Rob A.C. Bilo
Simon G.F. Robben
Rick R. van Rijn
Editors

Forensic Aspects of Paediatric Fractures

Differentiating Accidental Trauma from Child Abuse

Second Edition

Springer

Forensic Aspects of Paediatric Fractures

Rob A. C. Bilo • Simon G. F. Robben
Rick R. van Rijn
Editors

Forensic Aspects of Paediatric Fractures

Differentiating Accidental Trauma from Child Abuse

Second Edition

Springer

Editors
Rob A. C. Bilo
Veilig Thuis Rotterdam Rijnmond
Rotterdam, The Netherlands

Rick R. van Rijn
Department of Radiology and Nuclear Medicine
Amsterdam UMC location University
of Amsterdam
Amsterdam, The Netherlands

Department of Forensic Medicine
Netherlands Forensic Institute
The Hague, The Netherlands

Simon G. F. Robben
Paediatric Radiology
Maastricht University Medical Centre
Maastricht, The Netherlands

ISBN 978-3-031-12040-4 ISBN 978-3-031-12041-1 (eBook)
https://doi.org/10.1007/978-3-031-12041-1

This Springer imprint is published by the registered company Springer Nature Switzerland AG
The registered company address is: Gewerbestrasse 11, 6330 Cham, Switzerland

Foreword

Almost every child experiences a bone fracture in their lifetime. There are numerous situations that can lead to an accidental bone fracture. The major challenge for physicians is to recognize when the bone fracture is no longer an accidental event. What aspects indicate a forensic cause of the bone fracture? No medical textbook is available that answers this question as exhaustively as this book. The chapters are arranged according to the site of the bone fracture, and thus fit seamlessly with clinical practice. Several international radiologists, pediatricians, and forensic physicians, all experts in the field, have contributed. Differential diagnostic aspects are covered extensively, including congenital variants that can put physicians as well as radiologists on the wrong track. The book is packed with examples and is educationally well constructed. The book concludes with instructions for reading and interpreting medical literature including translations to Bayesian formulation. This book is an essential reference work in every emergency room and pediatric department and a textbook of the highest quality for every (pediatric) radiologist.

Elise van de Putte

Pediatrician Specialized in Social Pediatrics
Dutch Expertise Center for Child Abuse (DECCA)
Utrecht, The Netherlands

The original version of this book was inadvertently published without the foreword by Prof. Wilma Duijst. The foreword has now been included in the book.

Foreword

‘Break a leg’ is a common saying. The origin of this saying is unknown. Some state it refers to the world of dancers, actors and performers. The saying is meant to wish a person good luck. Others state the saying goes back to the seventeenth and eighteenth centuries. Breaking a leg meant having a child born out of wedlock. In this dark world we called this child ‘a bastard child’. This book is about the dark side of breaking a leg or whatever other bone in the body of a child. The principal question, after a broken bone is detected in a child, is whether the bone is broken due to a disease, due to bad luck (the wish did not work out well) or due to ‘the bastard child’ who annoyed his caregivers (the child is physically abused). Being able to make the distinction between these three options is crucial for paediatricians, forensic physicians, public prosecutors and judges. Making the distinction has consequences for the medical treatment, the decisions about the safety of the child and even the place for the child to live. And as having a disease or having bad luck is not a criminal offence, this distinction is crucial to determine whether someone has to be prosecuted for child abuse and if so who. In criminal court the distinction is the basis for the decision if someone is to be found guilty and has to be punished. To be able to make these decisions the professionals need scientific facts. This book provides these facts. From now on, making decisions about a broken bone in a child does not depend on whether the wish ‘break a leg’ uttered towards professionals worked out.

This book started out as a 200-page book and has now reached the status of a handbook in this field of expertise. I’m honoured to present this book to my forensic and judicial colleagues.

Wilma Duijst

Forensic Medicine and Criminal Law
Maastricht University
Maastricht, The Netherlands

Contents

Child Abuse, Non-Accidental Trauma, and Inflicted Injuries

1

Rob A. C. Bilo, Marloes E. M. Vester, Arjo A. J. Loeve, and Rian A. H. Teeuw

Contents

R. A. C. Bilo (✉)
Veilig Thuis Rotterdam Rijnmond (Center for the Reporting of Child Abuse, Domestic Violence and Elder Abuse), Rotterdam, The Netherlands

M. E. M. Vester
Forensic Physician, Voorhout, The Netherlands

A. A. J. Loeve
Department of Biomechanical Engineering, Faculty of Mechanical, Maritime and Materials Engineering, Delft University of Technology, Delft, The Netherlands
e-mail: A.J.Loeve@tudelft.nl

R. A. H. Teeuw
Department of Paediatrics, Amsterdam UMC, University of Amsterdam, Amsterdam, The Netherlands
e-mail: a.h.teeuw@amsterdamumc.nl

R. A. C. Bilo et al. (eds.), *Forensic Aspects of Paediatric Fractures*, https://doi.org/10.1007/978-3-031-12041-1_1

1.1 Defining Child Abuse

1.1.1 Child Abuse

There is no universally accepted definition of child abuse or any of its subtypes. Definitions will vary according to the circumstances under which the definitions are used, e.g. sociological, medical, political, cultural, scientific, or legislative. Definitions may also vary from concise and to the point, to comprehensive and more descriptive.

In this book we will use the definition of the World Health Organization (WHO), which was first drafted in 1999: 'Child abuse constitutes all forms of physical and/or emotional ill-treatment, sexual abuse, neglect or negligent treatment, or commercial or other exploitation, resulting in actual or potential harm to the child's health, survival, development or dignity in the context of a relationship of responsibility, trust or power' [1]. This definition includes both active (= abuse) and passive (= neglect) behaviour of parents and others towards children.

The terms 'non-accidental trauma' and 'inflicted injuries' will be defined and discussed in Sect. 1.1.6.

1.1.2 Physical Abuse

Physical child abuse is defined as the deliberate physical violent behaviour towards a child, committed by parents, caregivers, and other individuals (such as siblings, acquaintances, and teachers) that are in a position of relationship of responsibility, trust, or power towards the child. This leads to actual or potential physical injury that is the result of interactions (acts), or a lack of interactions (omissions), which should reasonably be within the control of a parent or person in a position of responsibility, power, or trust [1, 2]. Although the physical violent behaviour is always deliberate, the resulting physical injuries are often not intended to occur.

Physical abuse also includes the deliberate poisoning or suffocation of a child, but these acts can also take place with the motive to fabricate or induce illness in the child (Sect. 1.1.6) [2].

The severity of actions may range from a single incident without or with visible physical consequences to frequent physically aggressive behaviour, such as beating, punching, kicking, biting, and burning with or without visible injuries and/or scars, up to life-threatening and sometimes fatal consequences.

1.1.3 Neglect

Neglect is the failure to provide for the needs of the child in every aspect of a child's life: health, education, emotional development, nutrition, shelter, and safe living conditions, within the context of resources generally considered available to the family or carers. This includes the failure to properly supervise and protect children from harm [3]. The actions or the omissions of the parent/caregiver cause, or have a high probability of causing, harm to the child's health or physical, mental, spiritual, moral, or social development.

In physical neglect, the parent/caregiver is unable or unwilling to provide (with regard to the physical needs of the child) minimal adequate care (concerning, e.g. food, shelter, hygiene, sleep, and clothing) and suitable medical, dental, and mental health care. The parent/caregiver may also not take suitable precautions to ensure the safety of the child indoors and outdoors according to the nature and development of the child (no supervision, unsanitary, unsafe or unhealthy environment, no substitute care).

Other types of neglect are emotional and educational neglect. Emotional neglect occurs when parents/caregivers fall short in responsiveness and giving positive attention to the child. Allowing the child to witness violence between parents is also considered to be emotional neglect, but is sometimes also defined as emotional/psychological abuse [4].

Educational (or normative) neglect is defined as being unable or unwilling to have minimal concerns about the socialization of the child including the provision of suitable education for the child. It also includes exposing or involving the child in illegal acts that induce or promote delinquency or

antisocial behaviour in the child [4]. Educational neglect also includes not exercising appropriate and sufficient parental authority or not offering sufficient structure, while raising the child [5].

1.1.4 Emotional/Psychological Abuse

Emotional or psychological abuse is described as the systemic destruction of a person's self-esteem and/or sense of safety, acceptance and respect, increasing autonomy and clear boundaries, often occurring in relationships where there are differences in power and control [6]. There is an atmosphere, in which a child is bullied, hurt, and belittled. Psychological abuse includes threats of harm or abandonment, humiliation, deprivation of contact, isolation, and other psychologically abusive tactics and behaviours [7].

Synonyms of psychological abuse are, e.g. emotional abuse, verbal abuse, mental cruelty, intimate terrorism, and psychological aggression. When the abuse occurs in a residential care setting, it is often called systemic or institutional abuse [7].

1.1.5 Sexual Abuse

The WHO defines child sexual abuse as the involvement of a child in sexual activities that he or she does not fully comprehend, is unable to give informed consent to, or for which the child is not developmentally prepared and cannot give consent, or that violates the laws or social taboos of society [8]. According to the WHO child sexual abuse is evidenced by this activity between a child and an adult or another child who by age or development is in a relationship of responsibility, trust or power, the activity being intended to gratify or satisfy the needs of the other person. This may include but is not limited to:

- The inducing or forcing of a child to engage in any unlawful sexual activity
- The exploitative use of a child in prostitution or other unlawful sexual practices
- The exploitative use of children in pornographic performance and materials

1.1.6 Fabricated or Induced Illness by Parents/Caregivers

Fabricated or induced illness by parents/caregivers is defined as the deliberate fabrication or induction/production of physical or psychological symptoms in a child by a parent or caregiver [9, 10]. Fabricated or induced illness by carers (FII) (UK terminology) was formerly known as Munchausen syndrome by proxy [11]. It is now also known as paediatric condition falsification (PCF)/factitious disorder by proxy/medical child abuse [12–14].

In this form of child abuse the child has suffered, or is likely to suffer, significant harm through the deliberate action of its parent or caregiver. The symptoms of the child are attributed by the parent or caregiver to an illness or another medical cause [13, 15]. There are three ways (not mutually exclusive) of a parent or caregiver fabricating or inducing an illness in a child:

- Fabrication of signs and symptoms, including fabrication of the child's past medical history and the past medical history of other family members, including the perpetrator's medical history
- Fabrication of signs and symptoms and falsification of hospital charts, records, letters and documents, and specimens of bodily fluid
- Induction of symptoms/illness by a variety of means, e.g.:
  - The administration of prescribed and unprescribed medication
  - The administration of substances, that are freely available at home, which can be given to the child or applied to its skin
  - Starving the child leading to malnutrition
  - Smothering

An existing diagnosed illness in a child does not exclude the possibility of induced illnesses. The presence of a real existing illness can act as a stimulus for the abnormal behaviour and also provide the parent with opportunities for inducing or aggravating symptoms.

In order to determine whether the child's signs and symptoms are fabricated or induced, it is not necessary to have insight into the perpetrator's motives for the fabrication or induction. The diagnosis of fabricated illness is based upon the investigations of the child itself and the complete (medical) history. Potential motives of the suspected perpetrator can be evident or obscure and are of relevance to the treatment of the perpetrator him- or herself, which of course is crucial for the final prognosis of the safety and health of the child.

1.2 Epidemiology

Lord Laming stated in his 2003 report on the occasion of the violent death of Victoria Climbié about the incidence and prevalence of child abuse: '*I have no difficulty in accepting the proposition that this problem (deliberate harm to children) is greater than that of what are generally recognized as*

common health problems in children, such as diabetes or asthma' [16]. The exact incidence and prevalence of child abuse is not known. One important reason for this is that in nearly every study to establish the incidence and prevalence, researchers use their own definition. If a 'broad definition' is used, the incidence and prevalence will be higher than in case of a much narrower definition.

In the Netherlands (more than 17 million inhabitants with 180,000 births per year), the most recent and third Netherlands' Prevalence study on Maltreatment of children and youth (NPM-2017) showed that in 2017 90,000–127,000 children (26–37/1000 children) were recognized by professionals as victims of child abuse [17]. A study amongst primary and secondary school students showed that 270 of 1000 of primary school students had ever experienced a form of child abuse and 123 of 1000 secondary school students had been victimized during the secondary school period [18]. During the COVID pandemic the Netherlands instituted a nationwide lockdown, a study by the same group showed that in a three-month period an estimated 40,000 children (95% CI: 24.533–54.237 or 8–19/1000 children) were recognized as victims of child abuse [19]. This represents a significant increase and supports the theory that child abuse is related to stressful events. In these studies definitions of child abuse, comparable to the WHO definitions, were used.

More exact figures will probably never be known, because there will always be a dark number of unrecognized, and therefore unknown, cases, even with an accurate definition. A large review by Hillis et al. on the international prevalence of child abuse violence over the then past year included 38 articles from 96 countries found, depending on the used definition, a minimum prevalence of 50% for Asia, Africa, and Northern America [20]. A study based on United States national child protective services records (2003–2016) combined with census data led the authors to conclude that before the age of 12 years 32.4% of children were reported at least one time [21]. In this study, the probability of subsequent reports were 13.71% for 2 reports, 7.57% for 3 reports, 4.50% for reports, 2.80% for 5 reports, and 1.79% for 6 reports. Not surprisingly the data showed that children with more prior reports were more likely to be reported again.

Among professionals in the field of child abuse there is consensus that whatever the exact incidence and prevalence numbers might be, it is one of the main threats to the well-being of children.

1.3 Clinical Aspects

Each subtype of child abuse can have negative health consequences. In Table 1.1 an overview is given of some of the health consequences of child abuse, as summarized by the WHO and by Felitti et al. [22, 23]. On the one hand, these consequences are the immediate effect of the aggressive or negligent behaviour and are visible as, e.g. physical injuries or sexually transmitted diseases. On the other hand, these consequences are a delayed effect of the child abuse, manifesting in adolescent or adult life, like alcohol and drug abuse or delinquent, violent, and other risk-taking behaviour (Fig. 1.1).

Moreover, not all physical injuries will be noticed when they are present or, when they are noticed, be recognized as such.

Table 1.1 Health consequences of child abuse [22, 23]

Physical abuse	• Abdominal/thoracic injuries • Brain injuries/injuries to the central nervous system • Bruises and contusions • Burns and scalds • Disability • Fractures • Lacerations and abrasions • Ocular damage
Physical neglect	• Failure to thrive • Dental decay • Burns and scalds • Severe diaper rash • Consequences of inadequate medical care • Drowning
Sexual and reproductive	• Mucous membrane damage • Reproductive health problems (e.g. infertility) • Sexual dysfunction • Sexually transmitted diseases, including HIV/AIDS • Unwanted pregnancy • Constipation, enuresis, abdominal pain • Pelvic floor hypertrophy
Psychological and behavioural	• Alcohol and substance abuse • Cognitive impairment • Delinquent, violent, and other risk-taking behaviours • Depression and anxiety • Developmental delays • Eating and sleep disorders • Feelings of shame and guilt • Hyperactivity • Poor relationships • Poor school performance • Low self-esteem • Post-traumatic stress disorder • Psychosomatic disorders • Suicidal behaviour and self-harm
Other longer-term health issues	• Cancer • Chronic lung disease • Fibromyalgia • Irritable bowel syndrome • Ischemic heart disease • Liver disease • Obesity • Diabetes

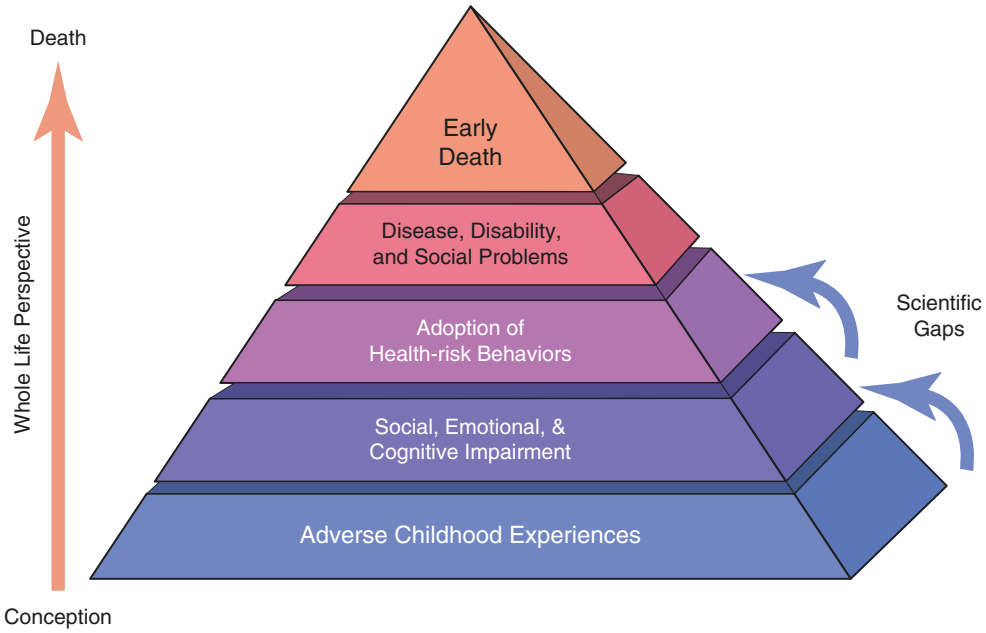


Fig. 1.1 The ACE Pyramid represents the conceptual framework for the ACE Study, which has uncovered how adverse childhood experiences are strongly related to various risk factors for disease throughout the lifespan (source: C. Whitfield, M.D., Centers for Disease Control and Prevention, http://www.cdc.gov/violenceprevention/acestudy/pyramid.html)

1.4 Defining Trauma and Injury

Trauma and injury are often used as synonyms both in daily practice and in the medical literature. This is confusing and in a forensic setting incorrect:

- A trauma is an event which can result in an injury.
- An injury is any wounding or physical damage (physical harm, bodily injury, physical injury) that results from a trauma.

It is important to recognize that cutaneous injuries may indicate damage to underlying structures, such as the skeleton, intra-abdominal organs, or intracranial (neurological) injuries.

1.5 Cause of Injury

1.5.1 Introduction

The cause (or mechanism) of an injury refers to the way a body part (skin, mucosa, or any other tissue: muscles, organs, and bones) is damaged. The cause of an injury should be differentiated from the manner (or mode) of an injury. The manner of an injury describes the circumstances under which the injury was sustained or the injury event happened (see Sect. 1.1.6).

An injury is caused by the (sudden) subjection of the body or body parts, e.g. the skin, the mucous membranes, the skeleton or internal organs, to amounts of energy that exceed the threshold of physiological tolerance. In other words, it concerns the exposure of the body to amounts of energy in which the loading (the process of transfer of energy during the contact) exceeds the maximum capacity of the body or parts of the body (and/or adjacent tissues) to absorb the transferred energy. This may occur with or without externally visible damage to the skin or the mucous membranes and with or without signs of damage to the skeleton or internal organs, e.g. fractures, intracranial bleeding, or intra-abdominal injuries [3, 24, 25].

Transfer of energy, leading to injuries, can happen due to mechanical trauma or in non-mechanical trauma (contact or near contact with physical agents) (Sect. 1.6–1.10) [25].

The nature of an injury, in other words the appearance, extent, and severity of an injury, not only depends on the type of trauma (mechanical or non-mechanical), but also on [26]:

- The amount of energy that is transferred during the contact
- The rate of energy transfer
- The duration of the exposure to the transferred energy
- The exposed body part(s)
- The size of the body surface over which the energy is distributed
- The nature of the ‘weapon’ used
- The structures under the skin (e.g. subcutaneous fat, muscle, bone, or internal organs)
- The age and (physical) developmental stage of the child
- The health status of the child and the presence of diseases

In addition to the foregoing, one should be aware that physical injuries in all body parts (including the skin and the skeleton) can also be caused by a lack, or excess, of one of the vital elements (e.g. oxygen, trace elements, vitamins, water, or warmth) [3, 24]. A lack or an excess of vital elements may even lead to death. An excess can be seen, for example in salt poisoning, hypervitaminosis, water poisoning (hyperhydration), or overheating (hyperthermia, heat stroke) [27–32].

1.5.2 Mechanical Trauma

Mechanical (blunt- or sharp-force) trauma is caused by transfer of energy due to static or dynamic loading [33, 34]. The difference between static and dynamic loading is best exemplified by the following statement of Burton: *'Consider the effect of a stationary bullet resting on your chest, compared to the effect of a moving bullet striking your chest. The stationary bullet exerts a static load on your chest. A moving bullet exerts a dynamic load'* [35].

1.5.2.1 Static Loading

In mechanics, a static load is defined as a non-varying load, e.g. a non-varying force exerted on a surface by the weight of a mass at rest. For that reason, a static load in mechanics is also known as a dead load.

In injury biomechanics and in forensic medicine, the terms static load and static loading are used slightly different. Static loading is defined as a relatively slow exertion (loading) of forces on body parts over a protracted period of time. In the paediatric literature, this protracted time is defined as more than 200 ms [36, 37]. Static loading occurs when a body part is squeezed and/or compressed, which may lead to injuries of soft tissues, bony tissues or sometimes to underlying tissues, like the intracranial or abdominal content. Just like in dynamic loading, the effect of static loading can be focal and limited to the point of compression or more extended in which not only the superficial, but also the underlying layers are damaged.

The type, severity, dimensions, and appearance of the injuries caused by static loading are not only determined by mass, weight, gravity, or force but also by:

- The surface of the compressing object (flat, curved, patterned, blunt, sharp, more or less flexible) and of the compressed skin (flat, curved, underlying tissues—connective tissue, fat, bone)
- The size of the contact surface between the compressing object and of the compressed skin
- The source that determines the applied load (gravity, human behaviour, accidental wedging)
- The load exerted on the skin

Gradual or repetitive build-up of loading or changes in loading of the skin during the time of exposure may happen, because the load that is exerted is not only determined by gravity but also by the amount of changes in pressure actively exerted on the skin, e.g. if a person is grabbed by another person, or if a person is overrun by a very slow driving car, or by movements of the person who is subjected to the load and actively resists the loading or passively changes position. This will lead to changes in the energy that is transferred from the compressing object to skin during the time of exposure (contact time) and will influence the final effect of the static load on the skin.

In physical assaults, static loading mostly will be caused by blunt-force trauma due to, e.g. compression or bending, but static loading of the skin may also be caused by pulling or twisting of the skin. Static loading may also happen in sharp-force trauma, when a sharp-pointed object (e.g. a needle) is first held and then pressed against the skin (often resulting a penetrating trauma of the skin and sometimes of the underlying tissues).

If static loading in blunt-force trauma leads to injuries of the skin and underlying tissues, including the vessels (subcutaneous veins or capillaries) and bones, the injuries are the result of direct damage by the distorting force (compressing, pulling, twisting) at the site of the distortion. The integrity of the skin and the underlying tissues may or may not be compromised during static loading.

The level of static loading is often expressed in terms of low and high pressure. These terms however are subjective and relative with a grey area between low and high pressure and depending on the context in which these terms are used. Pressure is the ratio between the exerted force and the exposed surface, in other words the distribution of force per square centimetre. The risk of injuries to the skin and the underlying tissues, for example bones or visceral organs, increases with increasing pressure (proportional relation).

In case of low-pressure static loading, e.g. in normal daily activities like handling a child, the loading caused by normal handling will not result in bruising or other skin injuries and usually not in injuries of underlying tissues, like bones. However this only accounts if there are no complicating factors added to the low-pressure loading, which decrease the capacity of the skin and the underlying tissues to absorb the transferred energy, e.g. a coagulation disorder, vascular disorders or disorders of the connective tissue, or the use of medication, e.g. corticosteroids and anticoagulants. Also, prolonged exposure to low-pressure loading, as sometimes can be seen in tight clothing, may lead to superficial skin injuries, like bruises (e.g. compression or torsion of the skin) and superficial abrasions (compression in combination with friction and shearing).

High-pressure static loading, as can be seen in accidents, e.g. resulting from prolonged wedging in motor vehicle accidents or in non-accidental trauma (e.g. in pinching, grabbing, or tying), may result in more extensive bruising or other injuries of the skin or compression injuries of underlying tissues, e.g. skull and rib fractures.

1.5.2.2 Dynamic Loading

Dynamic loading is the fast application or change of forces over a shorter period, e.g. less than 200 ms, often even less than 50 ms. Dynamic loading can be divided in dynamic impact and dynamic impulse loading.

Dynamic impact loading is defined as the application of an external force with a certain mass and velocity during a relatively short period of contact between the object and the skin/body. An injury caused by dynamic impact loading is also referred to as a kinetic injury: an injury caused by the exchange of energy during motion, leading to a transfer of energy during collision as long as the contact continues between the human body and the colliding object, e.g. an object or (parts of) another human body. Injuries due to dynamic impact loading are the result of a single impact or a series of impacts. In physical assaults, dynamic impact loading may occur in non-penetrating or penetrating blunt force or sharp force.

In dynamic impact loading, the following situations can occur during the impact between a body part and an object:

- The impacted body part is stationary, while the impacting object moves against the body part.
- The impacting body part is moving, while the impacted object is stationary.
- Both the body part and the object are moving either in the same direction with different speeds or in opposite directions.

The impact loading results in the body part changing shape with possible damage to the soft tissues, bony tissues, or the underlying tissues.

Dynamic impulse loading is the result of rapid (often repetitive) movements without (external) impact, but with a rapid alternation of acceleration and deceleration (inertial trauma), as can be seen in abusive head trauma or in some abdominal injuries due to shaking. Dynamic impulse loading has never been described as the cause of skin injuries or fractures of the calvarium, the base of the skull or orofacial bones. However, dynamic impulse loading can lead due to the rapid alternation to specific fractures, e.g. classical metaphyseal lesions (see Chap. 12). Dynamic impulse loading can occur combined with static loading: e.g. during shaking compressive forces on the ribs can result in rib fractures, but also in bruising on the thoracic wall.

1.5.3 Non-mechanical Trauma: Physical Agents

In non-mechanical trauma, injuries are the result of the transfer of energy during a direct contact of or a contactless transfer to the skin and/or other body parts, in which the skin and/or the other body parts are exposed to extremes in temperature (heat and cold) or to chemical or physical agents (acidic and alkaline chemicals, electricity, microwaves, and radiation). In a contactless transfer, the risk of injuries usually decreases with increasing distance between the body and the energy source, e.g. with radiation from an infrared heater. Chemical or physical agents may have a similar effect on the skin and the subcutaneous tissues as heat or cold: cutaneous burns, resembling burns, due to extremes in temperature (see Sect. 1.7.5).

1.5.4 Direct and Underlying Cause

While determining the cause of an injury, one should differentiate between the direct cause and the underlying cause. According to the Centers for Disease Control and Prevention, the direct cause is what produces the actual physical harm and the underlying cause is what started the chain of events that led to the actual physical harm (the injury) [38]. The direct and underlying causes can be the same or different.

If a child sustained bruising on the forehead after he or she stumbled while walking and hitting his or her head on a coffee table, the fall, caused by the stumbling, is the underlying cause (the action that started the chain of events leading to the injury, i.e. stumbling and falling), leading to the contact with the table, which is the direct cause of the bruising (the action that caused the actual physical harm, i.e. impact trauma). The cause of a certain injury does not have to say anything about the manner of that injury. Determining that the child fell and hit the table (causing a bruise to the forehead) does not define under which circumstances the stumbling took place. The stumbling may have been caused, for example by the unstable walking of the child (developmental level), just being wild during play, being pushed by another child during play or during a fight, or intentionally or unintentionally being pushed by an adult. The resulting injury will often be the same, because it is caused by an impact trauma (cause of injury), despite the different circumstances (manner of injury).

1.6 Manner of Injury

As stated before in Sect. 1.5.1, the manner of injury describes the circumstances under which the injury was sustained or the injury event happened. The manner can be divided in three types of circumstances [39]:

- Accidental trauma (often used synonyms: non-inflicted, non-intentional or unintentional, or non-abusive)
- Non-accidental trauma (often used synonyms: inflicted, intentional, deliberate, abusive, or negligent)
- Unexplained trauma (undetermined)

Using the term ‘accidental’ is factually misleading. It may suggest that the events leading to the injury were inevitable

and could not be avoided. In case of minors, especially younger children, most accidents (perhaps even all) are, at least in retrospect, preventable and sometimes even predictable [40]. 'Non-inflicted' is also a misleading term: the use of this term implies that the injury was not inflicted and that nobody was involved in causing the injury, although, for example in a motor vehicle accident, the driver is involved in causing the injury and the injury was inflicted. 'Non-intentional or unintentional' are more neutral terms: the injury event happened but there was no intention for it to happen, despite the fact that the incident could have been prevented if the necessary precautions had been taken. Despite the fact that the term 'accidental' is factually misleading, in this book this term will be used to describe the circumstances in which injuries resulted from an 'unintended, non-abusive' incident.

Using the term 'intentional' as synonymous for 'non-accidental', 'inflicted', or 'abusive' in the evaluation of injuries is also misleading. It would mean that the motivation of the perpetrator ('intentional: willingly, consciously, deliberately') to inflict an injury can be determined by evaluating certain characteristics of the injury. This is almost never possible based purely on the findings during the physical examination, except probably for pinch marks, bite-marks, or multiple stab wounds. In a physical assault, the action that led to an injury is almost always the result of a conscious decision of the perpetrator and therefore intended, but it is rarely the intention to inflict (serious) injuries. 'Inflicted' is a more appropriate term, because it states that the injury was the result of an action by a person (and perhaps an animal in bite-marks), without saying anything about the intention ('to inflict = to give or impose something unpleasant and unwanted, for example to inflict serious injuries'). In this book, the terms 'non-accidental' or 'inflicted' will be used to describe the circumstances in which injuries resulted from an abusive incident (an event resulting from violent behaviour of an adult towards a child).

The evaluation of the circumstance under which an injury or injuries were sustained, is a (forensic) medical task, in which injury characteristics and the patient's or parent's (clinical) history can be used to differentiate to a certain extent between non-accidental trauma/inflicted injuries, accidental trauma/non-inflicted injuries, and unexplained injuries. Beforehand disease, which can mimic injury, has to be ruled out. In each step of the evaluation, a carefully taken and comprehensive (clinical) history may add information, which may enable differentiation (see also Sect. 1.9). The following aspects should be taken into account during the evaluation, concerning the manner:

- The age and developmental level of the child
- Explanations given by the child (if possible), the parent(s), and others (people involved in the case, regardless of their background: professionally involved or not)
- Other (historical) physical signs and symptoms: e.g. stress-related physical signs or older bruises and/or other soft tissue injuries, fractures, head injury, and abdominal injury
- Past medical history and family history
- Additional findings during physical examination/forensic medical examination
- Findings during laboratory examination: e.g. testing of blood tests (e.g. blood clotting) or urine
- Findings during imaging: skeletal survey, CT/MRI, nuclear medicine, ultrasonography (brain, abdomen, total body)
- Assessment by social work/child protection services
- Inquiry by the police
- Data from (preferably evidence based) clinical and forensic paediatric literature about the differential diagnosis of physical findings and the possibility and probability of certain injuries in certain circumstances
- In case of suspicions of fatal abuse: findings during a forensic autopsy

Even if all medical findings and other data are properly evaluated, including an extensive (clinical) history, a comprehensive clinical examination, and/or forensic autopsy, it will not always be possible to draw reliable conclusions about the manner of the injury. For example, a young child (age 3) becomes a victim of a house fire and dies. The cause of the injuries and of the death of the child will probably be clear after autopsy, e.g. injuries due to heat and/or carbon monoxide intoxication. In other words, the injuries and death of the child were sustained due to the house fire. The following circumstances, under which the child sustained the injuries and died, should be considered to determine whether the house fire occurred in accidental or non-accidental circumstances:

- The fire resulted from a suddenly and unexpectedly malfunctioning of an electrical device. The parents were at home and tried everything to save the child. The manner of the injuries and the dying of the child can be defined as accidental and unintended.
- The child was left alone in house without any supervision and had easy access to matches. The parents were already warned by child protection to never leave the child alone at home. The injuries and the dying could have been prevented if the child had been taken care of in a proper way. The manner will be non-accidental, due to negligence.
- The fire was started deliberately. The manner of death will be manslaughter, if the death of the child was not intended to happen, or homicide, if the death of the child was intended to happen.

1.7 Cutaneous Injuries

1.7.1 Introduction

The most injured organ due to physical assault is the skin, irrespective of the age of the assaulted victim. The skin is also the easiest organ to examine in case of a suspicion of

Table 1.2 Cutaneous injuries caused by mechanical trauma [45]

Blunt-force trauma	Resulting injuries
• Closed injuries	• Erythema
	• Bruises
	• Petechiae
• Open injuries	• Abrasions
	• Lacerations and avulsions
	• Injuries due to a blunt penetrating trauma
Sharp-force trauma	**Resulting injuries**
• Open injuries	• Incisions and stab wounds
	• Gunshot wounds

non-accidental trauma. In paediatric patients, bruises are the most common injuries in physical assaults, followed by other cutaneous injuries, like abrasions and burns [41–44]. However, bruises and other cutaneous injuries due to non-accidental trauma are also common injuries due to accidental circumstances. An overview of cutaneous injuries resulting from mechanical trauma is given in Table 1.2.

When suspicious cutaneous findings are evaluated, a number of findings are seen more often in inflicted than in accidental injuries:

- Multiple injuries in various stages of healing
- Injuries of various kinds, e.g. bruises, abrasions, and burns
- Injuries on different body surfaces: front-back, left-right)
- Injuries with a clearly recognizable pattern, e.g. a hand-print or an iron
- Self-defence injuries: injuries that arise because the child adopts a body posture that reduces the risk of serious injury during a violent incident
- Injuries to body parts that would normally not be damaged in an accident or only in exceptional cases, e.g. the perianal area
- Injuries/bruises in non-mobile children, especially children under the age of 4 months old

1.7.2 Blunt-force Trauma: Closed Skin Injuries

1.7.2.1 Erythema

Erythema is defined as redness of the skin and/or mucous membranes caused by dilatation of the underlying capillaries. It is caused by a non-specific local reaction, which occurs with any cutaneous trauma (e.g. by heat, friction, rubbing, pressure, or by the application of irritating chemical substances), infection, or inflammation. Contrary to bruises and petechiae, erythema does blanch under diascopy, because there is no leakage of erythrocytes in erythema. In case of a traumatic cause, erythema will vanish within minutes to hours after the incident. For that reason, erythema is only rarely seen during the medical evaluation of suspect child abuse cases, unless the child is examined by a physician within minutes to hours after the incident that caused the erythema.

1.7.2.2 Bruises

A bruise is an injury of soft tissues (skin, underlying tissues, mucous membranes), in which vessels (capillaries and venules) are damaged by trauma (usually blunt-force trauma: collision/compression or stretching), causing leakage of erythrocytes into the surrounding interstitial tissues. Because of the leakage of erythrocytes, bruises will not blanch under diascopy. Synonyms that are used for bruises are hematoma, contusion, purpura, and ecchymosis. In this book the terms bruise or hematoma will be used.

Bruising occurs when the loading of soft tissues exceeds the maximum load-bearing capacity of these tissues. This means that traumatic bruising can occur in children with and without congenital or acquired medical conditions in which the maximum load-bearing capacity of vessels is diminished, in clotting disorders or in a combination of both, e.g. systemic disorders with clotting problems. In children with a medical condition trauma is often still needed to create bruising, but the acquired threshold value to cause bruising in these children is less than in children without such a condition. The manner of traumatic bruising in young children with or without a medical condition can be either accidental (injury, e.g. due to a fall during daily activities) or non-accidental (injury due to a human act or omission). In other words, finding a medical condition does not exclude non-accidental circumstances [45].

The total number of bruises (including bruises of the shins) in a young child due to age-appropriate motor behaviour (such as playing) (accidental bruises) typically ranges from a few to about 15 [46].

One should, however, realize that spontaneous bleeding and bruising can sometimes also occur in children with, e.g. immune related thrombocytopenia (ITP), sepsis, or Henoch–Schönlein purpura.

Cutaneous bruises generally are located superficially in the skin and subcutaneous tissues, with usually externally visible discoloration, changing in colour over time (days to weeks). More extensive extravasation of blood will be seen in areas with increasing laxity and loose subcutaneous elements in the tissues, e.g. bruising around the eyes is more obvious than bruising of the hand palm [47].

If bruising is found symmetrically on the whole body, this usually indicates an underlying condition. If symmetrical bruising is found on a limited part of the body, e.g. only in

the head-neck region or on the inside of the upper arms or thighs, inflicted injury is more likely than an accidental injury or a medical condition [45].

Traumatic bruising results from either blunt force (impact trauma, e.g. due to falling, bumping, or punching) or compressive force (compressing, tightening, or twisting of the skin, e.g. when squeezing the skin) [25].

Kemp et al. published the results of a systematic review of the medical literature concerning the circumstances under which bruises can occur in children (manner of bruising) [48]. They also evaluated the specific characteristics of bruises, related to the manner of injury. Accidental bruising occurred very rarely (<1%) in non-mobile children. Clustering of bruising was often found in children in whom the bruising was sustained in non-accidental trauma. Frequently, other types of recent and old injuries were found in these children such as scars from burns or (healing) scrapes, or scratches.

Pierce et al. showed that bruises on the trunk (chest, abdomen, back, buttocks, anogenital region, and hips), ears, and/or neck in children under 4 years of age (TEN-4) were indicative of non-accidental circumstances (specificity of 84%; sensitivity of 97%) [49]. The authors stated that the indicative nature of bruising at these locations applied if a statement about plausible accidental circumstances, including observation of the causing incident by an independent observer, was missing. Based on the findings of Pierce et al., it can be calculated the finding of a bruise in the TEN-region in a child under the age of 4 years is approximately 6 times more likely under the hypothesis of a non-accidental trauma than under the hypothesis of an accidental trauma. Pierce et al. further concluded that bruises in children under the age of 4 months, regardless of the location of the bruises, always are suspect for non-accidental circumstances, especially if no plausible accidental explanation is present or no evidence is found for a condition with an increased bleeding tendency.

In 2021, Pierce et al. published the results of the evaluation of an extended 'bruising clinical decision rule', TEN-4-FACESp (Table 1.3) [50]. The new BCDR has a sensitivity of 95.6% (95%CI, 93.0–97.3%), a specificity of 87.1% (95%CI, 85.4–88.6%), a negative predictive value (NPV) of 98.8% (95%CI, 98.1–99.3%), and a positive predictive value (PPV) of 63.9% (95%CI, 60.3–67.7%). Based on these new findings, it can be calculated that the finding of one bruise in the body parts covered by 'TEN-4-FACESp' in a child under the age of 4 years of age, or regardless of the location on the body in a child under the age of 5 months, is approximately 7.5 times more likely under the hypothesis of a non-accidental trauma than under the hypothesis of an accidental trauma (LR+ 7.41). Finding a bruise in a child under the age of 5 months is always suspect, irrespective of the location.

Table 1.3 TEN-4-FACESp [50]

T	Torso which includes chest, abdomen, back, buttocks, and genitourinary area
E	Ears
N	Neck
4	The 4 represents any bruising anywhere to an infant of 4 months or younger
F	Frenulum
A	Angle of jaw
C	Cheeks (fleshy)
E	Eyelids
S	Subconjunctivae
p	Patterned injuries

One of the most common explanations of bruising, according to the caregiver, in young and pre-mobile children is that the child itself was responsible for the occurrence of the bruising. Depending on the age and the level of development of a child, it is possible that an accidental fall occurs unnoticed as a result of the child's own actions. An accidental trauma in a young child with limited mobility will almost always be a non-serious and often observed event, such as bumping the head when rolling over or after lifting the head. For young children with this level of development, a 'spontaneous fall' from a short distance, e.g. from a changing table, can occur if the child turns over on the changing table. However, such a fall can only occur in case of lacking supervision: the child has been left in an unsafe situation.

A young pre-mobile child will have a limited control concerning the movements of the head and neck because the head is large compared to the rest of the body and the child does not have complete control over the neck muscles. There may be bumping contacts against persons or objects during the daily handling and care of a child, though there are no indications in the literature that bruising will occur due to these bumping contacts. It should be noted here that if a child has a clotting problem, bruising may occur during these bumping contacts.

It is possible that a young and pre-mobile child falls out of the hands of a parent/caregiver. This can be seen as a short distance fall (about 1.5 m). This type of accidental fall occurs regularly [51]. In such a fall, injuries (including bruising) can occur in those body parts that first come into contact with an object or surface during the fall. While being in the arms of the caregiver, the fall of the caregiver him/herself can increase the momentum of the fall and thereby the possible injuries.

With regard to the moment of the appearance of a bruise after a trauma and the possibilities for determining the age of a bruise based on visible characteristics, the following can be noted:

- Superficial bruises are usually visible as a discoloration soon after the causative event (almost immediately, up to minutes, e.g. on the forehead). With deeper bruising this

can take many hours to a few days (such as on a buttocks) [47, 52]. Furthermore, deeper bruising sometimes will not become visible, except when the skin is incised, e.g. during a forensic autopsy [47].
- Superficial bruises fade gradually and are usually no longer visible after 2 to 3 weeks.
- To date no scientific basis exists for dating bruises based on colour changes in young children, neither in visual assessment of findings during physical examination nor in review of photographic material [42].

1.7.2.3 Petechiae

Petechiae are small red, purple, or brown spots caused by minor bleeding (0.1–2 mm, pinpoint bleeding/punctate bleeding) in the skin, the mucous membranes, and/or the serosa surfaces, due to leakage of blood from damaged post-capillary venules. Because of leakage of erythrocytes in the surrounding tissues, petechiae will not blanch under diascopy.

Petechiae are common and can be caused by a large number of medical or traumatic conditions, ranging from minor to very serious [53]. Medical conditions include viral and bacterial infections, haematological disorders (e.g. immune thrombocytopenia, vitamin K deficiency), malignancies (e.g. leukaemia), vasculitis and inflammatory conditions (e.g. Henoch–Schönlein purpura), and disturbance of collagen synthesis, due to vitamin C deficiency, side-effects of some drugs (e.g. anticoagulants or some antibiotics). They can also be caused by heavy coughing (e.g. in whooping cough) or straining (e.g. in severe constipation). Petechiae can also be self-inflicted, e.g. due to suction, especially in elderly children. Petechiae in the skin around the eyes, sometimes combined with conjunctival haemorrhages may also occur in patients with eating disorders, due to self-induced vomiting [54].

In trauma the damage to the post-capillary venules is primarily due to an acute rise of the venous pressure in these venules. This sudden increase can result from several mechanisms:

- Back pressure, caused by mechanical obstruction of venous return to the heart that leads to over-distension and rupture of the thin-walled peripheral venules, which in its turn leads to rapid extravasation of blood, especially in lax tissues, such as the eyelid, or in unsupported serous membranes, such as the pleura and the epicardium (e.g. in strangulation or choking)
- Back pressure, caused by gravitational obstruction of venous return to the heart (e.g. in upside down hanging)
- Locally acting external colliding and/or compressing and/or (partly) crushing force action with or by a blunt object (e.g. in a slap mark)
- External, local acting, suction on the skin (e.g. 'love bite')

The manner of trauma can be accidental or non-accidental. 'Accidental' petechiae can occur e.g. in hanging upside down during play or sports on a horizontal bar, weightlifting, or in accidental drowning. 'Non-accidental' petechiae can be seen, e.g. in strangulation or choking but also in physical abuse. In a retrospective study of 506 children under the age of 6 years suspected of inflicted bodily injury, petechiae were present in 15.4% of the children for whom inflicted injury was deemed proven (54 of 350 children) and 1.9% of the children for whom an accidental trauma was deemed proven (3 of the 156 children). This corresponds to a likelihood ratio of almost 10 [48]. Petechiae can also be found as a result of, what can be considered to be a specific type of non-accidental trauma, medical procedures, in which local physical pressure is applied, e.g. due to a tight tourniquet or being held tightly.

Sometimes a medical condition and trauma coexist, e.g. in illnesses with vomiting and coughing (e.g. in pertussis), in which petechiae can be found in the head and neck region.

It is not possible to date petechiae based on externally visible characteristics. Petechiae due to trauma appear fairly quickly (within several seconds to minutes) after the loading and usually disappear within a couple of days up to about one week. Any accompanying redness that may occur simultaneously is visible almost immediately for up to 1 or 2 days.

1.7.3 Blunt-force Trauma: Open Injuries

1.7.3.1 Abrasions

An abrasion is a superficial injury to the skin, characterized by the traumatic removal, detachment, or destruction of the epidermis and sometimes underlying parts of the skin. Abrasions are also known as erosions, excoriations, or crab, scratch and scrap injuries.

Abrasions are located at the site of contact with the object and are caused by a blunt-force trauma, in which there is:

- Rubbing, sliding, scraping, wiping, or other lateral movement of the skin relative to an object with a high friction surface (e.g. a brick) or relative to a more or less sharp or pointed object (e.g. barbed wire, fingernail, tip of a nail or knife, piece of glass or animal-claws). In the lateral movement an object can be moving along the body surface, the body surface along the object or a simultaneous movement of object and body surface.
- Compression/crushing of the skin, where there is a force acting more or less perpendicular to the skin.
- A combination of both mechanisms.

Only rarely a child will sustain abrasions due to non-accidental circumstances. If these injuries are inflicted, the injuries will be found in particular in the head and neck region (head, face, mouth, and neck) and on or near the upper

arms [55, 56]. The most common injuries of this type are sharp, line-shaped injuries due to scratching with the nails or 'imprint' injuries, specifically nail imprints due to pinching. In elderly children, self-harm has to be ruled out.

If an abrasion is caused by a fingernail, the width and depth depend on the width and sharpness of the nail and the amount of pressure during the contact. If only pressure is applied and no lateral movement is made, the skin is often only superficially damaged and a superficial linear or curved (crescent-shaped) shape will be visible ('static fingernail imprint') [56]. A child usually sustains a fingernail abrasion in accidental circumstances. Newborns and young infants can have relatively long and sharp nails from birth. The child can scratch himself through non-directed movements, particularly in the face. These injuries are almost always limited in number and size, usually with a maximum of 0.5 cm long.

Dating of abrasions in children, either living or deceased, is not reliable if based on externally visible characteristics.

1.7.3.2 Lacerations and Avulsions

A laceration is a full-thickness injury of the skin and subcutaneous tissues, characterized by tearing of tissue in a frayed and irregular pattern and often associated with abrasions, contusions, and crushing of the wound margins. A laceration is also known as a tear or tear wound. A laceration is caused by blunt-force trauma (collision/compression or stretching—shearing force).

An avulsion is a laceration in which skin and subcutaneous tissues are not just separated but torn away from the underlying tissues. An avulsion is caused by the same mechanism as a laceration.

Usually lacerations and avulsion are sustained in accidental circumstances and are only rarely seen in non-accidental circumstances. A child may incur a laceration in non-accidental circumstances, e.g. while being whipped. Another non-accidental cause can be sexual abuse with penetration (digital, penile, object, etc.) resulting in a laceration of the hymen or the anal ring and surrounding tissues.

Dating of lacerations in children, either living or deceased, is not reliable, if based on externally visible characteristics.

1.7.3.3 Blunt Penetrating Trauma

A blunt penetrating trauma happens when a more or less pointed object pierces the skin. The diameter of the penetrating object may vary from a few millimetres (e.g. pin or nail) up to more than 10 cm (e.g. a wooden stake). The result of the piercing varies from deep narrow wounds that are sometimes hard to identify due to a small entry hole without clinical consequences via the same narrow wounds with penetrating and life-threatening injuries to underlying tissues to large injuries with extensive damage to underlying organs, e.g. in a blunt penetrating trauma of the abdomen [57].

Puncture wounds caused by blunt penetration should be differentiated from injuries caused by a sharp penetrating trauma (stab wound). As far as known from the literature and from case work done by the authors injuries due to a blunt penetrating trauma always occur in accidental circumstances.

1.7.4 Sharp-force Trauma

1.7.4.1 Incisions and Stab Wounds

An incision (incised wound), if caused by sharp-force trauma with a clean, sharp-edged object (e.g. a knife, a razor, or a glass splinter), is a slicing injury usually with sharp edges (clean cut), in which the injury is longer than deep, varying from superficial (paper cut) to significant (surgical incision). A sharp-edged incised wound will give little or no information about the object that caused the injury. If an incision is caused by a sharp serrated object (e.g. a bread knife), the incised wound will have laceration-like edges.

A stab wound (puncture wound or penetrating injury) is a deep, narrow injury, which is deeper than its length visible in the skin, caused by a sharp-pointed object puncturing the skin (e.g. needle, knife, or broken glass). A stab wound usually is sharp edged, except in case of a sharp serrated object. Stab wounds caused by sharp penetrating trauma should be differentiated from injuries caused by blunt penetrating trauma (puncture wounds).

Incisions and stab wounds are found in more than 10% of children with accidental injuries [58]. Incised wounds usually occur in accidental circumstances, usually due to broken glass (drinking glass, window pane), but may also occur due to cutting on paper or grass, a knife, broken glass, or lid of a can. Often these occur during household activities. Incisions can also be caused by sharp edges of equipment or tools during leisure time and work. Accidental incisions are often more irregular in shape. Deep penetrating accidental stab wounds can occur if a child falls on a knife or other sharp object, such as a sharp pencil or a knitting needle.

Inflicted incisions or stab wounds usually are caused with a knife, razor, or broken glass by a perpetrator. In children, this type of injury is only rarely inflicted. Nevertheless, one should consider non-accidental circumstances, if these injuries are found in children, especially if other suspicious injuries are found [59, 60]. An incised wound in the neck of a young child is highly suspicious for an attempted homicide or murder [61–63].

In case of self-inflicted injuries, any object can be used that can lead to incised wounds. 'Cutting/carving' with a sharp object (e.g. knives, razors, and glass fragments) is probably the most common form of self-infliction. The resulting injuries can exist on the entire body, but mostly

only on the wrists and forearms. It is more common in girls than in boys and can occur at any age, although it is usually seen in adolescents and young adults [64, 65].

Sometimes the term 'cut' is used. This term is confusing if used in a forensic setting, because this term is not well defined and commonly used for any injury in which the integrity of the skin is compromised. A 'cut' can result from either a blunt-force trauma (laceration, avulsion) or a sharp-force trauma (incision, stab wound).

1.7.4.2 Gunshot Wound

A gunshot wound (missile wound, velocity wound) is an injury caused by an object entering, and often leaving, the body at a high speed; typically a bullet or similar projectile. Often two wounds are found, one at the site of entry and one at the site of exit (through-and-through injury). Wound characteristics depend on the firearm (handgun, rifle, or shotgun) and ammunition (mass and design) used, bullet direction, range, and sequence of fire [66]. Analysis of gunshot wounds should, given the multitude of parameters involved, only be done by experts. Gunshot wounds may occur under accidental and non-accidental circumstances and are almost exclusively seen in countries with liberal firearm legislation like the United States, where it ranks third as cause of death for children [67–71]. Based on data from the Kids' Inpatient Database from 2000, 2003, 2006, and 2009 for children <19 years of age a total of 27,566 firearm-related injuries were recorded [72]. In children <5 years, most injuries were accidental (59.3%). In another study, it was shown that most fatal accidental shootings in children (89%) occur in the child's house while the child is playing with a loaded weapon [73].

1.7.5 Non-mechanical Trauma: Near Contact with Physical Agents

As already stated in Sect. 1.5.3, injuries are the result of the transfer of energy during a direct contact of or a contactless transfer to the skin and/or other body parts, in which the skin and/or the other body parts are exposed to extremes in temperature (heat and cold) or to chemical or physical agents (acidic and alkaline chemicals, electricity, microwaves, and radiation). In a contactless transfer the risk of injuries usually decreases with increasing distance between the body and the energy source, e.g. with radiation from an infrared heater. Chemical or physical agents may have a similar effect on the skin and the subcutaneous tissues as extremes in temperature (heat or cold): cutaneous burns, resembling heat or cold-related burns (Table 1.4). Chemical or physical agents may create heat at the moment of contact with the skin [74–76]. Besides external burns, internal burns may arise due to swallowing and inhalation of chemicals or electrocution.

Table 1.4 Injuries resulting from non-mechanical trauma

Trauma	Resulting injuries
Thermal	Heat: burns and scalds Cold: chilblains and frostbite
Chemical	Burns Allergic reactions (topical and generalized) Generalized poisoning manifestations
Electrical	Burns High- and low-voltage injuries
Electromagnetic and ionizing (radiation)	Burns

In this section, we will only shortly pay attention to the effects of thermal trauma, which may occur in accidental and in non-accidental trauma. Injuries due to accidental exposure to chemical and physical agents are rare in paediatric patients. As far as is known from the existing medical literature, injuries due to non-accidental exposure to these agents are extremely rare (chemical and electrical trauma), or even non-existing (electromagnetic and ionizing trauma).

In a thermal skin trauma, the damage to cells is caused by the transfer of thermal energy to the skin and/or the subcutaneous tissues, as a result of the exposure of tissue to high or low temperatures. The extent of the damage is determined both by the temperature and the duration of exposure. Thermal trauma can also be caused by low or freezing temperatures (cold-related injuries). Thermal trauma may result from:

- Direct contact (transfer of energy by conduction) with a dry, hot, and solid heat source (dry burns due to, e.g. iron or a curling iron), hot liquids (e.g. soup, hot tea), vapours, or gases (scalds or wet burns), and open fires (cigarette burns, fire and flame burns)
- Exposure of the skin and the subcutaneous tissues to the radiant heat of an object, e.g. the close proximity to a radiant fire or electrical heater or the prolonged sun exposure

1.8 Other Injuries

1.8.1 Introduction

In paediatric patients, the same type of injuries can be found in accidental trauma as in non-accidental trauma. However, with increasing age, injuries due to accidental trauma become more common than injuries due to non-accidental trauma.

Fractures probably are, next to bruises and other cutaneous injuries, the second most common injury in paediatric

patients due non-accidental trauma [77]. The cause and manner of fractures will be described in Chaps. 5 to 12. Intracranial and thoracoabdominal injuries can also be found in paediatric patients due to non-accidental trauma, but are less common than non-accidental cutaneous injuries and fractures. In this section, a short overview of thoracoabdominal injuries will be given without the intent of being complete. In Chap. 5 a short overview will be given of intracranial injuries, due to non-accidental trauma.

In paediatric patients, the most common cause of death, irrespective of the circumstances (accidental or non-accidental), is trauma [78]. Death due to accidental trauma is most commonly caused by intracranial injuries, followed by intrathoracic and intra-abdominal injuries as second and third most common cause [79, 80]. Death due to non-accidental trauma is also most commonly caused by intracranial injuries, but intra-abdominal injuries are the second most common cause [81, 82]. Rosenfeld et al. evaluated the findings in 678,503 children who were admitted with injuries, due to a physical trauma. Nineteen thousand one hundred and forty-nine children (3%) sustained injuries in a non-accidental trauma. According to Rosenfeld et al. non-accidental trauma is a major cause of death in young children, with polytrauma being common [83]. In 43% of trauma deaths in children under the age of 1 year and in 31% of children under the age of 5 years, death was due to non-accidental trauma, with traumatic brain injury being the most common cause (50%), followed by hollow viscus and thoracic injuries.

1.8.2 Thoracoabdominal Injuries

According to Milroy, thoracoabdominal injuries caused by blunt-force trauma are an important cause of morbidity and mortality in children [82]. Milroy also stated that isolated thoracic or abdominal injuries are less common than combined thoracoabdominal injuries and that thoracic injuries have a higher mortality than abdominal injuries.

Thoracoabdominal injuries are either caused by static or by dynamic loading (see Sects. 1.8.2.1 and 1.8.2.2) and can be sustained in accidental and in non-accidental circumstances. Usually thoracoabdominal injuries are sustained in accidental trauma, e.g. motor vehicle accidents [82]. When no plausible accidental explanation is given, non-accidental trauma should be considered in paediatric patients with blunt thoracoabdominal injuries, with intra-abdominal injuries being more common than intrathoracic [78, 82].

Sinha and Lander also stated that, even if a plausible cause of the injuries is present, neglect should be considered if the injuries were sustained in unusual circumstances, e.g. injuries due to a skateboard accident in an 8-year-old at 23:00 h.

Shenoi et al. evaluated the findings in 12,044 children with blunt-force trauma to the torso [84]. In 720 children (6%) the injuries were determined to be inflicted, in 9563 children (79.4%) unintentional (accidental), and in 148 children (1.2%) indeterminate. In 1613 children (13.4%), no data were found concerning the circumstances under which the injuries were sustained. In their study, children with accidental thoracoabdominal injuries had a lower median age than children with inflicted thoracoabdominal injuries (10 versus 14 years of age). There was no difference in mortality rates between both groups. The risk of pelvic fractures in the group of children with inflicted injuries was 96% less than the group with accidental injuries. Children with accidental injuries were more likely to be hospitalized.

1.8.2.1 Intrathoracic Injuries

(Intra)thoracic injuries are caused by static loading (compression) or to dynamic impact loading (blunt-force or penetrating trauma), irrespective of the circumstances under which the injuries are sustained (accidental or a non-accidental trauma).

Around 85% of all thoracic injuries in paediatric patients, that are serious enough to warrant medical attention and/or treatment, are due to blunt-force trauma (compression or impact), and around 15% are due to penetrating trauma (see also Chap. 7) [78, 85]. Blast injuries are very rare in paediatric patients.

Thoracic trauma may cause injuries of the intrathoracic organs (lungs, heart, aorta and great vessels, oesophagus, tracheobronchial tree), and of the structures of the chest wall. The most common injuries due to blunt-force trauma are fractures of ribs and sternum, contusions of the lungs or the heart, pneumothorax, and/or haemothorax [82, 86]. Thoracic injuries account for less than 10% of all paediatric trauma-related injuries but comprise up to 15% of paediatric trauma-related deaths [85, 86]. According to Milroy, mortality is higher in children with damage to the heart and to the aorta [82].

Several studies have shown that there is a clear difference between (intra)thoracic injuries sustained in accidental and in non-accidental trauma (see Sect. 7.3.3). Non-accidental intrathoracic injuries are more common in children under the age of 5 years than in children over the age of 5 years [87].

1.8.2.2 Intra-abdominal Injuries

Most intra-abdominal injuries are the result of a serious traumatic event, irrespective of the circumstances under which the injuries are sustained (accidental or a non-accidental trauma). The most common injuries are contusions and lacerations to the solid organs (liver, spleen, and kidneys), and less common injuries to the hollow viscera, irrespective of the circumstances.

In the United States in about 90% of the children with intra-abdominal injuries, these injuries are caused by blunt-force abdominal trauma [88]. In the remaining 10%, the injuries are due to several other causes, like sharp penetrating trauma or chemical trauma, such as the ingestion of objects like batteries or etching substances.

Intra-abdominal injuries in blunt-force abdominal trauma can be due to several causes [89–91]:

- Static loading (compression) with damage to hollow viscera ('bursting' injuries): if the abdomen is compressed, a hollow organ filled with liquid, air, or partially digested food can be compressed against a hard structure, e.g. the spine. This can lead to bursting of the organ, due to an increase in intraluminal pressure. 'Bursting' injuries most commonly occur in fluid-filled intestinal loops.
- Dynamic impact loading with damage to hollow viscera ('bursting' injuries): these injuries resemble the bursting injuries, due to static loading, and are usually caused by a direct blunt-force trauma to the abdomen, e.g. a 'single point blow' or a blow in the midline of the abdomen.
- Dynamic impact loading with damage to solid organs ('crushing' injuries): 'crushing' injuries of solid abdominal organs occur due to the impact of blunt-force trauma on the upper abdomen or on the lower ribs, in which the organs are violently and suddenly compressed against a hard structure, e.g. the spine or the ribs. This may result in lacerations and ruptures of the liver, spleen, pancreas, or kidneys. Bleeding in the intestinal wall, especially the duodenum wall, may also occur.
- Dynamic impact loading due to a rapid change of the velocity or direction of motion of the body, resulting in shearing and tearing forces created in areas of relative fixation inside the body [91]. This can occur in an event in which, e.g. something hits the child with a high speed and the child hits a solid object, e.g. a wall or a cupboard, resulting in a sudden and immediate deceleration. The sudden deceleration of the body and the inertia of the organs may lead to a sudden application of a large inertial load, causing the bowel to rupture on the antimesenteric side close to the posterior abdominal wall attachment point, e.g. at the Treitz ligament or the ileocecal junction [92, 93]. In such an event, the upper abdominal organ vascular supply can also tear off. Bleeding into or perforation of the small intestine may also occur.

Blunt-force trauma with damage to either hollow visceral and/or to solid organs can occur in accidental trauma, e.g. due to the impact of the end of a bicycle handlebar to the abdominal wall, or in non-accidental trauma, e.g. due to a punch or kick [81, 92, 94, 95].

Dynamic impact loading with a rapid change of the velocity or direction of motion of the body can occur in accidental trauma, e.g. in motor vehicle accidents, when the child is restrained with the aid of a two-point seat belt, or in motor vehicle versus pedestrian accidents or in a fall from a height [92]. It may occur when a child is violently thrown against a wall or on the floor. This may happen in non-accidental trauma, e.g. due to a bomb blast or during a physical assault, but is probably very rare [82].

In 1 to 8% of children who were hospitalized because of accidental blunt abdominal trauma, intra-abdominal injuries are found [92, 94, 95]. Intra-abdominal injuries were found in up to 65% of children who were hospitalized with a non-accidental blunt abdominal trauma [96]. In a systematic review concerning visceral injuries in paediatric patients due to non-accidental trauma, it was found that children with abdominal injuries due to non-accidental trauma were younger than children with abdominal injuries due to accidental trauma (2.5–3.7 years vs. 7.6–10.3 years) [87]. Lane et al. found that the rates of non-accidental abdominal trauma were higher for infants than for any other age group. They also found that in their study infants had higher rates of hospitalization because of non-accidental abdominal injuries, despite the fact that often toddlers are considered to be at highest risk for non-accidental abdominal injuries. They also found that more than 25% of all abdominal trauma in children <1 year of age was due to non-accidental trauma [97].

In children with non-accidental trauma duodenal injuries, especially in the third or fourth part, were commonly reported, but also injuries of the ileum and jejunum have been described. Duodenal injuries, due to accidental trauma, were not found in children under the age of 4 years [87]. Duodenal hematoma, caused by blunt-force abdominal trauma, may lead to obstruction of the lumen and may result in weakening and finally rupturing of the wall. Ruptures may present as peritonitis [82].

Injuries to liver, spleen, and pancreas are also frequently seen in non-accidental trauma [98]. Lane et al. even found that the organs that were most commonly injured were the liver (64% of hospitalizations), kidney (19%), and stomach/intestines (12%) [97].

Non-accidental blunt-force trauma, e.g. due to blows or kicks, may cause contusion, laceration, or transection of the pancreas [82]. The damage to the pancreas may be complicated by pancreatitis or pancreatic pseudocyst formation [82, 87].

Coexisting findings in children with inflicted abdominal injuries include malnutrition, fractures, burns, and head injuries [87].

Lane et al. stated that mortality rates of non-accidental abdominal trauma reported in the medical literature are 13–45% [97]. Maguire et al. found that the mortality from inflicted abdominal injuries was significantly higher than accidental injuries (53% vs. 21%) [87]. Post-mortem examination shows that often there has been more than 1 event in

which previous, unrecognized abdominal injuries were sustained [87, 99]. Gilbert-Barnes stated: 'Many of these children have received repeated blows to the abdomen, and careful examination and microscopic sampling of the abdominal contents has revealed extensive fibrosis confirming subacute or remote injury'.

Often it is stated that intra-abdominal injuries in a child are sustained in a fall. Carter and Moulton evaluated the findings in 180 paediatric patients under the age of 5 years with blunt-force abdominal trauma [100]. In 65 patients the intra-abdominal injuries were due to non-accidental trauma and in 115 patients due to accidental trauma (fall casualties). They found that non-accidental trauma should be considered, if the child was under the age of 5 years, had a hollow viscus, pancreatic and/or intracranial injury with a high injury severity score. They also found that in their population solid organ injuries and isolated splenic or renal injury were more likely in accidental than in non-accidental trauma.

Externally visible injuries are often absent in children with abdominal injuries, due to blunt-force trauma, irrespective of the circumstances under which the injuries were sustained [82]. Bruising can be absent in up to 80% of children with inflicted abdominal injuries [87].

It is not exactly known how often intra-abdominal injuries are sustained in non-accidental trauma. Estimates are that between 1% and 9% of children that are admitted to hospital because of non-accidental injuries will have intra-abdominal injuries [81, 101–104]. Because abdominal injuries in non-accidental trauma are often severe and arrive often late in hospital, there is a high rate of surgical interventions [105]. According to Sivit et al., around 5% of all abdominal injuries in need of a surgical intervention are sustained in non-accidental trauma [102].

1.9 Objectifying Suspicions of Inflicted Injuries and Non-accidental Trauma

Any suspicion of inflicted injuries/non-accidental trauma in a child should always be taken seriously. A correct and evidence-based interpretation of these signs and symptoms ('fact finding') is in the interest of the child but also in the interest of its parents/caregivers.

Some suspicions will be easy to reject, e.g. when the physical findings can be explained as disease-related symptoms or as injuries due to an accidental trauma, observed by an independent eye-witness. Other suspicions will be easy to confirm, e.g. when somebody admits to have inflicted the injuries or when an independent eye-witness observes the infliction of the injuries.

In many cases however, suspicions will require an elaborate investigation before a conclusion can be reached. This investigation requires taking of an extensive clinical history (see also Sect. 1.10). A forensic medical evaluation demands the same careful considerations as the making of a clinical medical diagnosis, with a meticulous weighing of alternative explanations, as in a clinical medical differential diagnosis.

The forensic medical evaluation of a suspicion is based on both a clinical and a forensic medical scientific framework and concerns the evaluation of cause and manner of the findings (Sects. 1.5 and 1.6). And, in the end, also of the motivation of the person who inflicted the injuries. The evaluation of the motivation (intention), however, is not a forensic medical task, but is the task of a behavioural analyst (forensic psychologist or psychiatrist) and/or of law enforcement.

Many signs and symptoms may lead to a suspicion of inflicted injuries in a child. A suspicion of injuries due to non-accidental trauma in a child may arise, based on individual findings or combinations of findings, e.g. bruises, fractures, and subdural haemorrhages. These individual findings or the combinations of these individual findings have their own clinical and forensic medical differential diagnosis and demand a careful differential diagnostic process, which is done in a systematic way.

In Tables 1.5 and 1.6, an example is given of a systematic approach of the diagnostic process in case of a suspicion. This process is based on the principles of diagnosis by exclu-

Table 1.5 Clinical scientific framework: clinical medical diagnosis

Step 1: Collecting as much clinical data as possible, incl. all data from the medical history	
• Whole body examination, incl. registration and photography of all external injuries and the absence of injuries	• The complete set of medical data is necessary, incl. all original source information (laboratory data, radiology, and retina photos)
• Registration of growth and development	• Only medical correspondence is insufficient
• Extensive neurological evaluation	
• Laboratory tests	
• Radiology (X-skeleton—RCPCH/ACR criteria, CT, MRI, US)	
• Ophthalmology	
• (Forensic pathology, neuropathology, ophthalmopathology)	
• Other data	
Step 2: Determination of the cause of the medical findings	
• Differential diagnosis of the individual and combined medical findings	
• Evaluating all the available medical data	
• Cause of medical findings	• Medical condition (congenital or acquired)
	• Trauma (trauma during or after birth)
	• Undetermined
Formulation of the weight of the evidence regarding the cause of the medical findings:	
• The individual findings or the combination of findings give no (or moderate or strong or very strong) support to hypothesis 1 (e.g. medical condition) against hypothesis 2 (e.g. trauma)	

Table 1.6 Forensic scientific framework (in case of trauma): forensic medical differential diagnosis

Step 3: Determination of manner of injury
• Medical findings (injuries and injury-patterns) compared to what is known in medical science
• Medical findings compared to the statements of parents, carers, or others to medical staff (medical history) & others (incl. police interrogations)
Manner of injury
• Trauma during birth
• Trauma after birth: accidental, inflicted
• Undetermined
Formulation of the weight of the evidence regarding the manner
• The individual findings or the combination of findings give no (or moderate or strong or very strong) support to hypothesis 1 (e.g. inflicted injury) against hypothesis 2 (e.g. accidental injury)
Step 4: Determination of mechanism of injury
• Medical findings (injuries and injury-patterns) compared to what is known in medical science
• Medical findings compared to the statements of parents, carers, or others to medical staff (medical history) and others (incl. police interrogations)
Mechanism of injury
• Static loading
• Dynamic impact loading: impact trauma (acceleration and/or deceleration trauma)
• Dynamic impulse loading: repetitive acceleration-deceleration trauma
• Undetermined
Formulation of the weight of the evidence regarding the mechanism
• The individual findings or the combination of findings give no (or moderate or strong or very strong) support to hypothesis 1 (e.g. dynamic impact loading) against hypothesis 2 (e.g. dynamic impulse loading)

sion and by inclusion and on the use of Bayes Theorem to formulate the conclusions, concerning the suspicion:

- Diagnosis by exclusion: a diagnosis reached by a process of elimination of other possibilities, related to the probability of these possibilities.

  A diagnosis by exclusion is a major component in the performing of a clinical or forensic medical differential diagnosis and necessary if the presence of a certain medical condition cannot be established with complete confidence from confirmatory physical examination, radiology, or laboratory testing.
- Diagnosis by inclusion: a diagnosis based on the results of confirmatory physical examination, radiology, or laboratory tests. In forensic medicine, statistical analysis of individual findings or combinations of findings offer the possibility of a diagnosis by inclusion under the condition that the results of the analysis of the findings do fulfil the normal statistical standards of accepting a diagnosis in clinical practice (see also Chap. 17).

  A diagnosis by inclusion is to a certain height also possible by applying the 'duck principle' as described by Minns and Brown in 2005 (Table 1.7) [106].

Table 1.7 Combinations of findings: the Duck principle [106]

	Characteristics	Conclusion
A bird	That waddles	Possible duck
	That waddles + swims on water	Suspected duck
	That waddles + swims on water + quacks	Strongly suspected duck
	That waddles + swims on water + quacks + has webbed feet	Few doubts of being a duck
	That waddles + swims on water + quacks + has webbed feet + a flat bill	Beyond reasonable doubt it is indeed: a duck!!

While evaluating a suspicion of inflicted injuries/non-accidental trauma, one should always keep in mind that, given the findings and circumstances, a possibility is not always a (medical) probability and a probability will not always be a (medical) possibility. A good example of the difference between certain possibilities and the probability of these possibilities was given in 2008 by David in an article on the evidence in non-accidental head trauma [107]. In this publication David gave two tables, one with causes of subdural bleeding and one with causes of retinal haemorrhages. Although David explicitly stated that he did also include causes in adults and that these were not relevant in children (possibilities without probability), the medical conditions in the tables are sometimes used as starting point in the differential diagnosis (*'diagnosis by exclusion'*) if inflicted head injury is suspected in a child. This leads to a confusion of tongues, in which possibilities and probabilities are used as synonyms. One might wonder how realistic (how probable) included possibilities like breakdancing, head banging, weightlifting, or boxing, described in the medical literature as causes of subdural bleeding in adolescents and adults, are as a cause of subdural bleeding in a young child under the age of 1 year. The same accounts for the listed caused by retinal haemorrhages. How realistic (how probable) is bungee jumping, high altitude, crushing injury to chest, or chest compression from safety belt as causes in a young child, despite their description in the adolescent and adult medical literature? Even if one looks at causes that were quoted by David and that could be relevant in infants, one should always ask how probable (how realistic), e.g. ECMO (ExtraCorporeal Membrane Oxygenation), diabetes, and sickle cell anaemia are as possible causes in infants, given the findings and circumstances in a specific child.

1.10 Characteristics of the Clinical History

Most physicians will positively identify injuries as inflicted when these injuries are of the most severe clinical category, such as extensive bruising or multiple fractures without identifiable medical history or cause in young, non-mobile chil-

dren. Problems arise mainly in children that sustained less severe injuries and have less obvious symptoms. To this category belong, e.g. mobile children that have some bruises or just one fracture without a clear clinical history [108].

1.10.1 Clinical History

A child is often not able to explain how (inflicted) injuries were sustained. This applies in particular to children in a life-threatening situation, making a conversation with the child (virtually) impossible. Besides, many children with serious inflicted injuries are preverbal. When children are able to relate the situation, there is a fair chance that they will keep silent out of, e.g. loyalty to the parents or out of fear for the perpetrator.

When inflicted injuries are suspected, it is important to pay attention to the clinical history of the child and the other family members. In case of inflicted injuries, it is possible that the child has sustained (multiple) previous trauma and has had prior hospitalizations. Various studies have shown that approximately 50% of all children in which child abuse was established had been seen by a physician for (in retrospect suspect) injuries [109]. Also, a child with inflicted injuries who returns to a non-safe home setting in which the infliction occurred, has a 30–50% chance to suffer additional injuries and an up to 10% increased risk for fatal violence [110].

Very regularly, earlier injuries and hospitalizations are found in other members of the family as well, such as the other parent or other siblings [111]. When compared to other men, it appears that men who use physical violence against their wife will frequently also use physical violence against their children. Furthermore, women who were physically assaulted by their husband appeared to be twice as likely to use physical violence themselves against their children compared to non-abused women. Likewise, 76% of the physically abused children allegedly used violence against a sibling [112].

1.10.2 The Origin of the Injuries

When a child makes a direct and spontaneous statement on how the injury was sustained, he or she will most likely tell the truth. This also applies to a witness making a statement regarding the origin of the injury. Yet, the statement of the witness should be closely examined, since the person will speak from his/her own set of values. Observed situations might be downplayed or, on the contrary, exaggerated. Also, the witness may serve his or her own self-interest by giving the statement.

The following items in the clinical history, concerning the origin of the injuries, can be considered as red flags for inflicted injuries:

- Contradictions between the statements of the child and the parent(s), between both the parents, or between parents and a witness.
- The absence of an explanation.
- Constantly varying statements, when further prompted or when taken on consecutive days.
- Different statements of parents to different people, or the withdrawal of statements.
- The absence of an adequate explanation for previous injuries detected physical or radiological examination.
- A statement in contradiction with the developmental stage of the child.
- A statement in contrast with the nature and/or location of the injury.
- A statement which only partially explains the injuries.
- A statement of the parents in which the child himself/herself or one of the siblings is stated to be responsible for the injury.

1.10.3 Delay in Seeking Medical Help

Another red flag for inflicted injuries can be a delay in seeking medical care. The latency period can vary from hours to days after the injury was sustained. Sometimes it may take weeks to months before injuries are ‘diagnosed’, e.g. in case of fractures. In these cases no treatment was sought initially, but injuries were incidentally recognized, e.g. during a complete workup because of a suspicion of child abuse. This is due to various reasons: shame, wrongly evaluated situation, hope for spontaneous recovery, and hope that the injury will no longer be recognized as resulting from child abuse. On the other hand, some accidentally sustained fractures cause only mild symptoms for which parents logically do not seek medical care.

Other red flags are the seeking of help by other persons besides the parent(s), such as the grandparents or a teacher. Or the seeking of help by the caregivers from others than their own general practitioner or paediatrician, thus a professional without previous knowledge of the child, without providing a plausible reason. Often this help is sought at odd times, such as during the evening at an ER.

1.10.4 Attitude and Reaction of the Parents/Caregivers

The attitude and reactions of parents vary and no typical pattern distinguishing between accidental and inflicted injuries

can be recognized. The contradiction between the severity of the injury and the reaction of the parent may indicate that the circumstances in which the injury was sustained are suspicious.

A parent may totally overreact to a minor injury. On the other hand, the carer may have hardly any or a very inadequate (remote, indifferent) reaction to (very severe) injuries.

A maltreating parent may react aggressively to innocent questions and the non-maltreating parent may react in a similar manner.

Sometimes parents can refuse further medical care when the possibility of child abuse/inflicted injuries is discussed.

When a physician speaks to the parents about a specific injury, he/she should be aware of the possible reactions of parents. Most parents realize that the physician doubts their statement and may suspect child abuse. This applies to parents who maltreat as well as to parents who do not maltreat. This may cause the parents to take a defensive attitude directly at the start of the interview. The reactions may vary from denial and a tendency to isolation and then proceed via anger, bargaining, and resignation to acceptance. Also, the physician will have to be aware that the parent to whom he speaks may be ignorant of the maltreating behaviour of the partner.

References

1. World Health Organization (WHO), International Society for Prevention of Child Abuse and Neglect (ISPCAN) (2006) Preventing child maltreatment: a guide to taking action and generating evidence. World Health Organization, Geneva
2. Hobbs CJ, Hanks HGI, Wynne JM (1999) Physical abuse. In: Hobbs CJ, Hanks HGI, Wynne JM (eds) Child abuse and neglect – a clinician's handbook. Churchill Livingstone, London, pp 63–104
3. World Health Organization (2006) Violence and Injury Prevention Team & Global Forum for Health Research - Unintentional child injuries in the WHO European Region. World Health Organization, Geneva
4. Baartman HEM, Hoefnagels C (2012) Emotionele mishandeling; een lastig te duiden begrip [Emotional abuse, a difficult to define concept]. Tijdschrift Kindermishandeling 2012:5
5. Van Wert M, Fallon B, Trocmé N, Collin-Vézina D (2018) Educational neglect: understanding 20 years of child welfare trends. Child Abuse Negl 75:50–60
6. Follingstad D, DeHart D (2000) Defining psychological abuse of husbands toward wives: contexts, behaviors, and typologies. J Interpers Viol 15:891–920
7. Doherty D, Berglund D (2008) Psychological abuse: a discussion paper. https://www.canada.ca/en/public-health/services/health-promotion/stop-family-violence/prevention-resource-centre/family-violence/psychological-abuse-discussion-paper.html. Accessed 5 Aug 2021
8. World Health Organization. Violence and Injury Prevention Team & Global Forum for Health Research (1999) Report of the Consultation on Child Abuse Prevention; Consultation on Child Abuse Prevention. World Health Organization, Geneva
9. Davis P, Murtagh U, Glaser D (2019) 40 years of fabricated or induced illness (FII): where next for paediatricians? Paper 1: epidemiology and definition of FII. Arch Dis Child 104:110–114
10. Royal College of Paediatrics and Child Health (RCPCH) (2012) Fabricated or induced illness by carers (FII): a practical guide for paediatricians. Royal College of Paediatrics and Child Health, London
11. Meadow R (1977) Munchausen syndrome by proxy. The hinterland of child abuse. Lancet 2:343–345
12. Bass C, Glaser D (2014) Early recognition and management of fabricated or induced illness in children. Lancet 383:1412–1421
13. London Child Protection Procedures (2017) Fabricated or induced illness. https://www.londoncp.co.uk/fab_ind_ill.html. Accessed 5 Aug 2021
14. Roesler TA, Jenny C (2008) Medical child abuse – beyond Munchausen syndrome by proxy. https://www.uptodate.com/contents/medical-child-abuse-munchausen-syndrome-by-proxy/print. Accessed 5 Aug 2021
15. Glaser D, Davis P (2019) For debate: Forty years of fabricated or induced illness (FII): where next for paediatricians? Paper 2: Management of perplexing presentations including FII. Arch Dis Child 104:7–11
16. Laming H (2003) The Victoria Climbié inquiry. Report of an inquiry. Presented to parliament by the secretary of state for health and the secretary of state for the home department by command of Her Majesty. http://www.victoria-climbie-inquiry.org.uk/
17. Alink L, Prevoo M, van Berkel S, Linting M, Klein Velderman M, Pannebakker F (2018) Nationale Prevalentiestudie Mishandeling van kinderen en jeugdigen (NPM-2017) [Prevalence study on Maltreatment of children and youth]. Leiden University, Institute of Education and Child Studies TNO Child Health, The Hague
18. Schellingerhout R, Ramakers C (2017) Scholierenonderzoek Kindermishandeling 2016 [2016 Pupils on abuse]. Wetenschappelijk Onderzoek- en Documentatiecentrum (WODC), Ministerie van Veiligheid en Justitie. The Hague
19. Vermeulen S, van Berkel S, Alink L (2021) Kindermishandeling tijdens de eerste lockdown [Child abuse during the first lockdown]. Instituut Pedagogische Wetenschappen, Leiden
20. Hillis S, Mercy J, Amobi A, Kress H (2016) Global prevalence of past-year violence against children: a systematic review and minimum estimates. Pediatrics 137:e20154079
21. Kim H, Drake B (2019) Cumulative prevalence of onset and recurrence of child maltreatment reports. J Am Acad Child Adolesc Psychiatry 58:1175–1183
22. Felitti VJ, Anda RF, Nordenberg D, Williamson DF, Spitz AM, Edwards V, Koss MP, Marks JS (1998) Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults. The Adverse Childhood Experiences (ACE) Study. Am J Prev Med 14:245–258
23. World Health Organization (WHO) (2002) World Report on Violence and Health – Child abuse and neglect by parents and other caregivers. World Health Organization, Geneva
24. Health Canada (2003) Injury surveillance in Canada: current realities, challenges. http://www.injuryresearch.bc.ca/docs/3_20090910_100541Report%20HC%20Inj%20Surveillance%20in%20Can%20Aug%202003.pdf. Accessed 5 Aug 2021
25. Bilo RAC, Oranje AP, Shwayder T, Hobbs CJ (2013) Evaluating suspicions skin findings in children. In: Bilo RAC, Oranje AP, Shwayder T, Hobbs CJ (eds) Cutaneous manifestations of child abuse and their differential diagnosis – blunt force trauma. Springer, Berlin, pp 25–61
26. DiMaio VJM, DiMaio D (2001) Blunt trauma wounds. In: DiMaio VJM (ed) Forensic pathology. CRC Press, Boca Raton, pp 91–116
27. Arieff AI, Kronlund BA (1999) Fatal child abuse by forced water intoxication. Pediatrics 103:1292–1295

28. el Awad ME (1994) Overheating in neonates in Saudi Arabia. East Afr Med J 71:805–806
29. Martos Sánchez I, Ros Pérez P, Otheo de Tejada E, Vázquez Martínez JL, Pérez-Caballero C, Fernández Pineda L (2000) Hipernatremia grave por administración accidental de sal común [Fatal hypernatremia due to accidental administration of table salt]. An Pediatr 53:495–498
30. Meyer-Heim A, Landau K, Boltshauser E (2002) Aknetherapie mit Folgen–Pseudotumor cerebri durch Hypervitaminose A [Treatment of acne with consequences – pseudotumor cerebri due to hypervitaminosis A]. Praxis 91:23–26
31. Quereshi UA, Bhat JI, Ali SW, Mir AA, Kambay AH, Bhat IN (2010) Acute salt poisoning due to different oral rehydration solution (ORS) packet sizes. Indian J Pediatr 77:679–680
32. Zhu BL, Ishida K, Fujita MQ, Maeda H (1998) Infant death presumably due to exertional self-overheating in bed: an autopsy case of suspected child abuse. Jpn J Leg Med 52:153–156
33. Ommaya AK, Gennarelli TA (1974) Cerebral concussion and traumatic unconsciousness. Correlation of experimental and clinical observations of blunt head injuries. Brain 97:633–654
34. Hymel KP, Bandak FA, Partington MD, Winston K (1998) Abusive head trauma? A biomechanics-based approach. Child Maltreat 3:116–128
35. Burton D (2007) Static v. dynamic loading: why the WTC towers fell so fast. http://www.burtonsys.com/staticvdyn/. Accessed 5 Aug 2021
36. Duhaime AC, Eppley M, Margulies S, Heher KL, Bartlett SP (1995) Crush injuries to the head in children. Neurosurgery 37:401–406. discussion 407
37. Prasad MR, Ewing-Cobbs L, Baumgartner J (1999) Crush head injuries in infants and young children neurologic and neuropsychologic sequelae. J Child Neurol 14:496–501
38. Center for Disease Control and Prevention (2007) Injury center. http://www.cdc.gov/ncipc/wisqars/nonfatal/definitions.htm. Accessed 16 Sep 2021
39. Nordic Medico-Statistical Committee (2007) Classification of external causes of injuries. NOMESCO, Copenhagen
40. WHO Regional Office for Europe (2013) In: Sethi D, Bellis M, Hughes K, Gilbert R, Mitis G, Galea G (eds) European report on preventing child maltreatment. https://www.euro.who.int/__data/assets/pdf_file/0019/217018/European-Report-on-Preventing-Child-Maltreatment.pdf Accessed 16 sept 2021
41. Hobbs CJ, Hanks HGI, Wynne JM (1993) Child abuse and neglect – a clinician's handbook. Churchill Livingstone, London
42. 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
43. 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
44. Hammond H (2009) Clinical assessment in suspected child abuse cases. In: Busutil A, Keeling JW (eds) Paediatric forensic medicine & pathology. Hodder Arnold, London, pp 1–23
45. Bilo RAC, Oranje AP, Shwayder T, Hobbs CJ (2013) Cutaneous manifestations of child abuse and their differential diagnosis – blunt force trauma. Springer, Cham
46. Labbé J, Caouette G (2001) Recent skin injuries in normal children. Pediatrics 108:271–276
47. Langlois NE, Gresham GA (1991) The ageing of bruises: a review and study of the colour changes with time. Forensic Sci Int 50:227–238
48. Kemp AM, Maguire SA, Nuttall D, Collins P, Dunstan F (2014) Bruising in children who are assessed for suspected physical abuse. Arch Dis Child 99:108–113
49. Pierce MC, Kaczor K, Aldridge S, O'Flynn J, Lorenz DJ (2010) Bruising characteristics discriminating physical child abuse from accidental trauma. Pediatrics 125:67–74
50. Pierce MC, Kaczor K, Lorenz DJ, Bertocci G, Fingarson AK, Makoroff K, Berger RP, Bennett B, Magana J, Staley S, Ramaiah V, Fortin K, Currie M, Herman BE, Herr S, Hymel KP, Jenny C, Sheehan K, Zuckerbraun N, Hickey S, Meyers G, Leventhal JM (2021) Validation of a clinical decision rule to predict abuse in young children based on bruising characteristics. JAMA Netw Open 4:e215832
51. Warrington SA, Wright CM (2001) Accidents and resulting injuries in premobile infants: data from the ALSPAC study. Arch Dis Child 85:104–107
52. Wilson EF (1977) Estimation of the age of cutaneous contusions in child abuse. Pediatrics 60:750–752
53. McGrath A, Barrett MJ (2019) Petechiae. https://www.ncbi.nlm.nih.gov/books/NBK482331/. Accessed 5 Aug 2021
54. Strumia R, Felitti F (2013) Skin signs due to self-induced vomiting. In: Strumia R (ed) Eating disorders and the skin. Springer, Berlin
55. Cairns AM, Mok JY, Welbury RR (2005) Injuries to the head, face, mouth and neck in physically abused children in a community setting. Int J Paediatr Dent 15:310–318
56. Saukko P, Knight B (2004) The pathology of wounds. In: Saukko P, Knight B (eds) Knight's forensic pathology. Arnold, London, pp 136–173
57. Bilo RAC, Oranje AP, Shwayder T, Hobbs CJ (2013) Blunt penetrating injuries. In: Bilo RAC, Oranje AP, Shwayder T, Hobbs CJ (eds) Cutaneous manifestations of child abuse and their differential diagnosis – blunt force trauma. Springer, Cham, pp 215–216
58. Coniglio MA, Bonaccorso A, Scillieri R, Giammanco G, Pignato S (2005) Incidenti domestici nell'infanzia. Risultati di un'indagine condotta nell'area etnea [Domestic injuries in childhood. Results of a survey carried out in a Sicilian area]. Ann Ig 17:261–267
59. Makkat S, Vandevenne JE, Parizel PM, De Schepper AM (2001) Multiple growing fractures and cerebral venous anomaly after penetrating injuries: delayed diagnosis in a battered child. Pediatr Radiol 31:381–383
60. Thomas M, Cameron A (1977) Rarity of non-accidental penetrating injury in child abuse. Br Med J 1:375–376
61. Navarro B, Urban R (2004) "Overkill" im Rahmen einer Neugeborenen-Tötung ["Overkill" in a case of neonaticide]. Arch Kriminol 213:129–137
62. Rougé-Maillart C, Jousset N, Gaudin A, Bouju B, Penneau M (2005) Women who kill their children. Am J Forensic Med Pathol 26:320–326
63. Tournel G, Desurmont M, Bécart A, Hédouin V, Gosset D (2006) Child barbarity and torture: a case report. Am J Forensic Med Pathol 27:263–265
64. Koblenzer C (1987) Psychocutaneous disease. Grune & Stratton, Orlando, USA
65. Louman S, Fredriks AM, van Bellegem ACM, Teeuw AH (2018) Zelfbeschadiging bij kinderen en adolescenten [Self-harm among children and adolescents]. Ned Tijdschr Geneeskd 162:D2609
66. Berryman HE (2019) A systematic approach to the interpretation of gunshot wound trauma to the cranium. Forensic Sci Int 301:306–317
67. DiMaio VJM (1998) Gunshot wounds: practical aspects of firearms, ballistics, and forensic techniques. CRC Press, Boca Raton
68. Bayouth L, Lukens-Bull K, Gurien L, Tepas JJ 3rd, Crandall M (2019) Twenty years of pediatric gunshot wounds in our community: have we made a difference? J Pediatr Surg 54:160–164

69. Cook A, Hosmer D, Glance L, Kalesan B, Weinberg J, Rogers A, Schultz CS, Gilligan CT, Gross B, Vernon T, Ward J, Osler T, Rogers F (2019) Population-based analysis of firearm injuries among young children in the United States, 2010–2015. Am Surg 85:449–455
70. Fowler KA, Dahlberg LL, Haileyesus T, Gutierrez C, Bacon S (2017) Childhood firearm injuries in the United States. Pediatrics 140:e20163486
71. Resnick S, Smith RN, Beard JH, Holena D, Reilly PM, Schwab CW, Seamon MJ (2017) Firearm deaths in America: can we learn from 462,000 lives lost? Ann Surg 266:432–440
72. Tseng J, Nuño M, Lewis AV, Srour M, Margulies DR, Alban RF (2018) Firearm legislation, gun violence, and mortality in children and young adults: a retrospective cohort study of 27,566 children in the USA. Int J Surg 57:30–34
73. Li G, Baker SP, DiScala C, Fowler C, Ling J, Kelen GD (1996) Factors associated with the intent of firearm-related injuries in pediatric trauma patients. Arch Pediatr Adolesc Med 150:1160–1165
74. Panke TW, McLeod CG (1985) Pathology of thermal injury – a practical approach. Grune & Stratton, Orlando
75. Pounder DJ (2000) Burns and scalds. In: Siegel J, Knupfer G, Saukko P (eds) Encyclopedia of forensic sciences. Elsevier Academic Press, London
76. Richardson AC (1994) Cutaneous manifestations of abuse. In: Reece RM (ed) Child abuse. Lea & Febiger, Philadelphia, pp 167–184
77. Loder RT, Feinberg JR (2007) Orthopaedic injuries in children with nonaccidental trauma: demographics and incidence from the 2000 kids' inpatient database. J Pediatr Orthop 27:421–426
78. Sinha CK, Lander A (2013) Trauma in children: abdomen and thorax. Surgery (Oxford) 31:123–129
79. Overly FL, Wills H, Valente JH (2014) 'Not just little adults' – a pediatric trauma primer. R I Med J (2013) 97:27–30
80. Yu YR, DeMello AS, Greeley CS, Cox CS, Naik-Mathuria BJ, Wesson DE (2018) Injury patterns of child abuse: experience of two Level 1 pediatric trauma centers. J Pediatr Surg 53:1028–1032
81. Cooper A, Floyd T, Barlow B, Niemirska M, Ludwig S, Seidl T, O'Neill J, Templeton J, Ziegler M, Ross A (1988) Major blunt abdominal trauma due to child abuse. J Trauma 28:1483–1487
82. Milroy CM (2014) Blunt abdominal and thoracic injuries in children. In: Collins KA, Byard RW (eds) Forensic pathology of infancy and childhood. Springer, Berlin, pp 291–325
83. Rosenfeld EH, Johnson B, Wesson DE, Shah SR, Vogel AM, Naik-Mathuria B (2020) Understanding non-accidental trauma in the United States: a national trauma databank study. J Pediatr Surg 55:693–697
84. Shenoi RP, Camp EA, Rubalcava DM, Cruz AT (2017) Characteristics and outcomes of acute pediatric blunt torso trauma based on injury intent. Am J Emerg Med 35:1791–1797
85. Sharma MS (2016) Pediatric thoracic trauma. https://emedicine.medscape.com/article/905863-overview#showall. Accessed 5 Aug 2021
86. Reynolds SL (2018) Pediatric thoracic trauma: recognition and management. Emerg Med Clin North Am 36:473–483
87. 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 37:430–445
88. Merten DF, Carpenter BL (1990) Radiologic imaging of inflicted injury in the child abuse syndrome. Pediatr Clin N Am 37:815–837
89. Clark RE, Clark JF (1989) The encyclopedia of child abuse. New York, Fact on File Inc.
90. Cooper A (1992) Thoracoabdominal trauma. In: Ludwig S, Kornberg AE (eds) Child abuse – a medical reference. Churchill Livingstone, New York, pp 131–150
91. Huntimer CM, Muret-Wagstaff S, Leland NL (2000) Can falls on stairs result in small intestine perforations? Pediatrics 106:301–305
92. Grosfeld JL, Rescorla FJ, West KW, Vane DW (1989) Gastrointestinal injuries in childhood: analysis of 53 patients. J Pediatr Surg 24:580–583
93. Hamilton A, Humphreys WG (1985) Duodenal rupture complicating childhood non-accidental injury. Ulster Med J 54:221–223
94. Ford EG, Senac MO Jr (1993) Clinical presentation and radiographic identification of small bowel rupture following blunt trauma in children. Pediatr Emerg Care 9:139–142
95. Cobb LM, Vinocur CD, Wagner CW, Weintraub WH (1986) Intestinal perforation due to blunt trauma in children in an era of increased nonoperative treatment. J Trauma 26:461–463
96. Ledbetter DJ, Hatch EI Jr, Feldman KW, Fligner CL, Tapper D (1988) Diagnostic and surgical implications of child abuse. Arch Surg 123:1101–1105
97. Lane WG, Dubowitz H, Langenberg P, Dischinger P (2012) Epidemiology of abusive abdominal trauma hospitalizations in United States children. Child Abuse Negl 36:142–148
98. Trokel M, Discala C, Terrin NC, Sege RD (2006) Patient and injury characteristics in abusive abdominal injuries. Pediatr Emerg Care 22:700–704
99. Gilbert-Barnes E (1991) Potter's pathology of the fetus and infant. Mosby
100. Carter KW, Moulton SL (2016) Pediatric abdominal injury patterns caused by "falls": a comparison between nonaccidental and accidental trauma. J Pediatr Surg 51:326–328
101. Caniano DA, Beaver BL, Boles ET Jr (1986) Child abuse. An update on surgical management in 256 cases. Ann Surg 203:219–224
102. Sivit CJ, Taylor GA, Eichelberger MR (1989) Visceral injury in battered children: a changing perspective. Radiology 173:659–661
103. Rothrock SG, Green SM, Morgan R (2000) Abdominal trauma in infants and children: prompt identification and early management of serious and life-threatening injuries. Part I: injury patterns and initial assessment. Pediatr Emerg Care 16:106–115
104. Roaten JB, Partrick DA, Bensard DD, Hendrickson RJ, Vertrees T, Sirotnak AP, Karrer FM (2005) Visceral injuries in nonaccidental trauma: spectrum of injury and outcomes. Am J Surg 190: 827–829
105. Sheybani EF, Gonzalez-Araiza G, Kousari YM, Hulett RL, Menias CO (2014) Pediatric nonaccidental abdominal trauma: what the radiologist should know. Radiographics 34:139–153
106. Minns RA, Brown JK (ed) (2015). Shaking and other non-accidental head injuries in children. Clinics in Developmental Medicine No. 162. Cambridge University Press, p. 17
107. David TJ (2008) Non-accidental head injury – the evidence. Pediatr Radiol 38:370–377
108. Kocher MS, Kasser JR (2000) Orthopaedic aspects of child abuse. J Am Acad Orthop Surg 8:10–20
109. Loder RT, Bookout C (1991) Fracture patterns in battered children. J Orthop Trauma 5:428–433
110. McClain PW, Sacks JJ, Froehlke RG, Ewigman BG (1993) Estimates of fatal child abuse and neglect, United States, 1979 through 1988. Pediatrics 91:338–343
111. Lindberg DM, Shapiro RA, Laskey AL, Pallin DJ, Blood EA, Berger RP (2012) Prevalence of abusive injuries in siblings and household contacts of physically abused children. Pediatrics 130:193–201
112. Baartman HEM (1993) Opvoeden met alle geweld: Hardnekkige gewoonten en hardhandige opvoeders [Raising children with violence – stubborn habits and tough nurtures]. SWP uitgeverij

General Aspects of Fractures in Children

2

Rob A. C. Bilo, Arjo A. J. Loeve, Simon G. F. Robben, and Rick R. van Rijn

Contents

R. A. C. Bilo
Veilig Thuis Rotterdam Rijnmond (Center for the Reporting of Child Abuse, Domestic Violence and Elder Abuse), Rotterdam, The Netherlands

A. A. J. Loeve
Department of Biomechanical Engineering, Faculty of Mechanical, Maritime and Materials Engineering, Delft University of Technology, Delft, The Netherlands
e-mail: A.J.Loeve@tudelft.nl

S. G. F. Robben (✉)
Department of Radiology, Maastricht University Medical Center, Maastricht, The Netherlands
e-mail: s.robben@mumc.nl

R. R. van Rijn
Department of Radiology and Nuclear Medicine, Amsterdam UMC, University of Amsterdam, Amsterdam, The Netherlands

Department of Forensic Medicine, Netherlands Forensic Institute, The Hague, The Netherlands
e-mail: r.r.vanrijn@amsterdamumc.nl

R. A. C. Bilo et al. (eds.), *Forensic Aspects of Paediatric Fractures*, https://doi.org/10.1007/978-3-031-12041-1_2

2.1 Introduction

2.1.1 Definition

A fracture is a partial or complete disruption of the continuity of bone or cartilage, due to mechanical forces exceeding the strength of the bone or cartilage to withstand these forces.

2.1.2 Epidemiology

Fractures regularly occur in children. Most fractures in children are due to accidental trauma [1].

Landin did several large studies in the Malmö region in Sweden [2, 3]. In 1983, he reported on a retrospective study regarding 8642 children. It concerned all fractures in children treated over a period of 30 years in Malmö (between 1950 and 1979). In 1997 he added more recent data to his original study. In the Malmö region, the chance to sustain a fracture between birth and the age of 16 was in the reported period 42% for boys and 27% for girls. The overall annual incidence of fractures in children turned out to be 2.1% (2.6 for boys; 1.7 for girls). This percentage did not differ significantly from the reported incidence of the annual incidence of 1.6% reported for boys and girls in an English study of children with fractures treated clinically as well as in outpatient clinics [4].

In the period after the reports of Landin in 1983 and 1997 and Worlock in 1986, the incidence of paediatric fractures, reported by other authors, only slightly fluctuated, depending on the studied population and the country of origin (Table 2.1) [4, 6, 9].

Rennie et al. found that the incidence of fractures increased with age [5]. They also found that most fractures were due to falls from a bed (height <1 m). Most fractures were found in the upper extremity. Fractures in the lower limbs were mostly due to a trauma in which the limb was twisted and to road traffic accidents.

Hedström et al. found that the most common fracture site was the distal forearm [6]. The most common type of trauma mechanism was a fall on the outstretched hand (FOOSH). The peak incidence occurred at 11–12 years in girls and at 13–14 years in boys, with a male-to-female incidence ratio of 2 to 1. They also found variations in mechanisms and activities with age, and over time. They noticed a slight increase of the incidence in the period between 1998 and 2007 compared to the first period they evaluated (1993 to 1997). According to them this partly could be due to changes in children's activity patterns over time.

Mäyränpää et al. noticed an increase in the incidence of fractures in the period between 1967 and 1983, but also noticed a significant decrease between 1983 and 2005 [7]. This decrease was largest in children between the ages of 10 and 13 years and most marked in hand and foot fractures. However, the incidence of forearm and upper arm fractures increased significantly (about one-third) in this period. Fractures were mainly due to falls when running or walking or falls from heights under 1.5 m. Fracture incidence peaked at 10 years in girls and 14 years in boys.

In 2010, Mathison and Agrawal reported an increasing incidence of paediatric fractures despite public health measures to prevent childhood injuries [9]. The incidence increased with age with a peak between the age of 12 and 15 years. Boys were over 50% more likely to sustain a fracture than girls. They concluded that traditional play activities continued to be the prevalent cause for fractures, but that there also was an increase in new sport and recreational activities, e.g. skateboarding, that carried significant fracture risk. They saw a higher incidence during the summer season. Fractures were seen more often in children from families with a low socioeconomic status, obese children, and children with risk taking behaviour. Furthermore it was seen in children with decreased bone density, eating disorders and inadequate nutrition, with chronic corticosteroid or performance-enhancing drugs use, and in smoking minors. Trauma from high risk taking behaviour, e.g. from sports, including extreme sports, accounted for a majority of fractures in middle and high school age children. Popular recreational play devices such as heelys, scooters, and all-terrain vehicles were highly associated with fractures [9, 10].

Table 2.1 Incidence of fractures in children

Author	Incidence/100	Male vs female	Age
Worlock and Stower [4]	1.6		
Landin [3]	2.1	2.6 vs 1.7	<16 years of age
Rennie et al. [5]	2.0	61 vs 39%	
Hedström et al. [6]	2.0	1.5 vs 1	<20 years of age
Mäyränpää et al. [7]	1.6	63 vs 37%	<16 years of age
Naranje et al. [8]	1.8		<18 years of age

Naranje et al. tried to identify the most common paediatric fractures per 1000 children between the ages of 0 and 19 years in 2010 by using 2010 National Electronic Injury Surveillance System (NEISS) database and 2010 US Census information [8]. They found that children between 10 and 14 years of age had the highest risk of sustaining fractures. Forearm fractures were the most common, accounting for 17.8% of all fractures, whereas finger and wrist fractures were the second and third most common, respectively. Finger and hand fractures were most common for age groups 10 to 14 and 15 to 19 years, respectively.

Wilkins and Aroojis stated that 6.8% of the fractures sustained by children in the first 16 years of their life is severe enough to require admittance to hospital [11]. Slightly less than 20% of children who visit a hospital for sustained injuries appear to have sustained a fracture. According to Naranje et al., most paediatric fractures can be treated on outpatient basis, with only 1 of 18 fractures requiring hospitalization or observation [8].

In conclusion, the incidence of fractures in childhood is high, approximately 2%. Boys fracture their bones twice as often as girls. The incidence increases with age with a peak incidence between 10 and 15 years. The distal arm is most frequently affected and FOOSH is the most common trauma mechanism.

2.2 Diagnosis and Differential Diagnosis of Fractures in Children

2.2.1 Clinical Presentation

Fractures, irrespective of the circumstances under which the fractures were sustained, are usually identified based on the medical history and the presence of clinical manifestations such as pain, swelling, inability to move, and abnormal alignment. For a fracture without clinical manifestations generally no medical help will be sought.

Pain will occur at the same time as the fracture. Swelling may occur immediately after the fracture occurred. Pain will increase when the afflicted body part is moved. This may be an active movement, such as when the child wants to move the afflicted body part, or a passive movement, e.g. when a parent or caretaker wants to change the diaper or bathes the child.

When there is no dislocation of the fracture parts, the acute inflammatory symptoms around the fracture may be limited to just a few days. Up to toddler age, children may be pain-free within a few days after the fracture has been sustained, whereas in older children and adults this may take much longer [12].

In young children, however, fractures frequently have an occult course [13, 14]. There are various reasons for this phenomenon. Non-mobile children have a limited movement pattern, which makes it harder to notice when a child does not move a body part over a longer or shorter period of time or whether its movement is somewhat restricted. Secondly, the periosteum acts as a splint, resulting in a lesser chance of mutual movement of the separated bone parts. And finally, if the fracture was inflicted, seeking medical advice is often postponed for a few days. During that period there may even be spontaneous recovery to such an extent that it is or seems no longer necessary to seek medical advice.

2.2.2 Differential Diagnosis

The differential diagnosis of fractures in a forensic paediatric evaluation demands differentiating between

1. Fractures or mimics of fractures (Sect. 2.4; Chap. 14).
2. Different causes of fractures (Sect. 2.5; Chaps. 5–14) in skeletally immature or mature patients and in normal or weakened bone.
3. Different circumstances under which the fracture can be sustained (Sect. 2.6; Chaps. 5–14).

Doctors involved in a forensic paediatric evaluation (e.g. paediatricians, radiologists, and forensic doctors) should work together in a structured manner. Table 2.2 provides an example of a structured approach. Doctors should follow, as far as possible and reasonable, all steps, before concluding what caused the fracture in a specific child and under which circumstances this fracture was sustained. Central to this diagnostic process is taking a detailed clinical history. Furthermore, the age and level of development of the child should be taken into consideration: the younger the child, the more limited his/her mobility, and the more probable that an injury, e.g. a fracture, was inflicted. In the differentiation, known trauma mechanisms and biomechanical aspects of fractures should also be taken into consideration (this chapter; Chaps. 5–12). Other factors that should be taken into account are the distribution of the fractures over the skeleton and the context in which the fractures were sustained (Chaps. 5–14).

During childhood, fractures are usually the result of accidents [15]. The differential diagnosis, apart from falls or accidents, witnessed by an independent person, or periosteal reactions that resemble a healing fracture, can be very comprehensive (Chaps. 5–14). One should realize that a suspicion of an inflicted fracture in a child also suggests the use of severe violence, probably by (one of) the parents. This emphasizes the importance of a structured approach, which should lead to a correct identification and prevent overhasty conclusions.

In this process, the (paediatric) radiologist is eminently important for an adequate diagnosis and protection of the

Table 2.2 Evaluation of fractures in young children

Step 1	Take a detailed clinical history Diagnostic procedures, focused on the initial signs and symptoms (medical history, radiology, laboratory)
Step 2	Fracture or mimic (Sect. 2.3)
If the diagnosis is a fracture	
Step 3	Describe the individual fracture(s) (Sect. 2.4)
	• Anatomic location, type of fractured bone, affected part of the bone. • Type of fracture and direction of the fracture line • Position and relationship of fracture components • Complications
	Describe all fractures
	• Number. • Distribution: – Axial and/or peripheral – Symmetric or asymmetric – Weight-bearing/non-weight-bearing parts of the skeleton • Age of the fractures and in case of multiple fractures differences in age/recent versus old (known and unknown) fractures
Step 4	Describe the skeleton
	• Configuration of the bones and the whole skeleton, i.e. the presence of underlying metabolic diseases and/or skeletal dysplasias • Findings suggesting skeletal lesions, like normal variants or 'wormian bones'
Step 5	Describe the child
	• Age and level of development • Known/suspected underlying pathology • Other injuries
Step 6	Test the plausibility of the clinical history by using evidence-based scientific data concerning
	• Probability of accidental versus non-accidental circumstances related to the age and level of mobility of the child • Fracture biomechanics

child at the moment that it is suspected that the fracture(s) was (were) inflicted. The radiologist is expected to be able to [16, 17]:

- Perform a correct radiological examination, according to the international standards.
- Detect the radiological abnormalities that are suggestive of (inflicted) fractures in both suspect and non-suspect cases.
- Distinguish between radiological abnormalities that are suspect for (inflicted) fractures and normal variants or disorders, simulating a healing or healed fracture.
- Reconstruct theoretically the causing trauma mechanism, based on the characteristics of the fracture and the known mechanisms, described in the medical literature.
- Evaluate whether the fracture and the known underlying trauma mechanisms are compatible with the given statements of the child and/or parents regarding its origin.
- Summarize which findings in the radiological evaluation could indicate accidental or non-accidental circumstances.
- Date fractures, based on the findings on imaging, within the limitations of scientific knowledge.

To fulfill these expectations it is essential that the radiologist who evaluates the characteristics of paediatric fracture(s) has sufficient knowledge of the clinical history of the patient, of known causing trauma mechanisms, and of paediatric radiology.

2.3 Fracture or Mimic

If clinical symptoms or findings on imaging are suggestive for a fracture, the first step in the (differential) diagnostic process, after taking a detailed clinical history and appropriate diagnostic procedures, is to exclude that the finding is a true mimic. A true mimic is defined here as a normal variant or a disorder, which appears on imaging simulates as a fresh or a healing or healed fracture [18]. In Table 2.3 an overview is given of normal variants and disorders that mimic healing or healed fractures. In Chap. 14, these normal variants and disorders are discussed extensively.

Some of the mimics, like Raine syndrome (OMIM #259775, osteosclerotic bone dysplasia), McCune-Albright syndrome (OMIM #174800, fibrous dysplasia), Alagille syndrome (OMIM #118450, arteriohepatic dysplasia) and metabolic disorders like the mucopolysaccharidoses may show findings on imaging that are similar to those showing in healing or healed fractures. Others may show findings suggestive of recent fractures, e.g. spondylometaphyseal dysplasia corner fracture type (OMIM #184255). Children with these syndromes/disorders however nowadays often are already recognized at birth or early in infancy because of the presence of significant additional findings.

If a real fracture is found in a child with a disorder with an increased risk for fractures due to weakening of the bone (e.g. osteogenesis imperfecta), this disorder should not be considered to be a true mimic of a fracture. There is a real fracture and a fracture is always caused by trauma in which the loading of the bone exceeded the maximum load-bearing capacity (Sect. 2.5.3.4: fatigue fractures in weakened bone). Finding a fracture in a child with a disorder does not automatically indicate under which circumstances the fracture was sustained (Sect. 2.6). Fractures due to weakening of bone can be considered to be a mimic of inflicted fracturing in child abuse, because an adequate clinical history may be lacking.

