the overall detection rate of non-accidental injury in children [180, 188–192], but really Authors think that the radiation exposition of nuclear medicine scan could be too high to be routinary accepted in clinical practice. Other imaging modalities can also be used to provide additional information when clinically indicated. Ultrasound examination of bone, for example, for ribs fractures, can help radiology in detecting fractures not clearly detectable in X-ray imaging [193–195].

In children aged more than 2 years the detection rate of occult bone injury is significantly lower and suspected bone injuries are best investigated using an X-ray of the suspected site of injury. For some children aged 2–3 years in whom occult bone injury is strongly suspected, radiological investigation with skeletal survey may be considered, leaving bone scan in doubt cases. The likelihood of detecting occult bone injury in a child aged more than 5 years is small because they are able to provide a better history and their bones require a greater force to cause injury. Consequently, for older children, radiographic skeletal surveys should be reserved for emergency or in other exceptional circumstances when the clinical suspicion is unusually high. Blood tests to evaluate bone metabolism should also be performed as a routine procedure when non-accidental bone trauma is suspected in children of all ages. The authors suggest tests such as serum calcium, phosphate, alkaline phosphatase, urea and electrolytes and serum vitamin D [196, 197]. Sometimes additional tests need to be considered. When an individual clinician lacks adequate knowledge and skill to collate this information and form a forensic opinion, it is imperative that he/she seek advice from a suitably experienced forensically trained colleague. Nuclear medicine bone scan demonstrates bone trauma by identifying the metabolic changes that occur within bone tissues as a consequence of trauma. Sometimes, the bone metabolic changes demonstrated may not be associated with a visible change in appearance on an X-ray examination.

An understanding of the physical principles of these imaging techniques is required to facilitate the identification of injuries that are not apparent on clinical examination. The changes that are demonstrated must be differentiated from normal variants and pathologies that mimic bone trauma. Some mimics of bone pathology require investigation with other laboratory tests.

The differences in the electron density of different body tissues and the properties of the different detector systems allow five different densities to be identified on conventional X-ray images or radiographs of humans. Bony trauma is most commonly identified when a gap, defect, break or alignment-abnormality is demonstrated in a bone or there are radiological signs of a healing bone injury present. Factors that influence whether bone injury will be demonstrated include the separation of the bony margins of the fracture, orientation of the fracture to the X-ray beam, position of the fracture in the body relative to other structures (is it obscured by overlying bones) and both the stage and extent of bony healing. Technical factors that can influence how well the bones are demonstrated include the resolution of the imaging system used, how well the child is immobilized, whether the images are coned to a region, the number of projections making up the examination and whether exposure factors appropriate for the size of the child are used. Coned views and different projections can be very important to identify subtle injuries of growth plates and ribs.

Femoral radiogram (AP view) demonstrating metaphyseal distal femoral fracture in a 6-month boy. Diffuse periosteal reaction of the distal femur after 15 days (AP and lateral views). Courtesy of Dr. Claudo Granata Giannina Gaslini Children's Hospital, Genoa

Plain radiographs are two-dimensional (2D) representations of 3D structures and this causes superimposition of body tissues and organs. This, together with the limited ability of plain radiographs to distinguish between different body tissues, can make identification of some structures difficult. However, when looking at bones in the extremities, this is less of a problem as there are fewer superimposed structures, and radiological orthogonal projections of bones help in fractures detection. Plain X-rays have the ability to produce images with very high spatial resolution (down to 100 μm or less). Despite this very high resolution, some bone fractures are not visible on plain

X-rays due to the orientation of the X-ray beam to the fracture and/or the small distance of separation of the fracture fragments. Computed tomography (CT) imaging produces images by using a collimated X-ray beam that is directed through the patient in multiple projections. The data collected from the multiple different projections allows mathematical computation of the attenuation of X-rays in individual points of the patient. A single slice used to take less than 1 min to acquire and a longer time to process. Current multidetector CT scans have a resolution of about 0.3 × 0.3 × 0.3 mm, can simultaneously acquire up to 320 slices in less than 0.5 s and are able to process all the data required to generate/construct the images in a few seconds. The improved temporal resolution reduces movement artefact and the improved spatial resolution in all imaging planes has resulted in an increased use of CT scans to identify complex fractures in multiple planes or oblique to the incident X-ray beam, with very good contrast resolution. CT scans can therefore demonstrate fractures that are not visible on conventional radiographs. Computed tomography utilizes ionizing radiation and involves a higher, more significant dose than plain radiographs. Where plain radiographs are able to differentiate five densities, CT has a much greater contrast resolution and can differentiate between tissues such as blood, white matter, grey matter and cerebrospinal fluid (CSF) in a CT scan image of the brain. The contrast between tissues of different structures can be manipulated by adjusting the image window width and level; this gives rise to terms such as 'bone window' settings. CT imaging allows superimposed structures to be individually identified and provides detail of bone anatomy which is very useful in the assessment of bones with a complex shape and around joints. CT very clearly shows focal areas of bone lysis/destruction, areas of bone sclerosis and periosteal new bone formation. CT is able to provide some information about soft tissues, particularly when there is localized soft tissue calcification or in case of subcutaneous emphysema, not easy to depict at X-ray when is of little amount. Ultrasound examinations and MRI studies usually provide superior images of soft tissues that are more likely to identify soft tissue pathology, especially in case of detection and in dating of haematomas. Forces causing bones fracture in infant is a part of diagnostic process. Planar micro-fractures at the metaphyseal-epiphyseal region in the immature primary spongiosum layer of immature long bones are believed to be caused by planar shearing forces. These injuries are referred to as classic metaphyseal lesions [198], but they are frequently referred to as 'bucket-handle' or 'corner' fractures [199]. Shearing forces occur during rapid acceleration and deceleration when an infant is shaken and when traction, compression or rotation forces are applied to cartilaginous epiphyses. While the X-ray appearance may differ depending on the projection of the X-ray beam, these radiological findings actually represent a single pathological process. Diaphyseal (shaft of long bone) fractures are more commonly seen in abused children than classic metaphyseal lesions. Diaphyseal fractures are commonly caused by accidental childhood trauma such as might be experienced during an accidental fall or sporting injury. In contrast to classic metaphyseal lesions, the site and type of diaphyseal fracture rarely contributes to differentiation of accidental from inflicted trauma. In some circumstances, for example, when the presence of a spiral fracture indicates that a torsional force has been applied, a discrepancy between the caregiver's offered explanation and the observed pattern of injury might suggest that the caregiver is not being truthful. Additional questions would need to be asked about the alleged mechanism of injury. Transverse fractures of the diaphysis can be caused by the application of force perpendicular to the shaft of the long bone (such as a direct blow) but can also be caused by bending forces transmitted along the shaft of the bone (Fig. 11.4). The periosteum is relatively poorly attached to the diaphysis of children's bones. Torsional and twisting forces can strip the periosteum from the cortex of diaphyseal bone, resulting in subperiosteal haemorrhage and resultant subperiosteal callus. When child abuse is diagnosed on the basis of skeletal injury, skull fracture is a common cause of presentation [164] (Fig. 11.5). Simple linear skull fractures occur as a result of both inflicted and accidental trauma

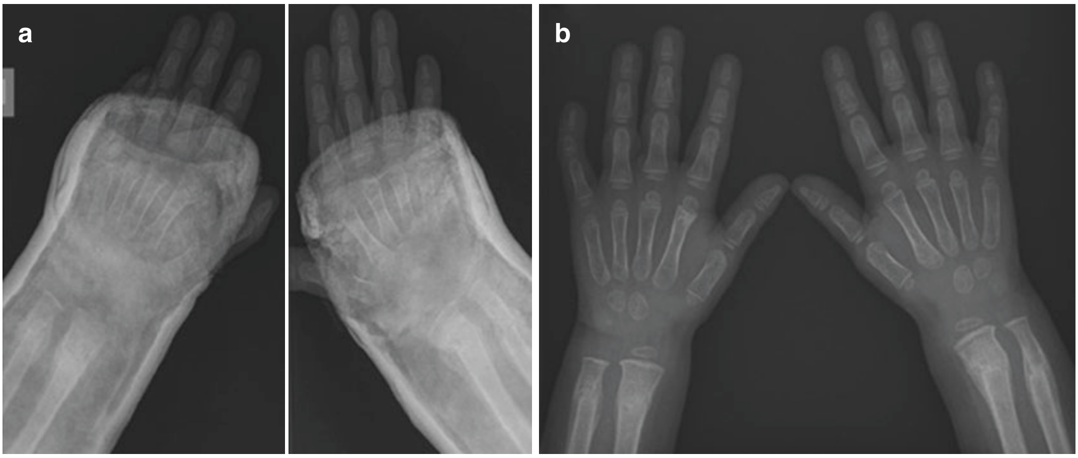


Fig. 11.4 Non-accidental fractures in a 4-year-old female (Authors' observation). (**a**) Bilateral fractures of distal radius and ulna with metaphyseal dorsal angulation and dislocation. (**b**) Nevertheless, the altered cortical bone profile, thanks to the physiological bone remodelling, may undergo to a complete resolution

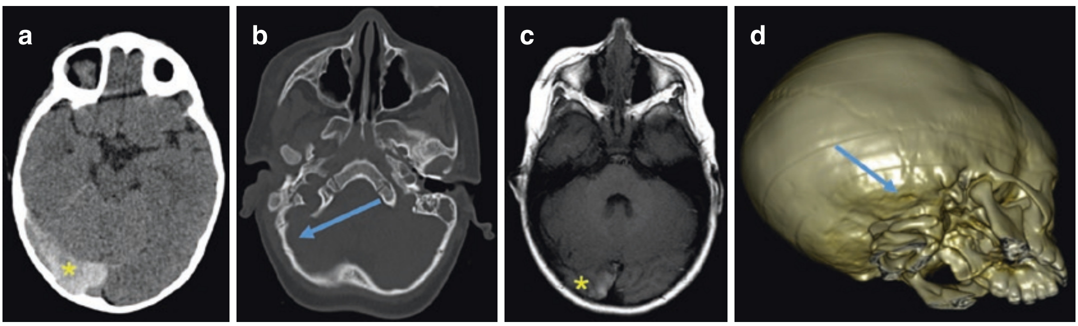


Fig. 11.5 Non-accidental brain injuries in a 2-year-old female (battered by her mother). (**a**) Brain CT scan shows right parasagittal and occipital epidural haemorrhage (∗) with ipsilateral tentorial and posterior falcine involvement; (**b**) small ipsilateral cortical lesion of the occipital/peri-mastoid skull (arrow) is present as well; (**c**) subarachnoid haemorrhage is still detected in brain MR, performed 3 month after with little haematic residuals (∗) along the occipital bone; (**d**) 3D VR reconstruction CT image shows an inflexible fracture of occipital bone (arrow)

[200–206] (Fig. 11.6). Non-parietal fractures, multiple and complex fractures, widely separated and depressed fractures and fractures associated with significant intracranial injury should all raise concern about possible inflicted injury. A single impact can result in a skull fracture that crosses a suture line. Rarely, a single impact may transmit forces such that two skull bones fracture some distance from the impact site. When bones on either side of the skull are fractured, multiple impacts and crush injury should be considered. Ping-pong fractures where there is deformity without a gap in the bone due to the pliability of the immature skull and depressed skull fractures can occur as a result of blunt impact with a contoured object in circumstances of accidental and inflicted trauma.

These fractures show transversal appearances, which are uncommon in accidental traumatic fractures in children.

Indeed, they usually show greenstick appearance, with eccentric far cortex comminution. Alternatively, but less commonly, they may show spiral appearance, caused by the involvement, during traumatic events, of diaphyseal long axis, thus resulting in oblique and longitudinal fracture rime. In this patient instead complete transverse fracture rimes are present and they can be caused

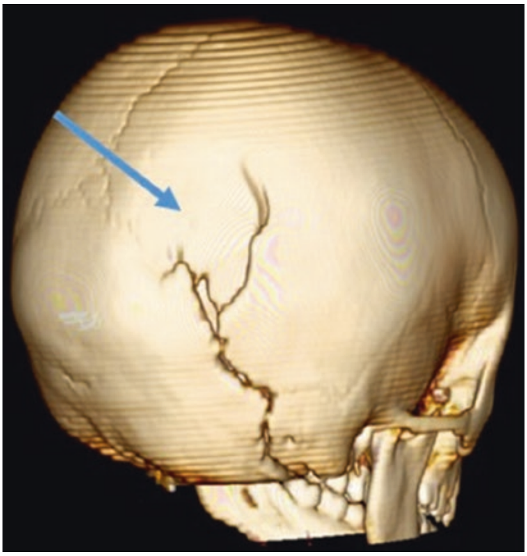


Fig. 11.6 18-month-old female. D: 3D VR reconstruction CT image shows a huge fracture of occipital bone (arrow)

by direct injury, probably painful, on the distal diaphyseal bones. Moreover, the bilateral fractures localized at the same bone portion may suggest a non-accidental injury, while they usually show different localization in the same bone, especially in cases of accidental distortion-caused fractures. Patient was treated by orthopaedic casts placement, but without surgical fractures reduction, which has lead, in 30 days, to abundant callus formation and bones deformities.

11.3 Conclusion

There are no particular bone injuries that are pathognomonic of child abuse [202–204, 207]. Although there are not bone injuries diagnostic of child abuse [208], however, some patterns of bone injury are seen much more frequently in assaulted children than in children who sustain accidental bone trauma. Classic metaphyseal lesions in children aged less than 2 years are seen far more commonly in abused children than in children experiencing trauma as a result of accidents [209].

Posterior rib fractures, fractures of the scapula, ends of clavicles, sternum and vertebral spinous processes suggest, but do not prove, injury caused by assault. Multiple bilateral symmetrical fractures, fractures judged to be of different ages, fractures of the hands and feet, vertebral body fractures, complex skull fractures and associated non-skeletal injury should raise suspicions of possible assault. Authors have referred to the 'specificity' of recognized patterns of bone injury to facilitate a diagnosis of child abuse [198, 210], but caution is imperative. Fractures seen predominantly in children known to have been abused have been categorized as 'specific' for child abuse whereas fractures occurring in both assaulted and accidentally injured children have been categorized as 'non-specific'. The most common types of fractures seen in assaulted children are fractures that are categorized as 'non-specific', including diaphyseal fractures and linear skull fractures. When forensic practitioners form an opinion about the cause of a child's bone injury, their existing knowledge base underpins opinion formation. Unfortunately, the existing literature is replete with examples of poor research methodology, dogmatic statements based on only a small number of cases and faulty reasoning by way of circular logic and other fallacies of logic. For example, the willingness of a forensic practitioner to diagnose child abuse in a toddler with an isolated spiral femur fracture is strongly influenced by the forensic practitioner's belief that in a non-ambulant child this fracture pattern is more likely to be caused by abuse than an accident. At times, circular logic, the child's poor social circumstances [211] and the presence of coexisting injuries influence the forensic practitioner's diagnosis in relation to fracture causation [212]. Knowledge of bone healing in children take a relevant part of diagnostic contribution in a suspected abusive fracture and several authors have made important contributions to our understanding of how bones recover from trauma. The times-since-injury implies particularly those working in a forensic context, are cautioned that these times are only estimates based on limited data and the understanding has been derived from the study of bone injury in children who have died as a result of accidental trauma. Histological analysis suggests that the healing process is usually no different when the cause of injury is inflicted trauma [213]. Bone healing is understood to occur in a number of phases. Firstly, the *induction phase* extends from the time of injury to the appearance of new bone at the fracture site. An inflammatory response initiated with the associated bleeding may last a few days and reveal itself on an X-ray in the form of soft tissue swelling with displacement and obliteration of normal fat and fascial planes. The initial soft tissue swelling is referred to as the pro-callus. A fracture line that initially appears sharp will change as healing progresses. Damaged bone is first resorbed as part of the inflammatory process which may blur the fracture margins. A nuclear medicine bone scan may detect increased blood flow due to inflammation and an MRI or ultrasound scan may detect soft tissue changes that are not yet evident on an X-ray. Soft or primary callus then starts to form with the fibrocartilaginous tissues laid down to stabilize the bones at the fracture site converting to loosely woven bone. In infants, this calcification/ossification is frequently seen as periosteal new bone and can occur within approximately 7–10 days but tends to occur later in older children (10–14 days after injury). Exuberant callus formation can be a sign of fracture instability and/or repetitive injury. Next, hard callus forms when disorganized periosteal and endosteal bone (sometimes called the provisional callus) begins to convert to lamellar bone with the bony trabeculae orientated along the lines of weight bearing/compression/tension. This phase begins in infants at 14–21 days at the earliest and peaks at 21–42 days. Remodelling occurs with gradual correction of deformity and resorption of the excess callus laid down as part of the initial healing process. This phase begins after bone union and may continue for 1–2 years after the injury. Remodelling of bone following bone fracture in children can result in a bone that heals completely and appears indistinguishable on X-ray from a bone that has not been injured.