Some disorders can be seen as true mimics, while also showing an increased risk of fractures. Menkes syndrome

Table 2.3 Normal variants and medical conditions, mimicking (healing) fractures (not all inclusive) (see also Chap. 14)

	Examples
Normal variants	• Subperiosteal new bone formation (shaft of femur, tibia and humerus, usually bilateral) in normal, healthy neonates and infants • Normal metaphyseal variants • Accessory growth centres • Unfused growth plate of the shoulder • Unfused apophysis of the fifth metatarsal • Accessory skull sutures • Accessory ossicles • Vascular/nutrient lines
Haematological disorders, malignancies, and benign tumours	• Sickle cell anaemia • Leukaemia • Ewing sarcoma • Osteoid osteoma and osteoblastoma
Congenital/genetic disorders	• Caffey's disease (infantile cortical hyperostosis) (OMIM # 114000) • Bone dysplasias • Metabolic disorders, e.g. mucopolysaccharidoses • Alagille syndrome (OMIM #118450) • Copper deficiency/Menkes syndrome (OMIM #309400) • Metaphyseal chondroplasia (type Schmid) (OMIM #156500) • Spondylometaphyseal dysplasia ('corner fracture type') (OMIM #184255)
Infections or healing/healed infections	• Osteomyelitis and chronic relapsing multifocal osteomyelitis • Congenital syphilis • Septic arthritis
Vitamin deficiencies	• Vitamin D deficiency (rickets) • Vitamin C deficiency (scurvy).
Vitamin overdose	• Hypervitaminosis A/vitamin A toxicity • Vitamin E therapy
Growth disturbance	• Harris lines: lines of increased bone density due to growth retardation or cessation (Sect. 5.12.2)
Medical intervention	• Prostaglandin E • Intra-osseous vascular access needles

(OMIM #309400) and copper deficiency, for example can be seen as true mimics, because of the presence of metaphyseal spurs, suggesting classical metaphyseal lesions and periosteal reactions, which appear as healing fractures. In both disorders however there is also an increased risk of fracturing, because of weakening of the bone (osteoporosis) due to disturbances in bone metabolism and for that reason may mimic inflicted fractures.

2.4 Fracture Description

As already stated in Sect. 2.1.1, a fracture is the partial or complete disruption of the continuity of a bone, due to mechanical forces exceeding the strength of the bone or cartilage to withstand these forces.

Fractures have different appearances on imaging:

- Most fractures are visible as a lucent (black) line on radiographs or CT.
- When a fracture is impacted, due to compressional forces, the overlapping fragments can produce a dense (white) line on imaging. In the spine these compressional forces result in a loss of height of vertebral bodies (compression fracture).
- Incomplete fractures may present as small interruption of the smooth continuous cortical curve of bones, like buckles or acute angulations.
- Avulsion fractures cause separation of small bony fragments from the metaphysis, or increased distance and/or malalignment of secondary ossification centres from the metaphysis.
- Fractures through a growth plate result in malalignment between epiphysis and metaphysis, with or without variations in thickness of the growth plate.
- Occult fractures are fractures that are present but not visible on imaging. This can occur when the fracture is small and the X-ray beam is not parallel to the fracture plane, or when the fracture is in exactly the same plane as the CT slice. Occult fractures usually become apparent on imaging 2 weeks later because of subperiosteal new bone formation and widening of the fracture line due to reparative bone resorption.

Fractures are described according to [19–23]:

- The anatomic location, the type of fractured bone, and the affected part of the bone.
- The type of fracture and the direction of fracture lines.
- The position and the relationship of the fracture parts.
- The complications.

2.4.1 Anatomic Location and Type of Bone

The first step in the description of a fracture is the correct anatomic identification of the fractured bone, the type of the fractured bone and, related to the type of bone, the part of bone that is affected.

2.4.1.1 Identification of the Fractured Bone

The identification of a fractured bone should be done according to generally accepted and standard anatomical terms.

2.4.1.2 Bone Type

Generally bone consists of trabecular bone and cortical bone and it contains the bone marrow. It offers support, regulates the calcium metabolism and production of blood cells.

There are five types of bones in the skeleton with different characteristics and different functions: long, short, flat, sesamoid, and irregular bones (Fig. 2.1) [24, 25].

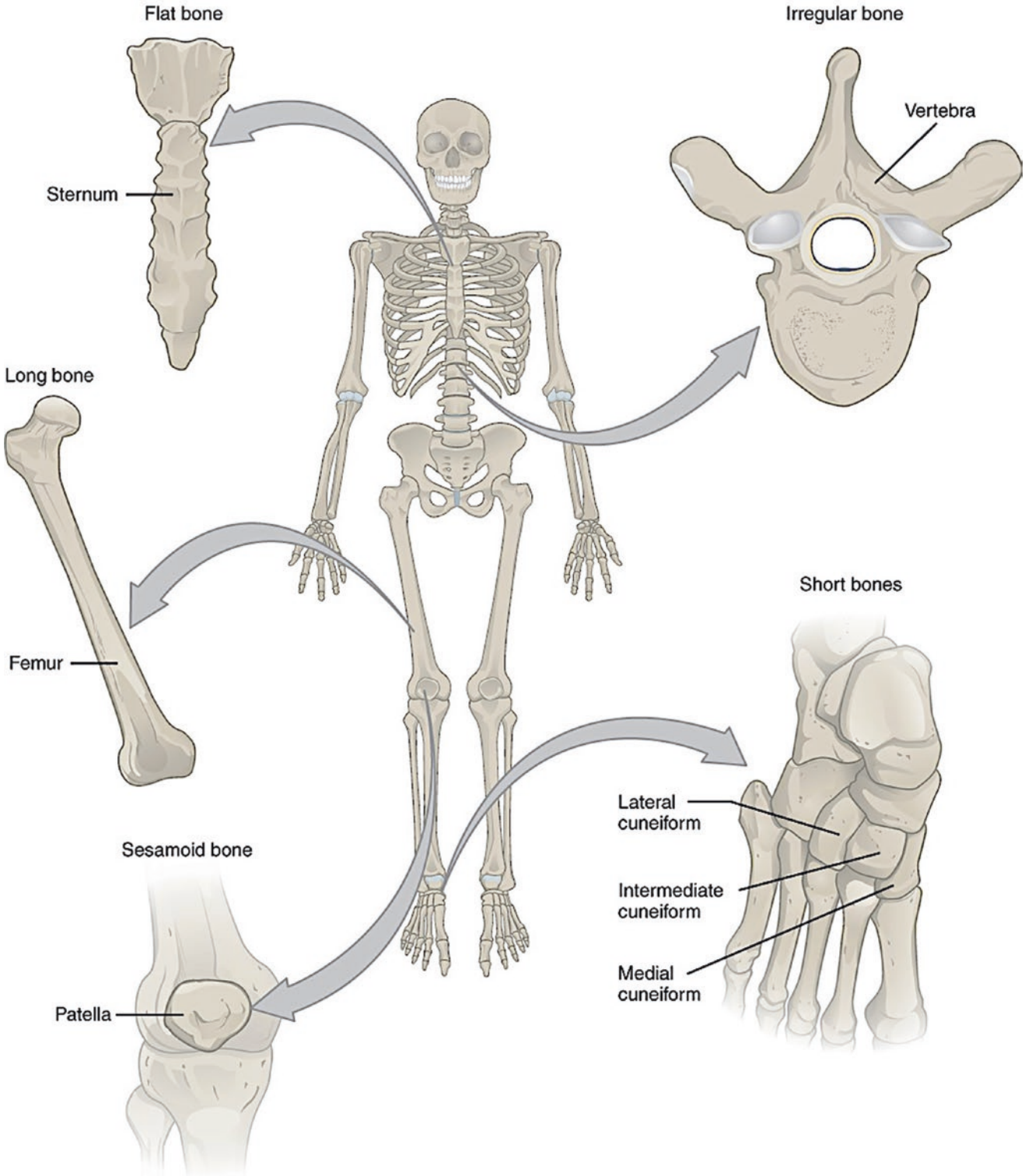


Fig. 2.1 Types of bones (OpenStax College, 2013: licensed under the Creative Commons Attribution 3.0 Unported license, https://commons.wikimedia.org/wiki/File:601_Bone_Classification.jpg)

Long Bones

Long bones are located in the appendicular skeleton (upper and lower limbs), have a cylindrical shape and are longer than they are wide. Long bones have several specific functions of which the most important are supporting the body weight and facilitating movement (articulation).

They consist of diaphyses, epiphyses, metaphyses, and physes (growth plates). Long bones typically have a wide

metaphysis with thin cortex and an abundancy of trabecular medullary bone whereas the diaphysis has a thick cortex and relative lack of trabecular bone (medullary cavity). The physis (growth plate) is situated between the epiphysis and metaphysis. At birth, virtually all epiphyses are cartilaginous and therefore not visible on radiographs. Ossification slowly progresses and is complete at adolescence. Some long bones, like the clavicle do not have a medullary cavity.

Typical long bones, like the humerus, radius, ulna, femur, tibia, and fibula, have two epiphyses (one at both ends), whereas some of the smaller long bones only have one epiphysis. Examples of smaller long bones are the phalanges, metacarpals, and metatarsals.

Short Bones

Short bones are located in the wrist and ankles. Short bones are more or less equal in length, width, and thickness and can have any shape. Most of these bones are named according to their shape, e.g. the carpals in the wrist (scaphoid, lunate, triquetral, hamate, pisiform, capitate, trapezoid, and trapezium) and the tarsals in the ankles (calcaneus, talus, navicular, cuboid, lateral cuneiform, intermediate cuneiform, and medial cuneiform).

The function of short bones is to provide support and stability in parts of the skeleton, that are intended for strength and compactness with limited movement.

Short bones behave like epiphyses, both in normal growth and in pathology: they are almost all cartilaginous at birth and have a slow progressive ossification, they have a poor vascularization and therefore are prone to malunion or osteonecrosis after a fracture.

Flat Bones

Flat bones are located in the skull (e.g. frontal, parietal, and occipital bone), the thoracic cage (sternum, ribs and scapula), and the pelvis (ilium, ischium, and pubis) and have a thin and curved shape with two prominent surfaces.

Flat bones form the boundaries of certain body cavities and their function is to provide protection for internal organs, like the brain, heart, lungs, and pelvic organs. Flat bones also provide large areas of attachment for muscles and are a major source of red bone marrow.

Sesamoid Bones

Sesamoid ('sesame seed shaped') bones are small and round or irregular bones, which are embedded in tendons and joint capsules. Sesamoid bones vary from person to person in number and placement. The most common locations are the tendons of the hands, knees, and feet. Examples of this type are the patella and the pisiform bone.

The function of sesamoid bones is to provide protection of the tendons and the joint capsules by absorbing and redistributing weight-bearing forces and in that way decreasing stress on and wear of the tendons [26].

Irregular Bones

Irregular bones are located in the axial skeleton (skull, spine, and pelvis). They vary in shape and structure and therefore do not fit into one of the other categories. They often have a complex shape. Examples of this type are parts of the skull, bones in the base of skull and some of the facial bones (e.g. temporal bone, zygoma, inferior nasal concha, mandibula), the vertebrae and parts of the pelvis (sacrum, coccyx, hip bone).

The function of irregular bones is to provide protection of internal organs, e.g. the vertebrae protect the spinal cord and the bones in the base of the skull protect (together with the flat bones of the skull) the brain.

A specific type of irregular bones are the so-called pneumatic bones, which are characterized by the presence of large air spaces, e.g. the maxilla, the mastoid, and the ethmoid. The function of these bones is not exactly known. These bones are relatively light and therefore considerably reduce the weight of the skull. These bones probably also play a role in the resonance of sound and in the temperature regulation of inspired air. Moreover, most of them are extremely thin-walled and therefore prone to fracture during trauma.

Because of the complex shape of irregular bones, it is often difficult to detect fractures with conventional radiographs and CT is preferred in cases with clinical suspicion but normal radiographs.

2.4.1.3 Affected Part of the Bone

The description of the affected part of the bone depends on the type of bone.

In case of a long bone fracture, e.g. a fracture of the femur, the anatomic location of the fracture can be specified as diaphyseal (proximal, middle, or distal part), metaphyseal (proximal, distal), physeal, or epiphyseal (intra-articular, extra-articular). The fracture can further be specified by describing more specific anatomic terminology of the location, like condyle, malleolus, plateau, fossa, and tuberosity [19].

In case of flat bone fractures, e.g. rib fractures, the anatomic location should be specified by giving the number, ranking and laterality of broken ribs, as well as the location of the fracture(s) in the broken ribs: at the costochondral junction, in the anterior, lateral and posterior costal arch, or near the head or neck of the rib. For research purposes more elaborate descriptions have been proposed, which could also be used in detailed legal reports [27].

In case of an irregular bone fracture, e.g. a vertebral fracture, the anatomic location can be specified as fractures of cervical, thoracic, or lumbar vertebrae and of the corpus, arch, or transverse/spinous process. In a clinical situation classification schemes such as the ThoracoLumbar Injury Classification and Severity Scale (TLICS) could be used [28].

The reader is referred for an extensive description of affected parts of the different bones to Chaps. 5–12.

Table 2.4 Type of fracture and direction of fracture lines in long bones

Complete fracture	Fracturing of a bone, causing separation into two or more pieces
Simple	• Only one single fracture line (a.k.a. single)
• Longitudinal	• The fracture line runs parallel to the long axis of the bone (a.k.a. longitudinal)
• Transverse	• The fracture line runs more or less perpendicular at an angle of less than 30 degrees in relation to the long axis of the bone
• Oblique	• The fracture line runs more or less oblique (diagonal) at an angle of over 30 degrees in relation to the long axis of the bone
• Spiral	• The fracture line 'circles' around the long axis (cork-screw), and the fracture line runs oblique in relation to the central axis
	With conventional radiology, it is not always possible to distinguish between an oblique and a spiral fracture.
Multifragmentory	• Multiple (three or more) fragments (a.k.a. comminuted or complex)
• Wedge–butterfly	• Triangular (shape of a butterfly wing) fragment between two larger bone fragments
• Segmental	• Fracture in two places with a 'floating and unattached' segment between two well-defined fracture lines
Incomplete–partial fracture	• The fragments are still partially joined and the fracture does not completely traverse the width of the bone
• Bowing	• The bone deformed past the point at which, based on the elasticity of the bone, spontaneous recovery is the rule • No radiologically visible cortical damage
• Greenstick	• The cortex at the tension side is damaged with an intact cortex and intact periosteum at the compression side
• Torus–buckle	• The cortex at the compression side is damaged with an intact cortex and intact periosteum at the tension side

2.4.2 Type of Fracture and Direction of Fracture Lines

The type of fracture, the direction of fracture lines, and the terms used to describe type and direction are determined mainly by the type of fractured bone. An overview of terms in long bones is given as an example in Table 2.4. The reader is referred to Chaps. 5–12 for more detailed information.

2.4.3 Position and Relationship of the Fracture Components

The position and relationship of the fracture components and the terms used to describe position and relationship again are determined mainly by the type of fractured bone. An overview of terms in long bones is given as an example in Table 2.5. The reader is referred to Chaps. 5–12 for more detailed information.

Table 2.5 Position and relationship of the fracture components

Fracture displacement	abnormal position of the distal fracture fragment in relation to the proximal bone
Non-displaced	• The fragments of the fracture are aligned
Displaced	• The fragments of the fracture are not aligned and there is a gap between the two ends of the bone
• Translated or ad latus	• The bone fragments are sideways displaced
• Angulated	• The fragments are displaced with an alteration of the normal axis of the bone, causing the distal portion to point in another direction than the proximal part: • Dorsal/palmar • Varus/valgus
• Rotated	• The distal component is rotated compared to the proximal component.
• Shortened	• The total bone length is reduced: • Impaction = telescoping of two fragments into each other, • Compression = crushing of two fragments, causing the broken bone to be wider or flatter in appearance, • Compression and wedging = (usually occurring in the vertebrae) the front portion of a vertebra in the spine has collapsed • Overriding = overlap of two completely displaced fragments (a.k.a. 'dinner fork' or 'bayonet' fracture)
• Avulsion	• A fragment of the bone is pulled off, often by a tendon or ligament

Table 2.6 Complications

Concerning the stability of the fracture	
Stable	• Fragments line up and are barely out of place.
Unstable	• Fragments tend to shift further out of place.
Concerning the integrity of overlying skin and soft tissues	
Closed	• The bone is broken, but the overlying skin has not been ruptured and remains intact (a.k.a. simple), without or with soft tissue injury.
Open	• The bone is broken and the overlying skin and soft tissues are either pierced by the broken bone or by the blow that has broken the skin at the time of the fracturing of the bone. The bone may or may not be protruded through the skin (a.k.a compound or complex). An open fracture carries a high risk of infection.
Concerning joint involvement	
No	
Yes	• Articular • Joint dislocation

2.4.4 Complications

In Table 2.6, an overview is given of possible complications of fractures concerning the stability of the fracture, the integrity of overlying skin and soft tissues and joint involvement.

2.5 Cause of Fractures (Mechanism)

2.5.1 General Aspects

In a forensic setting, a (physical) trauma is defined as an event which can result in an injury (physical harm, bodily injury, physical injury) (see Sect. 1.4). An injury is defined as any wounding or physical damage that results from the (sudden) subjection of the body or parts of the body to amounts of energy that exceed the threshold of mechanical tolerance, in other words, that are beyond the body's ability to absorb the transferred energy, with or without externally visible damage to the skin or the mucous membranes and/or with or without signs of damage to the skeleton or internal organs [29, 30]. In brief an (hard or soft tissue) injury is caused by loading of that tissue beyond its failure threshold (the maximum load-bearing capacity). In case of fractures (hard tissue injury): fracturing of bone or cartilage will occur when the loading of a bone or the cartilage exceeds the failure threshold.

Fracturing can occur in normal bone, but also in abnormal/weakened bone. In normal bone the loading will have to be substantial with a high transfer of energy. If a bone is fractured and there is no evidence of a trauma with a high transfer of energy, this may indicate the presence of generalized or more localized abnormal/weakened bone.

Although high and a low-energy trauma are well-accepted terms in the medical literature one should realize these terms are not well defined by exact numbers and measures, but by using general descriptions and comparing types of trauma, supported by examples of types of accidents. Like high and low pressure in static loading (Sect. 1.5.2.1). These terms should be considered subjective and relative with a grey area between low- and high-energy transfer and depending on the context in which these terms are used.

Often used synonyms are trauma (collisions) with high or low transfer of energy and high or low velocity trauma (collisions).

What type of injury is sustained, depends not only on the amount of transferred energy, but also on the specific characteristics of the trauma:

- Type of mechanical trauma:
  - Blunt force trauma: non-penetrating/penetrating.
  - Sharp force trauma: penetrating/non-penetrating.
  - Compression/crushing, tension, shearing and bowing, and combinations of these mechanisms, e.g. compression and bowing (see Chap. 12, concerning long bone fractures).
- Type of collision:
  - Moving object impacting static body or moving body impacting static object.
  - Body and object both moving: same direction (front to back collision), opposite direction (front to front collision), and/or angled (side to side collision).
- The amount of transferred (=absorbed and returned) energy.
- The nature of the object and the impact site on the body, including the structures underneath the skin.

2.5.2 High-Energy Trauma

A high-energy trauma is commonly described in the literature as a trauma in which the body of a person is exposed to the transfer of high amounts of energy. The transfer of energy can be mechanical or thermal (heat or cold, but also temperature generated by chemical agents or electricity) in origin.

Mechanically transferred energy can be converted into kinetic energy. Kinetic energy is the energy contained in a moving object or body. The amount of the transferred kinetic energy (KE) can be calculated and is determined by the mass and velocity of the moving body/object: $KE = ½ \times mass \times velocity^2$ (mass in kilograms, speed in metres per second, kinetic energy in joules). This formula shows that velocity is a more important determinant of the amount of transferred kinetic energy than mass. If the mass doubles, the transferred kinetic energy doubles but if the velocity doubles, kinetic energy quadruples.

A high-energy trauma usually will result in more serious injuries, like fractures, intracranial injuries, neck injuries, and/or injuries to internal organs, compared to a low-energy trauma.

Although exact numbers and measures concerning high- and low-energy trauma (collisions) are lacking guidelines for adults make use of examples like:

- Long-distance falls (at least 2–3 times body length).
- Motor vehicle accidents:
  - Motor vehicle versus pedestrian—speed of vehicle above 10 km/h.
  - Motor vehicle versus pedestrian—pedestrian run over or thrown.
  - Motor vehicle collision without seat belt—speed above 35 km/h.
  - Motor vehicle collision with seat belt—speed above 45 km/h.
  - Motor vehicle versus moped or motor collision with a speed difference above 35 km/h.
  - Accident in which the vehicle has been moved more than 7 metres.
  - Accident in which the engine or a wheel of the car has penetrated into the passenger compartment.
  - Distortion of the steering wheel.

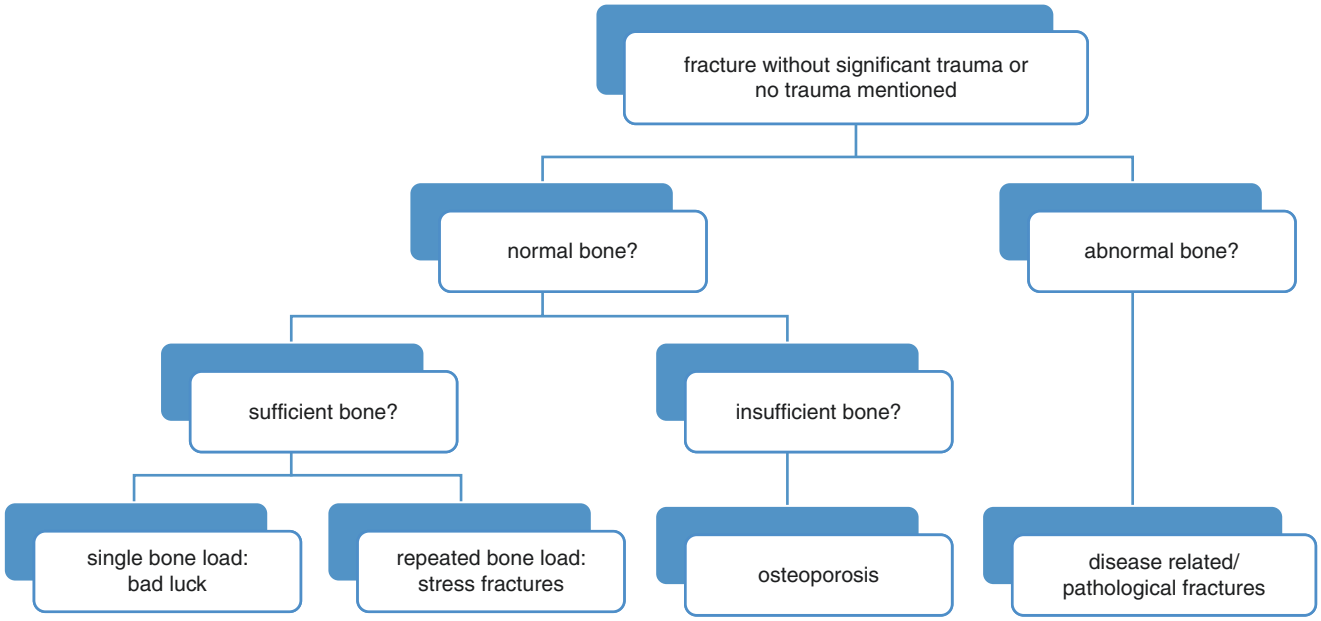


Fig. 2.2 Flow chart for the evaluation of a fracture when no (significant) trauma is reported

- Release of airbags.
- Accident with a car with an indentation of the passenger compartment above 35 cm on the victim's side and/or above 50 cm on the other side.
- Collision in which the front axle of a car has been moved backwards.
- A star break in the windscreen caused by the unrestrained passenger.
- Hair and/or blood on the interior mirror.
- Knocking over of the vehicle.
- Ejection from a vehicle.
- Helmet damage or no helmet worn.
- Seriously injured or deceased victim in the vehicle.
- Vehicle deformation.

The reader is referred to Chaps. 5–12 for an extensive description of fractures in children due to high-energy trauma.

2.5.3 Low-Energy Trauma

In the medical literature, a low-energy trauma is described in many different ways. This is usually done by using examples, just like in high-energy trauma, e.g. a slip, a trip, or a fall from standing height or less. Sometimes it is described as a trauma due to the transfer of an amount of energy of which it is assumed that such an amount would not result in a fracture. Finally it may be described merely by excluding the presence of a high-energy trauma [31].

If a bone is fractured and there is no evidence of a high-energy trauma or there is only a minor trauma or even no trauma mentioned in the medical history, one should start by answering the questions, which are visualized in Fig. 2.2.

2.5.3.1 Low-Energy Trauma: Normal Sufficient Bone—Single Load

Despite the fact that most fractures are caused by the transfer of high amounts of energy, caused by large mass or high velocity, one should realize that fractures can also be caused by a trauma that at first sight seems to be a low-energy trauma (Sect. 2.5.1). In such a seemingly low-energy trauma, it is often the case that the location where a small force acts creates a high torque around a rotation point, relatively far away from that location. This results in a large force at a fracture site close to the rotation point, due to the difference in moment arms, amplifying the small input force. Strictly speaking a small force acting over a large distance (force x distance = work) still creates a large transfer of energy. Nonetheless in a clinical setting this is considered to be a low-energy trauma. These fractures are sometimes characterized as 'bad luck'.

Probably the most common fracture in this category is the so-called toddler's fracture (Childhood Accidental Spiral Tibia (CAST) fracture), although Jaimes et al. are of the opinion that this fracture is the most common stress fracture (in biomechanics known as fatigue fracture) in young children [32]. The toddler's fracture is a non-displaced oblique fracture of the tibia shaft or the distal portion of the tibia. This type of fracture typically occurs between the age of

9 month and 3 years at the onset of ambulation [32]. A child with this fracture will refuse to bear weight on the fractured leg. The fracture probably is the result of an often not recognized acute more or less minor trauma, e.g. a torsional force when a toddler stumbles and falls on a positioned foot. According to Jaimes et al., toddler's fractures may also (less common) occur in other weight-bearing parts of the skeleton, like the fibula, the posterior part of the calcaneus, the base of the cuboid, and the talus [32].

In older children, a low-energy trauma in normal sufficient bone due to a single load may also result in a fracture: spraining an ankle during football is considered to be a low-energy trauma. The same movement, that results in spraining, may also result in a fracture of the tibia or fibula.

Some fractures, e.g. of the radius, ulna, and clavicula may also be the result of low-energy trauma in short distance falls due to falling on an outstretched hand (FOOSH) (see also Sects. 2.1.2, 8.2.2 and 12.5.3.3).

2.5.3.2 Low-Energy Trauma: Normal Sufficient Bone—Repeated Load

Repeated loading of normal sufficient bone may lead to an accumulated trauma to normal bone, resulting in overuse fractures due to a mismatch between on the one hand the burden of activity on bone and cartilage (the loading of the bone and cartilage) and on the other hand the load-bearing capacity of bone and cartilage (the intrinsic biomechanical properties and the intrinsic ability of the bone and cartilage to repair itself—the ability of bone and cartilage to absorb energy) [32, 33]. This type of fractures, due to overuse, caused by accumulated trauma, can be divided into avulsion fractures and fatigue fractures.

Avulsion Fractures

Avulsion fractures are more common in skeletally immature children and in adolescents. Examples of avulsion fractures in paediatric and adolescent patients are the classical metaphyseal lesion (Sect. 12.3.2) and the apophyseal avulsion fractures of the pelvis (Chap. 11).

Fatigue Fractures in Normal Bone: Stress Fractures

In the medical literature, fatigue fractures in normal bone and stress fractures are used as synonyms, describing (usually non-displaced hairline) fractures that occur in a bone after the bone has been subjected to repeated stresses (overuse—accumulated trauma), rather than one single sudden impact trauma. Stress-related fracturing occurs when muscles have become fatigued and are no longer able to absorb added/repeated shock. The overload of stress is then transferred to the bone, resulting in a stress fracture. None of these stresses would individually be large enough to cause a fracture, in a person without an underlying disorder with increased bone fragility, but the intrinsic ability of the bone to repair itself is exceeded by the repetitive character and frequency of the loading [33–35].

Stress fractures in adolescents and adults are most commonly sustained during sports, heavy physical exercise/labour. Most stress fractures occur in the weight-bearing bones of the lower leg and the foot (e.g. in runners: metatarsal bones). More than 50 percent of all stress fractures in adolescents and adults occur in the lower extremity [34].

Stress fractures also occur in paediatric patients. According to Griffiths, stress fractures had been described in children as young as 15 months [36]. He cited a 1942 article by Siemens, who '*described a case of bilateral fatigue fracture of the middle third of each fibula in a boy of 15 months*', and in his article himself described stress fractures in paediatric patients in the tibia (mostly in the proximal third), in the fibula and furthermore in the humerus, first rib, pelvis, medial sesamoid bone of the hallux, metatarsal bones (so-called march fractures), and femur.

The incidence of stress fractures in the paediatric population seems to be increasing, supposedly because more children are participating in organized and recreational sports at a younger age [32, 37–39]. Contributing factors to the increasing occurrence in paediatric patients is probably the combination of an underdeveloped musculoskeletal system/skeletal maturation, increased participation in competitive sport at a younger age, and increased duration and intensity of training, e.g. year-round activities [32, 38, 39]. Shelat and El-Khoury and Wu et al. also were of the opinion that many of the overuse injuries, including stress fractures, may go underreported and/or underdiagnosed, because they may also occur in other circumstances, than sporting, or are not recognized in imaging, because stress fractures can be radiographically occult [38, 39].

In older children and adolescents, mainly in athletes, fatigue fractures are found in the femur (inferior surface of the neck, shaft, and distal metaphysis), tibia, spine (spondylolysis), acromion, metatarsal and tarsal bones, pelvic bones, and upper extremity (e.g. little leaguer's shoulder or elbow, gymnast's wrist) [32, 38, 39].

Devas and Jaimes et al. mentioned that one should be aware that a stress fracture in a child might resemble an infection, e.g. osteomyelitis, or malignant bone tumour [32, 40]. Also benign bone tumours, like osteoid osteomas, may suggest stress fractures for an unexperienced radiologist. Therefore cross-sectional imaging and expert evaluation in these cases are mandatory.

2.5.3.3 Low-Energy Trauma: Normal Insufficient Bone

It is often stated that fractures in children usually are caused by trauma but that certain bone disorders, that 'weaken' the bone, also may cause fractures. This would concern bone disorders with normal but insufficient bone (= normal but not

enough bone), but also medical conditions with increased risk of fracturing due to weakened bone (see Sect. 2.5.3.4).

The statement that bone disorders may cause fractures is not correct. Bone disorders do not cause fractures, but may increase the risk of fracturing with a lower load.

A single load or repeated loading of normal but insufficient bone, e.g. in osteopenia and osteoporosis, can lead to overuse fractures due to a mismatch between the loading and the ability of the normal but insufficient bone to withstand the load.

In the medical literature, fractures in normal but insufficient bone are often referred to as spontaneous fractures: '*fractures that occur without a clear demonstrable external (= traumatic) cause'* [41]. Torwalt et al. described a 4-year-old boy with cerebral paresis and palsy after a non-accidental head trauma [41]. The post-mortem radiographs of the boy showed fractures at various stages of healing in the left humerus and both femurs, tibiae and fibulae. Based on a comprehensive investigation, child abuse, accidents, metabolic diseases, other primary and secondary bone diseases and pathological fractures could be excluded. They concluded that the multiple fractures in this boy were 'spontaneous' fractures secondary to osteopenia, although in our opinion another conclusion could also have been drawn. Based on the information given by the authors, one could also state that no cause was found and that inflicted injuries could not be excluded beyond reasonable doubt.

2.5.3.4 Low-Energy Trauma: Abnormal Bone

As stated in the foregoing section, medical conditions can lead to an increased risk of fracturing due to weakened bone. Just as in normal but insufficient bone fracturing may be caused by a single load or by repeated loading of the weakened bone.

In the medical literature, a fracture due to a medical condition with weakening of the bone is often referred to as a 'pathological' fracture: a fracture in bone tissue, that is pathological, weakened and remodelled by an underlying disorder, with altered or reduced mechanical and viscoelastic properties [42].

The list of medical conditions with an increased risk of fracturing due to weakened bone is very extensive. An overview of medical conditions (not all inclusive) with increased risk of fracturing is given in Table 2.7. Medical conditions that cause reduced bone strength can be congenital or acquired. These disorders are either generalized (diffuse, systemic) throughout the skeleton (e.g. osteogenesis imperfecta) or more localized (focal) (e.g. osteomyelitis or demineralization of one limb, resulting from prolonged immobilization). Disorders are either due to more or less benign disorders or to malignant disorders. Most 'pathological' fractures are due to benign tumours, but can also be due to tumour-like lesions, metabolic diseases (e.g. osteogenesis imperfecta, osteopetrosis), bone infections (e.g. osteomyelitis), and neuromuscular disorders. Pathological fractures due to malignant disorders (e.g. sarcomas, leukaemia, bone metastases) are much rarer [42–44]. According to Boyce and Gafni, many of these conditions are easily diagnosed or excluded with a thorough history, physical exam, and selected diagnostic tests [43].

The most important and probably most common medical condition with an increased risk of fracturing due to weakening of bone is osteogenesis imperfecta (OI). In 2013, Greeley et al. published the results of a retrospective study into the presence of fractures in 68 children with osteogenesis imperfecta [49]. They found that most fractures in children with OI occurred in the extremities: arms (17×) and legs (36×). Rib fractures were found in 15 children. In 13 out of 15 children with fractured ribs, they were diagnosed prenatally (before birth) or immediately after birth as the result of OI. The rib fractures of the 2 remaining children were diagnosed at the age of 14 and 43 months. This involved children with OI type 1 (plus a positive family history and blue 'eye white'). The number of fractures present at the time of diagnosis varied from 1 to more than 37, with 7 children (10%) having more than 2 fractures. All children with more than 2 fractions were diagnosed before or immediately after birth. Seventeen children (25%) were diagnosed after the first week of life, but before the age of 12 months. None of these children had more than 1 fracture at the time of diagnosis. Almost 75% of the children were diagnosed on the basis of the findings from the clinical examination. According to Greeley et al., finding multiple rib fractures is an unlikely finding in children under the age of 1 year with OI [49].

2.5.3.5 The Use of Terms like Spontaneous and Pathological Fractures in Forensic Paediatrics

From a clinical point of view, the use of terms like 'spontaneous' and 'pathological' in relation to the occurrence of fractures is understandable and even acceptable. However, from a biomechanical point of view the use of these terms as an explanation for the occurrence of a fracture or multiple fractures in a child with a bone disorder is an approach that is too limited, and as such incorrect. Fracturing of a bone is determined by the load exerted on the bone as well as the load-bearing capacity of the bone. 'Spontaneous' and 'pathological' only pertain to the capacity of the bone to absorb stress. Based on the use of these terms, one only and implicitly concludes that it would be possible for weakened bone to sustain a fracture not only with a minimal trauma or during normal care but even without a trauma.

A bone disease may decrease the maximum loading capacity/resistance of the bone against fracturing, resulting

in a lower resistance against loading and therefore in an increased risk of fracturing. A fracture in weakened bone is, just like in normal bone, caused by the exceeding of loading over the maximum load-bearing capacity of a bone (the capacity to absorb stress), in other words, it is caused by a trauma. As a result of the loading, the weakened bone breaks.

From a forensic point of view, the use of 'spontaneous' or 'pathological' may also lead to false certainties, related to the manner, when one has to differentiate between accidental and non-accidental circumstances based on these terms. By using these terms the manner of the fracture is not taken into consideration at all. When a fracture is found in a child, the presence of a disorder that results in a decreased capacity to absorb stress says nothing about the circumstances in which the stress was exerted. The medical history and the clinical/radiological symptoms may indicate whether the fracture was sustained in accidental and non-accidental circumstances.

Fractures in normal but insufficient bone and in weakened bone occur when the strength of a bone is reduced to a level that stresses that normally would not fracture a healthy bone will break the weakened bone (reduced maximum load-bearing capacity) [35, 50]. If fractures are found in a young and non-mobile child without any plausible explanation, a fracture in normal but insufficient bone or in weakened bone should be considered (Table 2.7). The determination of the possible presence of an underlying condition and the circumstances under which the fracture occurred are based on the medical history of the child (and the family), clinical examination (including laboratory examination), and radiological assessment [42].

Table 2.7 Medical conditions with an increased risk of fracturing (not all inclusive) [42–48]

Benign congenital disorders	
	Osteogenesis imperfecta and variants like Bruck syndrome
	Copper deficiency in infants
	Ehlers–Danlos syndrome
	Menkes syndrome
	Metabolic bone disease of prematurity
	Neuromuscular diseases, e.g. Duchenne muscular dystrophy
	Vitamin D-resistant rickets (or hypophosphataemic rickets)
	X-linked hypophosphatemia
	Liver defects, e.g. Alagille syndrome
	Malabsorption
	Familial osteoporosis
	Osteopetrosis
	Cole carpenter syndrome
	Congenital CMV infection
	Insensitivity to pain, e.g. in spina bifida and in congenital pain insensitivity
	Neurofibromatosis type 1
	Osteopetrosis
	Pycnodysostosis
	Idiopathic juvenile osteoporosis
Benign acquired disorders	
	Infections, e.g. osteomyelitis
	Vitamin D deficiency based on nutritional defects: Rickets
	Intoxications, e.g. with lead
	Prolonged reduced mobility, e.g. in cerebral palsy or posttraumatic
	Paediatric inflammatory bowel disease, e.g. Crohn's disease (due to, among others, direct effects of inflammation, prolonged use of glucocorticoids, and poor nutrition)
	Benign bone tumours, e.g. non-ossifying fibroma, osteochondroma, chondroblastoma, enchondroma, giant cell tumour, and osteoid osteoma
	Renal osteodystrophy, due to chronic renal diseases and concurrent vitamin D deficiency/rickets and secondary hyperparathyroidism
	Diabetes mellitus
	Mastocytosis
	Bone cysts, e.g. unicameral bone cyst and aneurysmal bone cyst
Iatrogenic conditions	
	Radiation therapy
Use of	Diuretics
	Glucocorticoids
	Anticonvulsants
	Antiretrovirals
	Methotrexate
	Bisphosphonates
	Prostaglandins
Malignant disorders	
	Leukaemia
	Isolated metastases/metastatic tumours (e.g. Wilms tumour, neuroblastoma)
	Ewing sarcoma
	Osteosarcoma
	(congenital) fibrosarcoma
	Eosinophilic granuloma
	Langerhans histiocytosis

2.6 Manner of Fractures (Circumstances)

As stated in the foregoing sections, a fracture is always caused by a trauma, whether the bone is normal, weakened, or overused. The circumstances under which a trauma occurs (manner) can be accidental or non-accidental. Trauma can occur intrauterine, during birth or after birth.

2.6.1 Intrauterine Fractures

As far as known from the medical literature intrauterine acquired fractures (fracturing of a foetal bone in utero) are only rarely reported [51–54]. The first descriptions are found in the medical literature in the early and mid-nineteenth century [55–57]. Some of the first descriptions, in the early 1900s, in radiology were by Smith and Snure [58, 59].

In utero fractures occur due to trauma in normal or in weakened bone, just like fractures that are sustained during and after birth. According to Morgan and Marcus, ultrasound is the best imaging modality for identifying foetal fractures of any aetiology, while biochemical and genetic tests can aid in the prenatal diagnosis of congenital disorders like osteogenesis imperfecta [53]. According to Dawson, the presence of signs of healing on imaging of normal bone within the first days after birth would rule out an obstetrical fracture [60].

2.6.1.1 Intrauterine Fractures in Normal Bone

Skull fractures, although rarely reported, are the most commonly described intrauterine fractures in medical literature [52–54]. These fractures can be true fractures with clearly recognizable fracture lines, but mostly reported are depressed skull fractures, so-called 'ping-pong' fractures, which are characterized by inward buckling of the calvarian bones (Sects. 5.3.3.1, 5.3.3.2 and 5.3.4.5) [61].

Isolated intrauterine femur fractures have also been described. Several authors described the fracture of the femur, after ruling out other possibilities, as occurring spontaneous/without any known trauma, others as being caused by maternal blunt force trauma of the pelvis, e.g. due to falls, motor vehicle accidents, or domestic violence [62–67].

Finally fractures of the tibia and fibula, spine and clavicula have been described (mostly as single case reports) as intrauterine acquired fractures, which were not disease related [60, 68–73].

2.6.1.2 Intrauterine Fractures in Weakened Bone

Intrauterine fractures in weakened bone have been described due to several medical conditions. Osteogenesis imperfecta is the most prevalent disorder, but other genetic/metabolic disorders can be found [53]. Dawson also mentioned chondrodystrophies and congenital syphilis [60]. Some of these disorders result in severe handicaps after birth or are not compatible with life intrauterine or after birth [74–78].

2.6.2 Fractures During Birth: Birth Trauma

2.6.2.1 General Aspects of Birth Trauma-Related Fractures

In older children, pain is often a more or less reliable indicator for the presence of a fracture. However, in neonates it is difficult to establish pain and therefore a fracture can only be diagnosed by carefully observing behaviour, muscle tone, heartbeat and symptoms such as nausea and vomiting or limited use of a body part [79].

Fractures resulting from birth are not always diagnosed immediately post-partum, unless there are obvious symptoms, such as a clearly visible swelling and/or abnormal position. It is quite likely that physicians will overlook some fractures due to the lack of obvious symptoms. Research by Morris et al. showed that there was a delay in diagnosis in the majority of children that had sustained a birth trauma-related femur fracture (Sects. 12.7.2.3, 12.7.3.3 and 12.7.4.3) [80]. Skull fractures are found in 5% of the children born by vacuum extraction, but are frequently overlooked unless a routine radiograph is made [81]. Clavicula fractures too are often diagnosed as late as several weeks after birth, due to the then present callus formation [82].

This delay in diagnosis can lead to wrongfully suspected non-accidental circumstances (child abuse). To a certain extent it is possible to differentiate between birth trauma-related fractures and fractures that are sustained after birth by carefully evaluating the presence of callus formation:

- Kogutt et al. stated that under normal circumstances, a fracture that is diagnosed 10 to 15 days after birth cannot be considered resulting birth, if there is no evidence of healing (subperiosteal new bone formation or callus formation) [83].
- Cumming reported that callus in the healing of birth-related fractures may be visible as early as 7 days after birth [84].
- Walters et al. evaluated 131 radiographs of presumed birth trauma-related clavicular fractures and rarely did find subperiosteal new bone formation before day 7 after birth, but it was most often present at day 10 [85]. Callus formation was rarely seen before day 9, but it was most often present at day 15.
- Fadell et al. reviewed retrospectively a total of 108 digital images of 61 infants with clavicular fractures [86]. Their findings are summarized in Table 2.8.

2.6.2.2 Incidence and Prevalence of Fractures Resulting from Birth

Jaarsma considered the incidence of birth trauma-related fractures to be 0.1–3.5% [79]. In the medical literature, a great number of studies can be found on fractures resulting from delivery (Table 2.9) [87–89]. Based on these publications it has been established that clavicular fractures are the most common birth-related fractures, followed by fractures of the humerus, skull, and femur. Rib fractures are only reported in exceptional situations (see Sect. 7.3.2) [91].

Table 2.8 Indicative timescale of healing in birth trauma-related clavicular fractures [86]

Feature of healing	First seen at day	Peak period (days)
Periosteal reaction	7	11–42
Callus	11	12–61
Bridging	20	22–63
Remodelling	35	49–59

Table 2.9 Fractures resulting from delivery (not all inclusive)

Author(s)	neonates (N)	Number of fractures (%)	Location	*N*	%
Rubin [87]	15,435	51 (0.35)	Clavicle Humerus Skull	43 7 1	84.3 13.7 1.6
Camus et al. [88]	20,409	123 (0.6)	Clavicle Humerus Skull Femoral shaft Epiphysis	105 7 7 2 2	85.4 5.7 5.7 1.6 1.6
Bhat et al. [89]	34,946	35 (0.1%)	Clavicle Humerus Femur Skull Orbit Epiphysis distal femur Dislocation elbow	16 7 5 4 1 1 1	45.7 20.0 14.3 11.3 2.9 2.9 2.9
Groenendaal and Hukkelhoven [90]	158,035	1174 (0.7)	Clavicle Humerus Femur No other fractures mentioned	Number of fractures not reported	

Bhat et al. found a higher incidence of fractures in cases without prenatal care, after a complicated delivery or after a Caesarean section [89].

In 2007, Groenendaal and Hukkelhoven drew attention in the Netherlands Journal of Medicine to the prevalence of fractures in term neonates [90]. They used data from Perinatal Registration Netherlands which contains data on term neonates <28 days old (n = 158.035). In 1174 children (0.74%) fractures were found. In 19% (n = 227) of cases, the cause of the fracture was not known: the vaginal birth had been either physiological and reported to be non-traumatic or there had been an uncomplicated Caesarean section; after the delivery there had been no cause for resuscitation and further diagnostics showed no indications for congenital bone diseases such as osteogenesis imperfecta or osteopenia. Twelve of the 227 children had sustained a humerus fracture without known cause, and 3 a femur fracture. The remaining 212 children had sustained a fracture of the clavicle. Groenendaal and Hukkelhoven suspected that the number of fractures in term neonates in The Netherlands would be higher than the 0.74% they found [90].

Many of the fracture-types that are found in non-accidental trauma have also been reported as occurring as birth trauma-related, usually in case reports. Hence, it is essential that in the immediate period after birth, a thorough obstetric history is taken. This history should also include the nurses notes, as often they will be the first to note that a child doesn't move a limb properly or shows pain during daily handling.

2.6.3 Fractures After Birth: Accidental Circumstances

While growing up, a child becomes more mobile and starts to discover the world around him. Due to this the risk of accidental injury increases [1].

Between the ages of 1 and 4 years and in older children (>10 years), a fracture is most commonly due to accidental circumstances [15]. In the group of children between the ages of 1 and 4 years, fractures of the upper extremities and the clavicle are most common, due to the reflex of the child to catch itself on the stretched arm when falling. In children over 10 years of age, the number of traffic accidents will be higher than in younger children [92] (Sect. 2.1.2).

2.6.4 Fractures After Birth: Non-Accidental Circumstances

2.6.4.1 Epidemiology

In children under 1 year of age, one will find fractures due to accidental circumstances only in a small minority of cases [92]. After bruises and burns, fractures are the most prevalent inflicted injuries [93, 94]. Fractures have been described in 55% of children who were victims of physical violence [83, 95].

The finding of inflicted fractures in children indicates the use of severe violence, which emphasizes the importance of a correct diagnosis. Sinal and Stewart found that fractures were the first sign of non-accidental trauma in 17% of the children with inflicted injuries [96].

Approximately 10% of children under the age of 5 years who because of an injury are seen by a physician in emergency departments in the United States have inflicted injuries, including fractures [97]. In children evaluated in emergency departments because of a suspicion of inflicted injuries, over 30% appears to have fresh or healing fractures [98].

In a study, in air force personnel in the United States, on child homicide between the ages of 1–15 years (average 3.9 years) it was found that 55% of these children had been seen by a physician because of physical injuries, including fractures, in the month prior to their death [99].

Rang poses that as many as 25% of all fractures in children of less than 3 years of age are inflicted and/or due to neglect [15]. According to Akbarnia et al., inflicted fractures occur predominantly in children of less than 1 year of age [100]. Based on various studies, it is estimated that 50 to 70% of all fractures in children of less than 1 year old are inflicted and/or due to neglect [101, 102]. It was also shown that children in this age group are at a high risk of sustaining inflicted injuries, including fractures, again, even after an intervention took place [103].

Unfortunately in these young, often non-mobile, children fractures are often not recognized because of several reasons. Firstly fractures in this young children can have an occult course, because fractures in these children often show no or hardly any clinically conspicuous symptoms such as swelling, redness, tenderness or pseudo-paresis [13, 14, 104]. Secondly in these children (inflicted) fractures remain not only unnoticed due to its occult course, but also because non-accidental circumstances are not or inadequately considered, or even rejected on non-plausible grounds:

- Between 1995 and 1999, Banaszkiewicz et al. carried out a retrospective study in all children under the age of 1 year which were brought into the emergency department of their hospital because of fractures [105]. The data of 74 children in total were re-evaluated. The average age of the children was 5 months (2 weeks to 1 year). Forty-six children had sustained a skull fracture. In 28 children, there was a fracture of the long bones. After analysis, it appeared that the attending physician failed to assess possible non-accidental circumstances correctly in nearly 30% of these children. In nearly 50% of children, the medical data did not show that non-accidental circumstances had even been considered, whereas in retrospect non-accidental circumstances would have been a plausible explanation in the differential diagnosis.
- Oral et al. carried out a similar retrospective study in 653 children of 3 years and younger who presented with a fracture over the period 1995–1999 [106]. The aim of their study was to establish whether in this group of children physicians inquired sufficiently into the circumstances, under which the fractures occurred. Revision showed that, based on the data in the dossier, in 42% of children it had not been possible to exclude that the fractures were inflicted (non-accidental injury/child abuse). The missing data concerned:
  - Information on the presence of (independent) eye-witnesses at the moment the fracture was sustained.
  - Information on previous injuries.
  - Revision of previous medical data.
  - Description of associated injuries.
  - An evaluation to see whether the reason provided and the injury of the child could be explained when taking into account the level of development of the child.
- Consequently, Oral distinguished four groups: accidental injury (63%), non-accidental injury (‘inflicted injury’) (13%), missed non-accidental injury (23%), and missed accidental injury (0.6%) [106]. Factors that had a positive influence on identifying non-accidental circumstances were the age of the child, the presence of multiple fractures, and an examination by a paediatrician.
- Carty and Pierce reported on a cohort of 467 children who were either presented at the or referred to the Alder Hey Hospital, Liverpool UK, with a suspected diagnosis of child abuse during a 13-year period (1984–1996) [107]. In their study group in 435 (93%) child abuse could be confirmed, in 51 (11.7%) of them chart reviews showed enough evidence that should have led to a correct diagnosis at the first presentation. In this group 6 (12%) children died and 10 (20%) survived with handicap, which was severe in 4 cases.

2.6.4.2 Specificity of Fractures, Sustained in Non-Accidental Circumstances

Although it is crucial for a responsible intervention, it is not always easy to differentiate between accidental and non-accidental circumstances, even if non-accidental circumstances are suspected [108]. According to Hobbs et al., non-accidental circumstances should be considered in case of [109, 110]:

- Multiple fractures in various stages of healing, even when no associated trauma is present, such as haematomas and (sub)cutaneous injuries.
- Damage to the epiphysis and metaphysis, possibly multiple as in the inflicted traumatic brain injury.
- Fractures of ribs (single or multiple), scapulae, and sternum.
- The presence of periosteal new bone formation.
- A skull fracture, with or without signs of intracranial trauma.
- Multiple and complicated skull fractures with a fracture width >3 mm.

Hobbs further stated that these fractures are more suspect than simple, uncomplicated fractures, shaft fractures of the long bones, and fractures of the clavicle and that fractures are more suspect when they occur simultaneously with other injuries, e.g. a simple fracture (e.g. of the humerus) combined with multiple unexplained haematomas [110].

In 1998, Kleinman presented an overview (Table 2.10) on the specificity of radiological findings regarding non-accidental trauma/inflicted fractures (child abuse) [111]. According to him, the highest specificity applied in infants. He also stated that non-accidental circumstances are likely when there is no explanation for the occurrence of fractures of average or low specificity or when the explanation does not correspond with the nature of the skeletal findings.

In a systematic review of the literature by Kemp et al., the predictive value of fractures as a sign of child abuse had been evaluated [112]. Other indications such as the child’s age or

Table 2.10 Specificity of skeletal injuries in child abuse

Specificity	Type of fracture/skeletal lesion
High specificity	Classic metaphyseal lesions (Metaphyseal corner fractures)
	Rib fractures, especially posterior
	Scapular fractures
	Spinous processes fractures
	Sternal fractures
Moderate specificity	Multiple fractures, specifically bilateral
	Fractures of different ages
	Epiphyseal separations
	Vertebral body fractures and subluxations
	Digital fractures
	Complex skull fractures
Common but low specificity	Subperiosteal new bone formation
	Clavicular fractures
	Long bone shaft fractures
	Linear skull fractures

Reprinted from Kleinman 1998 [111]. With permission

the injury that could lead to suspected child abuse were not taken into account. After a selection was made from 439 publications, 32 were analysed. Based on this systematic analysis, they concluded among others that rib fractures had the strongest correlation with non-accidental circumstances (child maltreatment). In 71% of cases (95% CI 42–91%) with rib fractures, the rib fractures were inflicted. They also found that none of the fractures was pathognomonic for child abuse. As such, the inflicted skeletal lesions may be similar to lesions found after an accident.

The determination whether a fracture was inflicted in a child not only depends on the characteristics of the fracture, as described in Sect. 2.4, or on the theoretical specificity of the fracture, as described in the foregoing text, but also on:

- The age and level of development of the child (Tables 2.11 and 2.12 and Chap. 13).
- The statements of the child, the parents, or the caregivers regarding the causing fracture.
- The theoretical reconstruction of the causing trauma, based on the known biomechanics.

When these are not compatible with the given statements of the child and/or the parents and/or caregivers about the causing trauma, non-accidental circumstances should be considered.

Performing the correct radiological examination and radiological dating of fractures is eminently important for an adequate diagnosis and protection at the moment that child abuse is suspected. Fractures as a result of violence can be found throughout the entire skeleton, are often present in multiple places, and may show various stages of healing on skeletal radiographs [92, 102, 121]. Since in cases of child abuse there is often a delay in seeking medical help, dating may be complicated by further loading of the fracture by movement, additional injuries, and renewed fractures. The more or less objective radiological dating (see Chap. 4) can spot inconsistencies regarding subjective dating, based on the medical history, and the explanation of the injury.

Table 2.11 Overview of the general motor development at key ages [113–119]

Age	Skill	General motor development
4 weeks	Control muscles of the eye	Positive head lag
16 weeks	Head balance	Stabile head balance Symmetric posture
28 weeks	Grip and manual manipulation	Sits and leans forward supported on the hands Stable stance when supported Asymmetric neck reflex disappears (22–26 weeks)
40 weeks	Control trunk and fingers: sitting, crawling, and picking	Sits without support Crawls Pulls up to stance Grip reflex at the feet disappears (40 weeks–18 months)
52 weeks	Control of legs and feet: the child stands erect and starts exploring	Walks holding on to one hand Walks along an object (such as coffee table or settee)
18 months	Control of larynx function: words and word combinations	Walks independently Able to sit up independently
24 months	Control of bladder and bowel functions	Is capable of running Can play football
36 months	Speaks in sentences	Can stand on one leg Jumps from the bottom step of the stairs
48 months	Understands numbers and shapes	Hops well on one leg Jumps forward on both legs
60 months	Child ready for school and prepared to play with other children	Hops equally well on either leg

Table 2.12 Windows of achievement for six gross motor milestones according to the World Health Organization [120]

	Box boundary (age in months)			
Motor milestone	Lower	95% CI	Upper	95% CI
Sitting without support	3.8	3.7–3.9	9.2	8.9–9.4
Sitting with assistance	4.8	4.7–5.0	11.4	11.2–11.7
Hand and knees crawling	5.2	5.0–5.3	13.5	13.1–13.9
Walking with assistance	6.0	5.8–6.1	13.7	13.4–14.1
Standing alone	6.9	6.8–7.1	16.9	16.4–17.4
Walking alone	8.2	8.0–8.4	17.6	17.1–18.0

2.6.4.3 The Value of Haematomas in the Differential Diagnosis Between Accidental and Non-accidental Circumstances

The little that is known about the presence of haematomas in relation to fractures in children has been learned through fractures that were sustained in non-accidental circumstances (inflicted fractures). In court procedures, it is sometimes claimed that haematomas are sustained at the same time as fractures ('the force required to cause a fracture will in all likelihood also result in haematomas') and that the absence of haematomas is proof that it took only very little force to break the bone and, as such, that the fracture must be due to disorders with an increased risk for fracturing, e.g. a metabolic disorder or from osteogenesis imperfecta. This opinion is based on one publication, in which this hypothesis was described, and is repeated regularly in court procedures, still without any evidence (see also Chaps. 15 and 16) [122, 123].

There is even ample evidence of the contrary. Mathew et al. conducted a prospective study into the presence of haematomas around the location of the fracture in 88 children that showed no signs of bone pathology with a total of 93 fractures (49 boys, 39 girls; age 12 months to 13 years and 11 months) [124]. All children were seen within 24 h after the fracture had been sustained. Only in eight fractures haematomas were found in the initial phase. No haematomas were found in fractures that showed no dislocation or in fractures that were well covered by soft tissue. In 13 other fractures, haematomas appeared within 24 h after hospitalization. Ultimately, 25 (28%) fractures were accompanied by haematomas 1 week after the fracture was sustained. According to Mathew et al., it is impossible to distinguish between fractures that are the result of bone disease and fractures resulting from child abuse based on the presence or absence of haematomas. It appears that in acutely sustained fractures in children, local haematomas are less common than one would expect; therefore, based on the absence of haematomas, non-accidental circumstances should never be excluded. Starling et al. also did not find any relation between fractures and the presence of haematomas [125]. After skull fractures had been excluded, it appeared that less than 10% of children had fracture-related haematomas. Peters et al. found that fractured bones that were most frequently associated with bruising were skull bones. The presence of bruising near the fracture site was uncommon in fractures of the extremities or the rib [126]. Valvano et al. found that the presence or absence of bruising was not useful in differentiating between inflicted and accidental fractures [127].

2.6.4.4 Perpetrators and Victims

Starling et al. were the first to initiate a study into the specific characteristics of perpetrators who cause fractures in children [125]. They evaluated the data of 194 children (age: 0–13.9 years; median 6 months) with in total 630 fractures. The median number of fractures per patient was 2 and the maximum was 31. In 153 children (79%), the perpetrator could be identified. Nearly 68% of perpetrators were male. Of all known perpetrators, 45% appeared to be the biological father.

Furthermore, there appeared to be a significant difference ($p = 0.003$) between the median age of the children who had been abused by a male (4.5 months) and by a female perpetrator (10 months). In 44 of the 194 children, the primary injury was non-accidental skull−/brain trauma. Since it is not known whether the age of victims of non-accidental skull−/brain trauma differs from that of children with other non-accidental fractures, further study was done after the children with non-accidental skull/brain trauma were excluded. However, this analysis still showed a significant difference ($p = 0.004$) between the median age of children abused by a male (5 months) or a female perpetrator (12 months).

2.6.5 Fractures After Birth: Other Circumstances

Fractures that cannot be classified as classical accidental or non-accidental fractures are fractures that are due to, e.g. medical procedures (Sect. 13.4.2), habit disorders (Sect. 13.3.1), or sports activities (e.g. Sect. 13.5.2).

References

1. Lyons RA, Delahunty AM, Kraus D, Heaven M, McCabe M, Allen H, Nash P (1999) Children's fractures: a population based study. Inj Prev 5:129–132
2. Landin LA (1983) Fracture patterns in children. Analysis of 8,682 fractures with special reference to incidence, etiology and secular changes in a Swedish urban population 1950–1979. Acta Orthop Scand Suppl 202:1–109
3. Landin LA (1997) Epidemiology of children's fractures. J Pediatr Orthop B 6:79–83
4. Worlock P, Stower M (1986) Fracture patterns in Nottingham children. J Pediatr Orthop 6:656–660
5. Rennie L, Court-Brown CM, Mok JY, Beattie TF (2007) The epidemiology of fractures in children. Injury 38:913–922
6. Hedström EM, Svensson O, Bergström U, Michno P (2010) Epidemiology of fractures in children and adolescents. Acta Orthop 81:148–153
7. Mäyränpää MK, Mäkitie O, Kallio PE (2010) Decreasing incidence and changing pattern of childhood fractures: a population-based study. J Bone Miner Res 25:2752–2759
8. Naranje SM, Erali RA, Warner WC Jr, Sawyer JR, Kelly DM (2016) Epidemiology of pediatric fractures presenting to emergency departments in the United States. J Pediatr Orthop 36:e45–e48
9. Mathison DJ, Agrawal D (2010) An update on the epidemiology of pediatric fractures. Pediatr Emerg Care 26:594–603
10. Ran Shin M, Fleming M (2019) Pediatric fractures in developing bone. https://now.aapmr.org/pediatric-fractures-in-developing-bone/. Accessed 15 July 2022
11. Wilkins KE, Aroojis AJ (2001) The present status of children's fractures. In: Beaty JH, Kasser JM (eds) Rockwood and Wilkins'

fractures in children. Lippincott Williams & Wilkins, Philadelphia, pp 3–20
12. Akbarnia BA, Campbell RM (1990) The role of the orthopedic surgeon in child abuse. In: Morrissy RT, Winter RB (eds) Lovell and winter's pediatric orthopaedics. Lippincott Williams & Wilkins, Philadelphia
13. Merten DF, Radlowski MA, Leonidas JC (1983) The abused child: a radiological reappraisal. Radiology 146:377–381
14. 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
15. Rang MC, Willis RB (1977) Fractures and sprains. Pediatr Clin N Am 24:749–773
16. Pierce MC, Bertocci GE (2006) Fractures resulting from inflicted trauma: assessing injury and history compatibility. Clin Ped Emerg Med 7:143–148
17. van Rijn RR, Nijs HGT, Bilo RAC (2009) Evidence based imaging in non-CNS non-accidental injury. In: Medina LS, Applegate KE, Blackmore CC (eds) Evidence-based imaging in pediatrics. Springer, Cham, pp 177–191
18. Keats TE, Anderson MW (2012) Atlas of normal roentgen variants which may simulate disease. Saunders, Philadelphia
19. Bolander S (2019) A systematic approach to describing fractures. JAAPA 32:23–29
20. Bell DJ, Gaillard F (2019) Fracture. https://radiopaedia.org/articles/fracture-1?lang=us. Accessed 18 Nov 2021
21. Meinberg EG, Agel J, Roberts CS, Karam MD, Kellam JF (2018) Fracture and dislocation classification compendium-2018. J Orthop Trauma 32(Suppl. 1):S1–s170
22. Lloyd-Jones G (2019) Introduction to trauma X-ray: fracture mimics. https://www.radiologymasterclass.co.uk/tutorials/musculoskeletal/trauma/trauma_x-ray_start. Accessed 18 Nov 2021
23. Murphy A, Hacking C (2019) Describing a fracture (an approach). https://radiopaedia.org/articles/describing-a-fracture-an-approach?lang=us. Accessed 18 Nov 2021
24. Lewis WH (1918) Part II osteology. In: Lewis WH (ed) Gray's anatomy of the human body
25. Jones J (2020) Bones types. https://radiopaedia.org/articles/bones-types. Accessed 18 Nov 2021
26. Mulligan EP (2012) Lower leg, ankle, and foot rehabilitation. In: Andrews JR, Harrelson GL, Wilk KE (eds) Physical rehabilitation of the injured athlete. Elsevier, Philadelphia, pp 426–463
27. Pinto DC, Love JC, Derrick SM, Wiersema JM, Donaruma-Kwoh M, Greeley CS (2015) A proposed scheme for classifying pediatric rib head fractures using case examples. J Forensic Sci 60:112–117
28. Vaccaro AR, Lehman RA Jr, Hurlbert RJ, Anderson PA, Harris M, Hedlund R, Harrop J, Dvorak M, Wood K, Fehlings MG, Fisher C, Zeiller SC, Anderson DG, Bono CM, Stock GH, Brown AK, Kuklo T, Oner FC (2005) A new classification of thoracolumbar injuries: the importance of injury morphology, the integrity of the posterior ligamentous complex, and neurologic status. Spine 30:2325–2333
29. Health Canada (2003) Injury surveillance in Canada: current realities, challenges. http://www.injuryresearch.bc.ca/docs/3_20090910_100541Report%20HC%20Inj%20Surveillance%20in%20Can%20Aug%202003.pdf. Accessed 5 Aug 2021
30. WHO Regional Office for Europe (2013) In: Sethi D, Bellis M, Hughes K, Gilbert R, Mitis G, Galea G (eds) European report on preventing child maltreatment
31. National Institute for Health and Care Excellence (NICE) (2017) Osteoporosis: assessing the risk of fragility fracture. Clinical guideline [CG146]. https://www.nice.org.uk/guidance/cg146. Accessed 11 Aug 2021
32. Jaimes C, Jimenez M, Shabshin N, Laor T, Jaramillo D (2012) Taking the stress out of evaluating stress injuries in children. Radiographics 32:537–555
33. Sanderlin BW, Raspa RF (2003) Common stress fractures. Am Fam Physician 68:1527–1532
34. American Academy of Orthopaedic Surgeons (AAOS) (2007) Stress fractures. https://orthoinfo.aaos.org/en/diseases%2D%2Dconditions/stress-fractures/. Accessed 18 Nov 2021
35. DeWeber K (2017) Overview of stress fractures. https://www.uptodate.com/contents/overview-of-stress-fractures. Accessed 10 Aug 2021
36. Griffiths AL (1952) Fatigue fracture of the fibula in childhood. Arch Dis Child 27:552–557
37. Bedoya MA, Jaramillo D, Chauvin NA (2015) Overuse injuries in children. Top Magn Reson Imaging 24:67–81
38. Shelat NH, El-Khoury GY (2016) Pediatric stress fractures: a pictorial essay. Iowa Orthop J 36:138–146
39. Wu M, Fallon R, Heyworth BE (2016) Overuse injuries in the pediatric population. Sports Med Arthrosc Rev 24:150–158
40. Devas MB (1963) Stress fractures in children. J Bone Joint Surg Br 45:528–541
41. Torwalt CR, Balachandra AT, Youngson C, de Nanassy J (2002) Spontaneous fractures in the differential diagnosis of fractures in children. J Forensic Sci 47:1340–1344
42. Canavese F, Samba A, Rousset M (2016) Pathological fractures in children: diagnosis and treatment options. Orthop Traumatol Surg Res 102:S149–S159
43. Boyce AM, Gafni RI (2011) Approach to the child with fractures. J Clin Endocrinol Metab 96:1943–1952
44. De Mattos CB, Binitie O, Dormans JP (2012) Pathological fractures in children. Bone Joint Res 1:272–280
45. Altman DH, Smith RL (1960) Unrecognized trauma in infants and children. J Bone Joint Surg Am 42-a:407–413
46. Mendelson KL (2005) Critical review of 'temporary brittle bone disease'. Pediatr Radiol 35:1036–1040
47. Wick JY (2009) Spontaneous fracture: multiple causes. Consult Pharm 24:100–102, 105–108, 110–102
48. Wong SC, Catto-Smith AG, Zacharin M (2014) Pathological fractures in paediatric patients with inflammatory bowel disease. Eur J Pediatr 173:141–151
49. Greeley CS, Donaruma-Kwoh M, Vettimattam M, Lobo C, Williard C, Mazur L (2013) Fractures at diagnosis in infants and children with osteogenesis imperfecta. J Pediatr Orthop 33:32–36
50. MacAusland WR (1972) Sprains, fractures, dislocations. In: Nardi GL, Zuidema GD (eds) Surgery, a concise guide to clinical practice. Little Brown, Boston, p 945
51. Alexander E Jr, Davis CH Jr (1969) Intra-uterine fracture of the infant's skull. J Neurosurg 30:446–454
52. Garza-Mercado R (1982) Intrauterine depressed skull fractures of the newborn. Neurosurgery 10:694–697
53. Morgan JA, Marcus PS (2010) Prenatal diagnosis and management of intrauterine fracture. Obstet Gynecol Surv 65:249–259
54. Veeravagu A, Azad TD, Jiang B, Edwards MSB (2018) Spontaneous intrauterine depressed skull fractures: report of 2 cases requiring neurosurgical intervention and literature review. World Neurosurg 110:256–262
55. Barker TH (1857) On intrauterine fractures: with an illustrative case. Br Med J 2:806–809
56. Brodhurst BE (1860) Cases of intra-uterine fracture with observations to show the analogy between fracture in utero and congenital distorsion
57. Murray GCP (1857) Intrauterine fractures. BMJ s4-1:865

58. Smith RR (1913) Intrauterine fracture: report of a case and a review of the literature. Surg Gynecol Obstet 17:346–349
59. Snure H (1929) Intra-uterine fracture – case report and review of roentgenologic findings. Radiology 13:362–365
60. Dawson GR Jr (1949) Intra-uterine fractures of the tibia and fibula; report of a case with correction by osteotomy and plating. J Bone Joint Surg Am 31a:406–408
61. Dupuis O, Silveira R, Dupont C, Mottolese C, Kahn P, Dittmar A, Rudigoz RC (2005) Comparison of "instrument-associated" and "spontaneous" obstetric depressed skull fractures in a cohort of 68 neonates. Am J Obstet Gynecol 192:165–170
62. Alonso JA, Wright DM, Sochart DH (2005) Intrauterine femoral fracture diagnosed at birth – maternal abdominal trauma versus non-accidental injury. Inj Extra 36:432–433
63. Arioz DT, Koken GN, Koken R, Kose KC, Cevrioglu AS (2008) Isolated intrauterine femoral fracture in an otherwise normal fetus. J Obstet Gynaecol Res 34:92–94
64. Bailey BA (2010) Partner violence during pregnancy: prevalence, effects, screening, and management. Int J Women's Health 2:183–197
65. Cook J, Bewley S (2008) Acknowledging a persistent truth: domestic violence in pregnancy. J R Soc Med 101:358–363
66. Pearsall AWT, Larkin JJ, Raasch W (1992) Intrauterine femur fracture. Orthopedics 15:947–950
67. Yu M, Xu D, Zhang A, Shen J (2018) Spontaneous fetal femoral fracture: a case report and literature review. J Int Med Res 46:1282–1287
68. Freedman M, Gamble J, Lewis C (1982) Intrauterine fracture simulating a unilateral clavicular pseudarthrosis. J Can Assoc Radiol 33:37–38
69. Heath PM (1912) Intra-uterine fracture of tibia and fibula, with absorption of bone. Proc R Soc Med 5:10–12
70. Hawthorne ES (1903) Fractured clavicle with ossific union in utero. Lancet 162:315
71. Nicole R (1954) Traumatische intrauterine Unterschenkelfraktur; Beitrag zur Frage der sog. Congenitalen Unterschenkel-Pseudarthrose [Traumatic intrauterine fracture of the lower leg; a contribution to the problem of so-called pseudarthrosis of lower-leg]. Bibl Paediatr 58:743–755
72. Scheier M, Peter M, Hager C, Lang T, Barvinek A, Marth C (2010) Spontaneous isolated midtrimester fracture of tibia and fibula in a normal fetus with in utero healing and good long-term outcome. Fetal Diagn Ther 28:58–60
73. Weinberg L, Wyatt JP, Busuttil A (2001) Traumatic intrauterine fetal spinal fracture following seat belt use: a case report. J Trauma 51:1195–1196
74. Dennis NR, Fairhurst J, Moore IE (1995) Lethal syndrome of slender bones, intrauterine fractures, characteristics facial appearance, and cataracts, resembling Hallermann-Streiff syndrome in two sibs. Am J Med Genet 59:517–520
75. Jain A, Kumar Jha B, Chopra A (2019) Case report: type 0 spinal muscular atrophy associated with fractures at birth. J Clin Neonatol 8:125–127
76. Lacson AG, Donaldson G, Barness EG, Ranells JD, Pomerance HH (2002) Infant with high arched palate, bell-shaped chest, joint contractures, and intrauterine fractures. Pediatr Pathol Mol Med 21:569–584
77. Marchesoni M, Helfer A (1955) An unusual case of multiple intrauterine Su di un raro Caso di fratture fetali multiple endouterine per osteogenesi imperfetta tipo Vrölik [fetal fractures caused by Vrölik's type of osteogenesis imperfecta]. Ann Ostet Ginecol 77:585–594
78. Rossbach HC, Dalence C, Wynn T, Tebbi C (2006) Faisalabad histiocytosis mimics Rosai-Dorfman disease: brothers with lymphadenopathy, intrauterine fractures, short stature, and sensorineural deafness. Pediatr Blood Cancer 47:629–632
79. Jaarsma AS (2007) Botbreuken bij pasgeborenen [Fractures in neonates]. Patient Care 34:9–12
80. Morris S, Cassidy N, Stephens M, McCormack D, McManus F (2002) Birth-associated femoral fractures: incidence and outcome. J Pediatr Orthop 22:27–30
81. Simonson C, Barlow P, Dehennin N, Sphel M, Toppet V, Murillo D, Rozenberg S (2007) Neonatal complications of vacuum-assisted delivery. Obstet Gynecol 109:626–633
82. Joseph PR, Rosenfeld W (1990) Clavicular fractures in neonates. Am J Dis Child 144:165–167
83. Kogutt MS, Swischuk LE, Fagan CJ (1974) Patterns of injury and significance of uncommon fractures in the battered child syndrome. Am J Roentgenol Radium Therapy Nucl Med 121:143–149
84. Cumming WA (1979) Neonatal skeletal fractures. Birth trauma or child abuse? J Can Assoc Radiol 30:30–33
85. Walters MM, Forbes PW, Buonomo C, Kleinman PK (2014) Healing patterns of clavicular birth injuries as a guide to fracture dating in cases of possible infant abuse. Pediatr Radiol 44:1224–1229
86. Fadell M, Miller A, Trefan L, Weinman J, Stewart J, Hayes K, Maguire S (2017) Radiological features of healing in newborn clavicular fractures. Eur Radiol 27:2180–2187
87. Rubin A (1964) Birth injuries: incidence, mechanisms and end results. Obstet Gynecol 23:218–221
88. Camus M, Lefebvre G, Veron P, Darbois Y (1985) Traumatismes obstétricaux du nouveau-né. Enquête rétrospective à propos de 20409 naissances [Obstetrical injuries of the newborn infant. Retrospective study apropos of 20,409 births]. J Gynecol Obstet Biol Reprod (Paris) 14:1033–1043
89. Bhat BV, Kumar A, Oumachigui A (1994) Bone injuries during delivery. Indian J Pediatr 61:401–405
90. Groenendaal F, Hukkelhoven C (2007) Botbreuken bij voldragen pasgeborenen [Fractures in full-term neonates]. Ned Tijdschr Geneeskd 151:424
91. van Rijn RR, Bilo RA, Robben SG (2009) Birth-related mid-posterior rib fractures in neonates: a report of three cases (and a possible fourth case) and a review of the literature. Pediatr Radiol 39:30–34
92. Worlock P, Stower M, Barbor P (1986) Patterns of fractures in accidental and non-accidental injury in children: a comparative study. BMJ 293:100–102
93. Cramer K (1996) Orthopedic aspects of child abuse. Pediatr Clin N Am 43:1035–1051
94. McMahon P, Grossman W, Gaffney M, Stanitski C (1995) Soft-tissue injury as an indication of child abuse. J Bone Joint Surg Am 77:1179–1183
95. Loder RT, Bookout C (1991) Fracture patterns in battered children. J Orthop Trauma 5:428–433
96. Sinal SH, Stewart CD (1998) Physical abuse of children: a review for orthopedic surgeons. J South Orthop Assoc 7:264–276
97. Holter JC, Friedman SB (1968) Child abuse: early case finding in the emergency department. Pediatrics 42:128–138
98. Hyden PW, Gallagher TA (1992) Child abuse intervention in the emergency room. Pediatr Clin N Am 39:1053–1081
99. Lucas DR, Wezner KC, Milner JS, McCanne TR, Harris IN, Monroe-Posey C, Nelson JP (2002) Victim, perpetrator, family, and incident characteristics of infant and child homicide in the United States Air Force. Child Abuse Negl 26:167–186

100. Akbarnia B, Torg JS, Kirkpatrick J, Sussman S (1974) Manifestations of the battered-child syndrome. J Bone Joint Surg Am 56:1159–1166
101. King J, Diefendorf D, Apthorp J, Negrete VF, Carlson M (1988) Analysis of 429 fractures in 189 battered children. J Pediatr Orthop 8:585–589
102. Leventhal JM, Thomas SA, Rosenfield NS, Markowitz RI (1993) Fractures in young children. Distinguishing child abuse from unintentional injuries. Am J Dis Child 147:87–92
103. Skellern CY, Wood DO, Murphy A, Crawford M (2000) Non-accidental fractures in infants: risk of further abuse. J Paediatr Child Health 36:590–592
104. 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 54:1107–1110
105. Banaszkiewicz PA, Scotland TR, Myerscough EJ (2002) Fractures in children younger than age 1 year: importance of collaboration with child protection services. J Pediatr Orthop 22:740–744
106. Oral R, Blum KL, Johnson C (2003) Fractures in young children: are physicians in the emergency department and orthopedic clinics adequately screening for possible abuse? Pediatr Emerg Care 19:148–153
107. Carty HM, Pierce A (2002) Non-accidental injury: a retrospective analysis of a large cohort. Eur Radiol 12:2919–2925
108. Taitz J, Moran K, O'Meara M (2004) Long bone fractures in children under 3 years of age: is abuse being missed in emergency department presentations? J Paediatr Child Health 40:170–174
109. Hobbs CJ, Hanks HGI, Wynne JM (1993) Child abuse and neglect – a clinician's handbook. Churchill Livingstone, London
110. Hobbs CJ (1989) ABC of child abuse fractures. BMJ 298:1015–1018
111. Kleinman PK (1998) Skelet trauma: general considerations. In: Kleinman PK (ed) Diagnostic imaging of child abuse. Mosby, St. Louis, pp 8–25
112. Kemp AM, Dunstan F, 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
113. Gesell A, Amatruda CS (1964) Developmental diagnosis. Hoever Medical Division, Harper & Row Publishers, New York
114. Egan DF, Illingworth RS, MacKeith RC (1969) Developmental screening. 0–5 years. In: Gardiner P, MacKeith R, Smith V (eds) Aspects of developmental and paediatric ophthalmology. S.I.M.P. with Heinemann Medical, London
115. Flehmig I (2007) Normale Entwicklung des Säuglings und ihre Abweichungen: Früherkennung und Frühbehandlung. Thieme Verlag, Stuttgart
116. Illingworth RS (1988) Basic developmental screening: 0–4 years. Wiley-Blackwell, Oxford
117. Illingworth RS (1983) Development of the infant and young child: Normal and abnormal. Churchill Livingstone, Edinburgh
118. Knobloch H, Pasamanick B (1974) Gesell and Armatruda's developmental diagnosis: the evaluation and management of normal and abnormal neuropsychologic development in infancy and early childhood. Harper & Row
119. Bilo RAC, Voorhoeve HWA (2017) Kind in ontwikkeling – een handreiking bij de observatie van jonge kinderen. Elsevier Tijdstroom, Maarssen
120. WHO Multicentre Growth Reference Study Group (2006) WHO Motor Development Study: windows of achievement for six gross motor development milestones. Acta Paediatr Suppl 450:86–95
121. Duhaime AC, Alario AJ, Lewander WJ, Schut L, Sutton LN, Seidl TS, Nudelman S, Budenz D, Hertle R, Tsiaras W 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
122. Paterson CR (1987) Child abuse or copper deficiency? Br Med J (Clin Res Ed) 295:213–214
123. Taitz LS (1991) Child abuse and metabolic bone disease: are they often confused? BMJ 302:1244
124. Mathew MO, Ramamohan N, Benet GC (1998) Importance of bruising associated with paediatric fractures: prospective observational study. BMJ 317:1117–1118
125. Starling SP, Sirotnak AP, Heisler KW, Barnes-Eley ML (2007) Inflicted skeletal trauma: the relationship of perpetrators to their victims. Child Abuse Negl 31:993–999
126. Peters ML, Starling SP, Barnes-Eley ML, Heisler KW (2008) The presence of bruising associated with fractures. Arch Pediatr Adolesc Med 162:877–881
127. Valvano TJ, Binns HJ, Flaherty EG, Leonhardt DE (2009) Does bruising help determine which fractures are caused by abuse? Child Maltreat 14:376–381

Radiology in Suspected Child Abuse

3

Rick R. van Rijn, Rutger A. J. Nievelstein, and Simon G. F. Robben

Contents

3.1 Introduction

Even before Kempe published his now classic article on 'the battered child syndrome' in 1962, radiologists drew attention to fractures that could really only be explained by the impact of external mechanical force [1]. In 1946, Caffey was the first to describe the relation between the presence of multiple fractures of the long bones and subdural haematomas in six children in whom no previous trauma was known [2]. He thought it remarkable that in a number of children no new anomalies were found while hospitalized; however, some children showed new manifestations as soon as they returned home. Based on the fact that in children subdural haematomas are usually of traumatic origin, he suspected that this combination had a traumatic origin. In 1953, Silberman established that the combination of injuries as described by Caffey had to have a traumatic background [3]. In 1955, Woolley was the first to conclude that the found anomalies were the result of 'intentionally' inflicted physical injuries [4]. In 1957, 11 years after his original publication, Caffey concluded that physical abuse by either one or both parent(s) could be a possible explanation for this combination of injuries [5].

The importance of radiological examination when there are suspicions of inflicted injuries was not just demonstrated

R. R. van Rijn (✉)
Department of Radiology and Nuclear Medicine, Amsterdam UMC, University of Amsterdam, Amsterdam, The Netherlands

Department of Forensic Medicine, Netherlands Forensic Institute, The Hague, The Netherlands
e-mail: r.r.vanrijn@amsterdamumc.nl

R. A. J. Nievelstein
Department of Radiology, University Medical Center Utrecht, Utrecht, The Netherlands
e-mail: R.A.J.Nievelstein@umcutrecht.nl

S. G. F. Robben
Department of Radiology, Maastricht University Medical Center, Maastricht, The Netherlands
e-mail: s.robben@mumc.nl

R. A. C. Bilo et al. (eds.), *Forensic Aspects of Paediatric Fractures*, https://doi.org/10.1007/978-3-031-12041-1_3

by the earlier-mentioned radiologists. Ellerstein performed routine radiological examinations in children suspected of being physically abused [6]. In 11.5% he found radiological indications for inflicted injuries. Approximately 20% of these children had fractures without any clinical manifestations.

Generally, fractures are the result of the more serious forms of physical abuse. Non-accidental fractures (inflicted fractures, fractures seen in child abuse) are similar to fractures sustained in an accident. Whether a fracture can be the result of non-accidental circumstances is determined by a combination of:

- The type of fracture.
- The age and level of development of the child.
- The manner in which the fracture must have been sustained (according to established biomechanical data).
- The statement of the child, the parents, or the caregivers, regarding the origin of the fracture.

Non-accidental circumstances are likely when the first three factors are contradicted by the fourth. The role of (paediatric) radiologists is of great importance and often conclusive in determining whether non-accidental circumstances are involved. In children below a certain age (see Sect. 3.2) who are suspected of being physically abused, it is indicated to do a skeletal survey. The purpose of the skeletal survey is:

- To detect occult fractures.
- To obtain additional information on clinically suspect abnormalities.
- To date fractures.
- To diagnose the underlying skeletal abnormalities that may provide an increased risk for fractures.

In the following paragraphs, the existing guidelines and quality criteria that apply to radiological imaging will be discussed.

3.2 Conventional Radiology

3.2.1 Guidelines

There currently are two major guidelines on imaging in case of suspected physical child abuse. The first is from the American College of Radiology (ACR) and the second is from the Royal College of Radiologists in collaboration with Society and College of Radiographers (RCR & SCoR). Both guidelines are discussed in detail and the minor differences are highlighted. In Europe, the European Society of Paediatric Radiology (ESPR) advises to adhere to the RCR & SCoR guideline.

3.2.1.1 American College of Radiology

According to the ACR, the use of specific imaging techniques in suspected physical child abuse will depend on the age of the child and the signs and symptoms presented [7]. For this purpose the ACR uses the following guidelines; for completeness, not just conventional radiology is mentioned:

1. Suspected physical abuse. Child ≤24 months of age. Neurological or visceral injuries not clinically suspected. Initial imaging evaluation.
   (a) A full skeletal survey (Table 3.1).
   (b) Tc-99 m bone scan whole body; can be a complementary/adjunctive examination for detecting skeletal trauma. It should only be used when the radiographic skeletal survey is negative but clinical suspicion remains high and search for further evidence of skeletal trauma is warranted.
   (c) There is no strong evidence to recommend universal screening with neuroimaging. However, clinicians should have low threshold for performing head CT or MRI in young children with suspected child abuse.
2. Suspected physical abuse. Child >24 months of age. Neurological or visceral injuries not clinically suspected. Initial imaging evaluation.
   (a) Initial imaging should focus on the areas of clinical concern. In children >2 years of age, skeletal survey is usually not done but may be performed based on clinical findings and the need to document the presence or absence of injuries.
   (b) There is no strong evidence to recommend universal screening with neuroimaging in the absence of clinical suspicion for AHT.
3. Child with one or more of the following: neurologic signs or symptoms, apnoea, complex skull fracture, other fractures, or injuries highly suspicious for child abuse. Initial imaging evaluation.
   (a) A full skeletal survey in all children <2 years of age in whom there is suspicion of abuse (Table 3.1).
   (b) Tc-99 m bone scan whole body (see point 1).
   (c) MRI scan of the head should be performed if the clinical presentation warrants further assessment.
   (d) MRI of the cervical spine should be strongly considered at the time of MRI brain imaging.
   (e) MRI of the total spine should be reserved for cases where the distinction between abusive and accidental trauma is not clear.
4. Child. Suspected physical abuse. Suspected thoracic or abdominopelvic injuries (e.g. abdominal skin bruises, distension, tenderness, or elevated liver or pancreatic enzymes). Initial imaging evaluation.
   (a) A full skeletal survey in all children <2 years of age in whom there is suspicion of abuse (Table 3.1).
   (b) Tc-99 m bone scan whole body (see point 1).

Table 3.1 Radiographic protocol for suspected child abuse [7, 8]

	ACR	RCR and SCoR
Skull[a]	AP Lateral Additional view when indicated: oblique or Towne view	AP Lateral
Cervical spine	AP Lateral	b
Thorax	AP and lateral, to include ribs and thoracic and upper lumbar spine[b]	AP, to include the shoulders Both obliques (to include all the ribs, left and right) Lateral to include the whole spine[c]
Abdomen, lumbosacral spine, pelvis	AP pelvis, to include the mid lumbar spine Lateral lumbosacral spine	AP abdomen and pelvis
Upper extremities	AP of the humerus AP radius/ulna	Where possible: AP of the whole arm (centred at the elbow if possible) Coned lateral elbow Coned lateral wrist In larger children: AP humerus (including the shoulder and elbow) AP forearm (including the elbow and wrist) Coned lateral elbow Coned lateral wrist
Lower extremities	AP of the femur AP tibia/fibula	Where possible: Whole AP lower limb, hip to ankle Coned lateral knee Coned lateral ankle Coned AP ankle (Mortise view) In larger children: AP femur AP tibia and fibula AP knee AP ankle Coned lateral knee Coned lateral ankle
Hands	PA	PA hand and wrist
Feet	AP/PA	AP
Follow-up	Approximately 2 weeks after the initial examination	Any of the abnormal or suspicious areas on the initial skeletal view plus the following views:
Chest		AP, to include the shoulders. Both obliques (to include all the ribs, left and right).
Upper extremities		Where possible: AP of the whole arm (centred at the elbow if possible) In larger children: AP humerus (including the shoulder and elbow) AP forearm (including the elbow and wrist)
Lower extremities		Where possible: Whole AP lower limb, hip to ankle In larger children: AP femur AP tibia and fibula

[a] Always part of a full examination, even if a head CT has been made. A linear skull fracture is not necessarily visible on the CT scan
[b] The addition of both oblique projections to the anteroposterior (AP) view of the rib cage may increase the yield of rib fractures
[c] For children under 1 year, this may be possible with one view, for larger children and those over 1 year, separate views will probably be required
[d] At this age, AP views of the cervical spine are hardly ever diagnostic and should only be made at the request of the radiologist

(c) CT or MRI of the head should also be performed in children with neurologic symptoms or risk factors for intracranial injuries.
(d) Contrast-enhanced CT of the abdomen is indicated in acute evaluation of the child with suspected abdominopelvic injuries. Routine CT scan screening for abdominal or chest injury is not recommended.

In other words: when child abuse is suspected, radiological examination is always advised in children <2 years old, and in children >2 years only when there are further serious external or internal injuries.

When the radiographs show any abnormalities, a view in a second plane should be made. A repeat skeletal survey should be performed approximately 2 weeks after the initial examination.

3.2.1.2 The Royal College of Radiologists and the Society and College of Radiographers

In September 2017, the RCR & SCoR formulated a British guideline, endorsed by the Royal College of Paediatrics and Child Health, for imaging when child abuse is suspected (Table 3.1) [8]. According to this guideline, a skeletal survey should be made in each child <2 years who is suspected of being subjected to child abuse. In view of the medical/legal implications of this examination, this skeletal survey should meet the highest technical standards and as such should be made by two trained radiographers. The examination should be performed under the supervision of a radiologist, who also safeguards the quality of the examination. The child is only allowed to leave the radiology department after the radiologist has approved the complete examination.

An important difference with the ACR protocol is the standard addition of oblique views of the ribs (Fig. 3.1a, b). Ingram et al. showed in a randomized control study that this increases the sensitivity of the detection of rib fractures by 17% (95% CI 2–36%) and the specificity by 7% (95% CI 2–13%) [9]. Hansen et al. described a series of 22 patients in which the oblique view changed the interpretation in 12 cases ($p = 0.02$) [10]. In these 12 cases, 19 rib fractures were found on the oblique views, and six fractures were excluded. All patients with rib fractures showed at least one fracture on the anterior-posterior and lateral views. A similar study by Marine et al found that in a group of 212 patients (106 patients with at least one rib fracture and 106 patients without rib fractures), when the four-view series were used as a gold standard, the sensitivity and specificity for any rib fracture on the two-view series was respectively 57% (range 47–70) and 99% (range 98–100) [11]. For posterior rib fractures this was almost identical at respectively 59% (range 53–67) and 99% (range 97–100). The reported confidence increased from 0.022 to 0.061 for the two-view series to 0.007 to 0.031 for the four-view series ($p < 0.001$).

The authors of this chapter have one comment with regard to the updated RCR & SCoR guideline and that relates to the use of AP radiographs of the whole limb. We feel that, based on our personal experiences, this should be discouraged as it can lead to insufficient radiographs in children who actually are too big for this approach. It is a well-known fact that in general skeletal surveys a substantial deviation from the protocols in use is seen, and adding an additional option to the process will certainly not lead to an improvement [12–14].

Although this book covers fractures and the imaging thereof, we feel that it is important to underline the importance of the complete workup of children, in whom child abuse is suspected. According to the guidelines all children under the age of 1 year and those children older than 1 year who have external evidence of head trauma and/or neurological symptoms should undergo neuroimaging. This should be done according to the flow chart shown in Fig. 3.2.

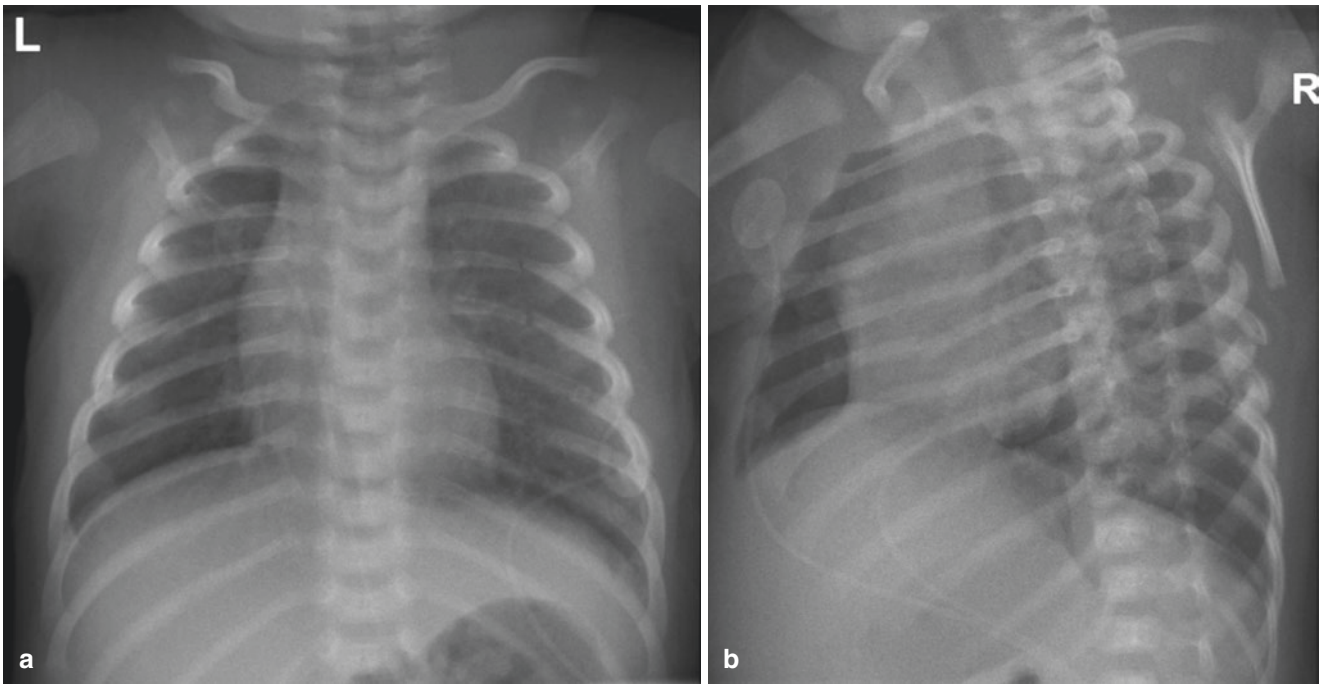


Fig. 3.1 Infant with bilateral recent rib fractures, although visible on the AP chest radiograph (**a**), the oblique chest radiograph (**b**) increases their visibility

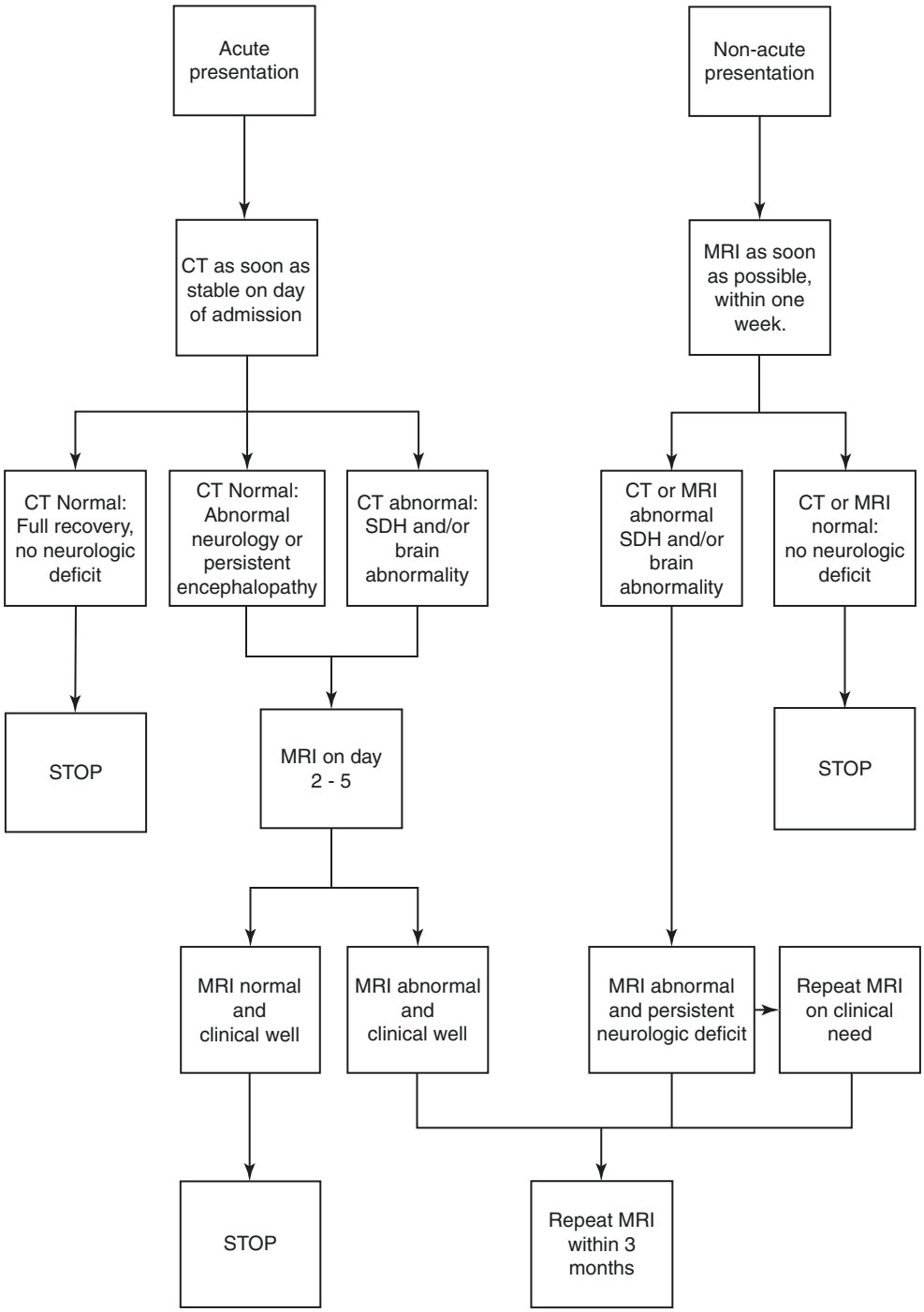


Fig. 3.2 Flow chart for neuroradiological imaging as proposed by the Royal College of Radiology and the Society and College of Radiography

3.2.1.3 Examination on Indication

Besides the indications in the earlier-mentioned guidelines, there are also further situations in which imaging may be indicated. Among these situations we would like to highlight young children with burns and drug-endangered children. In the first group it is known that a significant proportion are non-accidental burns, according to a systematic review the incidence in all children (aged 0–17 years) ranged from 0.5% to 24.6%, in children under the age of 13 this was up to 25%, with pooled data showing an incidence of 9.7% [15]. Hicks and Stolfi studied a small group of children with burns who underwent a skeletal survey, and they found that 5 (14%) out of 36 children had a positive skeletal survey [16]. Fagen et al. studied a group of 112 children with burns, mean age 15 months (range 1 month to 110 months) [17]. They grouped the children into three categories; non-accidental, indeterminate, and accidental. The outcome of the skeletal surveys were positive for respectively 15/45 (33%), 2/36 (6%), and 0/29 (0%). Degraw et al. studied a group of 97 children, under the age of 24 months, with burns who were referred for subspecialty child abuse evaluation [18]. Of these 97 children 18 (18.6%) were found to have occult fractures on the skeletal

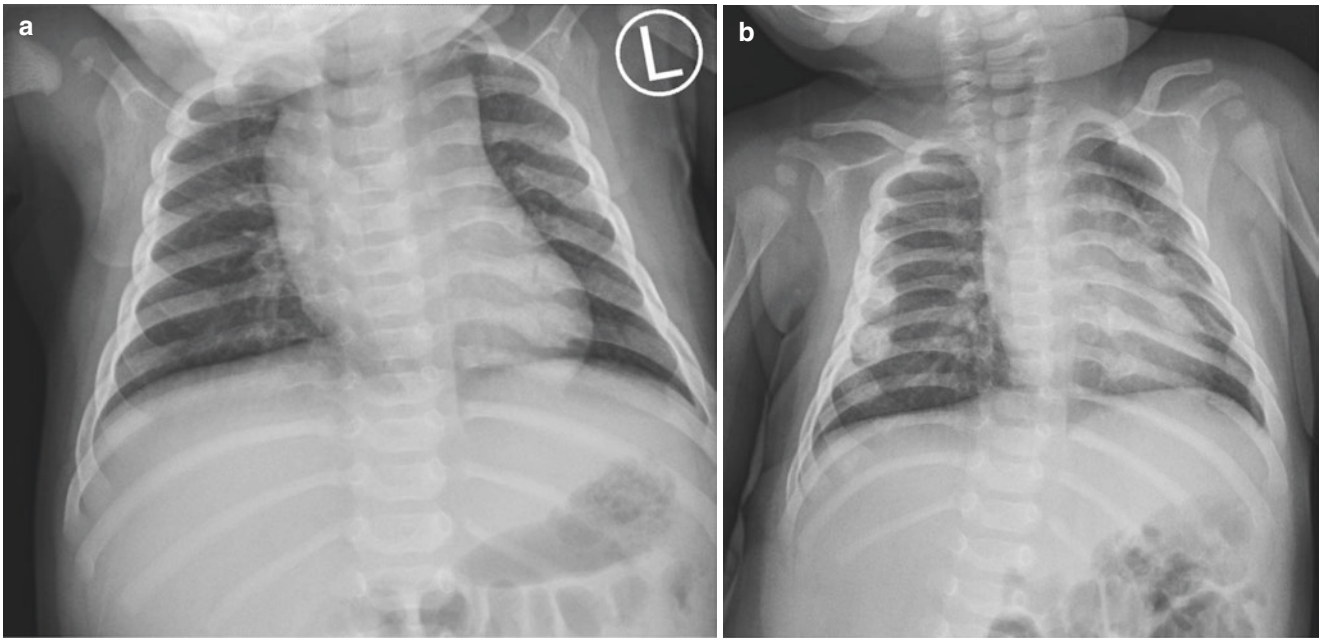


Fig. 3.3 (**a**) Three-month-old infant with multiple left sided posterior healing rib fractures. (**b**) the twin, who underwent a skeletal survey due to the findings in the sib, showed multiple bilateral healing rib fractures

survey. In the second group it is a well-known fact that in children who have parents with illicit drug use there is a higher risk for child abuse [19–21]. Howell et al. studied the yield of skeletal surveys in a group of 1252 children referred to their C.A.R.E. Team for drug endangerment. Of these children, 340 underwent a skeletal survey with 12 (4%) positive cases [19].

Studies have shown that physicians dealing with suspicions of child abuse are of the opinion that when one child of a family is abused, this is sufficient reason to subject the other children in the family to medical examination (Fig. 3.3a, b) [22]. In a retrospective analysis of 759 siblings of 400 index children, it appeared that in 37% of cases abuse was directed to all children and in 20% to one or several children in the family [23]. In a more recent study Lindberg et al. found that in a population of 134 siblings of index children referred to child advocacy teams, under 24 months of age, that in 16 (11.9%, 95% CI 7.5–18.5) at least one abusive fracture was diagnosed [24]. It is of interest to note that in none of these children associated findings were present on physical examination. Furthermore, they found that twins were at increased risk compared to nontwin contacts (odds ratio 20.1, 95% CI 5.8–69.9). The authors of this study concluded that '*A skeletal survey should be obtained in the contacts of injured, abused children for contacts who are <24 months old, regardless of physical examination findings*'. This is in keeping with the current protocol of the Royal College of Radiologists.

Sometimes the question arises whether or not to perform a skeletal survey in children aged over 24 months of age. A study by McNamara et al. showed that out of 325 skeletal surveys performed in children over 24 months of age (mean age 37.2 months, SD 16.5) 88 (27.1) were positive [25]. Of these, 88 showed known fractures only, in 1 case the authors state 'fracture without enough information' and in only 6 (2%) cases occult fractures were found. Of these 4 cases were physically abused, where in each case the diagnosis was already made, and in 2 cases a fall from a window caused the fractures. This study shows that in individual cases a skeletal survey over the age of 24 months can be obtained but the expected yield is low.

3.2.2 Adequacy of Examination

3.2.2.1 Number of Views

When child abuse is suspected, and the decision is made to continue with radiological examination, this should be conducted adequately. It should first be established that in young infants the so-called babygram (consisting of one anterior-posterior view and one lateral view) of the skeleton should be considered obsolete and an error of judgement (Fig. 3.4a, b). In diagnostic radiology, a babygram is inadequate when child abuse is suspected [26]. According to professional standards, this radiograph, preferably made on a mammograph, is only admissible in premature foetuses in which imaging is otherwise impossible.

It regularly happens that the radiological examinations performed do not meet the required standard. Offiah and Hall studied the quality of radiological examinations performed within the scope of child abuse that were submitted for re-evaluation to Great Ormond Street Children's Hospital (London, UK) [13]. They used three exclusion criteria:

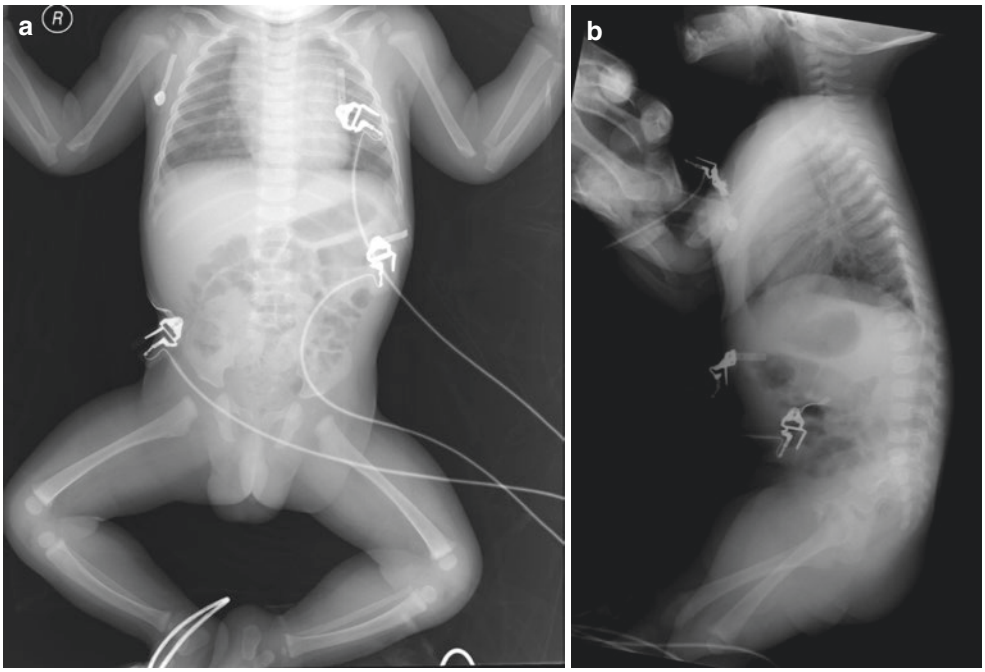


Fig. 3.4 (**a**) So-called babygram within the scope of a child abuse protocol. The use of a babygram for diagnostic purposes is obsolete when child abuse is suspected and should be considered a serious flaw with regard to living as well as deceased children. (**b**) Lateral view of a babygram

- Examinations in which only a selection of the produced radiographs was submitted for re-evaluation.
- Examinations of less than three radiographs (excluding babygrams).
- Examinations of children ≥2 years.

In total they admitted the skeletal surveys of 50 consecutive children to their study. Per child an average of 10 [2–13] radiographs were made. Hereby it should be mentioned that a professionally executed skeletal survey comprises 18–19 radiographs. In total, Offiah and Hall found 37 different combinations, including five babygrams. None of the examinations met the required standard. In general, hands and feet radiographs were absent. A study of Kleinman et al. from the United States confirmed the findings of Offiah and Hall [12]. As part of their study they inquired, by means of a questionnaire, in 155 paediatric hospitals which radiological protocol was used when child abuse was suspected. Of the 155 hospitals, 69% returned the questionnaire. Of these responders, 90.7% were members of the Society for Pediatric Radiology (SPR). Here too, a large variety was seen in the number of radiographs made. Van Rijn et al. evaluated the Dutch practices with regard to the radiological examination used in suspected child abuse, and found that only 7% of the reviewed skeletal surveys complied with the ACR criteria [14].

Hulson et al. performed a web-based survey among members of the European Society of Paediatric Radiology and found a considerable difference in practice across Europe, this was however before the ESPR adopted the guidelines of the Royal Collage of Radiologists [27]. Swinson et al. studied the effects of the publication of the guidelines of the British Society of Paediatric Radiology (followed by the guidelines for The Royal Collage of Radiologists and the Royal College of Paediatrics and Child Health) and compared their findings with the earlier-mentioned article of Offiah and Hall [28]. Their study still showed a considerable deviation in imaging, but significantly less so than in the earlier study. The publication of guidelines and education of the physicians involved seem to have a positive effect on the quality of imaging in cases suspected of child abuse. The same finding was reported by Patel et al. who evaluated the quality of radiographs in the skeletal surveys and found an improvement in the content of skeletal surveys [29]. More recently, Wanner et al. conducted an intervention study among members of the ACR in 69 different referring hospitals [30]. During this study they showed that after a 21-month intervention period there was a significant improvement in the total number of compliant views per skeletal survey.

3.2.2.2 Technique

Not only is it essential that the examination is complete, the techniques used are also of great importance.

When film-screen combinations are still used, a film with a speed of maximal 200 and a resolution of at least 10 line pairs per millimetre should be employed. The use of a grid is undesirable. When digital radiology is used (CR/DR), the optimal parameters for imaging a child skeleton should be chosen. When the radiograph is made, the extremities should be fully extended. Up to the present, the influence of digital radiology on the detection of subtle anomalies has not been investigated yet.

In view of the social and medical/legal implications, in this examination quality is of the essence. For this reason it is advised to perform this examination during office hours, unless a medical indication necessitates acute execution of the examination. In all cases, the (paediatric) radiologist must see the radiographs immediately after they have been made. The patient is only allowed to leave the department

after the radiologist has approved the examination and decided that no additional views were required.

The examination should be performed by at least two radiographers. From a judicial point of view, it is important that they can be traced via the initials on the radiographs. The radiology report needs to be complete and all abnormalities must be reported separately; furthermore, attention should be paid to dating the abnormalities (see Chap. 11). Finally, the report must systematically describe all findings in an orderly fashion and state whether the reported anomalies are suspect for inflicted injuries and whether it concurs with the provided clinical information.