Healing generally occurs more rapidly in younger infants but the rate at which bones heal and remodelling occurs varies with a number of factors: the anatomy of the injured bone; the site and nature of fracture; the degree of angulation and displacement of bone segments; the degree of immobilization of fractured bones during healing; stresses applied across the fracture during healing; and metabolic processes that enable healing of bone injury. Subtle fractures such as classic metaphyseal lesions and rib fractures may only be identified on plain X-rays once the healing process is well established, making bone scans or repeat X-ray examination essential in the evaluation of injury. With optimal healing conditions, a fractured bone may appear normal on an X-ray or nuclear bone scan examination in as short a time as a few months. Other bones, such as the skull, may not show radiological evidence of healing; dating skull fractures is notoriously imprecise. Table 11.3 identifies factors that may influence bone healing.

Table 11.3 Bone healing repairing

Factors promote bone healing	Factors that impair bone healing
Immobilization	Poor immobilization/instability
Early treatment	Delayed treatment
Simple fracture, two segments	Complex fracture, multiple segments
Good alignment, small gap (minimal displacement)	Separation, angulation or displacement of fracture segments
Good blood supply	Precarious/inadequate blood supply
Youth, good nutrition and good health	Old age, poor nutrition, poor health, local or generalized infection

When a fracture involves the growth plates or is allowed to unite with angulation or displacement, the bone may not grow normally resulting in a permanent deformity and/or limb shortening. When children's bones have been injured by inflicted trauma, other factors that might influence the healing process are a delay in presentation for medical treatment, coexisting nutritional deficiencies (possibly associated with neglect) and subsequent/repeated trauma at the fracture site. Two 1987 case reports by Perkins and Skirvin [214] and Spencer [215] reported faster healing of femoral fractures in children with coexisting severe head injury. These studies have not been replicated and the clinical significance of this association remains uncertain.

When forensic practitioners form an opinion about the cause of a child's bone injury, their existing knowledge base underpins opinion formation. Unfortunately, the existing literature is replete with examples of poor research methodology, dogmatic statements based on only a small number of cases and faulty reasoning by way of circular logic and other fallacies of logic. For example, the willingness of a forensic practitioner to diagnose child abuse in a toddler with an isolated spiral femur fracture is strongly influenced by the forensic practitioner's belief that in a non-ambulant child this fracture pattern is more likely to be caused by abuse than an accident. At times, circular logic, the child's poor social circumstances and the presence of coexisting injuries influence the forensic practitioner's diagnosis in relation to fracture causation [212]. Knowledge of bone healing in children take a relevant part of diagnostic contribution in a suspected abusive fracture and several authors have made important contributions to our understanding of how bones recover from trauma. The times-since-injury implies particularly those working in a forensic context, are cautioned that these times are only estimates based on limited data and the understanding has been derived from the study of bone injury in children who have died as a result of accidental trauma. Histological analysis suggests that the healing process is usually no different when the cause of injury is inflicted trauma [213]. Bone healing is understood to occur in a number of phases. Firstly, the *induction phase* extends from the time of injury to the appearance of new bone at the fracture site. An inflammatory response initiated with the associated bleeding may last a few days and reveal itself on an X-ray in the form of soft tissue swelling with displacement and obliteration of normal fat and fascial planes. The initial soft tissue swelling is referred to as the pro-callus. A fracture line that initially appears sharp will change as healing progresses. Damaged bone is first resorbed as part of the inflammatory process which may blur the fracture margins. A nuclear medicine bone scan may detect increased blood flow due to inflammation and an MRI or ultrasound scan may detect soft tissue changes that are not yet evident on an X-ray. Soft or primary callus then starts to form with the fibrocartilaginous tissues laid down to stabilize the bones at the fracture site converting to loosely woven bone. In infants, this calcification/ossification is frequently seen as periosteal new bone and can occur within approximately 7–10 days but tends to occur later in older children (10–14 days after injury). Exuberant callus formation can be a sign of fracture instability and/or repetitive injury. Next, hard callus forms when disorganized periosteal and endosteal bone (sometimes called the provisional callus) begins to convert to lamellar bone with the bony trabeculae orientated along the lines of weight bearing/compression/tension. This phase begins in infants at 14–21 days at the earliest and peaks at 21–42 days. Remodelling occurs with gradual correction of deformity and resorption of the excess callus laid down as part of the initial healing process. This phase begins after bone union and may continue for 1–2 years after the injury. Remodelling of bone following bone fracture in children can result in a bone that heals completely and appears indistinguishable on X-ray from a bone that has not been injured.

A radiological estimate of the time of injury involves evaluation of the appearance of soft tissues, the fracture line, the deformity and the callus. It is therefore only possible to offer an estimate of the possible date of injury within a range. However, when there are multiple sites of trauma it is often useful to assess whether the injuries are likely to have occurred at the same or different times. Repeated trauma raises suspicions of inflicted injury. In the systematic review of Prosser et al. [216], the dating of fractures in children identified only three articles that met their inclusion criteria. Of the 189 children reported (243 fractures), only 56 children were aged less than 5 years, the age group in which most fractures caused by assault occur. Kleinman's textbook (1998) offers opinion about dating fractures based on the author's personal experience. Offiah and Hall [217] recently added significantly to the literature about fracture patterns and bone healing in abused children. However, there remain few cases on which to base firm opinions about the features of healing fractures in children of different ages, in different bones and with different fracture patterns.

In addition to the physiological factors, the radiographic technique and imaging system resolution might not be optimized. It is important that high-resolution techniques be used and that imaging is performed by radiographers familiar with techniques used to optimize the imaging of children. Radiologists who are less familiar in interpreting children's X-rays might not detect signs of bone injury that are recognized more readily by experienced paediatric radiologists. Ultrasound is very operator-dependent and requires specialist skills. Its nature is more suited to localized examination of a clinically suspicious area rather than generalized screening for evidence of injury. However, it can be used to identify soft tissue changes when there is a focus of clinical concern such as bruising or swelling when the X-ray is normal.

References

1. Tardieu A (1860) Étude médico-légale sur les sévices et mauvais traitements exercés sur des enfants. Ann Hyg Pub Med Leg 13:361–398
2. Caffey J (1946) Multiple fractures in the long bones of infants suffering from chronic subdural hematoma. Am J Roentgenol Radium Ther 56:163–173
3. Caffey J (1972) The parent-infant traumatic stress syndrome; (Caffey-Kempe syndrome), (battered babe syndrome). Am J Roentgenol Radium Therapy, Nucl Med 114:218–229
4. Cameron JM, Johnson HR, Camps FE (1966) The battered child syndrome. Med Sci Law 6(1):2–21
5. Cameron JM, Rae LJ (1975) Atlas of the battered child syndrome. Churchill Livingstone, Edinburgh, London
6. Kempe CH, Silverman FN, Steele BF, Droegemueller W, Silver HK (1962) The battered-child syndrome. JAMA 181:17–24
7. Berkowitz CD (2017) Physical abuse of children. N Engl J Med 376:1659–1666
8. Block RW, Palusci VJ (2006) Child abuse pediatrics: a new pediatric subspecialty. J Pediatr 148(6):711–712
9. Adams G, Ainsworth J, Butler L, Bonshek R, Clarke M, Doran R, Dutton G, Green M, Hodgkinson P, Leitch J, Lloyd C, Luthert P, Parsons A, Punt J, Taylor D, Tehrani N, Willshaw H, Child Abuse Working Party (2004) Update from the child abuse working party: Royal College of Ophthalmologists. Eye (Lond) 18:795–798
10. Ross AH, Juarez CA (2014) A brief history of fatal child maltreatment and neglect. Forensic Sci Med Pathol 10(3):413–422
11. Adamsbaum C, Grabar S, Mejean N, Rey-Salmon C (2010) Abusive head trauma: judicial admissions highlight violent and repetitive shaking. Pediatrics 126:546–555
12. American Academy of Pediatrics, Section on Radiology (2000) Diagnostic imaging of child abuse. Pediatrics 105:1345–1348
13. American Academy of Pediatrics, Section on Radiology (2009) Diagnostic imaging of child abuse. Pediatrics 123:1430–1435
14. Pierce MC, Bertocci G (2008) Injury biomechanics and child abuse. Annu Rev Biomed Eng 10:85–106
15. Gao WM, Chadha MS, Berger RP, Omenn GS, Allen DL, Pisano M, Adelson PD, Clark RS, Jenkins LW, Kochanek PM (2007) A gel-based proteomic comparison of human cerebrospinal fluid between inflicted and non-inflicted pediatric traumatic brain injury. J Neurotrauma 24(1):43–53
16. Berger RP, Adelson PD, Pierce MC, Dulani T, Cassidy LD, Kochanek PM (2005) Serum neuron-specific enolase, S100B, and myelin basic protein concentrations after inflicted and noninflicted traumatic brain injury in children. J Neurosurg 103(1):61–68
17. Cicchetti D, Rogosch FA, Sturge-Apple ML (2007) Interactions of child maltreatment and serotonin transporter and monoamine oxidase A polymorphisms: depressive symptomatology among adolescents from low socioeconomic status backgrounds. Dev Psychopathol 19(4):1161–1180
18. Hecker T, Radtke KM, Hermenau K, Papassotiropoulos A, Elbert T (2016) Associations among child abuse, mental health, and epigenetic modifications in the proopiomelanocortin gene (POMC): a study with children in Tanzania. Dev Psychopathol 28:1401–1412
19. Zarate YA, Clingenpeel R, Sellars EA, Tang X, Kaylor JA, Bosanko K, Linam LE, Byers PH, Genet A (2016) COL1A1 and COL1A2 sequencing results in cohort of patients undergoing evaluation for potential child abuse. Am J Med Genet A 170(7):1858–1862
20. Maguire S, Mann MK, Sibert J, Kemp A (2005) Are there patterns of bruising in childhood which are diagnostic or suggestive of abuse? A systematic review. Arch Dis Child 90:182–186
21. Maguire S, Mann MK, Sibert J, Kemp A (2005) Can you age bruises accurately in children? A systematic review. Arch Dis Child 90:187–189
22. Vock R, Schellmann B, Schaidt G (1980) Isolated injuries of intestinal tract due to body maltreatment (author's transl). Z Rechtsmed 84(2):155–159
23. Global Burden of Disease (2013). World Health Organization, Geneva. http://www.who.int/topics/global_burden_of_disease/en/. Accessed 18 Nov 2017

24. Global Status Report on Violence Prevention (2014) WHO, Global status report on violence prevention 2014, Geneva. Accessed 15 Nov 2017
25. Child Maltreatment (2012) Washington: A.f.C.a.F. U.S. Department of Health and Human Services, Administration on Children, Youth and Families, Children's Bureau, 2012
26. U.S. Department of Health & Human Services, Administration for Children and Families, Administration on Children, Youth and Families, Children's Bureau (2016) Child maltreatment 2014. http://www.acf.hhs.gov/programs/cb/research-data-technology/statistics-research/child-maltreatment. Accessed 18 Nov 2017
27. Kleinman PL, Kleinman PK, Savageau J (2004) Suspected infant abuse: radiographic skeletal survey practices in pediatric health care facilities. Radiology 233(2):477–485
28. Chadwick DL, Chin S, Salerno C, Landsverk J, Kitchen L (1991) Deaths from falls in children: how far is fatal? J Trauma 31(10):1353–1355
29. Chadwick DL, Bertocci G, Castillo E, Frasier L, Guenther E, Hansen K, Herman B, Krous HF (2008) Annual risk of death resulting from short falls among young children: less than 1 in 1 million. Pediatrics 121:1213–1224
30. Dedouit F, Mallinger B, Guilbeau-Frugier C, Rougé D, Rousseau H, Telmon N (2011) Lethal visceral traumatic injuries secondary to child abuse: a case of practical application of autopsy, radiological and microscopic studies. Forensic Sci Int 206(1–3):e62–e66
31. Graupman P, Winston KR (2006) Nonaccidental head trauma as a cause of childhood death. J Neurosurg 104(4 Suppl):245–250
32. Hall JR, Reyes HM, Horvat M, Meller JL, Stein R (1989) The mortality of childhood falls. J Trauma 29(9):1273–1275
33. Jenny C, Isaac R (2006) The relation between child death and child maltreatment. Arch Dis Child 91(3):265–269
34. Serinelli S, Arunkumar P, Filkins JA, Gitto L (2017) Deaths due to child abuse: a 6-year review of cases in the Cook County Medical Examiner's Office. J Forensic Sci 62(1):107–118
35. World Health Organization (2013) Regional Office for Europe. European report on preventing child maltreatment. Edited by Dinesh Sethi, Mark Bellis, Karen Hughes, Ruth Gilbert, Francesco Mitis, Gauden Galea. Accessed 15 Nov 2017
36. Sidebotham P, Bailey S, Belderson P, Brandon M (2011) Fatal child maltreatment in England, 2005–2009. Child Abuse Negl 35(4):299–306
37. Spies EL, Klevens J (2016) Fatal abusive head trauma among children aged <5 years—United States, 1999–2014. MMWR Morb Mortal Wkly Rep 65:505–509
38. UNIcEF Innocenti Research Centre (2003) A league table of child maltreatment deaths in rich nations. Florence. Accessed 18 Nov 2017
39. Schnitzer PG, Gulino SP, Yuan YY (2013) Advancing public health surveillance to estimate child maltreatment fatalities: review and recommendations. Child Welfare 92(2):77–98
40. Krug EG, Mercy JA, Dahlberg LL, Zwi AB (2002) World report on violence and health. Biomedica 22(2):327–336
41. Lanzarone A, Nardello R, Conti E, Zerbo S, Argo A (2017) Child abuse in a medical setting: Case illustrations of two variants of munchausen sindrome by proxy. EMBJ 12(10):47–50
42. Argo A, Averna L, Triolo V, Francomano A, Zerbo S (2012) Validity and credibility of a child's testimony of sexual abuse: A case report. EMBJ 7(21):97–100
43. Girardet R, Lahoti S, Bolton K, Kellogg N (2016) Characteristics of cases submitted to a statewide system of child abuse experts. Child Youth Serv Rev 67:198–202
44. Lane WG, Dubowitz H, Langenberg P (2009) Screening for occult abdominal trauma in children with suspected physical abuse. Pediatrics 124:1595–1602
45. Trokel M, Discala C, Terrin NC, Sege RD (2006) Patient and injury characteristics in abusive abdominal injuries. Pediatr Emerg Care 22:700–704
46. Maguire SA, Upadhyaya M, Evans A, Mann MK, Haroon MM, Tempest V, Lumb RC, Kemp AM (2013) A systematic review of abusive visceral injuries in childhood—their range and recognition. Child Abuse Negl 37(7):430–445
47. Matthieu V, de Foort-Dhellemmes S, Desurmont M, Delestret I (2010) Confessed abuse versus witnessed accidents in infants: comparison of clinical, radiological, and ophthalmological data in corroborated cases. Childs Nerv Syst 26:637–645
48. Nimityongskul P, Anderson LD (1987) The likelihood of injuries when children fall out of bed. J Pediatr Orthop 7(2):184–186
49. Reddie IC, Bhardwaj G, Dauber SL, Jacobs MB, Moran KT (2010) Bilateral retinoschisis in a 2-year-old following a three-storey fall. Eye (Lond) 24:1426–1427
50. Reiber GD (1993) Fatal falls in childhood. How far must children fall to sustain fatal head injury? Report of cases and review of the literature. Am J Forensic Med Pathol 14(3):201–207
51. Dye DW, Peretti FJ, Kokes CP (2008) Histologic evidence of repetitive blunt force abdominal trauma in four pediatric fatalities. J Forensic Sci 53(6):1430–1433
52. Erck Lambert AB, Parks SE, Camperlengo L, Cottengim C, Anderson RL, Covington TM, Shapiro-Mendoza CK (2016) Death scene investigation and autopsy practices in sudden unexpected infant deaths. J Pediatr 174:84–90
53. Gaines BA, Shultz BS, Morrison K, Ford HR (2004) Duodenal injuries in children: beware of child abuse. J Pediatr Surg 39:600–602
54. Dashti SR, Decker DD, Razzaq A, Cohen AR (1999) Current patterns of inflicted head injury in children. Pediatr Neurosurg 31:302–306