When the complete skeletal survey confirms suspected non-accidental fractures, or when anomalies suspect for non-accidental fractures are found in routine radiographs of the child, this should be explicitly mentioned in the conclusion.

3.2.2.3 Technical Shortcomings in Making a Skeletal Survey

Even when the skeletal survey is made according to valid guidelines, there will be technical shortcomings that will complicate the evaluation or make it impossible to evaluate the radiological examination. In the retrospective study of Offiah and Hall it was shown that 35% of all images showed an artefact [13]. The most prevalent mistake was the presence of a hand to steady the child (32%). Other artefacts were e.g. the presence of drip-lines, buttons, and identification bracelets. In the study of van Rijn et al., artefacts were also frequently seen (17.5% of all radiographs); in these cases, the researchers frequently saw hands, drip-lines, diapers, and bracelets (Fig. 3.5a–h) [14].

It should be mentioned that when a child dies while hospitalized, it is not allowed to remove the drip-lines and tubes

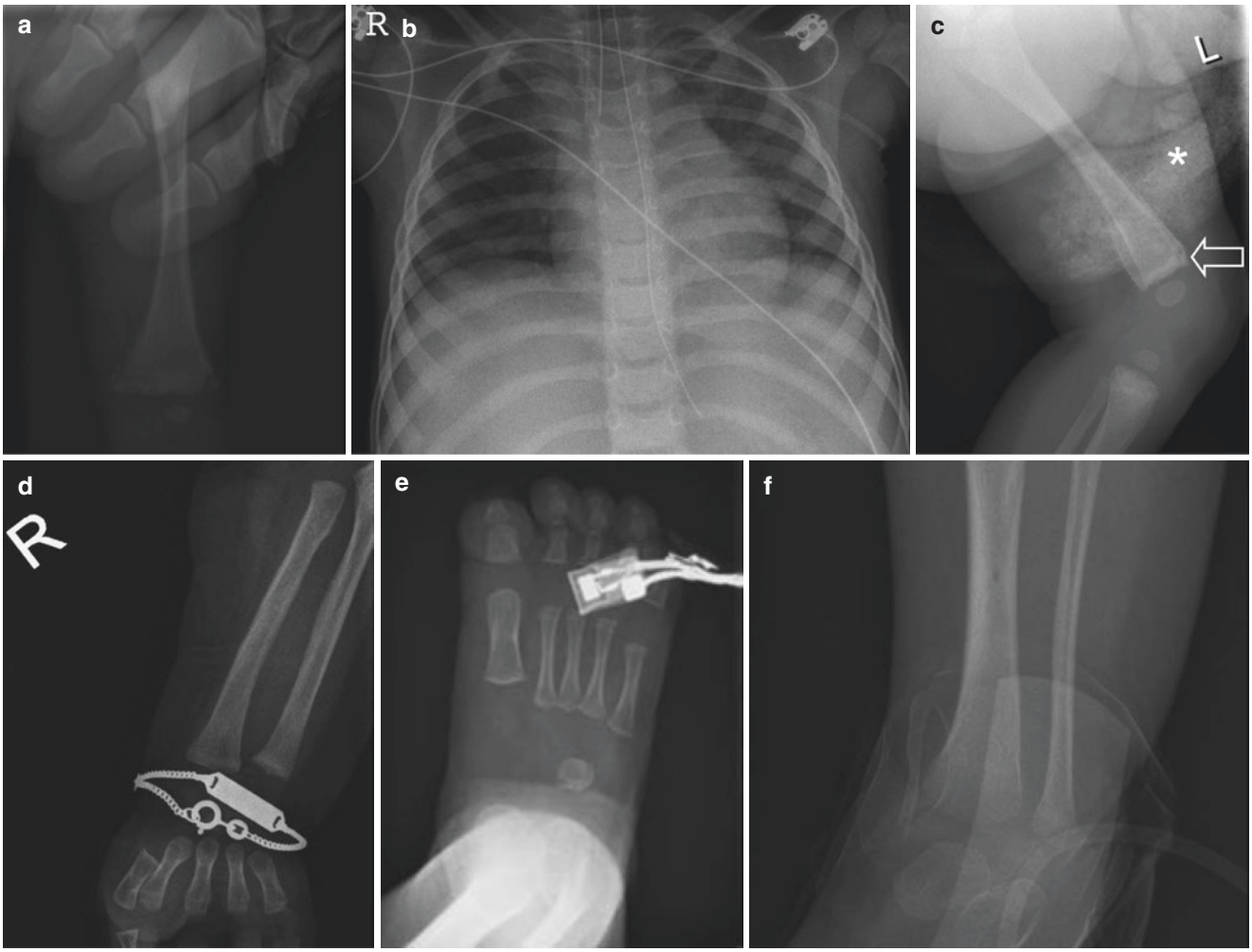


Fig. 3.5 (**a**) The hands of the radiographer are projected over the proximal femur metaphysis. (**b**) Although the gastric tube and trachea cannula cannot be removed, one should remove all other lines (situated outside the patent) as much as possible. (**c**) The diaper is clearly visible (asterisk) and can adversely influence the diagnosis. In spite of the presence of the diaper, a healing metaphyseal corner fracture with callus formation can be seen along the femoral shaft (*open arrow*). (**d**) Identification bracelet that nearly covers the distal metaphysis of the radius. (**e**) The position of the pulse oximeter makes it impossible to evaluate the phalanges of digits 3–5 of the foot. (**f**) Bandage used to stabilize the drip makes it impossible to evaluate the distal tibia and fibula. (**g**) Press studs of a baby suit projected over the left costal arch. (**h**) On the left side a radiograph of the dressed arm and on the right side the same arm after undressing, note the wrinkles of clothing that could cover or mimic a subtle fracture

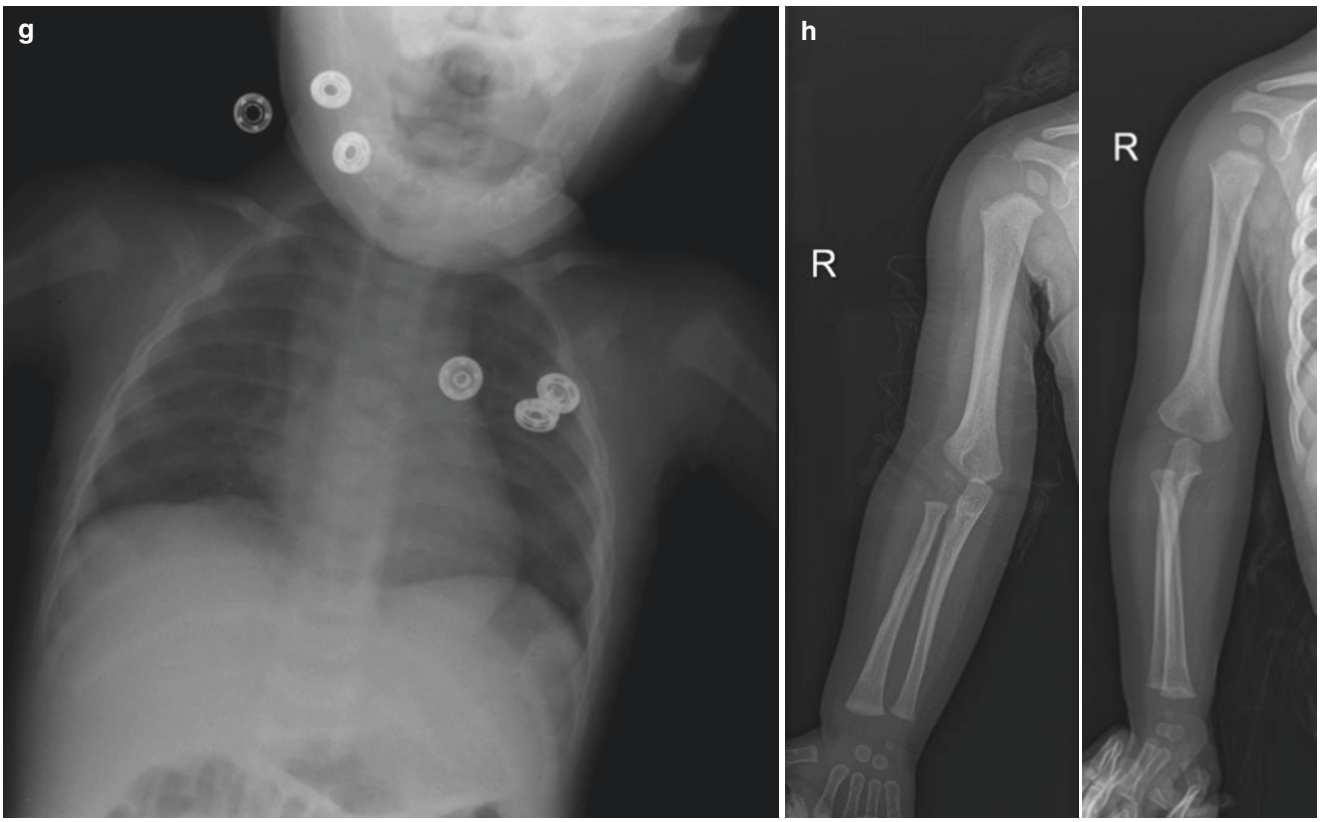


Fig. 3.5 (continued)

that have been inserted by physicians before autopsy (Fig. 3.6a, b). In this case, their presence on the skeletal survey is not considered to be a technical shortcoming.

3.2.2.4 Follow-Up Skeletal Survey

When a radiograph of a tender area found at physical examination does initially not show any anomalies, a follow-up radiograph after 2 weeks is indicated to show or exclude callus formation or a subperiosteal haemorrhage or an epiphyseal injury without dislocation (Fig. 3.7a, b). In the new RCR guideline, a limited follow-up skeletal survey is proposed (Table 3.1). In this follow-up skeletal survey, all abnormal or suspicious areas on the initial skeletal survey are visualized as well as a limited set of additional radiographs. Using this approach a significant reduction in radiation exposure is achieved.

As early as 1996, Kleinman et al. described this positive effect in their study that comprised 23 children who had sustained fractures [31]. In 61% of children, additional information was found. The number of confirmed fractures went up from 70 to 89, an increase of 27% ($p = 0.005$). According to Kleinman et al., repeating the examination also assisted in the dating of a number of fractures. Unfortunately, they did not mention which data they had used in this case [32].

Zimmerman et al. described the results of follow-up examinations in 48 patients [33]. In 46% of cases, the follow-up examination provided additional information. In 11 children, 27 fractures were found that had not been diagnosed earlier. These were mainly rib fractures (51%) and metaphyseal corner fractures (11%). Furthermore, in 15 children ambiguous anomalies were confirmed. In one child, in whom ambiguous fractures of the three metatarsals were seen, no indications for fractures were seen at follow-up examination. Consequently, the suspicion of child abuse could be rejected.

Harper et al. found that in a series of 796 follow-up skeletal surveys a total of 174 (21.5%) had new findings [34]. This included at least one new fracture detected in 124 cases (15.6%) and 55 cases (6.9%) where the findings were reassuring compared to the initial skeletal survey. The follow-up skeletal survey frequently affected the perceived likelihood of physical abuse.

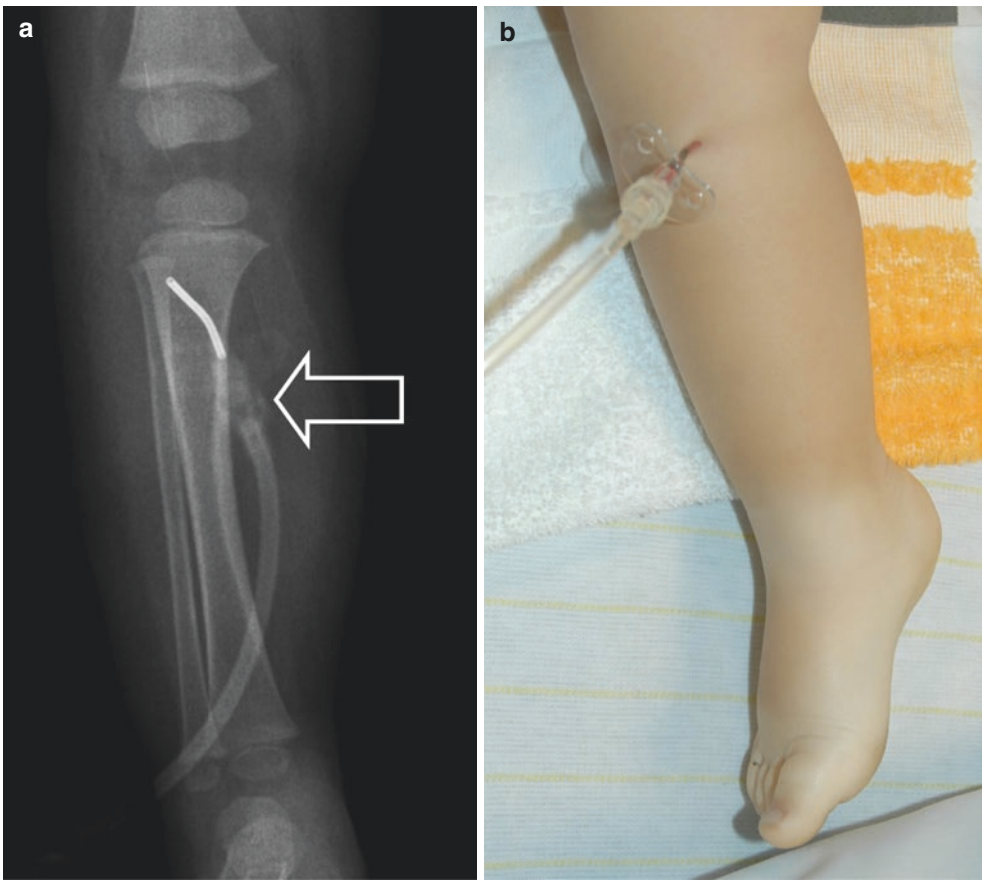


Fig. 3.6 (**a**) Post-mortem radiological examination with an intra-osseous vascular access needle in the right proximal tibia. After the patient has expired, it is, within the scope of trace investigations, not allowed to remove the needle. (**b**) Photo at autopsy shows the tibia needle in situ

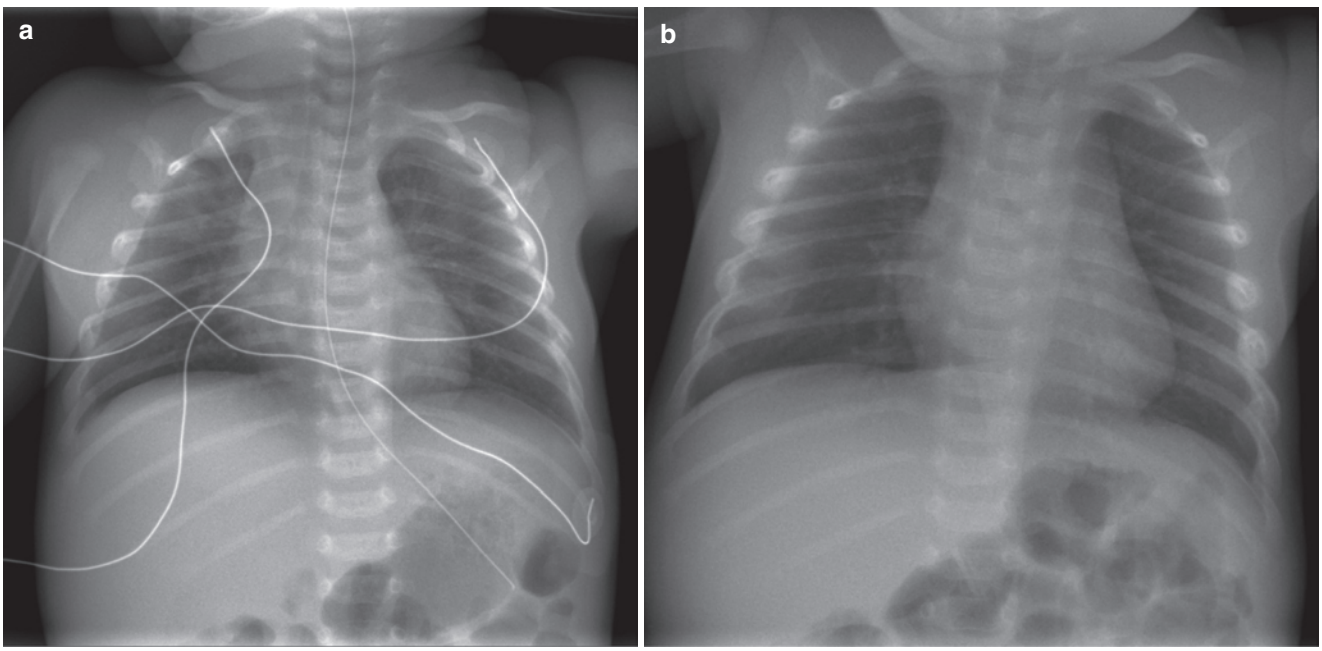


Fig. 3.7 (**a**) Initial chest radiograph shows an acute angle at the lateral aspect of the sixth rib on the left. On the right side a pleural thickening is seen. (**b**) Repeat radiograph after 14 days clearly shows callus formation on the lateral aspect of the third to sixth rib on the right and the fourth to seventh rib on the left

3.3 Bone Scintigraphy

Bone scintigraphy should not be used as an initial imaging technique in diagnosing child abuse (Fig. 3.8a–d). According to the guidelines of the ACR and RCR-SCoR, bone scintigraphy is only indicated when, after a full skeletal survey in which no anomalies are found, the presence of non-accidental fractures is still suspected [7]. It should be kept in mind that with the decreasing use of bone scintigraphy in children due to replacement by PET-CT or MRI, the experience in reading these studies also diminishes. This can result in less reliable outcomes of bone scintigraphy studies in daily practice.

Drubach et al. proposed the use of ^{18}F-NaF PET in the diagnosis of occult fractures [35]. In a study of 22 patients younger than 2 years, they showed that the conventional skeletal survey showed a total of 156 fractures versus 200 fractures with ^{18}F-NaF PET. ^{18}F-NaF PET had a higher sensitivity for rib fractures but a lower sensitivity for metaphyseal corner fractures. Since this publication, to the best of our knowledge, no other ^{18}F-NaF PET studies in the field of suspected child abuse have been performed.

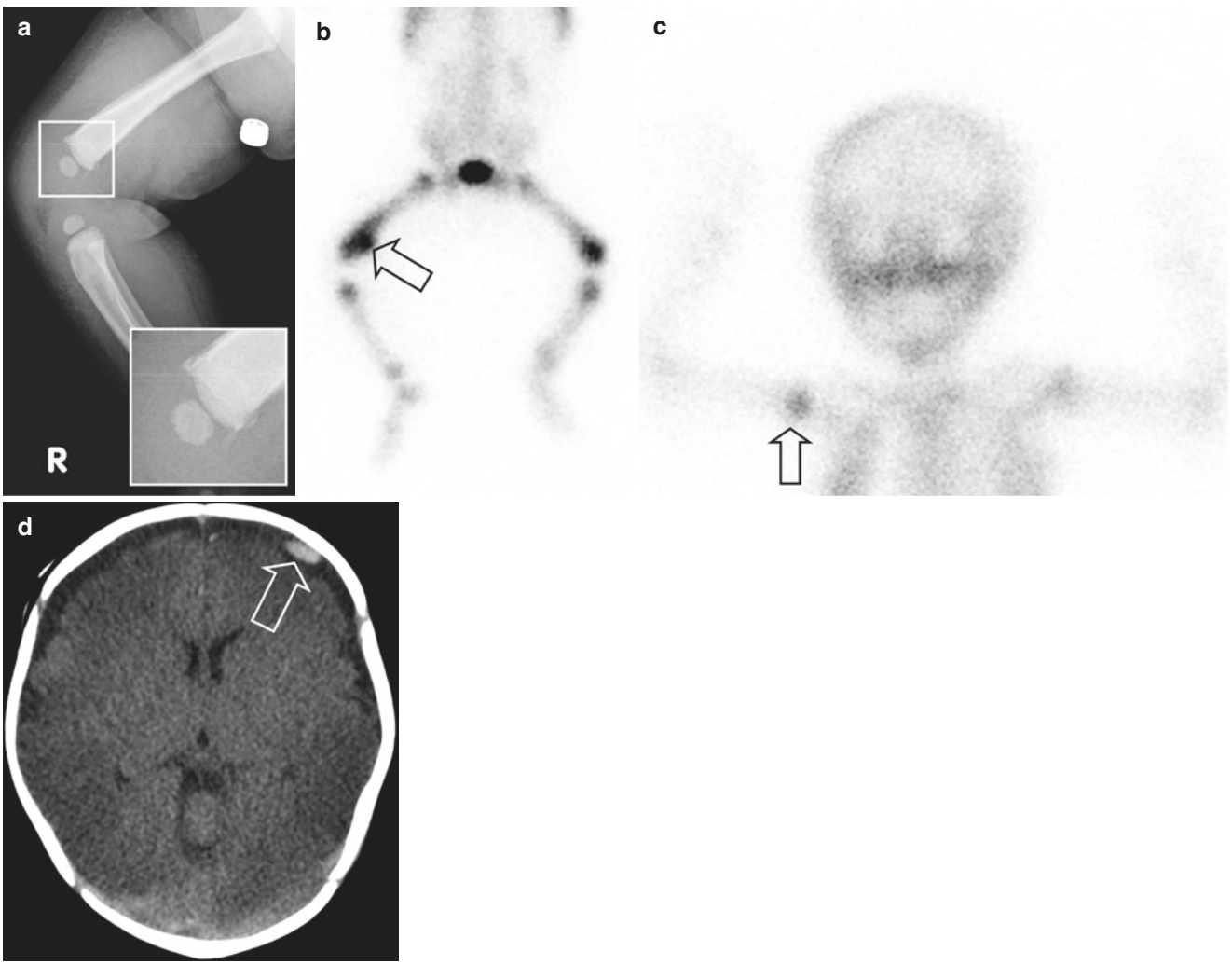


Fig. 3.8 (**a**) Three-month-old infant that had been presented at the emergency department with a swollen right knee. The radiograph shows a classic metaphyseal corner fracture (see inset); however, this had not been interpreted as such. (**b**) Bone scintigraphy, made the day after the radiograph, shows increased up-take around the right knee (*open arrow*). (**c**) Bone scintigraphy also shows increased up-take in the right proximal humerus (*open arrow*). This was not radiographed. (**d**) Five weeks after the initial examination, the patient presented again at the emergency department. However, this time she is in coma. A CT of the brain shows a subdural haemorrhage (*open arrow*) and bilateral diffuse ischaemic injuries

3.3.1 Effectivity of Bone Scintigraphy Versus Conventional Radiology

There are several publications which looked at the additional value of bone scintigraphy compared to a skeletal survey [36–42].

On behalf of the Welsh Child Protection Review Group, Kemp et al. compared the effectivity of bone scintigraphy and conventional radiology in cases of suspected child abuse [43]. Based on mainly case reports/series they came to the following conclusions: fractures will be overlooked in conventional radiology as well as in bone scintigraphy, bone scintigraphy is very sensitive for the detection of rib and acute fractures, whereas these may be overlooked in conventional radiology. And, when using bone scintigraphy, skull fractures, metaphyseal and epiphyseal fractures may be overlooked.

In a systematic review, Blangis et al. evaluated studies which assessed the value of bone scintigraphy after an initial negative skeletal survey, in total seven studies (with a total of 783 children) were included [44]. Based on the included studies, the authors concluded that a bone scan after a negative skeletal survey increased the summary absolute detection rates with an estimate of 10 percentage points. In approximately half of the children in whom non-accidental skeletal injuries were suspected the initial skeletal survey was negative. The summary number needed to scan with bone scintigraphy to detect one additional child with skeletal injury was 3. It is important to state that there was no assessment of the added value of bone scintigraphy to, as the standards dictate, a follow-up skeletal survey. Also looking at the included studies most studies suffered from methodological issues, e.g. retrospective, unclear patient selection and risk of inclusion bias. As a result the authors state that *'The quality of the reviewed evidence was low, pointing to the need for high-quality studies in this field'* [44]. So if this approach would be implemented it is to be suspected that the yield of bone scintigraphy would than be lower. Therefore, for now there is no indication to deviate from the ACR and SCR-SCoR guidelines and use bone scintigraphy, as a problem solving tool, only in exceptional cases.

3.4 Computed Tomography

Nowadays, CT is increasingly used in the primary evaluation of trauma victims. By now, studies in adults have shown that by using this technique relevant pathology will be found with a higher sensitivity and specificity, which leads to a significant improvement in the patient's prognosis [45–48]. However, in suspected child abuse CT in general has no place in the primary detection of occult fractures. A substantial percentage of fractures seen within the scope of suspected child abuse, e.g. rib fractures or CMLs, have neither from a diagnostic or a therapeutic point of view any need for additional imaging. The exception to this rule are fractures of the vertebrae, where it is essential to establish fracture stability. In these cases, in order to make a proficient evaluation, CT is required since this technique provides information on the stability of all three pillars of the spinal column.

Having said this CT has shown to be valuable in several anatomic areas which are relevant to potential child abuse cases, this is especially the case for rib and skull fractures. In recent years there have been several publications on the use of CT of the chest in the diagnosis of rib fractures, although there remains a discussion whether or not this should be done [49–52]. Sanchez et al., in a retrospective study, looked at 16 children under the age of 12 months with a total of 105 rib fractures [50]. Of these fractures 84% were seen on the first skeletal survey and 16% only after follow-up imaging, of which 11 out of 18 rib fractures were only seen on CT. Shelmerdine et al. retrospectively looked at 25 paired post-mortem skeletal surveys and post-mortem CT scans in a study population aged 1 month to 7 years [52]. In their study they found a total of 136 rib fractures at autopsy, three times as many rib fractures were correctly identified on CT compared to the skeletal survey (sensitivity 44.9% [95% CI 31.7–58.9] vs 13.5% [8.1–21.5]; difference 31.4% [23.3–37.8; $p < 0.001$]). Radiologists also reported a higher confidence when reporting on CT compared to the skeletal survey. In light of the radiation exposure of the chest CT, with the potential detrimental long-term consequences, it is not yet advised to perform a chest CT instead of the four-view chest radiographs. More research into the validation of low-dose chest CT scans is needed before routine chest CT imaging can be recommended.

In neurotrauma, CT is widely used for the primary evaluation of the patient. Over the past few years, authors in the radiological and paediatric literature increasingly argue the case for a standard head CT in all children of ≤2 years old who are suspected of being physically abused (Fig. 3.9) [53]. As CT is used more frequently, the question arises if conventional radiography of the skull is still needed. Culotta et al. performed a retrospective study in 167 children (median age 5 months) who were evaluated for potential AHT [54]. They found no significant difference ($p = 0.18$) between conventional radiography and CT. Sharp et al. performed a retrospective study in 94 infants (aged 24 days–23 months) in whom there was a suspicion of child abuse and in whom both conventional radiographs and CT of the head were made [55]. They found that in none of the cases conventional radiographs added findings over the findings on CT. Martin et al. performed a retrospective study in which they compared conventional radiographs, CT without 3D reconstruction, and CT with 3D reconstruction [56]. They found that CT with 3D reconstruction had a 100% sensitivity, specific-

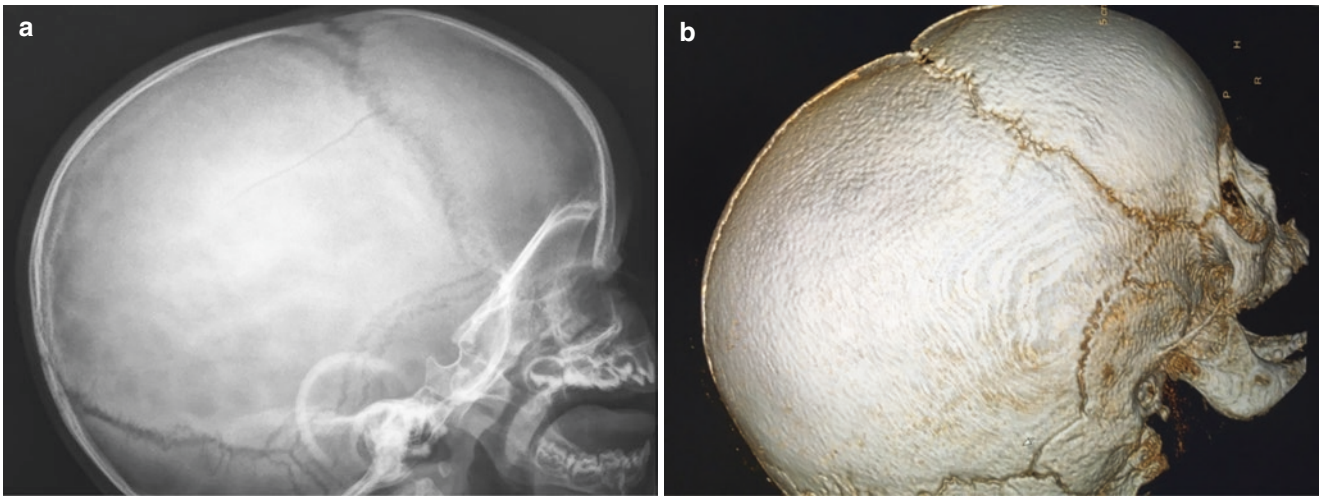


Fig. 3.9 (**a**) Small linear parietal fracture on the right side of the skull. (**b**) Shaded surface reconstruction of the CT scan. Note that as a result of the chosen setting the skull fracture appears to be shorter. This illustrates the need for proper assessment of the CT source data

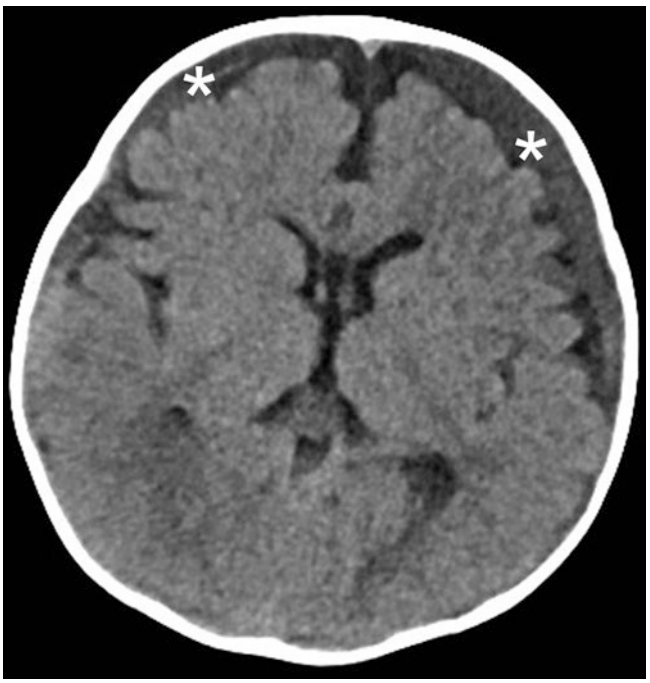


Fig. 3.10 CT of the head showing bilateral subdural haematomas in an infant with neurological symptoms and no clinical history of trauma

ity, PPV, and NPV (Fig. 3.10a, b). Based on these results the authors concluded '*Conventional radiographs (SRX) does not add further diagnostic information and can be omitted from the skeletal survey when CT with 3D reconstruction is going to be, or has been, performed*'. Penell et al. evaluated data from 158 infants who underwent both skull radiography and CT [57]. In their study population, they found 46 skull fractures on 3D CT and 40 on skull radiographs. The inter-rater reliability was higher for 3D CT ($\kappa = 0.95$) compared to skull radiographs (=0.65). Even though 5 fractures were identified on skull radiographs only, whereas 11 fractures were identified on 3D CT only, the authors concluded that omitting the skull radiograph is justified when a 3D CT of the skull is obtained. In an older study, Orman et al. retrospectively reviewed 250 paediatric cases (mean 7.82 years, range 4 days–17.4 years) with linear skull fractures on consensus reading by two experienced paediatric neuroradiologists [58]. Three reviewers (a third year resident, a fellow in neuroradiology, and a paediatric neuroradiologist) first reviewed the 2D dataset and later a combined 2D/3D dataset. They found that the combination of 2D&3D had a superior sensitivity and specificity (83.9% and 97.1%) compared to 2D only.

Based on this literature omitting the conventional skull radiographs, if a sufficient CT including 3D reconstructions is available, can be considered in cases of suspected child abuse.

3.5 Linear Slot Scanning

A relatively unknown and new technique, marketed as Statscan® (Lodox Systems, [Pty] Ltd., Sandton, South Africa), is linear slot scanning [59–73]. The Statscan® was initially developed to detect diamond smugglers working in the South African diamond mines [70, 74]. Since approximately 2000 it is also used in a trauma setting and in forensic facilities. This technique allows for full body imaging; for this a C-arm traverses the patient trolley along the Z-axis. As the C-arm can rotate up to a maximum angle of 100°, lateral and oblique radiographs are also possible (Fig. 3.11a, b). The system uses a collimated fan-beam and a linear bank of CCD cameras as a detector. The acquisition time of a scan is approximately 13 s [72]. Compared to a full skeletal survey

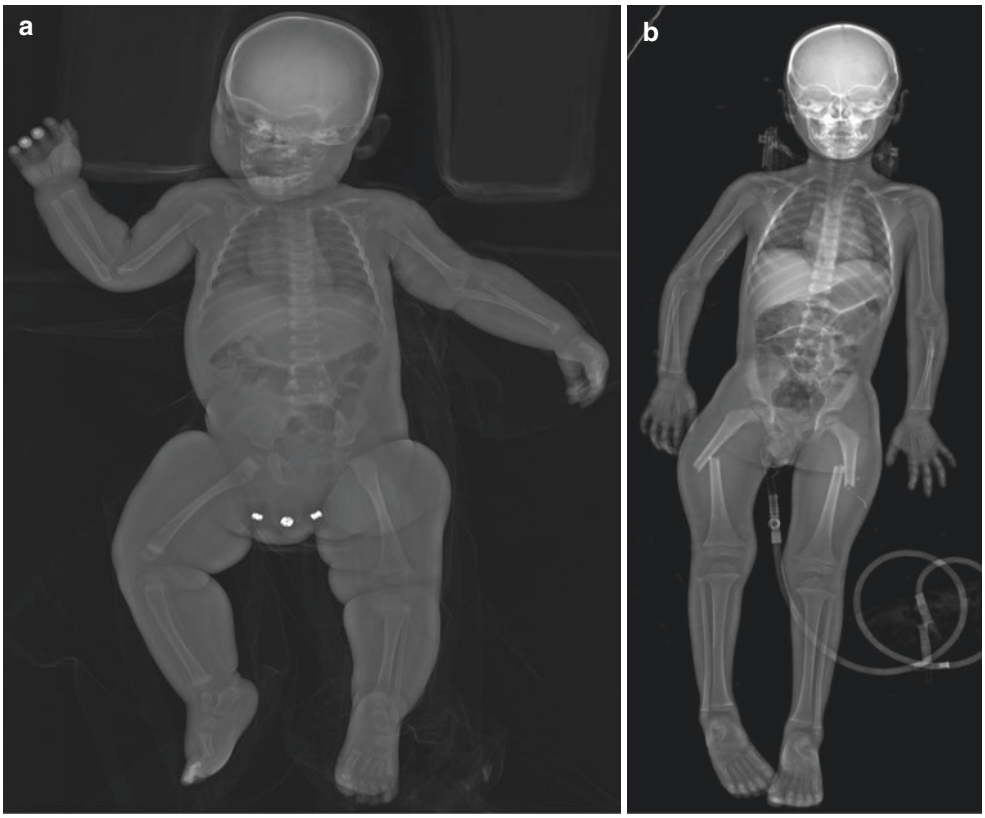


Fig. 3.11 Lodox image of a (**a**) neonate without fractures and (**b**) a child with bilateral femoral fractures (Courtesy of A. Speelman, Cape Peninsula University of Technology)

(in adults) the dose has been reported to be 65.0% to 94.0% less [72, 75]. Currently it has a noticeable install basis in the Southern part of Africa and in medical examiners offices throughout the United States [76].

There have been a few small studies into the use of Statscan in the paediatric trauma population [67, 68, 73]. The largest reported series is on 23 children where the AP linear slot bodygram showed 26 of 27 fractures (96%) in the study cohort. There is however no evidence with respect to the detection of, e.g. posterior rib fractures and metaphyseal corner fractures. In adults, a retrospective study in 245 consecutive trauma cases showed an overall sensitivity of 73% and a specificity of 100% [59]. However, in 50% of cases additional radiographs were obtained to provide a more detailed or an additional view for pre-operative planning. Spies et al. performed a post-mortem animal study using piglets comparing conventional radiology, Lodox, and CT to assess the sensitivity of Lodox for fracture detection [77]. A total of 586 fractures were created by blunt force trauma in 10 piglets, of which CT correctly detected 427 (73%), X-ray 294 (50%), and Lodox 245 (42%). Looking at just the ribs CT was most sensitive (84%) and Lodox least sensitive (50%).

Based on the literature evidence it could be concluded that if no CT scanner is available or to costly to acquire, linear slot scanning can be used to diagnose major trauma findings. For the detection of subtle fractures, dedicated spot radiographs will remain essential.

3.6 Magnetic Resonance Imaging

Up to the present, MRI is not widely used in the initial diagnosis of suspected child abuse. MRI is essential as a second stage imaging method in order to determine the prognosis in severe neurological trauma in intracranial as well as in spinal injuries (Fig. 3.12). In abdominal trauma, and more specifically in pancreatic injuries, MRI/MRCP may provide additional information on the intra-abdominal parenchymal organs (Fig. 3.13a, b).

With respect to fractures the use of MRI is limited. However, due to the relatively short scan times available in Short T1 Inverse Recovery (STIR), whole body imaging of children is possible. STIR is a sequence that yields a uniform fat suppression in the field of view leading to increased visualization of, e.g. bone marrow oedema. Clinical paediatric radiology has shown whole body STIR (WB-STIR) to be a sensitive technique for the detection of, e.g. bone metastases or foci of non-bacterial osteitis (Fig. 3.14a, b) [78–86]. Some authors suggest the use of WB-STIR for the detection of occult fractures in suspected child abuse (Fig. 3.15a–c) [33, 87, 88]. Besides a few case reports in which this technique has been described, two studies have been published in which WB-STIR and conventional skeletal surveys were compared [89, 90]. The first study comprised 16 children (average age 9 months; range: 1.5–37 months) that were suspected of being abused. The average time interval between

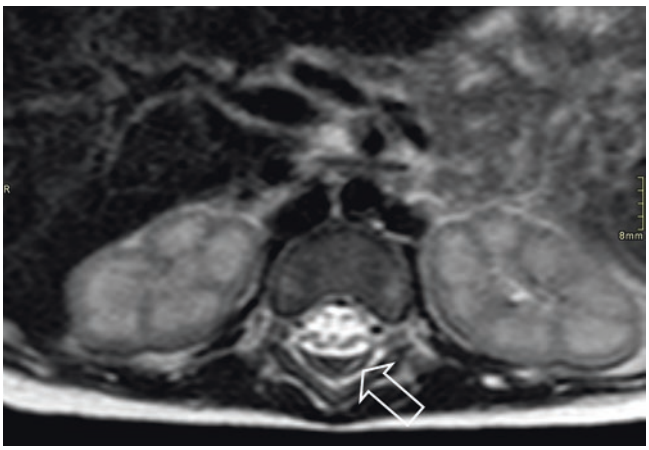


Fig. 3.12 Axial T2 weighted MRI showing an intraspinal subdural haematoma (arrow)

WB-STIR and conventional skeletal survey was 1.9 days (range: 0–13 days). The sensitivity of WB-STIR for rib fractures was 75% (33/44), CML 67% (2/3), metaphyseal fractures 100% (1/1), diaphyseal fractures 100% (6/6), and parietal skull fractures 100% (1/1). In total, 11 rib fractures were overlooked. However, all children had sustained multiple fractures and at least one rib fracture was detected by WB-STIR. In three children, WB-STIR showed fractures that had not been visible on the conventional skeletal survey. In the second study, the authors evaluated the additional value of WB-STIR and bone scintigraphy in addition to the initial conventional skeletal survey [90]. In this study 107 children under the age of 3 years who were suspected victims of child abuse were included. In this study, the skeletal survey had the highest sensitivity and specificity (88.4% [95% CI 82.0–93.1%] and 99.7% [95% CI 99.5–99.8%]),

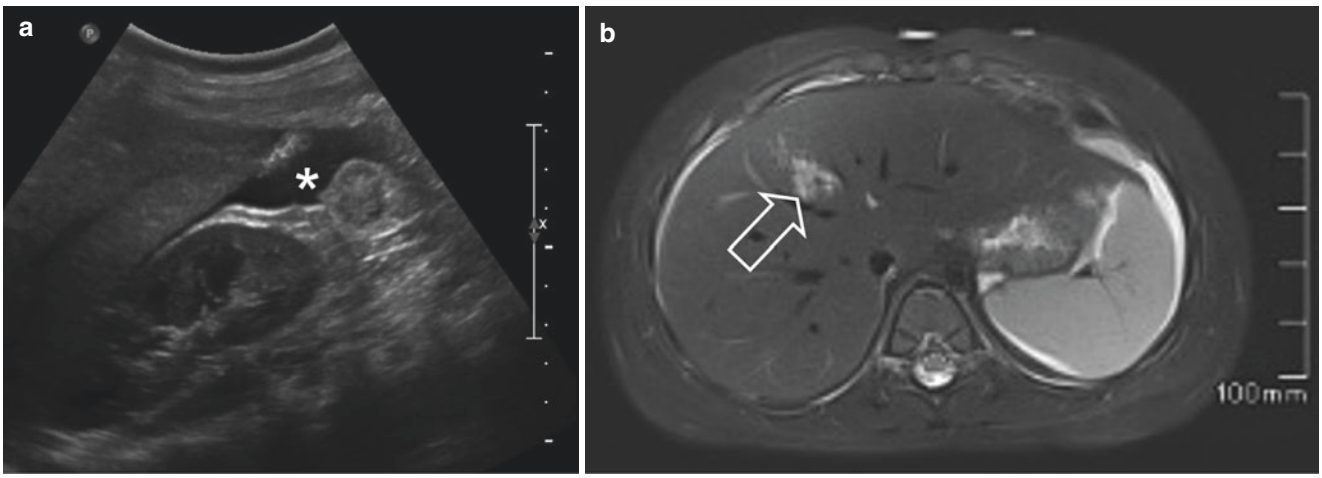
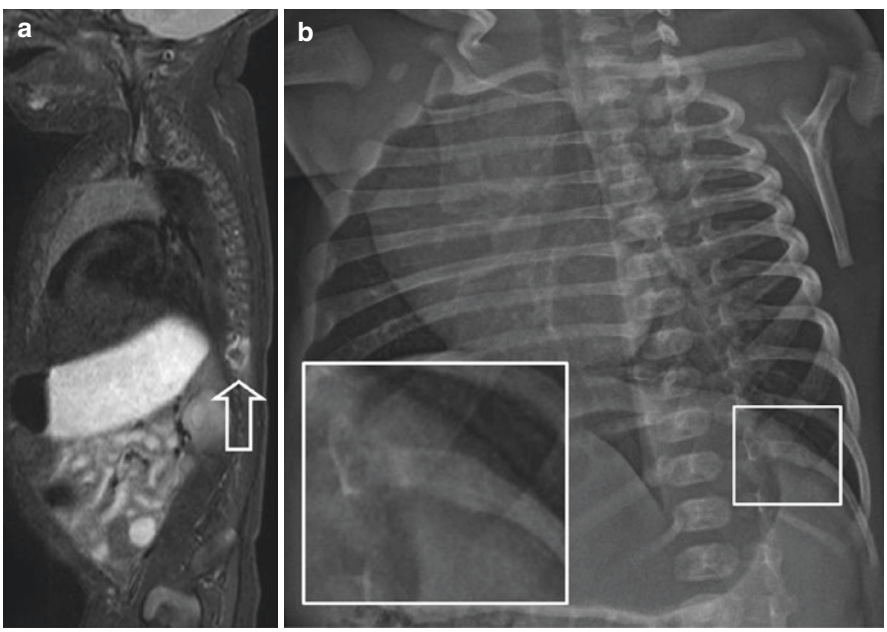


Fig. 3.13 (**a**) Positive focused assessment with sonography in trauma exam in a child with blunt abdominal trauma, fall with bicycle. (**b**) MRI shows a liver laceration in segment 4A (arrow)

Fig. 3.14 (**a**) Whole body MRI shows paravertebral oedema at the level of the 11th rib (arrow). (**b**) Follow-up oblique chest radiograph shows subtle callus formation of the posterior 11th rib (see inset)



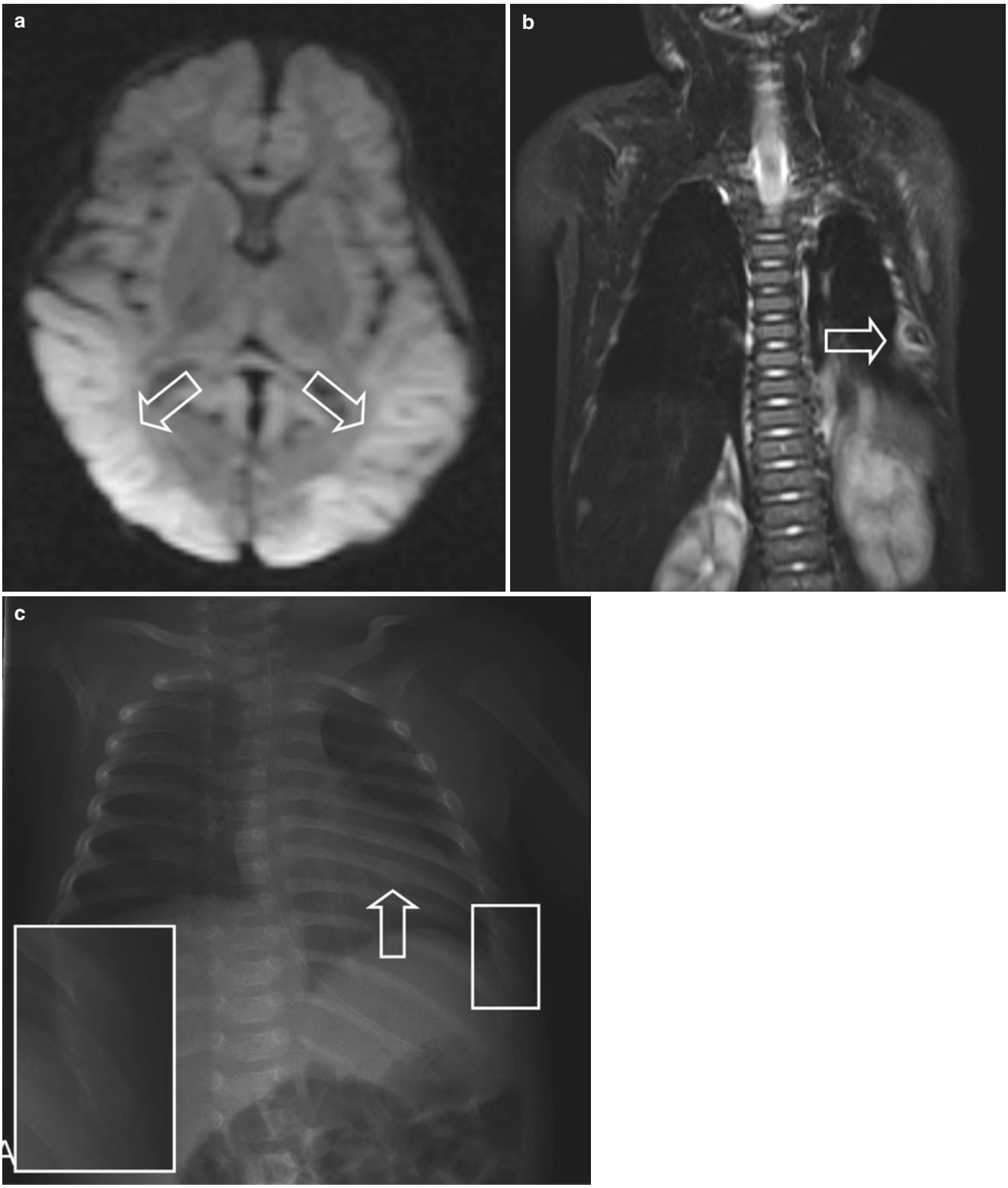


Fig. 3.15 (**a**) Two-month-old infant with severe inflicted traumatic brain injury. The diffusion-weighted views show areas of severe cytotoxic oedema (*open arrow*) resulting from hypoxia. (**b**) Cor STIR-weighted view of the chest shows increased signal intensity at the site of the seventh left rib (*open arrow*). This corresponds with a fresh fracture. (**c**) Oblique chest view, made 6 days after the MRI, shows callus formation at the site of the seventh left rib (*open arrow*). Furthermore, there are fractures visible at the costochondral junctions of ribs 7 and 8 (see inset)

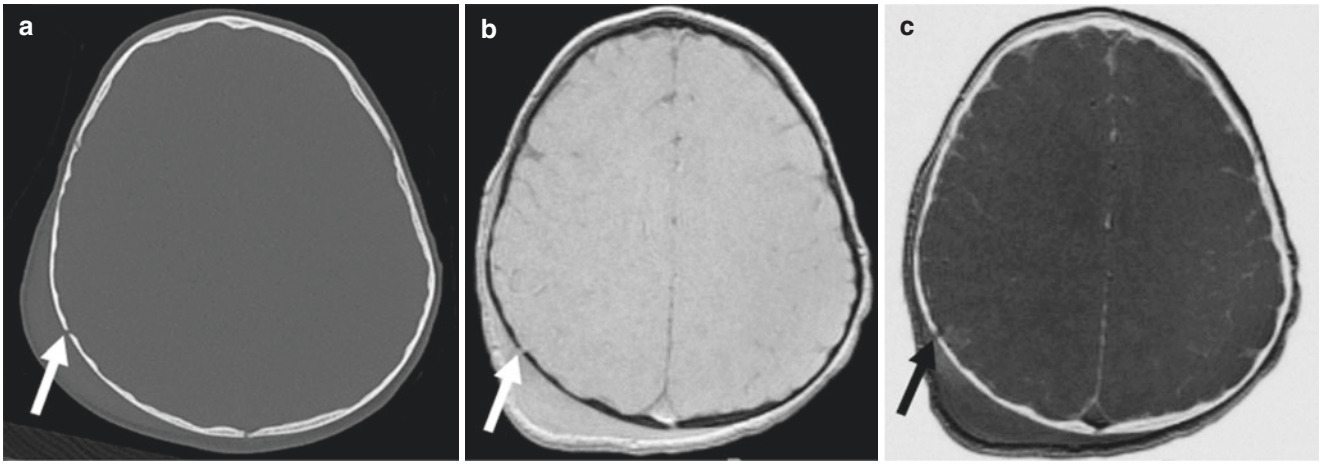


Fig. 3.16 (**a**) Infant with a right parietal skull fracture (arrow) with overlying haematoma. (**b**) Corresponding black bone sequence shows the fracture as well (arrow). (**c**) The inverted image shows an imaging resembling a CT scan (courtesy of M.H.G. Dremmen MD. PhD., Sophia Children's Hospital, Erasmus MC+, Rotterdam, the Netherlands)

followed by WB-STIR (69.9% [95% CI 61.7–77.2%] and 99.5% [95% CI 99.2–99.7%]) and bone scintigraphy (54.8% [95% CI 46.4–63.0%] and 99.7% [95% CI 99.5–99.9%]). The combination of the skeletal survey and WB-MRI had the highest sensitivity (95.9% [95% CI 91.3–98.5%]) and the combination of the skeletal survey and bone scintigraphy had the highest specificity (99.4% [95% CI 99.2–99.6%]). Based on their findings the authors concluded both WB-STIR and bone scintigraphy can be used in case of equivocal lesions. In many infants who are suspected to be a victim of child abuse MRI of the brain and spine will be obtained, and in these cases WB-MRI could be used as an 'add-on' to the standard brain and spine.

A relatively new development in the field of MRI is the use of the so-called 'black bone' sequence [91–93]. In 2012, Eley et al. were the first to present this sequence which consists of a low flip angle gradient echo MRI sequence providing high image contrast between bone and other tissues while at the same time reducing the contrast between soft tissues. There are only a handful of studies which have evaluated the sensitivity and specificity of this sequence compared to cranial CT as a gold standard. Dremmen et al. showed in a study of 28 children that 'black bone' MRI had a lower sensitivity (66.7% versus 100%) and specificity (87.5% versus 100%) (Fig. 3.16a–c) [94]. In a more recent study by Kralik et al., 'black bone' MRI showed an 83% sensitivity (95%[CI] 36–99%) and a 100% specificity (95%[CI] 88–100%) [95]. To date the evidence is insufficient to advocate replacing CT by 'black bone' MRI for the diagnosis of skull fractures.

3.7 Ultrasonography

In recent years, there have been some publications on the use of ultrasonography (US) in the diagnosis of fractures, due to non-accidental circumstances [96–100].

Marine et al. presented a descriptive retrospective study in 22 patients who had, based on the skeletal survey, a total of 39 CMLs [96]. In their population in 85% of cases the US exams were abnorma. The authors concluded that 'while a negative US does not exclude CML, US may have a role in either confirming or evaluating radiographically equivocal CMLs'. A recent retrospective study in 63 children by Karmazyn et al. focused on the accuracy of US for CMLs [97]. In this study, both in a group of children in whom the diagnosis CML was in doubt and in a group of children with radiologically proven CMLs US of the lower extremity was performed. Based on their findings the authors concluded that 'US has low sensitivity and high specificity in the diagnosis of CMLs in the lower extremities' and that 'US for CML may help substantiate the diagnosis' (Fig. 3.17a, b).

There are two publications on the use of US in diagnosing rib fractures, the first is by Kelloff et al. who describe a case of a 9-week-old infant [99]. The attending physician noted crepitus of the chest on physical exam and when the chest radiographs, including the obliques, were negative US was used as a problem solver. The follow-up skeletal survey showed healing rib fractures of the left sixth and seventh posterolateral ribs and right seventh, eighth, and ninth anterolateral ribs. Smeets et al. reported on a 9-month-old

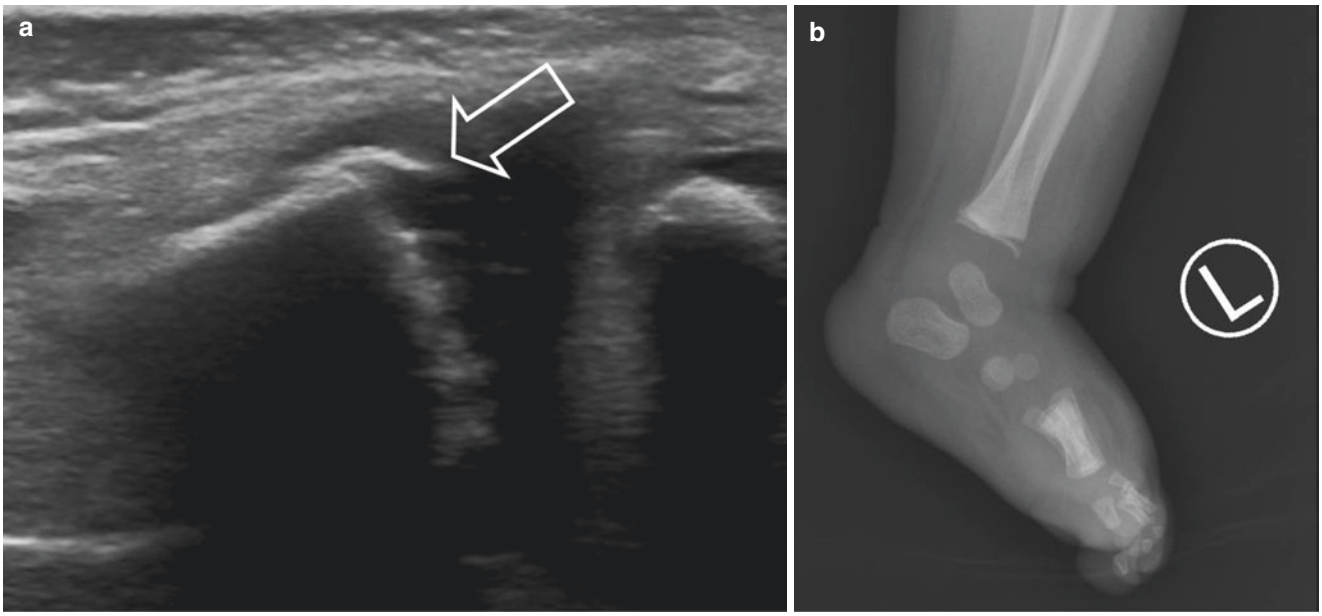


Fig. 3.17 (**a**) Ultrasonography of the distal tibia shows a metaphyseal corner fracture (**b**). Corresponding conventional radiograph of the ankle

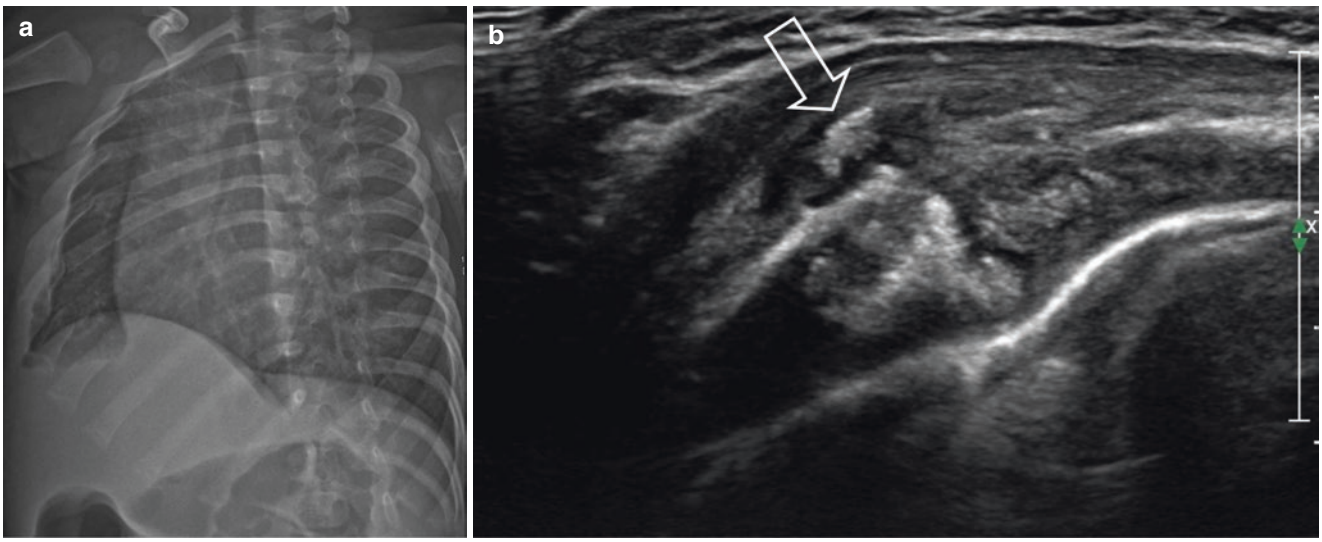


Fig. 3.18 (**a**) Chest radiograph shows multiple recent anterolateral right sided rib fractures. (**b**) Ultrasonography shows beginning callus formation (arrow)

girl that on a skeletal survey showed multiple healing rib fractures [100]. A soft tissue swelling was noted on the left lateral thoracic wall. US of this area revealed a subcutaneous haematoma overlying costochondral dislocations of all left lower ribs (Fig. 3.18a, b).

3.8 Bone Densitometry

In children presenting with fractures, the question of bone density in relation to bone fragility is often raised. In a publication from 1936 by Lachmann and Whelan, the authors stated that at least 20–40% of bone mass should be lost before it is visible on conventional radiography [101]. This is often mentioned in presentations and in court proceedings. However, the authors performed their experimental work on cadaveric bones and using film-screen combinations. The question is if this also applies to modern techniques and children. Recently, Rosendahl et al. performed a systematic review which addressed this question [102]. In their study, they couldn't find any evidence to support the statement that before it is noticeable on conventional radiography that 20–40% bone mass should be lost. Therefore, in order to make a statement on bone mass bone densitometry is neces-

sary. Within the world of bone densitometry historically the focus has been on middle aged women suffering from age-related osteoporosis, all techniques have been developed and initially validated for use in this specific population. However, once these techniques became more readily available it also found its way into the field of paediatrics.

The use of bone densitometry techniques in children is, without proper knowledge of its limitations, not without risks [103]. One of the obvious differences between adults and children is the propensity to grow for the latter group. As growth is a volumetric process techniques using a two-dimensional approach have the inherent problem of not only measuring a change in bone mass but also a change in size.

If bone densitometry is performed, the outcome of the study should not be reported as the *T*-score, as is customary in adults. In children the *Z*-Score should be adopted, this score not only adjusts for sex and racial background but also for age. One problem is that *Z*-score reference curves are in general only available for children aged 5 years and over, making it not useful for the age range in which children who are subject to physical child abuse tend to be [104, 105].

There are multiple techniques available to assess bone density, e.g. Dual-Energy X-ray Absorptiometry (DXA), Quantitative UltraSound (QUS), Digital Radiogrammetry (DXR), and Quantitative Computed Tomography (QCT) (Table 3.2) [106–108]. Of these, DXA is most widely in use and its use in children is recommended by the PDC. This technique measures bone mass as Bone Mineral Content (BMC, gram), Bone Mineral Density (BMD, gram/cm^2), or the derivative Bone Mineral apparent Density (BMaD, gram/cm^3) (Fig. 3.19).

With respect to reporting bone densitometry studies, the PDC states that terminology like 'T-score', 'Osteopenia', and 'Osteoporosis' based on densitometry studies only cannot be used in children. This as these terms are developed for studies in adult women. Therefore, in 2013 PDC revised the clinical criteria for the definition of osteoporosis in paediatrics. In this they defined osteoporosis as the finding of one or more vertebral compressions fractures (VCF) in the absence of local disease or high energy trauma, independently of densitometric results. Moreover, it was confirmed that, in the absence of VCF, only the combination of both a reduced bone mass for age and sex [BMC or BMD below 2 *Z*-score at the spine (L1–L4) and/or at the total body less head] and a significant history of fragility fractures (2 or more or 3 or more respectively by the age of 10 or below 19 years of age) is indicative of osteoporosis [109]. This statement is mainly supported by expert's opinion as the quality of evidence unfortunately was rated low.

A second aspect of reporting bone densitometry studies is the question whether the measurements should be corrected for length, body mass, skeletal age, or pubertal stage. This is especially the case in children with an underlying disease affecting one or more of these parameters. However, for every potential correction a potential error is also introduced. It is therefore important that the clinician who requests a bone densitometric study realizes the advantages but also the drawbacks of bone densitometry.

Finally and perhaps most important is the relation of fracture risk based on the bone densitometry findings. Although

Table 3.2 Overview of bone densitometry techniques (for comparison the natural background dose in the Netherlands is approximately 2.5 millisievert per year, whereas a transatlantic flight is approximately 0.05 millisievert) [107, 108]

Technique	Bone type	Skeletal site	Parameters	Radiation dose[a]
DXA[b]	Integral	Lumbar spine	BMC[c] (g/cm), BMD[d] (g/cm^2), BMaD[e] (g/cm^3)	0.0024–0.0040
		Hip	BMC (g/cm), BMD (g/cm^2), BMaD (g/cm^3)	0.0024–0.0054
		Total body	BMC (g/cm), BMD (g/cm^2)	0.0010–0.0034
DXR[f]	Integral	Hand	BMD (g/cm^2)	0.0001
QCT[g]	Cortical—trabecular	Spine (L1–L2)	BMC (g), vBMD (g/cm^3), CSA[h] (cm^2)	0.59–1.09
		Forearm	BMC (g), vBMD (g/cm^3)	0.01
pQCT[i]	Cortical—trabecular	Forearm/tibia	BMC (g), vBMD (g/cm^3), CSA (cm^2)	< 0.003
HR-pQCT[j]	Cortical—trabecular	Forearm/tibia	BMC (g), vBMD (g/cm^3), CSA (cm^2)	<0.005
QUS[k]	Integral	Heel, fingers, radius, tibia.	SOS[l] (m/s), BUA[m] (dB/MHz), AD-Sos[n] (m/s), BTT[o] (ms)	0
MRI	Cortical—trabecular	Central and appendicular skeleton	Trabecular parameters	0

[a] Radiation dose in millisievert
[b] Dual-energy X-ray absorptiometry
[c] Bone mineral content
[d] Bone mineral density
[e] Bone mineral apparent density
[f] Digital X-ray radiogrammetry
[g] Quantitative CT
[h] Cross-sectional area
[i] Peripheral quantitative CT
[j] High-resolution peripheral quantitative CT
[k] Quantitative ultrasonography
[l] Speed of sound
[m] Broadband ultrasound attenuation
[n] Amplitude-dependent speed of sound
[o] Bone transmission time

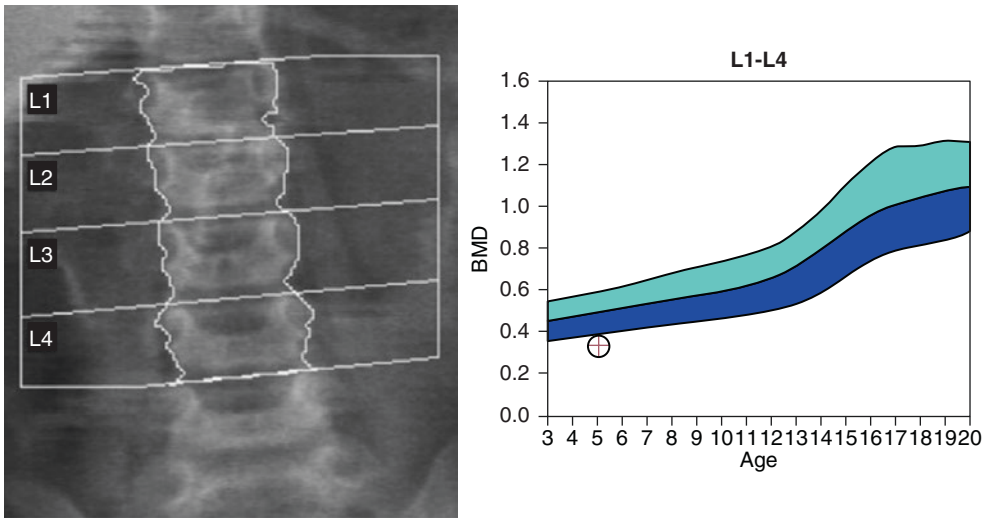


Fig. 3.19 Dual-energy X-ray absorptiometry of the lumbar spine of a five-year-old boy showing a low BMD for age

in adults a lot of strong evidence with respect to the predictive value of bone densitometry has been published, this is not the case for paediatrics [110–112]. Given the lack of consensus and limited amount of evidence, it would be unwise to use the outcome of bone densitometry studies as foundation for legal reports.

3.9 Post-Mortem Imaging

When a child dies suddenly and unexplained or in case of suspected unnatural death, the use of imaging techniques is part of the standard procedures of the post-mortem examination. The examination is of importance in the death of each minor. Although in physical child abuse skeletal lesions rarely have a life-threatening character, during autopsy they often are one of the strongest radiological indicators of physical child abuse. In all cases of sudden unexpected death in infancy (SUDI) defined as 'All cases in which there is death (or collapse leading to death) of a child, which would not have been reasonably expected to occur 24 h previously and in whom no pre-existing medical cause of death is apparent', post-mortem skeletal imaging according to the RCR guideline is mandatory. This can be complemented by a CT scan and/or MRI, even if an autopsy will be performed. The radiological examination is a very valuable addition to the autopsy and may direct the investigation [113, 114]. In the Netherlands, an evidence based national guideline for post-mortem imaging in a clinical setting of SUDI has been published [115, 116]. Although this guideline was specifically developed for a clinical setting, it can of course also be applied to a forensic medical setting. Recently, the European Society of Paediatric Radiology and the International Society of Forensic Radiology and Imaging issue a joint statement on a PMCT protocol [117] (Fig. 3.20). As mentioned earlier in this chapter, the so-called babygram (one single overview or two views) of the skeleton in young infants was shown to be inadequate when child abuse is suspected [26]. This examination should be considered obsolete, also in post-mortem evaluation. It is of interest to note that a 2009 American survey study into the use of skeletal surveys in a forensic post-mortem setting yielded rather poor results [118]. Of the respondents 29% reported the routine use of a babygram, 73% only 1–5 views, and only 5% more than 16 views. There thus seems to be a large discrepancy between the clinical setting and the post-mortem forensic setting.

During a full autopsy of a child, conspicuous fractures such as skull fractures or fractures of the long bones will generally not be overlooked. However, there is a greater risk that the more subtle skeletal anomalies may be overlooked, such as a CML, since the ends of the long bones are not routinely inspected at autopsy. There is also a reasonable chance that rib fractures (especially when located on the posterior side) will be overlooked [119].

When the sudden and unexplained death occurred in non-accidental circumstances, e.g. physical child abuse, it is not rare to find signs of earlier injuries at post-mortem radiological examination [120]. In a retrospective study of McGraw et al. of 106 consecutive post-mortem skeletal surveys, 14 children showed signs of inflicted skeletal injuries [121]. Sperry and Pfalzgraf describe a 9-month-old child whose death was initially contributed to cot death [122]. However, post-mortem examination showed healing clavicle fractures and a healing fracture of the humerus on the left. Extensive investigation revealed that 4 weeks prior to death a non-qualified chiropractor had treated the child for a 'shoulder dislocation'. It was very likely that this treatment

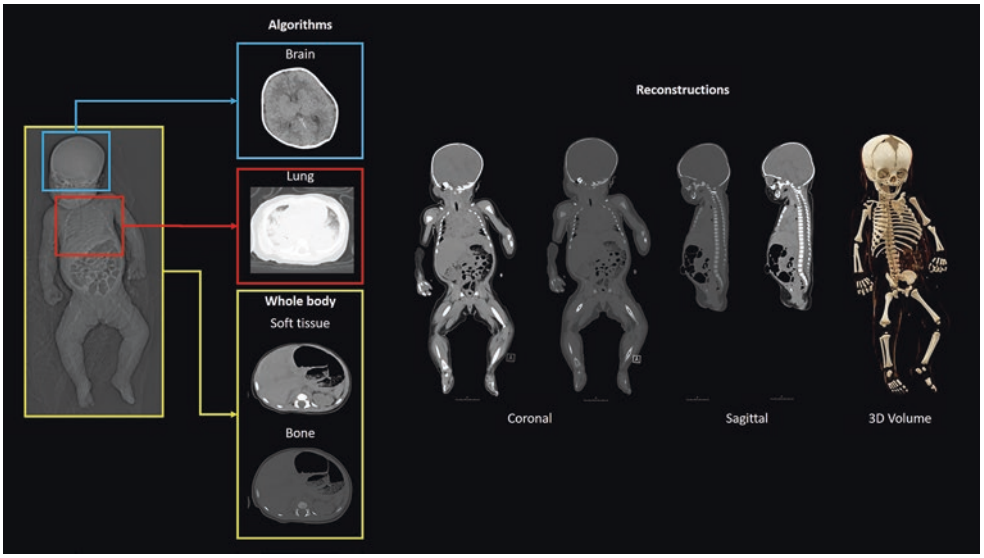


Fig. 3.20 Graphic representation of the post-mortem CT protocol as proposed by the European Society of Paediatric Radiology and the International Society of Forensic Radiology and Imaging [117]

was the cause of the fractures. Also, Ojima et al. describe the finding of fractures in a child who died suddenly and unexplained [123]. This child had undiagnosed osteogenesis imperfecta.

When it is decided that post-mortem examination with radiological techniques will take place, this should always happen prior to autopsy. It is important to recognize that imaging deceased children, due to rigor mortis or the legal inability to remove foreign objects, can present with difficulties and that the resulting quality can be lower compared to imaging a living child [124]. Preferably the images are evaluated by an experienced paediatric radiologist before the pathologist starts the autopsy. This enables the pathologist to take the radiological findings into consideration. Sometimes the pathologist will find fractures at autopsy that were not visible on the radiographs. When this is the case, the bone may be removed in its totality and be subjected to specimen radiography [125]. Specimen radiography should preferably be performed on a high-resolution system; in general a mammography system is the technique of choice (Fig. 3.21a–d).

An important development in clinical and forensic pathology is the use of post-mortem CT (Fig. 3.22a–c) and MRI (Figs. 3.23a, b and 3.24a, b) [126–128]. The use of these, clinically widely used, techniques is evident; also, for laymen it produces (when reconstructions are used) an image they can understand, and that is suitable for presentation in court cases. Furthermore, it provides calibrated three-dimensional measurements and long-term storage of images. However, post-mortem imaging also has its disadvantages. Firstly, obviously there is no blood circulation, which makes it difficult to use contrast media. A possible solution to this problem has been developed by the 'Virtopsy project' in Bern, where after perfusion with paraffin oil and with the use of a heart-lung machine it was still possible to produce an angiography [126]. A second, even more important problem is the interpretation of the CT and MRI images. Where radiologists are experienced in evaluating the images of living patients and pathologists are experienced in the performing and interpreting autopsies, there is little or no overlapping knowledge. This may lead to problems in interpretation; for example when air is seen in the portal system (Fig. 3.25). In living patients this is a rare finding, but in post-mortem CTs of critically ill patients, this is regularly found. Shiotani et al. described portal air in 33% of 190 post-mortem CTs [129].

To date most forensic PMCT studies have focused on the adult population [130–132]. Most paediatric post-mortem studies have focused on a very young population, including foetuses, making use of PMMRI which makes it difficult to incorporate their findings in a forensic setting [133].In children there only have been a few studies published on the use of PMCT in a forensic setting [134–137]. These three studies have shown somewhat conflicting results with respect to the sensitivity and specificity related to the cause of death. In a series of 18 children under the age of 2 years, in whom a cause of death was found at autopsy, Proisy et al. found that this was in accordance with PMCT in 15 cases (83%) [135]. Krentz et al. reported on a series of 26 children aged 0–12 years; in this series consisting of a mix of cases useful findings were more frequently detected by autopsy compared to PMCT (192 out of a total of 244 findings) [134]. Sieswerda et al. reported on a series of 98 children where CT and autopsy identified the same cause of death in 66/98 cases [136]. They found an important influence of the case mix with respect to the concordance between PMCT and autopsy, 59–67% con-

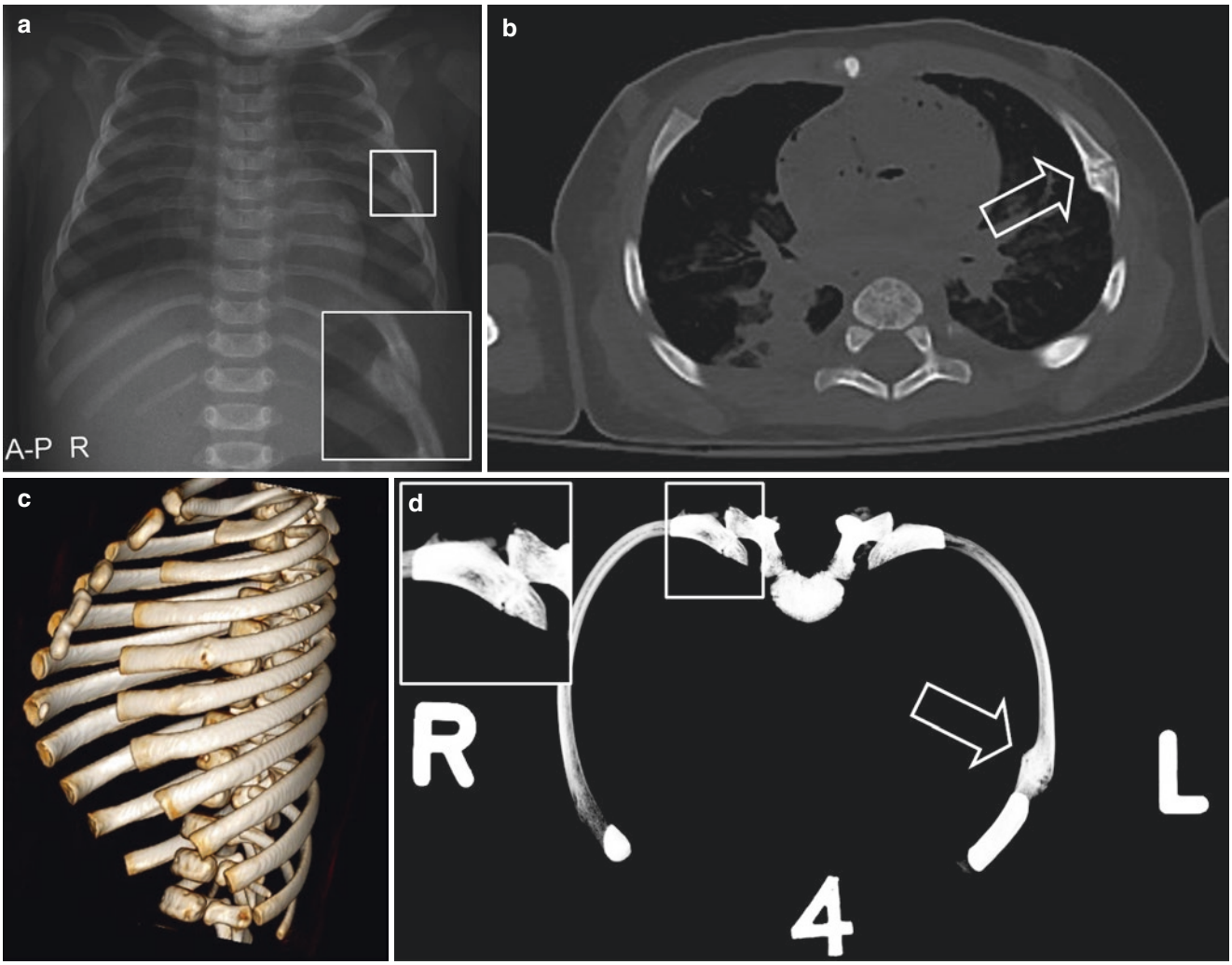


Fig. 3.21 (**a**) Chest radiographs shows multiple rib fractures with callus formation, among others on the lateral aspect of the fourth left rib (inset). (**b**) CT of the chest clearly shows the rib fracture of the fourth left rib. (**c**) 3D reconstruction of the chest in a bone window showing the rib fracture of the fourth left rib. (**d**) Specimen radiograph clearly shows the rib fracture of the fourth left rib (arrow) but also a healing fracture of the neck of the fourth right rib (inset)

cordance in unnatural deaths compared to 0% agreement to natural deaths. More recently, Shelmerdine et al. performed a retrospective study in 136 cases, 74 (54.4%) boys and 62 (45.6%) girls with a mean age of 2 years and 1 month (range: 2 days–14.7 years) [137]. In 77 cases, autopsy revealed a definitive cause of death; of these cases in 55 (71.4%) PMCT had a similar cause of death. For the whole population PMCT identified 40.4% (55/136) of the main pathologic findings. In this mixed study, containing unexplained deaths as well as forensic cases, there were depending on body area varying diagnostic accuracy rates. There were high diagnostic accuracy rates for neurologic findings (75.6%) and musculoskeletal findings (98.4%), whereas there were significantly lower rates for thoracic (64.7%), abdominal (53.8%), and cardiovascular (31.3%) findings. Overall it can be concluded that in all studies PMCT excelled in the detection of skeletal pathology and autopsy excelled in soft tissue findings. Despite the differences between these studies, it can be concluded that PMCT and autopsy should be considered to be complementary modalities.

Although post-mortem radiology is still in full development, and its values and limitations will have to be proven in the future, it seems obvious that after its successful introduction into the clinic it will now also find its place in pathology.

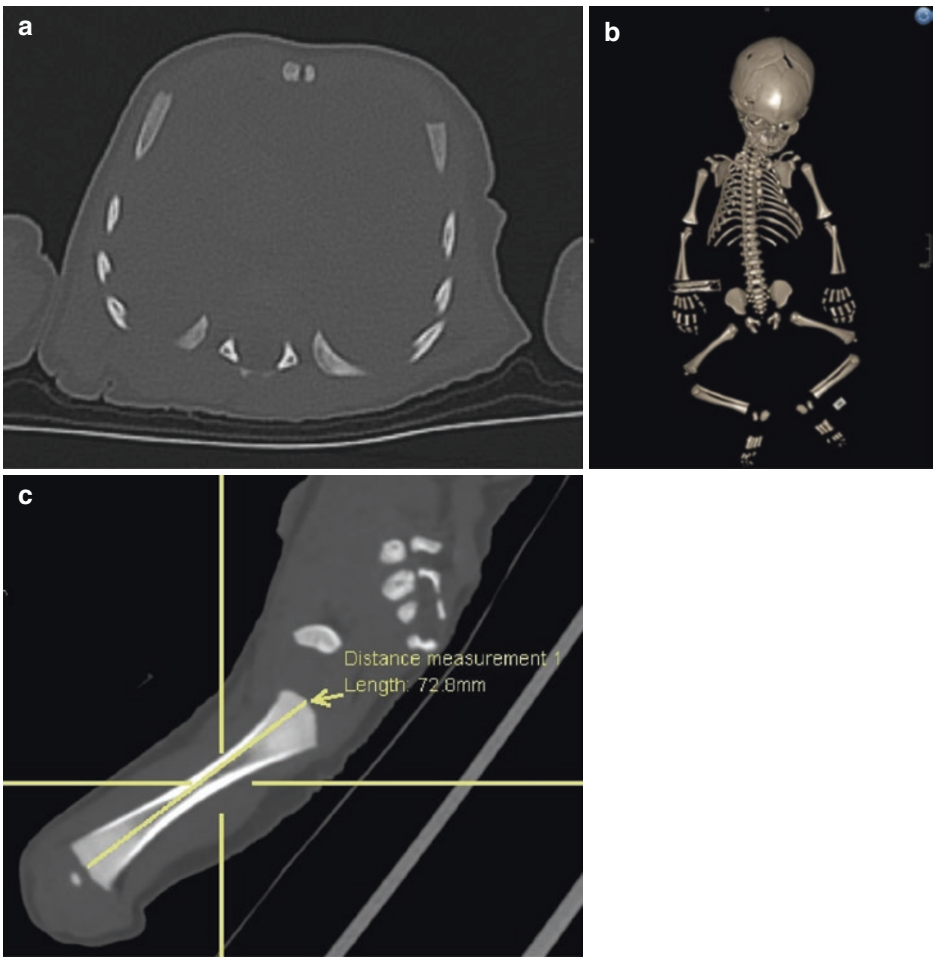


Fig. 3.22 (**a**) Found demised neonate, PMCT shows gasless lungs as a sign that the neonate died in utero. (**b**) 3D reconstruction shows a normally developed skeleton. (**c**) Based on the length assessment of the left femur the gestational age was calculated to be 37 weeks (SD 2.1 weeks) [151]

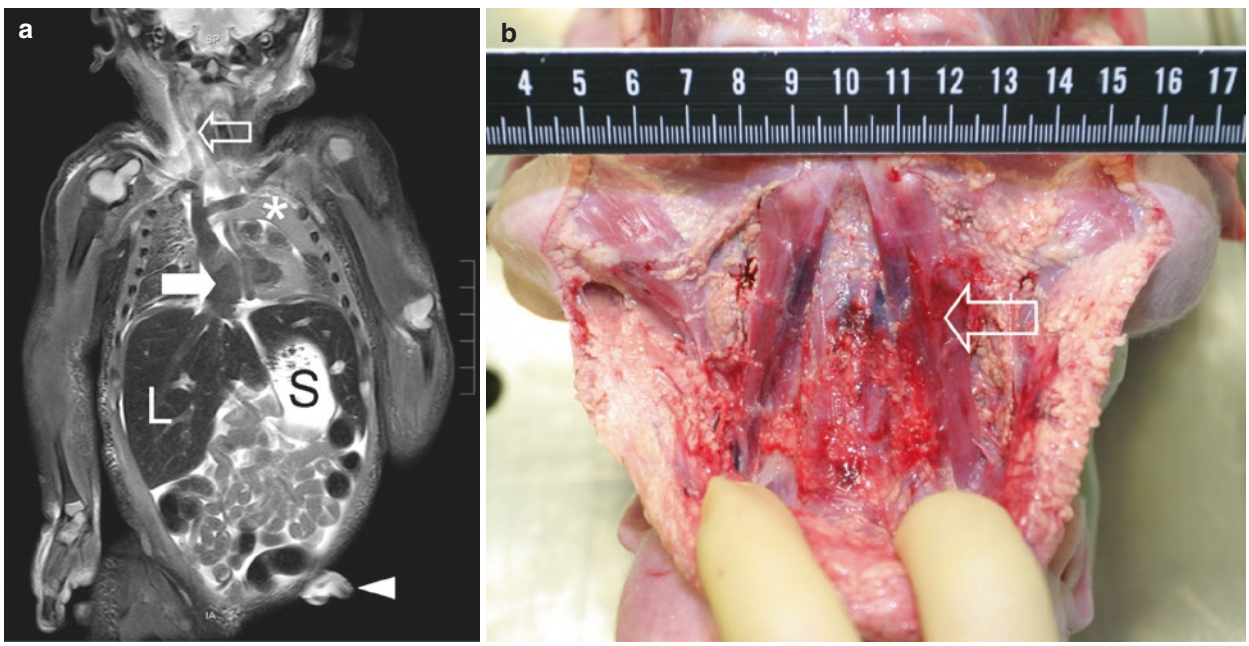


Fig. 3.23 (**a**) Deceased neonate, found in a refuse container. Post-mortem T2-weighted MRI shows oedema around the blood vessels in the neck (*open arrow*). Also, the neonatal anatomy is clearly visible. (* = thymus; L = liver; S = stomach; *arrow* = right atrium; *arrow point* = umbilical cord) (**b**) Autopsy (seen from above) shows a haematoma around the blood vessels on the right side of the neck (*open arrow*), possibly the result of strangulation

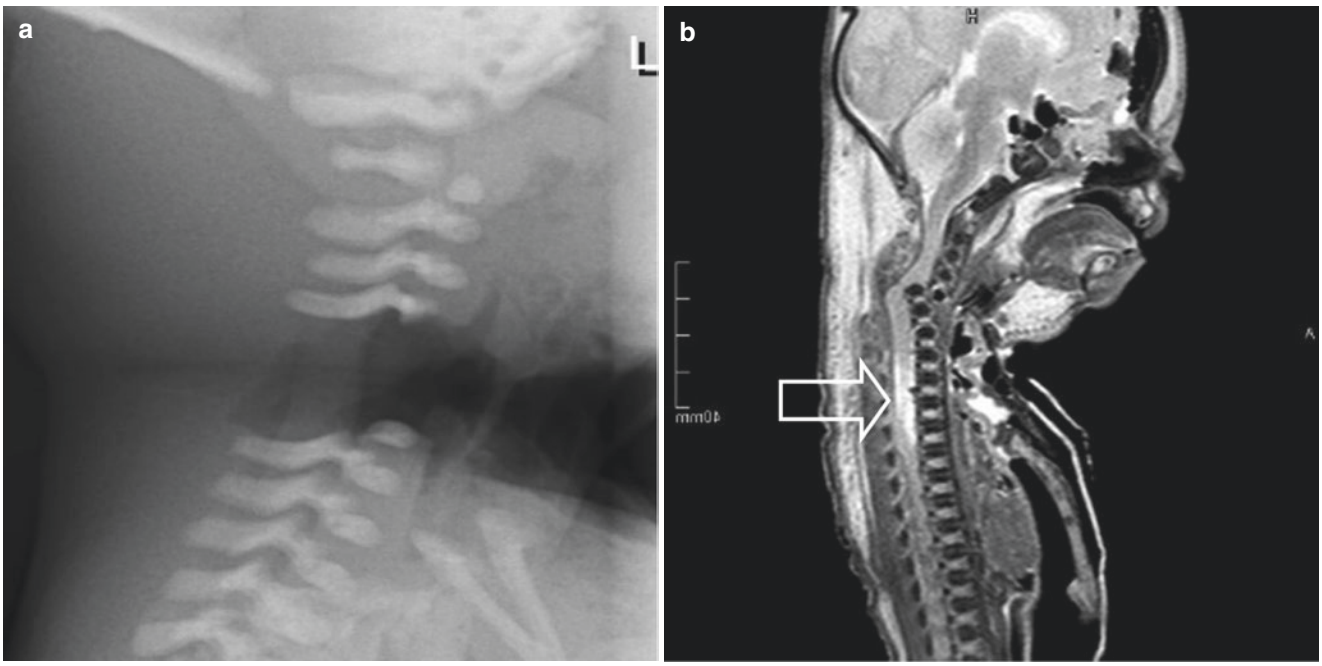


Fig. 3.24 (**a**) Fatal birth trauma C4-C5 distraction fracture. (**b**) Post-mortem MRI shows the known distraction but also a spinal cord transection (arrow)

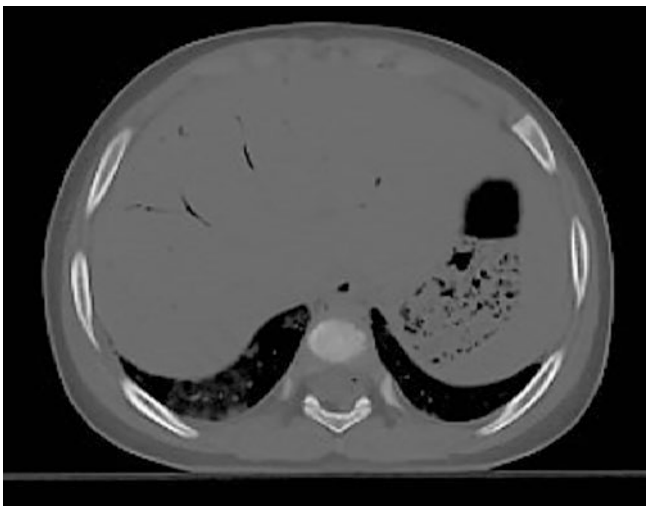


Fig. 3.25 Post-mortem CT shows portal air as a normal post-mortem finding

3.10 Radiation Dose in Imaging Suspected Child Abuse

Over the past decades, there are growing concerns about the small but potential adverse effects of ionizing radiation used in medical imaging [138–141]. There are two types of adverse effects related to the use of ionizing radiation, deterministic and stochastic. Deterministic effects are characterized by a dose-related increase in risk and associated severity of outcome. They only occur above a threshold dose and examples include radiation-induced dermatitis and cataract. Stochastic effects, on the other hand, are caused by a radiation-induced mutation or other permanent change in cells which otherwise remain viable. Examples of stochastic effects include cancer and hereditary effects. The probability of stochastic effects increases with dose without a threshold, which means that even a small X-ray dose has the potential to cause a base change in DNA, and that the severity of the outcome is not related to the dose. These small but not negligible health risks of the use of ionizing radiation is of particular concern in children as their tissues are more radiosensitive than adults and they have more years ahead in which cancerous changes might occur.

The term usually used to describe the effect of ionizing radiation is ‘effective dose’, which reflects a rough estimate of the whole body dose based on summed dose values to important critical organs and tissues within the exposed body area multiplied by ICRP103 weighting factors (wT). It allows for comparison of risks among various radiological imaging techniques and is measured in units of milliSievert (mSv). There are different ways to express radiation dose such as background equivalent radiation time (BERT), critical organ dose (COD), surface absorbed dose (SAD), dose area product (DAP), diagnostic acceptable reference level (DARLing), and effective dose (ED) [142]. In explaining effective dose to parents/caretakers the easiest is to use the background equivalent radiation time (BERT), in which the exposure is compared to the annual natural background radiation exposure such as natural radioactive substances in the air, soil, and environment to which the population is exposed

on a daily basis. Depending on where you live the annual background radiation exposure differs slightly, but on average this is 2.4 mSv per year [143]. There are numerous sources that expose us to radiation, in Table 3.3 some of these sources and the radiological exams used in evaluating child abuse cases are presented, Fig. 3.26 shows a break-down of the sources of natural back. The U.S. Nuclear Regulatory Commission (NRC) has an online calculator, focused on America, to calculate your personal annual radiation exposure, this might be useful in educating parents about the perceived risk of radiation [144].

Table 3.3 Overview of natural background radiation sources compared to a skeletal survey

Source	Radiation dose (mSv)	Weeks of natural background radiation
Coast-to-coast US flight [148]	0.035	0.75
Transatlantic flight [149]	0.08	1.7
Follow-up skeletal survey [141]	0.1	2.2
Skeletal survey [8, 139, 141]	0.06–0.2	1.3–4.3
Annual food intake [150]	0.3	6.5
Head CT [145]	1.6	34.7
Bone scintigraphy [141]	2.27	49.2
One year annual background radiation [143]	2.4	

Berger et al. investigated the effective dose of the skeletal survey based on the recommendations of the American Academy of Pediatrics and consisting of 15 different radiographs [139]. The radiographic examinations were acquired using an X-ray system with a digital flat panel detector and manually set technique factors optimized to provide a high-quality diagnostic image at the lowest possible radiation dose. Based on Monte Carlo simulations, the total effective dose of the 15 radiographs was estimated to be 0.2 mSv for both female and male infants. Similar results were found in a recent study published in 2019 by Rao et al. [141]. They investigated the effective dose of a skeletal survey compliant with the national guideline and based on the most recent

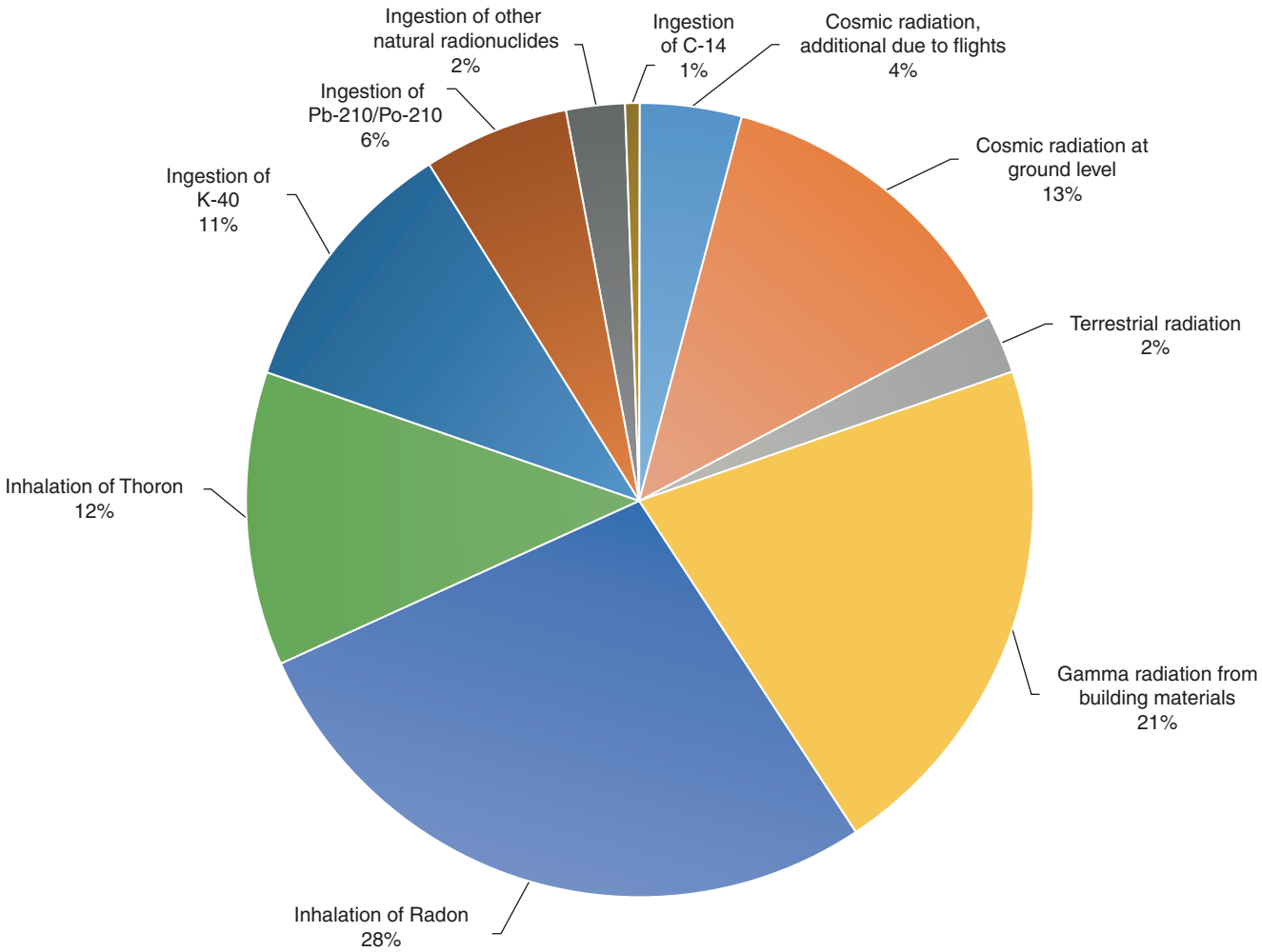


Fig. 3.26 Break-down of the sources of natural background radiation [152]. The first four causes are external radiation, the following two are caused by inhalation, and the last four are the result of ingestion of radio isotopes

ACR and RCR/SCoR guidelines. This skeletal survey included at least 14 different radiographs (19 in the absence of a paediatric radiologist), whereas the follow-up survey included fewer radiographs. The mean effective dose was 0.20 mSv (95% CI 0.18–0.22) for the initial skeletal survey and 0.10 mSv (95% CI 0.08–0.11) for the follow-up survey. They also looked at the mean effective dose of CT head, which delivered a mean effective dose of 2.49 mSv (95% CI 2.37–2.60). In a minority of patients in their study cohort a bone scintigraphy was performed, with a mean effective dose of 2.27 mSv (95% CI 2.11–2.43). Finally, in a recent newborn phantom study by Hampel et al. effective doses of approximately 0.06–0.09 mSv were found for a skeletal survey according to the RCR/SCoR guidelines [8]. Therefore, from these studies it can be concluded that (optimized) skeletal surveys deliver a relatively low effective dose of ionizing radiation, and that the benefits of early detection of physical abuse certainly outweigh the potential risks of the use of ionizing radiation. This seems to be true for the use of CT head (estimated dose 1.6 mSv [145]) in case of suspected abusive head trauma, although for non-acute head injury presentations MRI is preferred to keep the radiation dose to the child as low as reasonably achievable. In a large meta-analysis published in 2020, the authors concluded 'no evidence of an increased risk of all cancers was observed after X-ray exposure' [146]. For CT this isn't so clear cut, based on pooled results from studies on CT exposure during childhood, the life risk for leukaemia and brain tumours seems higher. However, these published studies all suffer from different levels of methodological limitations and given the fact that long-term data is needed the scans were made with significantly higher radiation exposures as are used in modern day scanners. For the true impact of the life risk of cancer after diagnostic paediatric CT, we will have to wait for the results of the ongoing European EPI-CT study [147].