55. Dias MS, Rottmund CM, Cappos KM, Reed ME, Wang M, Stetter C, Shaffer ML, Hollenbeak CS, Paul IM, Christian CW, Berger RP, Klevens J (2017) Association of postnatal parent education program for abusive head trauma hospitalization rates. JAMA Pediatr 171:223–229
56. Duffy SO, Squires J, Fromkin JB, Berger RP (2011) Use of skeletal surveys to evaluate for physical abuse: analysis of 703 consecutive skeletal surveys. Pediatrics 127(1):e47–e52
57. Evans KT, Knight B (1981) Forensic radiology, 1st edn. Blackwell Scientific Publications, Oxford
58. Gabaeff SC (2016) Exploring the controversy in child abuse pediatrics and false accusations od abuse. Legal Med:90–97
59. Sugar NF, Taylor JA, Feldman KW (1999) Bruises in infants and toddlers: those who don't cruise rarely bruise. Arch Pediatr Adolesc Med 153:399–403
60. Williams RA (1991) Injuries in infants and small children resulting from witnessed and corroborated free falls. J Trauma 31(10):1350–1352
61. Sheets LK, Leach ME, Koszewski IJ, Lessmeier AM, Nugent M, Simpson P (2013) Sentinel injuries in infants evaluated for child physical abuse. Pediatrics 131:701–707
62. Kellogg ND, American Academy of Pediatrics Committee on Child Abuse and Neglect (2007) Evaluation of suspected child physical abuse. Pediatrics 119:1232–1241
63. Kenney IJ (2001) Doubt, difficulties and practicalities in the diagnosis of non-accidental injury—a personal view. Imaging 13:295–301
64. 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
65. Rini MS, Colucci C, Bucci MB, Argo A (2017) Child abuse hidden in plain sight: The dentist obligations. Dental Cadmos 85(10):647–656
66. Saukko P, Knight B (2016) Fatal child abuse (Chapter 22). In: Knight's forensic pathology, 4th edn. CRC press, Taylor and Francis Group, New York, pp 475–495
67. Minns RA, Madea B, Kernbach-Wighton G (2014) Non-accidental head injury in children historical aspects of child abuse, definitions and incidence. In: Madea B (ed) Handbook of forensic medicine. Wiley Blackwell, Chichester, pp 681–701
68. Crichton KG, Cooper JN, Minneci PC, Groner JI, Thackeray JD, Deans KJ (2016) A national survey on the use of screening tools to detect physical child abuse. Pediatr Surg Int 32(8):815–818
69. Maguire S, Hunter B, Hunter L, Sibert J, Mann M, Kemp AM (2007) Diagnosing abuse: a systematic review of torn frenulum and other intra-oral injuries. Arch Dis Child 92:1113–1117
70. Levin AV, Christian CW, Committee on Child Abuse and Neglect, Section on Ophthalmology (2010) The eye examination in the evaluation of child abuse. Pediatrics 126:376–380
71. Rangarajan N, Kamalakkannan SB, Hasija V, Shams T, Jenny C, Serbanescu I, Ho J, Rusinek M, Levin AV (2009) Finite element model of ocular injury in abusive head trauma. J AAPOS 13:364–369
72. Royal College of Ophthalmology Child Abuse Working Party (1999) Child abuse and the eye. Eye (Lond) 13:3–10
73. Averna L, Argo A, Cascino FM, D'Anna T, Procaccianti P (2012) Suspected child abuse: Unusual death in western country. Suicide or homicide? EMBJ 11(6):52–54
74. Stanley P, Gwinn JL, Sutcliffe J (1976) The osseous abnormalities in Menke's syndrome. Ann Radiol 19(1):167–172
75. Lindberg DM, Shapiro RA, Blood EA, Steiner RD, Berger RP (2013) Utility of hepatic transaminases in children with concern for abuse. Pediatrics 131:268–275
76. Suzuki K, Fukushima T, Meguro K, Aoki T, Kamezaki T, Saitoh H, Enomoto T, Nose T (1999) Intracranial hemorrhage in an infant owing to vitamin K deficiency despite prophylaxis. Childs Nerv Syst 15(6–7):292–294
77. Schilling S, Wood JN, Levine MA, Langdon D, Christian CW (2011) Vitamin D status in abused and nonabused children younger than 2 years old with fractures. Pediatrics 127:835–841
78. Cranney A, Horsley T, O'Donnell S, Weiler H, Puil L, Ooi D, Atkinson S, Ward L, Moher D, Hanley D, Fang M, Yazdi F, Garritty C, Sampson M, Barrowman N, Tsertsvadze A, Mamaladze V (2007) Effectiveness and safety of vitamin D in relation to bone health. Evid Rep Technol Assess (Full Rep) 158:1–235
79. Gartner LM, Greer FR (2003) AAP clinical report on prevention of rickets and vitamin D deficiency: new guidelines for vitamin D intake. Pediatrics 111(4):908–910
80. Flaherty EG, Perez-Rossello JM, Levine MA, Hennrikus WL, American Academy of Pediatrics Committee on Child Abuse and Neglect, Section on Radiology, American Academy of Pediatrics, Section on Endocrinology, American Academy of Pediatrics, Section on Orthopaedics, American Academy of Pediatrics, Society for Pediatric Radiology (2014) Evaluating children with fractures for child physical abuse. Pediatrics 133(2):e477–e489
81. D'Eufemia P, Palombaro M, Lodato V, Zambrano A, Celli M, Persiani P, De Bari MP, Sangiorgi L (2012) Child abuse and osteogenesis imperfecta: how can they be still misdiagnosed? A case report. Clin Cases Miner Bone Metab 9(3):195–197
82. Cheung MS, Glorieux FH (2008) Osteogenesis imperfecta: update on presentation and management. Rev Endocr Metab Disord 9:153–160

83. Genetics Home Reference Osteogenesis imperfecta (2007). http://ghr.nlm.nih.gov/condition=osteogenesisimperfecta. Accessed 18 Nov 2017
84. Ojima K, Matsumoto H, Hayase T, Fukui Y (1994) An autopsy case of osteogenesis imperfecta initially suspected as child abuse. Forensic Sci Int 65(2):97–104
85. Singh Kocher M, Dichtel L (2011) Osteogenesis imperfecta misdiagnosed as child abuse. J Pediatr Orthop B 20(6):440–443
86. Maguire SA, Kemp AM, Lumb RC, Farewell DM (2011) Estimating the probability of abusive head trauma: a pooled analysis. Pediatrics 128(3):e550–e564
87. Wright JN (2017) CNS Injuries in Abusive Head Trauma. AJR 208(5):991–1001
88. Atwal GS, Rutty GN, Carter N, Green MA (1998) Bruising in non-accidental head injured children; a retrospective study of the prevalence, distribution and pathological associations in 24 cases. Forensic Sci Int 96(2–3):215–230
89. Bacopoulou F, Henderson I, Philip SG (2006) Menkes disease mimicking non-accidental trauma. Arch Dis Child 91(11):919
90. Banaschak S, Janßen K, Schulte B, Rothschild MA (2015) Rate of deaths due to child abuse and neglect in children 0–3 years of age in Germany. Int J Legal Med 129(5):1091–1096
91. Barr RG (2012) Preventing abusive head trauma resulting from a failure of normal interaction between infants and their caregivers. Proc Natl Acad Sci U S A 109(Suppl 2):17294–17301
92. Ellingson KD, Leventhal JM, Weiss HB (2008) Using hospital discharge data to track inflicted traumatic brain injury. Am J Prev Med 34(4 Suppl):S157–S162
93. Helfer RE, Slovis TL, Black M (1977) Injuries resulting when small children fall out of bed. Pediatrics 60(4):533–535
94. Hobbs CJ, Wynne JM (1996) Fractures in infancy: are the bones brittle? Curr Paediatr 6:183–188
95. Squier W, Mack J, Jansen AC (2016) Infants dying suddenly and unexpectedly share demographic features with infants who die with retinal and dural bleeding: a review of neural mechanisms. Dev Med Child Neurol 58(12):1223–1234
96. Shuman MJ, Hutchins KD (2017) Severe retinal hemorrages with retinoschisis in infant are not pathognomonic for abusive head trauma. J Forensic Sci 62(3):807–811
97. Christian CW, Block R (2009) Abusive head trauma in infants and children. Pediatrics 123:1409–1411
98. Bell E, Shouldice M, Levin AV (2011) Abusive head trauma: a perpetrator confesses. Child Abuse Negl 35:74–77
99. Bennett S, Ward M, Moreau K, Fortin G, King J, Mackay M, Plint A (2011) Head injury secondary to suspected child maltreatment: results of a prospective Canadian national surveillance program. Child Abuse Negl 35(11):930–936
100. Billmire ME, Myers PA (1985) Serious head injury in infants: accident or abuse? Pediatrics 75(2):340–342
101. Narang SK, Estrada C, Greenberg S, Lindberg D (2016) Acceptance of shaken baby syndrome and abusive head trauma as medical diagnoses. J Pediatr 177:273–278
102. American Academy of Pediatrics, Committee on Child Abuse and Neglect (2001) Shaken baby syndrome: rotational cranial injuries—technical report. Pediatrics 108:206–210
103. Caffey J (1972) On the theory and practice of shaking infants: its potential residual effects of permanent brain damage and mental retardation. Am J Dis Child 124:161–169
104. Caffey J (1974) The whiplash shaken infant syndrome: manual shaking by the extremities with whiplash induced intracranial and intraocular bleedings, linked with residual permanent brain damage and mental retardation. Pediatrics 54:396–403
105. Matshes EW, Evan RM, Pinckard JK, Joseph JT, Lew EO (2011) Shaken infants die of neck trauma not of brain trauma. Acad Forensic Pathol 1:82–91
106. Duhaime AC, Alario AJ, Lewander WJ et al (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
107. Spivack B (2001) Fatal pediatric head injuries caused by short-distance falls. Am J Forensic Med Pathol 22(3):332–336
108. Bhardwaj G, Chowdhury V, Jacobs MB, Moran KT, Martin FJ, Coroneo MT (2010) A systematic review of the diagnostic accuracy of ocular signs in pediatric abusive head trauma. Ophthalmology 117:983–92.e17
109. Biousse V, Suh DY, Newman NJ, Davis PC, Mapstone T, Lambert SR (2002) Diffusion-weighted magnetic resonance imaging in shaken baby syndrome. Am J Ophthalmol 133:249–255
110. Brogdon BG (2011) Child abuse. In: Thali M, Viner MD, Brogdon BG (eds) Brogdon's forensic radiology. CRC Press, Boca Raton, pp 255–278
111. Budenz DL, Farber MG, Mirchandani HG, Park H, Rorke LB (1994) Ocular and optic nerve hemorrhages in abused infants with intracranial injuries. Ophthalmology 101(3):559–565
112. Gabaeff SC (2011) Challenging the pathophysiologic connection between subdural hematoma, retinal hemorrhage and shaken baby syndrome. West J Emerg Med 12(2):144–158
113. Guthkelch AN (1971) Infantile subdural haematoma and its relationship to whiplash injuries. Br Med J 2:430–431
114. Kivlin JD, Simons KB, Lazoritz S, Ruttum MS (2000) Shaken baby syndrome. Ophthalmology 107:1246–1254

115. Odom A, Christ E, Kerr N, Byrd K, Cochran J, Barr F, Bugnitz M, Ring JC, Storgion S, Walling R, Stidham G, Quasney MW (1997) Prevalence of retinal hemorrhages in pediatric patients after in-hospital cardiopulmonary resuscitation: a prospective study. Pediatrics 99:E3
116. Gago L, Wegner R, Capone A, Williams GA (2003) Intraretinal hemorrhages and chronic subdural effusions: glutaric acidurie s type 1 can be mistaken for shaken baby syndrome. Retina 23(5):724–726
117. Goldman M, Dagan Z, Yair M, Elbaz U, Lahat E, Yair M (2006) Severe cough and retinal hemorrhage in infants and young children. J Pediatr 148:835–836
118. Kanter RK (1986) Retinal hemorrhage after cardiopulmonary resuscitation or child abuse. J Pediatr 108:430–432
119. Kivlin JD, Currie ML, Greenbaum VJ, Simons KB, Jentzen J (2008) Retinal hemorrhages in children following fatal motor vehicle crashes: a case series. Arch Ophthalmol 126:800–804
120. Lueder GT, Turner JW, Paschall R (2006) Perimacular retinal folds simulating nonaccidental injury in an infant. Arch Ophthalmol 124:1782–1783
121. Mei-Zahav M, Uziel Y, Raz J, Ginot N, Wolach B, Fainmesser P (2002) Convulsions and retinal haemorrhage: should we look further? Arch Dis Child 86:334–335
122. Mishra P, Naithani R, Dolai T, Bhargava R, Mahapatra M, Dixit A, Seth T, Kumar R, Saxena R (2008) Intracranial hemorrhagein patients with congenital haemostatic defects. Haemophilia 14(5):952–955
123. Pham H, Enzenauer RW, Elder JE, Levin AV (2013) Retinal hemorrhage after cardiopulmonary resuscitation with chest compressions. Am J Forensic Med Pathol 34(2):122–124
124. Sandramouli S, Robinson R, Tsaloumas M, Willshaw HE (1997) Retinal haemorrhages and convulsions. Arch Dis Child 76:449–451
125. Tyagi AK, Scotcher S, Kozeis N, Willshaw HE (1998) Can convulsions alone cause retinal haemorrhages in infants? Br J Ophthalmol 82:659–660
126. Weissgold DJ, Budenz DL, Hood I, Rorke LB (1995) Ruptured vascular malformation masquerading as battered/shaken baby syndrome: a near tragic mistake. Surv Ophthalmol 39:509–512
127. Binenbaum G, Mirza-George N, Christian CW, Forbes BJ (2009) Odds of abuse associated with retinal hemorrhages in children suspected of child abuse. J AAPOS 13:268–272
128. Binenbaum G, Forbes BJ (2014) The eye in child abuse: key points on retinal hemorrhages and abusive head trauma. Pediatr Radiol 44(4 Suppl):571S–577S
129. McCabe CF, Donahue SP (2000) Prognostic indicators for vision and mortality in shaken baby syndrome. Arch Ophthalmol 118:373–377
130. Shiau T, Levin AV (2012) Retinal hemorrhages in children. The role of intracranial pressure. Arch Pediatr Adolesc Med 166(7):623–628
131. Case ME, Graham MA, Handy TC, Jentzen JM, Monteleone JA, National Association of Medical Examiners Ad Hoc Committee on Shaken Baby Syndrome (2001) Position paper on fatal abusive head injuries in infants and young children. Am J Forensic Med Pathol 22(2):112–122
132. Duhaime AC, Gennarelli TA, Thibault LE, Bruce DA, Margulies SS, Wiser R (1987) The shaken baby syndrome: a clinical, pathological, and biomechanical study. J Neurosurg 66:409–415
133. Ludwig S, Warman M (1984) Shaken baby syndrome: a review of 20 cases. Ann Emerg Med 13:104–107
134. Christian CW, Taylor AA, Hertle RW, Duhaime AC (1999) Retinal hemorrhages caused by accidental household trauma. J Pediatr 135:125–127
135. Coats B, Binenbaum G, Peiffer RL (2010) Ocular hemorrhages in neonatal porcine eyes from single, rapid rotational events. Invest Ophthalmol Vis Sci 51:4792–4797
136. Puanglumyai S, Lekawanvijit S (2017) The importance of optic nerve sheath hemorrhage as a postmortem finding in cases of fatal abusive head trauma: a 13-year study in a tertiary hospital. Forensic Sci Int 276:5–11
137. Morad Y, Kim YM, Armstrong DC, Huyer D, Mian M, Levin AV (2002) Correlation between retinal abnormalities and intracranial abnormalities in the shaken baby syndrome. Am J Ophthalmol 134:354–359
138. Curcoy AI, Trenchs V, Morales M, Serra A, Pineda M, Pou J (2009) Do retinal haemorrhages occur in infants with convulsions? Arch Dis Child 94:873–875
139. Gleckman AM, Evans RJ, Bell MD, Smith TW (2000) Optic nerve damage in shaken baby syndrome: detection by beta-amyloid precursor protein immunohistochemistry. Arch Pathol Lab Med 124:251–256
140. Greiner MV, Berger RP, Thackeray JD, Lindberg DM (2013) Dedicated retinal examination in children evaluated for physical abuse without radiographically identified traumatic brain injury. J Pediatr 163:527–531
141. Parizel PM, Ceulemans B, Laridon A, Ozsarlak O, Van Goethem JW, Jorens PG (2003) Cortical hypoxicischemic brain damage in shaken-baby (shaken impact) syndrome: value of diffusion-weighted MRI. Pediatr Radiol 33:868–871
142. Phillips MD, Zimmerman RA (1999) Diffusion imaging in pediatric hypoxic ischemia injury. Neuroimaging Clin N Am 9:41–52
143. Ichord RN, Naim M, Pollock AN, Nance ML, Margulies SS, Christian CW (2007) Hypoxic-ischemic injury complicates inflicted and accidental traumatic brain injury in young children: the role of diffusion-weighted imaging. J Neurotrauma 24:106–118
144. Binenbaum G, Rogers DL, Forbes BJ, Levin AV, Clark SA, Christian CW, Liu GT, Avery R (2013)