References

1. Kempe CH, Silverman FN, Steele BF, Droegenmueller W, Silver HK (1962) The battered-child syndrome. JAMA 181:17–24
2. Caffey J (1946) Multiple fractures in the long bones of infants suffering from chronic subdural hematoma. Am J Roentgenol Radium Therapy, Nucl Med 56:163–173
3. Silverman FN (1953) The roentgen manifestations of unrecognized skeletal trauma in infants. Am J Roentgenol Radium Therapy Nucl Med 69:413–427
4. Woolley PV Jr, Evans WA Jr (1955) Significance of skeletal lesions in infants resembling those of traumatic origin. J Am Med Assoc 158:539–543
5. Caffey J (1957) Some traumatic lesions in growing bones other than fractures and dislocations: clinical and radiological features: the Mackenzie Davidson memorial lecture. Br J Radiol 30:225–238
6. Ellerstein NS, Norris KJ (1984) Value of radiologic skeletal survey in assessment of abused children. Pediatrics 74:1075–1078
7. Wootton-Gorges SL, Soares BP, Alazraki AL, Anupindi SA, Blount JP, Booth TN, Dempsey ME, Falcone RA Jr, Hayes LL, Kulkarni AV, Partap S, Rigsby CK, Ryan ME, Safdar NM, Trout AT, Widmann RF, Karmazyn BK, Palasis S (2017) ACR appropriateness criteria - suspected physical abuse-child. J Am Coll Radiol 14:S338–s349
8. The Royal College of Radiologists (RCR) and the Society and College of Radiographers (SCoR) (2017) The radiological investigation of suspected physical abuse in children. https://www.rcr.ac.uk/publication/radiological-investigation-suspected-physical-abuse-children. Accessed 5 July 2021
9. Ingram JD, Connell J, Hay TC, Strain JD, Mackenzie T (2000) Oblique radiographs of the chest in nonaccidental trauma. Emerg Radiol 7:42–46
10. Hansen KK, Prince JS, Nixon GW (2008) Oblique chest views as a routine part of skeletal surveys performed for possible physical abuse–is this practice worthwhile? Child Abuse Negl 32:155–159
11. Marine MB, Corea D, Steenburg SD, Wanner M, Eckert GJ, Jennings SG, Karmazyn B (2014) Is the new ACR-SPR practice guideline for addition of oblique views of the ribs to the skeletal survey for child abuse justified? AJR Am J Roentgenol 202:868–871
12. Kleinman PL, Kleinman PK, Savageau JA (2004) Suspected infant abuse: radiographic skeletal survey practices in pediatric health care facilities. Radiology 233:477–485
13. Offiah AC, Hall CM (2003) Observational study of skeletal surveys in suspected non-accidental injury. Clin Radiol 58:702–705
14. van Rijn RR, Kieviet N, Hoekstra R, Nijs HGT, Bilo RAC (2009) Radiology in suspected non accidental injury: theory and practice in the Netherlands. Eur J Radiol 71:147–151
15. Loos MHJ, Almekinders CAM, Heymans MW, de Vries A, Bakx R (2020) Incidence and characteristics of non-accidental burns in children: a systematic review. Burns 46(6):1243
16. Hicks RA, Stolfi A (2007) Skeletal surveys in children with burns caused by child abuse. Pediatr Emerg Care 23:308–313
17. Fagen KE, Shalaby-Rana E, Jackson AM (2015) Frequency of skeletal injuries in children with inflicted burns. Pediatr Radiol 45:396–401
18. Degraw M, Hicks RA, Lindberg D (2010) Incidence of fractures among children with burns with concern regarding abuse. Pediatrics 125:e295–e299
19. Howell S, Bailey L, Coffman J (2019) Evaluation of drug-endangered children: the yield of toxicology and skeletal survey screening. Child Abuse Negl 96:104081
20. Wells K (2009) Substance abuse and child maltreatment. Pediatr Clin N Am 56:345–362
21. Kelleher K, Chaffin M, Hollenberg J, Fischer E (1994) Alcohol and drug disorders among physically abusive and neglectful parents in a community-based sample. Am J Public Health 84:1586–1590
22. Campbell KA, Bogen DL, Berger RP (2006) The other children: a survey of child abuse physicians on the medical evaluation of children living with a physically abused child. Arch Pediatr Adolesc Med 160:1241–1246
23. Hamilton-Giachritsis CE, Browne KD (2005) A retrospective study of risk to siblings in abusing families. J Fam Psychol 19:619–624
24. Lindberg DM, Shapiro RA, Laskey AL, Pallin DJ, Blood EA, Berger RP (2012) Prevalence of abusive injuries in siblings and household contacts of physically abused children. Pediatrics 130:193–201
25. McNamara CR, Panigrahy A, Sheetz M, Berger RP (2021) The likelihood of an occult fracture in skeletal surveys obtained in

children more than 2 years old with concerns of physical abuse. Pediatr Emerg Care 38(2):e488–e492
26. Alexander R, Kleinman PK (2000) Diagnostic imaging of child abuse – portable guides to investigating child abuse. US Department of Justice, Washington
27. Hulson OS, van Rijn RR, Offiah AC (2014) European survey of imaging in non-accidental injury demonstrates a need for a consensus protocol. Pediatr Radiol 44:1557–1563
28. Swinson S, Tapp M, Brindley R, Chapman S, Offiah A, Johnson K (2008) An audit of skeletal surveys for suspected non-accidental injury following publication of the British Society of Paediatric Radiology guidelines. Clin Radiol 63:651–656
29. Patel H, Swinson S, Johnson K (2017) Improving national standards of child protection skeletal surveys: the value of college guidance. Clin Radiol 72:202–206
30. Wanner MR, Marine MB, Hibbard RA, Ouyang F, Jennings SG, Shea L, Karmazyn B (2019) Compliance with skeletal surveys for child abuse in general hospitals: a Statewide quality improvement process. AJR Am J Roentgenol 2019:1–6
31. 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
32. Prosser I, Maguire S, Harrison SK, Mann M, Sibert JR, Kemp AM (2005) How old is this fracture? Radiologic dating of fractures in children: a systematic review. AJR Am J Roentgenol 184:1282–1286
33. Zimmerman S, Makoroff K, Care M, Thomas A, Shapiro R (2005) Utility of follow-up skeletal surveys in suspected child physical abuse evaluations. Child Abuse Negl 29:1075–1083
34. Harper NS, Eddleman S, Lindberg DM (2013) The utility of follow-up skeletal surveys in child abuse. Pediatrics 131:e672–e678
35. Drubach LA, Sapp MV, Laffin S, Kleinman PK (2008) Fluorine-18 NaF PET imaging of child abuse. Pediatr Radiol 38:776–779
36. Blangis F, Poullaouec C, Launay E, Vabres N, Sadones F, Eugène T, Cohen JF, Chalumeau M, Gras-Le Guen C (2020) Bone scintigraphy after a negative radiological skeletal survey improves the detection rate of inflicted skeletal injury in children. Front Pediatr 8:498
37. Haase GM, Ortiz VN, Sfanakis GN (1980) The value of radionuclide bone scanning in the early recognition of deliberate child abuse. J Trauma 20:873–875
38. Sty JR, Starshak RJ (1983) The role of bone scintigraphy in the evaluation of suspected child abuse. Radiology 146:369–375
39. Jaudes PK (1984) Comparison of radiography and radionuclide bone scanning in the detection of child abuse. Pediatrics 73:166–168
40. 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
41. Bainbridge JK, Huey BM, Harrison SK (2015) Should bone scintigraphy be used as a routine adjunct to skeletal survey in the imaging of non-accidental injury? A 10 year review of reports in a single centre. Clin Radiol 70:e83–e89
42. Barlucea A, Silva F, Laguna R, Montalvan C (2010) Child abuse patterns: retrospective evaluation of clinical, radiographic and scintigraphic data in a Hispanic population. J Nucl Med 51:1655
43. 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 61:723–736
44. Blangis F, Taylor M, Adamsbaum C, Devillers A, Gras-Le Guen C, Launay E, Bossuyt PM, Cohen JF, Chalumeau M (2020) Add-on bone scintigraphy after negative radiological skeletal survey for the diagnosis of skeletal injury in children suspected of physical abuse: a systematic review and meta-analysis. Arch Dis Child 106(4):361–366
45. Scaglione M, Pinto A, Pedrosa I, Sparano A, Romano L (2008) Multi-detector row computed tomography and blunt chest trauma. Eur J Radiol 65:377–388
46. Sangster GP, Gonzalez-Beicos A, Carbo AI, Heldmann MG, Ibrahim H, Carrascosa P, Nazar M, D'Agostino HB (2007) Blunt traumatic injuries of the lung parenchyma, pleura, thoracic wall, and intrathoracic airways: multidetector computer tomography imaging findings. Emerg Radiol 14:297–310
47. Provenzale J (2007) CT and MR imaging of acute cranial trauma. Emerg Radiol 14:1–12
48. Geijer M, El-Khoury GY (2006) MDCT in the evaluation of skeletal trauma: principles, protocols, and clinical applications. Emerg Radiol 13:7–18
49. Ringl H, Lazar M, Topker M, Woitek R, Prosch H, Asenbaum U, Balassy C, Toth D, Weber M, Hajdu S, Soza G, Wimmer A, Mang T (2015) The ribs unfolded – a CT visualization algorithm for fast detection of rib fractures: effect on sensitivity and specificity in trauma patients. Eur Radiol 25:1865–1874
50. Sanchez TR, Grasparil AD, Chaudhari R, Coulter KP, Wootton-Gorges SL (2018) Characteristics of rib fractures in child abuse-the role of low-dose chest computed tomography. Pediatr Emerg Care 34:81–83
51. Sanchez TR, Lee JS, Coulter KP, Seibert JA, Stein-Wexler R (2015) CT of the chest in suspected child abuse using submillisievert radiation dose. Pediatr Radiol 45:1072–1076
52. Shelmerdine SC, Langan D, Hutchinson JC, Hickson M, Pawley K, Suich J, Palm L, Sebire NJ, Wade A, Arthurs OJ (2018) Chest radiographs versus CT for the detection of rib fractures in children (DRIFT): a diagnostic accuracy observational study. Lancet Child Adolesc Health 2:802–811
53. Stoodley N (2006) Controversies in non-accidental head injury in infants. Br J Radiol 79:550–553
54. Culotta PA, Crowe JE, Tran QA, Jones JY, Mehollin-Ray AR, Tran HB, Donaruma-Kwoh M, Dodge CT, Camp EA, Cruz AT (2017) Performance of computed tomography of the head to evaluate for skull fractures in infants with suspected non-accidental trauma. Pediatr Radiol 47:74–81
55. Sharp SR, Patel SM, Brown RE, Landes C (2018) Head imaging in suspected non accidental injury in the paediatric population. In the advent of volumetric CT imaging, has the skull X-ray become redundant? Clin Radiol 73:449–453
56. Martin A, Paddock M, Johns CS, Smith J, Raghavan A, Connolly DJA, Offiah AC (2020) Avoiding skull radiographs in infants with suspected inflicted injury who also undergo head CT: "a no-brainer?". Eur Radiol 30:1480–1487
57. Pennell C, Aundhia M, Malik A, Poletto E, Grewal H, Atkinson N (2021) Utility of skull radiographs in infants undergoing 3D head CT during evaluation for physical abuse. J Pediatr Surg 56(6):1180
58. Orman G, Wagner MW, Seeburg D, Zamora CA, Oshmyansky A, Tekes A, Poretti A, Jallo GI, Huisman TA, Bosemani T (2015) Pediatric skull fracture diagnosis: should 3D CT reconstructions be added as routine imaging? J Neurosurg Pediatr 16:426–431
59. Deyle S, Brehmer T, Evangelopoulos DS, Krause F, Benneker LM, Zimmermann H, Exadaktylos AK (2010) Review of Lodox Statscan in the detection of peripheral skeletal fractures in multiple injury patients. Injury 41:818–822
60. Deyle S, Wagner A, Benneker LM, Jeger V, Eggli S, Bonel HM, Zimmermann H, Exadaktylos AK (2009) Could full-body digital X-ray (LODOX-Statscan) screening in trauma challenge conventional radiography? J Trauma 66:418–422
61. Evangelopoulos DS, von Tobel M, Cholewa D, Wolf R, Exadaktylos AK, Zachariou Z (2010) Impact of Lodox Statscan

on radiation dose and screening time in paediatric trauma patients. Eur J Pediatr Surg 20:382–386
62. Flach PM, Ross SG, Ampanozi G, Ebert L, Germerott T, Hatch GM, Thali MJ, Patak MA (2012) "Drug mules" as a radiological challenge: sensitivity and specificity in identifying internal cocaine in body packers, body pushers and body stuffers by computed tomography, plain radiography and Lodox. Eur J Radiol 81:2518–2526
63. Fu CY, Wang YC, Hsieh CH, Chen RJ (2011) Lodox/Statscan provides benefits in evaluation of gunshot injuries. Am J Emerg Med 29:823–827
64. Fu CY, Wu SC, Chen RJ (2008) Lodox/Statscan provides rapid identification of bullets in multiple gunshot wounds. Am J Emerg Med 26:965.e965–965.e967
65. Huang HC, Fu CY, Hsieh CH, Wang YC, Wu SC, Chen RJ, Huang JC (2012) Lodox/Statscan facilitates the early detection of commonly overlooked extracranial injuries in patients with traumatic brain injury. Eur J Trauma Emerg Surg 38:319–326
66. Knobel GJ, Flash G, Bowie GF (2006) Lodox Statscan proves to be invaluable in forensic medicine. S Afr Med J 96:593–594, 596
67. Pitcher RD, van As AB, Sanders V, Douglas TS, Wieselthaler N, Vlok A, Paverd S, Kilborn T, Rode H, Potgieter H, Beningfield SJ (2008) A pilot study evaluating the "STATSCAN" digital X-ray machine in paediatric polytrauma. Emerg Radiol 15:35–42
68. Pitcher RD, Wilde JC, Douglas TS, van As AB (2009) The use of the Statscan digital X-ray unit in paediatric polytrauma. Pediatr Radiol 39:433–437
69. Whiley SP, Alves H, Grace S (2013) Full-body X-ray imaging to facilitate triage: a potential aid in high-volume emergency departments. Emerg Med Int 2013:437078
70. Whiley SP, Mantokoudis G, Ott D, Zimmerman H, Exadaktylos AK (2012) A review of full-body radiography in nontraumatic emergency medicine. Emerg Med Int 2012:108129
71. Yang L, Ye LG, Ding JB, Zheng ZJ, Zhang M (2016) Use of a full-body digital X-ray imaging system in acute medical emergencies: a systematic review. Emerg Med J 33:144–151
72. Beningfield S, Potgieter H, Nicol A, van As S, Bowie G, Hering E, Latti E (2003) Report on a new type of trauma full-body digital X-ray machine. Emerg Radiol 10:23–29
73. Douglas TS, Sanders V, Pitcher R, van As AB (2008) Early detection of fractures with low-dose digital X-ray images in a pediatric trauma unit. J Trauma 65:E4–E7
74. Solomons I (2015) Scanners to combat diamond theft at Debswana mines. Mining Weekly. https://www.miningweekly.com/article/hi-tech-scanners-to-expose-diamond-theft-2015-01-23
75. Mantokoudis G, Hegner S, Dubach P, Bonel HM, Senn P, Caversaccio MD, Exadaktylos AK (2013) How reliable and safe is full-body low-dose radiography (LODOX Statscan) in detecting foreign bodies ingested by adults? Emerg Med J 30:559–564
76. du Plessis M, Date-Chong M, Liebenberg L (2020) Lodox®: the invaluable radiographic solution in the forensic setting. Int J Legal Med 134:655–662
77. Spies AJ, Steyn M, Bussy E, Brits D (2020) Forensic imaging: the sensitivities of various imaging modalities in detecting skeletal trauma in simulated cases of child abuse using a pig model. J Forensic Legal Med 76:102034
78. Hargaden G, O'Connell M, Kavanagh E, Powell T, Ward R, Eustace S (2003) Current concepts in wholebody imaging using turbo short tau inversion recovery MR imaging. AJR Am J Roentgenol 180:247–252
79. Kellenberger CJ, Epelman M, Miller SF, Babyn PS (2004) Fast STIR whole-body MR imaging in children. Radiographics 24:1317–1330
80. Kumar J, Seith A, Kumar A, Sharma R, Bakhshi S, Kumar R, Agarwala S (2008) Whole-body MR imaging with the use of parallel imaging for detection of skeletal metastases in pediatric patients with small-cell neoplasms: comparison with skeletal scintigraphy and FDG PET/CT. Pediatr Radiol 38:953–962
81. Schooler GR, Davis JT, Daldrup-Link HE, Frush DP (2018) Current utilization and procedural practices in pediatric whole-body MRI. Pediatr Radiol 48:1101–1107
82. Merlini L, Carpentier M, Ferrey S, Anooshiravani M, Poletti PA, Hanquinet S (2017) Whole-body MRI in children: would a 3D STIR sequence alone be sufficient for investigating common paediatric conditions? A comparative study. Eur J Radiol 88:155–162
83. Albano D, Patti C, Lagalla R, Midiri M, Galia M (2017) Whole-body MRI, FDG-PET/CT, and bone marrow biopsy, for the assessment of bone marrow involvement in patients with newly diagnosed lymphoma. J Magn Reson Imaging 45:1082–1089
84. Arnoldi AP, Schlett CL, Douis H, Geyer LL, Voit AM, Bleisteiner F, Jansson AF, Weckbach S (2017) Whole-body MRI in patients with non-bacterial osteitis: radiological findings and correlation with clinical data. Eur Radiol 27:2391–2399
85. Leclair N, Thormer G, Sorge I, Ritter L, Schuster V, Hirsch FW (2016) Whole-body diffusion-weighted imaging in chronic recurrent multifocal osteomyelitis in children. PLoS One 11:e0147523
86. Smets AM, Deurloo EE, Slager TJE, Stoker J, Bipat S (2018) Whole-body magnetic resonance imaging for detection of skeletal metastases in children and young people with primary solid tumors – systematic review. Pediatr Radiol 48:241–252
87. Elterman T, Beer M, Girschick HJ (2007) Magnetic resonance imaging in child abuse. J Child Neurol 22:170–175
88. Stranzinger E, Kellenberger CJ, Braunschweig S, Hopper R, Huisman TAGM (2007) Whole-body STIR MR imaging in suspected child abuse: an alternative to skeletal survey radiography? Eur J Radiol Extra 63:43–47
89. Evangelista P, Barron C, Goldberg A, Jenny C, Tung G (2006) MRI STIR for the evaluation of nonaccidental trauma in children
90. Proisy M, Vivier PH, Morel B, Bruneau B, Sembely-Taveau C, Vacheresse S, Devillers A, Lecloirec J, Bodet-Milin C, Dubois M, Hamonic S, Bajeux E, Ganivet A, Adamsbaum C, Treguier C (2021) Whole-body MR imaging in suspected physical child abuse: comparison with skeletal survey and bone scintigraphy findings from the PEDIMA prospective multicentre study. Eur Radiol 31(11):8069
91. Eley KA, McIntyre AG, Watt-Smith SR, Golding SJ (2012) "Black bone" MRI: a partial flip angle technique for radiation reduction in craniofacial imaging. Br J Radiol 85:272–278
92. Eley KA, Watt-Smith SR, Golding SJ (2012) "Black bone" MRI: a potential alternative to CT when imaging the head and neck: report of eight clinical cases and review of the Oxford experience. Br J Radiol 85:1457–1464
93. Low XZ, Lim MC, Nga V, Sundar G, Tan AP (2021) Clinical application of "black bone" imaging in paediatric craniofacial disorders. Br J Radiol 94:20200061
94. Dremmen MHG, Wagner MW, Bosemani T, Tekes A, Agostino D, Day E, Soares BP, Huisman T (2017) Does the addition of a "black bone" sequence to a fast multisequence trauma MR protocol allow MRI to replace CT after traumatic brain injury in children? AJNR Am J Neuroradiol 38:2187–2192
95. Kralik SF, Supakul N, Wu IC, Delso G, Radhakrishnan R, Ho CY, Eley KA (2019) Black bone MRI with 3D reconstruction for the detection of skull fractures in children with suspected abusive head trauma. Neuroradiology 61:81–87
96. Marine MB, Hibbard RA, Jennings SG, Karmazyn B (2019) Ultrasound findings in classic metaphyseal lesions: emphasis on the metaphyseal bone collar and zone of provisional calcification. Pediatr Radiol 49:913–921
97. Karmazyn B, Marine MB, Wanner MR, Delaney LR, Cooper ML, Shold AJ, Jennings SG, Hibbard RA (2020) Accuracy of ultra-

sound in the diagnosis of classic metaphyseal lesions using radiographs as the gold standard. Pediatr Radiol 50:1123
98. Markowitz RI, Hubbard AM, Harty MP, Bellah RD, Kessler A, Meyer JS (1993) Sonography of the knee in normal and abused infants. Pediatr Radiol 23:264–267
99. Kelloff J, Hulett R, Spivey M (2009) Acute rib fracture diagnosis in an infant by US: a matter of child protection. Pediatr Radiol 39:70–72
100. Smeets AJ, Robben SG, Meradji M (1990) Sonographically detected costo-chondral dislocation in an abused child. A new sonographic sign to the radiological spectrum of child abuse. Pediatr Radiol 20:566–567
101. Lachmann E, Whelan M (1936) The roentgen diagnosis of osteoporosis and its limitations. Radiology 26:165–177
102. Rosendahl K, Lundestad A, Bjørlykke JA, Lein RK, Angenete O, Augdal TA, Müller LO, Jaramillo D (2020) Revisiting the radiographic assessment of osteoporosis-osteopenia in children 0–2 years of age. A systematic review. PLoS One 15:e0241635
103. van Rijn RR, Van Kuijk C (2009) Of small bones and big mistakes; bone densitometry in children revisited. Eur J Radiol 71:432–439
104. Kalkwarf HJ, Zemel BS, Yolton K, Heubi JE (2013) Bone mineral content and density of the lumbar spine of infants and toddlers: influence of age, sex, race, growth, and human milk feeding. J Bone Miner Res 28:206–212
105. Manousaki D, Rauch F, Chabot G, Dubois J, Fiscaletti M, Alos N (2016) Pediatric data for dual X-ray absorptiometric measures of normal lumbar bone mineral density in children under 5 years of age using the lunar prodigy densitometer. J Musculoskelet Neuronal Interact 16:247–255
106. Thodberg HH, van Rijn RR, Tanaka T, Martin DD, Kreiborg S (2010) A paediatric bone index derived by automated radiogrammetry. Osteoporos Int 21:1391–1400
107. Di Iorgi N, Maruca K, Patti G, Mora S (2018) Update on bone density measurements and their interpretation in children and adolescents. Best Pract Res Clin Endocrinol Metab 32:477–498
108. Adams JE (2016) Bone densitometry in children. Semin Musculoskelet Radiol 20:254–268
109. Gordon CM, Leonard MB, Zemel BS (2014) 2013 Pediatric position development conference: executive summary and reflections. J Clin Densitom 17:219–224
110. Martins A, Monjardino T, Nogueira L, Canhao H, Lucas R (2017) Do bone mineral content and density determine fracture in children? A possible threshold for physical activity. Pediatr Res 82:396–404
111. Wasserman H, Gordon CM (2017) Bone mineralization and fracture risk assessment in the pediatric population. J Clin Densitom 20:389–396
112. Clark EM, Ness AR, Bishop NJ, Tobias JH (2006) Association between bone mass and fractures in children: a prospective cohort study. J Bone Miner Res 21:1489–1495
113. Cohen MC, Whitby E (2007) The use of magnetic resonance in the hospital and coronial pediatric postmortem examination. Forensic Sci Med Pathol 3:289–296
114. Dedouit F, Guilbeau-Frugier C, Capuani C, Sévely A, Joffre F, Rougé D, Rousseau H, Telmon N (2008) Child abuse: practical application of autopsy, radiological, and microscopic studies. J Forensic Sci 53:1424–1429
115. Sonnemans LJP, Vester MEM, Kolsteren EEM, Erwich J, Nikkels PGJ, Kint PAM, van Rijn RR, Klein WM (2018) Dutch guideline for clinical foetal-neonatal and paediatric post-mortem radiology, including a review of literature. Eur J Pediatr 177:791–803
116. Klein WM, Duijst WLJM, Erwich JJHM, Hofman PAM, Kint PAM, Kroll JJF, Nikkels PGJ, Renken NS, Van Rijn RR, Rosier Y, Scheeren CIE, Stomp SJ, van der Valk P (2018) Klinische postmortem radiologie. https://richtlijnendatabase.nl/richtlijn/klinische_postmortem_radiologie
117. Shelmerdine SC, Gerrard CY, Rao P, Lynch M, Kroll JJF, Martin D, Miller E, Filograna L, Martinez RM, Ukpo O, Daly B, Hyodoh H, Johnson K, Watt A, Taranath A, Brown SD, Perry DH, Thorup Boel LW, Borowska-Solonynko A, van Rijn RR, Klein WM, Whitby A, Arthurs OJ (2019) Joint European Society of Paediatric Radiology (ESPR) and International Society for Forensic Radiology and Imaging (ISFRI) guidelines: paediatric post-mortem computed tomography (CT) imaging protocol. Pediatr Radiol 49:694–701
118. Laskey AL, Haberkorn KL, Applegate KE, Catellier MJ (2009) Postmortem skeletal survey practice in pediatric forensic autopsies: a national survey. J Forensic Sci 54:189–191
119. Norman MG, Smialek JE, Newman DE, Horembala EJ (1984) The postmortem examination on the abused child. Pathological, radiographic, and legal aspects. Perspect Pediatr Pathol 8:313–343
120. Kleinman PK, Marks SC Jr, Richmond JM, Blackbourne BD (1995) Inflicted skeletal injury: a postmortem radiologic-histopathologic study in 31 infants. AJR AmJ Roentgenol 165:647–650
121. McGraw EP, Pless JE, Pennington DJ, White SJ (2002) Postmortem radiography after unexpected death in neonates, infants, and children: should imaging be routine? AJR Am J Roentgenol 178:1517–1521
122. Sperry K, Pfalzgraf R (1990) Inadvertent clavicular fractures caused by "chiropractic" manipulations in an infant: an unusual form of pseudoabuse. J Forensic Sci 35:1211–1216
123. Ojima K, Matsumoto H, Hayase T, Fukui Y (1994) An autopsy case of osteogenesis imperfecta initially suspected as child abuse. Forensic Sci Int 65:97–104
124. Hughes-Roberts Y, Arthurs OJ, Moss H, Set PA (2012) Postmortem skeletal surveys in suspected non-accidental injury. Clin Radiol 67:868–876
125. Lorand MA, Fitzpatrick JJ, Soter DK (1996) Radiographic atlas of child abuse: a case studies approach. Igaku-Shoin Medical Pub, New York
126. Grabherr S, Gygax E, Sollberger B, Ross S, Oesterhelweg L, Bolliger S, Christe A, Djonov V, Thali MJ, Dirnhofer R (2008) Two-step postmortem angiography with a modified heart-lung machine: preliminary results. AJR Am J Roentgenol 190:345–351
127. Stawicki SP, Gracias VH, Schrag SP, Martin ND, Dean AJ, Hoey BA (2008) The dead continue to teach the living: examining the role of computed tomography and magnetic resonance imaging in the setting of postmortem examinations. J Surg Educ 65:200–205
128. Griffiths PD, Paley MN, Whitby EH (2005) Post-mortem MRI as an adjunct to fetal or neonatal autopsy. Lancet 365:1271–1273
129. Shiotani S, Kohno M, Ohashi N, Yamazaki K, Nakayama H, Watanabe K (2004) Postmortem computed tomographic (PMCT) demonstration of the relation between gastrointestinal (GI) distension and hepatic portal venous gas (HPVG). Radiat Med 22:25–29
130. Grabherr S, Egger C, Vilarino R, Campana L, Jotterand M, Dedouit F (2017) Modern post-mortem imaging: an update on recent developments. Forensic Sci Res 2:52–64
131. Ampanozi G, Thali YA, Schweitzer W, Hatch GM, Ebert LC, Thali MJ, Ruder TD (2017) Accuracy of non-contrast PMCT for determining cause of death. Forensic Sci Med Pathol 13:284–292
132. Grabherr S, Heinemann A, Vogel H, Rutty G, Morgan B, Wozniak K, Dedouit F, Fischer F, Lochner S, Wittig H, Guglielmi G, Eplinius F, Michaud K, Palmiere C, Chevallier C, Mangin P, Grimm JM (2018) Postmortem CT angiography compared with autopsy: a forensic multicenter study. Radiology 288:270–276
133. Thayyil S, Sebire NJ, Chitty LS, Wade A, Chong W, Olsen O, Gunny RS, Offiah AC, Owens CM, Saunders DE, Scott RJ, Jones R, Norman W, Addison S, Bainbridge A, Cady EB, Vita ED, Robertson NJ, Taylor AM (2013) Post-mortem MRI versus conventional autopsy in fetuses and children: a prospective validation study. Lancet 382:223–233

134. Krentz BV, Alamo L, Grimm J, Dedouit F, Bruguier C, Chevallier C, Egger C, Da Silva LF, Grabherr S (2016) Performance of post-mortem CT compared to autopsy in children. Int J Legal Med 130:1089–1099
135. Proisy M, Marchand AJ, Loget P, Bouvet R, Roussey M, Pele F, Rozel C, Treguier C, Darnault P, Bruneau B (2013) Whole-body post-mortem computed tomography compared with autopsy in the investigation of unexpected death in infants and children. Eur Radiol 23:1711–1719
136. Sieswerda-Hoogendoorn T, Soerdjbalie-Maikoe V, de Bakker H, van Rijn RR (2014) Postmortem CT compared to autopsy in children; concordance in a forensic setting. Int J Legal Med 128(6):957
137. Shelmerdine SC, Davendralingam N, Palm L, Minden T, Cary N, Sebire NJ, Arthurs OJ (2019) Diagnostic accuracy of postmortem CT of children: a retrospective single-center study. AJR Am J Roentgenol 212:1335. https://doi.org/10.2214/AJR.18.20534
138. Bajaj M, Offiah AC (2015) Imaging in suspected child abuse: necessity or radiation hazard? Arch Dis Child 100:1163
139. Berger RP, Panigrahy A, Gottschalk S, Sheetz M (2016) Effective radiation dose in a skeletal survey performed for suspected child abuse. J Pediatr 171:310–312
140. Hampel J, Pascoal A (2018) Comparison and optimization of imaging techniques in suspected physical abuse paediatric radiography. Br J Radiol 91:20170650
141. Rao R, Browne D, Lunt B, Perry D, Reed P, Kelly P (2019) Radiation doses in diagnostic imaging for suspected physical abuse. Arch Dis Child 104:863–868
142. Nickoloff EL, Lu ZF, Dutta AK, So JC (2008) Radiation dose descriptors: BERT, COD, DAP, and other strange creatures. Radiographics 28:1439–1450
143. United Nations Scientific Committee on the Effects of Atomic Radiation (2010) Sources and effects of ionizing radiation. https://www.unscear.org/docs/publications/2010/UNSCEAR_2010_Report.pdf. Accessed 5 July 2021
144. U.S. Nuclear Regulatory Commission (NRC) Personal annual radiation dose calculator. https://www.nrc.gov/about-nrc/radiation/around-us/calculator.html. Accessed 5 July 2021
145. Sheppard JP, Nguyen T, Alkhalid Y, Beckett JS, Salamon N, Yang I (2018) Risk of brain tumor induction from pediatric head CT procedures: a systematic literature review. Brain Tumor Res Treat 6:1–7
146. Abalo KD, Rage E, Leuraud K, Richardson DB, Le Pointe HD, Laurier D, Bernier MO (2021) Early life ionizing radiation exposure and cancer risks: systematic review and meta-analysis. Pediatr Radiol 51:45–56
147. Bernier MO, Baysson H, Pearce MS, Moissonnier M, Cardis E, Hauptmann M, Struelens L, Dabin J, Johansen C, Journy N, Laurier D, Blettner M, Le Cornet L, Pokora R, Gradowska P, Meulepas JM, Kjaerheim K, Istad T, Olerud H, Sovik A, Bosch de Basea M, Thierry-Chef I, Kaijser M, Nordenskjöld A, Berrington de Gonzalez A, Harbron RW, Kesminiene A (2019) Cohort profile: the EPI-CT study: a European pooled epidemiological study to quantify the risk of radiation-induced cancer from paediatric CT. Int J Epidemiol 48:379–381
148. Center for Disease Control and Prevention (CDD) Radiation from Air Travel. https://www.cdc.gov/nceh/radiation/air_travel.html. Accessed 5 July 2021
149. Public Health England (2011) Ionising radiation: dose comparisons. https://www.gov.uk/government/publications/ionising-radiation-dose-comparisons/ionising-radiation-dose-comparisons. Accessed 5 July 2021
150. U.S. Nuclear Regulatory Commission (NRC) Doses in Our Daily Lives. https://www.nrc.gov/about-nrc/radiation/around-us/doses-daily-lives.html. Accessed 5 July 2021
151. Scheuer JL, Musgrave JH, Evans SP (1980) The estimation of late fetal and perinatal age from limb bone length by linear and logarithmic regression. Ann Hum Biol 7:257–265
152. Rijksinstituut voor Volksgezondheid en Milieu Natural radiation in figures [Natuurlijke straling in cijfers]. https://www.rivm.nl/straling-en-radioactiviteit/straling-van-natuurlijke-oorsprong/natuurlijke-straling-in-cijfers. Accessed 5 July 2021

Fracture Dating

4

Hans H. de Boer, Simon G. F. Robben, and Rick R. van Rijn

Contents

4.1 Introduction

In a forensic medical setting the radiologist, pathologist, and/or anthropologist is often asked by the police or prosecution to assess the age of a fracture. In living victims, dating is focused on the time elapsed between the traumatic event and the imaging or examination of the fracture. In deceased victims, the fracture age may be used to assess the interval between the traumatic event and death of the victim. This information can be valuable in legal context for multiple reasons. First, it can be instrumental to police investigation in defining a timeline, which can be used to corroborate or falsify testimonies or to exclude specific scenarios or suspects. Second, it can help to establish the relation between the traumatic event and eventual death, making it possible to show inconsistencies between the more or less objective forensic medical dating and the subjective dating based on the clinical history and the reason provided for the injury [1]. Finally, in case of multiple fractures, dating of the lesions might help to differentiate between a single and multiple traumatic events. In children, depending on their age, the effects of trauma and the signs of subsequent healing may be apparent for months or even years.

Dating of any type of tissue damage is based on the tissue healing response, of which the start coincides with the moment of injury. Any dating method should abide to two basic principles. First, one should be able to recognize features that relate to a specific phase of the healing response. Second, these features should be time-dependent, which means their appearance and/or their disappearance must be limited to a specific amount of lapsed time. Healing responses may differ significantly between different tissue types, and it

H. H. de Boer
Victorian Institute of Forensic Medicine/Department of Forensic Medicine, Monash University, Southbank, VIC, Australia
e-mail: Hans.de.Boer@vifm.org

S. G. F. Robben
Department of Radiology, Maastricht University Medical Center, Maastricht, The Netherlands
e-mail: s.robben@mumc.nl

R. R. van Rijn (✉)
Department of Radiology and Nuclear Medicine, Amsterdam UMC, University of Amsterdam, Amsterdam, The Netherlands

Department of Forensic Medicine, Netherlands Forensic Institute, The Hague, The Netherlands
e-mail: r.r.vanrijn@amsterdamumc.nl

R. A. C. Bilo et al. (eds.), *Forensic Aspects of Paediatric Fractures*, https://doi.org/10.1007/978-3-031-12041-1_4

naturally follows that lesions can only be dated reliably if the forensic specialist is well acquainted with the biology and temporal progression of the healing response of the specific type of damaged tissue.

4.2 Biology of Fracture Repair

Fracture repair is a complex, as of yet incompletely understood biological process which includes a combination of cellular, chemical, and molecular processes. It goes beyond the scope of this chapter to present a full in-depth overview of this process. In this section, a summary of the biology repair is presented as far as relevant for fracture dating. There is a significant amount of literature in which this biology is presented in more detail [2–8].

4.2.1 Types of Fracture Repair

Fracture repair can be divided into two types. The first type, referred to as 'direct' fracture repair, occurs when the fracture is not displaced and is completely stable. As a result, no repair tissue is formed, and the fracture repair is limited to an increase of normal physiological bone remodelling. During this process, the number of so-called bone multicellular units (BMUs) increases at the fracture site. A BMU consists of bone-removing osteoclasts and osteoid-depositing osteoblasts, which in concert resorb old bone and fill the remaining defect with new bone tissue. As the BMUs traverse the fracture line, the fracture will be removed and replaced, eventually resulting in a total resolution of the fracture. From a radiological or histopathological point of view, direct fracture repair only produces a very minute, hardly distinguishable tissue response. This type of fracture repair may be encountered after surgical open reduction and internal fixation but is exceedingly rare in natural circumstances.

Most often fractures heal by means of indirect fracture repair. Here anatomical reduction or complete stability is not required, although a large fracture gap or excessive mobility between the fracture ends may have a negative impact on healing. Indirect fracture repair, which relies on several intermediate steps for its completion, is also referred to as secondary fracture repair. The healing response in case of indirect fracture repair is a sequential process, traditionally subdivided in an inflammatory, a reparative, and a remodelling phase.

4.2.1.1 The Inflammatory Phase

The inflammatory phase of indirect fracture repair starts immediately after the fracture. The disruption of blood vessels at the fracture site results in hematoma formation, with subsequent activation of the coagulation cascade. Activation of the coagulation cascade results in fibrin deposition. At the same time, the haematoma and the traumatically damaged tissue at the fracture site induce an inflammatory response with release of inflammatory mediators. This response is amplified by the osteocyte necrosis that is caused by the disruption and thrombosis of blood vessels. The following inflammatory response has many similarities to that seen in many other tissue types of the human body: an acute inflammatory response with polymorphic nuclear leukocytes is followed by a chronic inflammatory response with mononuclear inflammatory cells. Debris and necrotic tissue are removed by macrophages. Specific for bone tissue are the cells that are tasked with the removal of necrotic bone tissue: multinucleated osteoclasts. Radiologically and histologically, this can be appreciated as resorption of the fracture ends. Soon after the traumatic event, granulation tissue is formed. This heralds the next phase of fracture repair: the reparative phase.

4.2.1.2 The Reparative Phase

The reparative phase is characterized by cell growth and differentiation. This requires the influx of mesenchymal stem cells, which may differentiate in fibroblasts, chondroblasts, or osteoblasts. The differentiation of mesenchymal stem cells into a specific type of mesenchymal cell is dependent on local factors (e.g. local strain and hypoxia), and eventually gives rise to both enchondral and intramembranous ossification. Between the fracture ends, granulation tissue is gradually replaced by fibrous tissue and cartilage. This tissue lends stability to the fracture and is, in the absence of mineralized tissue, also referred to as 'soft callus'. Analogous to embryological skeletal development, the cartilaginous tissue in the soft callus is subsequently replaced by bone by enchondral ossification. At the same time, intramembranous ossification occurs at the periosteal surface of the fracture edges. This type of ossification may either be the result of the activation and differentiation of the osteoprogenitor cells that reside in the cambium of the periosteum, or osteoblast that differentiate from recruited mesenchymal stem cells. The result of the combined enchondral and intramembranous ossification is a bony cast surrounding and bridging the fracture. As the callus becomes more and more mineralized, it is also referred to 'hard callus'. Please bear in mind that the definition of 'soft' and ' hard callus' may differ in pathology and radiology literature. The fracture becomes more stable as the amount of mineralized tissue in the callus increases, eventually resulting in stability in each direction.

4.2.1.3 The Remodelling Phase

The bone tissue that is deposited by enchondral or intramembranous ossification is generally characterized by a haphazard and unorganized microarchitecture, called woven bone. This contrasts to normal bone tissue, which has a highly organized lamellar architecture. This specific lamellar

architecture is the result of continuous remodelling during life, in which the bone lamellae are oriented to meet the mechanical demands of the skeletal element, while using a minimum of tissue volume. This is also known as Wolff's law: the bone tissue of a healthy individual will adapt to the loads under which it is placed.

In order to regain its full function, the newly deposited bone tissue thus needs to be remodelled. This gradual replacement of woven bone tissue by lamellar bone tissue is similar to the normal physiological remodelling that takes place during life. It involves an intricate process in which the hard callus is resorbed by osteoclasts, and lamellar bone is deposited by osteoblasts. This remodelling is done by virtue of bone multicellular units, and dependent on the fracture, may take months or years, depending on the fracture and the age of the patient.

4.2.2 Factors that Influence Fracture Repair

Fracture repair may be influenced by a multitude of factors, resulting in either an impairment or acceleration of the process. These factors might be local, i.e. related to the fractured bone and its surrounding tissues, or systemic, i.e. related to the individual. Some of the most common and most relevant factors are listed in Table 4.1. A more detailed discussion than the one provided below can be found in various articles [48–50].

4.2.2.1 Local Factors

There are three major local factors that have an influence on bone healing. First, the position of the fracture ends, defined by fracture alignment and fracture gap. In general, a more anatomical position of the fracture leads to quicker repair. Consequently, malaligned or large fracture defects heal slower. A fresh fracture near an already healing fracture may considerably delay the healing process. A new fracture through an older untreated fracture can be identified by ample new bone formation and a clearly defined fracture line [9]. Repeated trauma may delay the resolution of soft-tissue injuries, periosteal new bone formation, hard callus formation, and remodelling [10].

Table 4.1 Selection of factors that influence fracture healing

Local factors	Systemic factors
Type of bone	Age
Fracture gap	Activity level
Fracture alignment	Nutritional status
Fracture stability	Health status (e.g. diabetes, obesity, use of medication)
Soft-tissue interposition	
Vascularity	
Infection	
Local bone disease	

Second, the stability of the fracture site, which has a less straightforward effect on healing. Whereas direct fracture repair requires absolute stability, early indirect fracture repair is enhanced by on-axis micro-movement. However, off-axis movement or an excessive amount of movement will prevent the deposition of bone tissue and can ultimately result in pseudoarthrosis development [11, 12]. Apparently, a 'perfect window' for adequate indirect fracture repair exists, and this forms the basis for semi-rigid immobilization of fractures such as casts, intramedullary nails, and external fixation devices. The beneficial or hampering effects of motion may affect various regions of the skeleton differently. For example, ribs are almost continuously in motion, not just because of the continuous process of breathing, but also through intermittent processes such as picking up and holding the child.

Third, the type and location of fractured bone has considerable effect on healing speed. Forearm fractures tend to heal faster than leg fractures, whereas peripheral appendicular bones, such as phalanges, tend to heal faster than proximal appendicular bone, such as the humerus [13]. Cancellous bone generally heals faster than cortical bone. Keep in mind that these are generalizations; they are by no means absolute.

Besides these three major factors, many others exist. Given the complexity of fracture repair, almost any local factor that has a direct or indirect effect on inflammation, cellular signalling, or tissue differentiation has an effect on the speed of fracture repair. Important ones to keep in mind are an impeded blood supply or denervation of the fracture site, pre-existing local bone disease, infection, and soft-tissue interposition. All are known to delay or even halt the fracture repair process.

4.2.2.2 Systemic Factors

As in local factors, any systemic factor that directly or indirectly affects the physiology of the repair process may affect its temporal progression. As such, systemic diseases that influence bone quality, such as osteoporosis, are known to impede fracture repair [14]. Also, the nutritional status seems to affect the moment of callus formation around the healing fractures; the poorer the nutritional status, the slower the development of new bone [15]. The same holds for vitamin deficiencies, or alcoholism [16, 17]. Among systemic factors that negatively influence fracture repair are furthermore those that are commonly related to vasculopathy. Examples hereof are smoking, obesity and diabetes or the use of steroids [18, 19].

A special comment should be made on the relation between the age of the individual and the speed of fracture repair. It is generally assumed that fractures tend to heal faster in younger individuals, and that this difference is limited to specific age groups. That is, young children

generally heal faster than older children or adults. The cause of these differences is poorly understood and probably many factors such as higher metabolism, vascularity, and mitotic activity in children play a role. Also, several genes and hormones involved in skeletal growth are involved in fracture healing. In children, which still experience skeletal growth, this osteogenic environment is a given, which facilitates fracture healing. In adults, the metabolic rate in bone is much lower, which impedes the speed of the healing response. However, in contrast to this, Prosser et al. maintain that as of yet there are no publications of scientific data that proof that fractures in young children (in particularly those of less than 1 year old) do indeed heal faster [20].

4.3 Dating Fractures

As described above, the general sequence of events in fracture repair is well known, and there is a considerate amount of data on the local and systemic factors that affect its speed. However, unlike the artificial and somewhat rigid description just given, fracture repair is a much less straightforward biological process; each phase gradually passes into the next. This in combination with local differences in the speed of healing may result in the simultaneous presence of several phases in one fracture. Second, while it is generally known which factors impede or accelerate fracture repair, there is hardly any knowledge on the extent in which they do so. Moreover, individuals commonly have a combination of impeding or accelerating factors, while the interplay between them is largely unknown. Despite these problems, the potential value of fracture dating has inspired various attempts to use either radiological or histological methods for the estimation of lapsed posttraumatic time.

4.3.1 Radiological Aspects of Fracture Dating

The radiological dating of fractures can either be done in living or deceased individuals. Especially when a victim is unable to provide an adequate clinical history, the radiological assessment of fracture repair can provide objective information on the age of the lesion. This is most often the case in children and as a result, virtually all research on the radiological dating of fractures has been aimed at this age group [13, 20–30]. For fracture dating follow-up radiological examination is valuable. In 1996, Kleinman et al. described the benefit of follow-up radiography in a study that comprised 23 children that had sustained fractures [31].

On conventional radiographs, the fracture repair process can be broken down in different phases (Figs. 4.1, 4.2, 4.3, and 4.4):

1. Soft-tissue swelling surrounding the fracture. This obviously depends on the fracture location, as for instance soft-tissue swelling surrounding a femoral or humeral fracture is much more distinct compared to a fracture of the phalanx of the hand or foot (Fig. 4.1a). In case of rib fractures, one should look for the presence of a focal pleural thickening (Fig. 4.2).
2. Presence of periosteal reaction, also called subperiosteal new bone formation. Subperiosteal new bone formation is especially seen in long bone fractures, while it may be more difficult to appreciate in rib fractures (Fig. 4.1b).

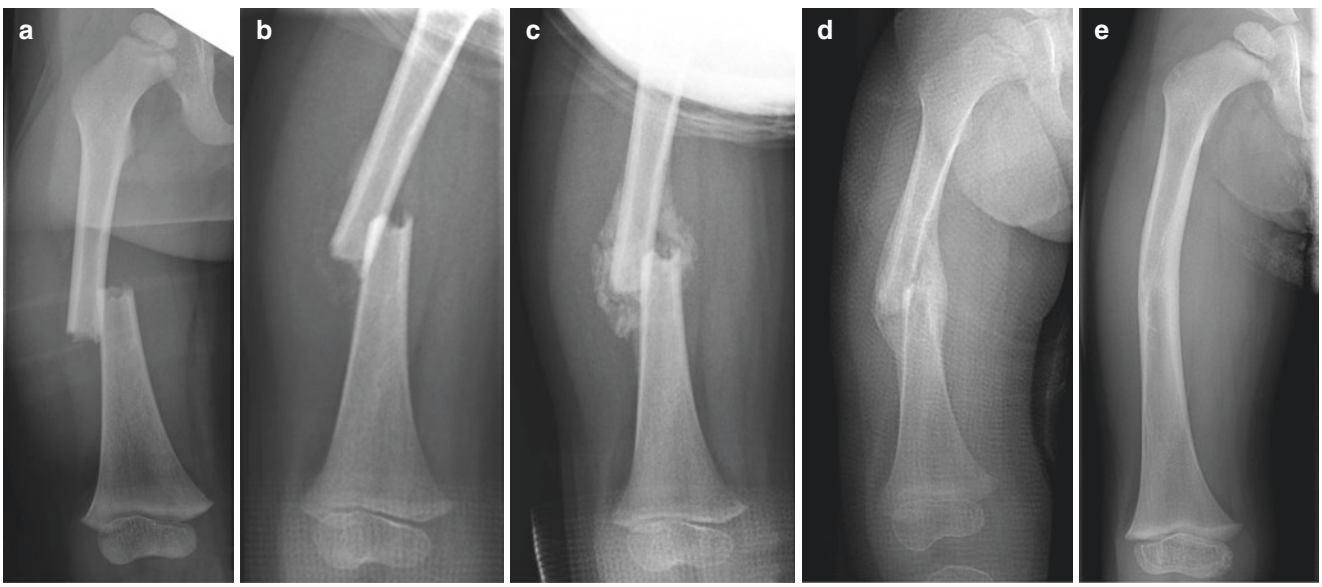


Fig. 4.1 Various phases of bone healing in a 1-year-old child, (**a**) transverse femoral fracture at day 1, (**b**) subperiosteal new bone formation and soft callus at day 12, (**c**) soft callus transforming to hard callus at day 18, (**d**) hard callus at day 45, and (**e**) remodelling after 11 months

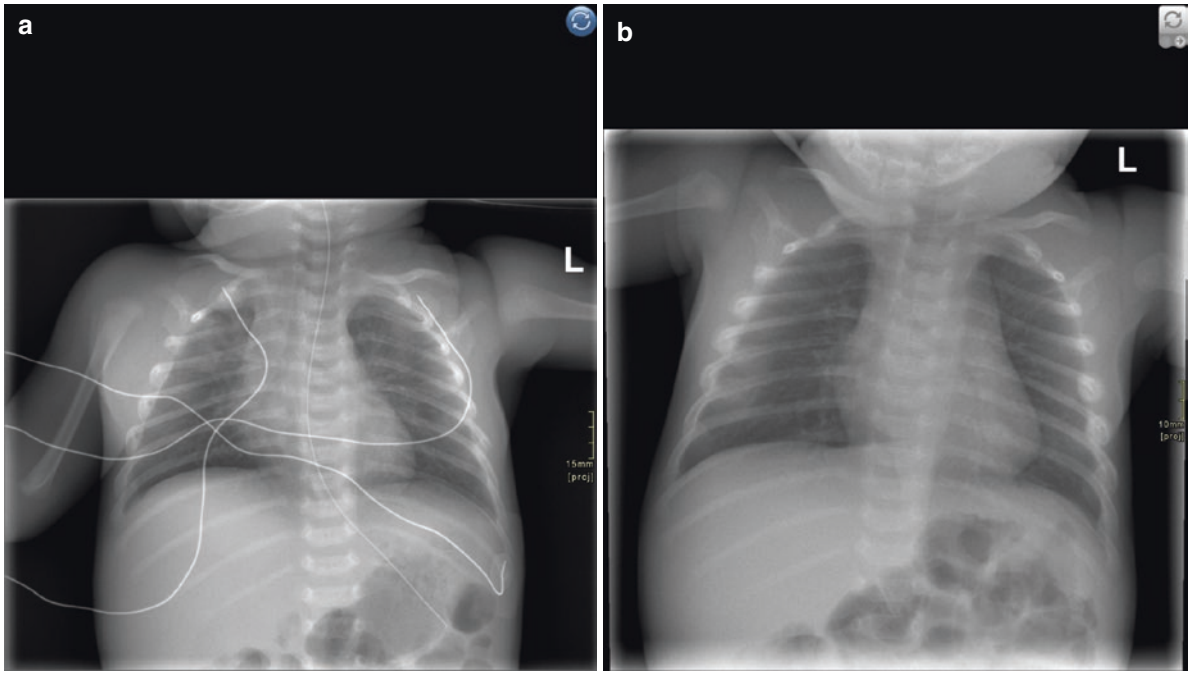


Fig. 4.2 (**a**) Chest radiograph showing bilateral pleural thickening and an acute rib fracture on the left side. (**b**) Follow-up radiograph shows bilateral healing rib fractures

Fig. 4.3 Lateral view of the lower arm showing widening of fracture line. (**a**) Fresh re-fracture of an old antebrachial fracture. Note the thin greenstick fractures of radius and ulna shaft. (**b**) Healing fracture 4 weeks later. Hard callus has formed, evident widening of both fracture lines

3. Soft callus formation. Radiologically defined as the deposition and organization of soft tissues with incipient mineralization to produce a 'fluffy' appearance adjacent to the fracture (Fig. 4.1c). Also the margins of the fracture line become more indistinct and the fracture line may occur wider on the radiograph, both because of bone resorption in the inflammatory and reparative phase of fracture healing (Fig. 4.3).
4. Hard callus formation. Mineralization of the soft callus and deposition of new bone produces a well-demarcated periosteal callus which surrounds the fracture (Fig. 4.1d) with a density that approximates the density of cortical bone. Endosteal hard callus may be visible as a zone of sclerosis at the fracture site (Fig. 4.4).
5. Bridging. The fracture line becomes indistinct, but callus is still clearly visible.
6. Remodelling. The fracture line is no longer discernible. The callus contour is smoothened and over time, only a deformity of the bone remains (Fig. 4.1e). This stage can even lead to a complete resolution of all findings related to the fracture.

Abovementioned stages are relatively well defined and sometimes used in daily clinical practice. For forensic purposes however, we need information on the accuracy with which radiologists can discriminate between them, and the exact time lapse related to them. Several papers that studied the healing phases of fractures in children also included interobserver variability (Table 4.2) [20, 23, 24, 27, 30]. The interobserver agreement varied from poor to excellent. Not

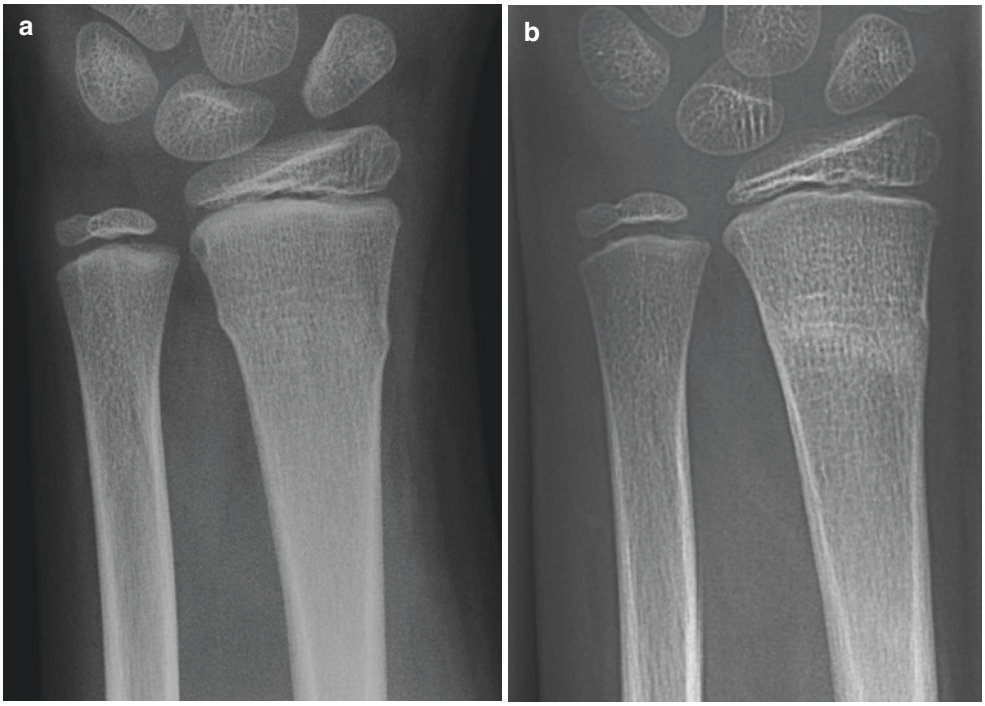


Fig. 4.4 An example of endosteal callus formation. (**a**) Torus fracture distal radius in an 8-year-old boy. (**b**) On follow-up imaging endosteal callus at the fracture site. No periosteal callus

Table 4.2 Agreement in the assessment of radiological healing features

Feature	Halliday [24]	Fadell [23]	Walters [27]	Prosser [20]	Warner [30]
Soft-tissue swelling	0.41–0.66			0.70	
Subperiosteal new bone formation	0.83–0.96	0.61	0.92	0.79	0.85
Definition of fracture line	0.46–0.64				
Presence or absence of callus	0.86–0.88	0.90	0.89		0.81
Well or ill-defined callus	0.17–0.76			0.77–0.80	
Presence of endosteal callus	0.34–0.73				
Bridging		0.69		0.82	0.69
Remodelling		0.86		0.83	

Abbreviations: *NR* not reported

all of the radiological features of fracture healing were addressed in every paper. The observer agreement of the definition of the fracture line was only mentioned in one paper [24] and was only moderate. Some papers make no distinction between soft and hard callus and just score the presence or absence of callus with a good to excellent interobserver agreement. The agreement on bridging and the discrimination between soft and hard callus varies between moderate and good. Overall, the presence of absence of callus or the presence of periosteal new bone formation had the best observer agreement.

The second important factor, next to observer agreement, is the accuracy of dating. For the relation between specific radiological features and lapsed time, most radiologist will refer to the chapter 'dating fractures' by O'Connor and Cohen, which contains a table with such information [10]. This table is, as stated by the authors, based on personal experience, and therefore is under Daubert criteria not admissible as evidence in the Court of Law. There have been several publications on dating fractures, most of them with relatively small populations (Table 4.3) [13, 20, 22–30, 32].

In 2005, Prosser et al. published a systematic review on dating paediatric fractures in which only three publications met their inclusion criteria [21, 22, 25, 28]. There were a total of 189 children, of which 56 were under the age of 5 years. The authors subsequently presented a table which links radiological healing features to specific time intervals. Given the limited number of included children, the relatively broad time ranges underscore the large variation in fracture repair speed. The effect of age on the speed of the process was noted in the described studies: the youngest individuals healed the fastest. Furthermore it must be kept in mind that considerate differences exist in the radiological appearance of fractures. For instance, skull fractures and metaphyseal corner fractures are known for healing with a little or no callus formation at all [31, 33]. In forensic case work the question is relevant if there is an influence of the literature used to base an age assessment on. Drury and Cunningham performed a study based on 112 long bone fractures in 96 children with an average age of 9.1 years (range 1–17 years)

Table 4.3 Overview of reported radiological healing features and associated time lapse

Author (year)	**Cumming [22]**	**Yeo [28]**	**Islam [25]**	**Halliday [24]**	**Malone [13]**	**Prosser [20]**	**Sanchez [26]**	**Walters [27]**	**Fadell [23]**	**Warner [30]**	**Hosokawa [29]**	**Crompton [32]**
Year	1979	1994	2000	2011	2011	2012	2013	2014	2017	2017	2017	2021
Fracture location	Clavicle, humerus, and femur	Femur	Radius and ulna	Long bones	Long bones	Clavicle and long bones	Ribs	Clavicle	Clavicle	Long bones	Femur	Femur
N (cases)	23	25	141	31	107	63	16	131	61	40	12	30
N (fractures)	23	25	141	37	107	82	23	131	61	59	12	30
Age (range)	New-borns	0–14 years	Mean 8 years—range 1–17 years	0–44 months	0–5 years	Mean 4.8 years	1–11 months	0–3 months	24 days (0–2 months)	9 days–12 months	New-borns	0–2 years
Radiological feature	**Peak (range)**	**Peak (range)**	**Peak (range)**	**Peak (range)**	**Peak (range)**	**Peak (range)**	**Peak (range)**	**Peak (first seen)**	**Peak (range)**	**Peak (range)**	**Peak (range)**	**Peak (first seen)**
Fracture gap widening			4–6 weeks 56% (2–8 weeks)									
Periosteal reaction presence	9–10 days (7–11 days)	1.6 weeks (1–3 weeks)	4–7 weeks 100% (2 weeks onwards)	(4–11 days)		22 (5–96 days)	1–3 weeks	10 (7+)	11 days (7–49)	9–49 days (7–130 days)	14 days (9–23 days)	7–11 days
First callus	7 days (9–10 days)	(1–3 weeks)	4–7 weeks 100% (2 weeks onwards)	(4–11 days)		No real peak (12–66)	3–5 weeks	15 (9+)	11 days (11–61)	9–26 days (9–130 days)	16 days (10–23 days)	15–26 days
Callus density > cortex			13 weeks 90% (4 weeks onwards)		38 days (15–75 days)	≥22 days (19–96 days)	5–7 weeks				21 days (16–32 days)	
Bridging		2.6 weeks (1.5–3.7 weeks)	13 weeks 50% (3 weeks onwards)	Earliest seen at 13 days, present on all films > 20 days	44 days (24–93 days)	≥36 (19–300 days)	7–9 weeks		22 days (20–63)	51–247 days (51–247 days)		
Completion			14 weeks (7 weeks onwards)		313 days (42–750 days)							

which they dated using the data described by respectively Islam et al., Malone et al., and Prosser et al. [34]. The study analysed fractures from a wide range of locations: 40 humeral, 13 ulnar, 10 radial, 11 metacarpal, 4 femoral, 5 tibial, 1 fibular, 23 metatarsal, and 5 proximal phalangeal fractures were included. This study, not surprisingly, showed differences in age assessment between all three methods. The authors concluded that great caution should be used when applying existing timetables to radiologically date a paediatric fracture of unknown age.

Messer et al. performed a systematic review on radiographic features of paediatric fracture healing and corresponding timelines [35]. This review included 10 studies that all had a retrospective design [13, 20, 23–30]. The number of children in each study varied greatly, from 12 [29] to 141 [25]. They found that the radiological features of healing and the timelines of common fracture healing variables differed significantly among studies for several reasons:

First, the age ranged from 0 to 17 years. It is unlikely that the timetable of a five-year-old child can be extrapolated to that of a neonate. Only 2 studies examined the effect of age [13, 28]. Second, various studies describe various bones or combination of bones. It is highly speculative that the healing of a rib fracture matches the healing phases of a femoral shaft fracture. Malone found that forearm fractures heal faster than leg fractures [13]. Third, the studies did not differentiate between various types of fractures. Only one study addressed fracture type (complete versus incomplete) with differences in healing between the two [30]. A torus fracture will heal with significant less callus than a fracture with displaced fragments. Fourth, because all the studies had a retrospective design the timelines may (in part) present hospital radiographic protocols, e.g. taking the first control radiograph only after 7 days. Fifth, there were eight studies focused on accidental injuries. Applying healing timelines generated from accidental trauma in children who are in good health and who receive adequate treatment to fractures in physically abused children who might suffer from malnutrition and multiple injuries is at least questionable. Finally, the radiological features of fracture healing varied considerably among the studies, but most used terminology was subperiosteal new bone formation, callus, bridging, and remodelling. Considering all abovementioned flaws, Messer et al. conclude that fracture healing timelines should be used with caution.

All in all, the current situation in radiological fracture dating is perhaps best illustrated by quoting Prosser et al. '*dating of fractures is an inexact science*' [20]. Each radiologist should be thoroughly aware of this whenever interpreting radiographs for the purpose of dating.

Next to the specific radiological stages of fracture healing at the fracture site, there are also changes in the rest of the fractured bone. Changes that manifest later during the healing phase and as such may provide additional information for the timeline. These include osteopenia, overgrowth, metaphyseal band-like lucencies, and growth lines. Osteopenia is caused by disuse during active (pain) or passive (cast) immobilization in combination with inflammatory-induced hyperaemia. Bone mineral density is difficult to appreciate on conventional radiographs but specific features are easier to recognize like subchondral epiphyseal lucency, metaphyseal band-like lucency, and appearance of Haversian canals in cortical bone (Fig. 4.5a, b).

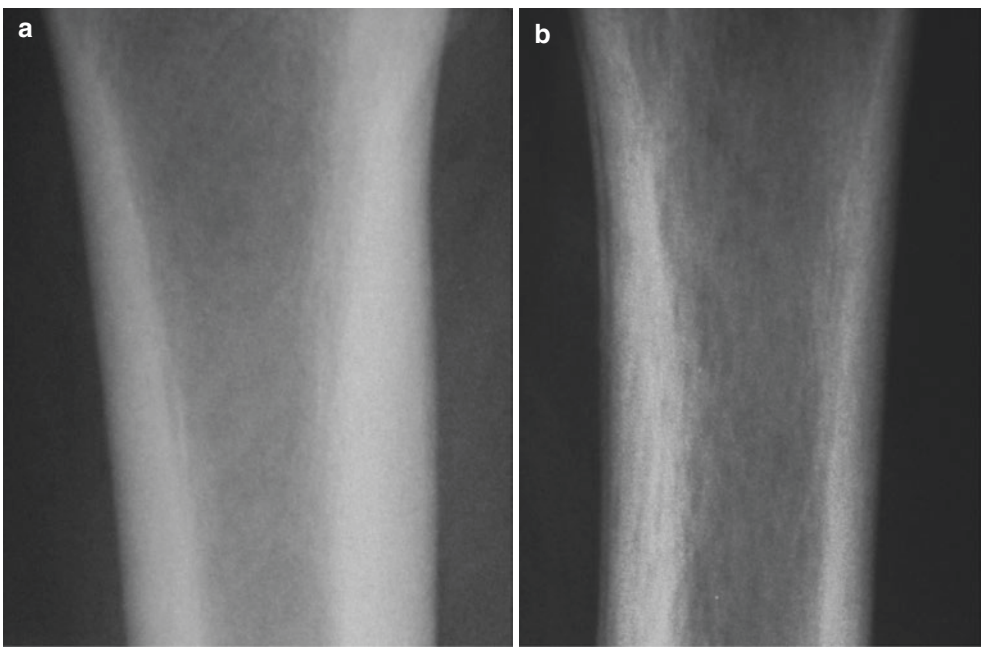


Fig. 4.5 Osteopenia in the femoral shaft of an 11-year-old girl with a left-sided slipped capital femoral epiphysis. (**a**) Right femur: normal shaft versus. (**b**) Left femur: appearance of Haversian canals due to disuse osteopenia

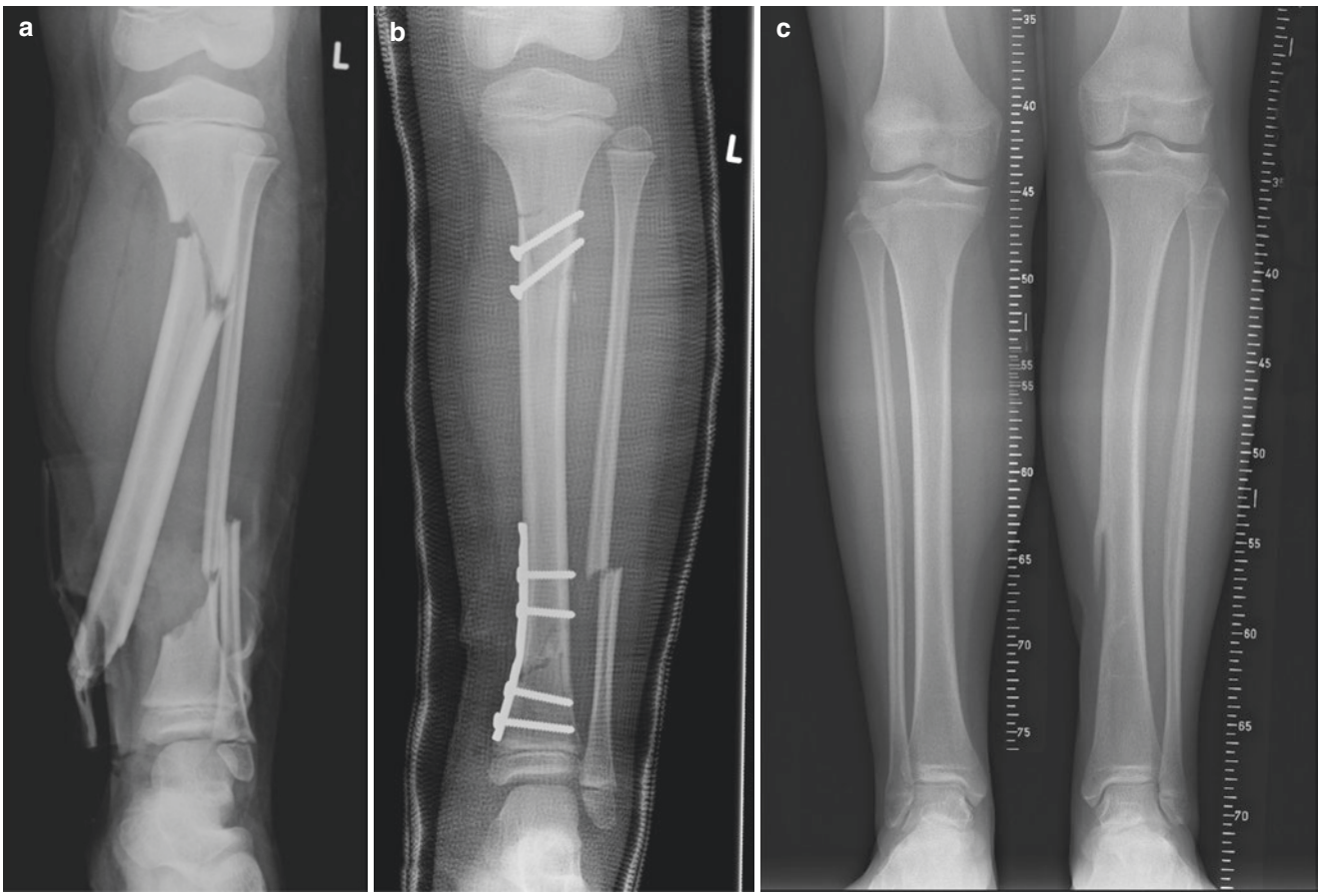


Fig. 4.6 A 6-year-old child with multiple fractures of the left lower leg (**a**) and after surgical reposition (**b**). At the age of 13, a significant leg length discrepancy has developed in the lower leg (**c**)

Overgrowth is present in the months after the fracture. It is probably a side-effect of inflammatory-induced hyperaemia, not only at the fracture side, but in the entire bone including the growth plates. It results in temporary overgrowth and thus increased lengthening. This phenomenon is especially seen in diaphyseal fractures (with a large hematoma) of long bones with a high growth rate such as femur, tibia, and humerus. The resulting leg length discrepancy may cause considerable invalidating effects (Fig. 4.6a–c).

Band-like metaphyseal lucency is a subset of focal intense osteopenia because of the high metabolic rate and high vascularity of the metaphysis, reflecting its specific task in longitudinal growth. Finally, growth lines (Parker-Harris lines) reflect a temporary arrest in longitudinal growth and probably formed in the first days after the fracture in the zone of provisional calcification. In time, as longitudinal growth recovers, they gradually 'grow away' from the growth plate. The distance from the growth plate to the growth line is an indication of the time elapsed since the fracture (Fig. 4.7a, b). However, growth lines are not pathognomonic for past fractures. They may also be silent witnesses of a variety of temporary metabolic disturbances, intercurrent diseases, intermittent medication or, in some cases, remain idiopathic.

4.3.2 Histological Aspects of Fracture Dating

Histological examination of a fracture in a forensic context is limited to deceased individuals. In most instances, it will relate to non-decomposed 'fresh' human remains, in which the fracture, including its surrounding soft tissues, is processed for histological slides. These slides, including both mineralized and non-mineralized tissue, naturally convey the most information on the healing phase of the fracture and are therefore preferred. In some cases, the remains are decomposed to such an extent that only skeletonized remains are available. The loss of the soft-tissue component and the potential microbial degradation and/or remineralization of the skeletal elements will complicate the analysis considerably. As most forensic pathological casework will relate to non-decomposed human remains, this paragraph will focus (unless otherwise specified) on the examination of fresh, non-decomposed human tissue.

The histology of fracture repair is described in most general pathology and orthopaedic pathology textbooks, and often these texts provide a crude estimation of the time related to various healing phases. Although such texts are valuable from an educational point of view, they do not suf-

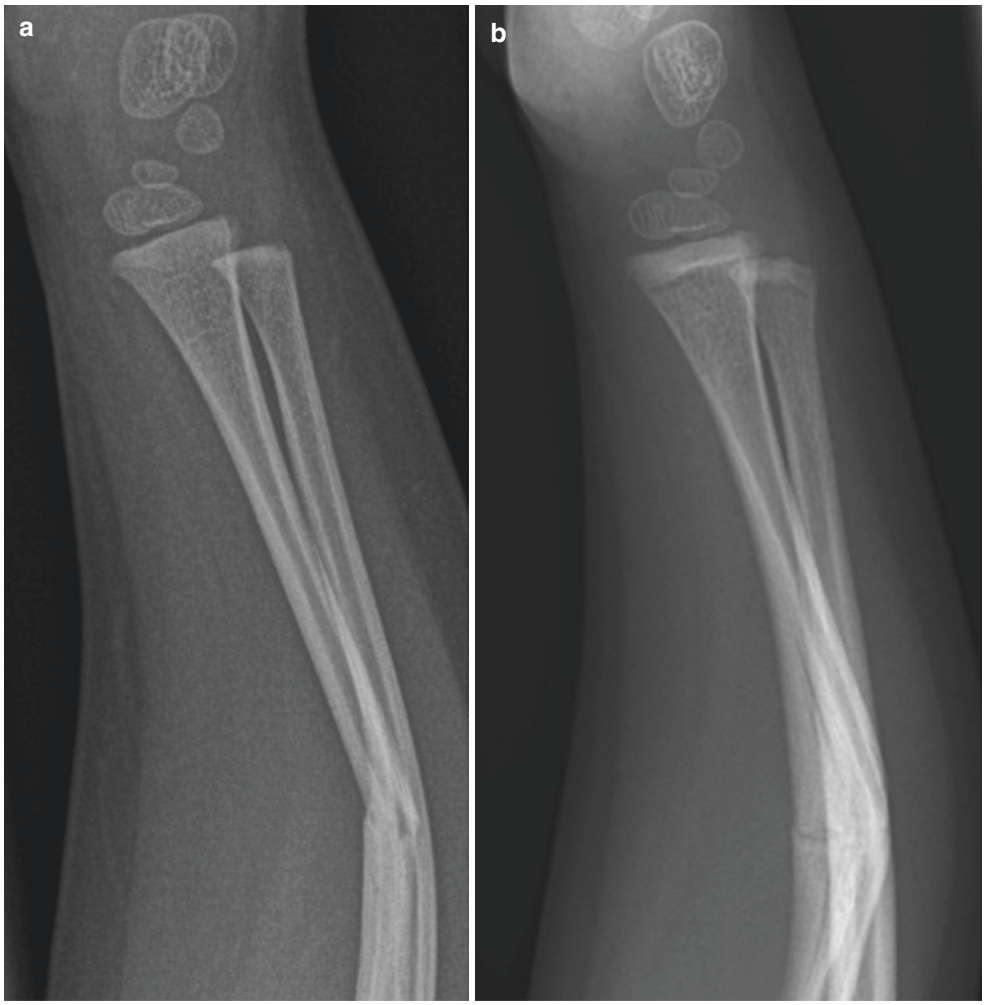


Fig. 4.7 (**a**) Lateral view of a fracture of the left lower arm. (**b**) Three months later, the fracture is healing and a growth line has formed at distal radius and ulna

fice as a basis of fracture dating in a forensic context. As in radiological fracture dating, the latter depends on the extent in which two basic premises are met: knowledge on the agreement in which histological healing features can be recognized reliably, and the extent in which these healing features are accurate representatives of lapsed time since the traumatic event.

The agreement of observers on their histological findings is a common point of discussion in any part of pathology, and medical scientific literature almost invariably shows that general agreement between pathologists is far from perfect. The partial alleviation of this problem by adhering to strict definitions does not only illustrate the need for such definitions, it also shows that histological examination is inherently subjective. Although this may come as no surprise, no data currently exists on the agreement between pathologists on the presence or absence of histological fracture repair features. Of course, many of these features are fairly well-defined such as the presence of hematoma, the formation of granulation tissue, or the apposition of osteoid. It may thus be expected that examiners agree considerably on their presence. Still, although texts on histological fracture examination seem to agree with this assertion, it lacks direct scientific evidence [8, 36].

Currently one exception to this rule exists, and it only relates to skeletonized tissue. A survey of non-decalcified, unstained histological slides of 22 fractures and 9 amputations showed a substantial agreement between three observers in the recognition of various healing phases [37]. Both ‘early’ features, such as the resorption of bone tissue adjacent to the fracture, and ‘late’ features, such as callus formation and callus remodelling, were considered reliably detectable. Histological staining of the sections with haematoxylin showed to have an effect on the visibility of some of the healing features, although our experience with the method suggests a less dramatic effect than the study shows.

Only a very limited number of studies focus on the exact timing of the emergence or disappearance of specific healing features, which is at least partially due to problems with the availability of adequate study material. Forensic material often lacks sufficient contextual data or cannot be used for research purposes, while obvious ethical constraints pre-

clude experimental human studies. Most of the limited data on the chronology of fracture repair therefore stems from various animal studies [38–42]. These studies are helpful for the development of new treatment strategies, but the obvious differences between the several animal models and humans disqualify this data for forensic purposes [43]. The studies that could serve a forensic purpose, i.e. well-documented human cases, are extremely scarce, usually outdated and mostly case descriptions [44–48].

In an attempt to circumvent the unavailability of direct data, authors have resorted to various research designs to devise a histological fracture dating methodology. In 2003, Klotzbach et al. made an attempt to date 44 paediatric fractures histologically [36]. Relatively recent fractures were dated by the presence of osteoblasts (reported to emerge at 2–4 days), osteoclasts (reported to emerge at 4–7 days), and the mineralization of osteoid (reported to emerge at 8 days). Older fractures were dated by comparing the amount of deposited bone with a known rate of bone apposition, which was derived from experimental human studies. Although elegant, the study had several important limitations: For instance, the source data did not come from a representative cohort (i.e. was mainly experimental and related to adult individuals). More importantly, the actual age of the studied lesions was unknown. The performance of the methodology thus remains to be tested.

In 2008, Malcolm reviewed the examination of fractures in autopsy pathology, and provided a chronology of several of the healing phases [8]. Cognisant of the many (un)known variables that affect fracture healing, he advocated the use of rather broad time intervals, especially in the later phases of healing. Instinctively, this more careful approach is more accurate than the detailed approach by Klotzbach et al., be it at the costs of specificity. However, the publication does not provide details on the source information of the time intervals. In addition, the performance of the dating methodology in a series of fractures of known age is not given.

In 2010, Maat and Huls published a chapter on the histological fracture dating in the first edition of this book [49]. This publication features a table that links specific healing features to specific time lapses, a reworked version is presented here as Table 4.4. Their time intervals are comparable

Table 4.4 The presence of histological healing features in infants, adopted from Naqvi et al. [52]

No. of fractures	11	10	11	16	14	6	13	10	7	8	5	6	8	8	8	5	6	5	4	4	4
Posttraumatic survival time	**1 h**	**2h**	**4h**	**12h**	**12-24h**	**24-36h**	**36-48h**	**3d**	**4d**	**5d**	**6d**	**7d**	**8-14d**	**15-21d**	**22-28d**	**29-35d**	**36-49d**	**50-70d**	**71-91d**	**92-112d**	**>122d**
Haemorrhage	11	10	11	16	14	6	11	6	4			1									
Osteocyte loss	2	10	11	16	14	6	13	8	4	5	2	2									
Fibrin formation			2	15	14	6	12	4	2												
Inflammatory reaction: polymorphs infiltration between the fracture ends				7	13	5	6	4	2												
Inflammatory reaction: macrophage infiltration between the fracture ends						3	8	9	4												
Granulation tissue formation						2	11	10	6	8	2										
Osteoclast activity at the fracture site							5	9	7	8	5	4									
Early condensation of mesenchyme							4	4	7	8	5	4									

(continued)

Table. 4.4 (continued)

into 'crude scaffold shapes'																					
Woven bone formation. Early primary callus including osteochondral tissue									2	8	5	6	7	1							
Bone trabeculae and cartilage nodules											2	5	8	8	7	2					
Calcification													5	8	8	5	6	5	4	4	4
Fracture union													2	7	8	5	6	5	4	4	4
Remodelling of woven bone into lamellar bone															3	5	5	5	4	4	4
Evidence of bone returning to normal																				4	4

The presence of histological healing features related to their posttraumatic survival time, as observed by Navqi et al. in 169 fractures in infants (<12 months). The total number of fractures studied per time interval is shown in the top two rows. The proportion of fractures with a particular healing feature is indicated by different colours: blue: less than 25%; green: 25–49%; orange: 5–74%; grey: 75–89%; black: 90–100%

to those given by Malcolm, but provide more detailed description on the defining healing features per healing phase. The source information for this table comes from an extensive literature review. Instead of reporting the time at which a healing feature is generally seen, the table reports the minimum amount of time needed for its emergence; the 'minimum posttraumatic survival time'. Theoretically, this alternative approach may circumvent the problems associated with interpersonal differences in healing speed. However, as each histological healing feature shows a more or less gradual increase before reaching peak intensity, the approach may result in considerable underestimation of the true age of the fracture. This increases in older fractures. Maat and Huls explicitly state that their table is compiled from data from healthy, non-operatively treated adult individuals, and that children may show a markedly quicker healing response. They furthermore list some histochemical stains that may aid the recognition of specific healing features. As the previous mentioned studies, the method has not been tested in a series of fractures of known age.

Although initially designed for fresh tissue, adaptations have made Maat and Huls' approach applicable to skeletonized remains as well [33, 37, 50]. This method for skeletonized material was tested by Cappella et al. [51] in five rib fractures and four skull fractures of known age. The method accurately estimated minimum posttraumatic survival in 8 out of 9 specimen, but indeed, significantly underestimated the true age of the fracture, especially in older fractures. This was particularly the case in the skull fractures, again emphasizing the influence of fracture location on the healing response. The (very) small number of cases however prevents large generalizations.

The largest and most comprehensive study in histological fracture dating stems from 2019 [52], and only included infants of 12 months of age or younger. Over a 32-year period, a total of 169 fractures with a known time of injury were studied. For a total of 14 healing features, ranging from local haemorrhage to restoration of the normal bone tissue, their absence or presence was determined and related to the posttraumatic survival time. The data confirms the sequential and time-related process of histological fracture repair, but also shows that many healing features may be encountered in fractures that differ considerably in terms of age. The results once again underscores the variability of the speed of the fracture repair process. The study included a wide variety of types and locations of fractures, and interobserver agreement of the described healing features was not tested.

All in all, various practitioners have tried to provide benchmarks for histological fracture dating, with the study by Navqi et al. currently providing the most comprehensive information for infants of less than 12 months of age [52]. As in radiological fracture dating, no fully tried and tested

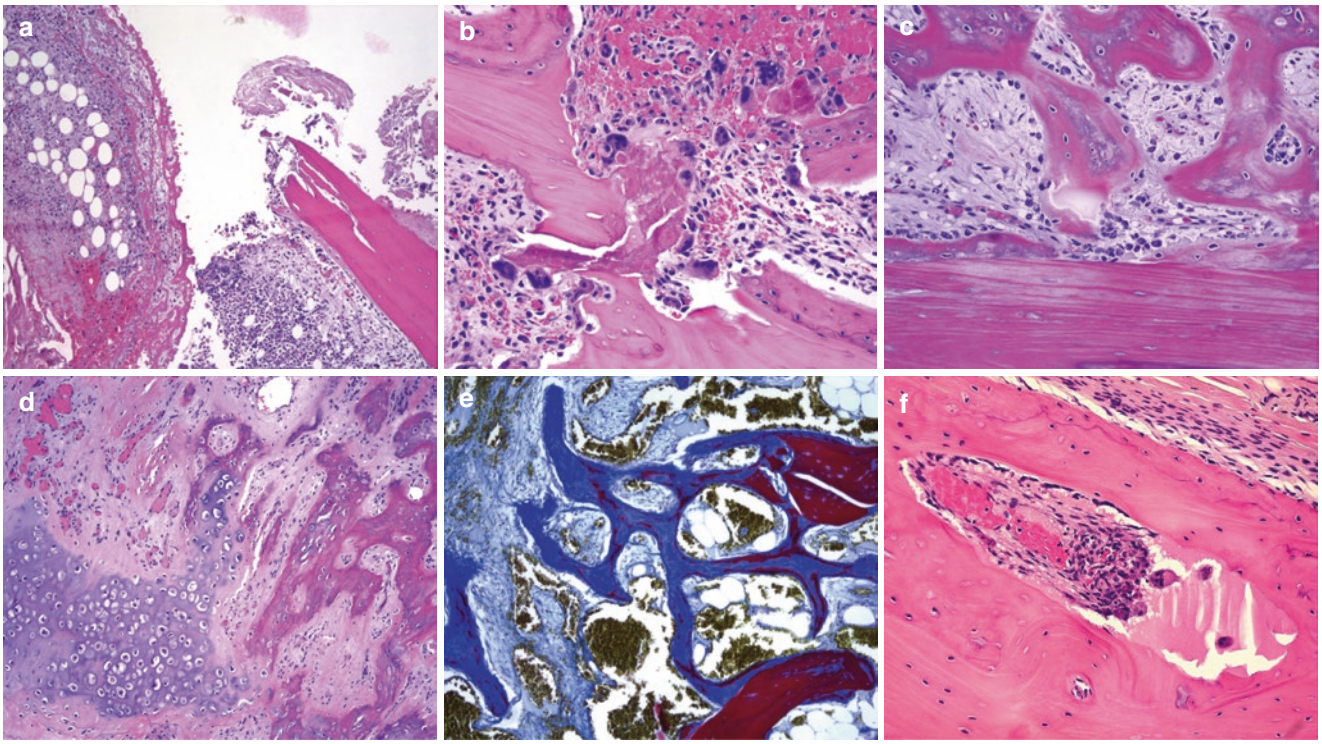


Fig. 4.8 Features indicative of various steps in the fracture healing cascade. (**a**) A recent fracture, with frayed fracture lamellae and early granulation tissue formation. (**b**) Granulation tissue and numerous multinucleated osteoclasts which remove necrotic bone fragments (recognizable by their empty osteocyte lacunae). (**c**) Early 'woven' bone formation directly adjacent to the cortex of a fractured rib. Note the difference between the highly organized lamellar bone and the haphazard architecture of the woven bone. The cuboid, plump cells lining the new bone are active osteoblasts. (**d**) Woven bone on the right side, but the lower left side of the panel shows chondroid tissue, indicating indirect fracture repair. (**e**) Masson trichome stained section, this type of stain helps to differentiate between the non-mineralized bone-precursor osteoid (blue) and mineralized bone tissue (red). (**f**) So-called 'bone multicellular unit', with multinucleated osteoclasts to the left and osteoid-depositing osteoblasts to the left

histological dating method exists, and practitioners should be fully aware of this when trying to determine the age of a fracture. Various laboratories are currently developing research strategies to examine fractures more systematically, and hopefully these efforts will provide the much-needed scientific evidence to advance the histological dating of fractures. Various histological features that are regularly used for dating are shown in Fig. 4.8a–f.

4.4 The Practice of Fracture Dating in Dutch Forensic Casework

Given all its limitations, it may come as no surprise that the majority of forensic specialists are reluctant to venture into fracture dating. Still, despite the scarcity of the evidence the radiological and histological examination of a fracture may provide information that may be valuable in a legal context. This especially holds in those cases in which clear hypotheses can be tested. An example might be a case in which multiple fractures were found, and the question arises to what extent they may be related to one or multiple events. Another example may be a situation in which a testimony places the fracture in a specific time frame. In such circumstances, the radiological and histological analysis may provide information to corroborate or refute the given scenario. In addition, detailed analysis of the fracture may give additional information on the health status of the individual.

For the sake of practicality, this chapter therefore ends with a description of the fracture dating approach in paediatric cases at the Netherlands Forensic Institute. It includes radiology, histology and uses an integrative approach for interpretation. It is noted that histology is especially helpful in the earlier stages of healing (i.e. the first four weeks of the healing response).

4.4.1 Radiological Imaging Before and After Autopsy

Each paediatric case that is submitted to the NFI receives a full body CT scan and a skeletal survey according to the guidelines of the Royal College of Radiology and Society and College of Radiographers [53]. A dedicated paediatric

radiologist discusses the results of the imaging with the pathologist prior to the autopsy. The radiologist and pathologist jointly make a decision on which fractures require further analysis during autopsy, for instance to confirm the presence of a fracture or for dating. All selected fractures are removed during autopsy, sometimes with the contralateral skeletal element for comparison. In some instances, this requires removal of the entire affected bone, although often excision of the fractured portion will suffice. After autopsy, fractures may be imaged a second time by conventional radiographs in two directions. There is a low threshold for radiological consultation for imaging directions or specific fields. The images are interpreted by a dedicated paediatric radiologist.

4.4.2 Histological Analysis

Each excised fracture is fixed in 4% formalin. As most fractures are too large for direct processing into paraffin embedded blocks, the fractures are sawn with a band saw. Preferably this results in one or more thick slices of about 4–5 mm thick, perpendicular to the fracture line (Fig. 4.9a, b). These thick slices are again formalin-fixed for at least another 24–48 h, decalcified in EDTA and processed into standard paraffin tissue blocks. Each block is used for at least one standard Haematoxylin and Eosin stained section, although various other stains may help to visualize the full spectrum of healing features. These may include Masson trichrome, Alcian blue, Von Kossa, period acid-schiff after diastase (PAS-D), Azan, and an iron stain. The histological interpretation is done by a dedicated histopathologist.

4.4.3 Interpretation

Interpretation of the radiological and histological images is guided by the context of the case and the forensic issue that is at stake. This issue forms the basis of two competing and mutually exclusive hypotheses, in line with the Bayesian approach that is now widely accepted as the standard of forensic evidence interpretation [54]. For example, if multiple rib fractures are found in a single individual, the issue at stake may be whether or not those fractures are related to a single event. The two competing hypotheses then could be: 'all rib fractures are explained by a single event' vs. 'the rib fractures are caused by at least two events'. In another example, a specific scenario may be put to the test. It may for instance be alleged that the fracture coincided with the death of the individual. In such a case the two competing hypotheses could be: 'the fracture is caused at the moment of death' vs. 'the fracture is caused prior to death'. The hypotheses are at the heart of the investigation and should be formulated carefully and preferably before the investigation.

A hypothesis-driven interpretation has several benefits over an approach in which each fracture is dated separately. First, it clearly defines the problem, providing clarity and focus. Second, it circumvents lengthy (and often impossible) determination of the age of each separate fracture. Rather, the aim is to describe to which extent the radiological and histological observations help to differentiate between the two competing hypotheses. In some cases, the radiological and histological observations may not be able to differentiate between the two hypotheses, in other words, the results of the analysis do not favour one hypothesis of the other. This may for instance be the case if two fractures show a similar healing phase and can therefore not be separated in time. This may also occur if the material is unsuited for analysis or there is insufficient scientific evidence to back up a statement. However, in other situations the results can provide useful information. For example, healing phases of two fractures can differ to such an extent that it becomes very unlikely that they occurred simultaneously. Or a fracture may show signs of re-fracture, indicating an initial and subsequent traumatic event. Or a fracture that was deemed to be perimortem may show signs of considerable healing. These examples

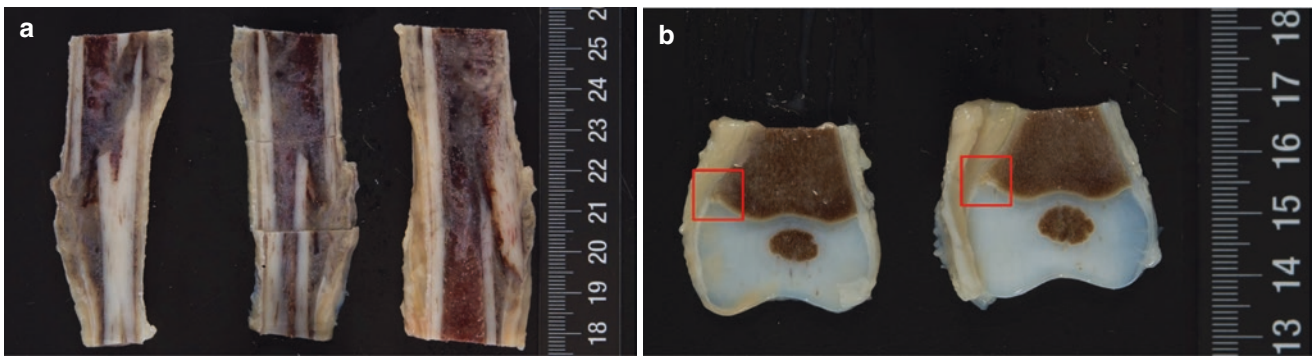


Fig. 4.9 Example of the excision of so-called 'thick slices' for the production of histological slides. Bar indicates centimetres. The fractured bone is initially sawn in halve, and subsequently lamellated in slices of approximately 3–4 mm thick. Careful photo documentation secures the chain of custody. (**a**) Healing tibial fracture; there is obvious displacement of the cortex and interposition of soft callus. (**b**) Distal metaphysis of a femur, suspected to have sustained a metaphyseal corner fracture (red box)

show that analysis of the healing response may still be useful, even if it remains impossible to put a specific date on a fracture.

The magnitude of the evidence, or the differentiating power of the observations, is expressed by the likelihood ratio. This likelihood ratio may be numerical, and then expresses the relative probability of the two hypotheses. A likelihood of twenty for instance indicates that the results are twenty times more likely when hypothesis 1 is true, than if hypothesis 2 is true. To indicate the uncertainties related to fracture dating, the likelihood ratio is usually expressed by a verbal term rather than by a number. As such, conclusions may for instance state that the findings provide 'weak', 'moderate', or 'strong' support for one hypothesis over the other. Each verbal term represents a range of numerical values and the use of a verbal term does require some uniformity on its meaning. More information on this can be found in the guidelines of the European Network of Forensic Science Institutes [55].

Eventually, it remains of great importance to realize that a statement on the age of a fracture require thorough knowledge of the biology of fracture repair, the affected individual, and the context of the case. As ever, it is best to err on the side of caution. Over-interpretation should be avoided.

4.4.4 Examples of Fracture Dating

Case 1: Two Posterior Rib Fractures

In a four-month-old infant, who died from extensive blunt force trauma to the head, two posterior rib fractures were also found. Due to the context of the case, the question arose as to these rib fractures could be related to the same event as the head trauma. Radiological analysis consisting of a skeletal survey (Fig. 4.10a) full body CT (Fig. 4.10b) did not show any healing features. At autopsy, the fractures showed haemorrhage, but no other signs of healing. Specimen radiographs showed recent posterior rib fractures (Fig. 4.10c) The ribs were excised (Fig. 4.10d) and processed into standard HE sections. Histological observation confirmed the presence of haemorrhage, no other signs of healing were noted (Fig. 4.10e). It was concluded that the radiological and histo-

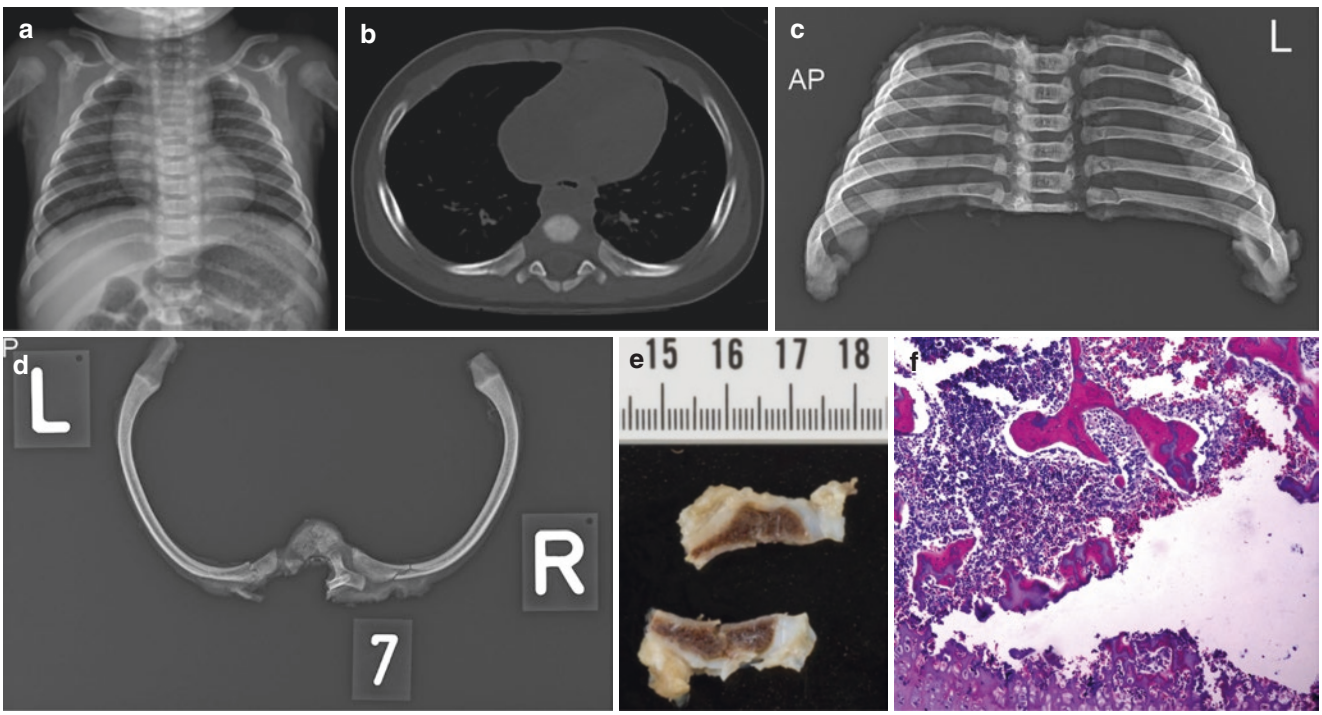


Fig. 4.10 (**a**) Post-mortem chest radiograph, on a diagnostic monitor posterior rib fractures there was as suggestion of left-sided posterior rib fractures of the seventh and eighth rib. (**b**) Post-mortem CT (axial image at the level of the seventh rib) showed bilateral posterior rib fractures. (**c**) Excised section of the chest (thoracic 3 to 8) shows bilateral posterior rib fractures of the seventh and eighth rib. (**d**) Detail radiograph of the seventh rib (note right and left are different from the specimen block) shows bilateral posterior rib fractures. Both fractures show no radiological signs of healing. (**e**) Excised and halved parts of a fractured posterior rib in a 4-month-old infant. No callus formation is visible. (**f**) Micrograph of a haematoxylin and eosin stained section of the fracture in the left panel. Besides minimal haemorrhage in the fracture cleft, no signs of vitality or healing are visible. This indicates that the fracture occurred during life, shortly (maximally several hours) before death

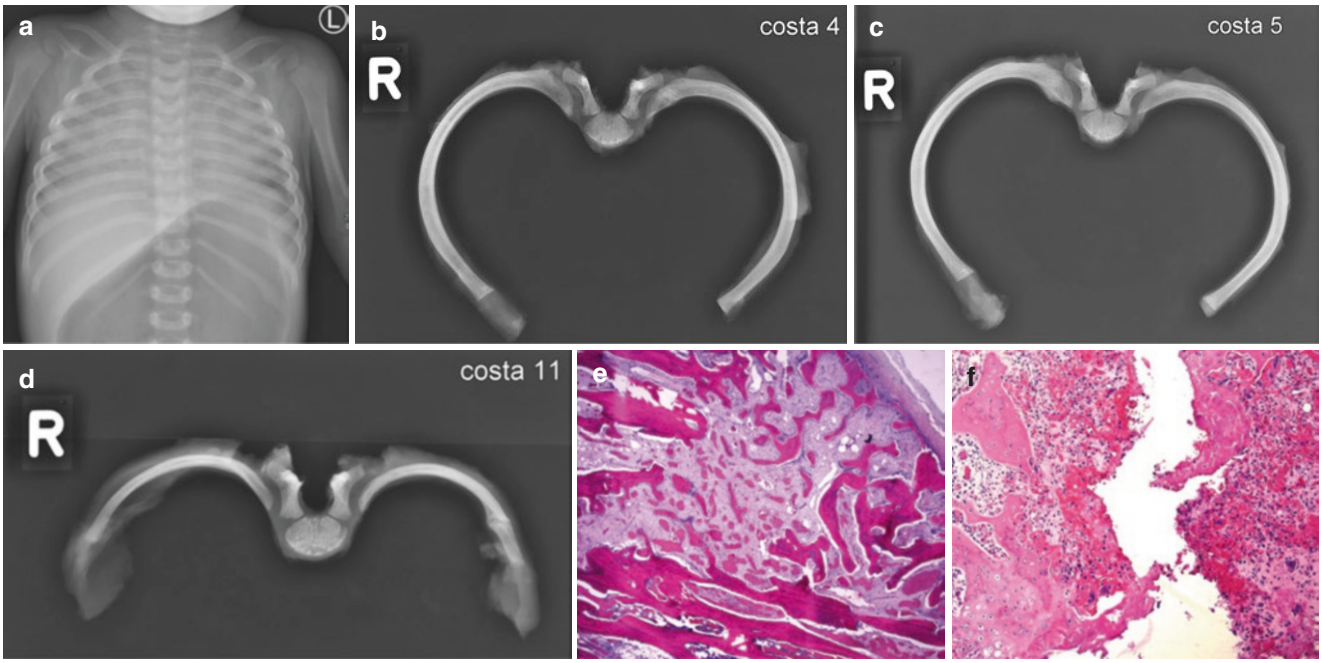


Fig. 4.11 (**a**) Post-mortem chest radiograph shows a healing rib fracture of the fifth rib right posterior and 11th rib left anterior. Specimen radiographs show (**b**) sclerotic posterior rib fracture of the fourth left rib, (**c**) healing rib fracture of the fifth rib right posterior, and (**d**) healing 11th rib left anterior. (**e**) Fifth rib right posterior fracture with advanced healing, as indicated by fibrous plug and extensive new bone formation. (**f**) Healing 11th rib left anterior in the same individual, also with advanced healing as indicated by the osteoclastic activity and copious new (woven) bone formation. However, the healing tissue is disrupted centrally, with haemorrhage and fibrin deposition, indicating a secondary traumatic event (re-fracture)

logical findings did not reveal any findings indicating a second traumatic event. It was stressed that these findings did not exclude a separate event shortly prior to the fatal head trauma.

Case 2: Multiple Rib Fractures

A 17-month-old infant was submitted for forensic autopsy. Skeletal survey (Fig. 4.11a) and full body CT (Fig. 4.11b) prior to the external and internal examination revealed multiple rib fractures, both anterior and posterior. The rib cage was excised for further analysis by specimen radiography and histology. The additional radiological analysis revealed a parietal linear skull fracture (Fig. 4.11c). Based on the radiology and autopsy results, various parts of the ribs were excised for histological analysis, to confirm the presence or absence of various regions suspected to be fractured, and to explore the extent in which the fractures could be related to a single event. Histology revealed several fractures that were not diagnosed at radiology or autopsy, especially at the costochondral junction. Besides rib fractures with advanced healing (Fig. 4.11d, left panel), also signs of recent trauma were found (Fig. 4.11e, right panel).

Case 3: A Skull Fracture

Histological analysis of a skull fracture, obtained at forensic autopsy. The sample was demineralized, embedded in paraffin and histologically stained with haematoxylin and eosin, Lawson Van Gieson and Masson Trichrome. An all over image of the fracture area with polarized light distinctly shows the discontinuation in the cranial vault (Fig. 4.12a). There is a connective-tissue plug visible in the fracture line. The middle-size enlarged view (Fig. 4.12b) clearly shows callus formation, only in a few places the callus is attached with sparse trabecular connections to the original cortex of the cranium. A detailed view shows the woven callus to be a deposit of primarily woven bone tissue (Fig. 4.12c). No remodelling into lamellar bone was observed. No blood extravasations and haemosiderophages were found, there was hardly any inflammatory infiltration. However, osteoclasts and their related Howship's lacunae were found at the fracture site (Fig. 4.12d). Although at autopsy the fracture was deemed perimortem, the histological observations indicate that that the fracture was sustained at least a couple of days before death.

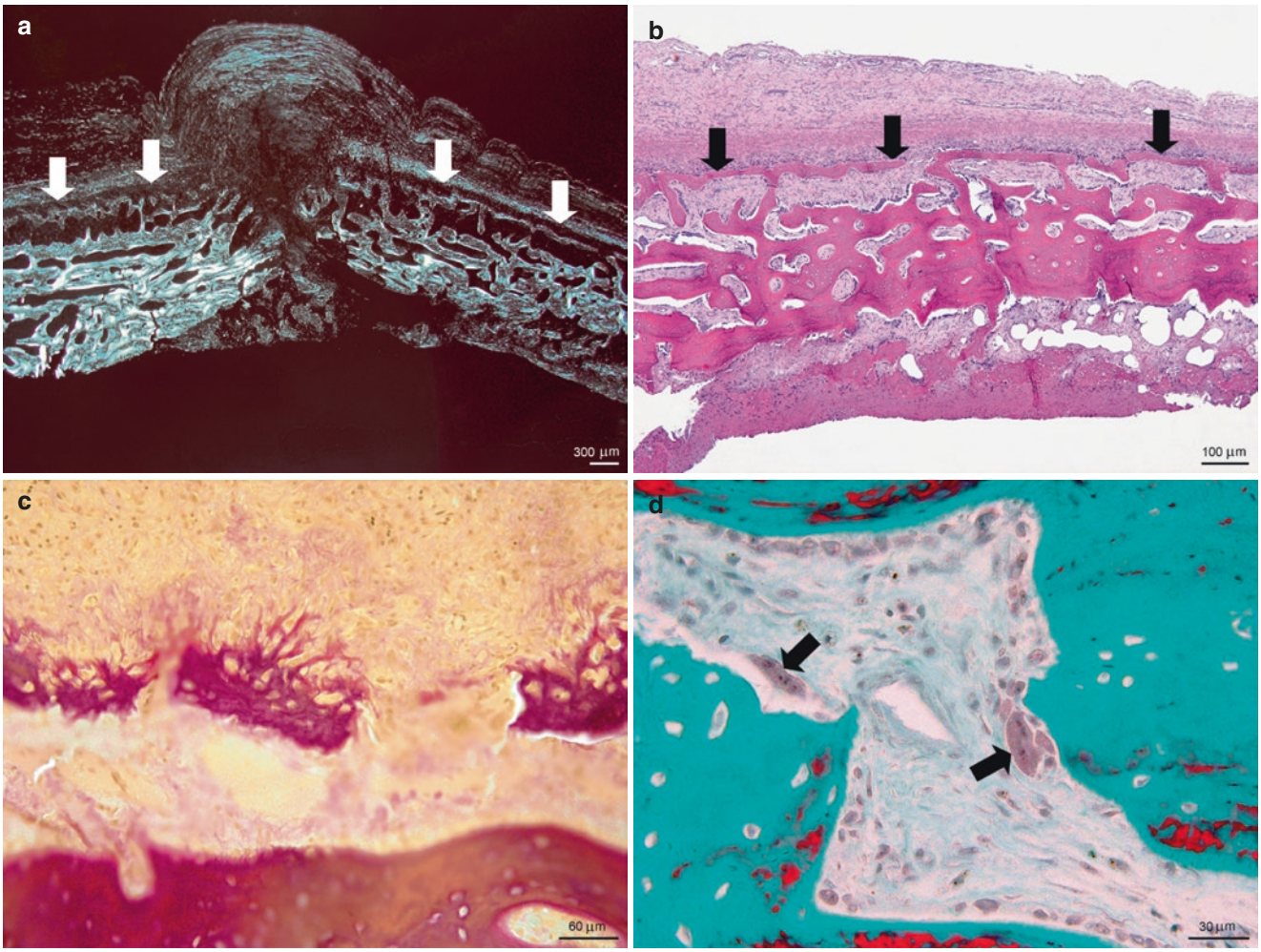


Fig. 4.12 (**a**) Discontinuity of the cranium with connective-tissue plug in the fracture cleft. Callus layer can still be separated from the marginal bone layer (arrows). Polarized light. (**b**) Detail of (**a**). Callus layer along the external trabecula of the cranium can still be separated (arrows). Haematoxylin-Eosin staining. (**c**) Depositions of primary bone tissue (disorganized), 'woven bones' in the callus layer. Lawson Van Gieson staining. (**d**) Howship's lacunae with multiple-nuclear osteoclasts in the fracture cleft (arrows). Trichrome staining according to Goldner

References

1. Taitz J, Moran K, O'Meara M (2004) Long bone fractures in children under 3 years of age: is abuse being missed in emergency department presentations? J Paediatr Child Health 40:170–174
2. Loi F, Córdova LA, Pajarinen J, Lin TH, Yao Z, Goodman SB (2016) Inflammation, fracture and bone repair. Bone 86:119–130
3. El-Jawhari JJ, Jones E, Giannoudis PV (2016) The roles of immune cells in bone healing; what we know, do not know and future perspectives. Injury 47:2399–2406
4. Frost HM (1989) The biology of fracture healing. An overview for clinicians. Part II. Clin Orthop Relat Res 00:294–309
5. Frost HM (1989) The biology of fracture healing. An overview for clinicians. Part I. Clin Orthop Relat Res 00:283–293
6. Marsell R, Einhorn TA (2011) The biology of fracture healing. Injury 42:551–555
7. Einhorn TA, Gerstenfeld LC (2015) Fracture healing: mechanisms and interventions. Nat Rev Rheumatol 11:45–54
8. Malcolm AJ (2008) Examination of fractures at autopsy. In: Rutty G (ed) Essentials of autopsy practice. Springer, London, pp 23–44
9. Hobbs CJ (1989) ABC of child abuse fractures. BMJ 298:1015–1018
10. O'Connor JF, Cohen J (1998) Dating fractures. In: Kleinman PK (ed) Diagnostic imaging of child abuse. Mosby, St. Louis, pp 168–177
11. Harwood PJ, Newman JB, Michael ALR (2010) An update on fracture healing and non-union. Orthop Trauma 24:9–23
12. Perren SM (1979) Physical and biological aspects of fracture healing with special reference to internal fixation. Clin Orthop Relat Res 00:175–196
13. Malone CA, Sauer NJ, Fenton TW (2011) A radiographic assessment of pediatric fracture healing and time since injury. J Forensic Sci 56:1123–1130
14. Cheung WH, Miclau T, Chow SK, Yang FF, Alt V (2016) Fracture healing in osteoporotic bone. Injury 47(Suppl. 2):S21–S26
15. Dreizen S, Spirakis CN, Stone RE (1964) The influence of age and nutritional status on 'bone scar' formation in the distal end of the growing radius. Am J Phys Anthropol 22:295–305

16. Gorter EA, Hamdy NA, Appelman-Dijkstra NM, Schipper IB (2014) The role of vitamin D in human fracture healing: a systematic review of the literature. Bone 64:288–297
17. Michael AR, Bengtson JD (2016) Chronic alcoholism and bone remodeling processes: Caveats and considerations for the forensic anthropologist. J Forensic Legal Med 38:87–92
18. Kurmis AP, Kurmis TP, O'Brien JX, Dalen T (2012) The effect of nonsteroidal anti-inflammatory drug administration on acute phase fracture-healing: a review. J Bone Joint Surg Am 94:815–823
19. Giannoudis PV, Hak D, Sanders D, Donohoe E, Tosounidis T, Bahney C (2015) Inflammation, bone healing, and anti-inflammatory drugs: an update. J Orthop Trauma 29(Suppl. 12):S6–S9
20. Prosser I, Lawson Z, Evans A, Harrison S, Morris S, Maguire S, Kemp AM (2012) A timetable for the radiologic features of fracture healing in young children. AJR Am J Roentgenol 198:1014–1020
21. Prosser I, Maguire S, Harrison SK, Mann M, Sibert JR, Kemp AM (2005) How old is this fracture? Radiologic dating of fractures in children: a systematic review. AJR Am J Roentgenol 184:1282–1286
22. Cumming WA (1979) Neonatal skeletal fractures. Birth trauma or child abuse? J Can Assoc Radiol 30:30–33
23. Fadell M, Miller A, Trefan L, Weinman J, Stewart J, Hayes K, Maguire S (2017) Radiological features of healing in newborn clavicular fractures. Eur Radiol 27:2180–2187
24. Halliday KE, Broderick NJ, Somers JM, Hawkes R (2011) Dating fractures in infants. Clin Radiol 66:1049–1054
25. Islam O, Soboleski D, Symons S, Davidson LK, Ashworth MA, Babyn P (2000) Development and duration of radiographic signs of bone healing in children. AJR Am J Roentgenol 175:75–78
26. Sanchez TR, Nguyen H, Palacios W, Doherty M, Coulter K (2013) Retrospective evaluation and dating of non-accidental rib fractures in infants. Clin Radiol 68:e467–e471
27. Walters MM, Forbes PW, Buonomo C, Kleinman PK (2014) Healing patterns of clavicular birth injuries as a guide to fracture dating in cases of possible infant abuse. Pediatr Radiol 44:1224–1229
28. Yeo LI, Reed MH (1994) Staging of healing of femoral fractures in children. Can Assoc Radiol J 45:16–19
29. Hosokawa T, Yamada Y, Sato Y, Tanami Y, Oguma E (2017) Subperiosteal new bone and callus formations in neonates with femoral shaft fracture at birth. Emerg Radiol 24:143–148
30. Warner C, Maguire S, Trefan L, Miller A, Weinman J, Fadell M (2017) A study of radiological features of healing in long bone fractures among infants less than a year. Skelet Radiol 46:333–341
31. 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
32. Crompton S, Messina F, Klafkowski G, Hall C, Offiah AC (2021) Validating scoring systems for fracture healing in infants and young children: pilot study. Pediatr Radiol 51(9):1682–1689
33. Steyn M, De Boer HH, Van der Merwe AE (2014) Cranial trauma and the assessment of posttraumatic survival time. Forensic Sci Int 244:e25–e29
34. Drury A, Cunningham C (2018) Determining when a fracture occurred: does the method matter? Analysis of the similarity of three different methods for estimating time since fracture of juvenile long bones. J Forensic Legal Med 53:97–105
35. Messer DL, Adler BH, Brink FW, Xiang H, Agnew AM (2020) Radiographic timelines for pediatric healing fractures: a systematic review. Pediatr Radiol 50:1041–1048
36. Klotzbach H, Delling G, Richter E, Sperhake JP, Puschel K (2003) Post-mortem diagnosis and age estimation of infants' fractures. Int J Legal Med 117:82–89
37. De Boer HH, Van der Merwe AE, Hammer S, Steyn M, Maat GJ (2012) Assessing post-traumatic time interval in human dry bone. Int J Osteoarch 24:98–109
38. Phillips AM (2005) Overview of the fracture healing cascade. Injury 36(Suppl. 3):S5–S7
39. Barnes GL, Kostenuik PJ, Gerstenfeld LC, Einhorn TA (1999) Growth factor regulation of fracture repair. J Bone Miner Res 14:1805–1815
40. Einhorn TA (1998) The cell and molecular biology of fracture healing. Clin Orthop Relat Res 1998:S7–S21
41. Brighton CT, Hunt RM (1991) Early histological and ultrastructural changes in medullary fracture callus. J Bone Joint Surg Am 73:832–847
42. Lu C, Miclau T, Hu D, Hansen E, Tsui K, Puttlitz C, Marcucio RS (2005) Cellular basis for age-related changes in fracture repair. J Orthop Res 23:1300–1307
43. Nunamaker DM (1998) Experimental models of fracture repair. Clin Orthop Relat Res 1998:S56–S65
44. Wingate Todd T, Barber CG (1934) The extend of skeletal change after amputation. J Bone Joint Surg Am 16:53–64
45. Todd TW, Iler DH (1927) The phenomena of early stages in bone repair. Ann Surg 86:715–736
46. Barber CG (1929) Immediate and eventual features of healing in amputated bones. Ann Surg 90:985–992
47. Barber CG (1930) The detailed changes characteristic of healing bone in amputation stumps. J Bone Joint Surg Am 12:353–359
48. Barber CG (1934) Ultimate anatomical modifications in amputation stumps. J Bone Joint Surg Am 16:394–400
49. Maat GJ, Huls N (2010) Histological fracture of fresh and dried bone. In: Bilo RA, Robben SG, Van Rijn RR (eds) Forensic aspects of paediatric fractures. Springer, Berlin, pp 194–201
50. De Boer HH, Van der Merwe AE, Maat GJ (2016) Survival time after fracture or amputation in a 19th century mining population at Kimberley, South Africa. In: Steyn M, Morris AG, Maat GJ, Morongwa NM (eds) Skeletal identity of Southern African populations: lessons from outside South Africa. South African Archaeological Society, pp 52–60
51. Cappella A, de Boer HH, Cammilli P, De Angelis D, Messina C, Sconfienza LM, Sardanelli F, Sforza C, Cattaneo C (2019) Histologic and radiological analysis on bone fractures: estimation of posttraumatic survival time in skeletal trauma. Forensic Sci Int 302:109909
52. Naqvi A, Raynor E, Freemont AJ (2019) Histological ageing of fractures in infants: a practical algorithm for assessing infants suspected of accidental or non-accidental injury. Histopathology 75:74–80
53. The Royal College of Radiologists (RCR) and the Society and College of Radiographers (SCoR) (2017) The radiological investigation of suspected physical abuse in children. https://www.rcr.ac.uk/publication/radiological-investigation-suspected-physical-abuse-children. Accessed 5 July 2021
54. Robertson B, Vignaux GA, Berger CEH (2016) Interpreting evidence: evaluating forensic science in the courtroom. Wiley, Hoboken, NJ
55. European Network of Forensic Science Institutes (2015) ENFSI guideline for evaluative reporting in forensic science. https://enfsi.eu/wp-content/uploads/2016/09/m1_guideline.pdf. Accessed 5 July 2021

Head

5

Rob A. C. Bilo, Simon G. F. Robben, and Rick R. van Rijn

Contents

R. A. C. Bilo (✉)
Veilig Thuis Rotterdam Rijnmond (Center for the Reporting of Child Abuse, Domestic Violence and Elder Abuse), Rotterdam, The Netherlands

S. G. F. Robben
Department of Radiology, Maastricht University Medical Center, Maastricht, The Netherlands
e-mail: s.robben@mumc.nl

R. R. van Rijn
Department of Radiology and Nuclear Medicine, Amsterdam UMC, University of Amsterdam, Amsterdam, The Netherlands

Department of Forensic Medicine, Netherlands Forensic Institute, The Hague, The Netherlands
e-mail: r.r.vanrijn@amsterdamumc.nl

R. A. C. Bilo et al. (eds.), *Forensic Aspects of Paediatric Fractures*, https://doi.org/10.1007/978-3-031-12041-1_5

5.1 General Aspects of Head Injuries

> Any blow to the head can transfer energy from the skin, through the skull and meninges, to the brain. When evaluating head trauma, the clinician should remember the anatomic layers of the head that may be affected: the skin, galea aponeurotica, periosteum, cranial bone, epidural space, dura mater, subdural space, arachnoid mater, subarachnoid space, and brain.
>
> Atabaki SM. [1]

5.1.1 Introduction

Head injuries are defined as injuries to the soft tissues of the orofacial region and the scalp and/or the bony tissues of the skull (calvarium, base, and orofacial bones) and/or the intracranial contents (meninges, brain) due to mechanical or non-mechanical trauma.

Most head injuries in paediatric patients are caused by mechanical trauma (Sect. 1.5.2), irrespective of whether these injuries concern soft tissues, bony tissues, or the intracranial content. On rare occasions paediatric head injuries, especially cutaneous injuries, are caused by thermal trauma, chemical trauma, electrical trauma, or electromagnetic and ionizing trauma (Table 1.4).

5.1.2 Injuries to the Scalp and the Bony Tissues of the Skull

In this chapter we will focus on injuries, due to mechanical trauma, to the scalp (Sect. 5.2) and the bony tissues of the skull (Sects. 5.3, 5.4, and 5.5). The reader is referred to Sect. 6.4, concerning fractures in the neck region (cervical vertebrae).

Mechanical trauma is caused either by static loading or by dynamic loading [2, 3].

In forensic medicine, static loading of the head is defined as a relatively slow exertion of forces on the head over a protracted period of time (more than 200 ms). This occurs when the head is squeezed and/or compressed, which may lead to injuries of soft tissues, bony tissues, or sometimes to the intracranial content. The results of static loading can be focal and limited to the scalp at the point of compression or more extended in which also the underlying layers (skull and intracranial structures) are damaged.

Dynamic (or rapid) loading is the impact of forces over a shorter period (<200 ms, often even less than 50 ms). Dynamic (or rapid) loading can be subdivided into two subtypes: dynamic 'impact' loading, and dynamic 'impulse' loading.