Patterns of retinal hemorrhage associated with increased intracranial pressure in children. Pediatrics 132(2):e430–e434
145. Binenbaum G, Christian CW, Ichord RN, Christian CW, Ichord RN, Ying GS, Simon MA, Romero K, Pollock AN, Forbes BJ (2013) Retinal hemorrhage and brain injury patterns on diffusion-weighted magnetic resonance imaging in children with head trauma. J AAPOS 17:603–608
146. Mills M (1998) Funduscopic lesions associated with mortality in shaken baby syndrome. J AAPOS 2:67–71
147. Hymel KP, Willson DF, Boos SC, Pullin DA, Homa K, Lorenz DJ, Herman BE, Graf JM, Isaac R, Armijo-Garcia V, Narang SK, Pediatric Brain Injury Research Network (PediBIRN) Investigators (2013) Derivation of a clinical prediction rule for pediatric abusive head trauma. Pediatr Crit Care Med 14:210–220
148. Levin AV (2010) Retinal hemorrhage in abusive head trauma. Pediatrics 126:961–970
149. Hobbs CJ (1984) Skull fracture and the diagnosis of abuse. Arch Dis Child 59(3):246–252
150. Hughes LA, May K, Talbot JF, Parsons MA (2006) Incidence, distribution, and duration of birth-related retinal hemorrhages: a prospective study. J AAPOS 10:102–106
151. Prange MT, Coats B, Duhaime AC, Margulies SS (2003) Anthropomorphic simulations of falls, shakes, and inflicted impacts in infants. J Neurosurg 99:143–150
152. Keats TE, Anderson MW (2006) Atlas of normal roentgen variants that may simulate disease, 8th edn. Mosby, Philadelphia
153. Prahlow JA, Rushing EJ, Bernard JJ (1998) Death due to a ruptured berry aneurysm in a 3.5-year-old child. Am J Forensic Med Pathol 19:391–394
154. Hughes-Roberts Y, Arthurs OJ, Moss H, Set PA (2012) Post-mortem skeletal surveys in suspected non-accidental injury. Clin Radiol 67(9): 868–876
155. Jenny C, Hymel KP, Ritzen A, Reinert SE, Hay TC (1999) Analysis of missed cases of abusive head trauma. JAMA 281:621–626
156. Jenny C, Committee on Child Abuse and Neglect (2006) Evaluating infants and young children with multiple fractures. Pediatrics 118(3):1299–1303
157. John SM, Jones P, Kelly P, Vincent A (2013) Fatal pediatric head injuries: a 20-year review of cases through the Auckland coroner's office. Am J Forensic Med Pathol 34(3):277–282
158. Jones M, Darwall D, Khalid G, Prabhu R, Kemp A, Arthurs OJ, Theobald P (2017) Development and validation of a physical model to investigate the biomechanics of infant head impact. Forensic Sci Int 276:111–119
159. Jones R, Flaherty EG, Binns HJ, Price LL, Slora E, Abney D, Harris DL, Christoffel KK, Sege RD (2008) Clinicians' description of factors influencing their reporting of suspected child abuse: report of the Child Abuse Reporting Experience Study Research Group. Pediatrics 122:259–266
160. Kleinman PK, Marks SC Jr, Richmond JM, Blackbourne BD (1995) Inflicted skeletal injury: a postmortem radiologic-histopathologic study in 31 infants. Am J Roentgenol 165(3):647–650
161. Kleinman PK, Nimkin K, Spevak MR, Rayder SM, Madansky DL, Shelton YA, Patterson MM (1996) Follow-up skeletal surveys in suspected child abuse. AJR Am J Roentgenol 167:893–896
162. Karibe H, Kameyama M, Hayashi T, Narisawa A, Tominaga T (2016) Acute subdural hematoma in infants with abusive head trauma: a literature review. Neurol Med Chir (Tokyo) 56(5):264–273
163. Karmazyn B, Lewis ME, Jennings SG, Hibbard RA, Hicks RA (2011) The prevalence of uncommon fractures on skeletal surveys performed to evaluate for suspected abuse in 930 children: should practice guidelines change? Am J Roentgenol 197(1):W159–W163
164. Keeling JW, Busuttil A (2005) Paediatric forensic medicine and pathology. Edward Arnold, London
165. van Rijn RR, Spevak MR (2011) Imaging of neonatal child abuse with an emphasis on abusive head trauma. Magn Reson Imaging Clin N Am 19:791–812
166. Mack J, Squier W, Eastman JT (2009) Anatomy and development of the meninges: implications for subdural collections and CSF circulation. Pediatr Radiol 39(3):200–210
167. Matschke J, Voss J, Obi N, Görndt J, Sperhake JP, Püschel K, Glatzel M (2009) Nonaccidental head injury is the most common cause of subdural bleeding in infants <1 year of age. Pediatrics 124:1587–1594
168. Vowles GH, Scholtz CL, Cameron JM (1987) Diffuse axonal injury in early infancy. J Clin Pathol 40(2):185–189
169. Geddes JF, Hackshaw AK, Vowles GH, Nickols CD, Whitwell HL (2001) Neuropathology of inflicted head injury in children. I. Patterns of brain damage. Brain 124:1290–1298
170. Geddes JF, Vowles GH, Hackshaw AK, Nickols CD, Scott IS, Whitwell HL (2001) Neuropathology of inflicted head injury in children. II. Microscopic brain injury in infants. Brain 124:1299–1306
171. Kemp AM, Stoodley N, Cobley C, Coles L, Kemp KW (2003) Apnoea and brain swelling in nonaccidental head injury. Arch Dis Child 88:472–476
172. Smith DH, Meaney DF, Shull WH (2003) Diffuse axonal injury in head trauma. J Head Trauma Rehabil 18:307–316
173. Parker JR, Parker JC Jr, Overman JC (1990) Intracranial diffuse axonal injury at autopsy. Ann Clin Lab Sci 20(3):220–224
174. Case ME (2007) Abusive head injuries in infants and young children. Leg Med (Tokyo) 9(2):83–87
175. Case ME (2014) Distinguishing accidental from inflicted head trauma at autopsy. Pediatr Radiol 44(4 Suppl):632S–640S