In dynamic impact loading of the head there are three possible situations:

- The head is stationary, while the impacting object is impacting against the head.
- The impacting head is moving, while the impacted object is stationary.
- Both the head and the object are moving in the same direction or in opposite directions.

The impact loading results in a momentary deformity of the head with possible damage to the soft tissues, bony tissues, or the intracranial content.

Dynamic impulse loading (inertial trauma) is the result of fast movements without impact (fast alternation of acceleration and deceleration, e.g. in shaking) of the head, which will mainly result in intracranial injuries and injuries to the spinal cord.

Injuries to the head may occur before, during, and after birth. The circumstances, under which injuries occur, can be either accidental or non-accidental.

The cause and manner of injuries to soft tissues of the scalp and of injuries to bony tissues will be discussed in the following sections.

5.1.3 Injuries to the Orofacial Soft Tissues and the Intracranial Content

Orofacial soft tissue injuries and injuries to the intracranial content are probably equally important as injuries to the scalp and the bony tissues of the skull, when evaluating head trauma. This was clearly highlighted by Atabaki [1].

Although these injuries are beyond the scope of this book, a short overview of inflicted orofacial soft tissue injuries is given in this section. An overview of intracranial injuries is given in Sect. 5.6.

The head is the least protected and the most vulnerable part of the body in case of trauma, irrespective of the circumstances (accidental or non-accidental) and irrespective of the age of the victim.

In 1946, Caffey was the first to report the relation between the occurrence of multiple fractures of the long bones and subdural hematoma in young children [4]. He suspected this combination to be of a traumatic origin. Three of the children, who were described by Caffey, also showed intraoral injuries. In 1966, Cameron et al. described 29 cases of fatal child abuse (mean age 14.5 months) [5]. Around 50% of the children had clearly visible abrasions, bruises, and bumps on the head, face, and neck (Table 5.1). Since the articles of Caffey and Cameron et al.there has been a plethora of publications on the subject of inflicted injuries in the head and neck region. Most of these publications point to the frequent occurrence of these injuries.

Several studies have shown that more than 45% of all children who sustained inflicted injuries (non-accidental circumstances, child abuse) have orofacial injuries [5–16]. The orofacial region is probably even the most frequently injured part of the body, due to non-accidental trauma [6, 12, 17, 18]. Around 75% of inflicted injuries in the orofacial region are found in children under the age of 3 years [17].

Most commonly reported inflicted orofacial soft tissue injuries are (often easy to identify) bruises, e.g. slap marks or pinch marks. Also bite marks, abrasions, and lacerations of lips and frenulum, burn injuries, and injuries to the ears can be found [9, 19].

Probably the main reason for the high incidence of inflicted injuries in this region, is that the head, and in particular the face, is the most visible part of the body by which someone is recognized and defined as a person. Moreover, human behaviour and emotions are recognized and interpreted through facial expressions. Therefore, physical aggression is mainly directed to this part of the body. In children this plays an even a greater role. When a child cries in a stressful situation, aggression may be directly targeted towards the face in general and the mouth in particular. Various authors are even of the opinion that the oral cavity is the most important target in physical assaults towards children because of the role of the mouth in feeding and communication, for example crying behaviour or unwanted verbal reactions of the child [18, 20].

Table 5.1 Injury location in 29 cases of fatal child abuse (irrespective of type of injury) [5]

Location	Percentage
Skull	79
Neck	52
Maxilla	49
Mandible	48
Upper lip	45
Frenulum	45

5.2 Injuries of the Scalp

5.2.1 Introduction

5.2.1.1 Anatomy of the Scalp

The scalp is that part of the skin of the head that extends from the external occipital protuberance and superior nuchal lines to the supraorbital margins. It forms a strong cover over the skull [21]. Compared to adults, the scalp of a young child is relatively thin. It consists of five layers: the skin (epidermis and dermis), a layer of dense connective tissue, a layer of strong fibrous tissue (galea aponeurotica), a layer of loose areolar connective tissue and the pericranium (periosteum) (Figs. 5.1 and 5.2) [21]. The superficial layers (skin, connective tissue, and galea aponeurotica) are firmly bound together and act as a single tissue layer. The layer of loose connective tissue allows the superficial layers to slide over the, firmly attached to the skull, epicranial aponeurosis.

5.2.1.2 Scalp Injuries

There are no reliable data about how often children sustain injuries to the scalp, although one may assume that minor head trauma due to accidental falls or other minor accidents happens on a daily base in mobile (young) children. Most commonly minor accidents will occur without causing significant and/or visible injuries (or skull fractures). As a result most of these children will not be examined by a doctor. More significant or visible bruising of the scalp is uncommon without significant head trauma or coexisting disorders like coagulation disorders, except in case of a subgaleal haematoma, which can be sustained due to a minor trauma [22].

5.2.1.3 Cause and Manner of Scalp Injuries

Most injuries to the scalp are caused by mechanical trauma, either static loading or dynamic impact loading (Sects. 1.5.2 and 5.1.2), followed by thermal trauma. Chemical trauma, electrical trauma, or electromagnetic and ionizing trauma of the scalp is very rare in paediatric patients. Irrespective of the cause of the scalp injuries, the manner of scalp injuries in children is either accidental or non-accidental. In this section, only injuries caused by mechanical trauma are discussed.

Static loading of the scalp occurs when the head is compressed. The results of static loading can be focal and limited to the scalp at the point of compression or more extended in which the underlying layers (skull and intracranial structures) are also damaged. Static loading may occur during childbirth or accidentally during traffic accidents, when the head is wedged for a period of time. It may also be inflicted when the head is violently squeezed during physical abuse.

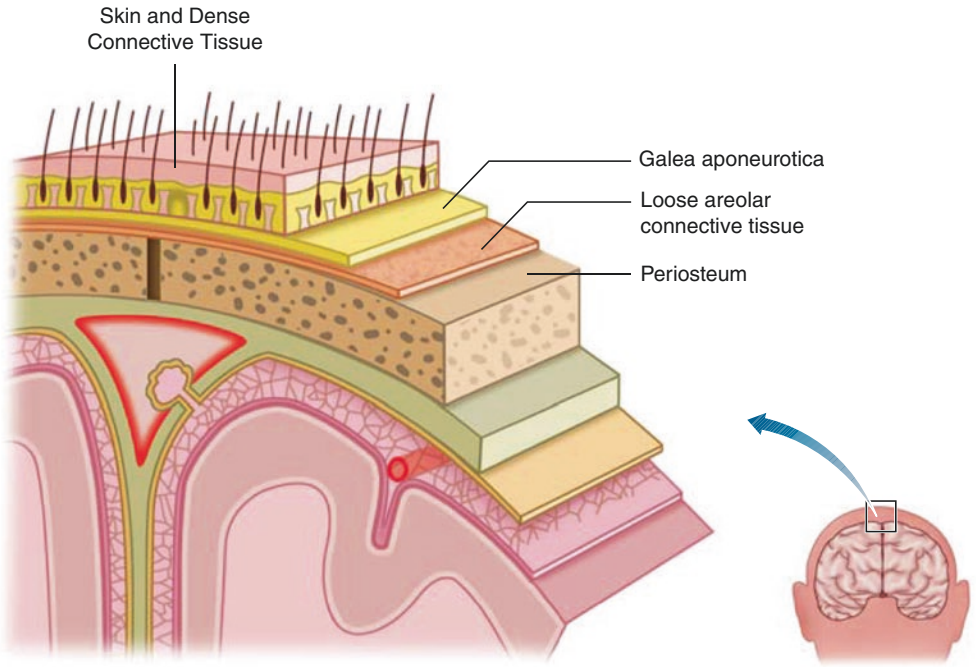


Fig. 5.1 Anatomy of the scalp

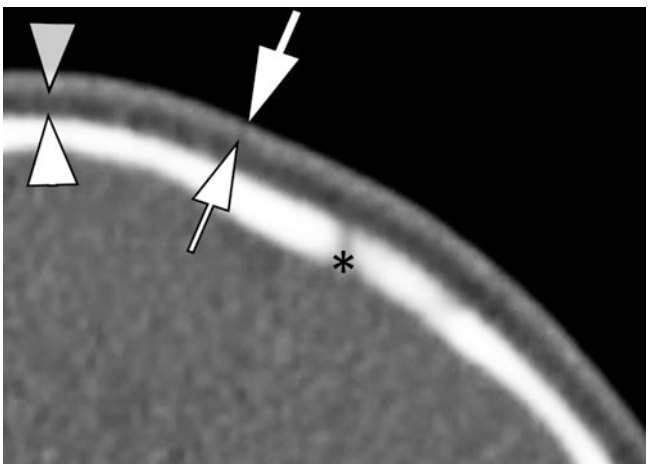


Fig. 5.2 Normal appearance of scalp on CT. Skin (between arrows) and hypodens subcutaneous fatty connective tissue (between arrowheads). Galea aponeurotica and periosteum are not visible. Asterisk = suture

Dynamic loading of the scalp usually consists of dynamic or rapid impact loading (Sect. 5.1.2). This results in deformation of the scalp and the head and may lead to damage to the scalp, skull, and/or intracranial structures. Dynamic impact loading may occur accidentally or may be inflicted. Inflicted impact loading is seen, e.g. when a child is hit with a hand or an object (moving object against stationary head) or by dropping or throwing the child on the floor or throwing the child against an object or a wall (moving head against stationary object). Dynamic loading of the scalp can also occur when traction is applied to the skin. This can occur due to either quick or continued slow hairpulling.

Scalp injuries in non-mobile children are most often sustained in non-accidental circumstances (inflicted injuries). In children under 2 years of age with limited mobility scalp injuries are most often sustained in accidental circumstances, usually accidental short-distance falls, e.g. from standing height, beds, or changing tables [23, 24]. The falling height will increase, when the child gets older, becomes more mobile and learns to climb. The risk of complicated accidents, e.g. due to toppling televisions, also increases. In older children (traffic accidents with pedestrian, bicycle, and motor vehicle) become more common [25].

5.2.1.4 Type and Severity of Scalp Injuries

The type and severity of scalp injuries are determined by the type of force applied to the scalp (= mechanical trauma), the amount of force, the angle at which the force is applied (perpendicular, tangential, or parallel to the surface of the scalp), and the type and surface of the object that strikes the head. Injuries may occur in only one layer, multiple layers, or in all layers plus the underlying bone structures of the skull and intracranial structures, like the dura and the brain.

5.2.2 Cutaneous and Subcutaneous Injuries of the Scalp

5.2.2.1 Introduction

Damage to one or all layers of the scalp is almost always the result of dynamic impact loading. The skin, however, may remain intact, in spite of damage to the deeper layers. Injuries

that can be found in the different layers are bruises, abrasions, lacerations and avulsions, subgaleal haematomas, and cephalhaematomas (Sect. 5.2.4).

5.2.2.2 Bruises

Bruising of the scalp is often not externally visible, unless there is at least some swelling [26]. During trauma the soft and pliable infant scalp and skull may remain intact, in spite of damage to the deeper layers. This is irrespective of the cause (static or dynamic impact loading) or the circumstances, making the identification of injuries difficult by visual inspection only. Bruising of the scalp may go unnoticed for hours to days in a child. Sometimes the deeper damage to the scalp is only found at autopsy [27, 28]. In other words, the absence of visible injuries in (living) children does not exclude head trauma [26, 28].

Bruising or swelling of the scalp can occur during and after birth. Birth-related bruising and swelling, e.g. a caput succedaneum or a swelling, induced by vacuum extraction, will rarely if ever lead to a suspicion of inflicted bruises. Bruising and swelling of the scalp, due to trauma after birth, can occur in accidental and non-accidental circumstances. Bruising of the scalp should be differentiated from, e.g. extensive Mongolian spots with involvement of the scalp [29]. Thakur and Kaplan described in an 18-month-old child the recurrent and unexplained swelling of different parts of the face and scalp, which initially led to a suspicion of inflicted blunt head trauma (child abuse), but finally was diagnosed as an atypical presentation of angioedema without occurrence of the swelling on other parts of the body and without urticarial [30].

5.2.2.3 Abrasions, Lacerations, and Avulsions

General Aspects

The scalp is extensively vascularized. The vessels are fixed within the scalp and are not able to retract when lacerated [31]. The fibrous fascia prevents vasoconstriction [21]. As a result of this, even minor scalp lacerations may frequently bleed profusely and lead to major blood loss or even haemorrhagic shock, particularly in children [21, 32]. Lacerations (or incision wounds) superficial to the galea show much less diastasis than injuries that cut through it because the galea holds the skin tight [21]. Open wounds like lacerations or incisions are a potential point of entry for infection, especially when associated with a skull fracture [32]. The veins do not have valves and open in the loose connective tissue beneath the galea. In this way, an infection can be transmitted from the scalp to the cranial cavity [21]. Total or partial avulsions of the scalp are rare in paediatric patients.

Abrasions, lacerations, and avulsions of the scalp can be sustained during birth, e.g. because of the placement or the removal of a scalp electrode [33]. Lacerations of the scalp can also occur due to other medical procedures during birth (mechanical birth-associated trauma) [34]. Teng and Sayre described the occurrence of superficial abrasions due to the use of a vacuum extractor [35]. Scalp lacerations and scalp incised wounds (mostly mild, very rarely severe) have been reported to occur during caesarean section [36–39]. Mathur and Marcus described the occurrence of a scalp laceration in a neonate due to the presence of infected cervical cerclage sutures at 20 weeks of gestation [40].

Trauma After Birth: Accidental Circumstances

After birth abrasions, lacerations, and avulsions of the scalp can occur due to accidental and non-accidental circumstances. Scalp injuries due to accidental circumstances have regularly been described:

- Mayr et al. described the findings in 103 children with highchair-related injuries: 68.9% of the children had sustained a simple contusion of the head or lacerations to the scalp or face, 15.5% a skull fracture, 13.6% a brain concussion, and 2.0% limb fractures [41].
- Alias et al. analyzed head injuries caused by fan blades in 14 children (mean age 7.9 years; range 1.0–12.2 years; most often school-aged boys) [42]. The circumstances under which the injuries were sustained included jumping on the upper bunk of a bunk bed, climbing on a ladder, climbing up onto a table, and being lifted by an adult. The injuries consisted of scalp lacerations, compound depressed fractures and multiple intracranial haemorrhages. One child died from severe head injuries.
- Agrawal et al. described two children with partial scalp avulsions sustained in accidental circumstances: a 4-year-old girl and a 1½-year-old boy [43]. The girl supposedly hit her head against the table, while playing at home. She sustained a partial avulsion of the scalp. There were no other physical findings. The boy fell off an auto rickshaw. He had a transient loss of consciousness and two episodes of vomiting. He also sustained a linear fracture of the occipital bone. There was no intracranial injury. There were no other findings.
- Scalp avulsions can also occur in paediatric patients with long hair, if the hair is caught in mechanical equipment, e.g. an uncovered farm engine, a tractor, or in a go-kart motor belt [44–48].

Trauma After Birth: Non-accidental Circumstances

Probably the most common non-accidental circumstances, resulting in abrasions, lacerations, and avulsions of the scalp in paediatric patients, are dog bites [48, 49]. Dog bites occur frequently and the head and neck are most frequently involved in paediatric patients [49–51]. According to Ng et al. the combination in a dog bite of crushing, tearing, and

perforation may result in scalp avulsion in severe cases [49]. Dog bites, penetrating the cranial vault, occur only occasionally and may go unnoticed on initial examination because penetrating wounds of the calvarium can be masked as a result of scalp displacement at the time of trauma and normalization of the scalp shape after penetration [50].

5.2.2.4 Traction Alopecia

Hair loss or baldness (alopecia) in children can result from many congenital and acquired (psycho)dermatological disorders, which in most cases are benign, self-limiting, and non-scarring and from trauma [52–56].

Alopecia may also result from trauma, e.g. traction alopecia or trichotillomania. The traction to the hair is applied either suddenly and in a short time (dynamic loading by rapid hair pulling) or continuously/chronically during a longer period (more or less static loading by continuous/chronic hair pulling). Chronic traction may lead to permanent alopecia.

Traumatic alopecia is most often seen in children, adolescents, and young adults [54, 57]. The alopecia can be self-inflicted or inflicted by others, e.g. parents, caregivers, or other children. In most paediatric cases traumatic alopecia will be unintentional (Table 5.2). Intentional violent hair pulling *however may result in traumatic alopecia and subgaleal bleeding* [61, 62]. *Violent hair pulling may in some cases lead to a massive subgaleal bleeding without creating alopecia* [63]. *DeRidder and Berkowitz described a toddler, who was admitted to the hospital because of* nonspecific gastrointestinal complaints, caused by pancreatitis, due to a pancreatic transection [64]. The toddler also had hair loss, most probably due to traction alopecia. No history of any witnessed trauma was given, and it was decided that the transection and the alopecia were inflicted.

5.2.2.5 Scalp Injuries, Skull Fractures and Intracranial Injuries

The scalp protects the skull against fracturing. Tedeschi showed that the risk of fracture of the skull increases tenfold when no scalp is present [65]. In accidental falls bruises of the scalp (and other injuries in the face) are seen regularly without being accompanied by skull fractures [66–71].

In general, fracture-related bruising is rare in children [72]. In skull fractures, however, fracture-related bruising/soft tissue swelling is relatively common [73, 74]. Scalp bruising and other scalp injuries may indicate skull fractures and focal or diffuse intracranial injuries, especially large and non-frontal (temporoparietal and occipital) scalp haematomas [75]. Peters et al. found scalp bruising or subgaleal bleeding near the site of a skull fracture in 43% of children with inflicted fractures [72]. Metz et al. evaluated the findings in 218 children under the age of 4 years (two-thirds of the children under the age of 1 year) and found clinically apparent soft tissue swelling in 73% of children with skull fractures [73]. They found radiologically apparent fracture-associated soft tissue swelling in 93% of the children with skull fractures.

In young children under the age of 2 years with a scalp injury, when there are no neurological symptoms, it is from a clinical viewpoint rarely indicated to perform diagnostic imaging, such as radiography or CT. From a forensic point of view, however, additional imaging, e.g. a skeletal survey and CT or MRI is indicated, if a plausible accidental explanation is lacking (see Chap. 3). This also applies when there are no neurological symptoms [76].

Table 5.2 Differential diagnosis of traumatic alopecia [58–60]

	Behaviour
Cultural, social, and cosmetic practices	Unintentional: • Certain styles of hairdressing (e.g. tight ponytails or tight braids)
Physiological habits	Unintentional: • Hair twirling, twisting, stroking, and pulling in infants and toddlers (habitual activity, like nail biting, and thumb sucking)
Habit disorders	Intentional or unintentional: • Self-mutilation—trichotillomania (self-inflicted hair loss)
Accidental trauma	Unintentional
Non-accidental trauma	Intentional: • Physical abuse—hair pulling • Fabricated or induced illness by caretakers

5.2.3 Subgaleal Haematoma

5.2.3.1 Introduction

A subgaleal haematoma (a.k.a. subaponeurotic haematoma) is a haematoma in the loose connective tissue between the galea aponeurotica and the periosteum of the skull (Fig. 5.3). The galea aponeurotica is located over the entire calvarium. The possible spreading of subgaleal blood is shown in Fig. 5.4. A subgaleal haematoma is in fact an abnormal accumulation of blood in a space which in physiological circumstances does not exist.

Most cases of subgaleal haematoma are reported in neonates [77]. The most common risk factors are instrumental delivery, prolonged second stage of labour, precipitate labour, coagulopathy, prematurity, large infants, foetal dystocia, and severe head moulding [78]. Subgaleal haematoma occurring beyond the neonatal period is rare [79]. If a child sustains a subgaleal haematoma, clinical symptoms do not have to occur immediately. Young children can show irritability, while older children can complain about headaches. In most cases, there will be an insidious onset of swelling of the scalp [79]. It may take up to 14 days before enough blood has collected in the subgaleal space to be visible or palpable to a parent/caregiver or during a physical examination by a doctor [79, 80]. Bleeding in the subgaleal space can lead to visible bruises

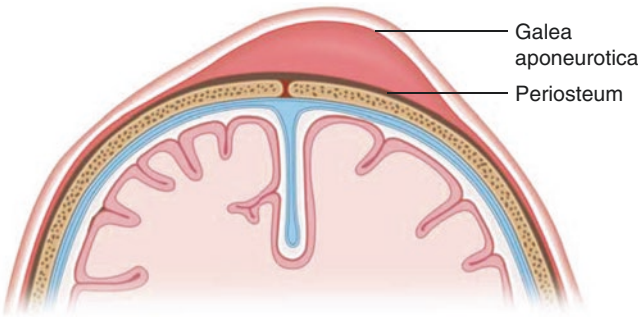


Fig. 5.3 Subgaleal haematoma. These haematomas are not confined to individual bones and therefore may cross sutures and fontanelles

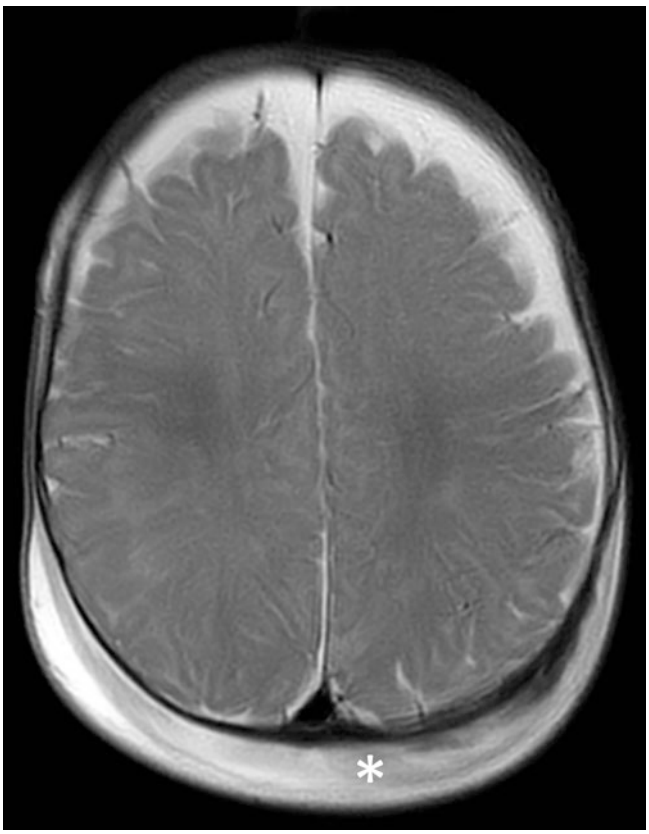


Fig. 5.4 T2-weighted MRI of a 6-month-old boy who had a dynamic impact trauma to the occipital skull. The subgaleal haematoma (asterisk) has mixed signal intensities and crosses the sagittal suture

around the eyes, behind the ears, from the helix of the ear (rim of the ear cup), and in the neck. Subgaleal bleeding may exist without externally visible bruising [62]. In that case, the subgaleal bleeding can be recognized by the presence of a swelling that can be felt by palpation of the hairy scalp.

In most children treatment will not be necessary, the swelling will resolve without treatment in a few days up to 5 weeks without complications, if there is no underlying medical condition [79, 81, 82].

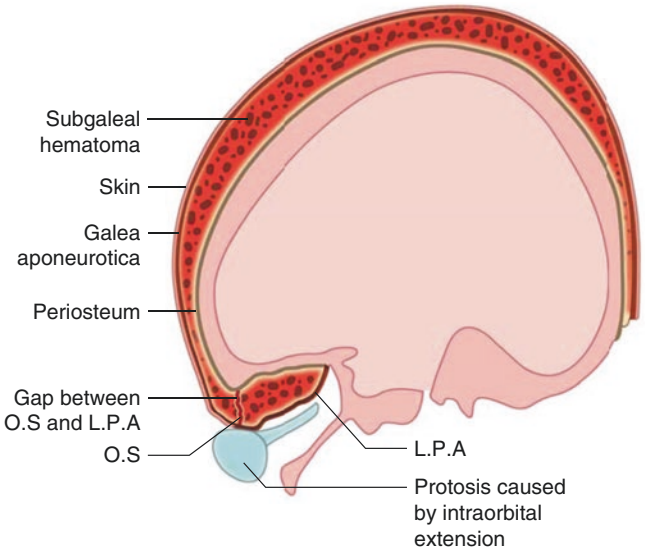


Fig. 5.5 Intraorbital extension of a large subgaleal haematoma through a gap between the orbital septum (O.S) and the levator palpebral aponeurosis (L.P.A), causing proptosis

Only rarely severe complications may occur. Because the potential subgaleal space basically covers the entire calvarium, a large amount of blood can accumulate in the space with only a limited increase in head circumference [80, 83]. This may lead to severe anaemia, an acute haemorrhagic shock on the basis of blood loss in subgaleal space (e.g. up to 50% of the total blood volume can be lost in the subgaleal space in a newborn), icterus due to significant hyperbilirubinemia and even death if it is not diagnosed on time [84–88]. Sometimes the haematoma migrates into the orbit, which can result in proptosis/exophthalmos (Fig. 5.5), and behind the zygoma to the neck on both sides [89–91]. If the blood migrates to the neck on both sides, tracheal compression can occur, even resulting in severe cases of obstruction and compromised breathing [90]. Local obstruction of the blood flow can also occur as a complication of local accumulation of blood/fluid under some pressure, which can make the skin locally vulnerable to, e.g. touching or rubbing, and sometimes even necrotic. Another very rare complication is the occurrence of an infection in a subgaleal haematoma. Barry described an infected subgaleal haematoma in an 8-month-old girl with a closed skull fracture after a fall of about 1 m [92]. The child was seen 1 day after the fall with a swelling of the head. Nine days after the fall incident, she was seen again in the hospital because of fever, irritability, and a significant increase of the swelling in the head. MRI showed a subgaleal abscess with osteomyelitis.

5.2.3.2 Cause of Subgaleal Haematoma

A subgaleal haematoma is caused either by traumatic separation of the aponeurosis/galea from the periosteum, resulting in tearing of the vessels crossing this potential space and

accumulation of blood in the subgaleal space or by migration of intracranial blood and/or liquor to the subgaleal space. Traumatic separation may be due to a minor or a major trauma [79, 81, 93].

Traumatic Separation of the Galea from the Periosteum

Traumatic separation of the galea from the periosteum (in other words, the tearing loose of the galea from the periosteum due to trauma) can be caused by traction on the hairy scalp (a more or less perpendicular force), by shifting of the galea and the periosteum relative to each other (a movement of the scalp and underlying soft tissue relative to the skull, whereby the galea shifts relative to the periosteum, caused by either a radial and/or tangential force, usually due to an impact trauma), or by a combination of traction and shifting. Haemorrhage in the originally virtual subgaleal space occurs, when the draining veins, which connect the veins of the hairy scalp with the intracranial dural venous sinuses, are damaged, due to the same mechanisms that caused the traumatic separation: traction, shifting, or both [80].

Migration of Intracranial Blood and/or Liquor to the Subgaleal Space

On rare occasions intracranial blood and/or liquor may migrate into the subgaleal space:

- Malek et al. described a 17-month-old patient, who fell from a height of 1.5 m and presented with an 8-mm temporal epidural haematoma, an overlying linear skull fracture, and a subgaleal hematoma without evidence of intraparenchymal injury or oedema [94]. There were no neurological symptoms. When the CT scan was repeated after 18 h, a near-complete resolution of the epidural haematoma was seen with an increase in the volume and spread of the subgaleal hematoma.
- Neely reported a 2½-year-old girl, who was struck on the left side of the head by a softball [95]. During physical examination, a soft-tissue swelling was felt over the left parietal bone. She had no seizures and no reported loss of consciousness (Glasgow coma scale 15). Serial CT scans showed the spontaneous decompression of epidural bleeding into the subgaleal space. The decompression was visible on serial CT scans as a simultaneous decrease in epidural blood and increase in subgaleal blood.
- Chida et al. described a similar course as Neely in a 4-month-old girl, who had fallen off the table on the floor [96]. On the first CT scan, made 4 h after trauma, an epidural haematoma and a left-sided parietal fracture were seen. Four hours after the first CT scan, the epidural haematoma was found to have increased in size. On day 2, it was seen that the blood discharged from the epidural location through the fracture into the subgaleal space. On day 5, the epidural blood had almost completely moved to the subgaleal space. The epidural and subgaleal blood had completely disappeared on the 12th day. No other complaints or symptoms were seen during admission.
- Yaka et al. described the extracranial decompression of an asymptomatic epidural haematoma in an 8-year-old girl [97]. The girl had fallen at the playground and struck her head on the ground 15 days before visiting the hospital with a swelling in the right parietal region. A CT scan was made, which showed a mixed density parietal epidural haematoma with a linear fracture overlying it. No other pathological intracranial findings, like a midline shift or an ipsilateral ventricular compression, were done on imaging. The girl remained asymptomatic after the fall. According to the authors, this was due to the spontaneous decompression of the epidural haematoma into the subgaleal space.
- Yamada et al. described a 6-year-old boy who presented at the hospital after a head trauma [98]. A CT scan, made at the initial presentation, showed a thin epidural haemorrhage in the right temporo-occipital area and diastasis of the right lambdoid suture. The boy showed no neurological or other symptoms. After a week he visited the hospital again, because of a massive fluctuant watery mass extending from the forehead to the right temporoparietal areas. Laboratory data revealed that he was anaemic. Follow-up CT showed a massive subgaleal haematoma around the cranial diastasis. Surgery was performed and the boy recovered completely and was discharged 9 days after surgery.

5.2.3.3 Manner of Subgaleal Haematoma

Intrauterine Trauma

A subgaleal haematoma is only very rarely sustained due to an intrauterine trauma. Only one case report was found. Assad et al. reported an intrauterine sustained depressed foetal skull fracture with massive subgaleal and subperiosteal haemorrhage, which required neurosurgical intervention with good clinical outcomes for both mother and infant [99].

Trauma During Birth

Although most cases of subgaleal haematomas are reported in neonates, subgaleal haematomas due to trauma during birth (neonatal subgaleal haematomas) in fact are rare. Gebremariam reported an incidence of 0.16–0.3% of all neonates [77]. Shimokaze et al. stated that neonatal subgaleal haematomas are found in about 0.04% in spontaneous deliveries and 0.6% in vacuum extractions [100]. Ekéus et al. found subgaleal haematomas in about 1.5% of the children born after using vacuum extraction [101]. However, in over

90% of the newborns with a subgaleal haematoma, the bleeding is caused by traction on the scalp due to using a vacuum extractor during vaginal delivery [79]. Usually, they contain a low volume of blood, often resolve spontaneously and in general have a good prognosis [100, 102].

A neonatal subgaleal haematoma can give rise to serious clinical symptoms, which can be potentially life threatening, because of the large volume of blood that can be present in a subgaleal haematoma, e.g. a hypovolaemic shock or disseminated intravascular coagulation and bleeding (Fig. 5.6) [79, 103–106].

Kilani and Wetmore evaluated the data of 34 neonates with a subgaleal haematoma [107]. In 31 neonates (over 90%) the haematomas were due to instrumental deliveries (vacuum in 21, vacuum followed by forceps in 8 and forceps in 8 neonates). In 17 children associated intracranial haemorrhage occurred (subarachnoid haemorrhage in 4, intraventricular haemorrhage in 4, intraparenchymal haemorrhage in 4, and subdural haemorrhage in 11 children). Skull fractures were found in six children, three of them had depressed fractures. Four children died, due to significant loss of blood volume loss with anaemia, coagulopathy, and shock requiring large volumes of blood and blood products transfusions. The presence of intracranial haemorrhage did not correlate with the severity of subgaleal haematoma or mortality, but the severity of the subgaleal haematoma correlated with mortality. In only four children minor neurological abnormalities were noted at discharge.

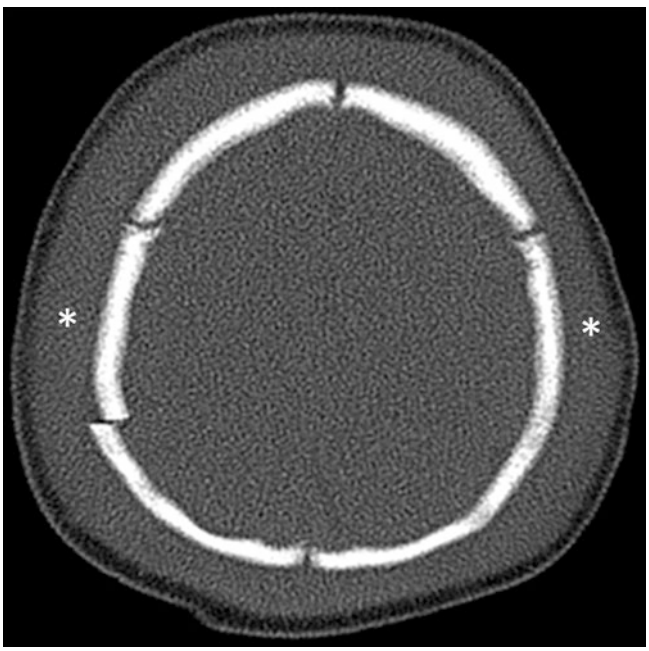


Fig. 5.6 Large circumferential subgaleal haematoma (asterisk) in a newborn causing hypovolemia. The skin and subcutaneous fat are normal (excluding caput succedaneum) and the hematoma crosses the sutures (excluding cephalhaematoma). Note the right parietal fracture

Santín-Amo et al. described that a neonatal subgaleal haematoma very rarely becomes chronic and then might require a surgical treatment [102].

Swanson et al. described the neonatal outcomes in 21 newborns with subgaleal haematomas over a 10-year period [105]. They found that 13 (62%) were born by instrumental vaginal delivery and that 10 neonates (48%) required resuscitation at delivery. The severity of subgaleal haemorrhage varied from mild in four infants (19%), via moderate in 10 (48%) to severe in seven (33%). Hypovolemic shock developed in 10 neonates (48%), encephalopathy in 13 (62%) and coagulopathy was present in five (24%). Three children died (14%). Long-term outcomes were good in the surviving infants.

Fareeduddin and Schifrin reported a case in which vacuum extraction was used during an elective caesarean delivery [108]. Although there is a clear relation between the presence of neonatal subgaleal haematomas and the use of vacuum extraction, Liu and Antaya reported the presence of four enlarging subgaleal haematomas in an 11-day-old boy born without the use of instruments during delivery [104]. They suspected that the provider's fingers caused the subgaleal haematomas during vaginal delivery.

Neonatal subgaleal haematomas, due to instrumental and/or traumatic deliveries, usually are recognized clinically shortly after birth. Sometimes it may take weeks to months before the swelling of the infant's scalp is diagnosed as a delayed presentation of a neonatal subaponeurotic (subgaleal) fluid collection [109]. Smith et al. evaluated 11 infants with a delayed presentation [109]. All infants underwent either successful vacuum delivery or failed vacuum delivery with subsequent forceps delivery or emergency caesarean section. All infants were otherwise well at presentation, and resolution of the scalp swelling occurred within weeks to months. In all the conditions had a benign course.

Trauma After Birth: Accidental or Non-accidental

Subgaleal haematoma occurring beyond the neonatal period is rare and is often associated with head trauma involving forces applied to the scalp causing emissary veins traversing the subgaleal space to be ruptured [79]. The circumstances under which a subgaleal haematoma can be sustained after birth are either accidental or non-accidental. It is not possible to differentiate between accidental and non-accidental circumstances merely based on the presence of a subgaleal haematoma.

Accidental Traction

A subgaleal haematoma occurring beyond the neonatal period may result from vigorous hair combing and tight hair braiding [79, 93, 110, 111]. Palmer described two girls aged 9 months and 10 years of age in whom traction caused by hair braiding resulted in a subgaleal bleeding [112]. In both

girls, the subgaleal haematoma became visible within 24 h after braiding. Vu describes the development of a massive subgaleal haematoma after hair braiding in a 9-year-old girl [79]. During physical examination on admission 11 days after the braiding she showed notable scalp swelling and bogginess frontoparietal as well as significant bilateral periorbital oedema. She was also anaemic. Onyeama describes the development of a subgaleal haematoma by braiding in a girl of 31 months. It was visible within 1 day and resolved in 2 weeks. Baker et al. reported the occurrence of a massive right frontotemporoparietal subgaleal haematoma after hair combing in a 14-year-old boy [110]. The boy presented with pain and swelling in the scalp for 6 days duration. The haematoma was drained 3 times and surgery was planned, but when he presented for the operation, the haematoma had resolved. In children in whom tight braiding resulted in a subgaleal haematoma, one should always consider a coexisting coagulation disorder (Sect. 5.2.3.4, Table 5.4).

Accidental Impact Trauma/Blunt Force Trauma

In 40% of children with a subgaleal haematoma one will find other signs and symptoms, suggestive of an impact trauma, including cutaneous injuries, skull fractures, or intracranial haemorrhages (Fig. 5.7a, b). The simultaneous presence of these physical findings is proof of impact trauma to the head. Often the trauma to the head is due to an accidental fall, e.g. with a bicycle, or when playing at home or in a playground, a traffic accident, a blow to the head by a swing, a karate blow, or a frying pan that fell on the head [89, 113].

In mobile children, there seems to be no relationship between the severity of the impact trauma, the severity and extent of subgaleal haematoma, the severity of other symptoms, and physical findings. Subgaleal haematomas in young children appear to occur due to a minor and perhaps sometimes even unnoticed trauma as well as major trauma [79–81, 93, 114].

Kuban et al. and Cooling and Viccellio described a number of infants and young children with a minor impact trauma leading to a subgaleal bleeding, which sometimes was massive, without any evidence of a skull fracture or a coagulation disorder [80, 114]. In these children, no specific underlying mechanism for the onset of bleeding was found. Haidar-El-Atrache et al. described a 7-year-old girl with scalp swelling, due to a subgaleal haematoma since 1 day [115]. There was no history of trauma or of easy bruising in the family. Neither signs of impact trauma, e.g. bruising, nor findings consistent with a coagulation disorder were found. They concluded that trivial impact trauma, which was not recollected by the child, may have caused the haematoma.

Even in a more or less minor trauma massive subgaleal haemorrhage can occur without an underlying skull fracture and/or without clotting problems. Oliveira Sillero described a 14-year-old boy who had fallen off his bicycle and hit the

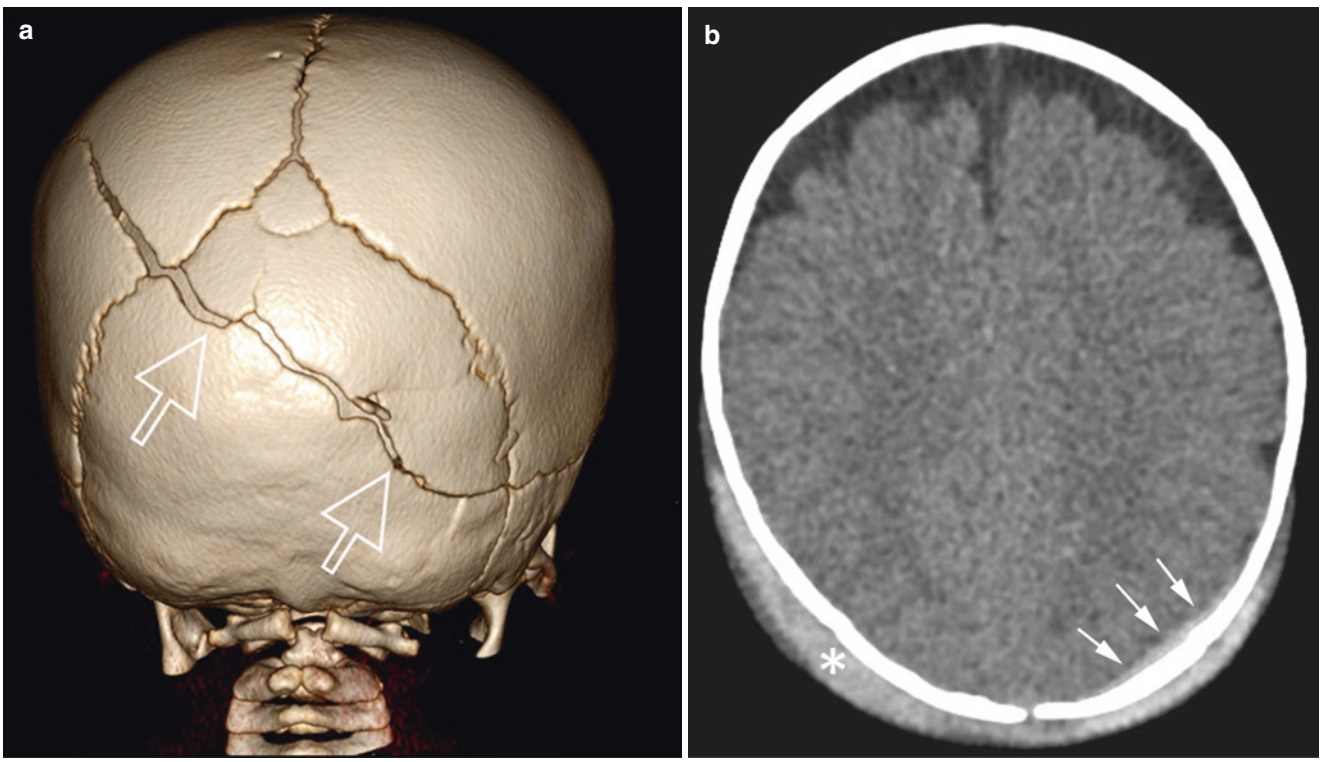


Fig. 5.7 This 6-month-old boy was said to have been struck on the head with a wooden toy block, thrown by a toddler. (**a**) Subgaleal haematoma (asterisk) and evidence of impact trauma: occipital fracture (arrows). (**b**) A and small epidural hematoma is also present (arrows)

ground with his head 14 days before he was admitted to the hospital [116]. His head was remarkably swollen. The CT scan showed a massive subgaleal haematoma. A total of 600 ml old blood was removed during drainage. Kichari and Gielkens described a 15-year-old boy with an increasing head circumference due to swelling to the hairy scalp after a minor trauma 4 days before admission [117]. An extensive circumferential subgaleal haemorrhage was seen on the CT scan. He had no neurological deficits. The haematoma disappeared within a few weeks. Takano et al. described a 9-year-old girl who sustained a subgaleal haematoma, after she struck the left side of her head on a doorknob [91]. She also had complaints of exophthalmos and diplopia.

In case of the simultaneous presence of a subgaleal haematoma and a skull fracture this usually will be a linear often parietal skull fracture without diastasis of the fracture line, directly located below the subgaleal haematoma [118]. The subgaleal haematoma most probably is due to the accumulation of blood from the fracture into the subgaleal space [118]. If there is only a skull fracture without intracranial injury, the child will be alert and active with no evidence of neurological problems. The only symptom may be irritability or headache caused by the subgaleal haematoma.

The simultaneous presence of a subgaleal haematoma and a nonlinear and more complex skull fracture or multiple skull fractures suggests a more serious trauma, irrespective of the circumstances, under which the trauma occurred (accidental or non-accidental) than in the case of the simultaneous occurrence of subgaleal haematoma and a linear fracture [118].

Non-accidental Traction

Many authors state that one always should consider non-accidental circumstances if a subgaleal haematoma is found in a paediatric patient, especially if hair pulling is suspected, although there are hardly any case reports, concerning hair pulling inflicted by a parent or caregiver to be found in the medical literature [111, 115, 119]:

- Hamlin stated that the most likely cause of childhood subgaleal haematoma is a vigorous hair-pull by the grip of an enraged adult and that finding this would be a clue to '*the battered-child syndrome*' [119].
- Seifert and Püschel described a 3½-year-old boy, who was brought to the hospital by his mother [63]. His forehead was swollen and bluish. A doughy swelling was present on the forehead as well as the rest of the calvarium. Some hours later, a massive bilateral periocular haematoma appeared. The mother was not able to explain what had happened. Sonographic examination showed an extensive subgaleal haematoma. Finally the mother's boyfriend admitted that he forcefully pulled the boy's hair.
- Shamji and Jacoby reported a 2-year-old girl, who was brought to the hospital by her mother because of a swollen head (64.5 cm circumference instead of normally 44–51 cm), which was due to a massive subgaleal haematoma [120]. 1500 ml of serosanguineous fluid was removed with two subgaleal drains. Further imaging showed a healing fracture of the distal humerus and occult fractures of the right tibia and wrist. Coagulation disorders were excluded. Child protection services confirmed the clinical suspicion of injuries due to non-accidental circumstances (child abuse). The subgaleal haematoma was explained as a non-accidental trauma, due to chronic hair pulling.

Several case reports have been published in which hair pulling was not done by a parent/caregiver, but by another child:

- Yip et al. described a 13-year-old boy with a 1-week history of progressive proptosis after his older sister pulled his hair during an argument [121]. On CT-scan a subgaleal hematoma and a right superior subperiosteal orbital hematoma were found.
- Fujisawa et al. described a 12-year-old girl with a subgaleal haematoma developed over her right cranium, due to hair pulling on the right side of her head pulled during a quarrel [122]. The subcutaneous swelling progressed to the forehead, and a marked exophthalmos developed on the left side.
- Edmondson et al. reported a case of an adolescent with delayed presentation of a massive subgaleal haematoma in the absence of any underlying haematological or anatomical abnormality, following a seemingly innocuous episode of hair pulling whilst play fighting [81].

Non-accidental Impact Trauma/Blunt Force Trauma

Also in case of a subgaleal haematoma due to an impact trauma non-accidental circumstances may play a role, e.g. if the child has been hit against the head with a hand or an object. According to Ota the simultaneous presence of a subgaleal bleeding with a more extensive/more complex skull fracture, a compound fracture or multiple fractures indicates a more serious incident (either accidental or non-accidental), than when a subgaleal haemorrhage is found in combination with a linear fracture [118]. If there are no clues in the medical history indicating a serious accident (preferably monitored by an independent observer), non-accidental circumstances should be considered.

5.2.3.4 Differential Diagnosis of Subgaleal Haematoma

Other Scalp Haematomas

In a neonate subgaleal haemorrhage must be differentiated from a caput succedaneum and cephalhaematoma (Table 5.3, Figs. 5.8 and 5.9). Subgaleal haemorrhage, unlike a cephalhaematoma, may cross the sutures and obscure the fontanelle [88]).

Table 5.3 Distinguishing features of different neonatal extracerebral fluid collections [87, 103, 123]

Feature	Caput succedaneum	Subgaleal haemorrhage	Cephalhaematoma
Location	Between the skin and the galea, at point of contact; can extend across sutures	Between the galea and the periosteum; may extend to orbits and to the neck; can extend across sutures	Between the periosteum and the skull, usually over parietal bones; restricted to the subperiosteal space; does not cross sutures
Characteristic clinical findings	Soft and poorly defined tissue oedema or serosanguineous fluid; pitting oedema that shifts with gravity	Diffuse, mobile, and elastic swelling; firm to fluctuant; ill-defined borders; may have crepitus or fluid waves	Fixed, elastic swelling with distinct margins; initially firm, more fluctuant after 48 h
Timing	Immediately visible after birth; maximum size and firmness at birth; resolve in 48–72 h	Progressive after birth; resolution over 2–3 weeks	Increases after birth for 12–24 h; resolution over 2–3 weeks
Volume of blood	Minimal	May be massive, especially if there is an associated coagulopathy	Rarely severe

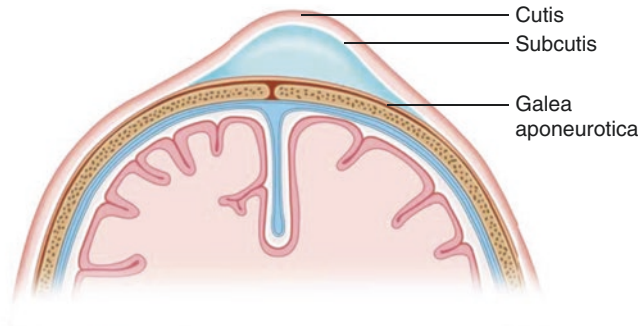


Fig. 5.8 Caput succedaneum. Fluid and/or oedema is located in the subcutaneous tissue between the galea aponeurotica and the skin. It may cross sutures and fontanelles

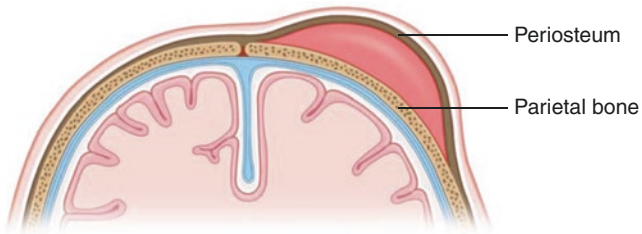


Fig. 5.9 Cephalhaematoma between the parietal bone and the periosteum. Cephalhaematomas do not cross sutures or fontanelles because the periosteum is confined to the bone

Coagulation Disorders

A subgaleal haematoma may occur after a minor trauma [79, 80]. However, if a subgaleal haematoma is found in a child after a minor trauma, one should always consider a coexisting or contributing underlying disorder, like a coagulation disorder [22, 79, 82, 84, 124]. In the medical literature, several coagulation disorders have been related to the occurrence of a subgaleal haematoma (Table 5.4).

Other Disorders

In the medical literature very rarely other disorders than coagulation disorders have been reported as coexisting or contributing medical conditions:

- Kirkpatrick et al. mentioned that a subgaleal haematoma should be differentiated from subgaleal infection and air from frontal sinusitis with bony erosion, and from an encephalocele or tumour erosion through the skull [82].
- Dahdaleh et al. reported a patient with sickle cell disease who presented with a sickle cell crisis that was complicated by the development of multiple acute epidural and subgaleal haematomas requiring surgical evacuation [145].
- Al-Tonbary et al. reported on a 2½-year-old boy with a progressive increase of the head circumference, due to a large subgaleal haematoma, which the boy sustained after a trivial trauma (falling on the ground while playing) 3 days before admission to the hospital [146]. Two days after admission, bruises started to appear over the entire body. The subgaleal haematoma and the bruising were the first signs of an acute myeloid leukaemia.

The Use of Anticoagulants

Children, who are treated with anticoagulants, like aspirin, for a prolonged period, are at risk of sustaining an extensive subgaleal haematoma, eventually resulting in significant anaemia, even in case of a mild trauma [79, 147].

5.2.3.5 Dating Subgaleal Haematomas

It is not possible to give an indication of the moment on which a child sustained a subgaleal haematoma based on the appearance of the first symptoms, on the moment on which the subgaleal haematoma became visible, or the characteristics of the externally visible ‘bruising’:

Table 5.4 Overview of coagulation disorders, related to the occurrence of a subgaleal bleeding

Disorder	Author	Patient	Symptoms
Factor I deficiency (fibrinogen deficiency, congenital hyperfibrinogenaemia, dysfibrinogenemia)	Prakash [125]	10-year-old girl	• Diagnosed at the age of 4 months with congenital hyperfibrinogenaemia • Subgaleal haematoma and proptosis after falling on her head
	Sharma et al., Khare et al. [126, 127]	5-year-old boy	• Gradual increase of the head circumference for last 1 month before admission • Medical history also showed the presence of a massive caput succedaneum at birth (full-term normal vaginal delivery) and prolonged profuse bleeding from minor injuries in the past
Factor VII deficiency	Pomeranz et al. [128]	6½-year-old girl	• Subgaleal haematoma and proptosis 9 days after a mild head trauma. • She fell on the back of her head, while roller skating
	Jenkins et al. [129]	10-year-old girl	• Delayed contralateral proptosis and external ophthalmoplegia after relatively minor right-sided forehead trauma
Factor VIII deficiency (haemophilia A) and factor IX deficiency (haemophilia B)	Alcover Bloch et al., Balliu Bada et al., Chia et al., Cohen et al., Rohyans et al., Radovanović et al., Talar et al. [130–135]	Neonates	• Massive subgaleal haematomas in neonates with haemophilia A or B with the risk of severe anaemia and a hypovolemic shock in the first days after birth, or even death (with or without instrument-assisted delivery)
	Talar et al. [135]	Neonate	Neonate delivered through caesarean section with a subgaleal haematoma
	Guirgis et al. [136]	5-year-old boy	• Large subgaleal and left subperiosteal orbital haematoma, resulting in progressive left-sided scalp swelling, marked proptosis of the left eye, visual loss (20/200 visual acuity), and an afferent pupillary defect after a mild trauma.
Factor X deficiency	Wetzel and Kingma [137]	Neonate	• Born after a non-traumatic, non-instrumented caesarean section with respiratory failure and severe metabolic acidosis secondary to subgaleal haematoma
Factor XIII deficiency	Kim and Taragin [22]	9-year-old girl	• Sudden head swelling, bilateral proptosis, extraocular muscle palsy, and progressive visual disturbance after hair braiding due to a subgaleal haematoma • Surgical intervention was needed
	Natarajan et al. [138]	6-year-old boy	• Tense subgaleal hematoma and proptosis 2 weeks after a minor head trauma
Von Willebrand (type 1)	Raffini and Tsarouhas [124]	17-year-old boy	• Subgaleal haematoma after hair braiding
Vitamin K deficiency	Ryan and Gayle [139]	Infant	• Exclusively breast-fed, no vitamin K prophylaxis at birth • Large subgaleal hematoma, resulting in hypovolemic shock, and death
Neonatal alloimmune thrombocytopenia	Borensztajn et al., Talar et al., Winkelhorst et al. [135, 140, 141]	Several neonates	• Severe subgaleal haematomas
Platelet function defect	Kirkpatrick et al., Hutspardol et al. [82, 142]	?	• No trauma occurred, spontaneous subgaleal haematoma
Kasabach-Merritt syndrome	Stalder et al. [143]	25-month-old boy	• Massive calcified subgaleal hematoma with secondary cranial deformity • Cranial deformity required surgical evacuation and reconstruction
Disseminated intravascular coagulation	Modanlou et al. [144]	Male neonate	• Massive subgaleal haematoma following the use of a vacuum extractor

- The first physical symptoms of a subgaleal haematoma (irritability, headache) do not have to occur immediately after the causing trauma.
- The first physical findings do not necessarily have to be immediately recognizable as ‘bruising’, e.g. around the eyes, behind the ears, and in the neck, because often only a non-specific swelling of the scalp or local redness becomes visible as the first physical sign.
- In infants and young children ‘bruising’ may take hours to days, before becoming visible, usually about 1–8 days after the causing trauma, before enough blood is collected in the subgaleal space to become visible or palpable for a

caregiver or during a physical examination [80]. However, the maximum period described in the medical literature is up to 14 days [79].

5.2.4 Cephalhaematoma

5.2.4.1 Introduction

A cephalhaematoma is an accumulation of blood or serosanguineous fluid under the periosteum of the skull (Fig. 5.9) [148, 149]. Cephalhaematomas are usually located over the parietal bones and can be found unilateral or bilateral. A cephalhaematoma can be sustained at any age, although the occurrence outside the neonatal period is extremely rare [150–153].

5.2.4.2 Cause and Manner of Cephalhaematoma

The accumulation of blood in the subperiosteal space is caused by a rupture of blood vessels crossing the periosteum due to shearing of the periosteum relative to the skull. The accumulation 'lifts' the periosteum from the skull and 'creates' a subperiosteal space, which in physiological circumstances does not exist. The blood cannot cross the sutures, because it is bound between the periosteum and the skull (Figs. 5.10 and 5.11a, b). When the blood accumulates, the pressure in the subperiosteal space increases and the accumulated blood finally will act as a tamponade and prevent further bleeding [148, 149].

Almost all cases of cephalhaematomas are found in neonates and occur due to traumatic pressure on the head during birth, e.g. caused by pressing the head against the maternal pelvis during labour or by the use of instruments [149]. The blood accumulates gradually and the cephalhaematoma may not be evident immediately after birth. In the setting of craniosynostosis, the blood may cross the affected suture [154]. Cephalhaematomas are found in about up to 2.5% of all living neonates [149, 155, 156].

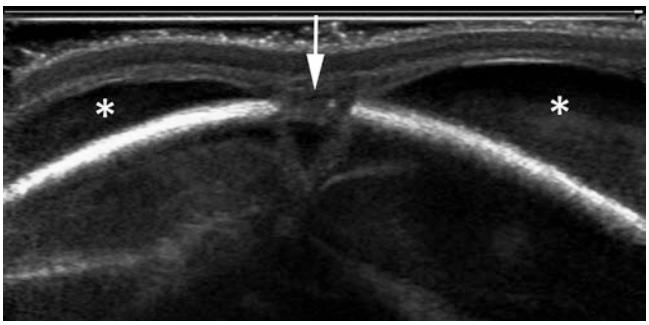


Fig. 5.10 Coronal ultrasonography image of a newborn with bilateral cephalhaematomas (asterisk). The cephalhaematomas do not cross the sagittal suture (arrow)

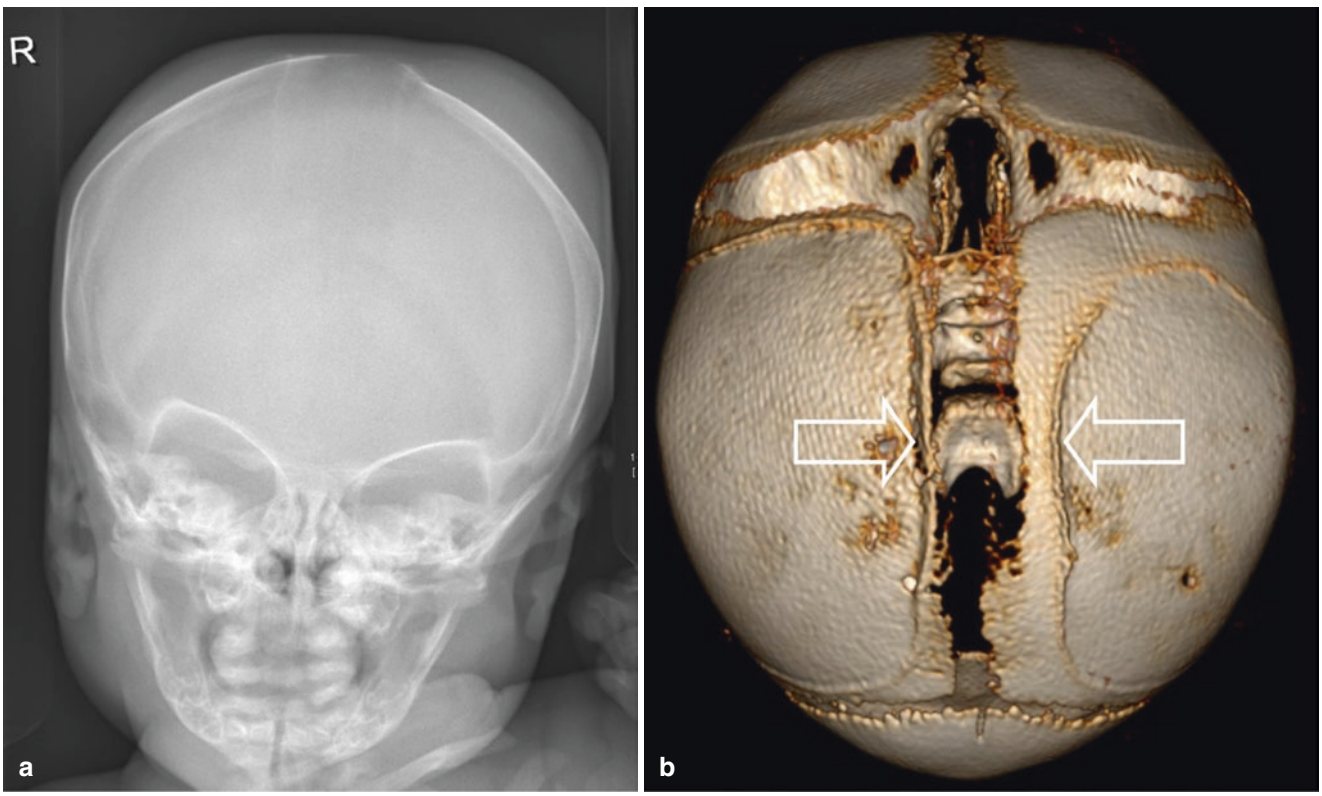


Fig. 5.11 Neonate with a bilateral swelling. (**a**) Skull radiograph shows bilateral soft tissue swellings. (**b**) 3D CT at the age of 6 weeks shows calcified ridges along the edges of the cephalhaematomas (arrows)

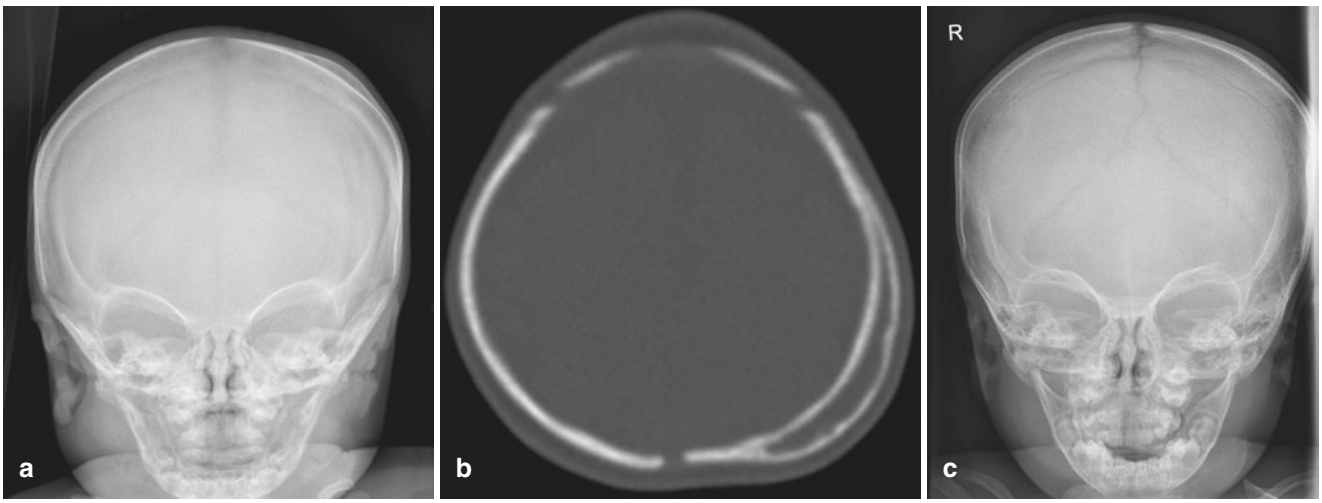


Fig. 5.12 Calcifying cephalhaematoma. Skull radiograph (**a**) and CT (**b**) at the age of 3 months. At the age of 12 months (**c**) the calcified cephalhaematoma has almost resolved and blended with the cortical bone

Treatment will not be necessary for most neonates [156]. Neonatal cephalhaematomas usually resolve spontaneously within 1 month, sometimes up to 3 months, although sometimes complications may occur [154, 157]. According to Raines and Jain, there is an increased risk of neonatal jaundice in the first days after birth [149]. Calcification is often seen and may undergo spontaneous remodelling (Fig. 5.12a–c) [157–159]. Calcification can start after 2–3 weeks [154]. Calcification may lead to asymmetry of the skull, secondary craniosynostosis, and/or deformation of the skull [155, 157, 158]. Spontaneous resorption of a calcified haematoma has also been reported [155, 160].

Sometimes a fracture is found underneath the haematoma. This fracture usually will be linear, but also the occurrence of a 'ping pong' fracture has also been described [161].

Wong and Cheah reported that hyperbilirubinemia or scalp infection can occur in children with a cephalhaematoma [156]. They reported a neonate with a cephalhaematoma, infected with *Escherichia coli*, resulting in an extensive deep-seated scalp abscess and septicaemia with *E. coli*. Zimmermann and Duppenthaler also reported the occurrence of an infected cephalhaematoma with *E. coli* in a 5-week-old infant [162].

The clinical course of a cephalhaematoma in a neonate can get complicated because of a coexisting coagulation disorder. Abdullah et al. described a 2-day-old girl with a cephalhaematoma with persistent abnormal coagulation tests, due to a combined factor V and VIII deficiency [163]. Salek et al. described a 2-day-old boy with a large cephalhaematoma of the right frontoparietal region [164]. On day 3 he showed pallor, poor feeding, and decreased activity. The boy was finally diagnosed with severe haemophilia A.

5.3 Calvarium Fractures

5.3.1 Introduction

The neurocranium consists of the skull cap/calvarium and the skull base and covers the cranial cavity, containing the brain. The calvarium is the dome-like upper part of the neurocranium. The skull base is the floor of the neurocranium (Sect. 5.4). The calvarium consists of the superior part of the frontal bone, the occipital bone, the parietal bones, and the temporal bones [165]. These bones are connected by syndesmoses (sutures) and these may cause confusion because they may be mistaken for fractures. A thorough knowledge of normal sutures (and their variants) is mandatory. More detailed information is found in Sect. 5.3.4.7 (Figs. 5.45, 5.46, 5.47, 5.48, 5.49, 5.50, 5.51, 5.52, 5.53, 5.54, 5.55, 5.56, 5.57, 5.58, and 5.59).

A trauma to the head can cause injuries to the covering skin (Sect. 5.2), the skull, e.g. fractures of the calvarium (this section), the base of the skull (Sect. 5.4), and/or orofacial bones (Sect. 5.5), and the intracranial content (Sect. 5.6). Children are more susceptible to head trauma than adults [166]. The size of a child's head is approximately 18% of the total body surface area in infancy, decreasing to about 9% in adults. Also, the body proportions do change. In infancy, the child's head is about 25% of the total body length, decreasing to about 12.5% in adulthood (Fig. 5.13). Above that, the skull of a child is thinner and more pliable, thus providing less protection to the brain than in adults [166].

Infants and young children regularly fall, and quite often they fall on their head [71]. If infants fall out of the hands of their parents or fall off a couch or changing table, this usually will be 'head first', with the head as the first part of the body

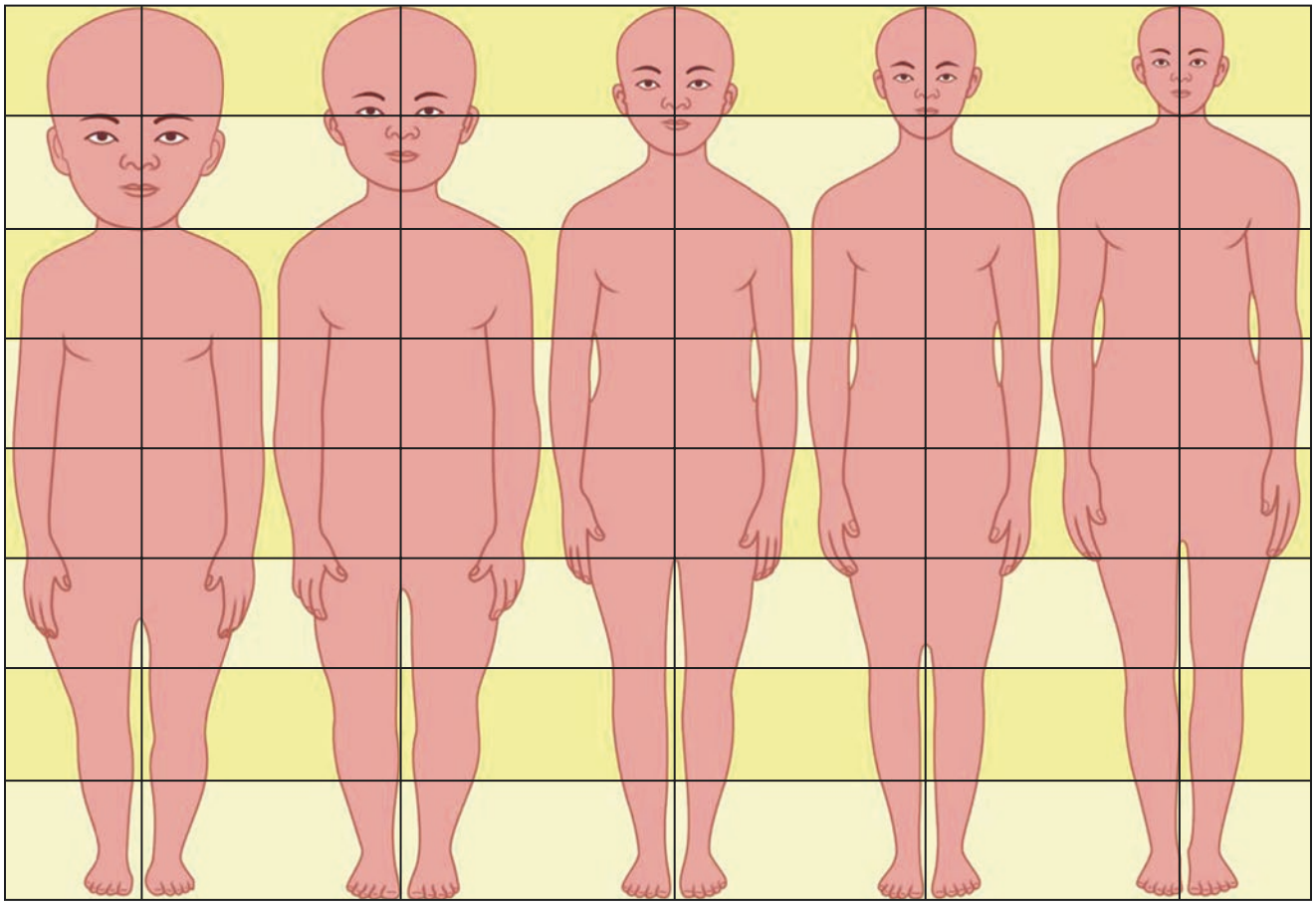


Fig. 5.13 Changing body proportions from infancy to adulthood, the human head becomes proportionately smaller and the legs become proportionately longer as humans mature. (Reprinted with permission: By Ephert—Own work, CC BY-SA 4.0, https://commons.wikimedia.org/w/index.php?curid=39752841 from 'Neoteny in humans' [167])

hitting the floor. This is due to the body proportions and the head being the heaviest body part in infants and young children. 80–90% of head trauma in paediatric patients can probably be classified as mild [166].

Fractures of the calvarium are the most common skull fractures in young children [168]. These are more common in younger children than in older children, irrespective of the circumstances (accidental or non-accidental—Sect. 5.3.3) [169]. Ibrahim et al. evaluated the findings in 285 children, aged 0–48 months with an accidental head injury from a fall [170]. They found that, despite similar injury severity scores, infants sustained more skull fractures than toddlers (71 vs. 39%). Of all children with skull fractures, 11% had no evidence of scalp and/or facial soft tissue swelling.

It is unknown how often a head trauma results in a calvarium fracture, although the incidence of skull fractures in children that present at the emergency department following head trauma ranges from 2 to 20% [171]. Fractures of the parietal bones are most common, followed by the occipital, frontal, and temporal bones. Generally, it will be linear fractures without dislocation (Sect. 5.3.4.2), followed by depressed fractures (Sect. 5.3.4.5) and basilar skull fractures (Sect. 5.4). Fractures of the calvarium usually do not cause any complications, unless they are accompanied by fragmentation causing bone-splinter damage to brain tissue or by epidural hematomas causing compression of the brain. Another possible complication of a calvarium fracture is the so called 'growing skull fracture' (Sect. 5.3.4.6).

The precise incidence of calvarium fractures however will be difficult to determine, because it is rarely indicated after a head trauma in a young child to perform a medical examination or additional diagnostic procedures, such as a radiograph or a CT scan, because most children do not show any disturbing symptoms. If a doctor is consulted, this consultation usually will be limited to a physical examination. Imaging of the skull, e.g. radiography or CT, is clinically not indicated, especially when neurological symptoms or physical findings indicating a fracture are lacking.

A fracture of the calvarium can be suspected because of the medical history or certain findings during the physical

examination. Older children may complain of a localized headache. Physical examination may reveal local swelling, a haematoma, a palpable fracture or physical findings, indicating a basilar skull fracture (Sect. 5.4). Metz et al. evaluated the findings in 218 children under the age of 4 years (two-thirds of the children under the age of 1 year) with a skull fracture and found clinically apparent soft tissue swelling in 73% and radiologically apparent fracture-associated soft tissue swelling in 93%of the children [73].

The majority of fractures of the calvarium will have little or no clinical consequences, due to the lack of disturbing clinical symptoms, physical findings, or complications. Despite the lack of these, fractures of the calvarium do have an indicative value. Their presence implies that considerable force has been exerted on the skull [172]. Therefore, from a forensic point of view, imaging of the skull is indicated—especially when the child is under the age of 1 year. This also applies when there are no neurological symptoms, even though a normal radiograph of the calvarium does not exclude the presence of a fracture or of intracranial injury. Finding a fracture of the calvarium does not always indicate that underlying structures such as dura, bridging veins, or brain have been damaged. A calvarium fracture can be observed during an operation or autopsy, while not necessarily being visible on a radiograph [173]. In 16 children with an epidural haemorrhage and a calvarium fracture, the fracture was visible on radiographs in 10 children, in four children it was seen during operation and in two during autopsy [174].

5.3.2 Cause of Calvarium Fractures

Fractures of the calvarium are caused by either static or dynamic (impact) loading of the head (Sect. 5.1) [2, 3]. In both types of loading the skull changes shape. This applies to children as well as to adults. In this section, only the effects of static and dynamic loading on the calvarium are discussed. Both types of loading can also lead to injuries of the scalp and the orofacial skin, the base of the skull, the orofacial bones, and of the intracranial content. These effects will be discussed respectively in Sect. 5.2 (injuries of the scalp), in Sects. 5.4 (basilar fractures) and 5.5 (orofacial bone fractures), and in Sect. 5.6 (intracranial injuries).

5.3.2.1 Static Loading

Defining Static Loading

Static loading is the relatively slow loading of forces exerted on the calvarium over a protracted period of time (above 200 ms). Static loading of the calvarium occurs when the calvarium is squeezed and/or compressed.

Injuries to the Head in Static Loading

Deformation of the calvarium, caused by static loading (squeezing and compression) can cause a more or less focal effect, e.g. a single line/linear fracture restricted to one calvarium bone (Sect. 5.3.4.2), symmetrical bilateral linear fractures (Sect. 5.3.4.3) or a depressed fracture/ping pong fracture (Sect. 5.3.4.5). Deformation due to static loading can also cause more diffuse multiple fractures in more than one skull bone and on more than one side (front, back, left, right) (Sect. 5.3.4.4). Finally, static loading can cause intracranial injuries (Sect. 5.6).

5.3.2.2 Dynamic Impact Loading

Defining Dynamic Impact Loading

Dynamic (or rapid) loading is the fast loading of forces over a shorter period (less than 200 ms, often even less than 50 ms). Dynamic loading can be divided in ‘impulse’ and ‘impact’ loading.

Dynamic impulse loading is the result of fast and repetitive movements of the head, without impact (acceleration—deceleration). Impulse loading of the head never causes fractures of the calvarium, the base of the skull, or orofacial bones.

Dynamic impact loading can be due to blunt force trauma (non-penetrating or penetrating) or to sharp force trauma (non-penetrating or penetrating). In dynamic impact loading, the head and an object can move relative to each other. The following situations can occur during the impact:

- Stationary head, impacted by a fast-moving object (acceleration trauma of the head and intracranial content), sometimes followed by falling, in which the head started moving and hits a stationary object, e.g. another object, a wall, or the floor (acceleration trauma followed by a deceleration trauma).
- Stationary object, impacted by the fast-moving head (deceleration trauma of the head and intracranial content).
- Both head and object are moving in the same direction during the impact, similar to a head-to-tail motor vehicle collision.
- Both head and object are moving in the opposite direction during the impact, similar to a frontal motor vehicle collision.
- Both head and object are moving with an oblique impact, similar to a side-impact collision.

Injuries to the Head in Dynamic Impact Loading

Fractures of the calvarium can be caused by dynamic impact loading [2]. Other injuries due to dynamic impact loading of the head are injuries to the scalp and the orofa-

cial skin (Sects. 5.2 and 5.5), the base of the skull (Sect. 5.4), the orofacial bones (Sect. 5.5), and the intracranial content (Sect. 5.6).

Child's Versus Adult's Calvarium in Dynamic Impact Loading

Calvarium fractures due to dynamic impact loading are regularly seen in children as well as in adults. Compared to a child's calvarium, the adult's calvarium is fairly rigid. The adult calvarium can cope with some deformation. However, when the deformation exceeds a certain point, no recovery is possible and a fracture will occur. Post-mortem research has shown that the adult calvarium can be indented a few centimetres before it resumes its original shape with or without fracture [175, 176]. This indentation may lead to considerable damage to the underlying tissue, even without a fracture being present.

A child's skull is made of thin and malleable bone tissue and does not have the rigidity and strength of the adult skull. Moreover, the calvarium bones of a child are separated by sutures that have not been fused yet. According to Lancon and Haines this makes the child's calvarium relatively resistant to fractures [28]. In their opinion fracturing of the child's calvarium takes a significant trauma. However, the question is whether this assumption is correct. Weber is of the opinion that a number of sites on the child's immature calvarium have an increased susceptibility to fracturing. This applies in particular to the parietal bone in infants [177].

In relation to the adult skull, these specific properties of the infantile skull enable it to tolerate greater deformation before it breaks. This deformation may even lead to a depression of the cranium without fracturing (the so-called ping-pong fracture/deformation—Sect. 5.3.4.5).

Deformation in Dynamic Impact Loading: Trauma-Related and Anatomy-Related Factors

The degree of deformation of the calvarium at the moment of fracturing and the nature and size of the fracture and the associated injuries will depend on trauma-related factors (the location of the impact and the amount of energy transferred during the impact) and on anatomy-related factors, concerning the scalp, the age of the child and the shape, build, thickness, and malleability of the calvarium at the point of impact and at other sites (after DiMaio and Dimaio [26]):

Trauma-Related Factors: Location of the Impact

The location of the impact determines only to a certain extent the location, nature, and extent of the skull fracture. Damage to the scalp, however, is an important indicator for the primary site of impact. For this reason, a precise registration of external injuries is always required, in particular, when inflicted injuries are suspected. In 80% of children with a calvarium fracture external injuries are found that indicate a head trauma. In 84% of children fractures were found ipsilateral and in 16% contralateral from the point of impact [174]. However, the absence of external injuries does not exclude a calvarium fracture.

A study of adults who had sustained a calvarium fracture showed that, depending on the place of impact, different types of calvarium fractures can result from equal amounts of energy. It is not clear whether this can also be applied to children and, if so, whether this is the same for every age group. An impact trauma on top of the cranium will usually lead to a calvarium fracture that may carry on into the temporal region or the base of the skull. A blow to the occipital region will usually lead to a linear fracture in the posterior cranial fossa. A blow to the temporoparietal region may cause a fracture that runs through the temporal bone to the base of the skull. A blow to the forehead causes a fracture that may run into the orbit and even into the maxilla [178].

Trauma-Related Factors: Amount of Energy Transferred At the Impact

The amount of energy, that is transferred at the impact, is determined by four elements:

- The shape, weight, and nature of the object. It may be a solid object that will not give way during contact (e.g. a hammer, concrete floor, or stone) or a more or less soft object with a surface that gives way at contact (e.g. a mattress or a floor covered with thick, soft carpet). In soft and yielding objects, the deformation of the surface will absorb a large part of the energy released at contact. Yet, the literature has shown that a child falling on a soft surface can also sustain a fracture [177]. On a solid and not flexible surface hardly any energy is transferred to the object.
- The velocity resulting from the speed of the head and the object at the moment of impact.
- A fixed or free-moving head. When the head can move freely, it will move along in the same direction as the impacting object. In this manner, part of the energy at impact is absorbed by the movement.
- The size of the impact surface. If the impact takes place on a limited surface, all energy released at impact will be concentrated at this surface. If the site of impact is larger, the energy will spread itself over this surface.

Anatomy-Related Factors: The Scalp

The calvarium is covered by the scalp, which consists of five layers: the skin (epidermis and dermis), a layer of fatty/connective tissue, a layer of strong fibrous tissue (the epicranial aponeurosis or galea), a layer of loose areolar connective tissue and the periosteum of the skull (pericranium) (Sect.

5.2.1.1) [21]. Tedeschi showed that when force is exerted on the calvarium, the scalp, if intact, will protect against fracturing [65]. Compared to when the skin is present, the risk for a fracture increases tenfold when no skin is present [65].

Anatomy-Related Factors: The Age of the Child

In a short-distance fall, children with open sutures and a thinner albeit more malleable skull will generally sustain a fracture less often than older children with closed sutures and a more rigid skull. Yet, children under 1 year of age can sustain a skull fracture in a relatively small trauma, in spite of the substantial malleability of their skull (Sect. 5.3.5). Children under 1 year of age than 1 year old are six times more likely to sustain a skull fracture than older children [179–181]. However, this will only rarely lead to serious or life-threatening intracranial injury (Sect. 5.3.5; see also Chap. 13). Ibrahim and Margulies concluded, based on an anthropomorphic dummy analysis, that toddlers (aged 2–4 years) might have fewer skull fractures and soft tissue injuries of the scalp and face than infants below the age of 1 year after the same short-distance fall, but that toddlers might be more vulnerable to neurological impairment/altered mental status (see also Sect. 13.3.2.2) [182].

Anatomy-Related Factors: Shape, Build, and Thickness of the Skull

The cranium is constructed of two layers of bone with a spongy structure in between (diploid). The inner layer of compact bone is the most vulnerable. On impact this layer may be damaged, whereas the outer layer does not suffer any damage. When the impact generates enough energy, the outer layer will fracture too and this may result in loose bone fragments (Fig. 5.14). Young children do not have a diploid structure of the parietal bone, leading to an increased risk for sustaining a fracture in this bone in a short-distance fall [177].

5.3.3 Manner of Calvarium Fractures

As stated before, calvarium fractures are regularly seen in children. Irrespective of the cause (Sect. 5.3.2), there may be various circumstances under which calvarium fractures are sustained (accidental or non-accidental circumstances). In relation to the manner the different moments in which the fractures can be sustained, should be taken into consideration: trauma before birth (Sect. 5.3.3.1), during birth (Sect. 5.3.3.2), and after birth (Sect. 5.3.3.3: static loading; Sect. 5.3.3.4: dynamic impact loading).

Trauma after birth can be due to accidental and non-accidental circumstances. Calvarium fractures, regardless of the circumstances (accidental or non-accidental), are more common in younger children than in older children [169]. Concerning these circumstances, differences are found between age categories [179, 181, 183–185]:

Fig. 5.14 Schematic representation of the relation between increasing levels of energy and type of fracture in dynamic impact loading

- In children under the age of 1 year, non-accidental circumstances (inflicted fractures) should always be considered, although trauma before birth or during birth due to the passage through the delivery channel or due to medical procedures during birth (the use of a forceps or vacuum extraction) or an accidental fall after birth should not be ruled out in advance.

- In children from approximately the age of 1 year (provided they are sufficiently mobile) up to the age of 4 years, accidental trauma due to falls during play seems to be most common.
- In children between the ages 4 and 14 years, the most common circumstances are accidental trauma in traffic accidents and non-accidental trauma in physical assaults.

Children with a bone disease can sustain a (calvarium) fracture at a lower level of loading than children without a bone disease.

5.3.3.1 Trauma Before Birth

Calvarium fractures, although rarely reported, are the most commonly described intrauterine fractures in medical literature [186, 187]. Intrauterine calvarium fractures can be caused by static loading and by dynamic impact loading.

Trauma Before Birth Due to Static Loading

Static loading is the most common cause of intrauterine calvarium fractures. Static loading can cause true fractures with clearly recognizable fracture lines, although this is rare [186]. Static loading most commonly causes depressed skull fractures, which are characterized by deformation of the calvarium, due to inward buckling of the calvarian bones without a recognizable 'true' fracture, so-called 'ping-pong' fractures (Figs. 5.15 and 5.16a, b) [186, 188, 189]. These fractures usually are found in the parietal and temporal bones [190], and should not be mistaken for fractures during birth (Section "Trauma During Birth Due to Static Loading", Fig. 5.17).

According to Preston et al., the finding of a depressed skull fracture in a neonate is rare, estimated between 4 and 10 in every 100,000 live births in western countries [191]. They, however, did not differentiate between depressed fractures/ping-pong fractures that were sustained before or

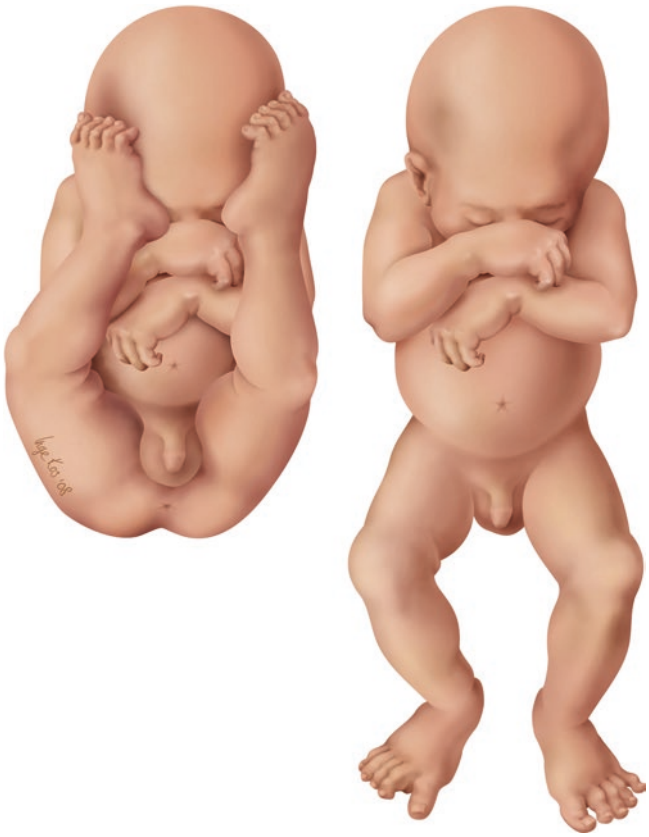


Fig. 5.15 Schematic drawing of the development of congenital impressions

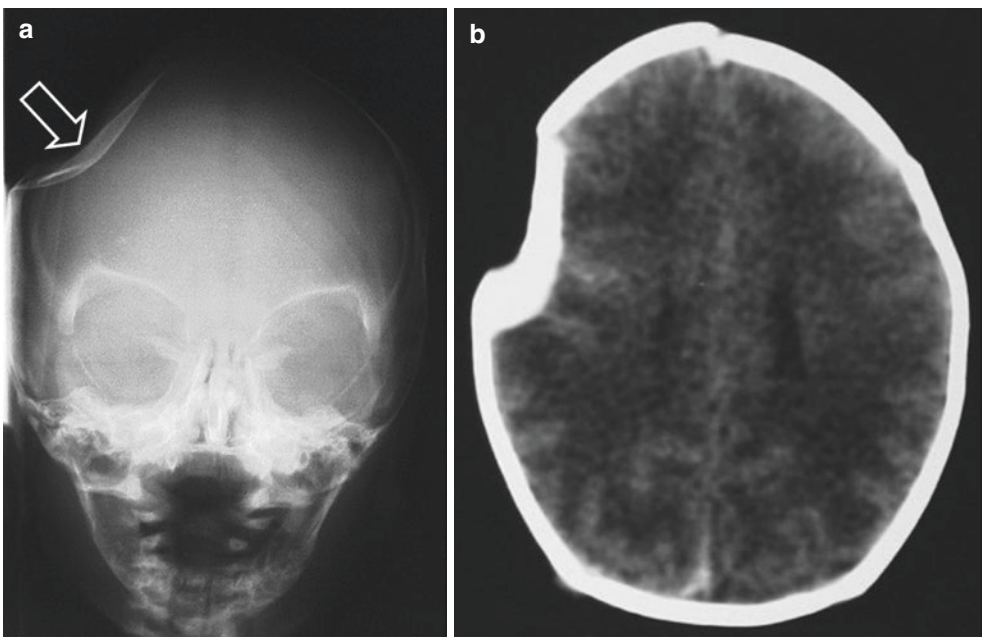


Fig. 5.16 Three-day-old boy after prolonged breech position and uneventful delivery. (**a**) At physical examination a depression of the right parietal bone was found, which was confirmed on a skull radiograph (arrow). (**b**) CT showed no soft tissue swelling or brain lesions (**b**). Given the history, this was considered a congenital abnormality caused by longstanding static loading

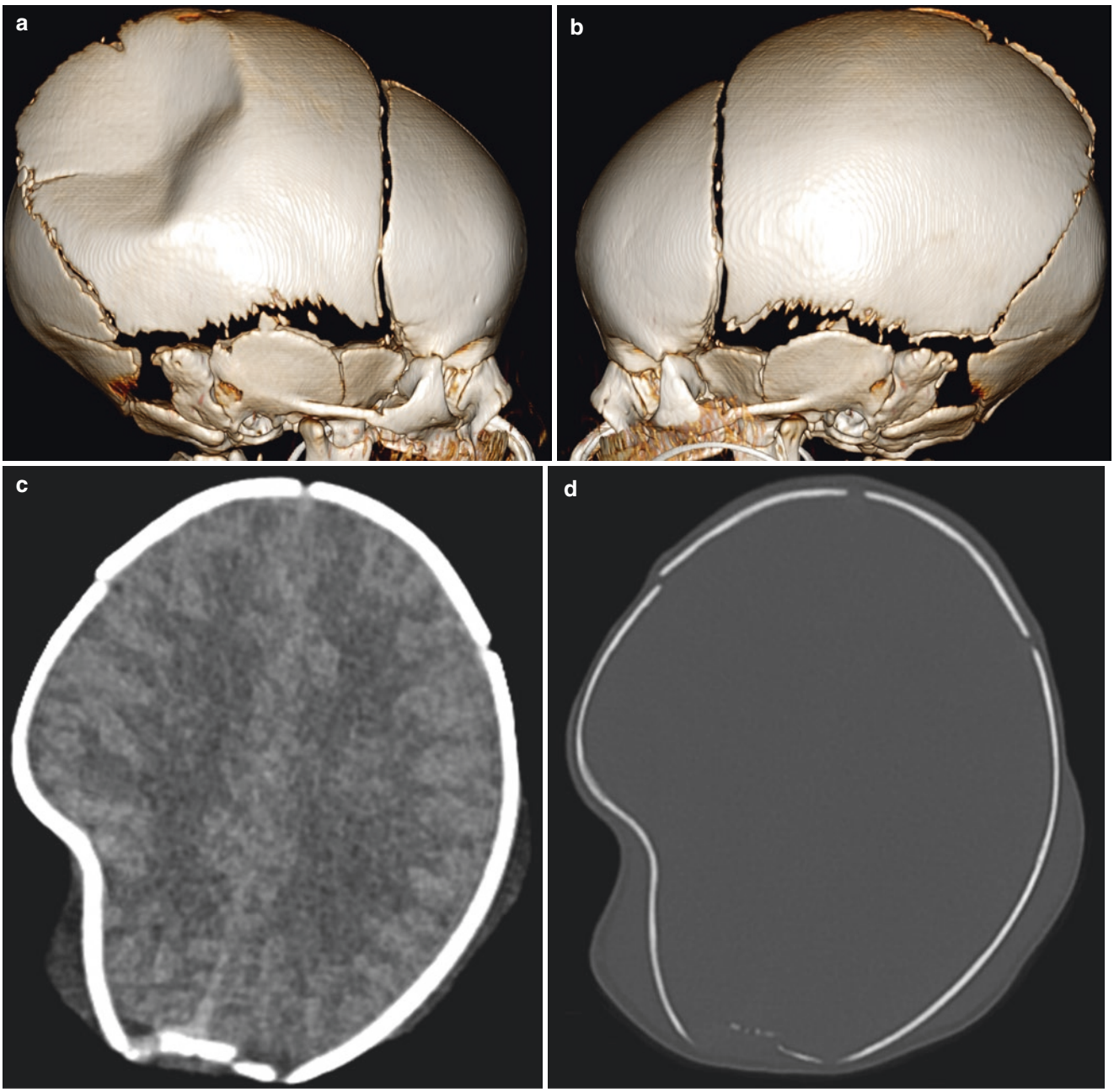


Fig. 5.17 Large depressed ('Ping-Pong') fracture in a term infant with a complicated delivery: slipped vacuum extractor followed by emergency caesarean section. The depression of the right parietal bone is well demonstrated on CT with 3D rendering (**a**) versus normal contralateral side (**b**). Corresponding slices (**c**, **d**) show posttraumatic soft tissue swelling but no intracranial damage. Uneventful recovery with gradual remodelling

during birth due to the use of instruments, although the authors stated that these types of fractures are due to instrumental delivery or pressure from the delivering doctor or midwife's hands during obstetric manoeuvres in a difficult delivery. Dupuis et al. retrospectively analyzed the data of 1,994,250 deliveries and found 68 neonates with depressed skull fractures [189]. Eighteen of these fractures were determined to be sustained intra-uterine. Fifty were determined to be due to the use of instruments during the delivery.

Brodhurst probably was the first to describe this type of intrauterine skull fracture: '*There are also numerous instances of fracture of the foetal skull on record, as well as of depression of the cranial bones*' [192]. According to Guha-Ray 'ping-pong' fractures are less rare in Africa among African women and possibly in other developing

countries than in Western countries [193]. Most intrauterine ping-pong fractures resolve spontaneously within 6 months without neurological deficits [194–198]. (Neuro)surgical interventions or non-surgical interventions are only rarely needed [199]. Intracranial bleeding (e.g. intraventricular haemorrhage), intracerebral injuries, and severe long-term neurological sequelae, like a developmental delay or spastic hemiplegia are rare and probably mainly found in case of maternal abdominal trauma, due to dynamic impact loading [200].

Based on their own experience with six neonates and a review of the literature, Alexander and Davis concluded that almost all depressed intrauterine calvarium fractures ('ping-pong' fractures) were caused by compression of the skull against the promontory of the sacrum of the mother [188]. This conclusion was consistent with what previously was supposed to be the cause [201–204].

Ben-Ari et al. evaluated the data of 29,137 births in 8 years and found depressed fractures in three neonates cases (0.01%) [205]. Two were determined to be intrauterine sustained. In one case, the right hand of the neonate showed slight shortening of the second digit and a more than usual flexor position of the fingers. The depression on the skull fitted the size of the hand and the authors suggested that prolonged pressure of the hand caused the depression of the skull. In another case, a large myoma of the left inferior wall of the uterus was thought to have caused the skull depression.

Nowadays, accepted explanations for the occurrence of intrauterine calvarium fractures, due to static loading, are continuous or prolonged compression of the child's head [186–188, 200, 206–208]:

- Against maternal bony structures (sacral promontory, L5 vertebra, pubic symphysis, ischial bone, asymmetric pelvis) or against uterine fibromas.
- By the child's own hands/fists or feet/heels.
- By a twin's hands/fists or feet/heels.

Sometimes no cause will be found.

Trauma Before Birth Due to Dynamic Impact Loading

Dynamic impact loading may involve (non-penetrating and penetrating) blunt force trauma and (non-penetrating and penetrating) sharp force trauma. The majority of trauma before birth, due to impact loading, occurs in accidental circumstances (motor vehicle/traffic accidents, followed by accidental falling). With the increasing number of traffic accidents the incidence of accidental in utero trauma may also be increasing. Impact loading can also occur in non-accidental circumstances, e.g. in physical assaults (including gunshot and stab wounds) or suicide attempts [209–211].

The incidence of trauma due to impact loading of the pregnant woman and her unborn child is not exactly known, but in the past the incidence of this type of trauma was estimated to be 6–7% [212, 213]. Injuries to the expectant mother, which carry a high risk for maternal and neonatal morbidity and mortality are abdominal trauma, pelvic fractures, and penetrating trauma [211].

In utero calvarium fractures due to maternal trauma have been mentioned in the medical literature for over a century [206]. Although it is possible for fractures to occur in every bone of the unborn child, calvarium fractures appear to be the most prevalent in utero trauma [212, 214]. In utero calvarium fractures can be found in all bones of the calvarium [215]. Multiple depressed calvarium fractures may also occur [216].

Calvarium fractures may be accompanied by serious injuries that are sometimes incompatible with life. Intracranial (subdural/subarachnoid, intraventricular) haemorrhages, cerebral oedema, hypoxic and ischaemic damages, and parenchymal injuries have been reported [215, 217, 218].

Trauma Before Birth Due to Dynamic Impact Loading: Accidental Circumstances

The majority of calvarium fractures in utero are related to severe maternal injuries (fractures of the pelvis). As a result of the fracture and dislocation of the pelvic bones, the calvarium is pressed against the sacrum with great force [206]. The highest risk in case of blunt abdominal trauma is during the third trimester, when the skull has descended into the pelvis [187].

The occurrence of calvarium fractures in the unborn child is often accompanied by severe maternal trauma. Weigel described the case of a pregnant woman who died from haemorrhage, due to a ruptured uterus after a motor vehicle accident [219]. Sadro et al. reported a case of detachment of the placenta due to a blunt abdominal trauma, which also resulted in intrauterine fractures of the calvarium/lethal foetal head injury [220].

Severe maternal injuries, however, are not always present. Stafford et al. described eight cases of in utero foetal trauma (two children had sustained calvarium fractures with cortical lacerations and contusions) [218]. Six of the eight children were stillborn, and the other two died during the first postnatal day. In all cases, the mother survived, usually with only limited injuries. At least five of the mothers were unrestrained at the time of the accident, three of whom experienced abdominal impact against the steering wheel but no external abdominal injuries. Their study showed that lethal placental or direct foetal injury, e.g. of the head, can occur even though maternal injuries are minor or insignificant.

Härtl and Ko described the case of a 19-year-old pregnant woman who had been involved in a traffic accident and had no significant injuries [206]. Due to foetal distress, it was decided to perform a Caesarean section. The child was found

to have a linear fracture in the left parietal bone plus a scalp haematoma on the left side at the location of the fracture. Both mother and child had an excellent recovery. The authors assumed that the fracture was caused by blunt trauma directly through the abdominal wall during the accident.

Assad et al. described the occurrence of a depressed calvarium fracture with a massive subgaleal and subperiosteal haemorrhage in an unborn child after a motor vehicle accident, requiring neurosurgical intervention with good clinical outcomes for both the expectant mother and unborn infant [99].

Trauma Before Birth Due to Dynamic Impact Loading: Non-accidental Circumstances

Tattoli et al. reported the case of a 46-year-old woman, 34 weeks pregnant, who attempted suicide by jumping from a flyover, immediately after a probably deliberate traffic collision with the guardrail [211]. She had fractures of five lumbar vertebrae and three ribs with pulmonary contusions, but had no other injuries. Following the mother's stabilization, the foetal heart tones were detected as abnormal and the patient had an emergency caesarean section delivering a stillborn male infant. Neither alcohol nor drugs were found in the mother who had been diagnosed with an unspecified episodic mood disorder. She recovered completely from her injuries. At autopsy of the newborn, a massive subarachnoid haemorrhage with deformity of the skull was found, caused by maternal blunt abdominal trauma following a car accident and fall.

Moscote Salazar et al. described the occurrence of a penetrating head trauma with skull fracture and intraventricular haemorrhage in a foetus after his mother had violently self-inserted a blunt object through the vagina [210]. The integrity of the intrauterine membranes was disrupted, which resulted in preterm labour. After birth, the child was surgically treated (debridement of the scalp and surgical management of the fracture), but the child died 4 weeks later, due to neonatal sepsis. Only a few intrauterine head stab wounds have been reported [209, 221–223].

Grubb reported a 26-year-old Hispanic woman, 30 weeks pregnant, admitted to the hospital with multiple stab wounds in the abdomen and upper extremities, inflicted by her husband [223]. She and her unborn child were managed non-surgically by serial examinations and continuous foetal monitoring. At 34 weeks she was admitted to the hospital in labour. An energetic girl (weighing 2540 gm) was delivered spontaneously. An Apgar score of 8 was noted after 1 and 5 min. A poorly healed laceration, about 5 mm long, was noted over the infant's left parietal bone and closed spontaneously on the second day of life.

Schultz et al. and Avenarius et al. described the same case: a 19-year-old woman in her 29th week of pregnancy who was stabbed in the lower abdomen during a robbery [221, 222]. Two hours later, an emergency caesarean delivery was performed. The APGAR scores of the newborn were low: 2, 5, and 6 at 1, 5, and 10 min, respectively. The child had a 2-cm-long bleeding wound above the right ear and an extensive intracerebral haemorrhage in the region of the right thalamus and temporal lobe with a visible 5–6-cm long incision channel, stretching about 1 cm beyond the midline. A large, right-sided subdural hematoma near the laceration site and intraventricular bleeding had occurred. Surgical evacuation of a subdural hematoma was undertaken. The child developed a post-haemorrhagic hydrocephalus and a left spastic hemiparesis.

Gallo et al. reported the case of a 20-year-old woman who was stabbed in the lower abdomen when she was 30 weeks pregnant [209]. She and her unborn child were managed non-surgically by serial examinations and continuous foetal monitoring. Spontaneous vaginal delivery occurred at term with good maternal and foetal outcomes. The child had a right temporal swelling, which was interpreted as a subcutaneous haemangioma. At the age of 2 years and 6 months, the child had a pulsating bulge in the right temporal region. Clinical examination and imaging were indicative of a typical growing skull fracture, due to a bone defect and dural tear with a good neurological and aesthetic outcome (see also Sect. 5.3.4.6).

Disease-Related Intrauterine Calvarium Fractures

Disease-related intrauterine calvarium fractures are only very rarely described in the medical literature.

Plotkin described the occurrence of intrauterine calvarium fractures in osteogenesis imperfecta type II [224]. Bar-Yosef et al. described a neonate with multiple congenital calvarium fractures and intracranial bleeding [225]. He also had multiple skin folds suggesting a connective tissue abnormality. The child was diagnosed with Ehlers-Danlos syndrome type VIIC. Solomons et al. also described a neonate with congenital calvarium fractures and skin lacerations at birth, who was diagnosed with Ehlers-Danlos syndrome type VIIC (Dermatosparaxis Ehlers-Danlos syndrome) [226].

5.3.3.2 Trauma During Birth

Trauma During Birth Due to Static Loading

Calvarium fractures due to birth trauma usually are caused either by static loading due to the delivery itself caused by pushing/compressing the child's head against the mother's pelvis or due to medical procedures during the delivery caused by the use of instruments (forceps—compression, vacuum extractor—traction). In uncomplicated deliveries, skull/calvarium fractures, however, are very rare.

Rubin did a prospective study on 15,435 births and only found only one skull fracture [227]. Two other studies showed 11 skull fractures in a total of more than 51,000

births [228, 229]. In an article (letter), Groenendaal and Hukkelhoven reported 1174 fractures in 158,035 births that were due to birth trauma, none of which were skull/calvarium fractures [230].

Most calvarium fractures that result from birth are uncomplicated linear fractures in the parietal bone. This kind of fracture almost always concurs with a difficult delivery or externally applied mechanical force. For example, skull fractures are found in 5% of children that had had vacuum extraction [231]. The risk of sustaining a skull fracture when vacuum extraction is used increases considerably when the cup releases unexpectedly and has to be re-applied (Fig. 5.17a, b). Bruising of the scalp may indicate the presence of a calvarium fracture. The risk also increases with maternal age, primigravida, and macrosomia. Yet, a simple linear fracture may also occur in a normal spontaneous vaginal birth without specific complications or the use of forceps or vacuum extraction [232].

Ben-Ari et al. evaluated the data of 29,137 births in 8 years and found depressed fractures in three neonates cases (0.01%) [205]. Two were determined to be intrauterine sustained. Only one of these fractures was determined to be instrument (forceps) related.

Dupuis et al. found a depressed fracture immediately after birth in 1 in 26,000 neonates [189]. This fracture may be sustained in utero (Sect. 5.3.3.1) or during birth due to the use of instruments during the delivery. Complicated skull fractures occur mainly with forceps deliveries, but depressed fractures have also been reported with excessive manipulation during a Caesarean section or vacuum extraction [233, 234]. A growing skull fracture has been reported twice as resulting from vacuum extraction [235, 236]. Rupp et al. described circular fractures and/or elevation of the outer layer of the skull, subperiosteal and intra-osseous haemorrhages, and epidural and subdural haemorrhages as complications of a vacuum extraction [237].

A Caesarean section seldom leads to skull fractures. Alexander et al. found 418 children with injuries in a total of 37,110 Caesarean sections [238]. Six of them sustained a skull fracture due to complicating factors prior to the Caesarean section, such as complications resulting from an earlier effort at a vaginal delivery.

There is a considerable chance that a linear fracture is not detected directly after birth. Complex skull fractures are usually visible immediately after birth and are often accompanied by marked and acute intracranial injuries [233].

During the first months it is, based only on the radiological evidence of the fracture, generally impossible to differentiate whether the calvarium fracture resulted from trauma during or after birth, irrespective of the circumstances (accidental or non-accidental) under which the fracture was sustained [184]. Calvarium fractures in children under the age of 1 year tend to heal without notable callus formation. An uncomplicated linear fracture that was sustained during delivery will no longer be well visible after 2 months, due to fading of the fracture lines and will no longer be visible after 6 months [184].

The chance of a skull fracture after an uncomplicated delivery is negligible, especially when the new born does not show any visible swelling or neurological symptoms. Of the children with a scalp haematoma, 10–25% may have a calvarium fracture [239, 240].

Disease-Related Calvarium Fractures Due to Trauma During Birth

For the incidence of calvarium/skull fractures as birth trauma in children with congenital disorders, such as osteogenesis imperfecta or Menkes disease, we refer to Chap. 14.

5.3.3.3 Trauma After Birth: Static Loading

Accidental Circumstances

Most head injuries are caused by accidents involving dynamic impact loading (acceleration and/or deceleration) [241, 242]. Head injuries due to static loading are much less common than those caused by dynamic impact loading [241, 242]. Accidental static loading most commonly occurs due to accidents, in which the child's head is more or less immobilized against a rigid structure, e.g. due to wedging. In traffic accidents static loading often is combined with dynamic impact loading, e.g. impact of the head, while being hit by a moving vehicle, followed by wedging of the head for a period of time or when a wheel runs over the head, in which the head lies more or less stationary and is pressed against a rigid structure.

Immobilization/wedging can result in bilateral compression of the child's head, which most commonly occurs at the site of temporal bones, leading to static loading of the head in a transverse axis [241]. Due to the compression/static loading, the skull is deformed relatively slowly, eventually leading to crushing of the head and, according to Duhaime et al. associated damage to the intracranial structures, such as the brain [243]. According to Gonzalez Tortosa et al., bilateral compression produces basilar fractures that cross the midline structures, however, without usually producing significant cerebral damage [241]. López-Guerrero et al. described the findings in 19 children with crushing head injuries (13 boys and 6 girls; mean age 4.1 years) [242]. All children had externally visible injuries (scalp injuries or bleeding from the nose, ears, or throat). Basilar fractures were seen in most cases with extension to the calvarium in 11 children. Although Gonzalez Tortosa et al. stated that bilateral compression did not produce significant cerebral damage, López-Guerrero et al. found associated intracranial lesions in 14 children, including two with diffuse axonal injury [241, 242]. Eleven children were initially unconscious. Six children had cranial nerve defects in addition to

impaired hearing. Surgery was needed in three children. Only seven children had long-term sequelae.

Duhaime et al. reported on seven children between the age of 15 months and 6 years that had sustained crush injuries [243]. They all suffered basilar fractures and six of them had multiple and often extensive fractures of the calvarium. The authors did not report whether the 7th child, who died soon after arriving at the hospital due to transection of the cervicomedullary myelum, had sustained any other fractures besides the earlier mentioned basilar fracture. Four children were victims of traffic accidents, and had been run over by a reversing car. In the three other children there was, according to the authors, static loading when the child climbed on a heavy object or pulled at a heavy object that consequently dropped on the head of the child (solid stone front of a fireplace, 27-inch television, 45 kg clock). However, the question is whether in these three children one can speak of static loading. It could also be dynamic impact loading, in which the child falls on the floor with its head more or less stationary on the underlying surface and the object falling on the child and crushing the head. This can be compared to the effects of a fall from great height, which may also lead to multiple and extensive fractures of the calvarium.

According to Partrick et al. driveway-related trauma in children (being struck or run over in a driveway), resulting in crush injuries of the head/closed head injury, carried a tenfold increase in mortality in children under 5 years of age when compared with all other paediatric pedestrian accidents with a mortality rate of only 2% [244].

Prasad et al. evaluated the data concerning crush injuries of the head in eight children (ages 13–32 months) [245]. They found that all children sustained pronounced head injuries, characterized by multiple fractures throughout the calvaria, extra-axial haemorrhages, and parenchymal contusions. Cranial nerve injuries were found in three and hemiparesis in two children. One year after the trauma, five of the six children had a good recovery. The authors concluded that the neuropsychological outcome after brain injury produced by static loading of the head was more favourable than from traumatic brain injury due to dynamic loading.

According to Takeshi et al., serious crush injuries of the head usually are fatal. They also pose that the prognosis of this type of injury, either lethal or excellent, depends on the extent to which the skull and brain have been able to withstand the force [246]. Six of the seven children (three boys, four girls; average age: 5.9 years) they described had sustained skull fractures. In six children the head had been run over by the wheel of a car. The head of one child had been crushed by press machine. In four children multiple linear fractures of the calvarium were found and in six children a basilar fracture was found. Four children died. The three surviving children only had cranial nerve palsies, which recovered completely.

Brison et al. concluded the same as Takeshi et al. concerning the fatal course of crush injuries of the head in agricultural accidents, especially in pre-school children, due to being run over by agricultural machinery as a bystander or as an extra rider who fell from the machine [246, 247].

Non-accidental Circumstances

Static loading of the head, due to compression, can occur in non-accidental circumstances. In several (unpublished) cases, one of us dealt with (RB), compression of the child's head was mentioned, e.g. by wedging of the child's head between the arm and trunk of a caregiver or by sitting or standing by a parent/caregiver on the child's head. However, in these cases it could not be determined which part of the injuries to the head were caused by the compression or by other mechanisms, e.g. dynamic impact loading. No reports were found in the medical literature, in which non-accidental compression irrefutable has led to (intra)cranial injuries.