176. Smit A, Cain T (2011) Assessment and interpretation of bone trauma in children (Chapter 4). In: Gall J, Payne-James J (eds) Current practice in forensic medicine. John Wiley and Sons, Hoboken, NJ
177. Harwood Nash CE, Hendrick EB, Hudson AR (1971) The significance of skull fractures in children. A study of 1,187 patients. Radiology 101(1):151–156
178. Shapiro R (1972) Anomalous parietal sutures and the bipartite parietal bone. Am J Roentgenol Radium Ther Nucl Med 115:569–577
179. Schaber B, Hart AP, Armbrustmacher V, Hirsch CS (2002) Fatal pediatric head injuries caused by short distance falls. Am J Forensic Med Pathol 23(1):101–103. author reply 103–5
180. Sty JR, Starshack RJ (1983) The role of bone scintigraphy in the evaluation of the suspected abused child. Radiology 146:369–375
181. Yason D, Jane JA, White RJ, Sugar O (1968) Traumatic subdural hematoma of infancy. Long-term follow-up of 92 patients. Arch Neurol 18(4):370–377
182. Young JY, Duhaime AC, Caruso PA, Rincon SP (2016) Comparison of non-sedated brain MRI and CT for the detection of acute traumatic injury in children 6 years of age or less. Emerg Radiol 23(4):325–331
183. Mehta H, Acharya J, Mohan AL, Tobias ME, LeCompte L, Jeevan D (2016) Minimizing radiation exposure in evaluation of pediatric head trauma: use of rapid MR imaging. Am J Neuroradiol 37:11–18
184. Ryan ME, Jaju A, Ciolino JD, Alden T (2016) Rapid MRI evaluation of acute intracranial hemorrhage in pediatric head trauma. Paediatr Neuroradiol 58(8):793–799
185. American College of Radiology (2006) Practice guidelines for skeletal surveys in children. Revised 2016 (Resolution 10) www.acr.org/SecondaryMainMenuCategories/quality_safety/guidelines/pediatric/skeletal_surveys.aspx. Accessed 18 Nov 2017
186. British Society of Paediatric Radiologists (1999) Standard for skeletal surveys in suspected non-accidental injury (NAI) in children. www.bspr.org.uk/nai.htm. Accessed 18 Nov 2017
187. Government of Western Australia (2009) (Updated 2012 and 2015) Diagnostic imaging pathways—paediatric, injury (non-accidental). www.imagingpathways.health.wa.gov.au. Accessed 18 Nov 2017
188. Kemp AM, Butler A, Morris S, Mann M, Kemp KW, Rolfe K, Sibert JR, Maguire S (2006) Which radiological investigations should be performed to identify fractures in suspected child abuse? Clin Radiol 61(9):723–736
189. Mandelstam SA, Cook D, Fitzgerald M, Ditchfield MR (2003) Complementary use of radiological skeletal survey and bone scintigraphy in detection of bony injuries in suspected child abuse. Arch Dis Child 88:387–390
190. Conway JJ, Collins M, Tanz RR, Radkowski MA, Anandappa E, Hernandez R, Freeman EL (1993) The role of bone scintigraphy in detecting child abuse. Semin Nucl Med 23:321–333
191. Haase GM, Ortiz VN, Sfakianakis GN, Morse TS (1980) The value of radionuclide bone scanning in the early recognition of deliberate child abuse. J Trauma 20:973–975
192. Jaudes PK (1984) Comparison of radiography and radionuclide bone scan in the detection of child abuse. Pediatrics 73:166–168
193. Barsness KA, Cha ES, Bensard DD, Calkins CM, Partrick DA, Karrer FM, Strain JD (2003) The positive predictive value of rib fractures as an indicator of nonaccidental trauma in children. J Trauma 54(6):1107–1110
194. Bulloch B, Schubert CJ, Brophy PD, Johnson N, Reed MH, Shapiro RA (2000) Cause and clinical characteristics of rib fractures in infants. Pediatrics 105(4):E48
195. Cadzow SP, Armstrong KL (2000) Rib fractures in infants: red alert! The clinical features, investigations and child protection outcomes. J Paediatr Child Health 36:322–326
196. Morild I, Gjerdet NR, Giertsen JC (1993) Bone strength in infants. Forensic Sci Int 60(1–2):111–119
197. Perez-Rossello JM, Feldman HA, Kleinman PK, Connolly SA, Fair RA, Myers RM, Gordon CM (2012) Rachitic changes, demineralization, and fracture risk in healthy infants and toddlers with vitamin D deficiency. Radiology 262:234–241
198. Kleinman PK (1998) Diagnostic imaging of child abuse, 2nd edn. Mosby, St. Louis, MO
199. Kleinman PK (2008) Problems in the diagnosis of metaphyseal fractures. Pediatr Radiol 38(13):388–394
200. Gill JR, Andrew T, Gilliland MGF, Love J, Matshes E, Reichar RR (2014) National Association of Medical Examiners Position Paper: recommendations for the postmortem assessment of suspected head trauma in infants and young children. Acad Forensic Pathol J 4(2):206–213
201. Leventhal JM, Thomas SA, Rosenfield NS, Markowitz RI (1993) Fractures in young children. Distinguishing child abuse from unintentional injuries. Am J Dis Child 147(1):87–92
202. Piteau SJ, Ward MG, Barrowman NJ, Plint AC (2012) Clinical and radiographic characteristics associated with abusive and nonabusive head trauma: a systematic review. Pediatrics 130(2):315–323
203. Plunkett J (2001) Fatal pediatric head injuries caused by short-distance falls. Am J Forensic Med Pathol 22(1):1–12
204. Roach JP, Acker SN, Bensard DD, Sirotnak AP, Karrer FM, Partrick DA (2014) Head injury pattern in children can help differentiate accidental from non-accidental trauma. Pediatr Surg Int 30(11):1103–1106
205. Shugerman RP, Paez A, Grossman DC, Feldman KW, Grady MS (1996) Epidural hemorrhage: is it abuse? Pediatrics 97(5):664–668

206. Worlock P, Stower M, Barbor P (1986) Patterns of fractures in accidental and non-accidental injury in children: a comparative study. Br Med J (Clin Res Ed) 293(6539):100–102
207. Reece RM, Sege R (2000) Childhood head injuries: accidental or inflicted? Arch Pediatr Adolesc Med 154(1):11–15
208. Kemp AM, Dunstan E, Harrison S, Morris S, Mann M, Rolfe K, Datta S, Thomas DP, Sibert JR, Maguire S (2008) Patterns of skeletal fractures in child abuse: systematic review. BMJ 337:a1518
209. Leventhal JM, Edwards GA (2017) Flawed theories to explain child physical abuse: what are the medical-legal consequences? JAMA 318(14):1317–1318
210. Reece RM (1994) Child abuse: medical diagnosis and management. Lea & Febiger, Philadelphia
211. Levey EJ, Gelaye B, Bain P, Rondon MB, Borba CP, Henderson DC, Williams MA (2017) A systematic review of randomized controlled trials of interventions designed to decrease child abuse in high-risk families. Child Abuse Negl 65:48–57
212. Schwend RM, Werth C, Johnston A (2000) Femur shaft fractures in toddlers and young children: rarely from child abuse. J Pediatr Orthop 20(4):475–481
213. Giardino AP, Alexander R (2005) Child maltreatment: a clinical guide and reference, 3rd edn. GW Publishing Inc., St. Louis
214. Perkins R, Skirvin A (1987) Callus formation in the rate of healing of femoral fractures in patients with head injuries. J Bone Joint Surg Br 69:521–524
215. Spencer RF (1987) The effect of head injury on fracture healing. A quantitative assessment. J Bone Joint Surg Br 69B(4):525–528
216. Prosser I, Maguire S, Harrison SK, Mann M, Sibert JR, Kemp AM (2005) How old is the fracture? Radiological dating of fractures in children: a systematic review. AJR Am J Roentgenol 184:1282–1286
217. Offiah AC, Hall CM (2009) Radiological atlas of child abuse. Radcliffe Publishing, Oxford and New York