5.3.3.4 Trauma After Birth: Dynamic Impact Loading—Accidental Circumstances

As stated before most calvarium fractures and other head injuries, including intracranial injuries, are caused by dynamic impact loading with acceleration and/or deceleration due to a direct impact of the head against a hard object or surface. Calvarium fractures are common injuries in young children and can be sustained in accidental or non-accidental circumstances [241, 242, 248, 249].

Accidents: Falls

In a young child, an accidental fall, in which only the effect of gravity is involved, will usually lead to an impact trauma of the head, being the first to be impacted. This is due to the disproportional size and the disproportionately high weight of the head (see also Sect. 5.3.1). If a calvarium fracture is found in a young child, an accident of sufficient magnitude, will be enough to explain the occurrence of the fracture adequately [249].

In the medical literature a short-distance fall usually is defined as a fall over a distance of less than about 1 m (around 3 ft), an intermediate-distance fall as a fall over a distance of between about 1 m and 3 m (10 ft) and a long-distance fall as a fall over a distance over 3 m [170].

For practical reasons we defined falls in this section as follows:

- Short-distance fall: Fall over a distance of less than 1.5 m (5 ft).
- Intermediate-distance fall: Fall over a distance between 1.5 and 3 m (5–10 ft).

- Long-distance fall: Fall over a distance of than 3 m (10 ft).

Furthermore, the results of studies concerning certain short-distance and intermediate-distance falls will be discussed combined because these studies either did not differentiate between short- and intermediate-distance falls, or described falls over a distance of less than 1 m up to 3 m.

Falls, irrespective of their distance of falling, can also be divided into uncomplicated and complicated falls. An uncomplicated fall is defined as a fall on a flat surface in which only the effect of the gravity is involved. A complicated fall is defined as a fall on a flat or uneven surface, in which extra loading of the head/skull is added to the effect of gravity. This extra loading may occur:

- At the initial moment of a fall, e.g. falling from the arms of a caregiver, from a swinging swing or with a baby walker.
- During falling, e.g. when falling off a bunk bed, in which the child comes into contact with parts of the bed while falling, or when falling with a baby walker from the stairs. This can also occur when a caregiver falls with a child on his/her arms.
- At the landing, e.g. falling on a non-flat surface or falling on objects, like toys. This can also occur, when a caregiver falls with a child and falls fully or partly on the child.

In case of extra loading at the initial moment of a fall or during falling this may result in a combination of the loading due to height of the fall, caused by the gravity, with a 'forward momentum' and the 'propulsive force exerted by the moving swing, baby walker or caregiver' (Fig. 5.18a, b) [249].

Most accidental falls, especially in young children, are preventable if adequate precautions are taken, adequate supervision is present and, in case of the use of equipment like baby bouncers, baby walkers, or car seats, the safety standards are maintained [250].

A delay in seeking medical care in children with injuries is often considered to be supportive for a non-accidental aetiology. Metz et al. evaluated the occurrence of delayed seeking of medical care (frequency and patterns) in 210 children under the age of 4 years (2011–2012) with accidental skull fractures [251]. Delays were defined as seeking medical care after a period of 6 h or more after the accident. 'Minor accidents' included falls <4 ft and low force trauma, while 'major accidents' included higher height falls and major force events. Delays were less likely with major accidents (4.9%), than with minor accidents (25.8%) (RR = 0.32 (0.15–0.70)). Children came to care for soft tissue swelling (STS) (39%), the injury event (36.2%), altered consciousness (15.2%), and vomiting (10.5%). Delayed onset of STS (78.6%) caused most delayed care. Early STS was firm, (17.6%) vs. delayed (5.0%), as opposed to soft or fluctuant. Delayed care seeking is common for minor, but not major accidental infant and toddler skull fractures. Most followed delayed onset of signs and symptoms. Metz et al. concluded that delayed seeking of medical care in children with skull fractures in itself does not imply non-accidental circumstances.

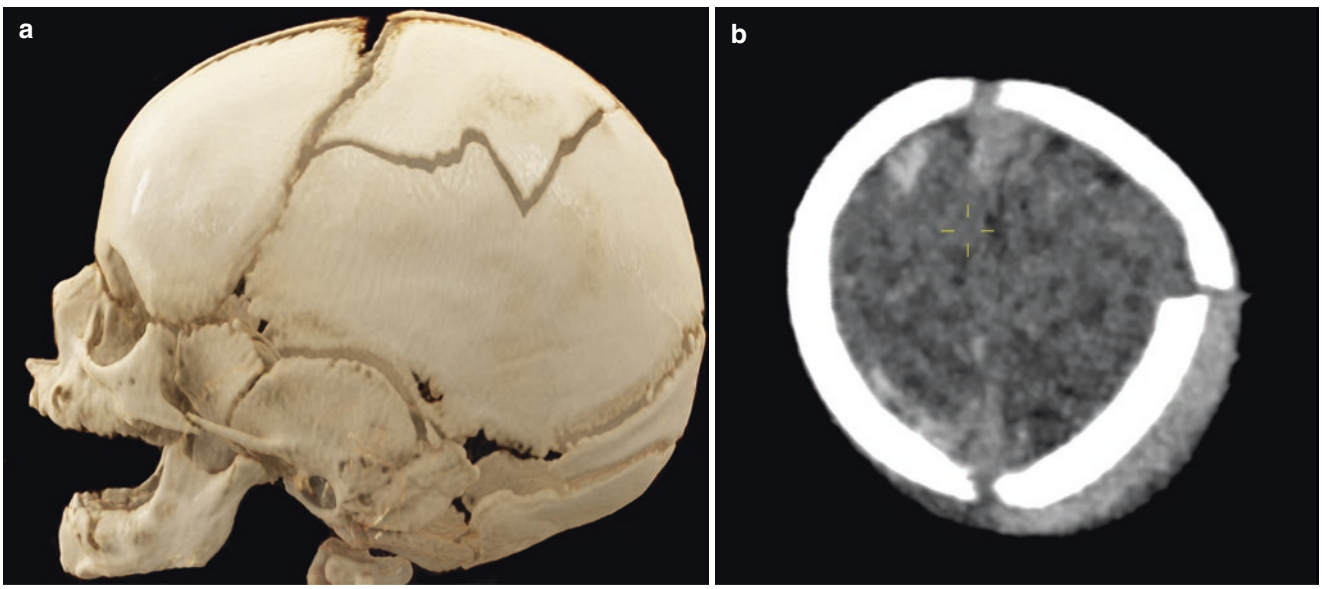


Fig. 5.18 Complicated fall resulting in a simple zigzag fracture (**a**) in a 4-week-old infant. His mother twisted her ankle and fell while carrying the child in a baby wrap carrier (see also Fig. 5.20b). She could not prevent the head from hitting the pavement. Despite the subgaleal hematoma and subarachnoid blood (**b**) the infant did not have neurological symptoms

Accidental Short-Distance Falls

An uncomplicated short-distance fall is defined as a fall in which only the effect of gravity is involved and in which usually the fall occurs over a distance of less than 1.5 m [66, 67, 69, 71, 249, 252]. In other words: an uncomplicated fall is a short-distance free fall on a flat surface. The fall originates from a position in which the child stands still or lies still and the fall is the result of the child's own movement patron in accordance with its level of development, e.g. a situation in which the child falls from a changing table because it turns over, or when a child falls over while standing because it loses balance. Data on how often a child sustains a skull fracture after an uncomplicated fall have been derived from fall studies in living and in deceased children. According to Offiah and Hall every loading that is added in a short-distance fall to the effect of gravity increases the chance that the child sustains a fracture of the calvarium [249].

Uncomplicated Short-Distance Falls in Living Children

Many authors emphasize that the occurrence of a calvarium fracture (and/or intracranial injuries) after an uncomplicated short-distance fall will be rare, unless there are complicating factors [66, 67, 69, 71, 252, 253]. Their opinion probably is correct, but calvarium fractures, sustained due to an uncomplicated short-distance fall, hardly ever will cause alarming clinical symptoms (Fig. 5.19) (Sect. 5.3.1). Hence, no medical help usually will be sought and therefore no additional examination will be done. Consequently, falls, irrespective of the circumstances, may result in a larger number of calvarium fractures than one would deduce from data in the literature. No consensus exists about the minimal distance a child must fall to sustain a skull fracture.

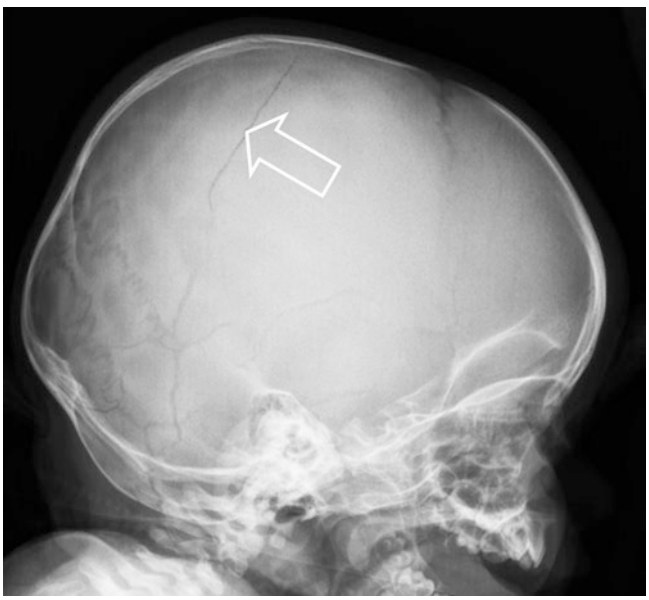


Fig. 5.19 Eight-month-old infant with a soft tissue swelling of the head after a fall from a bed. Radiological examination shows a linear fracture of the skull (open arrow)

Data on how a living child sustains a calvarium fracture after an uncomplicated fall can be derived from the evaluation of data concerning accidental falls, which were more or less observed by independent bystanders or happened in more or less 'controlled' circumstances:

- Helfer et al.: 246 children under the age of 5 years with a history of a short-distance fall [66]. The group consisted of 161 children whose parents filled out a questionnaire when they saw a physician for a fall over a distance of less than 90 cm (bed or settee) and 85 children who had fallen from their crib/cot or from the examination table during their stay in hospital. Two children (1%) in the group that had fallen outside the hospital had sustained a calvarium fracture. Both children were younger than 6 months of age. In the children who had fallen while hospitalized, one calvarium fracture (1%) was found. The majority of children did not have any externally visible injuries.
- Nimityongskul and Anderson: Seventy-six children, age range from neonate to 16 years, who had fallen out of bed, crib/cot, or chair while being hospitalized [69]. Fifty-seven children were under the age of 5 years and 23 children were under the age of 1 year. The measured fall distance was between 30 and 100 cm. Most children had superficial injuries (haematomas of the scalp and abrasions/lacerations of the face). One 12-month-old child had an uncomplicated fracture of the occipital bone.
- Lyons and Oates: Two hundred and seven children under the age of 6 years who had fallen from their crib/cot (n = 124) or bed (n = 83) [67]. The fall distance ranged from 65 cm (lowered side rail) to 110 cm (side rail up) in a fall from a crib or cot, and from 50 to 85 cm (including side rails) in a fall from a bed. In 31 children there were visible injuries, in 26 of these children injuries to the head were visible. None of the children who fell from a bed sustained a fracture. In the group of children who fell from their cot/crib only one child (<1%), aged 10 months, had an uncomplicated linear skull fracture.
- Tarantino et al.: One hundred sixty-seven children under the age of 10 months (average age 5.2 months, 56% male), who fell over a distance of less than 1.25 m and for that reason were presented at the emergency department [70]. They excluded all falls from baby walkers and car seats, falls down the stairs and all accidents resulting from walking, running, or climbing. They also excluded children that had fallen on objects or on whom a caregiver had fallen. Fifty-five percent of children fell out of bed, 20% fell from the arms of a parent/caregiver (being dropped), 16% fell from a couch and 10% fell in a different manner. Eighty-five percent of children had no or minimal injuries. The remaining children (n = 25) had

more or less severe head injuries or other injuries: 16 of them had a closed-head injury, of whom 12 had a calvarium fracture, and seven had a fracture of one of the long bones. Infants that dropped from the arms of the caregiver had the highest risk of sustaining significant injury (odds ratio 6,4 versus rolling from a bed or other surface). They concluded that the biomechanics of a fall from the arms of a caregiver may be different from other kinds of short-distance falls.

- Warrington and Wright: Requested parents of 6-month-old children, enrolled in the Avon Longitudinal Study of Parents and Children, to describe every accident since birth using questionnaires [71]. They asked the parents to describe the type of fall, the distance of the fall, the injury and, the medical help given (in case this was sought). The number of forms returned was 11,466 (81% of the total study cohort), in 2554 children, a total of 3357 fall incidents were reported. Fifty-three percent of children fell out of bed or from the settee, and 12% fell from an arm while being carried or when the person who carried the child fell down while holding the child. In the remaining children, a large diversity of falls was seen: from a table, chair, or changing table, from a baby bouncer etc. In less than 1% the circumstances of the fall were not reported. Seventy-six percent of children fell only once, and in 5% it was thrice or more. The number of falls increased with the age of the child. Less than 25% occurred before the age of 4 months. Only 14% of children sustained visible injuries, of which 56% were haematomas. In 97% of the injury was visible on the head. Less than 1% (21 children) sustained a concussion or fracture. One-hundred and sixty-two children were taken to hospital after their fall, and 18 children were hospitalized. In the hospital, a calvarium fracture was diagnosed in three children. However, this was no reason for hospitalization. Calvarium fractures were never seen after a fall from a bed or settee. None of the children suffered intracranial injuries such as subdural or epidural haemorrhages.
- Johnson et al.: Seventy-two consecutive children under the age of 5 years (ranging from 4 months to 4 years and 9 months), who presented at the emergency department because of an accidental head trauma after falling [254]. They collected data on the distance of fall in a free fall or falling down the stairs, the surface area of the landing and the length of the child. The distance of the fall ranged from 50 cm to 3 m. Most children fell less than 1 m. Of the 72 children, 49 children fell on a hard surface and 23 on a soft surface (covered in carpet). In 52 children the fall resulted in a visible injury to the head (35 on hard surface, 17 on soft surface—there was no significant difference). There were visible head injuries in all children that had fallen over a distance of more than 1.5 m, and in 95% of the children that had fallen over a distance of more than 1 m. In 32 children (44%), radiological imaging of the skull was done. In four cases a calvarium fracture was seen, of which three were linear. Two of the children with a linear fracture had fallen over a distance of more than 1 m. One child sustained a fracture in a fall of 80–90 cm against the stone edge around a fireplace. The fourth child sustained a basilar fracture in a fall of over 3 m from a window on the first floor. The authors concluded that children rarely sustain serious injuries in accidents in and around the home. They maintain that skull (calvarium and basilar) fractures are rare and occur only in less than 5% of all accidents. In their opinion, it takes a fall of at least 1 m or, in lesser distances, on a limited impact area to cause a skull fracture.
- Thomas et al.: One hundred twelve children under the age of 1 year with head trauma. In 96 children radiological imaging of the head was done. According to the parents, 32 children fell over a distance of less than 1 m [255]. Six children, that belonged to the group of 80 children that had fallen over a distance of less than 1 m, were diagnosed with a calvarium fracture. According to the parents, two children with a skull fracture had fallen from a height of less than 30 cm. In four of the six children that had sustained a fracture the physicians were sufficiently concerned to report the child to the child protection service. When additional examinations were performed, two of the children were found to have further fractures. Based on the statements of the parents, it appeared to be impossible to predict which children had sustained skull fractures. The presence of external injuries or neurological symptoms appeared to be an unreliable indicator for skull fractures. The reported distance of the fall was also not indicative. Therefore, Thomas et al. are of the opinion that in children under the age of 1 year that present with head trauma, radiological imaging of the head (X-ray) should routinely be done [255]. In their study, it led to the identification of four children with a skull fracture due to non-accidental circumstances (child abuse).
- Hughes et al.: Four hundred sixty-three children under the age of 48 months, who attended a hospital because of a head trauma due to a witnessed fall [253]. The authors investigated the relationship between fall height and the occurrence of head injuries, including the severity of the head injuries. The falls concerned a height of falling less than 3 m. Forty-seven children had a skull fracture or intracranial injuries, 416 children had minor head injuries. No calvarium fractures or intracranial injuries were recorded in children who fell over a distance of less than 60 cm, based on the height of the head centre of gravity. Skull fractures or intracranial injuries were more likely in children under the age of 12 months in case of an impact trauma to the temporal/parietal or occipital region, impact onto wood, and falls from a carer's arms, particularly

when on stairs. The authors did not find a significant difference between the mean fall heights of children who had a simple skull fracture (n = 17) versus those who had a complex fracture or ICI (n = 30). According to the authors, the following should always be evaluated in young children with a head injury after a reported fall: the age of the child, the child's position prior to the fall, the estimated height of falling, the location of the impact on the calvarium, and the landing surface.

- Hajiaghamemar et al. used an integrated approach and combined the findings of case evaluation, anthropomorphic reconstruction, and finite element simulation to predict the occurrence of skull fractures and the thresholds for the occurrence [248]. They found that infant falls from 0.3 m had a low probability (0–54%) to result in parietal skull fracture, particularly with carpet impact (0–1%), that head-first falls from 0.9 m had a high probability of fracture (86–100%) for concrete impact and a moderate probability (34–81%) for carpet impact and that the probabilities of fracture in 0.6 m falls were dependent on impact surface. Occipital impacts from 0.9 m onto the concrete also had the potential (27–90% probability) to generate parietal skull fracture.

The foregoing data show that calvarium fractures can be sustained in uncomplicated short-distance falls and these data also show that uncomplicated short-distance falls rarely if ever lead to (well-documented) serious or life-threatening intracranial injuries.

When a calvarium fracture is the result of an uncomplicated short-distance fall, e.g. a fall from a bed or a changing table, the occurrence of other fractures, such as rib fractures or a mid-shaft fracture of one of the extremities, is unlikely. In a non-accidental calvarium fracture, e.g. because a parent or caregiver deliberately has hit the child's head against the wall, or when a parent or caregiver at the end of his/her wits has thrown the child to the floor, it will nearly always lead to a more complicated combination of injuries than in an uncomplicated short-distance fall, e.g. calvarium fractures combined with intracranial injuries or with injuries in other locations of the body. The medical history, as told, will not be able to explain the (combination of the) injuries and their location. In other words, an accidental calvarium fracture, due to an uncomplicated short-distance fall can nearly always be explained based on the medical history.

In addition to the medical history, the fracture characteristics may provide only limited opportunities to further differentiate between accidental and non-accidental fractures. Hobbs evaluated 89 children under the age of 2 years old with calvarium fractures [256]. Sixty of them had sustained fractures due to accidental causes. The remaining 29 were victims of child abuse. Table 5.5 gives an overview of the differences between both groups.

Table 5.5 Characteristics of accidental and non-accidental calvarium fractures in children under the age of 2 years [256]

	Accidental	Non-accidental
Type of fracture	Generally simple and linear, uncomplicated	Multiple or complex Depressed fracture (Sect. 5.3.4.5) Growing fracture (Sect. 5.3.4.6)
Fracture width	<3 mm (never >5 mm)	>3 mm
Location	Generally, a fracture in one bone Mainly parietal Rarely other locations	More than one bone Mainly parietal and occipital Sometimes frontal or temporal or in the anterior cranial fossa or the medical cranial fossa
Intracranial injury	Rare	Frequently, combined with other fractures

Experimental Uncomplicated Short-Distance Falls in Deceased Children

Nearly every young child has ever fallen, probably even more than once, on its head from a standing position or from limited height, e.g. from a changing table or from a stroller. Since no consensus existed on whether children would sustain calvarium fractures from falling from these low heights or about the minimum distance a child must fall on its head to sustain a calvarium fracture, Weber did experimental research with deceased children under the age of 8.2 months. In his first article in 1984, he described three test series each with five children who he dropped in free fall from a height of 82 cm on several surfaces (stone-tile surface, carpeted floor, foam-supported linoleum floor) [177]. Hereby, the horizontally positioned body and the parieto-occipital part of the skull hit the surface simultaneously. In all cases autopsy showed linear skull fractures of the parietal bone. One child sustained bilateral fractures. In three children the fractures run across the sutures. Based on this study, he concluded that skull fractures can be sustained in a fall from a changing table. He also concluded that when non-accidental circumstances are suspected, differentiation with an accidental fall is only possible when the whole picture is taken into consideration.

In a second article, Weber described a follow-up study on another 35 children who he dropped on a soft surface [257]. For 10 children, a 2-cm thick foam rubber mat was used and for the other 25 a once folded, 8 cm-thick blanket. Weber found a calvarium fracture in one child in the rubber mat group (two linear fractures in the left parietal bone). In the other group, he found bowing fractures in four children (linear fractures or ping-pong fractures).

In interpreting Weber's data, one must be aware of the fact that a living child will fall differently from the dead body of a deceased child, due to active muscle tension and, when old enough, a falling reflex. Yet, Weber's studies show that it is possible to sustain a skull fracture in an uncomplicated fall from a height under 1 m.

Complicated Short-Distance Falls

Most falls in infants and young children will be uncomplicated short-distance falls. A complicated short-distance fall is defined as a fall over a distance of less than 1.5 m, in which extra loading of the head/skull is added to the effect of gravity.

Samuel et al. analyzed fall mechanisms in 595 children between 0 and 2 years of age and identified eight types of falls: from ground-level, down-stairs, from a bed, from a changing table, from furniture, from adult hold, from a playground device, and from a stroller/baby carriage [258]. All these falls can be defined as short-distance falls, but some can be considered to be complicated short-distance falls, e.g. adult hold falls or falls from a stroller/baby carriage. In their study population, the risk of clinically significant traumatic brain injury was very low. This was found in only one child. They did not find an association between the type of fall and the severity of the injury.

Burrows et al. evaluated the data of 1775 children under the age of 6 years (median 18 months, 54.7% boys) with head injuries from falls [259]. They found that children with complicated falls had the greatest chance of intracranial injuries or a skull fracture. This concerned short-distance falls in children, who fell from the arms of a parent or caregiver (mean age 12 months) or who fell from infant or child products (mean age 21 months) and long-distance falls in children who fell from a building (e.g. a window, wall, or attic) (mean age 3 years).

In 2001, Plunkett published a study entitled 'Fatal Pediatric Head Injuries Caused by Short-distance falls' [260]. In this paper, he presented data on 75,000 cases from the US National Electronic Injury Surveillance System (NEISS), from January 1988 to June 1999, and based on this presented data on fatal short-distance falls from playground equipment (e.g. see-saw, swing, and monkey bar). Plunkett reported 18 fatal cases, the ages of the children ranged from 1 to 13 years (five children aged between 12 and 24 months of age, five children aged between 25 months and 25 years of age, and eight children aged between 6 and 13 years), the reported fall distance varied from 55 cm. to 3 m, where in several cases (falls from a swing) there also was an angular acceleration in play. In 12 cases, the accident was witnessed by an independent third witness, in the 6 non-independently witnessed cases four children were under the age of 24 months. Based on the data presented in this article Spivack calculate a death rate of 1.3 deaths per 100,000 of such falls [261]. In 2008 Chadwick wrote, based on previously published publications, that '*The best current estimate of the mortality rate for short falls affecting infants and young children is <0.48 deaths per 1 million young children per year*' [262]. It is safe to assume that the incidence of fatal cases based on the publication by Plunkett is an overestimation. This as registries are biased towards more serious cases, as most falls will never be reported and thus not included in the NEISS database. This paper led to several letters to the editor but here the main focus was on either the reported lucid interval, the presence of retinal haemorrhages, or the extrapolation of the findings in this publication to cases of suspected AHT [261, 263, 264]. But in all letters, the authors commented that this study supported the general notion that it is extremely rare that a short-distance fall of a child will result in death from a head injury.

Falls from the Arms of Parent or Caregiver

Warrington and Wright and Tarantino et al. also looked into the consequences of a fall from the arms of a parent or caregiver [70, 71].

Warrington and Wright studied the incidence of falling in 11,466 non-mobile children in a home setting by a questionnaire [71]. The incidence of one or more falls was 22%. In nearly 12% of falls, the infant was dropped out of the arms of a person or a person fell carrying the infant, resulting in an incidence of 3.4%. At least one of these infants sustained a calvarium fracture but otherwise the authors did not specify the injuries of these infants. They merely state that serious injuries (concussion or fractures (either long bones or calvarium)) did not result from falls from a bed or settee but were the result of complex accidents (e.g. fall from the arms, changing unit, table, bouncer, and baby walker).

Tarantino studied 167 infants ≤10 months who presented to the emergency department and who rolled off a bed, changing table or other surfaces, or were dropped by the caretaker [70]. Only 15% sustained significant trauma (long bone fractures or closed head injury (75% of which had calvarium fractures)). Infants who were dropped by the caretaker had significantly more significant injuries (32%) than infants who rolled from a bed or other surfaces (10%). None of these accidental falls resulted in intracranial haemorrhage.

Minns reported that infants, as early as 5 weeks old, when they are held with one hand against the shoulder of a caregiver, are able to arch backwards, effectively diving from a caregiver's shoulder, falling approximately 1.5 m on the floor [68]. As a result of such a fall, they may sustain focal bruising, extensive calvarium fractures, and focal contusion of the brain, not accompanied by a concussive element or other encephalopathy or delay in seeking treatment. A good medical history and careful investigation of the circumstances will provide ample information to differentiate between accidental and non-accidental skull/brain trauma.

In 2004, Bechtel et al. published an article with the title 'Characteristics that distinguish accidental from abusive injury in hospitalised young children with head trauma' [252]. In 2005, Lueder responded to this article, regarding the presence of retinal haemorrhages in a number of accidental falls [265]. In their answer to Lueder's letter, Bechtel et al. described a number of situations in

which children had fallen from the hands of a parent or another caregiver and consequently sustained calvarium fractures and other injuries, e.g. epidural haemorrhages (Table 5.6) [266].

Falls on Objects

In the medical literature mostly case reports and only limited epidemiological studies are found about relatively serious injuries, e.g. calvarium fractures or intracranial haemorrhages, after short-distance falls, which were complicated, because of falling head first on a non-flat surface or falling on objects, like toys, electrical plugs, or other objects:

- Wheeler and Shope: 7-month-old girl who fell out of bed and sustained an uncomplicated, simple depressed calvarium fracture with a size of 2 × 4 × 0.5 cm in the right parietal bone (so-called ping-pong fracture—Sect. 5.3.4.5) [267]. She appeared to have fallen over a distance of approximately 60 cm on top of a metal toy car and was found lying on the floor crying. Nobody saw the fall. After the fall the father noted a depression on the right side of her head. In hospital she had a normal physical and neurologic exam, with no evidence of external trauma except for the depression on the right side of the head. There were no signs of underlying brain damage, retinal haemorrhages, or other fractures.
- Thompson et al.: 1-month-old boy with a depressed calvarium fracture and a thin right frontoparietal subdural haematoma [268]. The boy was sleeping on his mother's chest while she was lying in bed. The mother fell asleep and rolled over causing the child to fall off her chest and off the bed (height: 86 cm). He struck his head on a humidifier that was adjacent to the bed and landed supine on the carpeted floor. The subdural haematoma was determined to be consistent with an impact trauma. The boy was clinically well and had shown no neurological symptoms.
- Six children were described after falling on an electrical plug [269–272]. Ages ranged from 6 months to 2½ years. All patients had penetrating injuries to the skull and most of them had accompanying dural tears. All had depressed skull fracture in the frontal or parietal region. Neurological outcome was excellent in all cases.
- George and Round: 3-year-old boy, who had tripped and fallen onto a metal model of the Eiffel Tower, resulting in penetration of the skull and the brain parenchyma (11 mm) [273]. The tower became rigidly lodged into his skull. He had no neurological symptoms. He was operated and left the hospital the following day.

Table 5.6 Injuries in children who fell from the arms of a parent or another caregiver [265, 266]

Age	Distance	Context	Witnesses?	Findings at examination
1 month	1 m	Fell from the father's arms, who was lying on the bed	+	• Right-sided calvarium fracture • Epidural haemorrhage • Retinal haemorrhages in one eye (right eye)
4 month	1 m	Fell from the arms of an older child	?	• Left-sided calvarium fracture • Intra-retinal haemorrhages in the posterior pole of the left eye
4 month	1.25 m	Fell from mother's arms and hit its head against the edge of the table	+	• Right-sided calvarium fracture • Intracranial haemorrhage • Intra-retinal haemorrhages around the optical disc and arcs
8 month	60 cm	Fell from mother's arms, who was lying on the settee	?	• Left-sided calvarium fracture • Epidural haemorrhage • One intra-retinal haemorrhage in the left eye

Accidental Short- and Intermediate-Distance Falls

As already mentioned before, in this section the results of studies concerning certain short-distance and intermediate-distance falls will be discussed combined, because these studies either did not differentiate between short- and intermediate-distance falls, or described falls over a distance of less than 1–1.5 m as well as falls up to 3 m, e.g. falls with bouncy chairs or car seats, standing on an elevation, or from high chairs or shopping carts.

Falls from Furniture

Thompson et al. evaluated the findings in 79 children, aged 0–4 years, who presented at an emergency department with a history of a short-distance fall from furniture [268]:

- No injuries: 15 children
- Minor injuries, e.g. lacerations and contusions (Abbreviated Injury Scale (AIS) 1): 45 children
- Moderate injuries, e.g. fractures (AIS 2): 17 children
- Serious injuries, e.g. subdural haematomas (AIS 3): 2 children

There were no children with injuries classified as AIS 4 or higher (life-threatening injuries) and no fatalities. None of the evaluated children had moderate or serious injuries to

multiple body regions. Children with AIS 2 or 3 injuries tended to have fallen from greater heights, had greater impact velocities, and had a lower body mass index than those with AIS 1 or no injuries.

Falls From or with Stroller

Stroller-related falls (synonyms: perambulator, pram, baby carriage), in particular in children under the age of 1 year, are not rare and usually result from incorrect use [274–276]. Injuries are mainly found in the head and neck area, including calvarium fractures and intracranial injuries:

- Couper et al.: One hundred forty-nine infants and young children with stroller-related injuries, who had either fallen of the stroller or had their fingers entrapped in the stroller [274]. One hundred five children were between the age of 9 and 15 months. Eighty nine children (60%), who had fallen off the stroller, had either injuries to the head, face or teeth, or a concussion.
- Powell et al.: A retrospective review of data for children 3 years old and younger from the National Electronic Injury Surveillance System of the United States Consumer Product Safety Commission for 1994–1998 [277]. They estimated that there were almost 65,000 children (median age 11 months) with stroller-related injuries treated in hospital emergency departments in this period. Most injuries involved the head (44%) or face (43%). Injury diagnoses included contusions or abrasions (38%), lacerations (24%), closed head injuries (22%), and extremity fractures (3%). Two percent of the children had to be admitted. Seventy percent of these children were admitted because of head trauma.
- Fowler et al. retrospectively analyzed data from the National Electronic Injury Surveillance System for children 5 years of age and younger treated in emergency departments (1990–2010), who sustained an injury associated with a stroller or carrier [278]. They estimated that in the United States. around 17,000 children under the age of 5 years are treated annually in emergency departments, concerning stroller- or carrier-related injuries. 70–75% were stroller-related injuries. These were most common in boys (52.4%) and in children under the age of 1 year of age (42.0%). The head (43.0%) and face (31.0%) were most commonly injured. The most common diagnoses were soft tissue injuries (39.4%) and traumatic brain injuries/concussions (24.6%).
- Vilke et al.: One hundred twenty-one children with stroller-related injuries. Most children were 0–6 months of age. 47.5% of the children fell off the stroller [279]. Fifty-nine percent of the injuries were head injuries, 18% facial injuries. No child died.
- Tripathi et al.: Two hundred forty-eight children under the age of 6 years (median age 12.5 months) with stroller-related injuries (retrospective review, tertiary paediatric hospital in Singapore) [280]. Most injuries (97.6%) were due to blunt force trauma. The circumstances of sustaining injuries were fall/tripping in 221 children. Most children (91%) had injuries to the head, face and neck. 144 children (58.1%) had superficial injuries, 69 children (27.8%) sustained open wounds, and 16 children (6.5%) had a haematoma of the scalp. 17 children (6.9%) suffered fractures or dislocations of a limb or digit. Two children had moderate to severe head injuries. Most of the injuries (197 cases, 79.4%) occurred despite adult supervision.

According to Watson and Ozanne, the risk for serious injuries is considerable, since by far the majority of children (96% of children in their study) who had fallen from a perambulator fell on their head, although serious intracranial injuries have only been described in case reports (see earlier in this section) [276]. It concerns typically injuries that originate from impact, e.g. epidural haemorrhages, but the impact does not have to lead to a calvarium fracture, e.g. Lee and Fong [275] described the occurrence of an epidural haemorrhage in a 10-month-old girl after falling from a stroller. The girl had no skull fracture.

Permanent injuries as well as death are extremely rare [274–277]. Watson and Ozanne-Smith reported one child, that, according to the authors, died due to falling from a stroller. In their review of the literature, Lee and Fong found three children that died after their parents reported a fall from a stroller [275, 276]. In the end, two of the children were classified as victims of child abuse.

Arnholz et al. described the origin of bilateral skull fractures in a 6-week-old baby who had fallen from a perambulator from a height of approximately 90 cm on top of his/her head on concrete steps [281]. As associated injuries 'two separate and symmetrical areas of scalp haemorrhage' were found. Arnholz et al. point out that bilateral fractures are rarely the result of an accident and for that reason should be seen as extremely suspect of child abuse [281]. Their findings correspond with Weber's experiments with deceased children (See the section on 'Experimental Uncomplicated Short-Distance Falls in Deceased Children' earlier in this section) [177, 257].

Falls from Baby Carriers, Bouncy Chairs, Baby Bouncers, and Car Seats

A baby carrier is a supporting device worn by an adult for holding a young child close to the torso (Fig. 5.20a–c). Baby carrier-related falls in young children are less common than, e.g. stroller-related falls, but are not rare and, just like stroller-related falls, usually result from incorrect use [278]. Injuries are mainly found in the head and neck area, including calvarium fractures and intracranial injuries.

Fig. 5.20. (**a**) Baby carrier, (**b**) baby wrap carrier, and (**c**) bouncy chair

- Fowler et al. retrospectively analyzed data from the National Electronic Injury Surveillance System for children 5 years of age and younger treated in emergency departments (1990–2010), who sustained an injury associated with a stroller or carrier [278]. They estimated that in the United States around 17,000 children under the age of 5 years are treated annually in emergency departments, concerning stroller- or carrier-related injuries. 25–30% were baby carrier-related injuries. These injuries were most common in boys (52.5%) and in children under the age of 1 year of age (89.0%). The head (61.5%) and face (24.7%) were most commonly injured. The most common injuries were soft tissue injuries (48.1%) and traumatic brain injuries/concussions (34.9%). Carrier-related injuries resulted in more hospitalizations (6.5%) than stroller-related injuries (2.4%).
- The Centres for Disease Control (CDC) reported 26 fatalities associated with infant carrier-related injuries over a period of 5 years resulting in an annual fatality rate of approximately 5 [282].
- Wickham and Abrahamson analyzed the data of 131 children under the age of 1 year (mean age 6.9 months) with head injuries, due to accidental circumstances [283]. Seventeen of these 131 children sustained a head trauma

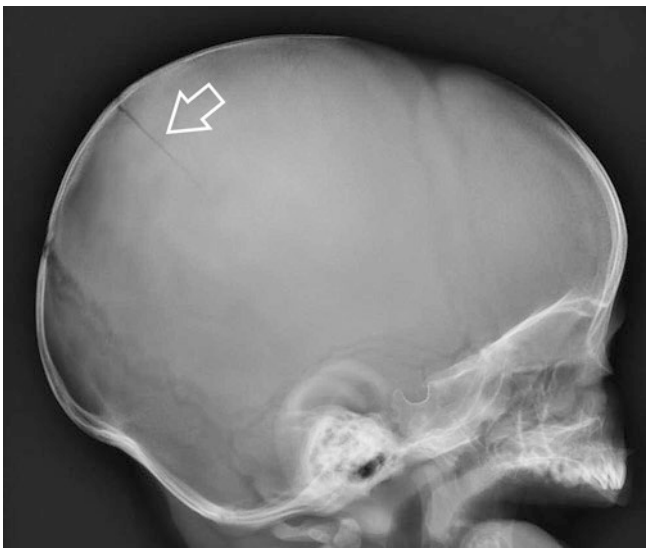


Fig. 5.21 Infant who fell with a bouncy chair from a table resulting in a simple linear parietal fracture (arrow). Uneventful recovery

due to a fall with a bouncy chair (11 children) (Fig. 5.20c) or a car seat (6 children). 13 falls occurred while the child was seated in the bouncy chair or car seat which was placed on a raised surface (Fig. 5.21): all bouncy chair falls and two car seat falls. Four car seat falls occurred while being moved from car to house. Fourteen children fell on a solid surface. Only one child had sustained a skull fracture as a result of the fall. There were no serious or life-threatening injuries. Most and probable all falls could have been prevented.

Greenberg et al. described the findings in 62 children under the age of 18 months (mean age 4.4 months) with infant carrier car seat-related falls (Fig. 5.22) and concluded that falls from infant carriers occurred regularly [284]. In almost 90% of these falls involved children who were unbuckled in their car seats. Twenty-two children had to be hospitalized, including 6 children who had to be admitted to a paediatric intensive care unit. Thirteen children had intracranial injuries: subdural haematoma in 8, epidural haematoma in 3, subarachnoid haematoma in 1 and cerebral contusion also in 1 child. Eleven of these 13 children had a skull fracture. Ten children had isolated skull fractures.

There are several case reports concerning injuries due to unsafe use of baby bouncers (e.g. [250, 285, 286]). Unfortunately, the literature is not clear on the definition of a baby bouncer, which makes it difficult to compare the described findings. A baby bouncer usually is considered to be a playing device made for children that are well able to keep their head upright, but are yet unable to walk (Fig. 5.23). Claydon reported the case of a fatal fall from a baby bouncer [250]. A 5-month-old boy had fallen after two other children had rocked the child in the bouncer. At the time of the fall,

Fig. 5.22 Baby carrier car seat

Fig. 5.23 Baby bouncer

the boy's head was no more than 60 cm (2 ft) from the floor. Clayton assumed, that pivoting about the central point provided by the seat of the bouncer increased the momentum of the head before it struck the ground and so the injury would be more severe than a straightforward fall back from his own height to the ground. After the fall the boy cried loudly. He died 7 h after the fall. At autopsy, a large epidural haemorrhage was found on the left side, without an associated skull fracture.

Falls from High Chairs

High chair-related accidents regularly occur in children under the age of 3 years. The majority of those children sustained head injuries, but in the (albeit limited) literature, serious or even life-threatening injuries have rarely been described:

- Watson and Ozanne-Smith: Eighty-three percent of high chair-related injuries occurred as a result of falls [276]. Seventy-five percent of the children who fell from a high chair landed on their head. Only 25% of the children was wearing any form of safety restraint. They reported one child that died from a fall from a high chair.
- Mayr et al.: One hundred three children with high chair-related accidents. Most commonly the children tried to stand up in the chair before falling off, but also tipping over the chair was reported regularly [41]. Only one child was wearing a safety restraint. Skull fractures were present in 15.5%, simple bruising of the scalp or lacerations of the scalp or face in 68.9%, brain concussion in 13.6%, and limb fractures in 2%.
- Both Powell et al. and Kurinsky et al. retrospectively analyzed data from high chair-related injuries that were treated in emergency departments from the National Electronic Injury Surveillance System (NEISS) [287, 288]. Approximately 9000 children ≤3 years were seen annually. Falling was the most common injury mechanism (93–94%). Most injuries were in the head/neck region (44–59%) and the face (28–39%) and concerned closed head injuries (21–37%), bruises and abrasions (33–36%), lacerations (19–25%), and fractures (8–9%). Admission to the hospital was required for 2–3% of the children. No fatalities were recorded, but the authors state that the NEISS does not register outcomes after admission to the hospital.

According to all cited authors most of the accidents could have been prevented by following the safety instructions and using safety restraints.

Falls from Shopping Carts

Shopping cart-related accidents are common in children, especially in children under the age of 5 years, who account for around 85% of all cart-related accidents [289]. Injuries mainly occur due to falling from carts and tip-overs of carts tipping, but also other circumstances can occur like becoming entrapped in a cart, falling off a cart while riding on the outside, striking against a cart, and being run over by a cart. Shopping cart-related accidents can result in severe injuries and probably even death. Injuries to the head and neck are most common and occur in around 75% of the accidents:

- The Centers for Disease Control and Prevention (CDC) reported on infant carrier-related fatalities over a period of 5 years. One child died due to blunt force trauma to the head [282]. A grocery cart containing an infant carrier in which the infant was riding overturned and the child impacted, probably head first, to the ground.
- Both Smith et al., Martin et al., and Wright et al. retrospectively analyzed data from the US National Electronic Injury Surveillance System concerning shopping cart-related injuries in children under the age of 15 years who were treated in US emergency departments from 1990 to 2011 [287, 289, 290]. An average of approximately 24,000 children were reported annually. Injury rates decreased markedly with increasing age and were highest among children under the age of 5 years (97.8 per 100,000 persons/year), followed by children between 5 and 9 years (13.9 per 100,000 persons/year), and children between 10 and 14 years (2.9 per 100,000 persons/year) [289]. Head and neck injuries were most common and occurred in 74–80% of the children. Almost 3% of the children required a hospital admission, most of them (93%) were between 0 and 4 years of age. Among the 0–14 year old children, fractures accounted for 45% of hospital admissions, followed by internal injury (22%) and concussion (17%). The annual concussion/closed head injury rate per 10,000 children increased from 0.64 in 1990 to 2.02 in 2011 [290].
- Smith et al.: Sixty-two children (4 months to 10 years of age; mean age 2.8 years), presented at the emergency department because of shopping cart-related accidents over a period of 15 months (prospective research) [291]. The majority of children had sustained the injury by falling out of the shopping cart in 58%, followed by toppling over of the cart in 26%. The sitting position was associated with tip-over injuries, and standing in the cart basket was associated with falling from the cart. Injuries due to falling out of the cart occurred at all ages, whereas injuries due to toppling over accounted for 40% of shopping cart-related injuries among children under the age of 2 years. Eleven children (18%) had fractures, of which five (8%) had skull fractures. Forty-nine children (79%) sustained head injuries, 9 children (14%) had lacerations, and 30 (48%) had superficial injuries (bruises or abrasions). Smith et al. concluded that accidents with

shopping carts can lead to serious and potentially life-threatening injuries, although there were no cases of (intra)cranial injury, in spite of falling on a solid (often concrete) surface [291]. No intracranial haemorrhages were found.

- Vilke et al.: One hundred twenty children under the age of 14 years with shopping cart-related injuries [279]. Most children were around 1 year of age. Seventy percent of the children fell off the shopping cart. Head injuries accounted for 53% of the injuries and facial injuries for 12%. No child died.

Falls from Bunk Beds

Bunk bed-related injuries are common in children and may be sustained by falling from the top bed, the bottom bed, or from the ladder. The fall may occur during sleep, when getting out of bed or while playing. The majority of children who fall from a bunk bed, will sustain head injuries, including facial injuries:

- Selbst et al.: Sixty-eight children with bunk bed-related injuries, of whom 47 were younger than 6 years (prospective research) [292]. Injuries were sustained most often when the child fell from the top bed (38 children), fell off the ladder (7 children), or fell off the bottom bed (8 children). Injuries occurred during sleep (19 children), getting in or out of the bunk bed (13 children), or playing in or near the beds (28 children). Of those injured while asleep, 13 of 19 children were younger than 6 years. Head injuries were present in 35 children and injuries to the extremities in 16 children. The most common injuries were lacerations in 27 children and soft tissue bruises in 19 children. Eight children had concussions and seven children had fractures. One child had a skull fracture and a subdural haemorrhage. Six children (9%) required admission to the hospital. Head and face injuries were significantly more likely if the top bed had no side rails. Injuries were more serious in case of falling off the top bed.
- Macgregor: Eighty-five children with bunk bed- and conventional bed-/cot-related injuries, of whom 66 were under the age of 6 years (prospective research) [293]. A majority had fallen out of bed while sleeping. Twenty-five children sustained a fracture, 27 a head injury, 12 a laceration requiring treatment, and 21 sustained a soft tissue injury to a limb. Injuries were more serious in case of falling off the top bed. There were no children with calvarium fractures, in spite of the fact that a number of children showed notable neurological symptoms (e.g. unconsciousness, drowsiness, or vomiting). In none of the children an intracranial haemorrhage was found, not even in case of a complex fall, e.g. if the child during fall hits another piece of furniture before landing on the ground [293].
- Mayr et al.: Retrospective analysis of 218 children with bunk bed-related accidents. The most common circumstances were falls from the top bed during sleep in 35.1%, while playing in 34.4%, and falling off the ladder in 23.2% [294]. Ninety-one children had major injuries, including 3 polytrauma, 7 calvarium fractures, 44 cerebral concussions, 33 long bone fractures, 2 Lisfranc injuries, and 2 lacerations of the spleen. Eighteen children had fractures in other locations than the long bones or the calvarium, 89 children soft tissue injury and sprains, 18 skin lacerations, and 2 tooth fractures. Almost 25% of the bunk bed-related accidents occurred in children under 3 years of age.
- Belechri et al.: Injuries in 197 children (aged 0–14 years) with falls from bunk bed compared to injuries in 1684 aged-matched children with falls from conventional beds (research period 1996–1998) [295]. Injuries in children with bunk bed-related falls generally were more serious than those in children with conventional bed-related falls, with overrepresentation of brain injuries, fractures, multiple injuries, and injuries requiring hospitalization.
- Mack et al: Retrospective analysis of data from the 2001 to 2004 National Electronic Injury Surveillance System, concerning non-fatal, unintentional bunk bed-related injuries in children, aged 0–9 years (around 23,000 children annually, including 14,600 children under the age of 6 years [296]. 1 in 4 children was injured in a fall from the upper bed. Injuries were fractures, lacerations, contusions and abrasions, and internal injuries. The most commonly injured body region in these children was the head and neck.
- D'Souza et al.: Retrospective analysis of data from the 1990 to 2005 National Electronic Injury Surveillance System, concerning non-fatal, unintentional bunk bed-related injuries in children and adolescents, aged 21 years or younger (around 35,790 children and adolescents annually, including 14,600 children under the age of 6 years) [297]. The most common type of injury was lacerations in almost 30%, followed by abrasion in 24% and fractures in almost 20%. The most commonly injured body region was the head and neck in 27.3% in all age groups.

In spite of the high number, the severity and diversity of the injuries that occur when children (and adolescents) fall from a bunk bed, intracranial injuries are only rarely reported in the medical literature [292, 294, 295, 298]. In none of the before mentioned studies, the death of a child after a fall from a bed bunk was reported.

Falls During Playing and Sporting Activities

Calvarium fractures can occur during daily activities, like playing or sporting:

- Ono et al.: Forty-two children under the age of 15 years with playground equipment-related head injuries (median age 5 years; 26 boys, 16 girls) [299]. The injuries were slide-related in 20 children, swing-related in 11, and jungle gym-related in 5. Injuries were sustained in one child each, due to the use of monkey bars, iron bars, and a trampoline. In six children the circumstances were not known, except that they were playground equipment related. Falls ranged from a height of 1.2–2.5 m. Most of the falls occurred on hard soil or concrete. Skull fractures were found in 30 children, acute epidural haemorrhages in 9, acute subdural haemorrhages in 5, subarachnoid haemorrhages in 3, concussion in 8, and contusion in 3.
- Illingworth et al.: Two hundred twenty-five children with skateboard (Fig. 5.24) injuries [300]. Nine-two fractures were found, of which most involved the upper limb. Lacerations, bruises, abrasions, and injuries to joints and soft tissues were the next most frequent injuries. Concussions were found in four children, of whom one also had a skull fractured. Minor head injuries were found in 8 and soft tissue injuries of the head and neck in 23 children.
- Russell et al.: Sixty-four children with (Fig. 5.24) injuries (median age 14.5 years; 84% male) [301]. Fifty-one children sustained head injuries, including 32 intracranial haemorrhages, 31 skull fractures, and 17 concussions. All children survived. The authors compared the findings in children with longboard injuries with the findings in children with skateboard injuries during the same period and found that head injuries were more common in the longboard group (Fig. 5.24).

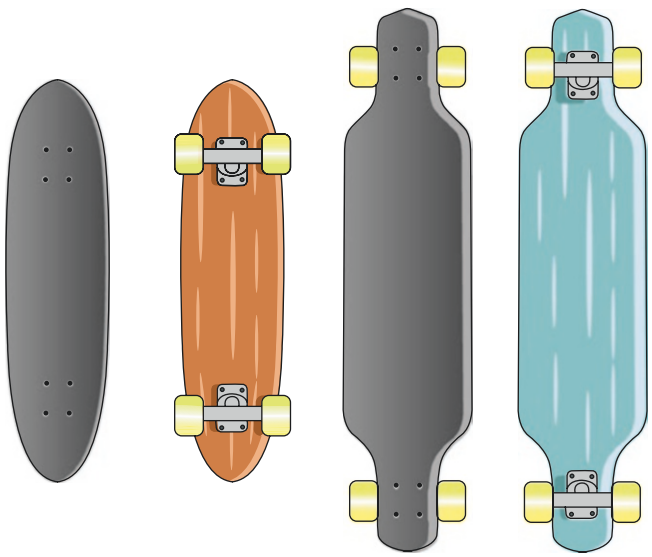


Fig. 5.24 Left: classic skateboard; right: longboard

Accidental Long-Distance Falls

Long-Distance Free Falls and Falls From Windows, Balconies, and Rooftops

According to the American Academy of Pediatrics (AAP) falls off heights (windows, roofs, and balconies) represent an important cause of child injuries and death and in the United States, approximately 140 deaths from falls occur annually in children under the age of 15 years [302]. An estimated three million children require emergency department care for fall-related injuries. According to Stone et al. the leading cause of death in children are injuries, sustained in accidental circumstances and falls are the most common type of injuries due to accidental circumstances (unintentional injuries) in the United States [303].

The majority of children with injuries due to long-distance falls are under the age of 5 or 6 years and fall over a distance of 3–7 m (one or two floors), most commonly in or in the direct vicinity of the home, and mostly during the warm seasons [304–307]. Parents usually do not witness the fall of their child, unless the parents are directly involved in the falling [305].

The fall distance necessary to cause injuries in children in a long-distance free fall has been and still is a continuous subject of discussion. Most injuries sustained by a child with a long-distance fall are injuries to the head and neck. The most common injuries, besides visible injuries, like bruising and abrasion, are fractures of the calvarium, which may be accompanied by intracranial findings, like subdural, subarachnoid and epidural haematoma, or cerebral contusions [304, 306–310]. Basilar fractures can also occur [308]. The risk for a fatal course increases with increasing height, for example a fall from a balcony, roof, stairs, diving board, or from an open window or tree (Fig. 5.25a, b) [304]. Mortality rates are relatively low [307, 308, 311].

In long-distance falls intracranial injuries are the most common cause of death [304, 308, 312]:

- Meller and Shermeta: 48 children who had vertically fallen from heights (e.g. windows, walls, and roofs) [313]. Most children (67%) fell from a distance of 3.5 m (12 ft) but 33% of the children fell from a height between 3.5 and 11 m (12–36 ft). The mean age of children with serious injuries was 7.5 years. Twenty-seven percent of children under 3 years of age had a documented injury, as opposed to 67% of children over 3 years of age. The mortality rate was relatively low (2%) and 4% of the children had long-term sequelae.
- Williams: Evaluation of the physical findings in 398 consecutive paediatric victims of falls [310]. In the end, 106 children were selected for further evaluation. In these, the fall had been witnessed by another person than the carer, and the circumstances of the fall had been documented. The physical findings in these children are specified in

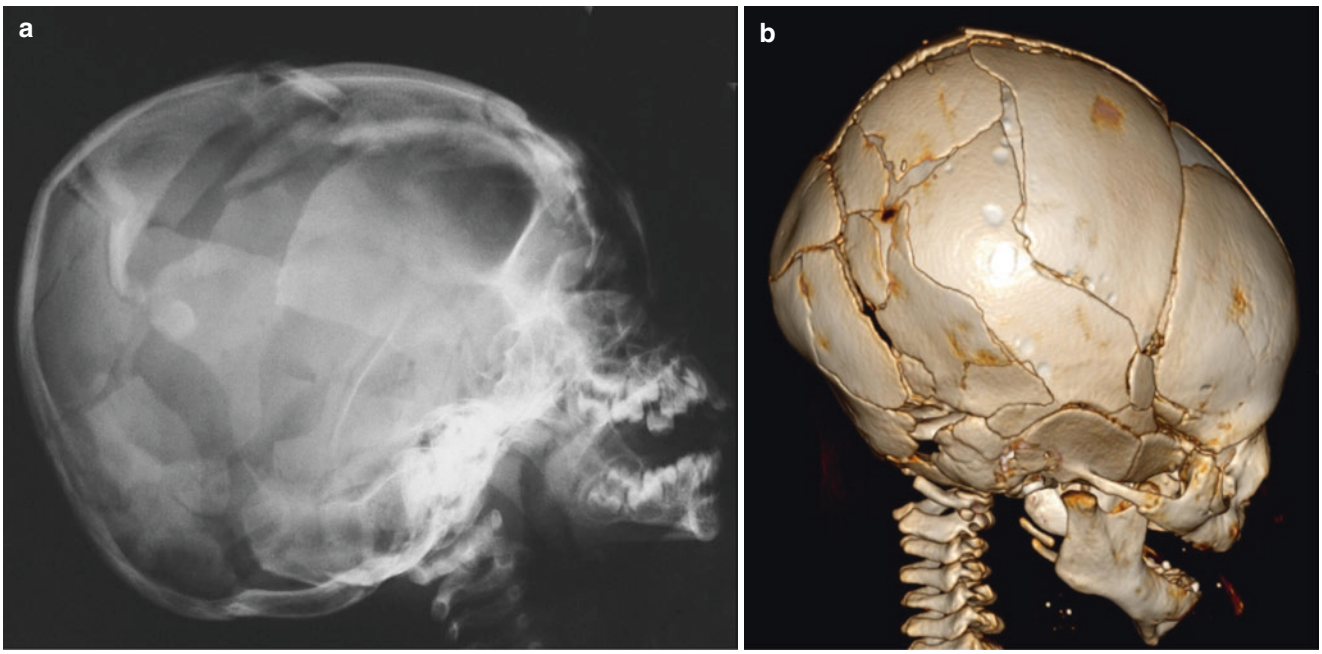


Fig. 5.25 (**a**) Skull radiograph of an infant who died after being thrown from the fifth floor of an apartment building by her caretaker. Extensive comminuted diastatic skull fractures. (**b**) 3D CT reconstruction of another patient with extensive comminuted fractures

Table 5.7 Injuries in falls witnessed by others than the carer (distance of falling 0.5–20 m) [310]

Severity of injury	*N*	Injuries	<3 m	>3 m
None	15		8	7
Mild	77	• Haematomas, abrasions • Simple fractures	24	43
Serious	14	• Calvarium fractures: depressed or compound/ comminuted • Intracranial haemorrhages Brain oedema	3	11

Table 5.7. In the group with the independent eye witness, there were 44 children that had fallen over less than 3 m. In this group, three children had sustained a small, depressed fracture without loss of consciousness from falling against an edged surface. None of these children died. In the group of children whose fall had been witnessed by an independent observer, one child died after a fall of over 20 m (70 ft). Williams concluded that 'infants and small children are relatively resistant to injuries from free falls, and falls of less than 3 m (10 ft) are unlikely to produce serious or life-threatening injury'.

- Musemeche et al.: Seventy children (mean age 5 years; 68% boys) with falls of 3 m (10 ft) or higher or of at least one story (1985–1988) [306]. Seventy-eight percent took place in falls from two stories or less and usually took place at home or near the home. Most children sustained a single major injury, most commonly involving the head or the skeleton. All children survived.
- Lehman and Schonfeld: Ninety three children fallen from windows and 58 fall from other structures (balconies, fire escapes, and roofs) in a 3-year period [311]. Over 70% fell from a second-story window. Most children were under 3 years of age, and playing at the time of the fall. Although the morbidity rate was high (two-thirds of children had at least one fracture, more than 30% of the children required the intensive care unit, and 10% were permanently more or less neurologically impaired), the mortality rate was relatively low (0.7%).
- Lallier et al.: 64 children (aged 1–18 years; mean age 7.4 years; 45 boys, 19 girls) were admitted to hospital because of a long-distance fall [304]. Fifty children fell from 6 m (20 ft) or less (two stories) and 14 fell from a distance above 6 m: 15 children fell from a balcony, 13 from a window, 9 from a tree, 6 from a roof, 6 from stairs, 3 from a diving board, and not further specified in 12 children. Sixty percent took place at home and during the summer months. Two children had no injuries, 55 children had only one system injury, and 7 multisystem injuries. Major injuries included head trauma (39%), musculoskeletal (34%), abdominal (12%), maxillofacial (8%), and spine (6%). Only one child died after a fall over 15 m (50 ft).
- Murray et al.: Retrospective review of children, aged 0–14 years [309]. Children who fell over a distance of 4.5 m (15 ft) or less than had a higher incidence of intracranial injuries and a lower incidence of fractures of the extremities than children who fell over a distance above 4.5 m. Skull fractures were the most common injuries and

were associated with an increase in intracranial injuries in both subgroups.

- Stone et al.: 86 children under the age of 15 years, admitted to hospital, because of falls from windows (1991–1997), compared to 1277 children under the age of 15 years, admitted to hospital, because of other falls [303]. Children, aged 0–4 years had a higher rate of falls than children, aged 5–14 years: 14.6/100,000 vs. 2.0/100,000. Boys were twice as likely to fall as girls. The mortality rate for falls from windows was 4.7%, compared to 0.07% for all other falls.
- Kim et al. and Wang et al.: Retrospective analysis of 729 children, aged 15 years or younger with fall-related injuries due to accidental circumstances (1992–1998) [308, 314]. In 393 children the fall was classified as a short-distance fall (under 4.5 m/15 ft) and in 336 as a long-distance fall (4.5 m or above). Ninety-eight children had a fracture of the calvarium and 93 had a basilar fracture. A cerebral contusion was found in 26 children, a subarachnoid haemorrhage in 25, a subdural haematoma in 22, and an epidural haematoma in 12. Brain injuries were found with equal frequency in children with falls over a distance of 4.5 m or less and falls above 4.5 m [314]. Common extracranial injuries were fractures of the upper extremity in 6.2%, fractures of the lower extremity in 5.6%, pulmonary contusion in 1.8%, pneumothorax in 1.1%, liver laceration in 1.1%, bowel injury in 1.0%, and splenic injury in 2.1%. Orthopaedic and thoracic injuries resulted more commonly from high-level falls, whereas abdominal injuries were as likely to occur after a low-level fall. Long-distance falls were associated with a higher mortality rate than a fall of 4.5 m or less (2.4 vs. 1%; overall mortality rate 1.7%). Four children died from a fall under 4.5 m, all because of intracranial injuries and intracranial hypertension. All four had a GCS of three when arriving at the hospital and had abnormal CT findings. Fifty percent of the children, who died after a fall over a distance above 4.5 m, died of intracranial injuries and 50% of severe extracranial injuries.
- Vish et al.: Ninety children (mean age 2 years; 55 boys, 35 girls) were admitted to hospital because of a fall from a window [307]. Ninety-eight percent of the children fell over a distance of four stories or less. Head injuries and fractures of the extremities were most common. Three children died, and three children were discharged to a rehabilitation centre.
- Mayer et al.: Thirty-one children under the age of 16 years with head injuries and/or multiple trauma due to falling from windows or balconies [305]. Eighty-four percent of the children was between 0 and 5 years of age. All six children who died belonged to this age group. Twenty-seven children fell from the third-floor or lower. Twenty-one children fell at home. Fifteen children climbed on furniture before falling. According to the authors the fall was due to dangerous balcony or house constructions in 20% of the falls. Except for three cases parents did not witness the fall. One mother jumped out with her child and two mothers threw their children out of the window. Two children attempted suicide.
- Melo et al.: 58 children under the age of 6 years (mean age was 2.8 ± 1.4 years) with severe head injuries due to falls from windows, presenting with a Glasgow Coma Score (GCS) of eight or less (2000–2005) [315]. Almost half of the children had a GCS of 5 or less and just over 60% had a Paediatric Trauma Score (PTS) of 3 or less. The mortality rate was 41% (24/58) and most of them (88%; 21/24) died within 48 h after falling. According to the authors, severe head injuries due to falls from windows carry a high risk of mortality in children under the age of 6 years.
- Al et al.: Five hundred thirty-eight patients, aged 3 months to 98 years (mean age 12.4 ± 3.22 years) with injuries due to an accidental fall from heights (1–20 m; mean distance 3.2 ± 2.4 m), divided into falls from rooftops, balconies, stairs, and simple falls (elevator shafts, rock cliffs, bunk beds/berths, chairs, donkey/horses, and cribs) [312]. Over 50% of patients were under the age of 6 years old, 70% were under the age of 10 years, and 83.5% were under the age of 20 years old. The most common injuries were to the head with linear fractures being the most common injury in over 20% of all patients, followed by intracranial haemorrhages, brain oedema, and cerebral contusions. The mortality rate was 2.2%, and was highest among the patients who fell from flat-roofed houses. All patients who died had head injuries. The mortality rate in children under the age of 10 years was 1.5%. The mean distance of falling in this age group of children who died was 4 m, although fatalities were more common, when children fell from distances over 4 m, or when the child's head hit a hard surface, such as concrete.
- Shields et al.: 86,500 balcony fall-related injuries (estimated), treated in US hospital EDs from 1990 through 2006 [316]. The distance of falling ranged from 1.5 m (5 ft) to 26.5 m (87.5 ft). Thirty-seven percent of the patients were under the age of 18 years. These patients were more likely to sustain a concussion/closed head injury or skull fracture than patients 18 years and older.
- Kocak et al.: One hundred thirty-three children under the age of 18 years (median age 4 years, 50% between 2 and 7 years of age) with accidental falls from a height of at least 1 m (prospective study—1 year period) [317]. Ninety-five children were between 0 and 6 years of age. Parents were not present at the moment of falling in 55% of children in this age group. Thirty-eight percent of all children fell off a balcony. The mean fall distance was 2.9 ± 2.5 m. Median Glasgow Coma Score was 15 (50%

had a GCS of 14 or 50), and the median Paediatric Trauma Score was 10 (50% had a PTS of 9–11). The risk of losing consciousness increased with increasing distance of falling. Head injuries were the most common injuries in 63%. Multiple injuries were found in 17.3%.

- Pérez-Suárez et al.: Fifty-four paediatric patients (age ranging from 0.4 to 14 years; mean age 6 years) with falls of 2 m and above, admitted to hospital (intensive care unit) (retrospective study—10 years period) [318]. Twenty-eight of the children (51%) were (often unsupervised) pre-school children, 12 (22%) were above the age of 12 years. Ninety-two percent fell off a building (e.g. windows or balconies). Most of the falls occurred during playing. Six of the 12 children between 12 and 14 years of age attempted suicide. Head injuries, ranging from mild to severe, were found in 38 children (70%), including calvarium fractures in 25 children (46%) and intracranial injuries (subdural and epidural haematomas; brain contusion) in 30 children (56%). Seven children (12%) died, all due to falls over 9 m (two children due to brain injuries, two due to hypovolemic shock, and three to cardiorespiratory arrest).

These papers show that accidental long-distance falls in children have high morbidity (27–97%), especially head injuries, and a relatively low overall mortality (0–4.7%). However, mortality increases sharply in patients with severe brain damage, up to 41%.

Long-Distance Falls from Playing Equipment

Several studies have been published concerning more or less long-distances falls from playing equipment, e.g. from ropes, vines, and tree houses:

- Albanese et al.: Twenty-six children, of whom 18 fell from ropes, and 8 from vines (all onto packed dirt) (retrospective study) [319]. Fourteen children fell over a distance of one story or less, 8 from two stories, and 4 from three stories. No differences were found in injury severity score, or length of hospital stay with respect to the height of falling. Head injuries were the most common injuries in 58% of the children, followed by long bone fractures in 42%, axial skeletal fractures in 23%, and intra-abdominal visceral injuries in 8%. One child died due to intracranial injury after a two-story fall.
- Randazzo et al.: Retrospective analysis of data from the National Electronic Injury Surveillance System, concerning tree house-related injuries, due to falls or jumps (1990–2006) in (estimated) 47,351 paediatric patients of 19 years and younger, who were treated in an emergency department [320]. Fractures were the most common injury (36.6%), and the upper extremities were the most commonly injured body part (38.8%). The risk of sustaining a head injury was the highest in children under the age of 5 years. The risk of sustaining a fractures was higher in falls of jumps over 3.5 m (10 ft) than in falls of jumps under 3.5 m.

Long-Distance Falls from Stairs

Almost all parents have experienced at some time that their young child fell downstairs. This means that annually the number of falls down the stairs must be very high. Usually, it results in little or no injuries. This is probably why the paediatric literature contains only a few publications on this type of accident and the occurrence of skull fractures in these accidents, despite the fact that falling down stairs most probably is the most common type of long-distance falls in children, followed by playground-related falls and bicycle-related falls [321]. Most of the studies concerning stairway-related falls show that long-distance free falls cause more serious and more injuries than stairway falls from the same vertical distance downstairs:

- Joffe and Ludwig: Three hundred sixty-three children (1 month to 18.7 years; mean age 55 months; 54 children under 1 year of age), presented to a paediatric emergency department with injuries due to falling downstairs (prospective study) [322]. Children with inflicted injuries were excluded from the study. 10 children were carried by their parents or carer. Twenty-four children were in a baby walker when they fell downstairs. The majority of children sustained only minor superficial injuries. Seventy-three percent of the children had injuries to head and neck, irrespective of the severity of the injuries. Twenty-eight percent had injuries to the extremities (mostly distal) and 2% had injuries to the trunk. Children under the age of 4 years were more likely to sustain injuries to the head injuries than children above that age. Only 2.7% of the children had injuries to more than one body part. Six percent of the children had fractures. Seventy-two percent of these children had fractures of extremities. Six children (28% of children with fractures) had a skull fracture, all of them were under the age of 3 years. Four of the children with skull fractures belonged to the ten children that fell from their parents' or carers' hands while going downstairs. A cerebral concussion was found in three children. Intracranial haemorrhages or cerebral contusions were not seen. In none of the children, the injuries were life threatening. No child needed intensive care. No association was found between the number of steps fallen down and the number and/or the severity of injuries. Children who fell down more than four steps had no greater number or severity of injury than those who fell

down less than four steps. According to the authors, a fall downstairs consists of mild-to-moderately severe initial impact followed by a series of low-energy (most probably) non-injurious falls down the remaining steps. The first fall is the longest by height: the height of the child itself plus the number of steps of the stair. Joffe and Ludwig concluded that different circumstances should be suspected when multiple, severe, truncal, or proximal extremity injuries are noted in a child who reportedly fell down stairs [322].

- Chiavello et al.: Sixty-nine children (under the age of 5 years; median age 2 years) presented to a paediatric emergency department with stairway-related injuries (prospective study—2-year period), including three children who fell while being carried by a parent/carer [323]. Children with baby walker-related accidents and children suspected to have inflicted injuries were excluded. Most injuries were not serious. Ninety percent of the children had head and neck injuries, irrespective of the severity, 6% injuries of the extremities, and 4% of the trunk. There were no children with injuries to more than one body region. Fifteen children sustained serious injuries such as concussions (11 children—16%), skull fractures (5 children—7%), cerebral contusion (2 children—3%), subdural haematoma (1 child—1%) and fracture of the second cervical vertebra (1 child—1%). The three children who were carried had sustained serious injuries: one child had a skull fracture, one child had a skull fracture and a brain contusion, and one child had a subdural haematoma. This was also the child that had sustained a fracture of the second cervical vertebra. These injuries occurred in a fall while being carried downstairs by an adult. Chiaviello et al. [323] concluded that head and neck injuries are the most prevalent injuries, and that it is rare to have injuries on more than one body part, but they also concluded that serious stairway-related injuries might be more common than reported before their article.
- Docherty et al.: Two hundred thirty-nine children (aged 0–15 years; mainly toddlers), visiting the emergency department, because of falls downstairs (retrospective analysis) [324]. Two hundred sixteen of the 239 children (90%) had sustained one or more than one injury. One hundred sixty-five children (69%) had minor injuries to the head and face, 29 minor soft tissue injuries to other body parts. Twenty-three children had fractures: fractures of the clavicle in 9, wrist in 4, elbow in 1, femur in 4, and tibia in 5 children. Eight children were admitted to the hospital. None of the head injuries needed an intervention. Injuries to more than one body part were found in only 8 children (3%). Seventeen children (7%) were dropped while being carried on the stairs by a parent or carer and all 17 children sustained injuries, of whom 5 had a fracture of the skull, 1 of the tibia, and 2 of the femur. The likelihood or severity of the injuries was not associated with the number of stairs, but young children who fell downstairs with their carer or were dropped while being carried downstairs should get a close evaluation. Docherty et al. concluded (just like Joffe and Ludwig) that different circumstances should be suspected when severe injuries to the trunk and extremities truncal or injuries involving more than one body region are found in a child, who reportedly fell downstairs [322, 324].
- Pomerantz et al.: Four hundred eighty-nine children (under the age of 5 years) were hospitalized because of falling either from furniture (318 children; most common falls: 33% from beds, 18.9% from couches; 17.9% from chairs) or from stairs (171 children) (retrospective study, 11 years) [325]. Head injuries were much more likely in falls from stairs compared to falls from furniture (64.3 vs. 38.1%), including skull fractures (39.8 vs. 20.1%). Injuries to the upper extremities were much more likely in falls from furniture compared to falls from stairs (33.3 vs. 9.9%), including humerus fractures (30.8 vs. 9.4%).
- Zielinski et al.: Retrospective analysis of data from the National Electronic Injury Surveillance System of the US Consumer Product Safety Commission from 1999 through 2008, concerning stair-related injuries in (estimated) 931,886 children (under the age of 5 years) [326]. In this period, the number of the number of stair-related injuries decreased significantly. Approximately 75% of children had injuries to the head and neck region, and 2.7% of patients were hospitalized. Soft tissue injuries were found in almost 35% of the children. Children who fell while being carried accounted for almost 25% of the injuries in children under the age of 1 year.
- Pennock AT et al.: Sixteen children (7–51 months of age; average age 14.5 months), who sustained a fracture due to falling down stairs, while being carried by a carer (retrospective study, 2004–2012) [327]. None of the children had a fracture of the skull. Fifteen children had a fracture of the lower extremity, of whom 8 had a fracture of the femur. The majority were buckle fractures, but all diaphyseal femur fractures were spiral. One child had a fracture of the ulna. Detailed histories from the person who carried the child showed that they 'missed a step' due to the child being carried in front, obscuring the vision of the carer (Fig. 5.26).
- Hibberd et al.: Three hundred seventy-two children (aged 0–13 years) with bruises from accidental (unintentional) trauma (falls from less than 1 m, falls from 1 to 2 m, falls from standing height or less and hitting an object during fall, stairs or impact, crush, sports, or motor vehicle collision) [328]. The children had 559 injury incidents, result-

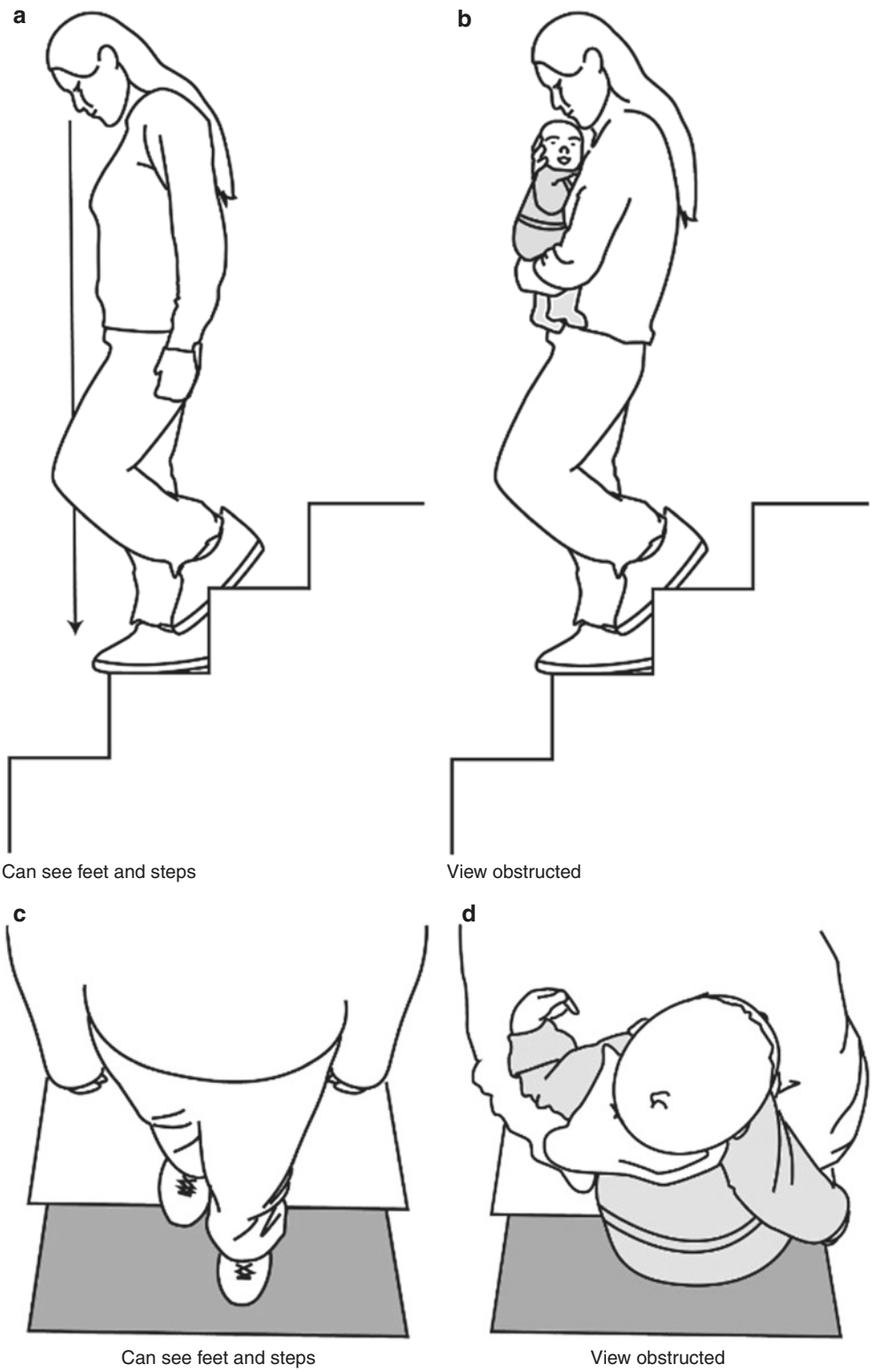


Fig. 5.26 (**a**) Side view of the caretaker's unobstructed view on the stairway. (**b**) Side view of the child obstructing the caretaker's view. (**c**) Bird's eye view of the caretaker's unobstructed view of the stairway. (**d**) Bird's eye view of the child can obstruct the caretaker's view of the stairway. (Reprinted under open access from A.T. Pennock et al. Stair falls: caregiver's "missed step" as a source of childhood fractures [327])

ing in 693 bruises. Stair falls resulted in 3 or more bruises only with falls involving 10 or more steps.

Injuries to the head (including fractures of the skull) are probably the most common injuries in stairway falls in children. Serious intracranial injuries, however, are only very rarely described. Only Chiavello et al. reported the occurrence of intracranial injuries (see earlier in this section) [323]. As far as could be derived from the medical literature there is only one isolated case report concerning a fatal stairway fall in a young child. Lantz and Couture reported an almost 8-months-old infant, who sustained an acute subdural

haematoma, and a severe haemorrhagic retinopathy, according to the authors, due to an unobserved stairway fall (basement stairway, six carpeted steps, total vertical height 1.40 m) [329]. The infant eventually died due to intracranial injuries. According to the Greeley and the RCPCH and RCO, the case report cannot be considered as proof that young children can sustain fatal injuries due to stairway falls, because Lantz and Couture did not give sufficient evidence that non-accidental circumstances (inflicted injuries/abuse) were excluded properly [329–331].

Falls with Baby Walkers, Including Walker Falls from Stairs

Accidents with baby walkers (Fig. 5.27) occur regularly in young children up to 1 year of age. The number of baby walker-related injuries is decreasing in the United States since the implementation of federal mandatory safety standards in 2010 [332]. Nevertheless, despite the decline in injuries, baby walkers remain an important and preventable source of injury among young children.

Baby walkers are not inherently dangerous, but become dangerous because of lacking supervision by parents/carers [333]. In the eighties of the last century, discussions started on the risks of sustaining calvarium fractures and other severe injuries (including severe or even fatal head injuries), due to falls downstairs while using a walker and on the banning of walkers because of this risk [333–338]. In the eighties 30–50% of infants, placed in walkers, experienced at least one accident or sustained injuries due to the use [333, 339].

Fig. 5.27 Baby walker This media file is in the public domain in the United States. This applies to US works where the copyright has expired, often because its first publication occurred prior to January 1, 1925, and if not then due to lack of notice or renewal

Most baby walker-related injuries are bruises, abrasions, and minor cuts and occur due to various mechanisms: going head over heels, falling down the stairs or from an elevation, or by crushing of fingers. The most serious injuries occur when falling down the stairs or from an elevation:

- Kavanagh and Banco: 150 infants (aged 5–15 months) using infant walkers, surveyed in a 3-months period [340]. In 47 infants a walker-related accident occurred, leading to closed head injuries, fractures, lacerations, tooth evulsion, and soft palate perforation.
- Wellman and Paulson: Retrospective chart review of infants with baby walker-related injuries over a 23-month period [341]. Ninety-seven percent of the children sustained injuries to their head or face. The majority of the injuries were relatively innocent. Sixty-eight percent of the injuries were the result of falling down steps.
- Fazen and Felizberto: Forty-two infants (aged 8–12 months) using various types of baby walkers [342]. Twenty-one infants (50%) experienced at least at least one accident involving a tip-over, a fall downstairs, or finger entrapment. Two children sustained head and neck injuries after falling downstairs in a walker, that were serious enough to require medical management. Stairway and finger entrapment accidents occurred most commonly before the age of 7 months, while tip-overs were much more likely to occur after the age of 8 months.
- Stoffman et al. sent questionnaires to evaluate the use of baby walkers. Of the 152 responding families 82% reported the use of a baby walker. The incidence of baby walker-related falls was 36 and 8% needed medical care. To determine what proportion of head injuries in children under 24 months of age who presented to an emergency department were related to the use of baby walkers, the authors reviewed the charts of 52 such children. Baby walkers were involved in 42% of the head injuries in children under the age of 12 months and in none of the children aged 12–24 months. All baby walker-related injuries, including skull fractures in three children, were sustained in falls downstairs.
- Rieder et al.: Prospective study of baby walker-related injuries in infants presenting to the emergency department of a large paediatric hospital during a 1-year period [343]. They included 139 children with injuries, 29 of these were fractures. Falls downstairs accounted for 123 injuries (89%). The most severe injuries occurred in falls downstairs. Most frequent injuries were closed head injury (67%) and skull fractures (14%).

- Coats and Allen: Retrospective study of all infants under the age of 2 years attending an accident and emergency unit [344]. Twenty-two baby walker-related injuries were found in a total of 1049 attending infants. Skull fractures were found in three infants. These were considered to be the most serious injuries. Most injuries occurred due to falling downstairs in the walker. Injuries due to the use of baby walkers occurred with a similar frequency as injuries due to road traffic accidents.
- Partington et al.: One hundred twenty-nine children under the age of 2 years with head injuries (retrospective clinical review, 3-year period) [345]. Nineteen children sustained baby walker-related head injuries (mean age at time of injury 8.7 months). Almost 95% sustained head injuries due to falling downstairs. Nine children (almost 50% of the children with walker-related head injuries) had a calvarium fracture.
- Mayr et al.: One hundred seventy-two infants (aged 7–14 months) with baby walker-related injuries (retrospective study, 3.5-year period). One hundred twenty-five infants (73%) had bruises and lacerations of the head, including four teeth luxation). Nineteen children (11%) had a skull fracture, of whom 15 had a calvarium fracture and 4 a basilar fracture. 23 children (13%) had a concussion of the brain. Three children had a fracture or distortion of the arm. The authors recommended a general ban on the sale and manufacture of baby walkers [336].
- Chiavello et al.: Sixty-five children (aged 3–17 months, 95% under the age of 1 year) (prospective study, 3-year, 8-month period) with baby walker-related injuries [334]. In 46 children (71%) the injuries were due to stairway falls, in 14 children (21%) to tip-overs, in 2 children (3%) to falls from a porch. Three children (5%) sustained burn injuries. In 97% the injuries were located in the head and neck region. In 6% there were injuries on the extremities en in 3% on the trunk. Most injuries were minor. In 19 children (29%) significant injuries were found: fractures (skull, c-spine), intracranial injuries (concussion, intracranial haemorrhage), and full-thickness burns. Five children had intracranial haemorrhages. One child with a skull fracture, subdural haemorrhage, and a fracture of the cervical spine died. After excluding the children with walker-related burn injuries, all serious injuries were found in children who had fallen downstairs.
- Petridou et al.: Forty-nine infants (80% under the age of 10 months) with baby walker-related injuries (retrospective analysis, 12-month period) [346]. Most injuries, especially in children under the age of 10 months, were sustained in falls downstairs. Most injuries were found in the head and neck region and were minor (bruises and abrasions), although three children sustained fractures and one child a burn injury.
- Smith et al.: Two hundred seventy-one children (aged 4–20 months, mean age 9.2 months, mean age 8 months; 1 child was 36 months) with baby walker-related injuries [347]. Ninety-six percent fell downstairs, 1.5% from an elevated surface (e.g. curb or porch). The rest were injured in another way (falling out the walker, burning or shutting finger in door, while sitting in the walker). One hundred fifty-nine children had bruises and abrasions, 35 had concussions/head injuries, and 33 had lacerations. Twenty-six children sustained calvarium fractures (17 parietal, 8 frontal, and 1 occipital), three clavicular fractures, and one a radius and ulna fracture. Three children had a depressed calvarium fracture with an accompanying intracranial haemorrhage, of which two were subdural haematomas. Two of these three children had a second skull fracture without depression. The calvarium fractures all occurred in the group of children that had fallen downstairs. Falls downstairs ranged from 1 to 30 steps. A fall over 10 steps resulted in a significantly increased risk on a skull fracture. Supervision was present in 78% of cases, including supervision by an adult in 69% of cases.
- American Academy of Pediatrics (2001): In 1999, an estimated 8800 children under the age of 15 months were treated in emergency departments in the United States for baby walker-related and from 1973 through 1998 34 walker-related deaths were reported. Based on these data the AAP recommended a ban on the manufacture and sale of mobile baby walkers.

In summary, baby walkers are often used in everyday life but carry a high risk for injuries (up to 50%). Most injuries involve the head and neck regions. Especially falls from stairs with baby walkers occur often and have a high risk for severe injuries to the head.

Accidents: Toppling Televisions and Other Falling Heavy Objects

Injuries in children which are caused by toppling televisions or other falling heavy objects are compared to injuries sustained in other accidents rare [348]. However, because of the weight of some televisions, compared to the size of young children severe and sometimes fatal injuries can result [348, 349]. Various publications report a high morbidity and mortality in children due to toppling televisions. Wide-screen cathode ray tube (CRT) televisions are notorious, especially when placed on unstable cupboards, often with wheels, dressers or other furniture that the child can climb on [349–355]. Although most bulky CRT televisions in family homes have been replaced by flat screen televisions (LCD, LED), accidents with CRT televisions still are reported regularly. According to several recent studies, the number of toppling television-related accidents is even still increasing [356–

358]. In 2018, Safe Kids Worldwide stated that every 3 weeks a child dies from injuries, caused by toppling televisions [359].

Duhaime et al. defined the cause of head trauma, due to falling of heavy objects as static loading (‘*the child climbed or pulled on a heavy object, which then fell over with the child and landed on the child’s head*’) [243]. This type of accident, however, has more in common with dynamic loading, as found in accidental falls. In such a situation it is not rare for a double impact to occur: first, the moment that the child falls off the cupboard on top of its head and then the moment that a heavy object, like a television and/or the cupboard topple(s) over on the child. Both contact forms lead to dynamic impact loading. All children in Duhaime’s study had basilar cranial fractures.

Injuries due to toppling televisions are predominantly found in children between the age of 1 and 4 years and mostly concern injuries to the head and [354, 356, 360]. The most common injuries are head injuries (skull fractures, intracranial haematomas) (Table 5.8).

The most common cause of death in these children is severe head (skull and intracranial) injuries [352, 354, 356]. Children that died as the result of injuries due to toppling televisions instantly showed clinical symptoms and were in near immediate need of intensive care.

- Bernard et al. reported on 73 accidents with toppling televisions in children (boys 31; girls 42) (average age 36 months; SD ± 25.4 months) [350]. The data were derived from a retrospective analysis of incident files of the US Consumer Product Safety Commission (CPSC) data systems and The Children’s Hospital of Alabama inpatient medical records. Twenty-eight children (boys 10; girls 18) (average age 31 months; SD ± 22 months) died due to the accident. The head was the most prevalent anatomical location for injuries (externally visible injury, skull fractures, and intracranial injuries) (72%). Of the 14 deceased children, who were further investigated by the CPSC, 13 died from head injuries, while the remaining child died from generalized crushing injuries (injuries in which several body parts and organs are seriously damaged and/or crushed).
- DiScala et al. evaluated the findings in 183 children under the age of 7 years (76% between the age of 1 and 4 years) (almost 60% boys), hospitalized because of injuries caused by toppling televisions (data from the US National Pediatric Trauma Registry) [351]. 68.3% of the 183 children had head injuries, and 43.7% had injuries to one or more body parts or organs. More than a quarter of children had injuries with an injury-severity score of 10–75. Approximately one-third of the children had to be admitted to an intensive care unit. Five children died due to massive intracranial haemorrhages.
- Scheidler et al. evaluated the findings in 43 children, aged 0–16 years, and found that the most common injuries were to the head, abdomen, and arms/legs (fractures) [354]. The majority of television-related injuries occurred in toddlers, aged 1–3 years, who were left unattended at home. Five children died, all resulting from head injuries. Four children sustained an abdominal trauma, and in three children a surgical intervention was indicated. None of the children with abdominal trauma died.
- Jea et al. reported on seven children, aged 18–36 months (average 22 months), with injuries due to toppling televisions. Four children sustained a calvarium fracture, two children a basilar fracture [352]. Skull fractures were found most often in children under the age of 24 months.
- Yahya et al. reported on 18 children (13 boys, 5 girls), aged 12 months to 10 years (mean 44 months) with injuries caused by toppling televisions [355]. Radiological findings were 16 skull fractures, 3 epidural haematomas, 3 small subdural haematomas, one intracranial haemorrhage (not otherwise specified), and three venous obstructions of the transverse-sigmoid sinus. Three children had cranial nerve deficits and three had otorrhoea, otorrhagia, or haemotympanum.
- Ota et al. analyzed 26 children (mean age 40 months) who were injured by a falling television. There were 14 head injuries (8 fractures and 4 intracranial bleedings) and 9 children with an injury of an extremity (5 fractures). Nine children were hospitalized, including two patients admitted to the intensive care unit [353].
- Marnewick et al. analyzed the findings in 13 children under the age of 15 years with injuries, caused by toppling televisions [348]. Nine children had head injuries and one child died.
- Befeler et al. evaluated the findings in 26 paediatric patients (19 boys, 7 girls). 85% of the incidents occurred in 2–4 year old children [360]. Twenty children had head

Table 5.8 Anatomical location of injuries and ‘injury severity score’ in toppling televisions [351]

Anatomical location of the injury	*N*	%
• Skull/brain	58	31.7
• Arms or legs	28	15.3
• Face, abdomen, skin	17	9.3
• Combination of more than two injuries: skull/brain, face, chest, abdomen, arms, legs, skin	80	43.7
• Total head/neck area	125	68.3
Injury severity score	*N*	%
• 1–9 (mild)	127	69.4
• 10–15 (moderate)	32	17.5
• 16–24 (severe)	13	7.1
• 25–75 (life-threatening)	7	3.8
• Unknown	4	2.2

injuries, ranging from concussions to skull fractures and subdural, subarachnoid, and intraparenchymal haemorrhages. No child died.
- Eren et al. described 86 children (47 boys, 39 girls), aged 9 months to 8.5 years (mean age 38.8 ± 19.5 months), with injuries, all caused by toppling CRT televisions [358]. Nineteen children had skull fractures and 12 had intracranial haemorrhages. One child had permanent neurological damage and one child died.

Therefore, injuries sustained from toppling televisions often cause head trauma that is potentially serious and even life threatening. Mortality rates up to 38% have been reported.

Accidental Circumstances: Traffic Accidents

Severe trauma, such as a motor vehicle accident or motor vehicle versus pedestrian accidents, may cause head injuries, including calvarium fractures and intracranial injuries. However, in those cases, the accident is almost always observed by an independent spectator and the patient's history will be supported by statements of this spectator and the history corresponds with the injuries found. Therefore, independently observed accidental calvarium fractures will rarely if ever be confused with calvarium fractures due to non-accidental circumstances. For a comprehensive overview regarding the origin of calvarium fractures accompanied by intracranial injury and other fractures and possible death based on accidental causes, we refer to Sect. 5.6 and Chap. 13.

Accidental Circumstances: Daily Activities

Injuries to scalp, calvarium, and intracranial contents can also occur due to ceiling fans:

- Alias et al. (2005): Fourteen children with head injuries due to fan blades in (mean age 7.9 years; range 1.0–12.2 years; most often school-aged boys). The circumstances under which the injuries were sustained included jumping on the upper bunk of a bunk bed, climbing on a ladder, climbing up onto a table, and being lifted by an adult. The injuries consisted of scalp lacerations, compound depressed fractures, and multiple intracranial haemorrhages. One child died from severe head injuries.
- Furyk et al.: One hundred thirty-six paediatric patients with ceiling fan-induced head injuries. Most children had no physical findings [361]. If present, lacerations of the scalp were most common. Skull fractures were found in seven children. Most of these fractures were palpable. The circumstances included getting in and out of the upper bunk bed, jumping from furniture, or being held up by an adult.
- O'Donnell: A 12-month-old girl with a large scalp laceration and a depressed curvilinear calvarium fracture and underlying haemorrhagic contusion of the brain. The girl was held up by an adult and struck on the head by a ceiling fan [362].
- Hoz et al.: Twenty-nine paediatric patients with head injuries due to metallic ceiling fans [363]. Most of the injuries occurred while climbing on or jumping from furniture. Most children were between 2 and 5 years of age. Compound depressed skull fractures were most common, often associated with intracranial injuries and pneumocephalus.

5.3.3.5 Trauma After Birth: Non-accidental Circumstances

In non-accidental circumstances, a calvarium fracture can be caused by a direct impact due to a considerable blunt force trauma, e.g. when being punched with a fist or in a contact with a flat surface, like being hit with a shelf or thrown on the floor or against a wall.

Epidemiology

Calvarium fractures are sustained in non-accidental circumstances only in a relatively small part of all paediatric calvarium fractures, although, according to some authors, skull/calvarium fractures are the second most common fracture occurring in non-accidental circumstances [181, 364, 365].

Johnstone et al. evaluated 409 children under the age of 13 years; only 3% of skull fractures were sustained in non-accidental circumstances [366]. However, this percentage seems to increase dramatically as the studied population gets younger. Hobbs found that 33% (29 of 89 children) of calvarium fractures in children under the age of 2 years of age occurred in non-accidental circumstances [256]. Loder and Bookout evaluated the data of children under the age of 16 months that had sustained fractures in non-accidental circumstances and found skull fractures in 35% of children [367]. Leventhal et al. studied 93 children under the age of 3 years with skull fractures; 80% was under the age of 1 year [368]. In the group of children under the age of 1 year, 27% of skull fractures occurred in non-accidental circumstances (child abuse). Reece stated that 80% of inflicted skull fractures occur in infants under the age of 1 year [369]. According to Kleinman and Barnes 7–30% of all inflicted fractures in children are skull fractures and 10–13% of all inflicted injuries concern skull fractures [184]. Merten et al. found a comparable percentage, slightly less than 10% (67 children with a skull fracture) of a total of 712 children with inflicted injuries [370]. Neither Kleinman nor Merten et al. differentiated for age. Pandya et al. found that calvarium fractures in children under the age of 18 months were more often sustained in accidental than non-accidental circumstances [371]. According to Kleinman et al. skull fractures are found in 41% of children that had died as a result of non-accidental circumstances (physical violence) [180].

Differential Diagnosis, Based on Type of Calvarium Fractures

No type of calvarium fracture offers the possibility to differentiate a non-accidental fracture (an inflicted injury) from an accidental fracture [372]. Even if a history of trauma is lacking in a child who is diagnosed with a calvarium fracture and a soft tissue swelling of the scalp, this does not indicate an inflicted injury, because, e.g. subgaleal and subperiosteal haematomas may develop gradually in the course of several days (up to weeks) (Sect. 5.2).

The most common inflicted calvarium fracture is a unilaterally localized, simple linear fracture of the parietal bone without depression. However, this also happens to be the most prevalent skull fracture in accidents [169, 368, 373].

When the fracture is bilaterally present or when there are multiple fractures with depression and diastasis >3 mm, one should consider non-accidental circumstances, especially with an ambiguous clinical history. Also, in depressed fractures, fractures with diastasis of the fracture lines and occipital fractures, one should consider physical violence as a possible cause (Table 5.5) [28, 256, 368, 370, 374].

However, the presence of the earlier-mentioned characteristics or locations of calvarium fractures, taken out of their context, can never be considered as absolute proof of non-accidental circumstances [375, 376]. Meservy et al. evaluated 134 children under the age of 2 years and found that in 39 infants (29%) the calvarium fracture was inflicted [377]. The parietal bone was the most common fracture site in both accidental fractures (91.3%) as in non-accidental fractures (87.5%). They also did not find differences in the occurrence of impression fractures, diastatic fractures (≥3 mm), or complex fractures between children who were involved in an accidental trauma compared to children with a non-accidental trauma. The age of the child also offered no possibility of differentiation between accidental and non-accidental circumstances. Meservy et al. did also find that multiple or bilateral fractures or fractures that cross the sutures were more common in or highly suspect for non-accidental circumstances [377]. The literature seems to confirm the finding of Meservy et al. concerning the crossing of sutures (e.g. [28, 374, 377]). However, fractures that continue into the adjacent bone are also found in accidental circumstances [177, 378].

5.3.3.6 Medical Differential Diagnosis of Calvarium Fractures Due to Trauma After Birth

In the radiological differential diagnosis one should be aware of so-called pseudo-fractures, such as impressions of blood vessels, but also different aspects of sutures and connective tissue fissures (Sect. 5.3.4.7) [379]. Also, super-positioned externally localized objects may cause confusion. For example, this may the case with plaids or hair bows. In Chap. 14, normal variants, resembling calvarium fractures, will be dealt with. Also, congenital and acquired disorders with an increased risk of fracturing will be described in Chap. 14.

5.3.4 Types of Calvarium Fractures

5.3.4.1 Introduction

The type of calvarium fracture that is sustained due to trauma is largely dependent on the same trauma- and anatomy-related factors that determine whether static or dynamic impact loading will result in a fracture: the amount of transferred energy during the impact, the location of the impact, and the shape of the impacting object (Sect. 5.3.2) [380]. Calvarium fractures can be classified according to several criteria [166, 380, 381]:

- The anatomical fracture location: Fractures in a single bone (frontal bone, parietal bones, temporal bones, occipital bone) or in more than one bone, e.g. frontoparietal or parietooccipital.
- The integrity of the skin: Closed versus open fractures. In a closed fracture the skin, covering the fracture, is intact, while in an open (or compound) fracture there is an associated laceration of the skin and the bone fragments may be visible. In penetrating injuries there is not only a calvarium fracture, but also a laceration of the skin and injury to the dura (Fig. 5.28a–c). This results in an injury that has an open connection between external and intracranial environments, presenting a considerable risk for infection. A pointed object is more likely to penetrate the skull than a hard and flat surface, such as the surface of the floor or a wall.
- The characteristics of the fracture: Wiersema et al. proposed a 3-stepped classification system for a standardized description of paediatric skull fractures [382]. The steps describe consecutively the category, the pattern, and the description:
  - Category: Three types: Simple, complex, and comminuted.
  - Pattern: Adds detail to the category, like linear, curvilinear, stellate, or diastatic.
  - Descriptor: Describes additional features of the fracture, e.g. depressed, displaced, and degree of healing.

Category is the basis of this classification system (Fig. 5.29a–c):

A simple fracture is defined as any fracture with two terminal ends, regardless of its course, length, and severity. They may cross one or more sutures, travel along a suture, follow a curved path as long as it has only two terminal ends.

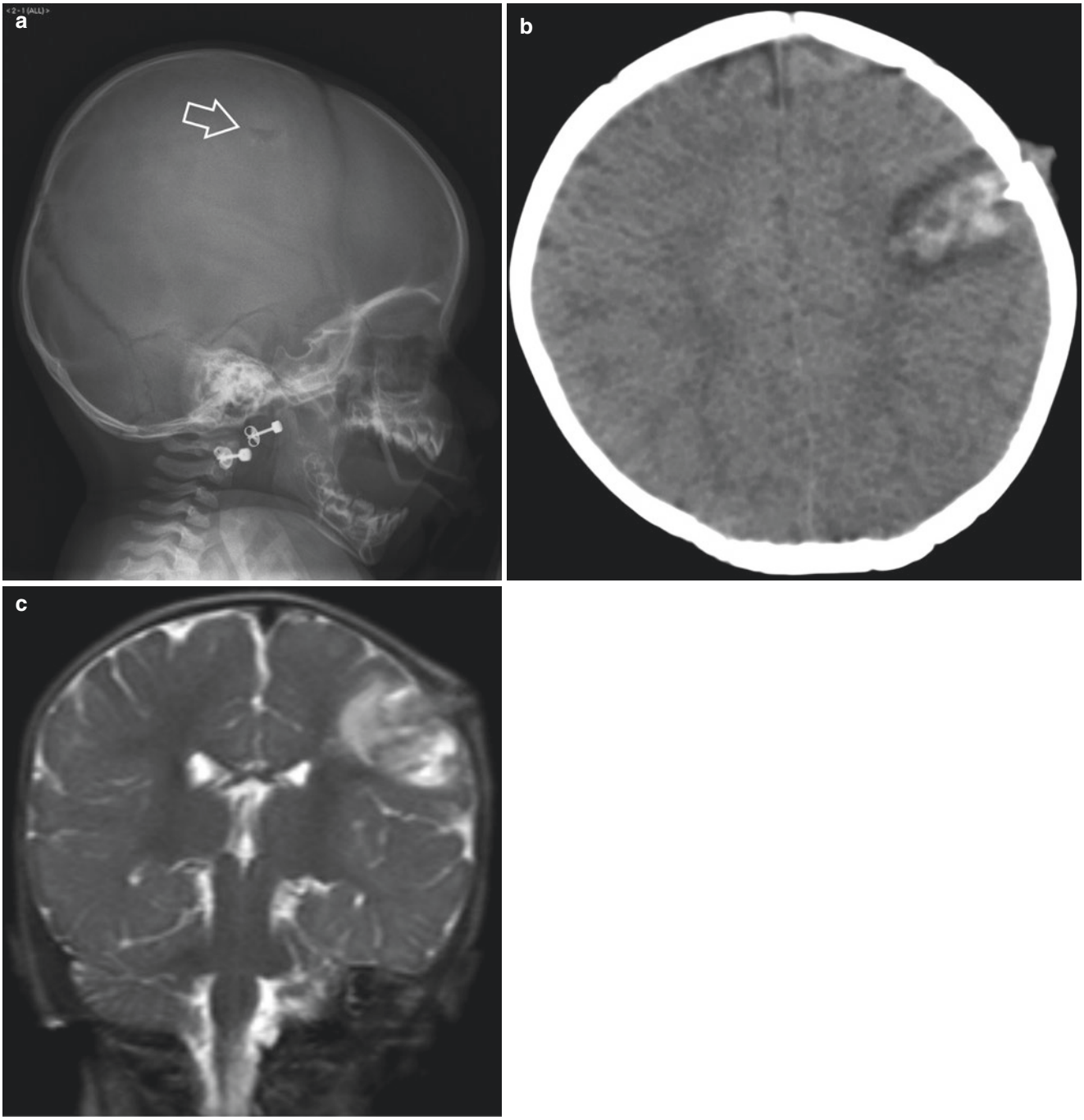


Fig. 5.28 An infant with a penetrating injury caused by a forcefully introduced key. (**a**) A small defect on the skull radiograph depicts the entry of the key (arrow). (**b**) CT and (**c**) MRI show the intracranial haemorrhage and skin laceration

A complex fracture has three or more terminal ends, regardless of its complexity. A comminuted fracture is defined as a fracture that results in an island of bone.

Patterns of fractures are commonly occurring fracture characteristics adding details to the basic categories like linear (more or less straight), curvilinear, stellate, diastatic, and zigzag (Fig. 5.30a–f). The term diastatic is bivalent, some authors consider diastatic fractures as fractures with diastatic enlargement (>4 mm) [383], others define diastatic fractures as fractures that involve a suture line, either partially or wholly [384].

Descriptors describe additional features of the fracture that do not fit in categories or patterns, like depression, displacement, and stage of healing (Fig. 5.31a, b).

Every type of calvarium fracture may potentially develop into a ‘growing fracture’ (Sect. 5.3.4.6).

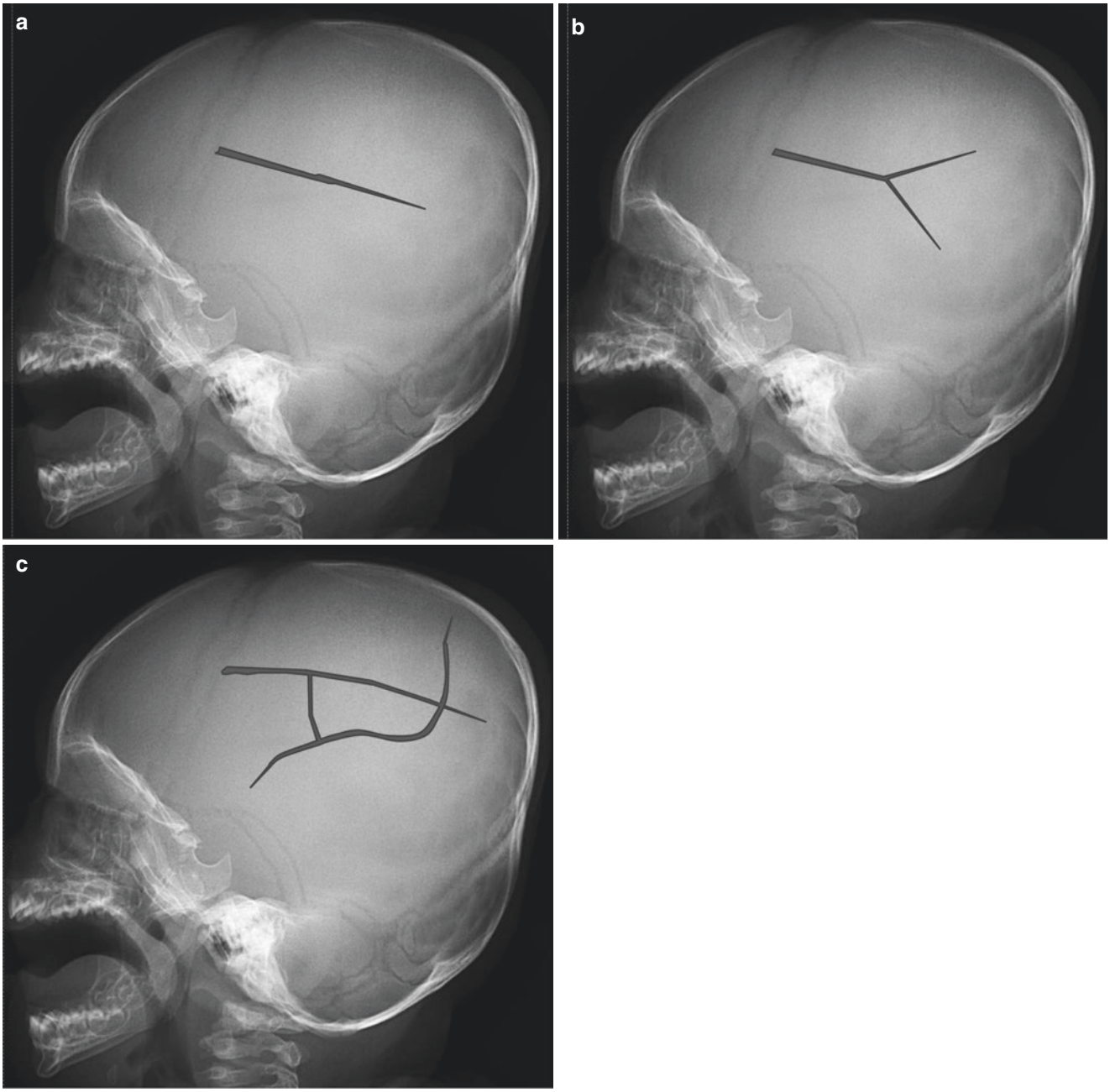


Fig. 5.29 The three basic categories of skull fractures: (**a**) simple, (**b**) complex, and (**c**) comminuted

5.3.4.2 Simple (Single Line)/Linear or Curvilinear Calvarium Fractures

The most prevalent type of fracture of the cranium is the single-line linear fracture. Wiersema et al. defined this type of fracture as '*a simple fracture as any fracture with two terminal ends, regardless of its course, length, and/or severity. A simple fracture may cross one or more sutures, travel along a suture, or assume a curved path as long as it has only two terminal ends*' (Figs. 5.29a, 5.30a, b, d–f, and 5.32a–d). Of all skull fractures in children, 74–90% are single-line/linear fractures. This type of fracture is usually restricted to one skull bone. Linear fractures may be present bilaterally and symmetrically. Linear fractures result from a contact with a large flat object, in which the impact of a blunt trauma spreads over a large area, e.g. the fall from the arm of a parent/caregiver that results in the head of the child falling onto the floor (Fig. 5.33a, b) [385]. This is a typical example of relatively 'low velocity' dynamic impact loading [26].

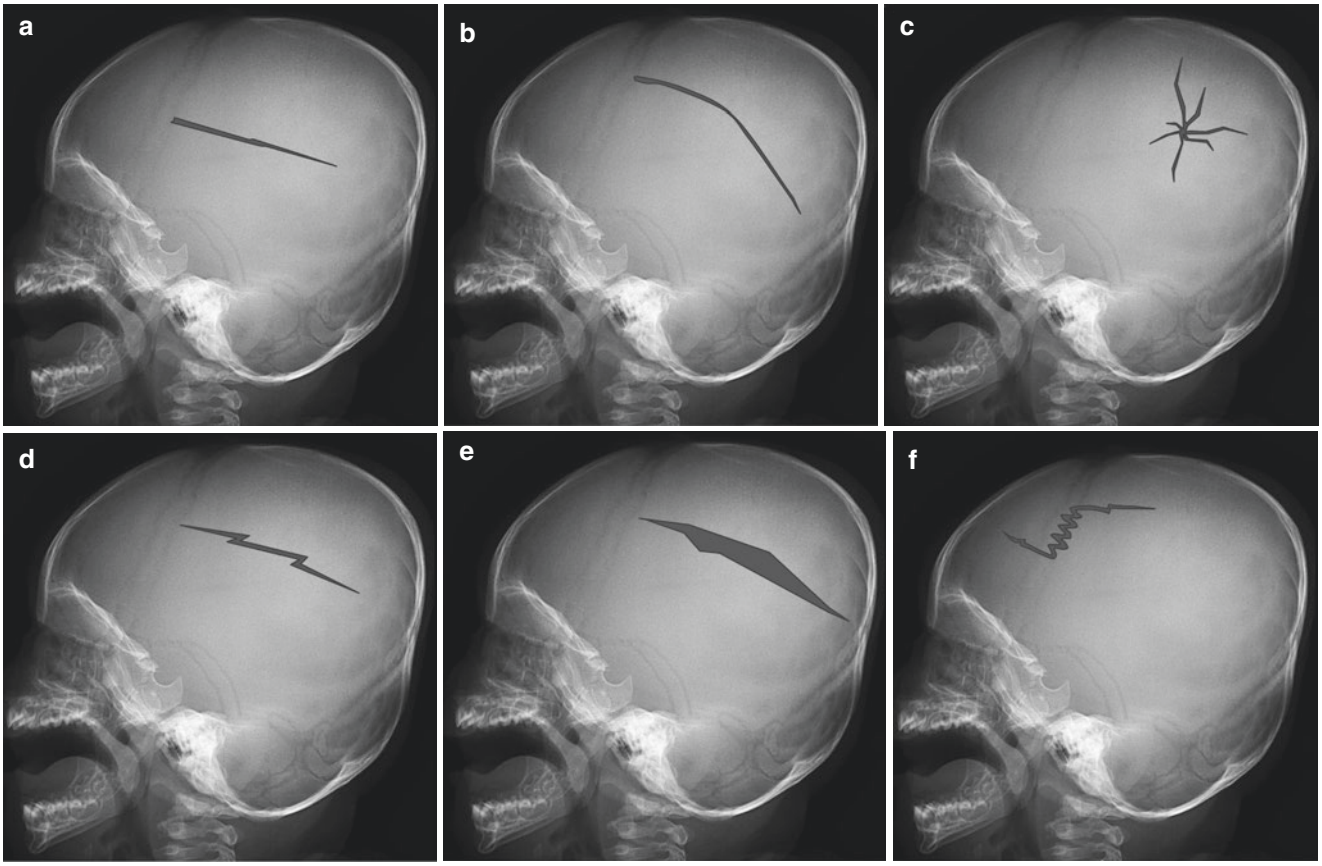


Fig. 5.30 Patterns of skull fractures. (**a**) Simple linear, (**b**) simple curvilinear, (**c**) complex stellate, (**d**) simple zigzag, (**e**) simple linear diastatic, because wider than 4 mm, and (**f**) simple zigzag diastatic, because partially running along the coronal suture

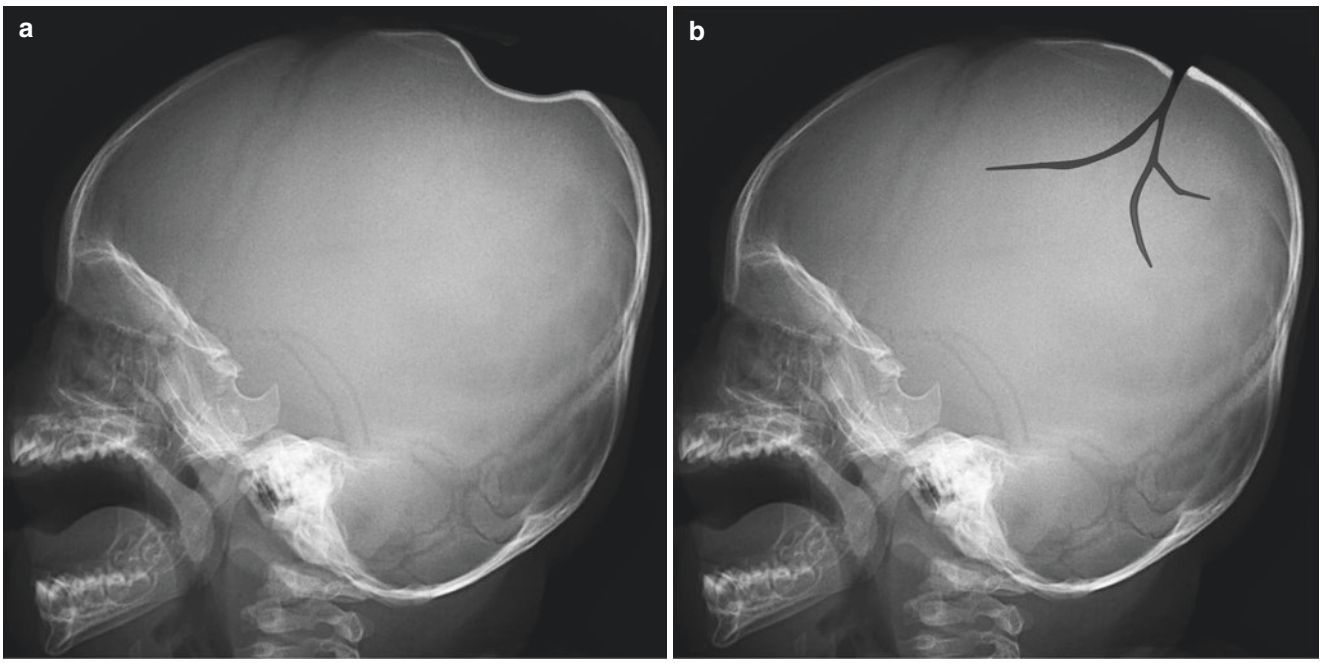


Fig. 5.31 Descriptors of skull fractures. Depressed (Ping-Pong) fracture (**a**), complex curvilinear displaced fracture (**b**)

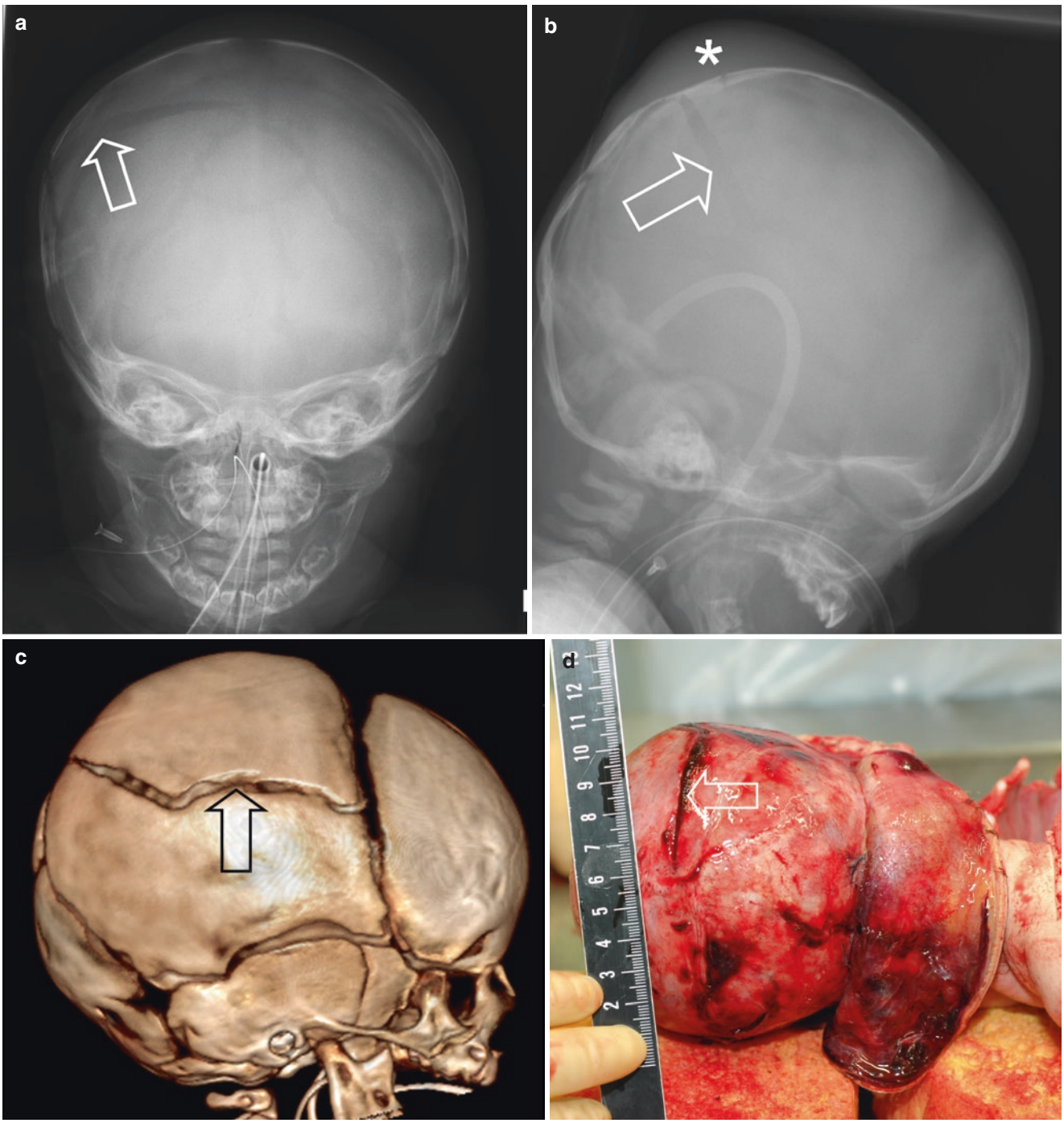


Fig. 5.32 (**a**) Infant who, according to the clinical history, had fallen from the changing table (85 cm high). When presented at the emergency department she was comatose. Five days later she died from neurological trauma. The anterior-posterior skull view shows a bilateral linear fracture that transgressed multiple sutures (open arrows). (**b**) Lateral skull view shows besides the fracture in the parietal bone (open arrow) a clearly visible soft-tissue swelling corresponding to a post-traumatic haematoma (asterisk). (**c**) The fracture is visible on the three-dimensional CT reconstruction (open arrow); furthermore, conform the child's age, the sutures are still visible). (**d**) At autopsy the fracture in the parietal bone is clearly visible (open arrow)

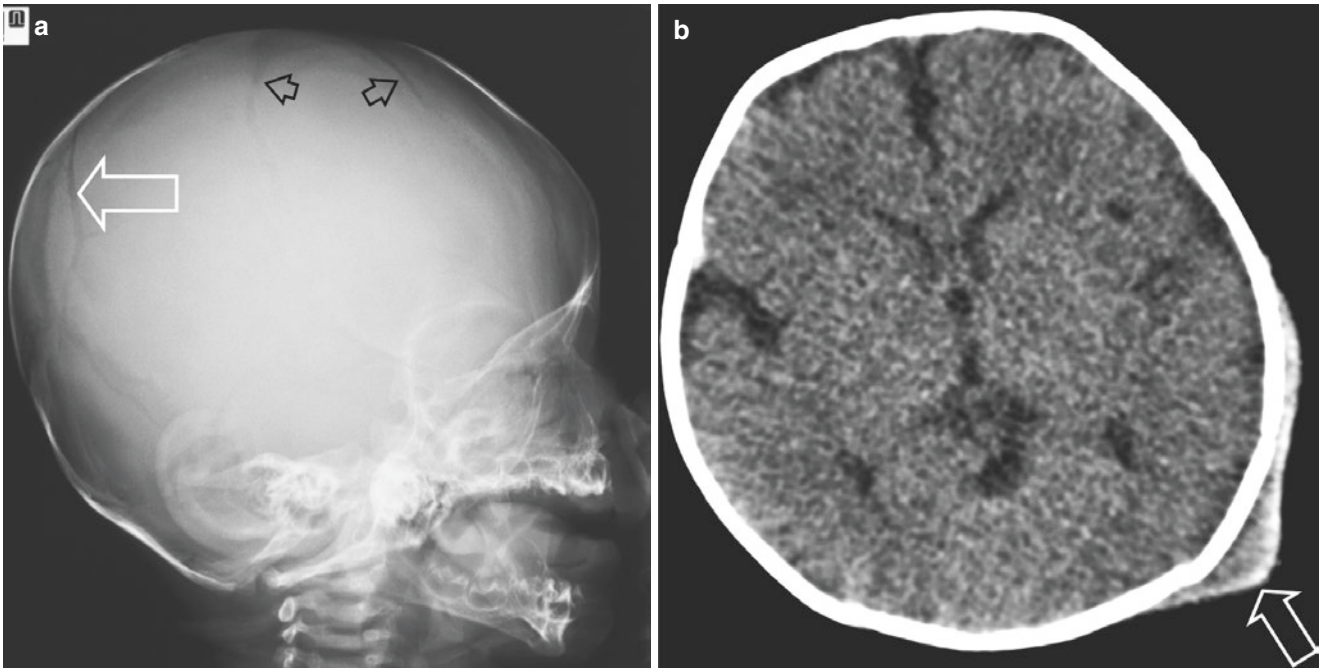


Fig. 5.33 (**a**) Two-month-old baby who, according to the clinical, had fallen from the arms of his 7-year-old sister. The fall had not been witnessed. The lateral view of the skull shows a parietal linear fracture (white arrow). The small black arrows indicate both coronal sutures, wide apart because of rotation of the skull. (**b**) Additional CT in this patient shows post-traumatic soft-tissue swelling (arrow) but no intracranial pathology

When the head impacts on an object with a large flat surface, the skull curvature flattens under the influence of the contact. The skull surface bows inwards, whereas the surrounding area bows outwards in a wave-like manner (Fig. 5.34) [178, 385]. The outward bowing of the skull may occur at a relatively large distance from the primary site of contact. Hence, the location of a linear fracture does not have to correspond with the place of contact [373]. After the skull has been deformed by the impact, it will try to resume its normal shape. At the moment that the inwardly bowed part resumes its normal shape, the fracture will spread from its original location into the direction of the place of impact as well as into the opposite direction. This may result in a fracture line that reaches the original place of contact or extents even further [26].

Although linear fractures are usually confined to one skull bone, it is possible that the fracture extends into the adjacent skull bone. In most linear fractures, external injuries are found, such as swelling of the overlying tissues or a haematoma. Sometimes a subgaleal haematoma is seen (Sect. 5.2.3). The extent of the subgaleal haematoma may be such that it leads to anaemia [1].

In approximately 15–30% of linear fractures intracranial injury is found (Sect. 5.6) [171]. Linear fractures tend to show diastasis (Sect. 5.3.4.6). However, in most patients linear fractures heal without any problems.

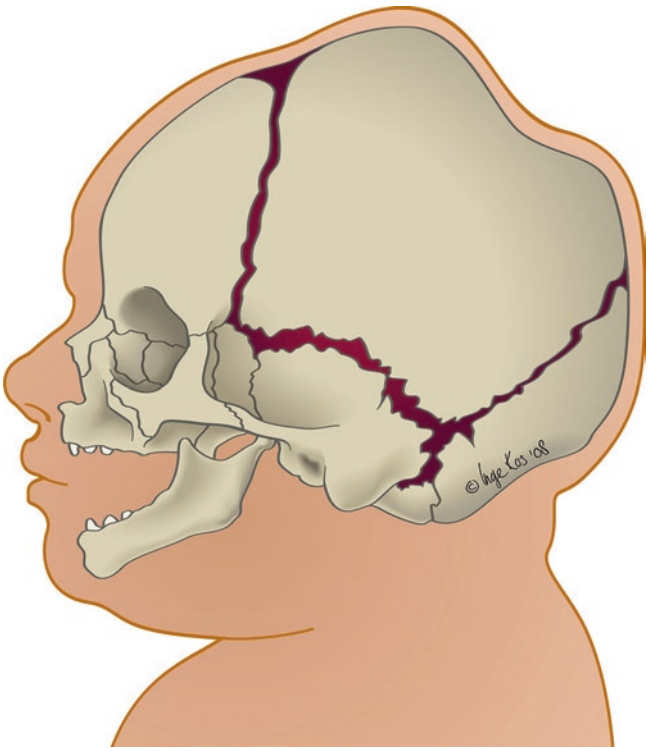


Fig. 5.34 Schematic representation of the wave pattern of skull deformation after contact with a relatively large surface. At the impact site, there is inward deformation whereas peripherally the skull bows outwards

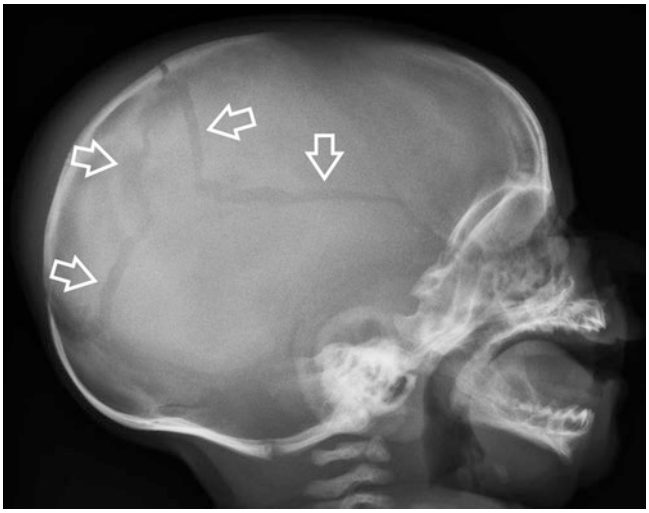


Fig. 5.35 Bilateral parietal diastatic linear fracture (arrows) crossing the midline in a 4-week-old infant who fell from the arms of his mother because she slipped from the second step of the stairway. Skeletal survey was normal. No retinal haemorrhages. After a short period of drowsiness there was an uneventful recovery

Table 5.9 Recommended evaluation for an infant with bilateral calvarium fractures (after O'Hare [386])

Assessment	Noteworthy features
Detailed history	• Characteristics of the fall: fall height, trajectory, impact surface, complicating factors • Independent eyewitness
Complete physical exam	• Skin findings (incl. scalp): bruises, abrasions, swelling, pain. • Orofacial findings • Other signs of trauma (thorax, abdomen, extremities)
Psychosocial assessment	• Prior and current child protection involvement • Prior and current injuries • Prior and current poor child care, concerning this child and other children in the family • Prior and current domestic violence between family members (between parents, between children)
In case of suspicion of non-accidental circumstances or if medically indicated	
• Non-contrast cranial CT, incl. 3D reconstruction	
• Complete skeletal survey	
• Complete laboratory work-up	

5.3.4.3 Symmetrical or Bilateral Linear Calvarium Fractures

Sometimes bilateral and nearly symmetrical linear fractures are found in children. These can be due to accidental and non-accidental circumstances (Fig. 5.35) [386].

The occurrence of bilateral and nearly symmetrical linear fractures can be explained in various ways:

- Single contact trauma at the centre of the vertex: Arnholz et al. described a 6-week-old child with symmetrical biparietal fractures after a fall from a perambulator [281]. The fractures did not pass the sagittal suture. The fall was observed by an eyewitness. The child landed 'head first' on the centre of the calvarium on a concrete base. The child also had two separate bilateral subgaleal haematomas in the parietooccipital area [281]. The same mechanism can occur when a child is hit against the wall with great force and the energy transferred at the impact spreads symmetrically over, e.g. the parietal skull bones.
- Single unilateral (one-sided) impact trauma: According to Kleinman and Barnes bilateral fractures, crossing the midline, can also be caused by a single and unilateral impact trauma [184].
- Impact trauma at two different moments: According to Offiah and Hall, an impact trauma at two different moments would be more likely in a non-accidental than in accidental circumstances [249].
- Bilateral compression: Hiss and Kahana did a post-mortem forensic analysis of four young children with bilateral calvarium fractures [387]. They concluded that symmetrical fractures appear to be more often the result of bilateral compression between two surfaces (static loading) than of a one-sided localized dynamic impact trauma. Offiah and Hall described this mechanism as the effect of a single 'crushing force, applied simultaneously to both sides of the head', e.g. by standing on the child's head [249].

In Table 5.9, an overview is given of the recommended evaluation to differentiate accidental from non-accidental circumstances [386]. If bilateral and symmetrical calvarium fractures are suspected, one should consider accessory sutures in the differential diagnosis [386].

5.3.4.4 Complex and Comminuted Calvarium Fractures

Wiersema et al. defined a complex fracture as '*a complex fracture is any fracture that has three or more terminal ends, regardless of its level of complexity*' (Figs. 5.29b and 5.30c), and a comminuted fracture as '*a comminuted fracture is defined as any fracture that results in an island of bone. The island of bone must be surrounded on all sides by fracture*' (Fig. 5.29c and 5.36a) [382]. Several patterns and descriptions of complex and comminuted calvarium fractures can be found in children:

- Complete or incomplete circular/concentric fractures may occur around the point of impact, due to blunt force trauma, occurring when a solid object hits the head with a high-velocity impact. Concentric fractures are typical bowing fractures: the circles are formed on the outer surface of the skull at the junction of the inward and outward bowing part of the skull, as a result of the extreme bowing at the point of impact (Fig. 5.36b) [26, 388].

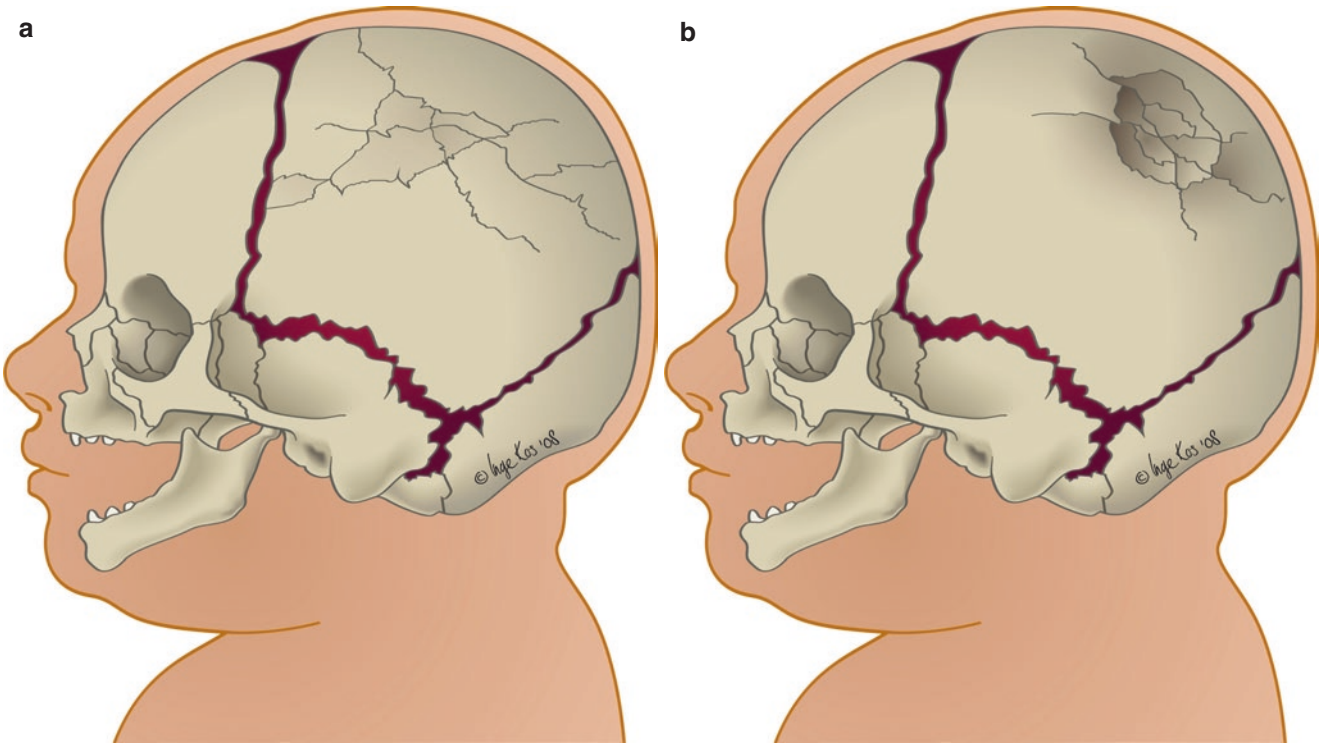


Fig. 5.36 Schematic representation of a comminuted fracture (**a**) and a comminuted stellate depressed fracture (**b**)

- Star-shaped or stellate fractures occur when a flat object impacts on a bowed bone with a (very) high velocity. At the point of impact, the bone suffers an impression that results into a number of fractures that all originate from the inward-bowing point of impact [385]. Star-shaped and circular fractures may both be present.
- Complex fractures with signs of crushing occur due to one impact with a very high transfer of energy, e.g. in a long-distance fall, or due to multiple impacts to the head, e.g. when the skull is repeatedly hit with a hammer. In this type of fracture, the skin may or may not be intact (Fig. 5.37).

According to Offiah and Hall, complex bilateral skull fractures are a strong indication of non-accidental circumstances (inflicted injury), unless there is an acceptable medical history with regard to serious accidental circumstances [249]. Complex bilateral skull fractures are probably due to more than one impact on both sides of the head or to a crushing force, applied simultaneously to both sides of the head.

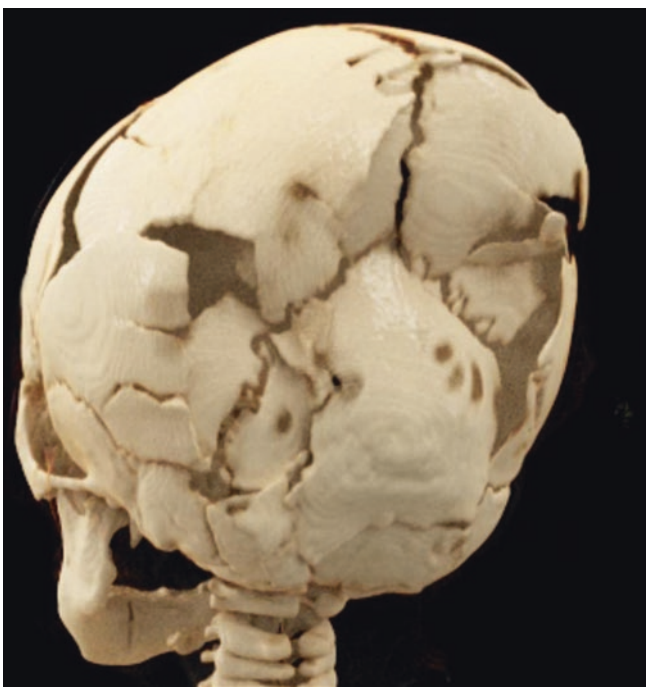


Fig. 5.37 Infant who was the victim of abusive head trauma. Cinematic rendering shows the extent of the comminuted diastatic depressed skull fracture with elevated fragments

5.3.4.5 Complete and Incomplete Depressed Calvarium Fractures

A depressed calvarium fracture is a fracture in which a part of one of the calvarium bones has buckled inwards. A depressed fracture can be complete, with inward buckling (depression) and clearly recognizable fracture lines ('true fracture'), or incomplete, with an inward buckling and no recognizable fracture lines ('ping-pong or celluloid fracture'). A ping-pong fracture is usually only seen only in infants, generally under the age of 6 months, due to the larger malleability and elasticity of the immature calvarium/skull, because of not being ossified [373]. In a ping-pong fracture,

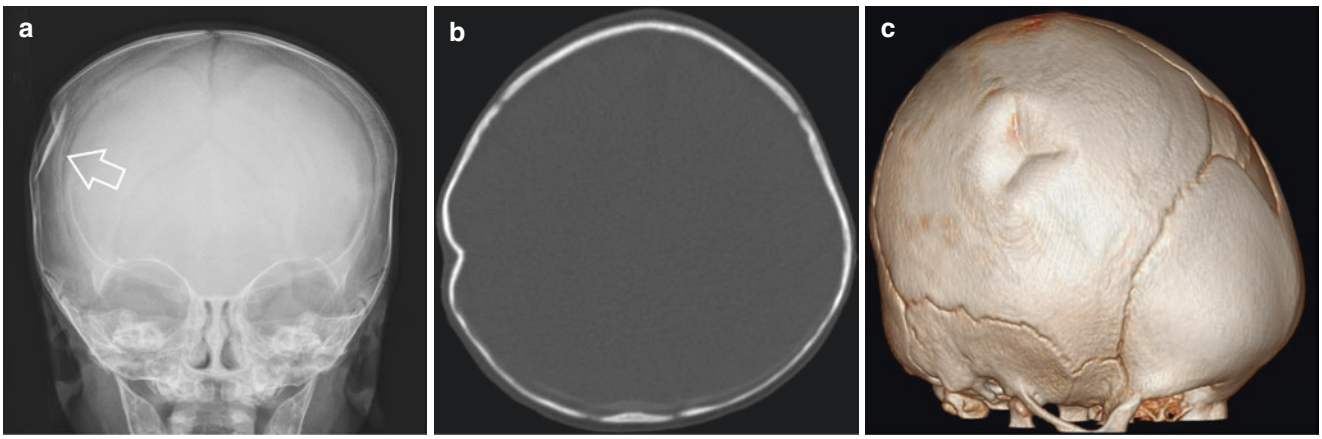


Fig. 5.38 Infant who fell from a couch. (**a**) AP skull radiograph shows a ping-pong or celluloid fracture of the right parietal bone (arrow). (**b**) CT confirms the location of the fracture. (**c**) 3D reconstruction

part of one of the calvarium bones transforms from convex to concave (Fig. 5.38a–c) [389, 390].

A child can sustain a depressed calvarium fracture before, during, and after birth (Sects. 5.3.3.1–5.3.3.3). This type of fracture can be caused by static loading (compression) or by dynamic impact loading (blunt force trauma). The circumstances under which a child sustains a depressed calvarium fracture, can be accidental or non-accidental, but in rare instances may be due to medical procedures, like the use of instruments during birth (Sects. 5.3.3.1–5.3.3.3). Irrespective of the circumstances, a depressed fracture, due to dynamic impact loading, can occur when an object with a small surface (e.g. a hammer or the heel of a shoe) hits the calvarium and a high transfer of kinetic energy takes place, or when an object (irrespective of the size of the object) hits only a small part of the skull with a high transfer of kinetic energy.

According to Erşahin et al., a depressed calvarium fracture can be found in 7–10% of all children that are admitted to hospital with a head trauma and in 15–25% of all calvarium fractures in children [391].

Luckett probably was the first to describe a depression of the calvarium without a fracture line [392]. He used the term ping-pong ball indentation to describe this. He described a 5½-months-old infant, who, supposedly, had fallen from a low couch (less than 2 ft–50 cm), striking the left side of the head (Figs. 5.38c and 5.39). He described the following: '*We obtained a dozen ping-pong balls and by holding them firmly in the hand and striking both sides consecutively against a solid flat surface reproduced the same tri-cornered depression nineteen times out of twenty-four. The other five depressions were quadrilateral. This tendency to form tri-cornered depressions from a blow produced by a flat surface seems to be characteristic of hollow spherical bodies with thin walls, and might have some medicolegal value relative to injuries of the vault*' [392].

Erşahin et al. evaluated the data of 357 boys (67%) and 173 girls (33%) (ages ranging from 1 day to 16 years; mean age 6.1 years) [391]. They found that the depressed fracture was most commonly sustained in an accidental fall.

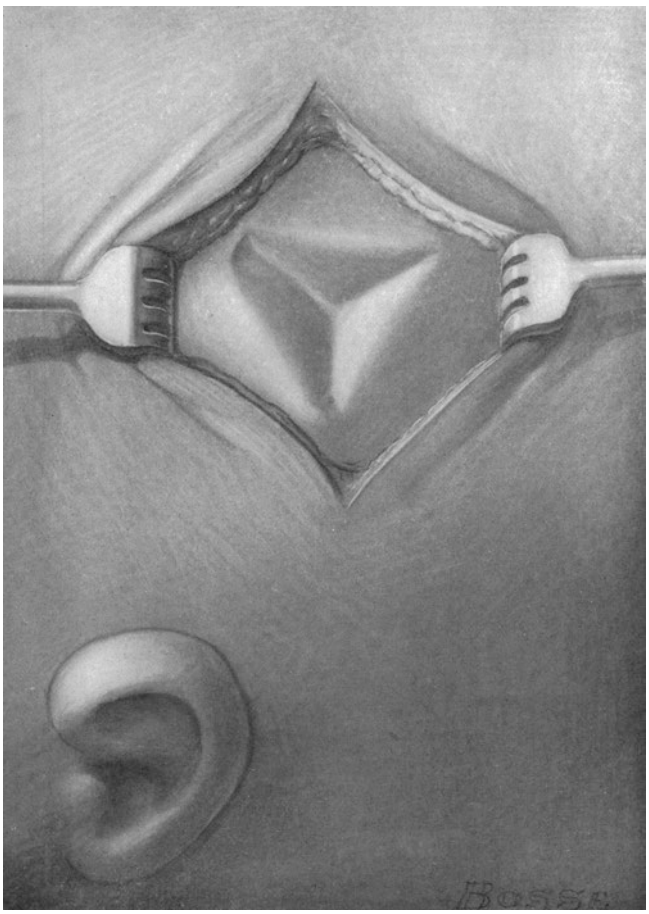


Fig. 5.39 Tri-cornered impression of skull without fracture [392]

Zia et al. reported the case of a 7-week-old boy with a ping-pong fracture, who was involved in a road traffic accident [393]. The boy had a right sided skull depression, which was palpated during physical examination and confirmed on a CT scan. No fracture line was seen.

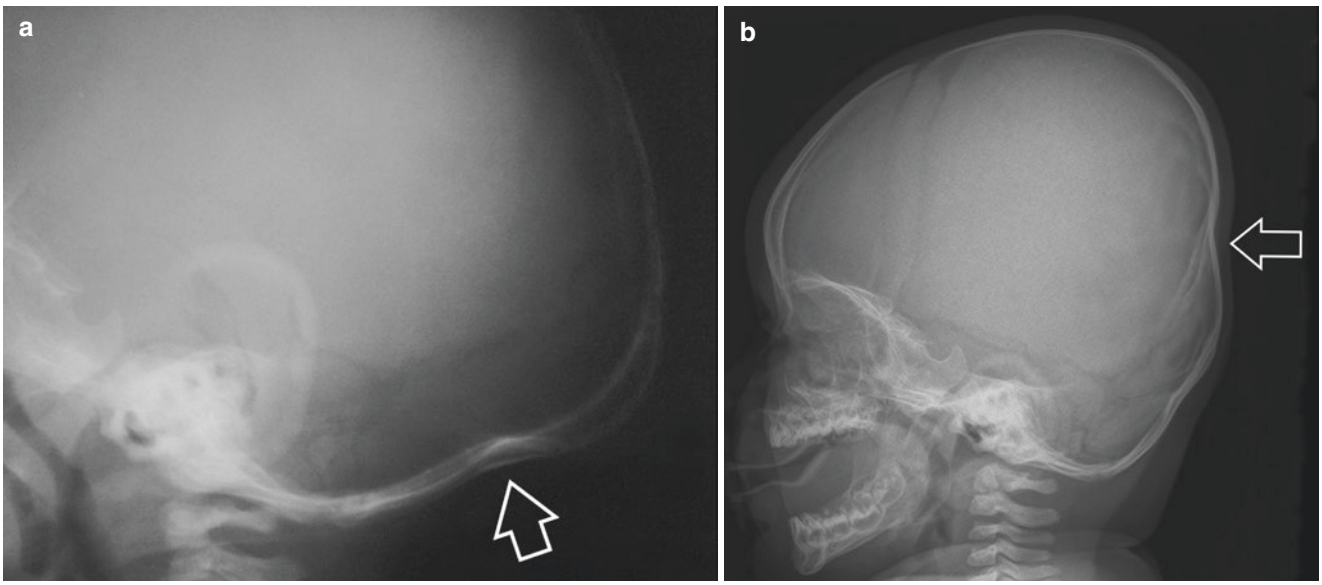


Fig. 5.40 Mimics of depressed fractures (arrows). (**a**) Bathrocephaly and (**b**) postural non-synostotic brachycephaly

In a depressed fracture, there is hardly any deformation of the skull besides the primary point of impact [178, 385]. At the point of impact a fracture is sustained, possibly with fragmentation. The depression results from the inability of the inner layer of the bone to absorb the inward bowing adequately. The depression may reflect the shape of the object. Sometimes there is only a depression in the outer layer, whereas the inner layer remains intact [26]. Sometimes a compound fracture can occur, in which the skin is broken and the damaged part of the calvarium is splintered.

In most depressed fractures no complications will occur. Erşahin et al. found that 66% of the 530 paediatric patients they evaluated had open (or compound) fractures and that the risk of sustaining a compound fracture increased with age [391]. Brain lacerations were found more often in compound fractures (29%) than in closed fractures (15.5%). In their study compound fractures were associated with a worse outcome and a higher incidence of intracranial lesions and cortical lacerations. They also found that the deeper the depressed bone, the higher the risk of both dural tear and cortical laceration and the worse the prognosis. A depressed fracture increases the risk for posttraumatic seizures. Schutzman and Greenes found intracranial injuries in about 30% of children with a depressed fracture [171]. Besides intracranial haemorrhages, compression of the underlying brain tissue, laceration of the brain parenchyma, and intraparenchymal bone fragments may occur [197, 391].

Depressed fractures may be simulated by intrauterine compression (Figs. 5.15 and 5.16), postural non-synostotic brachycephaly, and bathrocephalie (Fig. 5.40a, b).

5.3.4.6 Growing Fractures of the Calvarium

Introduction

A growing skull fracture is a fracture that enlarges over time due to herniation of intracranial tissue through the defect (Figs. 5.41a–c and 5.42a, b). In 1816 John Howship, a British surgeon, was the first to describe the occurrence of a growing fracture of the calvarium in a 9-month-old child, who was '*playing near a flight of stairs, fell down, and was taken up at the bottom in a state of insensibility. There was no wound upon the head; but on examination, there was a broad line parallel with the coronal suture, where the right parietal bone was depressed for the length of three inches, and the breadth of one inch. At this part from the form of the depression, it was supposed that the head had struck against the edge of the stairs*' [394]. He referred to this condition in a case report (case 10) as '*partial absorption of the parietal bone resorption, arising from a blow on the head*'. Following his first description other terms that were used for the same condition are traumatic cephalhydrocele, traumatic meningocele, cephalhydrocele, leptomeningeal cyst (because of the frequently present relation with a cyst-like mass filled with cerebrospinal fluid), meningocele spuria, fibrosing osteitis, cerebrocranial erosion, diastatic fracture, cranial-burst fracture, and finally growing skull fracture [383, 395, 396].

Most calvarium fractures that are sustained during childhood heal without any complications. Most cases concerning growing calvarium fractures are reported in children under the age of 3 years. About 50% of all cases are found in children under the age of 1 year [397]. The literature reports an incidence that ranges from 0.05 to 1.6% of all calvarium fractures. A growing fracture is hardly ever seen

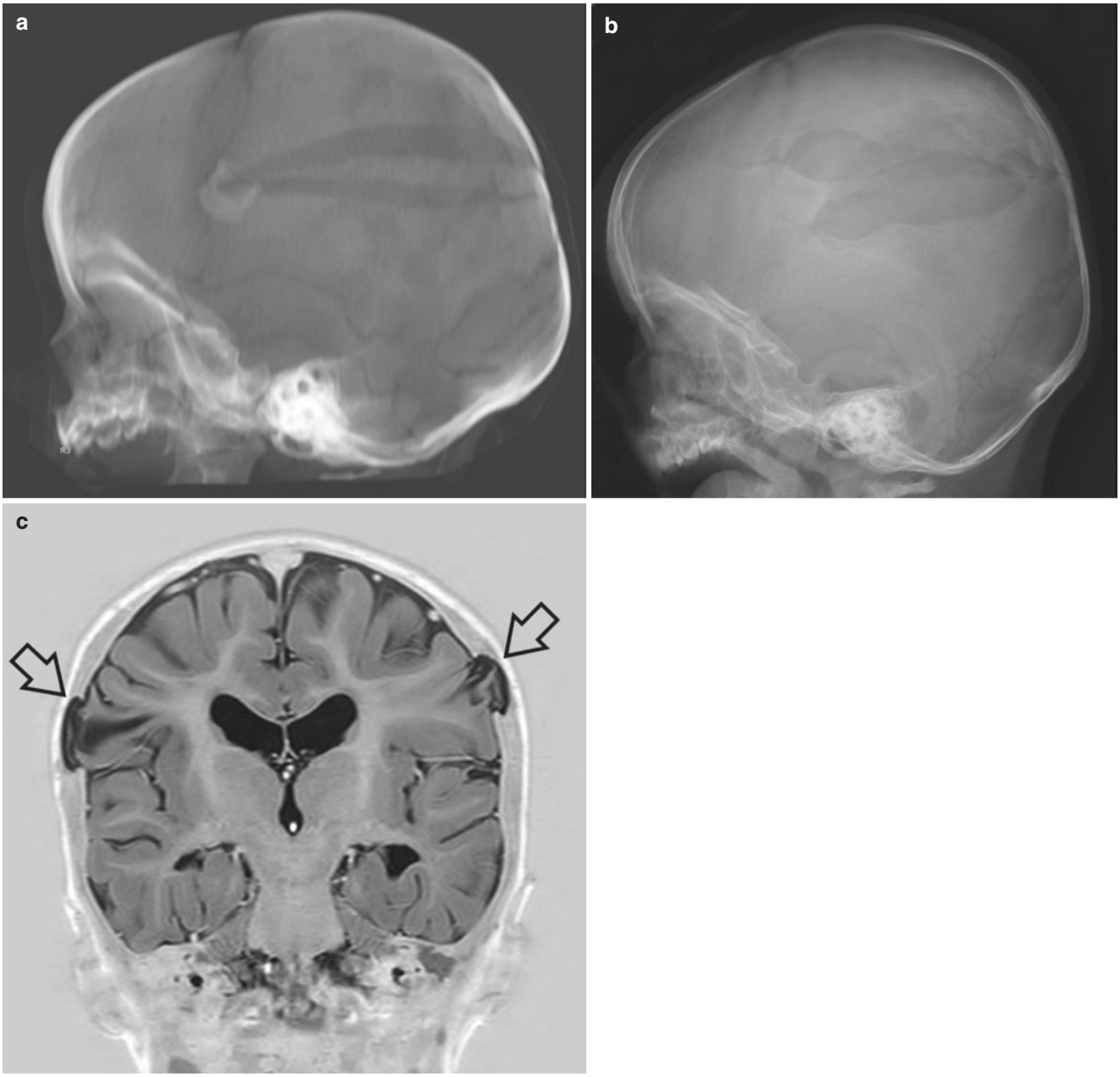


Fig. 5.41 An infant who fell from the hands of the caretaker. (**a**) radiograph shows bilateral diastatic parietal fractures. (**b**) At the age of 6 months the fractures have increased in width due to (**c**) herniation of intracranial structures into the fractures (arrows)

in children above the age of 8 years [397–402]. There may be a considerable delay in time between the occurrence of the clinical findings and the moment the diagnosis is made [403, 404]. Sometimes the diagnosis is not made until the patient is above the age of 60 years [405, 406]. Consequently, in certain cases it is impossible to relate to the initial trauma. After reaching their maximal size, growing fractures tend to remain stable for the rest of one's life [400].

Most growing fractures are found in the calvarium, in particular in the parietal bone (50%) [407]. Sometimes growing fractures can be found at the base of the skull or in the roof of the orbit. It is very rare for a growing fracture to be present in the posttraumatic diastasis of a suture [408]. Generally, it concerns linear fractures. Normally, a depressed fracture will not develop into a growing fracture [409]. However, a linear fracture that originates from a depressed fracture can develop into a growing fracture [410]. In a fracture with a diastasis of over 4 mm, there is an increased risk for the development of a growing fracture (Fig. 5.43a, b) [411, 412].

Clinical symptoms develop gradually, unless in the acute phase there is a cranial-burst fracture with acute herniation

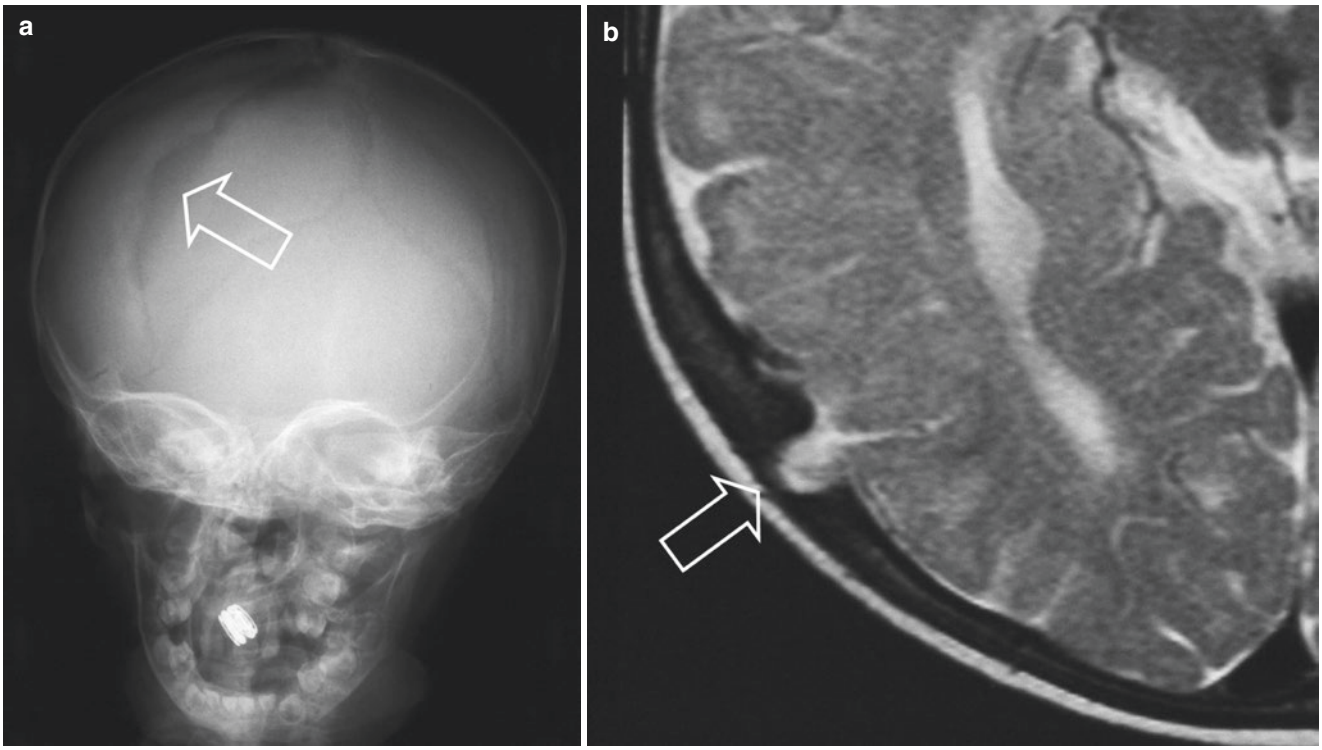


Fig. 5.42 (**a**) Girl with a growing skull fracture. The skull view shows a diastatic fracture on the right dorsal parietal side. (**b**) Pre-operative MRI shows a dural defect and prolapsed meninges and brain tissue in the diastatic fracture (arrow)

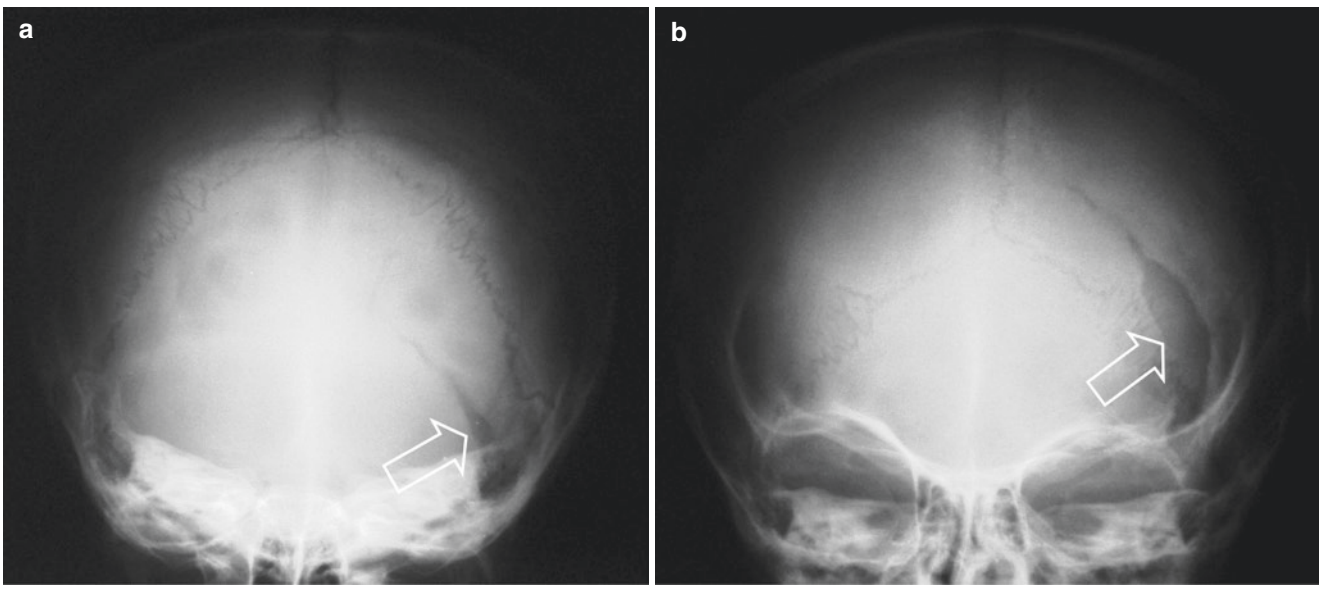


Fig. 5.43 (**a**) Infant who was presented at the emergency department after a fall on the head. The skull view showed a diastatic fracture on the left side (open arrow). (**b**) Follow-up view after 3 months, clearly shows the growing skull fracture (open arrow)

of intracranial tissue through the fracture towards subgaleal, or if there is a dura defect with a high risk for herniation and development of a growing fracture. It seems that in acute situations MRI imaging is the most reliable manner to show dura defects [383]. The MRI images enable instant evaluation of damage to the dura, and an immediate referral of the patient for surgical correction, so as to prevent additional damage [396].

Children may present with gradual increase in the subgaleal mass, headache, and signs of neurological pathology. Pezzotta et al. did a retrospective study of the literature on 132 children with a growing fracture [407]. They established

that normally the initial clinical symptoms were the development of seizures (40%), focal neurological deficits (43%), unconsciousness (38%), or combinations of the aforementioned. Asymptomatic presentation was more common in frontal-parietal and frontal-parietal-occipital locations. In 50% of children, the delay between the occurrence of the fracture and appearance of the first symptoms ranged from a day to a year. There seems to be a proportional relation between the severity of the neurological deficits and the size and 'growing time' of the defect [399]. The externally visible lesions of a growth fracture are a cyst-like non-firm swelling, visible sometime after the initial trauma, with an underlying palpable bone defect [408].

The diagnosis is based on the clinical presentation and radiological images. In order to avoid neurological complications, immediate recognition and early treatment are required [413]. Treatment is always surgical and directed at reducing the herniated brain tissue and repair of the damage inflicted to skull and dura. It may be necessary to place a shunt to alleviate the cyst and to treat local dilatation of the ventricles [408].

The severity of the underlying trauma is a risk factor for the child. A linear fracture combined with haemorrhagic contusion foci in the underlying brain tissue suggests a trauma severe enough to cause dura lacerations. The presence and severity of the associated damage determine the risk of complications.

In a growing fracture there is nearly always underlying brain damage. At the place of the fracture scar tissue may develop in brain tissue and meninges. Cyst-like changes at the place of the fracture may be the result of encephalomalacia. Posttraumatic aneurysms and subdural haematomas have also been reported in relation to growing fractures [413, 414]. In all children they examined, Muhonen et al. found damage to the cortex at the location of the fracture, although, without signs of increased intracranial pressure [404]. Although in most children signs of damage to the underlying brain tissue can be found, this finding is not a prerequisite for developing a growing fracture [397].

A growing fracture of the base of the skull may cause eye proptosis or cerebrospinal fluid leakage from the nose or the ear.

Cause of Growing Fractures

The exact cause of growing fractures is still under discussion. It appears that calvarium fractures are not inclined to show diastasis when the underlying dura is intact. The occurrence of a growing fracture seems to depend on many factors: head trauma resulting in a large fracture, the presence of a dura laceration, damage to the parenchyma at the location of the skull fracture and the dura laceration, and damage sustained at the time of maximal brain growth [396, 408].

Muhonen et al. are of the opinion that herniation of brain tissue/leptomeningeal cyst, without indications for increased intracranial pressure, points to physiological growth and to pulsations of the cerebrospinal fluid as the cause of diastasis/growth of the fracture [404]. The force of the pulsations widens the skull fracture. The pulsations also push intracranial tissue into the fracture line. This makes it impossible for the osteoblasts to migrate to the fracture; hence, there is no new bone formation and consequently no healing. Finally, there is resorption of the adjacent bone as a result of the continuous pressure of the tissue herniation through the defect in the bone [408].

It seems that insufficiently closed dura lacerations during craniotomy can also lead to growing fractures of the skull. These findings support the idea that traumatic damage to the dura is the most important risk factor in the development of a growing fracture [408, 415]. In children, the dura is firmly attached to the skull preventing wide fracture lines. Therefore, when a diastatic fracture (≥4 mm) is present in a young child there is an increased risk for the development of a growing fracture.

Manner of Growing Fractures

Growing fractures usually occur after serious head trauma. This trauma can occur before, during, and after birth. The circumstances can be accidental or non-accidental, but the occurrence has also been described due to medical procedures.

Trauma Before Birth

There are case reports on the origin of growing fractures following the occurrence of calvarium fractures/damage to the calvarium in utero.

Moss et al. described the case of a full-term male infant with a right parietal caput succedaneum and cephalhaematoma at birth [416]. The boy also had bilateral parietal fractures and a one-sided leptomeningeal cyst at birth. At the age of 2 weeks, a linear lesion measuring 3 × 7 cm in the parietal bone was found. The initial linear fractures probably were caused by a blunt force trauma, a blow to the mother's abdomen 2–3 weeks before birth.

Gallo et al. described the occurrence of a growing fracture due to sharp penetrating trauma [209]. A 20-year-old pregnant woman was stabbed in the lower abdomen at the 30th week of gestation. The stabbing did cause a bony interruption and a dural tear with parenchymal injury in the foetus, resulting in a slowly developing growing fracture of the calvarium. When the child was born, a right temporal swelling was seen, which was interpreted as a subcutaneous haemangioma. At the age of 2½ years, a pulsating bulge in the right temporal region was noticed and imaging showed a typical growing skull fracture.

Trauma During Birth

The occurrence of a growing fracture of one of the calvarium bones has been described in case of a difficult delivery with vacuum extraction [234, 235, 417, 418].

Trauma After Birth

Growing fractures of the calvarium most commonly occur an accidental circumstance, e.g. due to falling or a traffic acci-

dent. Occasionally after a non-accidental trauma (child abuse). The occurrence of a growing fracture has also been reported as a complication after neurosurgery for corrective cranial vault reshaping [415].

Hobbs evaluated 89 children under the age of 2 years with skull fractures. In 60 cases he found accidental circumstances [256]. In the remaining children (n = 29), the fractures were due to non-accidental circumstances (child abuse). In the group of children with accidental trauma, he did not find but one growing fracture, whereas six of the abused children did have a growing fracture (Table 5.5). Hobbs's results seem to contradict the results of the study of Donahue et al., they evaluated 13 children with a growing fracture, ranging in age from 1 to 17 months with an average age of 5.7 months [383]. Seven children had suffered serious injuries in traffic accidents, and in five children the fractures were due to a non-accidental trauma (child abuse). In one child the physicians were not clear about the cause. The children in the study of Donahue et al. were all seen when acute [383]. They showed a conspicuous haematoma of the scalp and a Glasgow Coma Score of 10 points or less, indicating recent serious head trauma.

When the data of Hobbs and Donahue et al. are combined, they show that in young children head trauma with herniation of intracranial tissue (either in the acute phase or at a later stage) is the result of severe head trauma [256, 383]. It must be possible to objectify the circumstances of the trauma in order to accept accidental circumstances. If not possible, one must consider non-accidental circumstances in this group of young children.

5.3.4.7 Normal Variants Simulating Fractures

Sutures

Sutures and synchondroses are fibrous and cartilaginous connections between the separate bones that form the skull. In young children, the sutures are flexible allowing them to adapt to increased intracranial pressure (Fig. 5.44a–e).

The largest sutures are well known (the big five): metopic, coronal, sagittal, squamous, and lambdoid sutures and can be seen both on conventional radiographs as 3D CT images

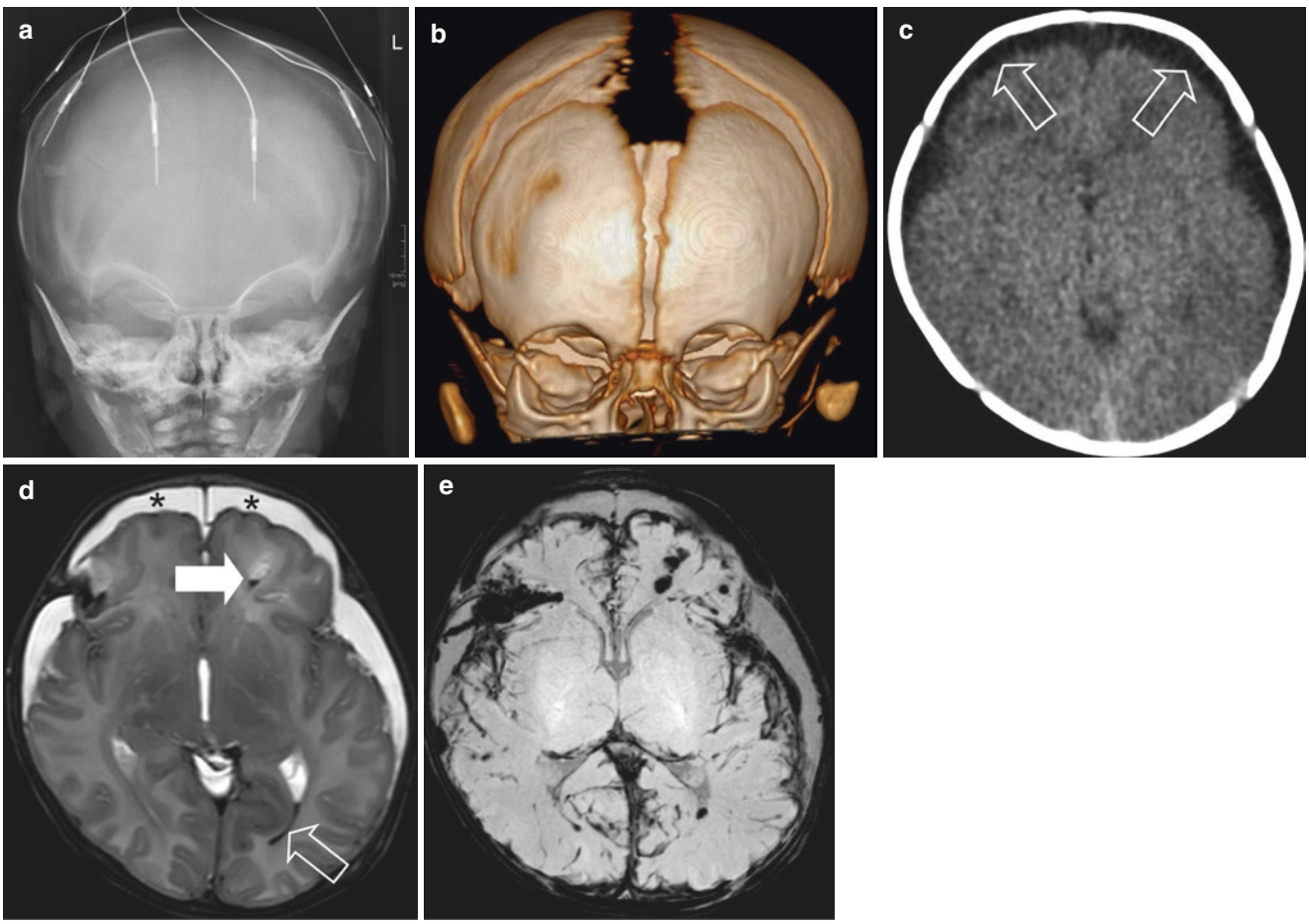


Fig. 5.44 Infant who was presented at an emergency department in a comatose state. (**a**) Skull radiograph shows widening of the sutures. (**b**) 3D-CT showed no skull fractures. (**c**) CT shows bilateral subdural hematomas (arrows). (**d**) T2-Weighted MRI shows bilateral subdural hematomas (asterisk), a laceration in the frontal lobe (arrow), and blood in the lateral ventricle (open arrow). (**e**) Susceptibility-weighted MRI shows haemosiderin depositions in the subdural haematomas, the laceration, and the lateral ventricle

(Fig. 5.45a–e) [419, 420]. But there are many other smaller sutures to complicate interpretation (Fig. 5.46a, b). And on top of that many normal variants (e.g. accessory sutures) make interpretation even more difficult [421–425].

Accessory Sutures

Most accessory sutures are found in the occipital and parietal bones. These can be considered as membranous remnants between separate ossification centres or focal persistence of the stripe-like foetal ossification pattern (Fig. 5.47a–c).

The Occipital Bone

The occipital bone develops from six primary ossification centres (Fig. 5.48a, b) and the interparietal bone (Inca bone) can even be subdivided in additional secondary ossification centres (Fig. 5.49a, b) [426, 427]. Therefore, the occipital bone is particularly prone to the formation of accessory sutures and bones. Well-known mimickers of fractures are the mendosal suture, the superior and inferior midline (or median) fissures, the innominate suture, and Kerckring's ossicle:

The *mendosal suture* is the most prominent and most frequent accessory suture (Figs. 5.48, 5.49, 5.50, and 5.51). It is a remnant of the suture between the supraoccipital and interparietal bones and always seems to originate from the mastoid fontanel (Fig. 5.48).

The *superior midline fissure* is a remnant of the suture between the ossification centres of the interparietal bones. It is seen in up to 21% of newborns and closes by the age of 5 months (Figs. 5.49 and 5.55) [428].

The *innominate suture* runs between the exoccipital and supraoccipital bones (Fig. 5.48) These sutures usually do not cause confusion on 3D-CT, but are difficult to discriminate on conventional radiographs, simulating fractures (Fig. 5.51). They gradually fuse with age and on conventional radiographs this fusion is complete by 4 years of age, except for the mendosal sutures that persist until 6 years of age [429].

Two remnants of development of the supraoccipital bone may persist after birth, Kerckring's ossicle and the midline occipital fissure: *Kerckring's ossicle* is an ossification centre of the supraoccipital bone, in the midline posteriorly of the foramen magnum (Figs. 5.48 and 5.52). It appears around week 16 of gestation and usually fuses with the rest of the occipital bone antenatally but sometimes persists after birth [424, 430].

The inferior midline occipital fissure (or median occipital fissure) runs from the posterior rim of the foramen magnum in posterior direction and should be less than 2 cm in length (Fig. 5.49) [424, 430]. A longer fissure would not be consistent with normal embryogenesis and therefore represents a fracture (Fig. 5.53) [424]. All fissures around the foramen magnum

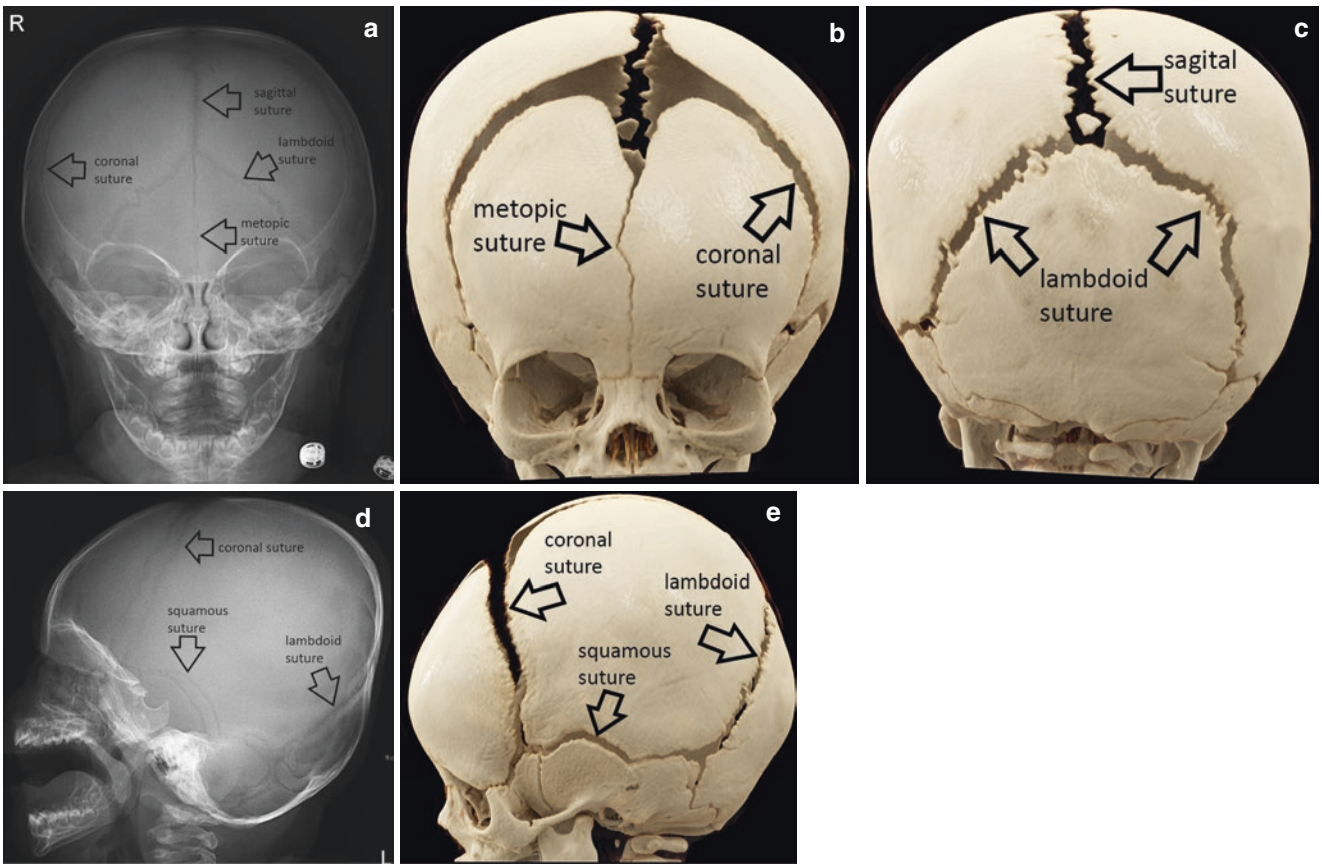


Fig. 5.45 'The big five' sutures on radiographs (**a** and **d**) and 3D-CT (**b**, **c**, **e**)

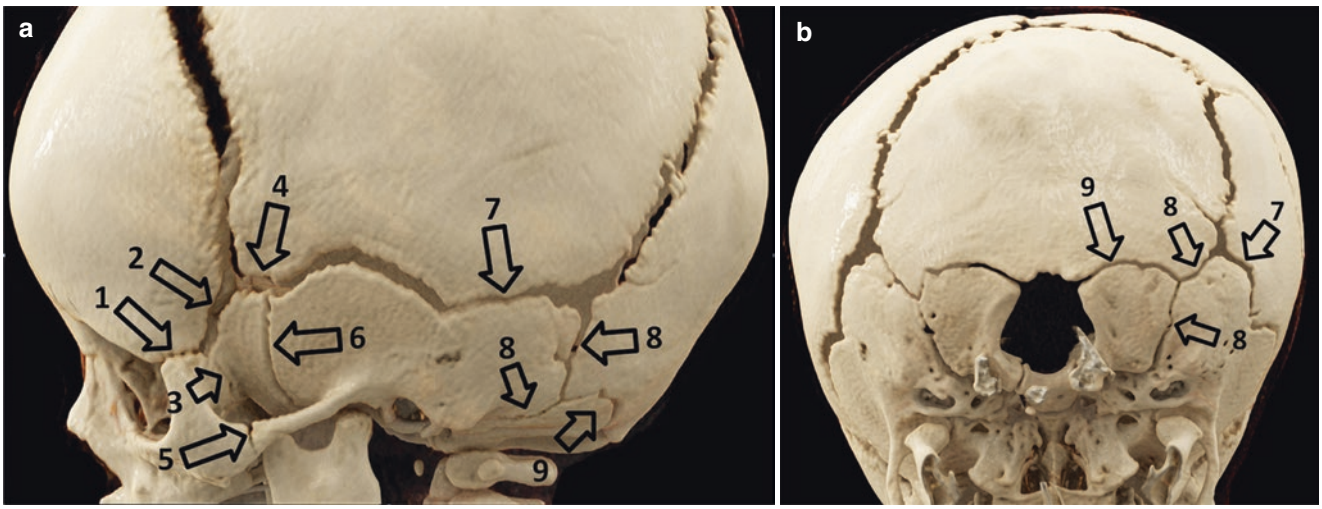


Fig. 5.46 The minor sutures on 3D-CT lateral (**a**) and caudal view (**b**). See also Fig. 5.67. 1 = Frontozygomatic. 2 = Sphenofrontal. 3 = Sphenozygomatic. 4 = Sphenoparietal. 5 = Temporozygomatic. 6 = Sphenosquamosal. 7 = Parietomastoid. 8 = Occipitomastoid. 9 = Innominate (posterior intraoccipital)

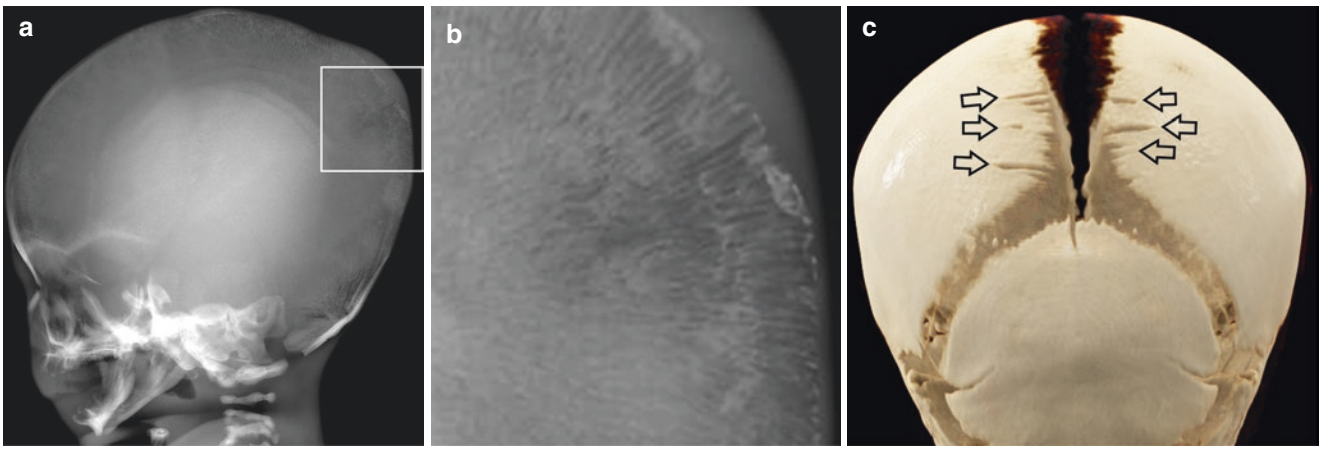


Fig. 5.47 (**a**) Lateral radiograph of the skull of a 20-week-old foetus with mammography technique. Insert is depicted in (**b**) showing the stripe-like pattern of ossification. (**c**) Persistence of some of these membranous stripes can result in accessory sutures as shown on a 3D-CT of a term new-born

close before 4 years of age and a persistent hypoattenuating line of any length that is seen extending from the foramen magnum beyond 4 years of age indicates a fracture [423].

The Parietal Bone

The parietal bone shows fewer variants than the occipital bone due to its less complicated embryogenesis: the parietal bone ossifies from two centres whereas the occipital bone has at least six centres [426, 431]. Two variants will be discussed:

Obelion fissure. The obelion marks the point along the sagittal border of the parietal bone where a parietal notch may appear, at approximately one-third of the sagittal suture from the posterior fontanel. This may eventually result in a (obelion) fissure, a small parietal fontanel or parietal foramina. The obelion fissure extends outwards from the sagittal suture for 1 cm or more and is present at birth in approximately 25% of the children to disappear soon after birth [432, 433].

Occasionally it is seen in routine skull radiographs where it may simulate a fracture (Fig. 5.54a, b). It is seen to be a good advantage on CT (Fig. 5.55).

Accessory parietal suture. This is a remnant of the space between the two foetal ossification centres of the parietal bone, usually one above the other resulting in a horizontally orientated accessory suture [431, 434]. They are often bilateral and symmetric [423]. Usually, they extend from the lambdoid suture into the parietal bone for only several centi-

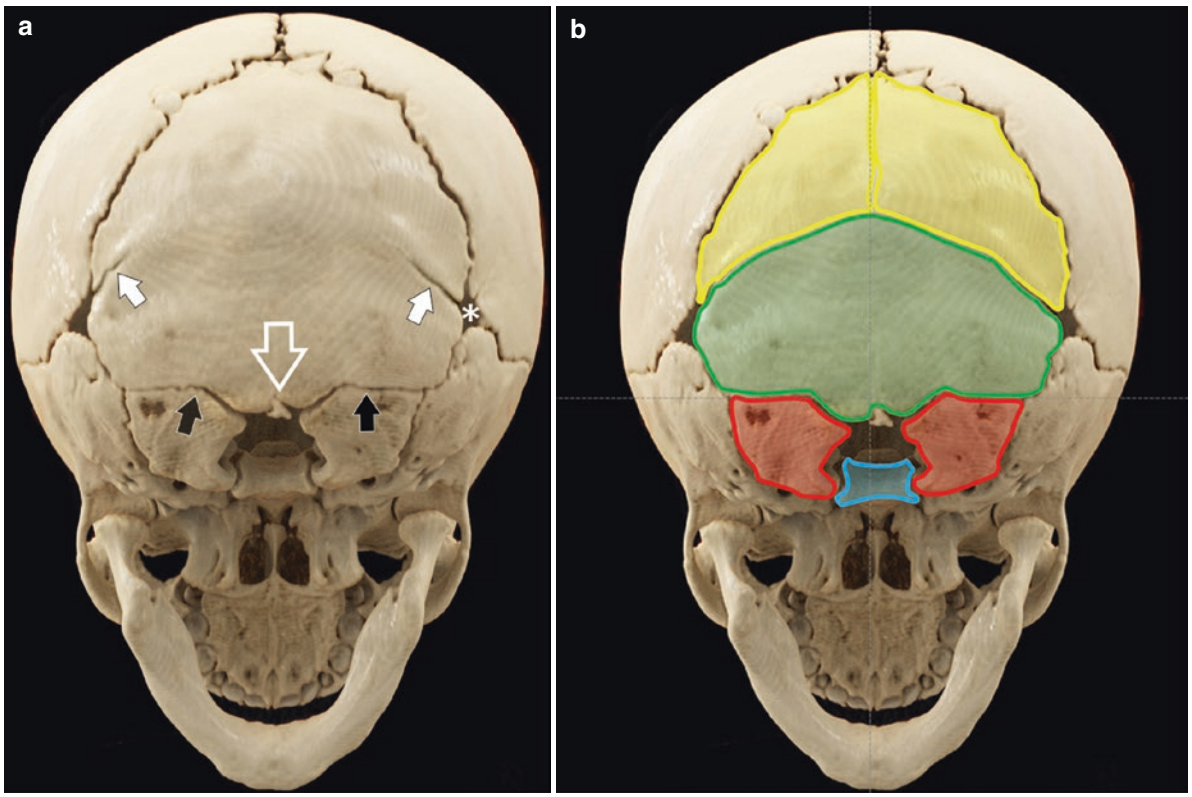


Fig. 5.48 (**a**) 3D-CT of the occipital bone in a 6-month-old infant showing the major ossification centres of the occipital bone: single basoccipital (blue), two exoccipital (red), single supraoccipital (green), and two ossification centres of the interparietal bone (= os inca, yellow). (**b**) At the posterior side of the foramen magnum Kerckring's ossicle is present (arrow). Also, well visualized are the mastoid fontanel (asterisk), mendosal suture (small white arrows), and the innominate suture (small black arrows)

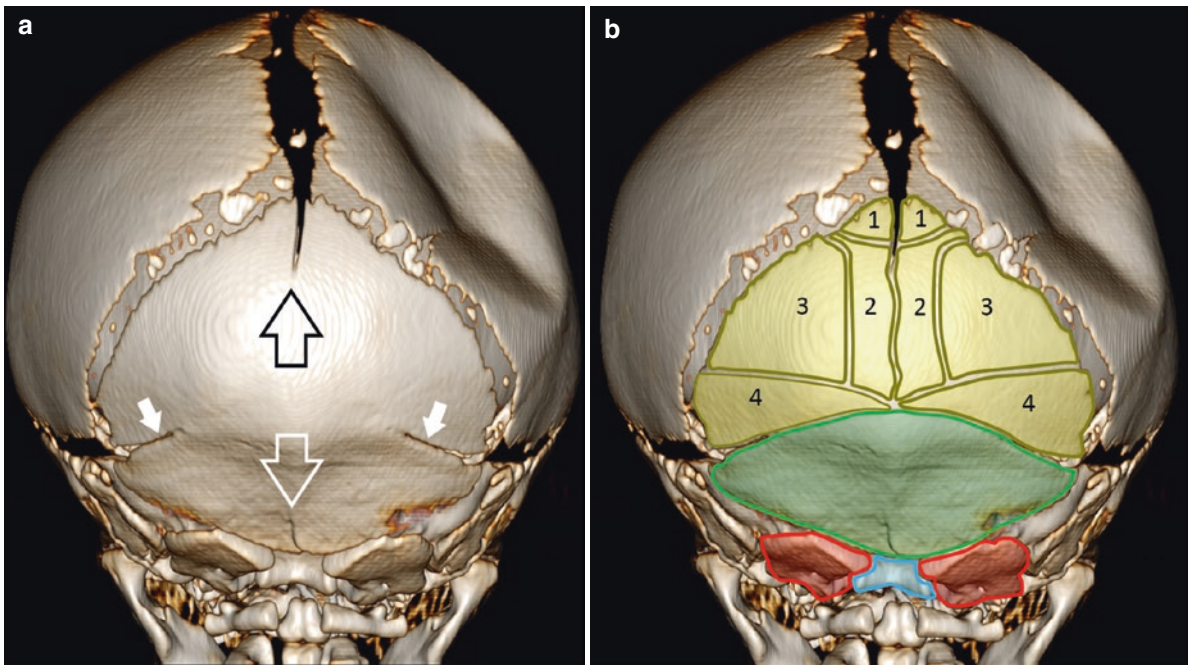


Fig. 5.49 (**a**) 3D-CT of a neonate with a birth-related parietal impression fracture showing the mendosal sutures (small white arrows), the superior median occipital fissure (black arrow), and the inferior median occipital fissure (white arrow). (**b**) Detailed ossification centres of the interparietal bone (yellow) are schematically drawn: 1 = preinterparietal. 2 = medial secondary interparietal. 3 = lateral secondary interparietal. 4 = primary interparietal

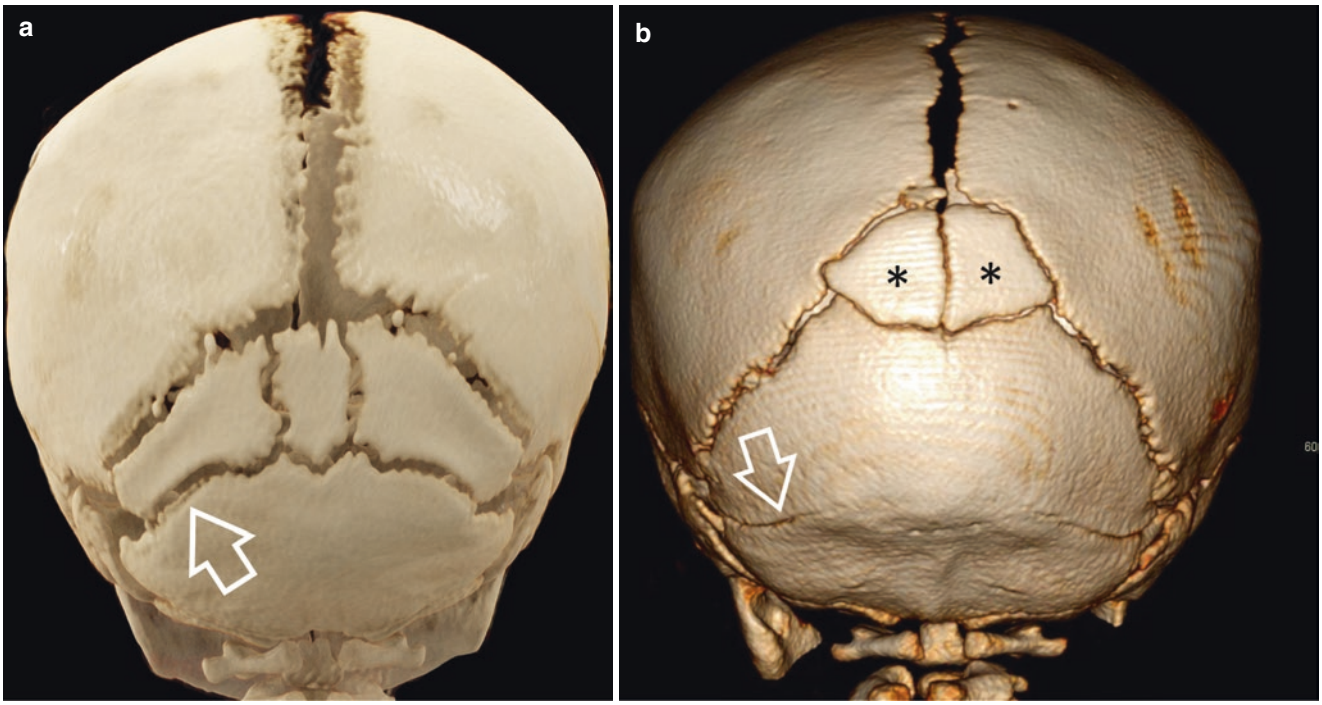


Fig. 5.50 3D-CT's of 2 patients with accessory occipital sutures. Arrows indicate the mendosal suture. (**a**) Infant whose mother while carrying him in a baby wrap carrier resulted in a parietal fracture (not shown). The numerous accessory sutures in the occipital bone should not be misinterpreted as fractures. Knowledge of the embryology of the occipital ossification centres (Fig. 5.49) makes it clear that this is a multi-segmented os inca (interparietal bone) with separate lateral secondary interparietal bones and fused medial secondary interparietal bones. (**b**) Another infant with separate large pre-interparietal bones (asterisk)

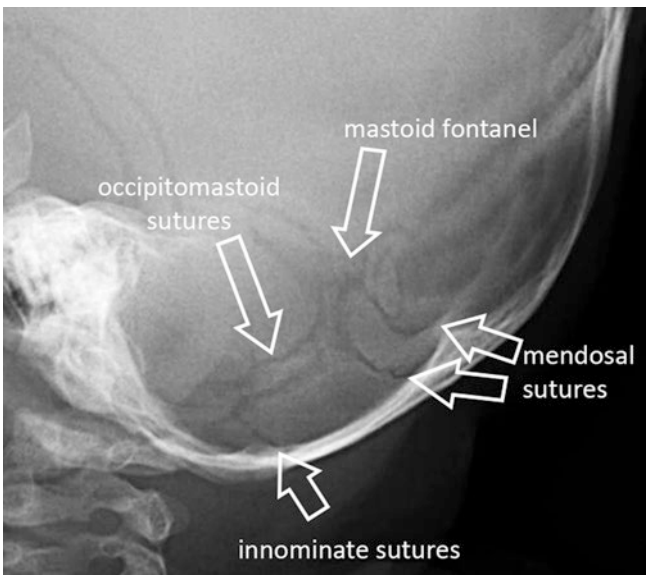


Fig. 5.51 Lateral radiograph of the skull of a normal infant showing occipital sutures

Fig. 5.52 CT MIP image of Kerckring's ossicle (indicated by arrow)

metres (Fig. 5.56) but sometimes extend all the way to the coronal suture (Fig. 5.57a, b).

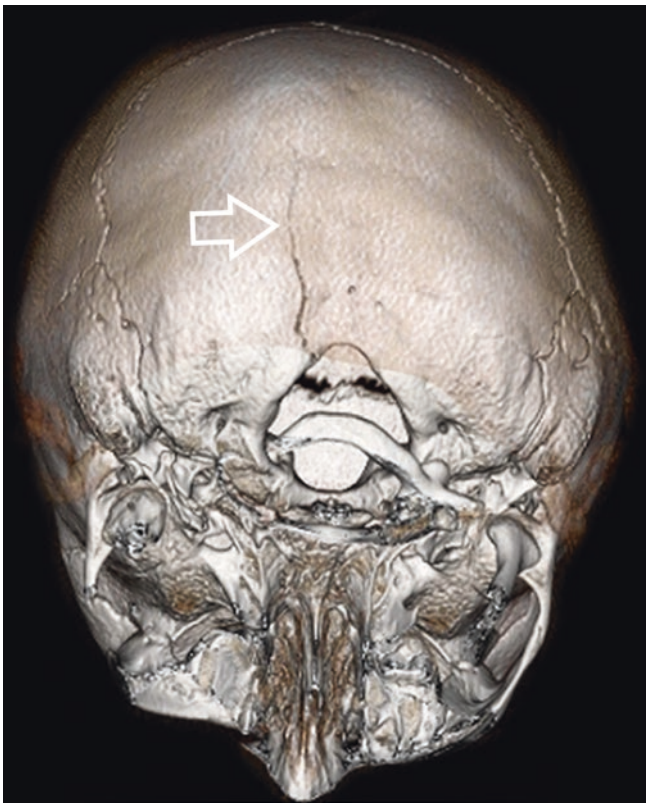


Fig. 5.53 3D-CT, posterior view. Fracture of the occipital bone extending into the foramen magnum (arrow). It exceeds a length of 2 cm and is therefore not the inferior midline occipital fissure

Occasionally, the ossification centres lie next to each other resulting in a vertically oriented accessory suture [435]. The combination of vertical and horizontal accessory sutures may result in complex sutural patterns [436]. These are very rare (Fig. 5.58a–d).

Fractures Versus Accessory Sutures

Differentiating fractures from sutures and artefacts remains a diagnostic challenge in childhood. Solid knowledge of the anatomy and variations of sutures is as important as common radiological sense: some basic guidelines to distinguish fractures from accessory sutures are given in Table 5.10 [421, 423].

Although 3D-CT is superior to discriminate accessory sutures from fractures, it is important to realize that movements even during a fast CT scan may create motion artefacts that simulate fractures (Fig. 5.59).

5.3.5 Calvarium Fractures and Intracranial Injuries

Calvarium fractures and intracranial injuries are only correlated to a limited degree. Calvarium fractures may be present without any intracranial injury, but can be associated with several intracranial injuries, like epidural or subdural bleeding or contusion of the brain [437]. On the other hand, there may be intracranial injury without a skull fracture. This applies to accidental as well as to non-accidental circumstances [26, 170, 184, 438]. In Sect. 5.6, the relation between

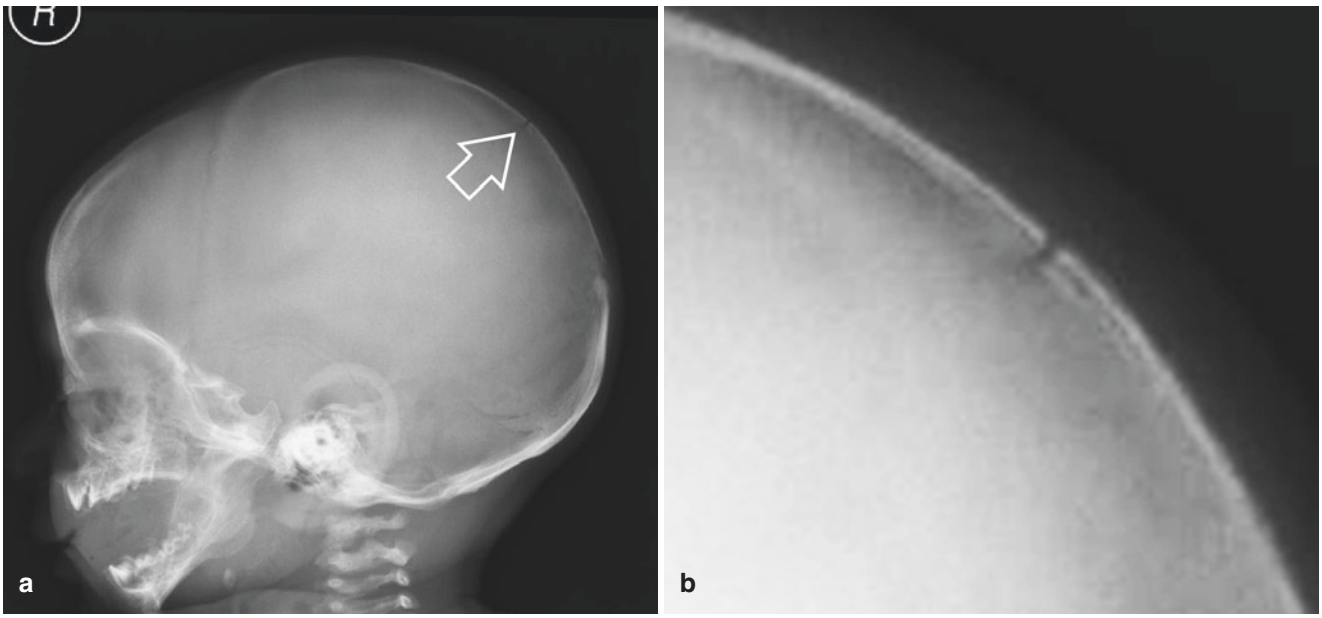


Fig. 5.54 Newborn infant. (**a**) Incidental finding of obelion fissure on lateral skull radiograph. (**b**) Detail shows the fissure to a better advantage, simulating a fracture

Fig. 5.55 Neonate with multiple bruises, a torn frenulum and on the skeletal survey two posterior rib fractures and several metaphyseal corner fractures. Obelion fissure (black arrow) ending in a small parietal foramen. The contralateral side also has a subtle obelion fissure. Also, note the simple linear fracture (white arrow) and superior median occipital fissure (open arrow)

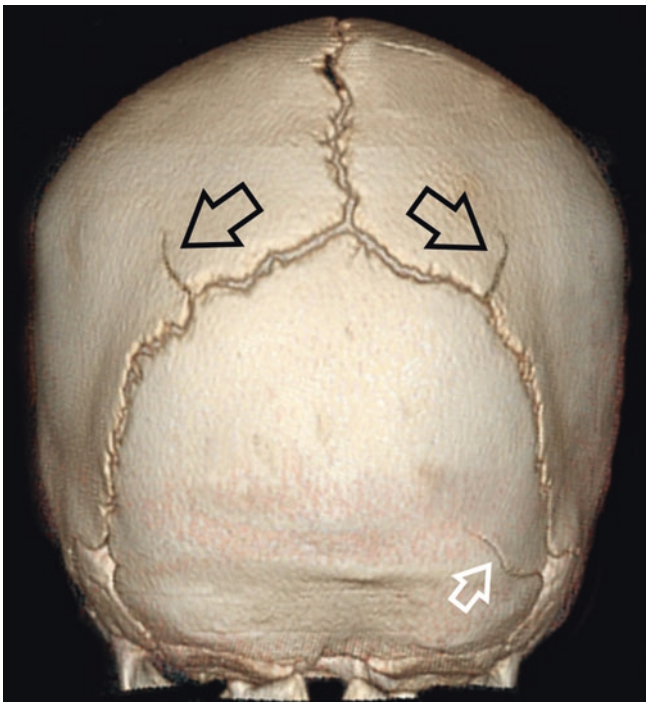


Fig. 5.56 Two accessory parietal sutures (black arrows) in a 3-month-old boy who had a workup for non-accidental injury because of unexplained bruises on the buttocks. Also, note the mendosal suture on the right side (white arrow). These should not be mistaken for fractures

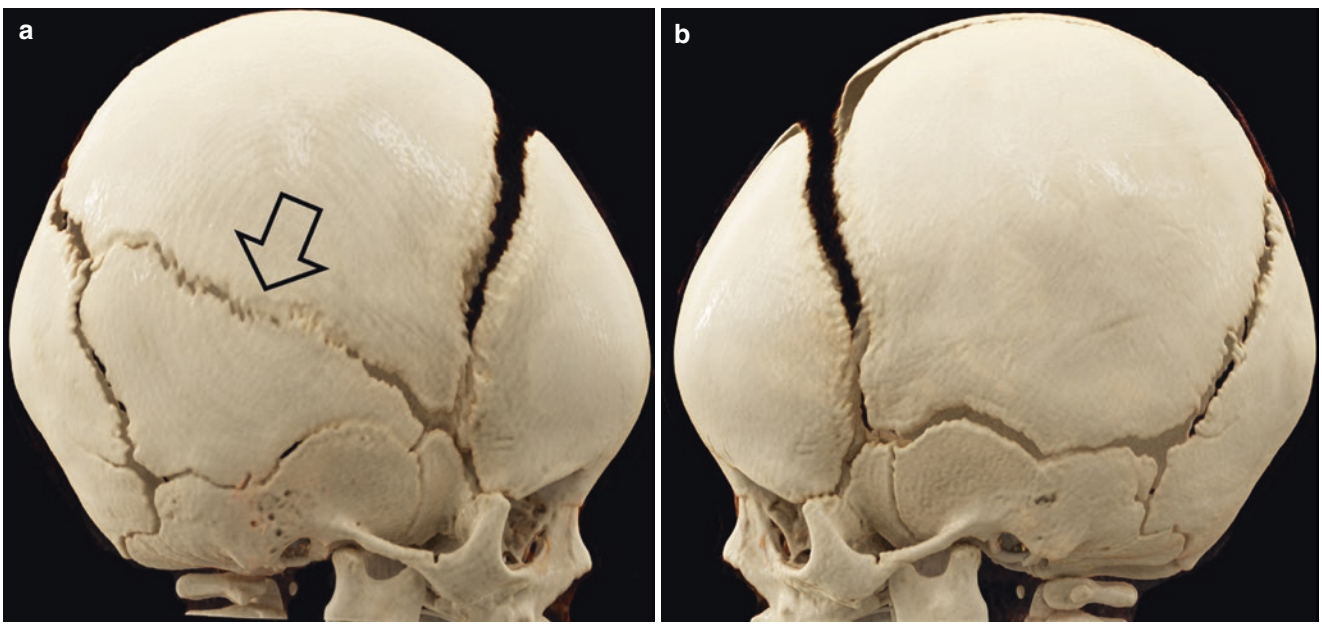


Fig. 5.57 (**a**) Complete intraparietal suture on the right side (arrow). (**b**) Normal left side for comparison. This was an incidental finding in an infant with subdural hematomas, hypoxic-ischemic brain lesions, and retinal haemorrhages without a history of trauma. She expired after the cessation of treatment because of poor neurological prognosis. The judicial autopsy showed no additional traumatic lesions

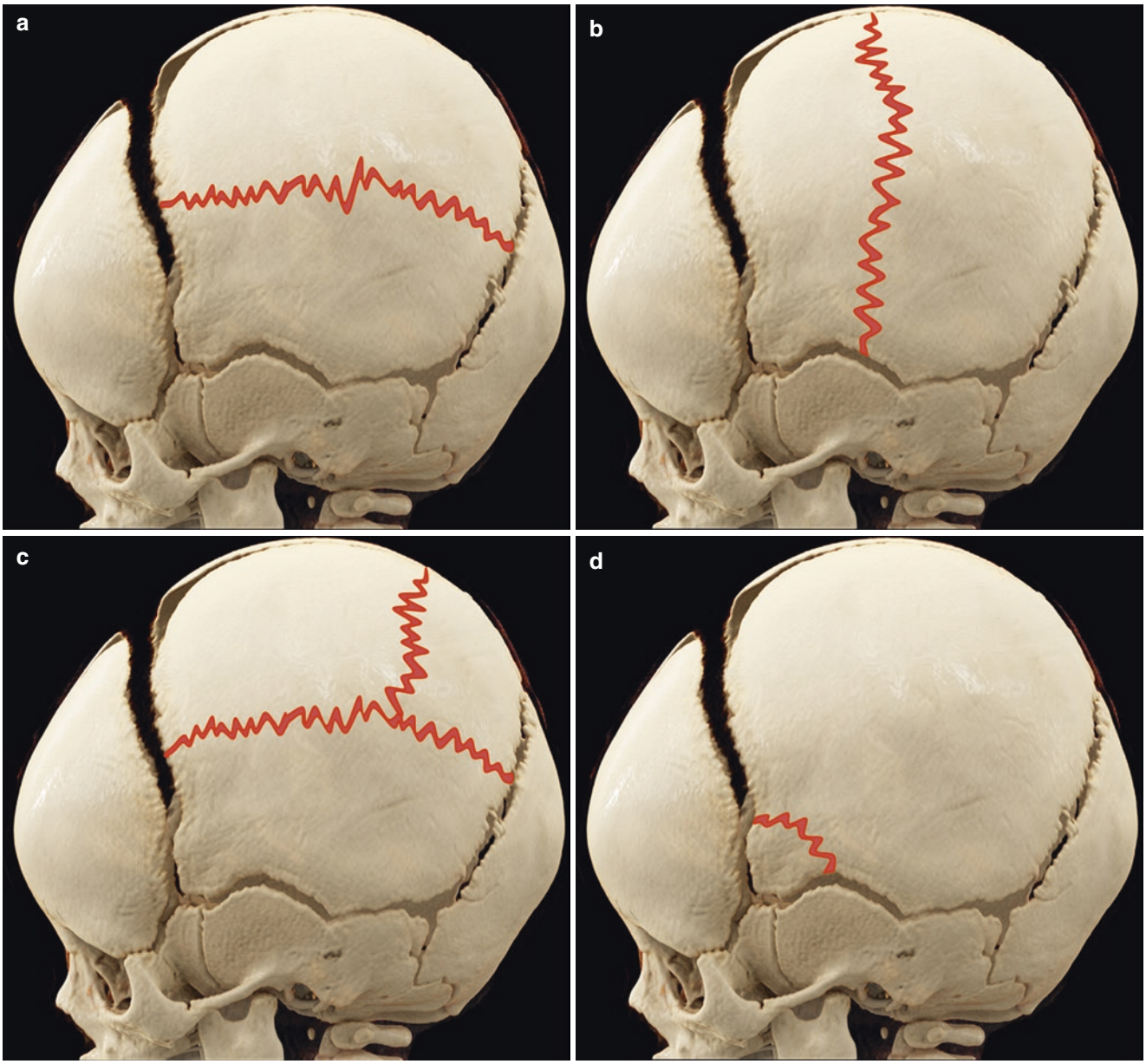


Fig. 5.58 Schematic representation of vertical and horizontal accessory parietal sutures

Table 5.10 Distinguishing features of fractures and accessory sutures

Fracture	Accessory suture
Straight course	Zigzag/interdigitating
Nonsclerotic borders	Sclerotic borders
May bifurcate	Do not bifurcate
Cross sutures	Merge with sutures
Increase in diameter as they approach a suture	Uniform in diameter
Cause diastasis of sutures	No diastasis of sutures
Unilateral	Bilateral
Asymmetrical if bilateral	Fairly symmetrical
Focal soft tissue swelling	No soft tissue swelling
Focal intracranial haemorrhage	No haemorrhage

calvarium fractures and intracranial injuries will be dealt with more extensively.

5.3.6 Dating of Calvarium Fractures

In living children, an indication of the age of a calvarium fracture can be given by analyzing certain findings from physical examination (abnormalities of the hairy scalp/soft tissue swelling) and characteristics of the fracture, which are visible during imaging (conventional radiology, CT, MRI) [249, 372].

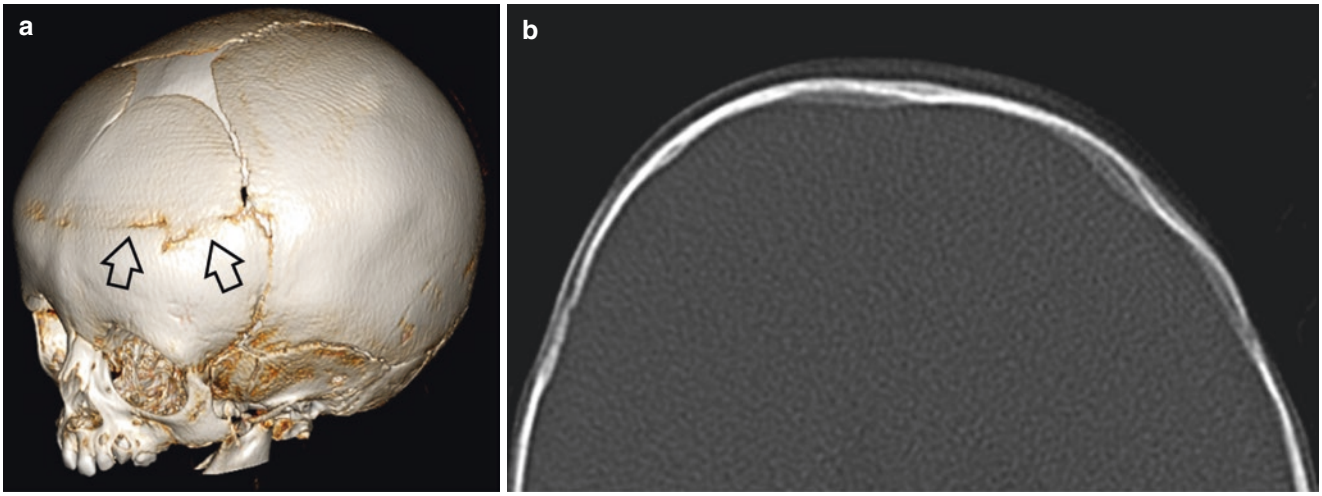


Fig. 5.59 The mother of this infant suspected that her partner abused the child and a workup for non-accidental injury was performed. (**a**) The 3D-CT was interpreted as a possible left frontal fracture. (**b**) The native slices demonstrated motion artefacts (e.g. blurring of the skin and ghosting of calvarium) that were caused by an ultrashort jerking movement of the head during spiral CT scanning. No other abnormalities were found on CT and the skeletal survey was normal

5.3.6.1 Soft Tissue Swelling

As previously stated, calvarium fractures are caused either by static loading (squeezing or compression) or by dynamic impact loading (blunt force trauma). A soft tissue swelling may occur at the location of the contact or of a calvarium fracture. The swelling may be caused by the direct effect of the impact trauma, which resulted in damage to the scalp and underlying soft tissue, and/or by bleeding at the site of the fracture.

The soft tissue swelling can be seen immediately, but can also develop gradually over a period of a few hours to days. In case of a gradual development, the swelling can be noticed after a while because, e.g. a soft or spongy swelling is felt while touching the head during daily activities, like caring for the child. The swelling generally begins to decrease in size after 7–10 days.

The clinical detection of swelling of the hairy scalp can be helpful in determining when a skull fracture has occurred, but both the presence and the absence of soft tissue swelling during a physical examination can lead to an incorrect interpretation. The late detection of soft tissue swelling, for example regularly occurs in single linear fractures. Soft tissue swelling may be visible on the CT scan without being clinically visible (Fig. 5.60a–c). However, it is possible that in children with fatal head injuries no evidence of damage to the scalp is found during physical examination or during the CT scan, while abnormalities were found in and under the scalp during autopsy on the outside of the skull [439]. Kleinman and Silvera (2015), however, are of the opinion that, if no soft tissue swelling is visible over the fracture on a CT scan, an acute fracture is unlikely and that the fracture was sustained at least a few days before the CT scan was made [372].

The foregoing data show that the development of soft tissue swelling only enables a rough estimation of the moment on which a calvarium fracture was sustained, varying from some days to longer than a week before the fracture is diagnosed by imaging. And if a subgaleal haemorrhage occurs, it may take even weeks before the swelling becomes clinically visible and gives rise to imaging (Sect. 5.2.3).

5.3.6.2 Radiological Characteristics

The dating of calvarium fractures by using the findings of a single radiological examination is unreliable. Repeated imaging combined with clinical findings, like soft tissue swelling, may provide a more reliable indication of the moment at which the fracture was sustained (Fig. 5.60c).

Calvarium fractures show a different healing pattern than other fractures. No callus is formed during the healing process. A fresh fracture will have sharp edges that fade during the healing process. Calvarium fractures do not heal as fast as other fractures. In young children, the healing process may be faster than in older children.

In the case of birth-related calvarium fractures, the first radiological signs indicating recovery (fading of the edges of the fracture) are often not visible until 4–6 weeks after birth [440]. A simple linear fracture, sustained during birth, is often no longer distinctly visible after 2 months and has almost always completely disappeared after 6 months [441].

In calvarium fractures that are sustained after birth, the edges of the fracture will be sharply defined in the first 2 weeks after the fracture occurred. After this period, fading of the edges takes place, after which the fracture will eventually close and no longer be visible (Fig. 5.61). This fading can be visible for weeks to months and is therefore not a reliable

indicator of the moment of sustaining [442]. It may even take as long as a year before a fracture is no longer visible on radiographs [184]. A simple fracture may no longer be visible on imaging within 1–2 months, while complex fractures may require many months to fully heal. With complex fractures, some parts can heal faster than others [249, 372]. In other words, the time, in which healing occurs, varies, depending on the type of fracture (simple or complex) and between children [443].

The fracture lines in diastatic fractures grow towards each other, unless a growing fracture occurs as a result of underlying damage to the dura [404, 444, 445].

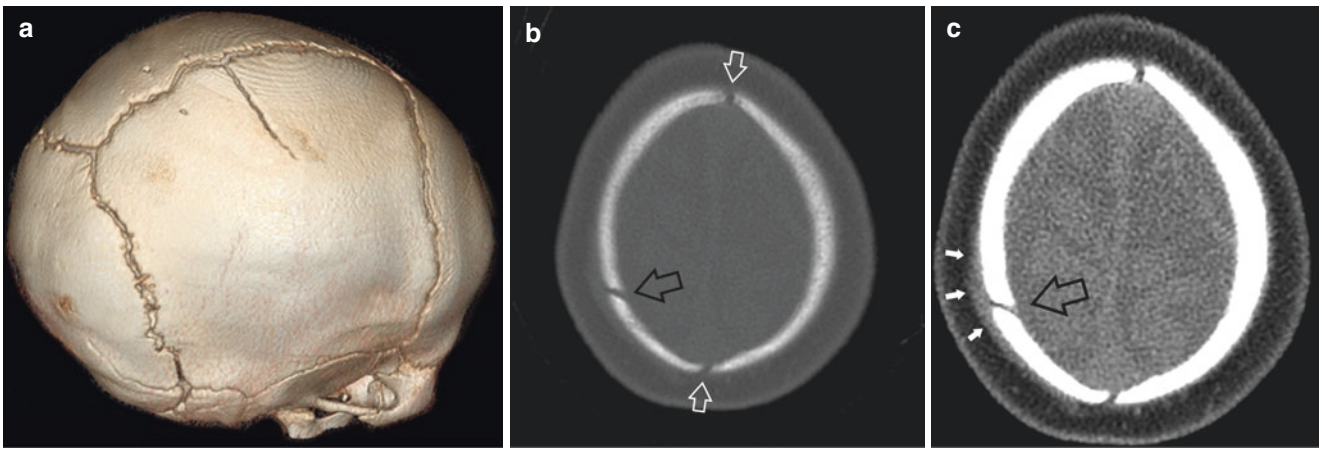


Fig. 5.60 A 3-month-old boy with a bruise on the left buttock of unknown cause. A skeletal survey showed multiple CMLs and suspicion of a skull fracture. (**a**) CT shows a simple linear structure in the right parietal bone on 3D reconstruction. (**b**) This proves to be a fracture and not an accessory suture (black arrow) because of the sharp edges compared to the sagittal suture (white arrows). (**c**) The subperiosteal soft tissue swelling (white arrows), which confirms a recent traumatic origin, was not palpable at physical examination

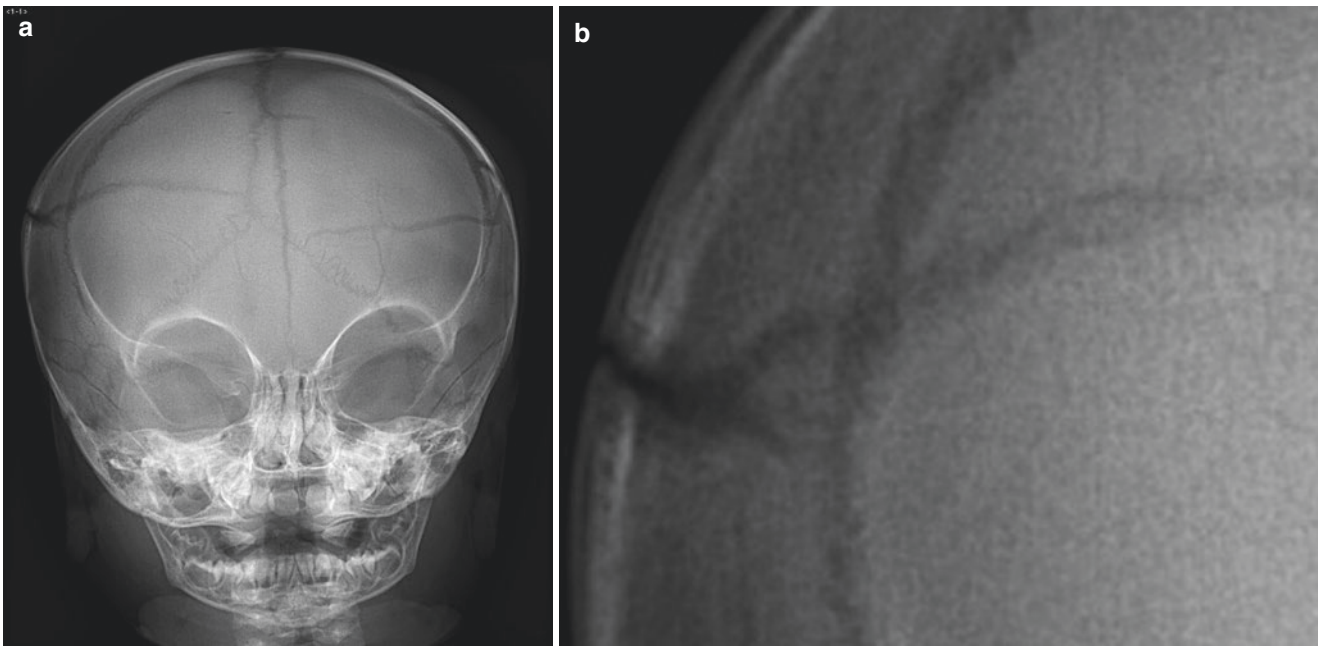


Fig. 5.61 (**a**) Bilateral parietal fractures in a 6-month-old boy as a coincidental finding on a skull radiograph that was made for craniosynostosis. (**b**) The right-sided fracture is not recent and has blurred margins. (**c**) The fracture on the left side is relatively fresh with sharp borders. (**d**) On MRI there a focal haemorrhagic abnormalities on the left side, but no abnormalities (anymore) on the right. The child was placed in foster care

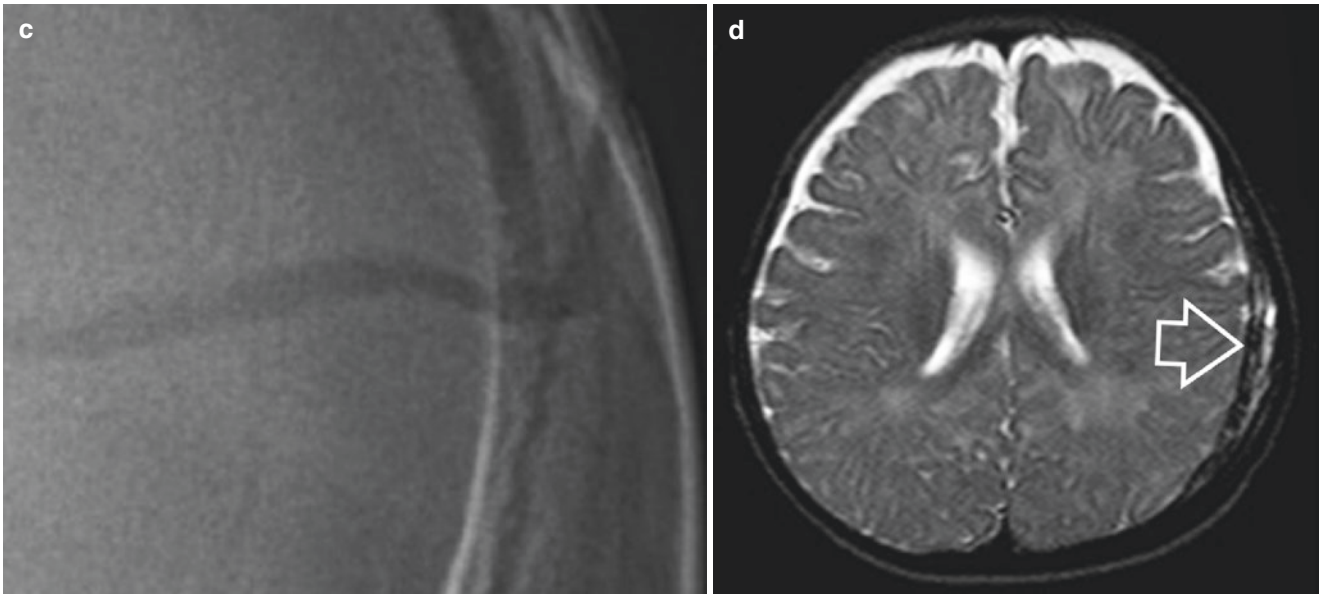


Fig. 5.61 (continued)

It is almost impossible in the first months after birth to differentiate between a calvarium fracture due to a birth trauma or a trauma, sustained after birth, irrespective of the circumstances (accidental or non-accidental) solely on the basis of the radiological characteristics of a skull fracture [184].

5.4 Basilar Fractures

5.4.1 Introduction

The skull base is the floor of the neurocranium and is (from anterior to posterior) composed of the frontal bone, ethmoid bone, sphenoid bone, parietal bone, temporal bone, and occipital bone. Basilar fractures or basal skull fractures may involve any of these bones.

Basilar fractures are relatively rare in children. A basilar fracture can be found in 6–14% of all children with a head trauma, that requires medical intervention (regardless of the circumstances of the trauma). The posterior and middle parts of the base (occipital bone, sphenoid bone, and/or temporal bone) are fractured more often than the anterior part [446]. It is possible for a growing fracture to develop in the base of the skull [417].

Basilar fractures are more complicated than calvarium fractures due to the presence of related structures such as cranial nerves, inner ear structures, and sinuses. Basilar fractures are commonly associated with intracranial injuries. Perheentupa et al. found concomitant intracranial injuries in 43% of 63 paediatric patients (mean age 10.7 years; range 1–18 years) and early neurological deficits, due to traumatic brain injury, in 33% [447]. Ten percent had permanent neurological deficits. Most basilar fractures were due to accidental circumstances (motor vehicle accidents/road traffic accidents). A basilar fracture may lead to loss of cranial nerve functions, such as facial numbness/paralysis, anosmia (decreased sense of smell), visual defects/nystagmus, and hearing loss. The fracture may also lead to incarceration of the cranial nerves. A basilar fracture can also pose a risk for meningitis [166].

Clinical signs may be nausea, vomiting, and general malaise. Also, unconsciousness, seizures, and loss of neurological functions can occur. In a child with a basilar fracture, various physical findings can be found at physical examination, that are considered to be almost pathognomonic, such as the 'Battle sign', 'racoon eyes', haemotympanum, and leakage of cerebrospinal fluid from ear and nose.

The 'Battle sign' is a haematoma directly behind the ear on the mastoid process and is an indication for a fracture of the middle part of the base of the skull in the posterior cranial fossa. If the pars petrosa of the temporal bone fractures, there is often deformation of the external auditory canal which may cause a rupture of the tympanic membrane. This may lead to leakage of cerebrospinal fluid. On inspection, the tympanic membrane may show discolouring, due to the presence of blood behind the tympanic membrane (haemotympanum). With further posterior extension of the fracture, involving the sigmoid sinus, the tissue behind the ear and over the mastoid process may assume a blue-brown colour as a result of blood that collects underneath the fascia. This is called 'Battle sign' [448–450]. Although the 'Battle sign' is usually visible 8–12 h after the fracture is sustained, it may also take as long as 48–72 h [451, 452].

'Racoon eyes' or periorbital ecchymosis is a haemorrhage of the loose connective tissue around the eyes, which causes a red to purple swollen ring around the eye, similar to the rings around the eyes of a racoon. It is a clinical symptom indicative of a basilar fracture in the anterior cranial fossa

[453]. According to McLaurin, the presence of racoon eyes should be considered evidence for a skull base fracture in the anterior cranial fossa, irrespective of finding a fracture on a radiograph or CT scan [446]. Racoon eyes occur when blood seeps from a fracture in the anterior cranial fossa in the loose connective tissue of the orbit. The haemorrhage is sharply outlined due to the connection between the periosteum and the bony margins of the orbit. Usually, racoon eyes are bilateral, since blood seeps via the paranasal sinus into the contralateral orbit. Racoon eyes will show within a few hours, but a time delay from 48 to 72 h has also been reported [451, 454].

There may also be loss of cerebrospinal fluid or blood from the nose (rhinorrhoea) or ear (otorrhoea) or loss of smell due to damage to the terminal filaments of the olfactory nerve at the cribrous lamina [449, 453]. Rhinorrhoea is not necessarily instantly present. It may take some time (days to weeks) after the fracture was sustained [446].

5.4.2 Cause and Manner of Basilar Fractures

A basilar fracture usually is sustained in accidental circumstances, due to a blunt force trauma to the back of the head, such as a blow or a fall. A basilar fracture may also occur as a continuation of a fracture of the cranium, in an impact trauma at the top of the head or a blow in the region of the temporoparietal bone that resonates through the temporal bone into the base of the skull [178]. Furthermore, these fractures can also occur in static loading crush injuries in traffic accidents or in dynamic loading crush injuries in a fall from a great height (above 3 m) [71, 243, 246, 304]. As a result of static loading crush injuries, the skull is deformed relatively slowly and there may be damage to the intracranial structures, such as the brain [243]. In some traffic accidents, there is a combination of dynamic impact loading (e.g. head against car while being hit by a car) and static loading (e.g. when the wheel runs over the head; hereby the head lies more or less stationary and is pressed against a rigid structure). Duhaime et al. reported on seven children between the age of 15 months and 6 years that had sustained crush injuries [243]. They all suffered basilar fractures, six had multiple and often extensive fractures of the cranium. The researchers did not report whether the 7th child, who died soon after arriving at the hospital, due to a transection of the cervicomedullary myelum, had sustained any other skull fractures besides the earlier-mentioned basilar fracture. Four children were victims of traffic accidents and had been run over by a reversing car. In the three other children, there was static loading when the child climbed on a heavy object or pulled at a heavy object that consequently dropped on the head of the child (solid stone front of a fireplace, 27-in television, 45 kg clock). However, the question is whether in the case of these three children one can speak of static loading. It could also be dynamic impact loading, in which the child falls on the floor with its head more or less stationary on the underlying surface and the object drops on the child (see Sect. 5.3.2). This can be compared to the effects of a fall from great height, which may also lead to multiple and extensive fractures of the cranium. According to Takeshi et al., serious crush injuries of the head usually are fatal [246]. The authors also pose that the prognosis of this type of injury, either lethal or excellent, depends on the extent to which the skull and brain have been able to withstand the force. Six of the seven children (average age: 5.9 years) they described had sustained skull fractures. In six children the head had been run over by the wheel of a car. In four children multiple linear fractures of the cranium were found and in six children a basilar fracture.

Basilar fractures probably only very rarely occur due to non-accidental circumstances. While screening the literature no case reports in paediatric patients were found in which was described that the fracture was inflicted.

5.4.3 Differential Diagnosis of Basilar Fractures

Racoon eyes should not be confused with an orbital haematoma/'black eye'. Racoon eyes may be distinguished from a normal 'black eye' by its sharply defined margins and the moment at which the 'black eye' appears. A normal 'black eye' is usually instantly visible with only rarely a delay of a few hours at most. Racoon eyes are generally visible after a few hours, possibly even after as much as 2–3 days. Moreover, in a normal 'black eye', bleeding and swelling may spread to the front and face, whereas racoon eyes will be restricted to the direct vicinity of the eye.

Although some consider racoon eyes to be indicative or even to be evidence of a basilar fracture, Gumus states that the finding can also be a sign of diseases like amyloidosis, Kaposi's sarcoma, multiple myeloma, and metastatic neuroblastoma [455].

5.5 Orofacial Bone Fractures and Dental Trauma

5.5.1 Introduction

Orofacial fractures are less common in paediatric patients than in adult patients [456, 457]. Approximately 5–15% of all facial fractures occur in children [456]. As children grow up, the risk to sustain orofacial fractures increases [458]. Less than 1% of orofacial fractures occur in children under the age of 5 years. Two peaks can be seen in the occurrence of orofacial fractures. The first peak occurs at the age of 6–7 years, and is associated with the beginning of school attendance. The second peak occurs at 12–14 years, and is probably due to increased physical activity and participation in sports during puberty and adolescence. Orofacial fractures

are more often minimally displaced or non-displaced in children than in adults [456].

Fracturing can occur in all orofacial bones: mandible (Sect. 5.5.4), maxilla and zygomatic arch (Sect. 5.5.5), orbit (Sect. 5.5.6), nasal bones and nasal septum (Sect. 5.5.7), and frontal bone (Sect. 5.5.8).

Although there seems to be an age-dependent distribution of orofacial fractures, mandible, and nasal/nasal septum fractures are the most common orofacial fractures in paediatric patients, irrespective of the circumstances, gender, and age [456, 458, 459]. Fractures of the frontal skull and orbital roof are found more often in newborns to children aged 5 years, midface and mandible fractures in children aged 6–16 years and nose and mandible in adults (cited from Oppenheimer et al.) [460]. Imahara et al., just like McCoy et al. and Enlow, found that cranial and central orofacial injuries were more common among toddlers and infants, and mandible injuries were more common among adolescents [458, 461, 462]. Hoppe et al. found that in male patients and the older age groups, the mandible was most commonly fractured orofacial bone, while in female patients and in younger age groups the orbit was most commonly fractured [463]. Alcalá-Galiano et al. found that older children sustained more severe orofacial fractures than younger children [456].

5.5.2 Cause of Orofacial Bone Fractures

Fracturing of orofacial bones in paediatric patients usually requires a significant force, in other words fracturing usually requires a trauma with a high energy transfer (dynamic impact loading). This can either be a blunt force or a (blunt or sharp) penetrating trauma. Penetrating trauma will not be dealt with in this book.

Alcalá-Galiano et al. are of the opinion that not only the circumstances are closely age-related but also the cause: young children, who are constantly supervised and usually live in a protected environment, most commonly sustain orofacial fractures from trauma with a low energy transfer (low-velocity forces) (e.g. accidental short distance falls—Sect. 5.3.3.4), while older children more commonly have injuries due to trauma with a high energy transfer (high-velocity forces), e.g. motor vehicle accidents, sporting activities, physical assault (Sect. 5.6.3) [456]. However, it is not clear from their review on what grounds non-accidental circumstances were excluded in young children, which makes it uncertain how often or even whether young children sustain orofacial fractures due to a trauma with a low energy transfer.

5.5.3 Manner of Orofacial Bone Fractures

5.5.3.1 Accidental and Non-accidental Circumstances

Orofacial fractures can be sustained during and after birth, although almost all orofacial fractures are due to a trauma after birth. In Table 5.11, an overview is given of the results of several studies concerning the circumstances under which orofacial fractures are sustained. Most are due to accidental circumstances, although the circumstances varied with gender and age (Sect. 5.5.3.2). Orofacial fractures due to birth trauma are extremely rare and have been described in a single case report in the mandibula (Sect. 5.5.4), zygomatic arch (Sect. 5.5.5), and orbit (Sect. 5.5.6).

5.5.3.2 Manner, Fracture Location, Gender, and Age of Sustaining

Several authors found that the circumstances under which certain orofacial fractures are sustained varied with gender and age [456, 458, 463–465]. Imahara et al. stated that the proportion of patients with orofacial fractures increases substantially with increasing age [458].

Mericli et al. compared the data of children with craniofacial fractures due to non-accidental (violence-related) and other (nonviolence-related) circumstances [465]. This retrospective study concerned the data of 1528 paediatric patients with skull and/or facial fractures in a major urban children's hospital from 2000 to 2005. Patients with isolated skull fractures were excluded, leaving 793 patients. In 98 (12.5%)

Table 5.11 Accidental and non-accidental circumstances, concerning orofacial fractures

Circumstances	Alcalá-Galiano (2008)	Imahara (2008)	Mericli (2011)	Hoppe (2014/2015)	Allred (2015)
Accidental			87.5%	56.5%	
• Motor vehicle accidents	5–80.2%	55.1%			30.7%
• Sports-related	4.4–42%				24.4%
• Other accidental causes (e.g. falling, bicycle accidents)	7.8–48%				
• Falls		8.6%			
• Other causes (not specified)	1–4.8%				
Non-accidental			12.5%	43.5%	
• Violence/assault	3.7–61.1%	11.8%			13.7%

patients the fractures were due to non-accidental circumstances and in 695 patients due to other nonviolence-related circumstances. Patients with violence-related fractures were more likely to be male and older. They also found that nasal and mandible angle fractures were more common in patients with violence-related fractures, while skull and orbital fractures were more common in patients with nonviolence-related fractures.

In two publications, concerning 353 paediatric patients under the age of 18 years with orofacial fractures (of a total of 3147 patients over a 12-year-study period), eventually, due to insufficient data, 285 patients with a total of 431 fractures were included [463, 464]. Orofacial fractures were most commonly due to accidental circumstances in female paediatric patients (motor vehicle accidents) and in the younger age groups (falls, pedestrian vs. motor vehicle), while the fractures in male paediatric patients and in the older age groups were more commonly due to non-accidental circumstances (physical assault). In male patients and the older age groups, the mandible was most commonly fractured, while in female patients and in younger age groups the orbit was most commonly fractured. Hoppe et al. concluded that the differences in circumstances, fracture locations, and concomitant injuries (Sect. 5.5.3.3) between sexes and different age groups likely reflected the differing activities between the different (gender and age) groups [464]. Hoppe et al. identified 124 paediatric patients (43.5%) who sustained fractures due to non-accidental circumstances (assault—interpersonal violence) [463]. Patients with fractures due to non-accidental circumstances were more likely to be boys and more likely to have sustained a mandibular fracture.

5.5.3.3 Manner and the Occurrence of Associated Injuries

Orofacial fractures in the paediatric population usually result from severe trauma, resulting, due to the severity of associated injuries, in substantial hospital resource use, morbidity, and mortality [456, 458, 459].

Compared with patients without orofacial fractures, patients with fractures exhibited substantial injury severity, hospital lengths of stay, ICU lengths of stay, ventilator days, and hospital charges [458]. Patients with orofacial fractures had more severe associated injuries to the head and chest and considerably higher overall mortality than patients without orofacial fractures.

Allred et al. found associated injuries in almost half of the evaluated patients with the majority involving cerebral trauma (14.7%) or the extremities (9.3%) [459].

In their review of the medical literature, Alcalá-Galiano et al. found that associated injuries were found in 10–88% of paediatric patients with orofacial fractures [456]. The likelihood of associated injuries depended on the complexity and on the location of the fracture. Midfacial and mandibular fractures had a higher risk of other injuries than other orofacial fractures, because the finding of midfacial and mandibular fractures implies a trauma with a high energy transfer. They also found that the most commonly associated injuries in cases of midfacial and mandibular fractures were injuries of the neurocranium and/or the central nervous system.

Mericli et al. found that patients with violence-related fractures had fewer associated serious injuries and lower morbidity (lower rate of hospital admissions and intensive care unit admissions) than patients with nonviolence-related fractures [465].

Hoppe et al. found that intracranial haemorrhage was the most common concomitant injury across most age groups [464]. In another study, Hoppe et al. found that patients with violence-related fractures were less likely to have other systemic injuries such as spinal fractures, intracranial fractures, long bone fractures, and pelvic/thoracic fractures [463]. These patients also had a higher Glasgow Coma Scale (14.7 vs. 12.8) and age (16.0 vs. 12.8 years) and a significantly lower hospital length of stay (2.9 vs. 7.9 days). According to the authors, the differences in presentation between non-accidental and accidental circumstances probably result from the fact that the forces, causing injuries in interpersonal violence, are likely directly directed to the craniofacial skeleton and therefore other organ systems are spared.

Owusu et al. found associated intracranial injuries in 756 of 8848 cases of paediatric mandible fracture (8.5%) and cervical spine fractures in 393 (4.4%) [466].

5.5.4 Mandibular Fractures

Although mandibular fractures are uncommon in children, these fractures probably are, together with nasal bone and nasal septum fractures, the most common orofacial fractures in paediatric trauma patients, irrespective of the gender, age, and circumstances of sustaining [456]. Children tend to have only one mandibular fracture site compared to usually more than one fracture site in adults. The most common fracture sites were condylar (often bilateral) and subcondylar, followed by fractures at the parasymphysis, angle, and symphysis [456]. The symphysis itself (symphysis menti) should not be mistaken for a fracture (Fig. 5.62a, b). Also at birth 2–4 mental ossicles may be present at the caudal side of the symphysis menti that should not be mistaken for avulsion fractures (Fig. 5.63a–c). These ossicles become incorporated into the intramembranous bone when the symphysis menti converts from a syndesmosis to a synostosis during the first postnatal year.

Mandibular fractures are either caused by static loading (compression) or dynamic impact loading (blunt force

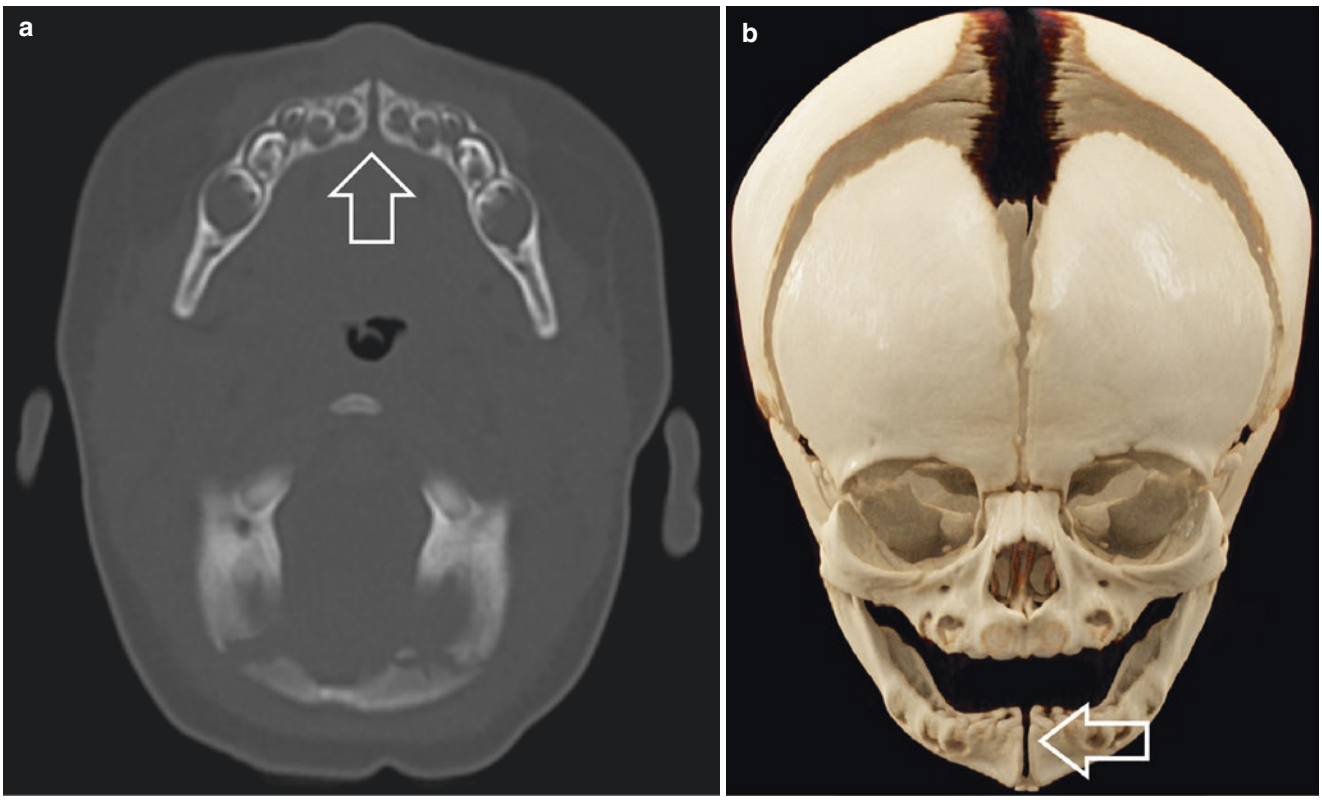


Fig. 5.62 Normal skull of a 3-week-old infant who died of sepsis. (**a**) The mandibular symphysis is indicated by the arrow on axial CT. (**b**) Mandibular symphysis on 3D CT. This should not be mistaken for a fracture. Note the small ossicles near the symphysis, better delineated in Fig. 5.63

trauma), usually a high-energy transfer trauma, except in infants and young children in whom the fracture can be due to a low-energy transfer trauma, like a short-distance fall [467, 468]. Several researchers (Glazer et al. [469]) found that associated injuries were more common in young children [470]. The mandibular is typically affected in Caffey's disease (see also Sect. 14.5.6) and these periosteal appositions may simulate healing fractures (Fig. 5.64a, b).

Most mandibular fractures are due to an accidental trauma after birth [458, 471]. The following circumstances are reported in the medical literature [458, 463, 466, 467, 471–474]:

- Birth trauma:
  - Traumatic vaginal delivery, including use of forceps
  - Caesarean section
- Trauma after birth:
  - Accidental circumstances: (low distance) falls, motor vehicle accidents, bicycle accidents, sporting activities
  - Non-accidental circumstances: child abuse, physical assault
- Unknown/not reported in several cases.

5.5.4.1 Mandibular Fractures in Children Under the Age of 2 Years

Mandibular fractures in children under the age of 2 years are rare with an estimated frequency of 0.9–2.6% [472]. Up to 2016, less than 30 cases were reported in neonates and infants in the medical literature.

Mandibular Fractures in the Neonatal Period

Mandibular fractures are extremely rare in neonates. Some of the risk factors are underlying congenital disorders, birth weight over 4000 g, experience of the midwife/obstetrician, midforceps delivery, or shoulder dystocia [474]. If a mandibular fracture is found in a neonate, it is almost always due to an often not specified traumatic delivery [474–479]. Sometimes the use of forceps during a vaginal delivery is mentioned [480, 481]. A mandibular fracture, sustained during a caesarean section, was reported by Priest [482].

One should, however, always think of other (accidental or non-accidental) circumstances, even in neonates. Gopalakrishnan et al. described a 1-day-old neonate, who sustained a single fracture of the mandibular symphysis after vaginal delivery, immediately after birth, due to falling off the delivery chair on the ground [467]. York described a 3-week-old neonate who sustained the fracture due to a car

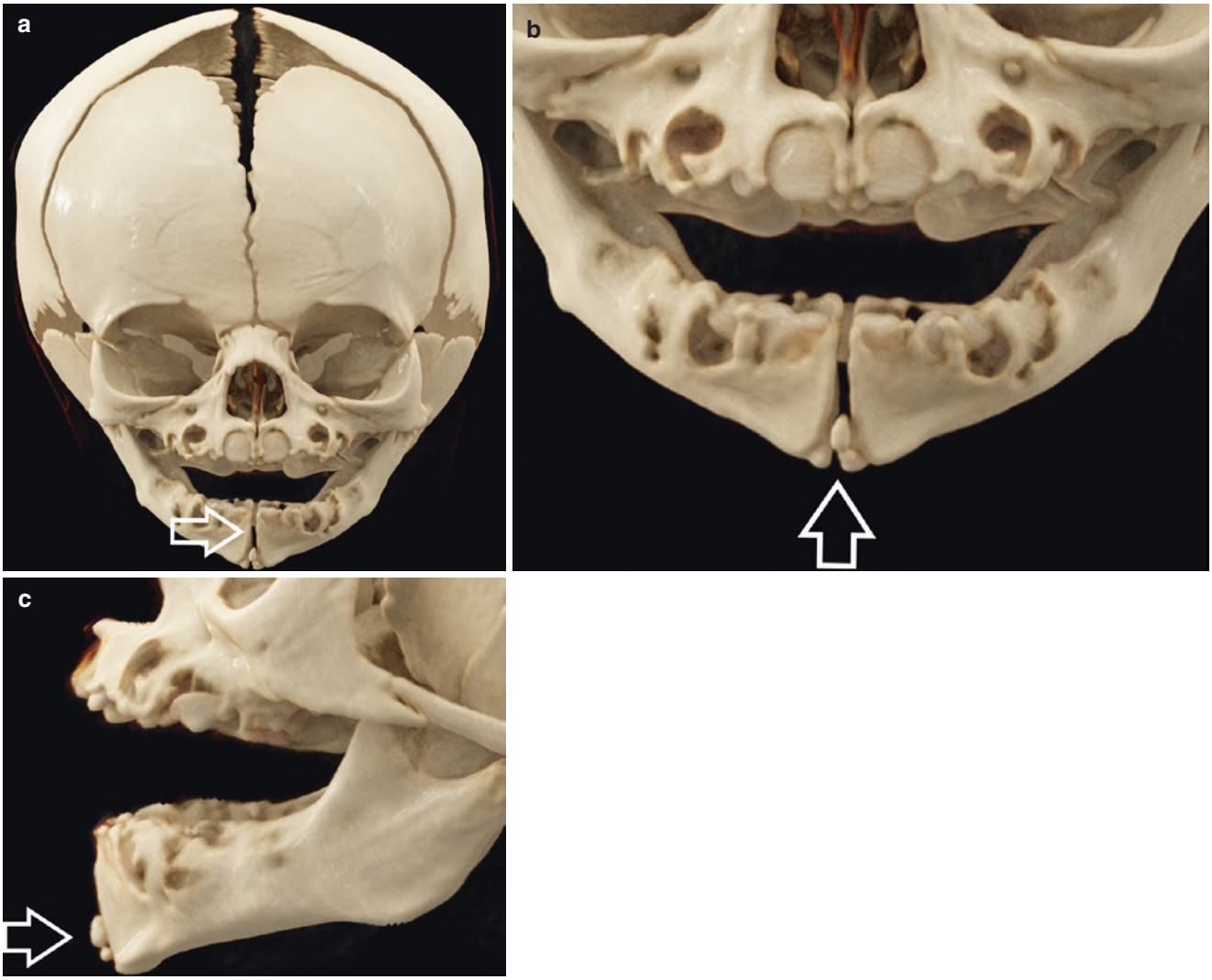


Fig. 5.63 Normal skull of a 2-week-old infant who was evaluated for sudden death. No abnormalities were found. (**a**) The mandibular symphysis is clearly seen (arrow). (**b**, **c**) Three mental ossicles are present at the caudo-ventral side of the symphysis (arrows)

accident [483]. Chidzonga described the occurrence in a neonate, who was assaulted by its mother, who was suffering from a postpartum psychosis [484, 485].

Mandibular Fractures Between 1 Month and 2 Years of Age

Almost all mandibular fractures in children between 1 month and 2 years of age are considered to occur in accidental circumstances (Fig. 5.65a, b). Lustmann and Milhem described seven cases of mandibular fractures in infants (six boys, one girl) due to short-distance falls [472]. All were located in the symphysis region. It is not clear on what grounds non-accidental circumstances were excluded in these children.

- Knoche et al. showed the importance of a scene investigation, if there is doubt about the circumstances [486]. They described a 3-month-old girl with a unilateral mandibular fracture, who allegedly fell from her swing onto her face. The mother explained that she had put her baby in an electric swing, which was placed on a wooden stool. According to the mother a few minutes after starting the mechanical swing it toppled off the stool. The girl landed face down on the carpet overlying a ceramic tile floor. CPS and law enforcement were involved and conducted a scene investigation. They found out that the swing was placed on four small wooden circular barstools, about 45 cm. high. The swing initially appeared stable, but became unstable after being turned on. Above that, the plastic grippers on the base of the swing were worn off in three locations. It was concluded that the fracture was consistent with the accidental circumstances, as described by the parents and as re-enacted during the scene investigation.
- Kim et al. reported an 11-month-old infant, who sustained a fracture of the symphysis of the mandibula

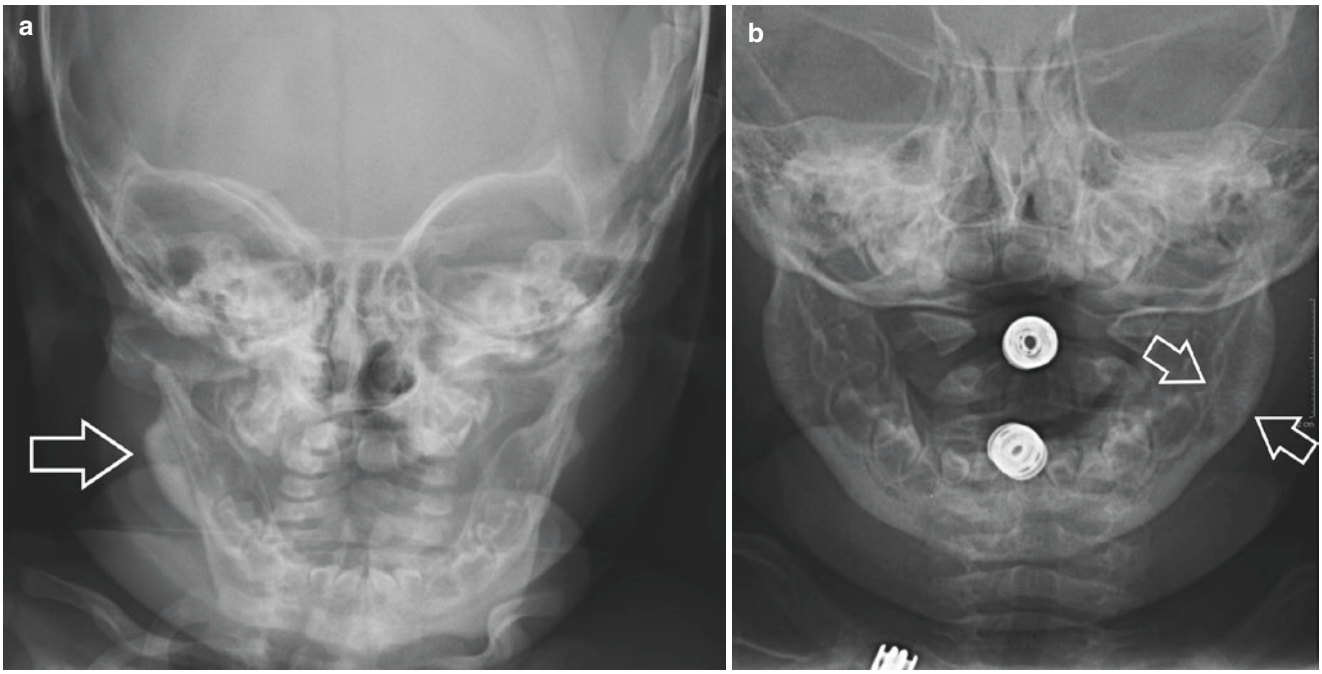


Fig. 5.64 Periosteal new bone formation in Caffey's disease may simulate healing fractures. (**a**) Infant with focal periosteal new bone formation (arrow). (**b**) Another infant shows generalized hyperostosis (between arrows)

with perioral swelling and bruising around the area of the symphysis, due to falling down from a baby carriage [468].

- Letelier et al. reported a 22-month-old girl with multiple fractures of the mandibula (bicondylar, left body, and right parasymphyseal region) due to a motor vehicle accident [487]. She was expelled from her mother's arms through the windscreen after crashing against a road barrier and found conscious 10 m away from the car. She had a normal Glasgow Coma Score (15) and had a right periorbital haematoma, severe right hemifacial oedema, abrasions, mainly on the right side of her face and on her chin, a laceration of the inner side of the lower lip, gingival wound of upper front teeth zone, avulsion and luxation of several teeth. There were no intracranial injuries reported.
- Siegel et al. evaluated the data of 73 paediatric patients, who were seen in a 10-year period, with a mandibular fracture [488]. They found that young children relatively commonly sustained this type of fracture due to non-accidental circumstances (Fig. 5.66a, b). They also found that associated injuries were more common in young children with other inflicted fractures, but that inflicted mandibular fractures were not associated with other injuries.
- Alberth et al. described a 7-month-old boy with an isolated mandibular fracture, in whom it was suspected that the fracture was inflicted [489].
- Schlievert reported the case of a 6-month-old child who sustained a mandibular fracture due to a direct blow [473].

5.5.4.2 Mandibular Fractures in Children Above the Age of 2 Years

Mandibular Fractures in Children Between 2 and 12 Years of Age

Owusu et al. found that in children under the age of 12 years mandibular fractures were most commonly sustained in accidental circumstances [466]. Around 30% were due to falls. The most frequent fracture site in this age group was the condyle.

Chan and Au-Yeung [490] reported a 7-year-old boy with a very uncommon combination of bilateral mandibular condyle and external acoustic canal fractures after falling from his step (scooter).

Mandibular Fractures in Children Older than 12 Years of Age

The mean age of children with mandibular fractures is around 14 years, irrespective of the circumstances under which the fractures were sustained [466].

In children between 13 and 18 years of age mandibular fractures were most commonly sustained (around 40%) in non-accidental circumstances (assault) [463, 466]. The angle of the mandibula was the most frequent fracture site in this age group.

Owusu et al. not only found a difference in age, but also between male and female patients [466]. In male patients, the angle was the predominant site, and in male patients the fracture was most commonly sustained in non-accidental circumstances. In female patients, the condyle was the most frequent site, and the fracture most commonly was sustained in accidental circumstances (falls).

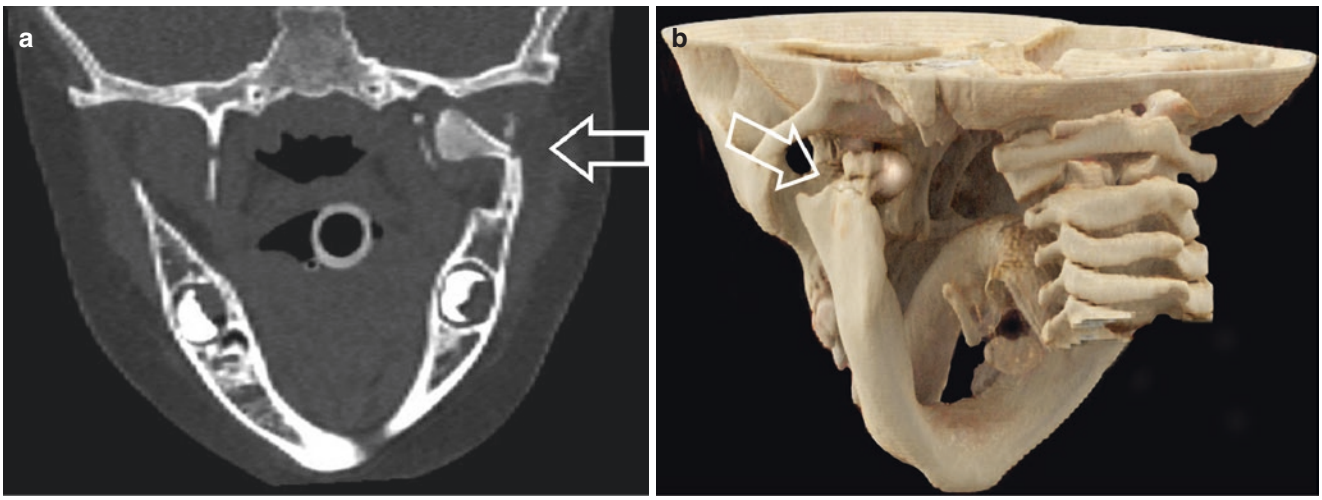


Fig. 5.65 Unilateral condylar fracture in a child who fell forward on her chin during potty training. Arrows indicate the fracture in coronal CT (**a**) and 3D rendering (**b**)

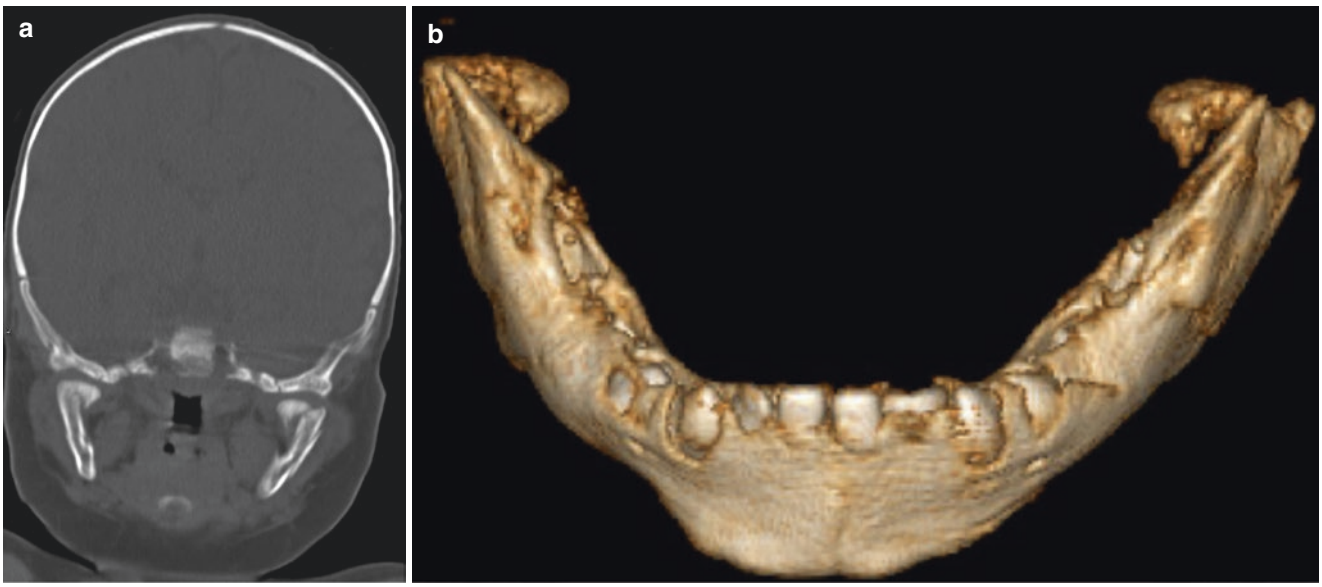


Fig. 5.66 A 5-month-old Infant who underwent a non-accidental injury workup because of facial bruises after a fall. (**a**) Bilateral condylar fractures are seen on coronal CT reconstructions. (**b**) 3D rendering of the fractures

5.5.5 Midfacial (Maxillary and Zygomatic Arch) Fractures

Midfacial fractures are rare in young children and may range from simple minor fractures of the zygoma or the zygomatic arch to complex orbital-zygomaticomalar fractures, involving the zygoma and/or maxilla and/or the orbital rim/floor [456, 491]. Midfacial fractures become more common with increasing age, when the face, according to Alcalá-Galiano et al. undergoes a downward and forward projection with the midface becoming more prominent and less protected by the frontal bone [456]. Sutures around the midface may be mistaken for fractures, e.g. intermaxillary, zygomaticomaxillary, frontonasal, and nasomaxillary sutures (Fig. 5.67a–c). The zygoma forms a large part of the anterolateral wall of the orbit, the lateral margin of the infraorbital rim, and the anterior segment of the zygomatic arch. It articulates with the superior portion of the maxilla and the frontal, temporal, and sphenoid bones. The zygomatic arch is predominantly formed by the zygomatic process of the temporal bone which articulates with the temporal process of the zygoma forming the arch [491]. The maxilla, also known as the upper jaw, is the central bone of the midface. Midfacial fractures can be sustained during and after birth.

If sustained during birth the cause most probably has been compression (static loading). Noyola-Frias et al. described the occurrence of a fracture of the zygomatic arch and right

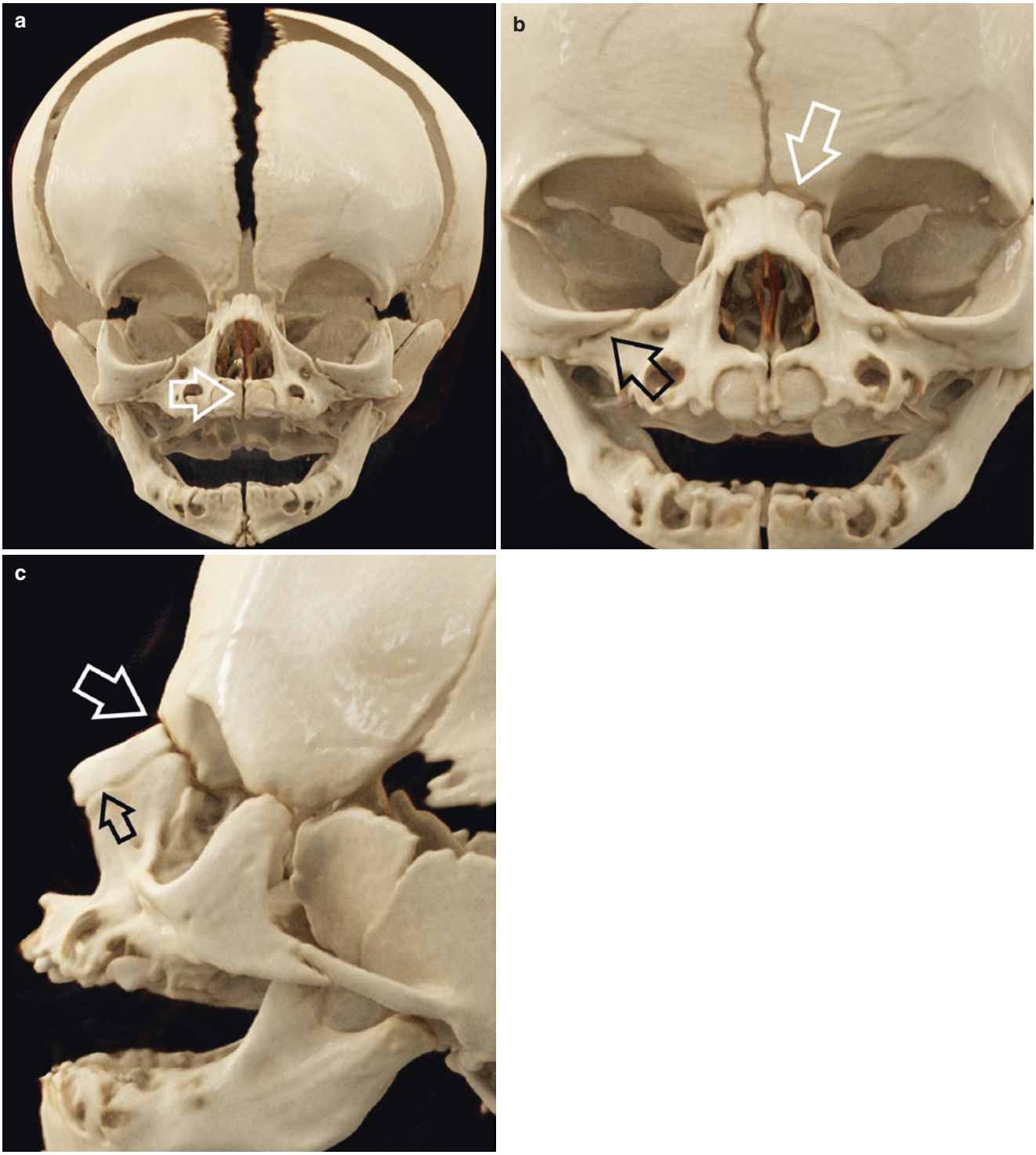


Fig. 5.67 Normal sutures in an infant's face. Intermaxillary suture (arrow in **a**), frontonasal suture (white arrow in **b** and **c**), zygomatomaxillary suture (black arrow in **b**), and nasomaxillary suture (black arrow in **c**)

frontomalar region in a 2-day-old girl due to the use of a forceps during vaginal delivery, because of a persistent transverse position [492].

If sustained after birth, zygomatic arch fractures/midfacial fractures usually are caused by an impact trauma with a high energy transfer, almost always a blunt force trauma [456, 493]. Usually, fractures in this region will be sustained due to accidental circumstances (motor vehicle accidents, falls, or sporting activities).

Non-accidental circumstances (fights, interpersonal violence) are rarely described in the medical literature and if described mostly concern adolescents [464, 494]. Only one

publication was found in which a zygomatic arch fracture in a child was suspected to be inflicted [495]. Maxillary fractures have also been reported as being inflicted in children [9, 496].

Kao et al. evaluated the findings in 218 paediatric patients (aged 0–18 years, average age 11.5 years), who presented with a total of 410 midface fractures (retrospective study) [497]. 80.7% of the fractures were sustained in accidental circumstances. Most common were motor vehicle accidents in 56 patients (25.7%), during sporting activities in 35 patients (16.1%) and falls in 22 patients (10.1%). In 32 patients (14.7%) the fractures were due to physical assault/battery. In 7 patients (3.2%) the occurrence was animal related. In three patients (1.4%) the fractures were classified as gunshot wounds. The most common manners by age were falls in patients, aged 0–7 years, motor vehicle accidents in patients, aged 7–13 years, and physical assault/battery in patients, aged 13–18 years.

5.5.6 Orbital Fractures

5.5.6.1 General Aspects of Orbital Fractures

An orbital fracture is a fracture of one or more than one of the bones that form the bony orbit, which surrounds and protects the eye. The orbit consists of thickened orbital rims and relatively thin inner walls (roof, floor, lateral, and medial wall) (Fig. 5.68). The zygoma and maxilla form the largest part of the medial, inferior, and lateral orbital margins as well as almost all of the orbital floor [491].

Orbital fractures can be divided into orbital rim fractures, orbital floor fractures, orbital roof fractures, and fractures of the medial and lateral orbital wall [498–500]. In paediatric patients, the medial wall and floor are the commonest sites affected [501].

Orbital fractures can occur during and after birth.

5.5.6.2 Orbital Rim Fractures

An orbital rim fracture is a fracture of the bony outer edges of the orbit (Fig. 5.69a, b) [498]. The rim consists of very thick bone and fracturing of the rim only occurs in a trauma with a high energy transfer, usually a blunt force trauma with a direct impact on the face or periorbital region, e.g. in a motor vehicle accident. Because of the type of trauma, rim fractures are often associated with other extensive injuries to the surrounding orofacial soft tissues and bones, and sometimes the eye, the optical nerve, eye muscles, and the brain [502].

5.5.6.3 Orbital Floor Fractures

Floor fractures can be divided into direct and indirect orbital floor fractures. Approximately 24% of floor fractures are associated with ocular injury [456]. Orbital floor fractures usually are caused by a direct impact trauma to the front of the eye and periorbital region, in which the energy, that is transferred during the impact, transmitted downward through the orbital soft tissues to the least resistant part of the orbit, the thin orbital floor [456].

Direct Orbital Floor Fractures

In a direct floor fracture both floor and rim are fractured. The floor fracture is an extension of the rim fracture, in which,

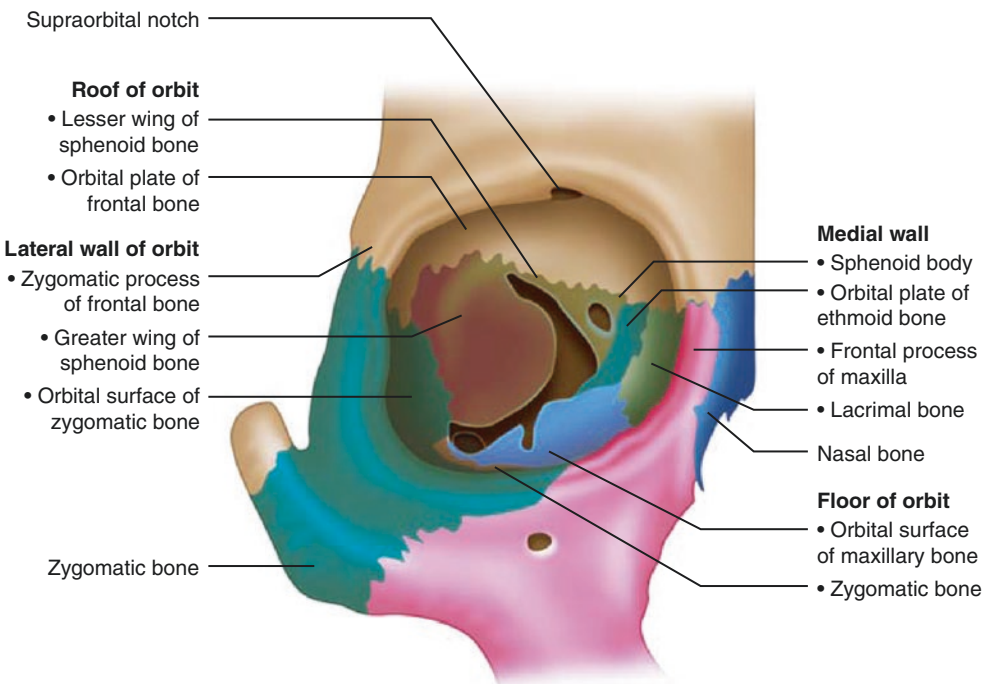


Fig. 5.68 Schematic drawing of the inner walls of the orbit

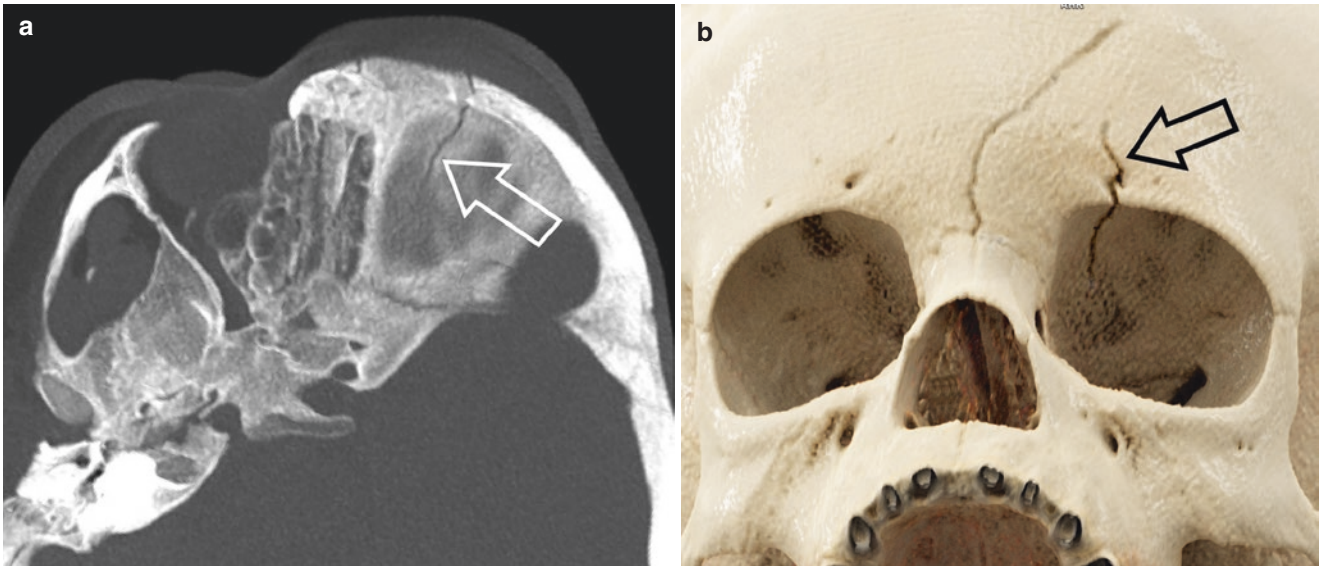


Fig. 5.69 CT of a 3-year-old girl who fell from the stairs. (**a**) MPR MIP image and (**b**) 3D reconstruction show fracture line (arrows) running through the orbital rim. Also, note another fracture line in the frontal bone ending at the frontonasal suture and the monocle haematoma

due to an impact on the rim, the floor is pushed backward and the bones of the floor are buckled downwards. The fracture may also result in damage to the muscles and nerves around the eye. Muscles and other structures around the eye can get caught between the fractured part, keeping the eyeball from moving properly.

A direct floor fracture can be sustained in accidental circumstances, e.g. as a result of an impact to the face and periorbital region in motor vehicle accidents, sporting activities, and daily activities. The fracture can also occur in non-accidental circumstances, e.g. in a physical assault [500].

Indirect Orbital Floor Fractures

An indirect floor fracture occurs when, due to blunt force trauma to the front of the eye and periorbital region (usually a direct blow to the eyebrow and upper cheek-bone), the thin orbital floor buckles or breaks, while the orbital rim remains intact [503].

In the medical literature indirect, orbital floor fractures are also referred to as 'pure internal floor fractures', isolated orbital floor fracture or 'orbital blow-out fractures' [504, 505]. A blow-out fracture, however, can occur in one or more than one bone of the orbital walls [154, 506]. The orbital floor is the most common location of blow-out fractures, although fractures of the floor are often associated with a fracture of the medial wall [154, 502].

An indirect floor fracture/blow-out fracture is usually caused by a blunt force trauma with a high energy transfer to the eye and orbit. Often this concerns a direct blow to the eye and the periorbital region with an object larger than the diameter of the orbital rim [456, 500, 502]. Due to the blow, the intra-orbital pressure suddenly increases. Decompression of the increased pressure occurs by rupturing of the eyeball, and, if the eyeball does not rupture, fracturing of one or more of the bones of the orbital walls at their weakest point, without fracturing the rim [154, 500, 502]. This increased pressure will spread equally over all orbital walls. The floor with a thickness of only 0.5–1 mm is the thinnest bone and weakest point of the orbit and will fracture first. Joseph and Glavas also mention another theory, namely that compression of the inferior orbital rim causes direct buckling of the orbital floor, as can be seen in a direct floor fracture [502]. According to the American Association for Pediatric Ophthalmology and Strabismus (AAPOS) the release of the transferred energy, resulting in buckling or breaking of the floor, may protect the eye from more serious damage [503].

This may result in herniation of the intra-orbital tissues into the antrum, which could result in a growing fracture of the side of the orbit [407]. There may also be haemorrhage into the orbit, which will present as a nasal bleed on the side of the fracture [507].

According to Klenk and Kovacs, blow-out fractures of the orbital floor are rare in children under 8 years of age, due to the anatomical characteristics of growing bone at an early age [508]. Zygomatic fractures often accompany a blow-out fracture of the orbital floor. There must be severe blunt trauma in the medical history [507].

In children and adolescents a specific type of indirect floor fracture has been described, the so-called 'trapdoor fracture', which is a greenstick-like fracture of the orbital floor, linear in form and hinged medially, allowing herniation of orbital contents (orbital fat, inferior rectus muscle) through the fracture and then entrapping these herniated contents, due to recoiling to near anatomical position [456, 504, 506,

509]. Due to this entrapment the tissues, which are caught in the fracture, immediately lose their blood supply. Also, the eye movements will be severely restricted [503]. A variant of the trapdoor fracture is the 'white-eyed' blow-out fracture, characterized by restriction of upward gaze caused by inferior rectus muscle entrapment within the fracture [510].

An indirect floor fracture can be sustained in accidental and in non-accidental circumstances. Wang et al. analyzed the findings in 41 paediatric patients under the age of 18 years (mean age 12.7 years) with orbital blow-out fractures [511]. In 43.9% (n = 18) the fractures occurred due to non-accidental circumstances (physical assault), e.g. a blow with a fist or elbow. Accidental circumstances were found in 56.1% of the patients: motor vehicle accidents in 29.3% (n = 12), falls in 17.1% (n = 7), and sporting activities in 9.7% (n = 4). Egbert et al. found that children above the age of 12 years were more likely to sustain orbital floor fractures due to interpersonal violence than children under the age of 12 years [512]. Hatton et al. found that in paediatric patients nearly 50% of blow-out fractures occurred during sporting activities, e.g. by being hit by a (base)ball or tennis ball or by another player [499]. The fracture, however, can also occur in non-accidental circumstances, e.g. in a physical assault.

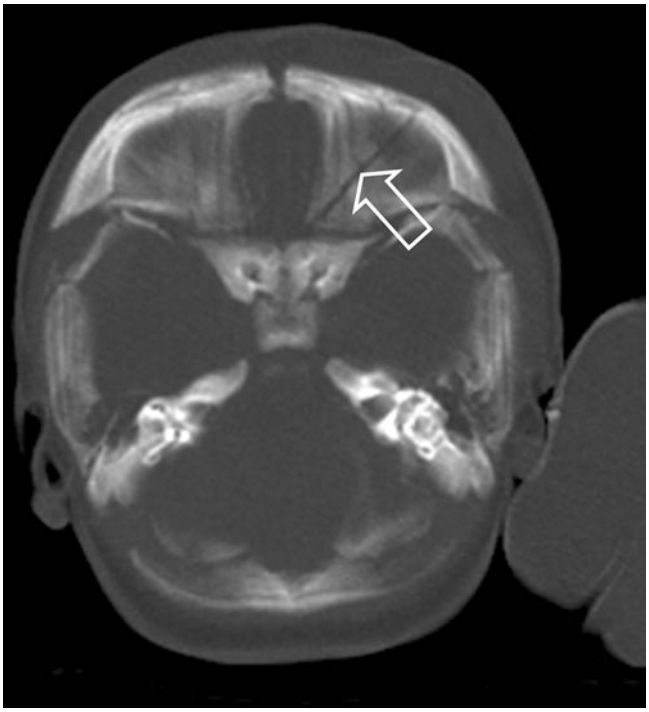


Fig. 5.70 A 3-month-old infant girl who sustained severe neurological trauma and presented in coma at the emergency department. CT of the orbit showed a left orbital roof fracture (arrow) with accompanying soft tissue hematoma. Interrogation by the police revealed that the girl had been hit by a steel pétanque ball

5.5.6.4 Orbital Roof Fracture

Orbital roof fractures are uncommon in children and are typically associated with trauma to the forehead/frontal bone These fractures are often extensions of superior orbital rim fractures (Fig. 5.70). Isolated non-displaced orbital roof fractures are most commonly seen in children and may occur in much more trivial events than in adults, in whom fracturing is associated with trauma with a high transfer of energy, e.g. falls from heights or motor vehicle accidents [513].

- Messinger et al. evaluated the findings in 23 children (aged 3.3 ± 1.6 years) with fronto-orbital trauma (retrospective study, 2 year and 9 month period). Twenty children had fractures in one orbital roof and 3 had fractures in both [514]. Only three children had large, displaced roof fractures. Most children had no associated facial fractures. Only seven children had associated maxillofacial fractures.
- Greenwald et al. evaluated the findings in 32 children with a total of 36 roof fractures (retrospective study, 5-year period) [515]. Sixteen fractures were isolated fractures (75% linear) and 20 (usually comminuted) were associated with more extensive damage to the skull. Haematomas of upper eyelids usually developed hours after the trauma. Isolated fractures typically occurred in younger children (mean age 2.8 years), most sustained in falls from a height under 3 m (10 ft). Only in one case, the fracture was sustained during birth. According to Greenwald et al. roof fractures in children are common, but frequently overlooked [515].
- Koltai et al. evaluated the findings in 40 children, aged 1–16 years, with orbital fractures [516]. Fourteen children had fractures of the roof, 10 of the floor, and 2 of the medial wall. 14 children had mixed fractures. The mean age (4.8 ± 3.3 years) of the 14 children with roof fractures was significantly less than the mean age (12.0 ± 4.2 years) of the 26 children with other orbital fractures. Roof fractures had a significantly greater likelihood of associated neurocranial injuries.
- Ng et al. reported two boys 5 and 6 years with accidental orbital injuries due to falling on handlebar-mounted bicycle hand brakes, perforating in both boys the left upper eyelid [517]. The 5-year-old boy sustained an orbital roof fracture and penetrating brain injury. The 6-year-old boy had an orbital haemorrhage. In both boys surgery was needed.
- Steyn reported the findings in the skeletal remains of a 3.5-year-old boy, who died of a massive cranial fracture, with multiple injuries present to the rest of the body [518]. The body was exhumed to look for signs of chronic, long-term abuse. Besides the massive cranial fracture, a fracture in the orbital roof was found.

5.5.6.5 Orbital Fractures Due to Trauma During Birth

Orbital fractures due to birth trauma have been described only a couple of times in the medical literature [228, 515]. Greenwald et al. reported 36 orbital roof fractures in 32 children over a 5-year period. In only one case the roof fracture was due to birth trauma [515]. Bhat et al. evaluated the data of 34,946 live-born babies over an 11-year period [228]. They found 35 cases of bone injuries with only one case of an orbital fracture.

5.5.6.6 Orbital Fractures Due to Trauma After Birth

General Aspects of Orbital Fractures Due to Trauma After Birth

According to Gerber et al., orbital fractures are the third most common facial fractures in children [501]. According to Bales et al., 5–25% of orofacial fractures are orbital fractures [519]. Nevertheless, orbital fractures are rare in children, irrespective of the fractured bone(s) [456, 460, 501, 518]. In the acute phase, the externally visible signs of orbital fractures in children are abrasions of the eyelid, haematomas, and oedema [507] (Figs. 5.69 and 5.70).

- Losee et al. did a retrospective review of the findings of 74 paediatric patients under the age of 18 years (average age 8.6 years) with a total of 81 orbital fractures [520]. They found that in their population 90.5% of the orbital fractures were due to non-accidental circumstances. Paediatric patients who sustained orbital fractures from activities of daily living were, on average, 6 years old, whereas paediatric patients who sustained orbital fractures in non-accidental circumstances were twice as old, with an average age of 13.6 years.
- Gerber et al. evaluated the findings in 24 consecutive paediatric patients under the age of 18 years (mean age 13.5 years) (retrospective study, 2005–2010) with orbital fractures. Fifty-eight percent of the patients ($n = 14$) had isolated floor fractures [501]. The remaining patients had fractures of other orbital and/or facial bones. Thirty-eight percent ($n = 9$) had indirect floor fractures (blow-out fractures) and 46% ($n = 11$ had trapdoor fractures). Most fractures were sustained in accidental falls.
- Barh et al. evaluated the findings in 52 paediatric patients under the age of 18 years (range 2–18 years, mean age 10.9 years) with orbital fractures and found that 81% of the floor was a fracture site. In almost 50% a trapdoor fracture was found [521].
- Depending on the location of the fracture, fracturing of the orbit can cause severe damage to the eyeball or the optic nerve. In 50% or more of the orbital fractures, that are sustained after birth, there is also (intra)ocular damage [499]. It is also possible that ocular muscles get incarcerated in the fracture [522–525].

Orbital fractures due to trauma after birth are caused either by direct trauma (blunt force trauma, impact trauma) or by indirect trauma (trauma, due to deformation) to the globe, the orbital, facial, or cranial bones and can be sustained in accidental and non-accidental circumstances, irrespective of the bone(s) that are fractured [456, 460, 518, 526].

In case of accidental trauma, usually, a blunt force trauma with a high energy transfer will be mentioned in the medical history, most commonly with an impact directly unto the orbit (Fig. 5.71a–c) [499, 500]. In such a trauma the globe is more resistant to perforation than the bony orbit is to fracture [491]. Usually, these are fractures of the orbital floor and medial side of the maxilla [499]. This may happen in sporting activities in adolescents, e.g. when the face or the periorbital region is hit by an object larger than the eye socket, like a baseball or a softball. It may also occur in a motor vehicle accident when the face/periorbital region hits the dashboard or steering wheel during the collision. If this happens there are often associated injuries to the eye/eyeball, tear duct, periorbital bones and sinuses, and brain can occur [500].

When a child presents with an orbital fracture, and no blunt force trauma with a high energy transfer is mentioned, one should always consider non-accidental circumstances (physical violence), e.g. when the face or the periorbital region is hit by a fist.

Gender and Orbital Fractures Due to Trauma After Birth

Boys sustain orbital fractures more often than girls [503]. Losee et al. evaluated the findings of 74 paediatric patients under the age of 18 years and found orbital fractures in 53 males and 21 females [520]. Hink et al. described 591 orbital fractures and associated ophthalmic and craniofacial injuries in 312 paediatric patients, in whom orbital fractures were diagnosed by CT scan: 62% ($n = 192$) were boys and 38% ($n = 120$) were girls [527]. Sirichai and Anderson evaluated the data of 41 children, aged 8 months to 15 years (retrospective study, 10-year period) with orbital fractures and found a clear male predominance [528]. 33 of the children were male. Firriolo et al. evaluated 152 paediatric patients with floor fractures: 80.3% ($n = 122$) were boys and 19.7% ($n = 30$) were girls [529]. Barh et al. [521] evaluated the findings in 52 children, aged 2–18 years, with orbital fractures: 39 boys and 13 girls.

Location of the Orbital Fractures and Age of Sustaining

Alcalá-Galiano et al. found that orbital floor and orbital rim fractures were rare in young children, and that this was due

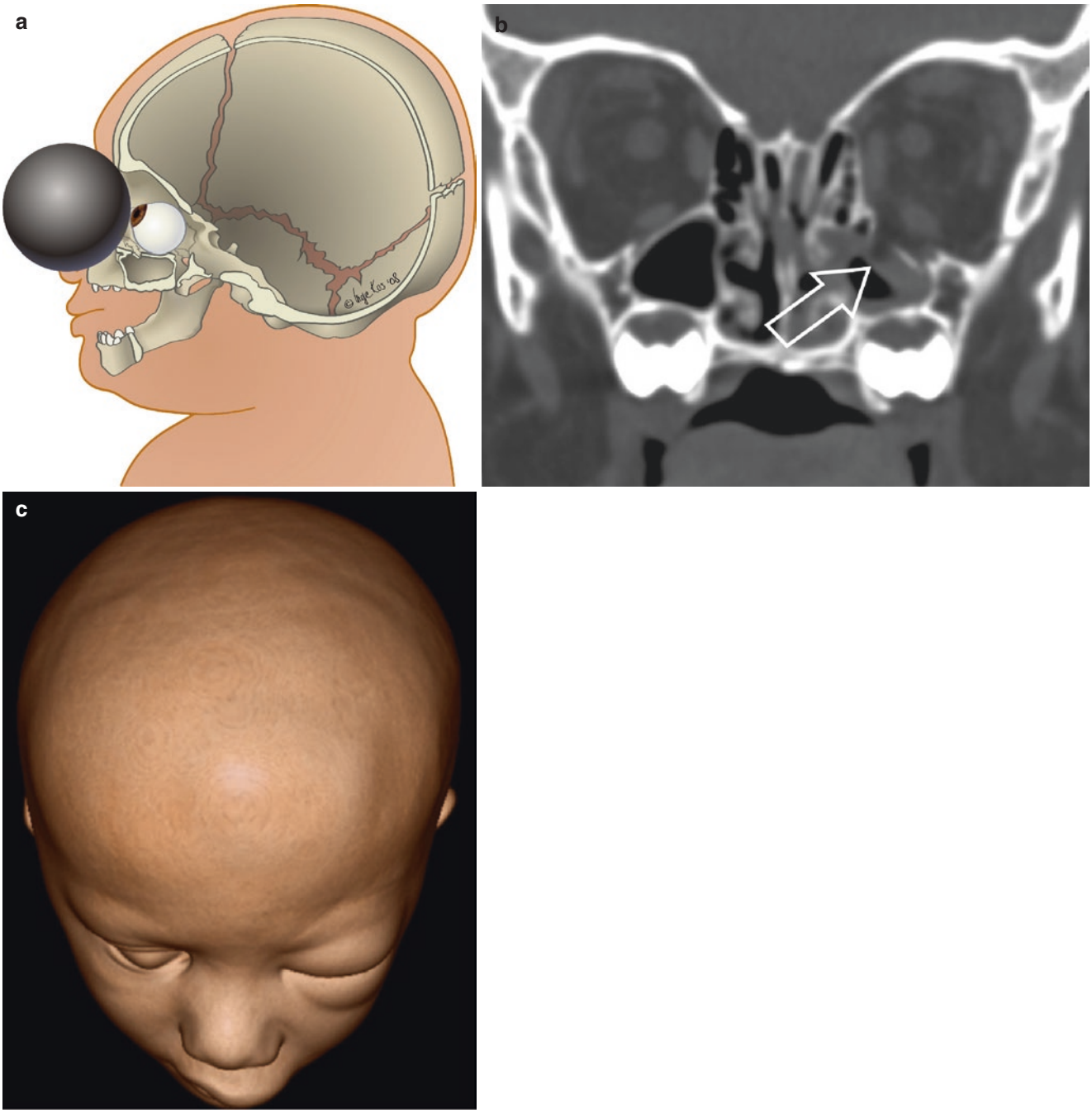


Fig. 5.71 Blow-out fracture in a 3-year-old girl. Mechanism (**a**) orbital floor fracture with depression and herniation of orbital fat (arrow in **b**) and resulting in soft tissue haematoma (monocle sign) (**c**)

to the anatomical development of the orbit and periorbital bones and sinuses [456]. In young children blunt force trauma on the frontal bony usually is transmitted directly to the orbital roof, while with increasing age the frequency of orbital floor fractures increases. The authors also stated that after the age of 7 years, orbital fractures mainly will affect the medial and lateral walls and the floor. According to them a blow-out fracture of the orbital floor is a common fracture in children of this age. Koltai et al. evaluated the findings in 40 paediatric patients between the age of 1 year and 16 years with orbital fractures (retrospective case series) [516]. Roof fractures were found in 14 children with a mean age of 4.8 ± 3.3 years. Ten children had floor fractures, 14 children had mixed fractures, and two children had fractures of the medial wall. These 26 children had a mean age of 12.0 ± 4.2 years. The authors also found a significantly greater likelihood of associated neurocranial injuries in children with roof fractures. According to the

authors, roof fractures occur primarily in younger children due to the proportionally prominent forehead and the lack of frontal sinus pneumatization. Lower orbital fractures occur primarily in older children because of the increased vulnerability of the face due to growth and the pneumatization of the paranasal sinuses.

Sirichai and Anderson evaluated the data of 41 children, aged 8 months to 15 years (retrospective study, 10-year period) [528]. Orbital floor and multiwall fractures were most common, with medial wall fractures as the second most common site. Most fractures were sustained during sporting activities (more often with increasing age). Blunt force trauma due to falls was more common in young children (in their study all 10 years of age or less). Fractures of the orbital roof and lateral wall were more common in young children (Fig. 5.70) (decreasing in frequency with increasing age), while those of the orbital floor and medial wall occurred at any age, although those of the medial wall were more common among older children. Sirichai and Anderson stated that these locations and patterns of fractures changed with increasing age, due to changes in behaviour and activities, and due to growth and development of the craniofacial skeleton [528].

Hink et al. described 591 orbital fractures and associated ophthalmic and craniofacial injuries in 312 paediatric patients (192 boys, 120 girls), aged 4 months to 16 years (average 7.3 years), in whom orbital fractures were diagnosed by CT scan (retrospective study, 2002–2011) [527]. They found that in their population orbital fractures associated with other craniofacial fractures were more common than isolated orbit fractures (internal fractures and fractures involving the orbital rim but without extension beyond the orbit) (62 vs. 38%). Roof and medial wall fractures were most common (30% and 28%, respectively), followed by orbital floor (24%) and lateral wall (18%) fractures. Orbital roof fractures were the most common fractures in patients under the age of 8 years, whereas orbital floor fractures were the most common fracture in patients above the age of 8 years. Associated neurologic injuries were more common (23%) than associated ophthalmic injuries (20%).

Yang et al. evaluated the findings in 177 paediatric patients under the age of 18 years (retrospective study, 2004–2014) with orbital wall fractures [530]. The floor was the most common fracture site, irrespective of the age of the patient. A floor fracture was present in 50% of the children under the age of 7 years (pre-school age) and in 64.4% of the patients, aged between 7 and 18 years (school age). The male-to-female ratio and the circumstances under which the fracture was sustained showed significant differences between the two age groups. The sex distribution in the preschool group was equal (52.0% boys) whereas in the school age group the overwhelming majority of patients were boys (93.4%, $P < 0.001$). Accidental falls from heights were responsible for 42.9% of the orbital fractures in children under the age of 7 years, while physical violence (non-accidental trauma) was responsible for 49.3% of the orbital fractures in patients, aged between 7 and 18 years. Associated injuries and facial fractures were found more frequently in patients above the age of 7 years.

5.5.7 Nasal Bone and Nasal Septum Fractures

Nasal bone and nasal septum fractures are caused by dynamic impact loading, usually blunt force/impact trauma. Blunt force trauma to the nose occurs regularly in children, but not all children who sustained a blunt force trauma to the nose are in need of medical treatment [531]. The nasal bones are the most prominent bones of the orofacial skeleton and above that the least protected and least resistant against impact trauma [532]. This also explains why, compared to other orofacial bones, the nasal bones, just like the mandibula, so often fracture, despite the fact that the nasal cartilage is relatively compliant [456].

The circumstances, under which the trauma to the nasal bones/nasal septum can occur, are either accidental or non-accidental.

Accidental circumstances are most common and concern motor vehicle accidents, sporting activities, and daily life accidents like falls [533, 534]. Desrosiers and Thaller found inflicted nasal/nasal septum fractures in 15% of the evaluated paediatric patients [533]. Liu et al. found nasal fractures due to interpersonal violence in 10% of their cases [534].

Borner et al. found a decrease in fall-related injuries with increasing age, whereas accidental and non-accidental blunt force to the nose increased with age [531]. They also found that male patients had a higher risk for soft tissue injuries and frontobasal fractures (individual or combined fractures of the orbital roof, the posterior wall of the frontal sinus, the roof of the ethmoid sinus, or the roof or wall of the sphenoid sinus).

Blunt force trauma to the nose may lead to externally visible superficial (abrasions) and deeper (lacerations) cutaneous injuries. It may also lead to haemorrhage and fractures of the bony or cartilage part of the nose [535, 536]. After direct blunt force trauma, a haematoma or the development of an abscess in the nose septum is a rare complication.

In young children, injuries to the cartilage of the nasal septum are rare, irrespective of the circumstances, under which the damage was sustained (accidental or non-accidental). In the medical literature, there are only a few case reports on inflicted nasal bone and nasal septum injuries [537, 538]. However, according to Nathanson, fractures of the nasal bones and the nasal cartilage of young children strongly suggest non-accidental circumstances [539]. This is certainly true when there is no serious trauma in the medical history.

Precious et al. described three children, aged 11, 14, and 17 years, who had sustained nasomaxillary injury at least 8 years earlier as a result of physical beating [538].

Canty and Berkowitz described 20 children (aged, 2 months to 15 years; mean age, 7 years) with a post-traumatic haematoma of the nasal septum [537]. Nasal fracture was present in three children. In two children (under 2 years old), a septum haematoma (and the consequent development of an abscess) resulted from non-accidental circumstances (child abuse). Compared to children that presented with a septum haematoma and developed an abscess after a minor and isolated nasal trauma (14 children, aged 1–14 years old) and after a sports injury (four children, over 10 years old), the children with inflicted injuries were all young (under 2 years old). Moreover, the children with inflicted injuries had sustained severe additional injuries in the head and neck region (face, neck, nose) and the patient history recorded earlier inflicted injuries (child abuse).

Inflicted injuries of the nose, and of other inflicted injuries to the face, are often accompanied by other extracranial injuries, such as fractures and haematomas on trunk and extremities [540].

5.5.8 Frontal Bone Fractures

According to Alcalá-Galiano et al. fractures of the frontal bone are common in young children because of the prominence of the forehead, overhanging the face [456]. In children under the age of 7 years fractures of the frontal bone tend to extend superiorly in the calvarium or across the orbital roof. According to Alcalá-Galiano et al., orbital roof fractures are considered skull fractures, and these are often associated with neurocranial injury [456].

5.5.9 Dental Trauma, Dental Neglect, and Gingival Injuries

5.5.9.1 Dental Trauma

Introduction

Dental and periodontal trauma regularly occurs in children. Epidemiological studies have shown that overall (children and adults), the incidence pro year of dental trauma is around 4.5% [541]. It is estimated that almost 50% of all children will sustain some kind of dental or periodontal trauma during childhood [542–544]. According to Andreasen and Ravn about 30% of children and toddlers will have injuries of the primary dentition (boys vs. girls = 1:1) [542]. Widmer, however, is of the opinion that in two out of three children one may find damage to the deciduous teeth [545]. In one out of five children over 6 years of age/adolescents/adults, there is damage to the permanent teeth [541, 545]. Dental trauma to the permanent dentition is recognized at least twice as often in boys than in girls [542]. Faus-Damia et al. found a prevalence of dental injuries of 6% in children aged 6–18 years [546]. They also found that boys between 12 and 18 years of age sustained more injuries than girls.

Dental injuries can be injuries to the enamel (chipping) and fracturing of the crown and root (Fig. 5.72a–c). Periodontal injuries include gingival bruising, luxation (displacement) of teeth (intrusive, extrusive, lateral), and avulsion of teeth (Fig. 5.73a–c). Most of the injuries are minor, most commonly due to chipping of only the enamel. Fracturing of the crown also occurs regularly, but is less common. The most commonly injured teeth are the maxillary central incisors, followed by the maxillary lateral incisors and the mandibular incisors [541, 543].

Many of these injuries will not be reported or seen by a dentist, because they are minor or are not recognized as such. Subtle damage to the teeth can easily be missed by non-dentists or by inexperienced dentists. If injuries are found or suspected, the assistance of an experienced (paediatric) dentist should be sought [547].

Cause and Manner of Dental Trauma

Dental trauma in children is almost always caused by blunt force trauma (impact trauma). The severity of the impact ranges from minor, resulting, e.g. in chipping, to major, resulting, e.g. in fractures of the root or tooth avulsion, combined with other severe injuries in the orofacial region.

The circumstances of the trauma may be accidental (falls, traffic accidents, sports-related) or non-accidental (altercations, physical abuse, assaults, and torture) [288, 545, 548]. In pre-schoolers and school-aged children dental injuries most commonly are sustained in accidental falls at home, whereas in adolescents contact during sports (e.g. collisions, elbowing, or falls) and altercations are the most prevalent circumstances [541, 543, 549]. Also, free-time activities, like bicycle rides, walking, roller skating, and skateboarding play an important role in the occurrence in adolescents [549].

According to Widmer, dental injuries are found in 30% of victims of child abuse [545]. However, differentiating between non-accidental and accidental traumatic dental injuries is very difficult, perhaps even impossible, if the injury has to be assessed separately from the context (medical history, age, and developmental level of the child) [545, 550].

Discoloured teeth, indicating pulp necrosis, may be the result of an earlier trauma [546, 551, 552]. The differential diagnosis of discoloration should include a history of exposure to tetracycline or heavy metals during enamel formation [540]. In this situation, one will find discoloration of all teeth that were formed during the exposure. Wright and Thornton pointed at dentinogenesis imperfecta in the differential diagnosis of discoloration, caused by child abuse [553].

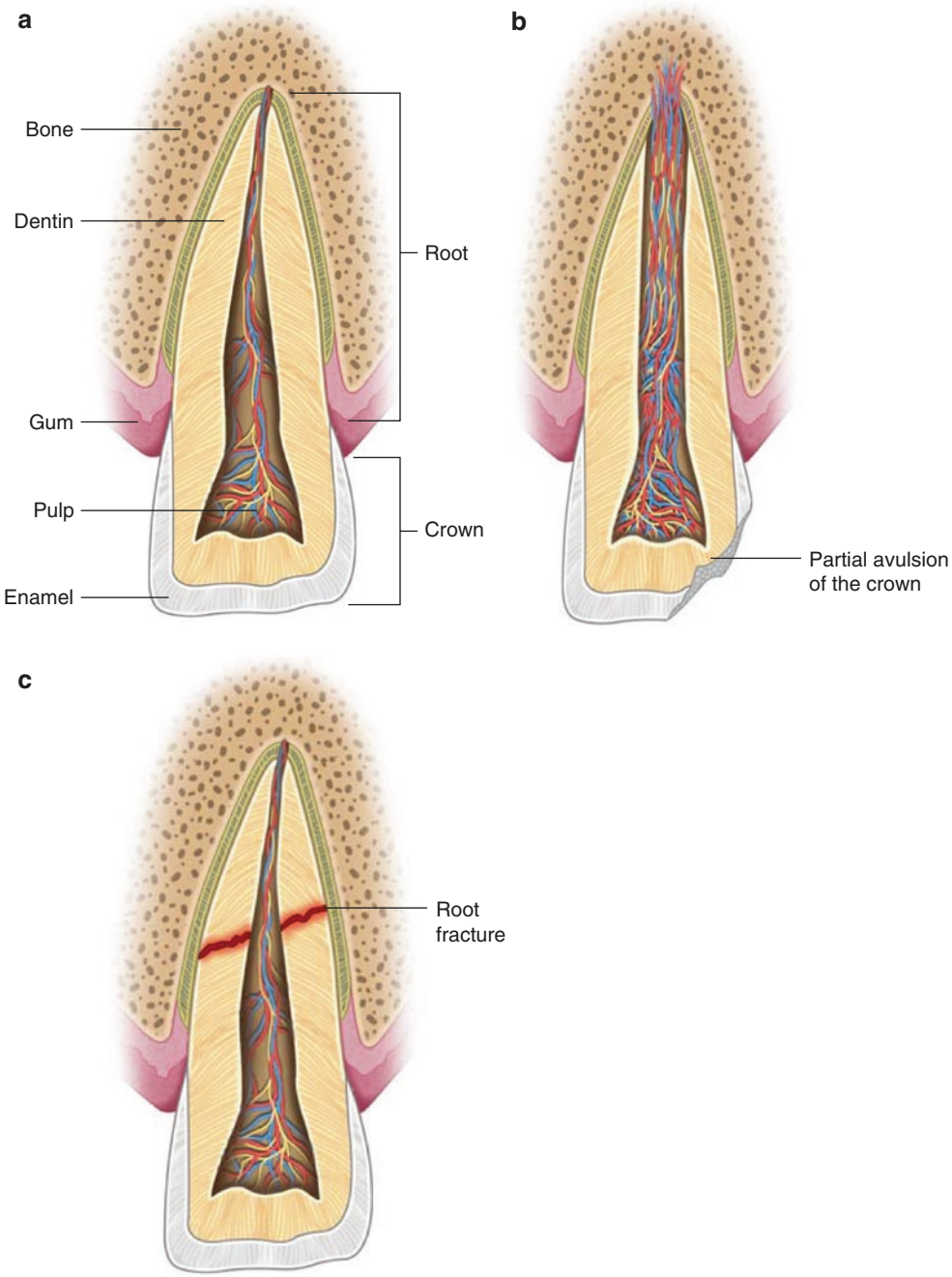


Fig. 5.72 Schematics of (**a**) a normal tooth, (**b**) partial avulsion of the crown with secondary inflammatory changes and (**c**) root fracture

Intrusion and forced extraction of teeth have also been reported in the paediatric medical literature. Kaplan described the extraction of canines within certain cultures for treating paediatric diseases [554]. Edwards et al. described the extirpation of primary canine tooth follicles ('ebinyo') by traditional healers in infants in rural areas of eastern Africa to prevent high temperature, vomiting, loss of appetite, and diarrhoea and reported this in five infant siblings, who apparently had been subjected to 'ebinyo' as infants before immigrating to the United States [555]. Carrotte reported a family in which the parents over the years had extracted various permanent incisors from three of their six children as punishment for misbehaviour [556]. One parent held the child, while the other removed the teeth.

In adolescents self-injury should be considered in case of dental fractures. Gantha et al. reported a 14-year-old boy, who visited the Department of Paediatric Dentistry together with his parents, because of broken upper front teeth [557]. The parents revealed that the boy himself broke his teeth by hitting with a hammer. The parents stated that the boy was constantly bullied in the school by his peers because of the abnormal size of his teeth.

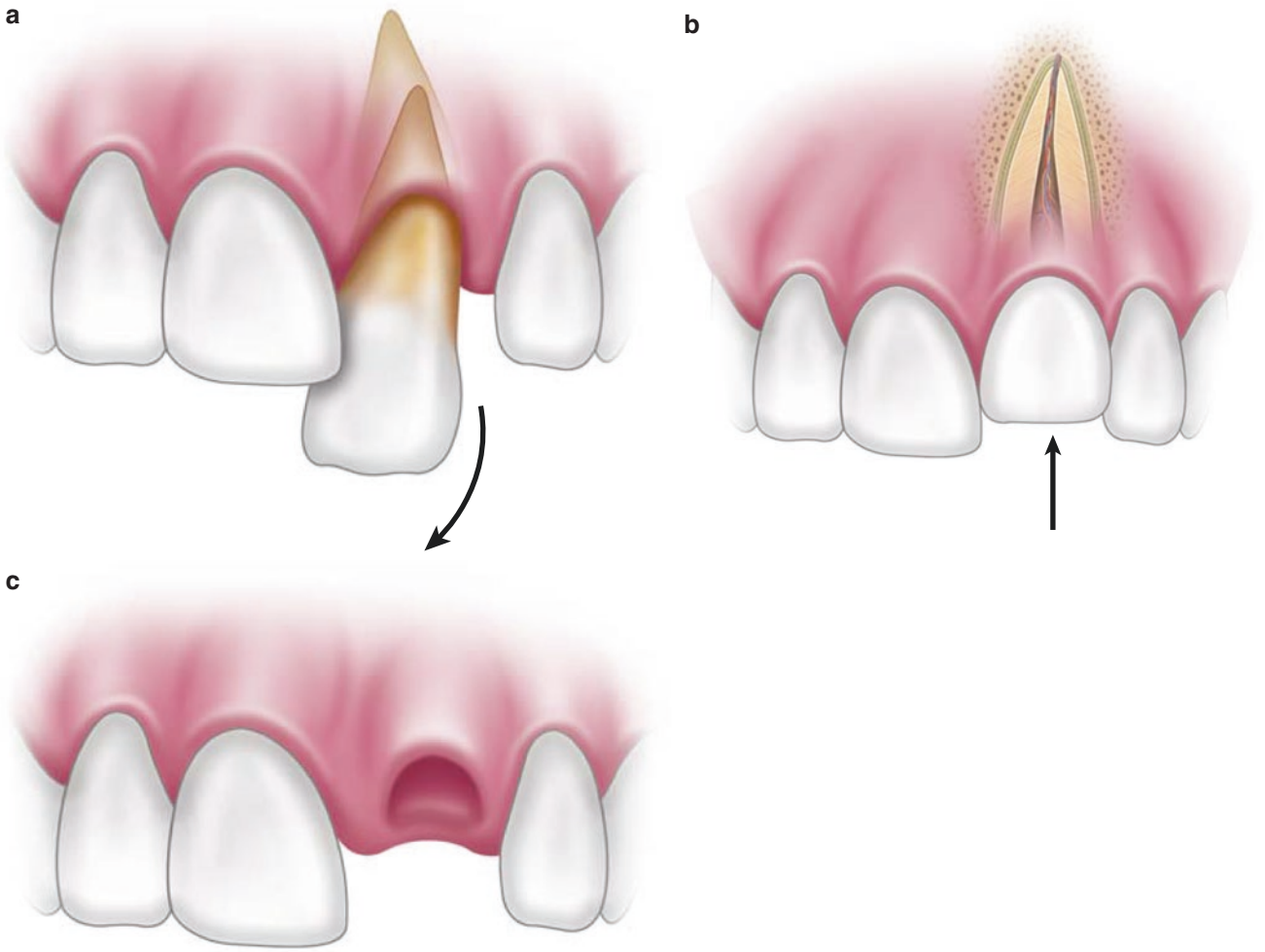


Fig. 5.73 Schematic of (**a**) extrusive displacement, (**b**) intrusive displacement, and (**c**) complete luxation

5.5.9.2 Dental Neglect

Dental neglect is defined by the American Academy of Pediatric Dentistry (AAPD) as '*willful failure of parent or guardian to seek and follow through with treatment necessary to ensure a level of oral health essential for adequate function and freedom from pain and infection*' [558]. The British Society of Paediatric Dentistry (BSPD) defines dental neglect as '*the persistent failure to meet a child's basic oral health needs, likely to result in the serious impairment of a child's oral or general health or development*' [559]. In other words, dental neglect is the failure on the part of the parent(s)/caregiver(s) to provide preventive dental care in the form of adequate hygiene or in the prevention of caries. It also includes not seeking adequate dental help for caries, infections, or any other abnormalities of the teeth or supporting structures, which makes normal eating difficult or impossible, causes chronic pain, slows down the growth and/or development of the child and makes it difficult or impossible for the child to participate in all kinds of daily activities, such as play or school [20].

Important signs of dental neglect include the occurrence of caries even conspicuous for laymen, and negligence in seeking medical/dental assistance for infections, bleeding, or trauma to the head and neck region. According to Lourenço et al. and Harris caries (dental decay) is probably the most significant sign of neglect in children [560, 561]. Dental neglect may occur in isolation, but could also be a significant sign of other types of neglect or more generalized child maltreatment [562, 563]. Research done by Greene et al. showed that victims of child abuse and neglect had an eight times higher risk of poor permanent teeth [564].

According to Ramazani dental caries is the most prevalent infectious disease in the paediatric population [563]. A 2003 survey, cited by the British Dental Association (BDA), showed that by the age of 5 years, 43% of UK children had obvious decay [562]. In 2005, an epidemiological study of dental neglect in young people reported a prevalence of between 40 and 50% in 15- and 16-year-old adolescents at secondary schools in a deprived inner-city area [562].

The BDA stated that even extensive decay will not always indicate neglect and that the finding of caries should never be interpreted in isolation but always assessed in the context of the child's medical and social history and developmental stage [562]. Hinchliffe mentions lack of knowledge or difficulty in understanding or complying with home dental care or dietary needs by parents or carers [565]. This may cause dental problems in the child, resembling those that are found in neglect, but cannot be considered, according to Hinchliffe as deliberate neglect, e.g. a child with rampant caries with a parent/caregiver who is unaware that caries may be associated with poor oral hygiene, sweet diet, and drinks.

5.5.9.3 Gingival Injuries

Accidental or inflicted dental injuries are often combined with bruising or lacerations of the surrounding gingiva. Trauma caused by a blow or an object striking the child's face may lead to gingival injuries without apparent dental injuries.

Traumatic damage to the gingiva may be found as a result of child abuse. Iatrogenic, accidental, and artificial damages must be taken into account in the differential diagnostics of gingival damage. This may involve the effect of chemical substances such as aspirin or peroxide, thermal stimuli such as hot or cold food, and physical causes such as flossing, piercings, and self-inflicted injury. Iatrogenic and accidental causes frequently appear to be self-limiting, whereas artificial abnormalities often appear to be chronic [566].

5.6 Intracranial Injuries

5.6.1 Introduction

Head injuries and intracranial injuries are sometimes used as synonyms in case of the physical findings, due to head trauma, irrespective of whether these were sustained in accidental or non-accidental circumstances. This is incorrect from a forensic point of view. The term 'head injuries' should only be used as a generic term for unspecified injuries to the head, which can be injuries to the soft tissues of the orofacial region and/or the scalp and/or the bony tissues of the skull (calvarium, base, and orofacial bones) and/or the intracranial contents (meninges, brain), caused by mechanical or non-mechanical trauma (Sect. 5.1.1). The use of the term 'intracranial injuries' should be limited only to injuries of the intracranial contents (Sect. 5.6.2).

If a child sustained a head trauma and the trauma resulted in injuries, the exact character and location of all injuries to the head should always be specified, because this may help to evaluate the circumstances under which the trauma was sustained (accidental or non-accidental). Because the focus of this book is on (forensic aspects of) fractures in children, in this section injuries to the intracranial contents and the differential diagnosis of these findings will only shortly be described.

Traumatic brain injury is the most common cause of permanent disabilities and death in paediatric patients (Chap. 1). This is irrespective of the circumstances (accidental or non-accidental) under which the trauma was sustained [567–572].

5.6.2 Trauma-Related Intracranial Findings

The most prevalent trauma-related intracranial findings are due to the accumulation of blood either in physiological circumstances existing or in non-existing spaces (= haematoma). This accumulation is the result of extravasation of blood from damaged vessels (veins, arteries, capillaries) (= bleeding or haemorrhage). An overview of the most common trauma-related intracranial findings is given in Table 5.12 and Fig. 5.74a–h.

5.6.3 Cause of Trauma-Related Intracranial Findings

Trauma-related intracranial findings can be divided into damage due to primary and to secondary causes.

Primary trauma-related intracranial damage is damage that occurs immediately at the moment of the traumatic event and is caused by mechanical trauma:

(a) Static loading of the scalp, skull, and intracranial contents, due to squeezing and compression, may lead to injuries of the intracranial contents.

Table 5.12 Dynamic loading and head injuries [380, 573]

Mechanism	Intracranial findings
Impact loading	• Injuries of the scalp • Fractures of the calvarium, base, and orofacial bones • Epidural bleeding, at the site of the calvarium fracture, often with mass effect • Subdural bleeding, often not at the site of the calvarium fracture, often with mass effect • Often localized axonal injury
Impulse loading ('inertial trauma')	• Subdural bleeding, often a thin layer over both hemispheres, no or only limited mass effect • Acute encephalopathy • Often diffuse axonal injury
Impact or impulse or both (mechanism undetermined)	• Subarachnoid haemorrhage • Brain contusions and lacerations, incl. related intracerebral haemorrhage • Subdural effusions, incl. haemorrhage

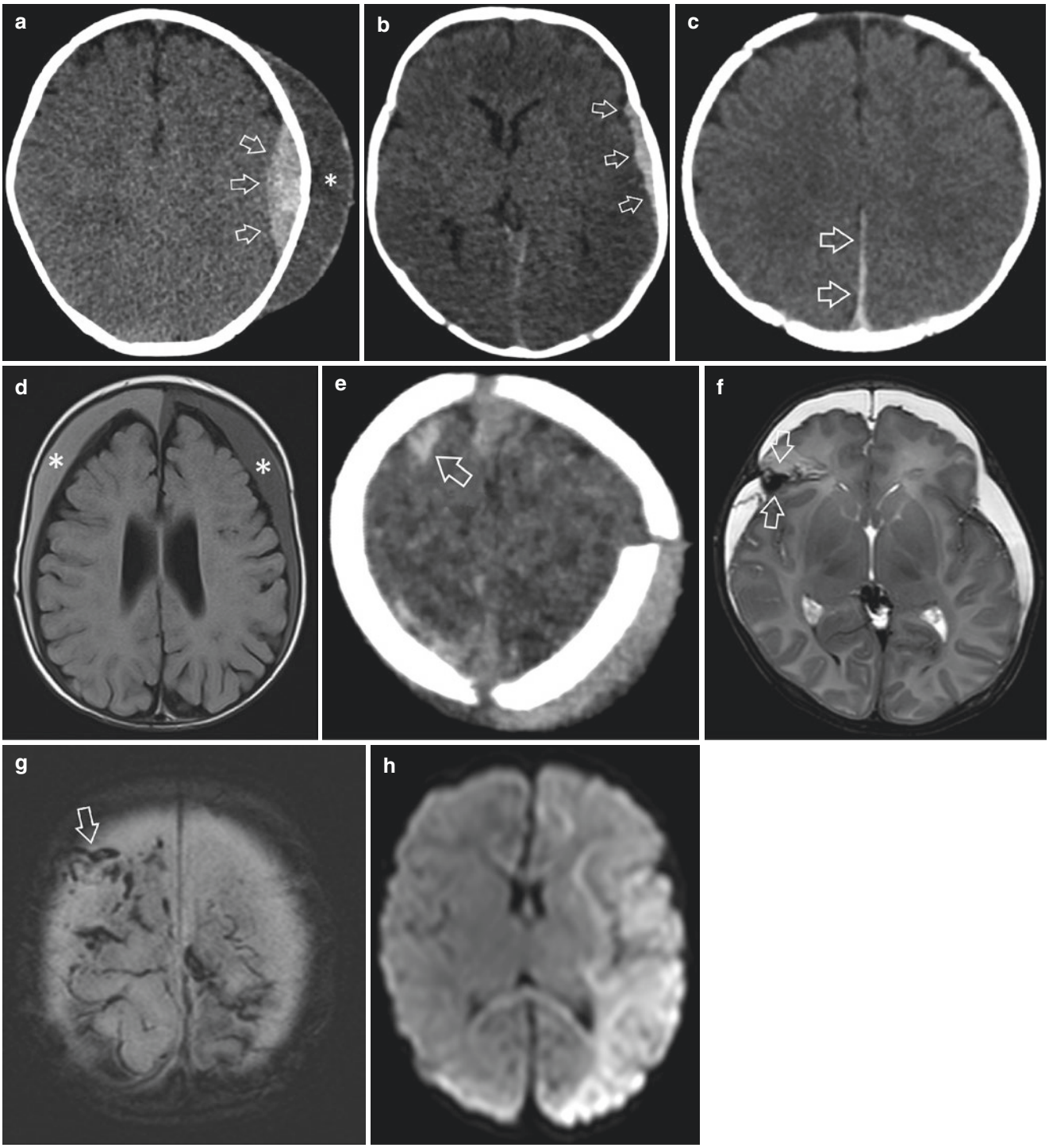


Fig. 5.74 Spectrum of intracranial lesions in accidental and non-accidental injury. (**a**) Epidural hematoma (arrows) and accompanying large cephalhaematoma (asterisk). (**b**) Small acute subdural haemorrhage (arrows). Also, note the subtle loss of grey/white matter differentiation which is suspected of hypoxic-ischemic encephalopathy. (**c**) Small acute subdural hematoma in posterior interhemispheric fissure (arrows). (**d**) Large chronic bilateral subdural hematomas with different signal intensities on this FLAIR MRI image. (**e**) Subarachnoid blood in sulcus (arrow). Note the left parietal skull fracture and soft tissue swelling of the scalp. (**f**) Rupture of the right frontal lobe (between arrows). (**g**) Ruptured and thrombosed bridging veins at the vertex. The arrow depicts the 'tadpole' sign, composed of a thrombosed vein ending in an ellipsoid haemorrhage. (**h**) Large area of diffusion restriction left parieto-occipital on DWI MRI image as an early sign of hypoxic-ischemic encephalopathy

(b) Dynamic impact loading of the scalp, skull, and intracranial contents, due to blunt force trauma (non-penetrating or penetrating) or to sharp force trauma (non-penetrating or penetrating).
(c) Dynamic impulse loading of the scalp, skull, and intracranial contents, due to fast and repetitive movements of the head, without impact (acceleration—deceleration).
(d) Combination of impact and impulse loading.

In Table 5.12 an overview is given of head injuries, due to dynamic impact and dynamic impulse loading.

Clinical symptoms of primary damage may vary from almost non-existing to very severe. The damage can be life threatening or even not compatible with life. Symptoms can occur immediately after the event or may take a while to develop or to be recognized.

Secondary trauma-related intracranial damage is reactive damage that occurs as a (non-specific) response of the brain tissue to a traumatic event, that caused the primary damage. Secondary damage not only occurs in trauma, but may also occur due to diseases, in which the brain is involved. Secondary damage is caused by the occurrence of a lack of oxygen in the brain tissue (hypoxic-ischemic damage).

Clinical symptoms of secondary damage can occur almost immediately or within minutes after the provoking trauma, but can also develop after a time interval of hours to days [574–576].

Oedema is one of the most devastating secondary symptoms. Oedema causes an increase in the volume of the brain tissue. This has a number of negative effects [576]. In the first place, it leads to an increase in intracranial pressure, which can manifest itself with symptoms like vomiting, lowering or loss of consciousness, a bulging fontanel, broadening of cranial sutures, rapid increase of the head circumference, and epileptic seizures. Oedema and the increase of the intracranial pressure can negatively affect the blood supply to (parts of) the brain, which may lead to additional damage to the brain tissue. A lack of supply of oxygen to the brain can also arise as a result of cerebral respiratory problems and a spasm in the vessels in response to bleeding in the head. Oxygen deficiency can lead to hypoxic damage to the axons (hypoxic/ischemic encephalopathy) in addition to traumatic damage to the axons (traumatic encephalopathy). Secondly, the space in the skull is limited. When the volume of the brain increases, there is a risk of impaction of parts of the brain, which in turn causes more damage and increases the risk of death [576].

The severity of the brain damage and the ultimate prognosis is determined by both the damage that results from the primary trauma and the secondary damage that occurs in response to the primary trauma.

Finally, the presence of subdural/subarachnoid blood can lead to irritation of the cerebral cortex, which can trigger epileptic seizures. This may result in a status epilepticus, which may cause a serious disruption of the oxygen supply to the brain, leading to permanent damage to the brain tissue.

In other words, a (serious) disruption of the oxygen supply to the brain can occur as a result of oedema formation, vasospasm, and epileptic seizures/status epilepticus. These reactions can occur separately or in combination.

In Table 5.13, an overview is given of primary and secondary damage in head trauma.

Table 5.13 Primary versus secondary damage in head trauma

Primary injuries	Secondary findings
• Injuries to the scalp • Fractures of the skull and orofacial bones • Intracranial extra-axial and intra-axial bleeding • Axonal injuries (diffuse or localized) due to mechanical trauma	• Oedema • Increased intracranial pressure • Hypoxic-ischaemic encephalopathy • Axonal injuries (diffuse or localized) due to hypoxia/ischaemia • Thrombosis

5.6.4 Manner of Trauma-Related Intracranial Injuries

Trauma-related intracranial injuries can occur before, during, and after birth. In this section, no attention will be given to intracranial injuries sustained in the uterus.

5.6.4.1 Trauma-Related Intracranial Injuries Sustained During Birth

Head trauma can occur during birth, due to compression of the skull and intracranial content, while passing the birthing canal, or due to using a forceps. It may also occur during birth by pulling (traction) on the scalp and skull during a vacuum extraction.

Subdural Haematoma

The most prevalent and most important perinatally acquired intracranial injury, concerning the differential diagnosis of intracranial injuries, sustained after birth, is a subdural haematoma (SDH). In 2008, David stated concerning the occurrence of perinatally acquired SDH the following [577]:

> Thus, without wishing to oversimplify matters, there appear to be two rather distinct recognised categories of subdural haemorrhage occurring at birth:
>
> 1. A common, asymptomatic, self-limiting and rapidly resolving (completely disappeared before 4 weeks of age) category, found in close to 10% of babies.
> 2. A rare, symptomatic category, often requiring treatment.
>
> There are those who wish to speculate that there exists a third and fourth category, as described above, but these remain hypothetical.

3. A large subdural haematoma occurring at birth that causes little or no symptoms at all at first, but is detected much later, either coincidentally or because of chronic and possibly mild symptoms.
4. A small (or larger) but asymptomatic subdural haematoma occurring at birth, re-bleeding, resulting in significant fresh haemorrhage at a later date.

No new scientific evidence has been published since 2008, in which the statements concerning the hypotheses, formulated under 3 and 4 have been proven. However, Rooks et al. showed that SDH is found in almost 50% of all neonates, instead of the 10%, mentioned by David [578].

SDH is less common in caesarean sections than in vaginal birth, with a higher risk in forceps or vacuum delivery than in spontaneous vaginal birth [579–584]. Birth-related SDH has been described in vaginal birth as well as in caesarean sections [578, 584].

Most of the perinatally acquired subdural haematomas are asymptomatic. If a perinatally acquired SDH causes symptoms, in most neonates these will be present within 12–24 h after birth, but it may take up to 4 days [585–588]. In subarachnoid and intraparenchymatous bleeding it may take as long as 11 days, before the first symptoms appear [586].

Retinal Haemorrhages

Retinal findings play an important role in the forensic medical evaluation of head injuries and the radiologist must scrutinize the eye in every child which is suspected of a non-accidental injury. Unfortunately, only extensive retinal haemorrhages and detachments can be seen on MRI (Fig. 5.75a, b) and therefore the role of the radiologist is limited compared to an ophthalmologic examination by an ophthalmologist with experience in non-accidental injury.

RH can be sustained during birth. Birth-related flame/splinter-shaped RH resolves within 2 weeks (usually within 2–3 days) after birth. Larger dot/blot-shaped haemorrhages can be found up to 6 weeks after birth, but usually resolve within 4 weeks [589]. Laghmari et al. (2014) analyzed the presence of retinal haemorrhages in 2031 healthy newborns within 24 h after birth [590]. In over 30% of the newborns RH was found. All children with RH were examined again after 1 and 4 weeks. They found that the RH in two-thirds of the newborns resolved within 1 week and all RH resolved within 4 weeks. Birth-related RH are rarely serious or very extensive [590–593].

It should be mentioned that the correct interpretation of the RH severity (extent) can provide an indication of the circumstances, under which the RH were sustained (accidental or non-accidental). RH severity is defined by the combination of the number and type of RH and the distribution of the haemorrhages in the retina: the degree of spread over the quadrants (top, bottom, left, right), from the posterior pole up to the ora serrata and in relation to the retina (intraretinal, preretinal, and subretinal). The finding of extensive RH in all quadrants, from posterior pole up to the ora serrata, in all layers of the retina and in large numbers (too numerous to count) is much more likely in non-accidental trauma than in accidental trauma [593–595]. It should be noted that the reverse does not apply: a limited number of RH or the absence of retinal haemorrhages does not rule out non-accidental trauma or does not make an accidental event more likely.

5.6.4.2 Trauma-Related Intracranial Injuries Sustained After Birth

Intracranial injuries, as summarized in Table 5.14, and RH can be sustained after birth in accidental or in non-accidental circumstances.

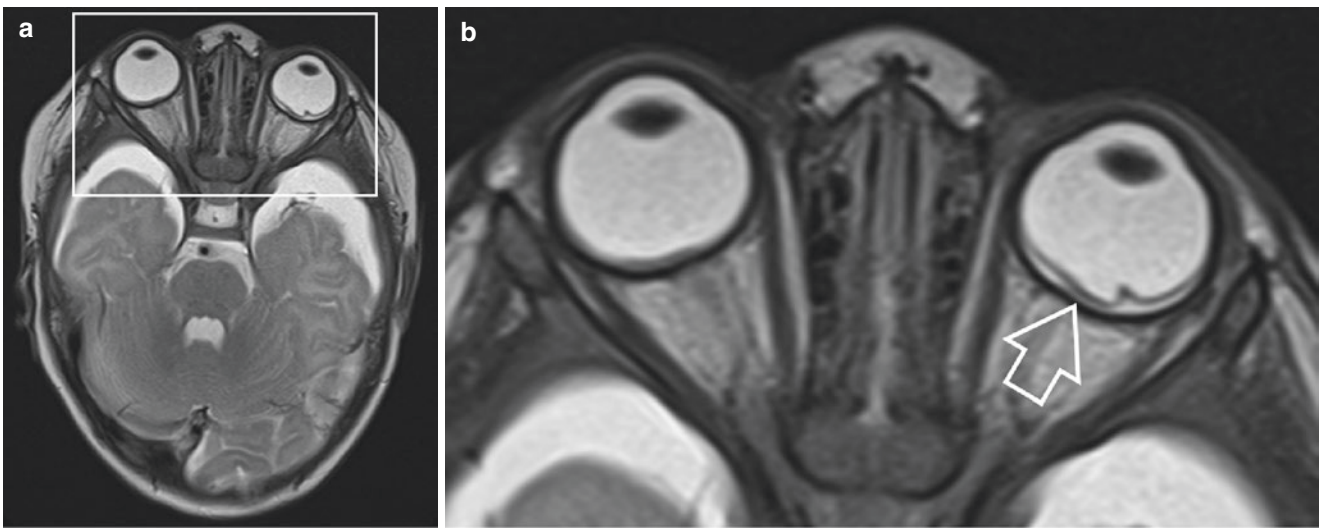


Fig. 5.75 (**a**) T2-weighted MRI of a 3-month-old girl with extensive subdural effusions and extensive retinal lesions. (**b**) Detail of (**a**) showing the retinal detachment of the left eye (arrow). Also, note the "wavy" thickening of the retina in the right eye

Table 5.14 Trauma-related intracranial findings

Intracranial extra-axial bleeding	
• Epidural bleeding	Bleeding between the skull and the dura mater. An epidural haematoma is in fact an abnormal accumulation of blood in a space which in physiological circumstances does not exist
• Subdural bleeding	Bleeding between the dura mater and the arachnoid membrane. A subdural haematoma is in fact an abnormal accumulation of blood in a space which in physiological circumstances does not exist
• Subarachnoid bleeding	Bleeding in the space between the arachnoid membrane and the pia mater surrounding the brain
Intracranial intra-axial bleeding	
• Intracerebral/ intraparenchymal bleeding	Bleeding in the brain tissue
• Intraventricular bleeding	Bleeding in the ventricles
Parenchymatous lesions	
• Axonal injury	Diffuse or focal
• Oedema	

Table 5.15 Manner of intracranial injuries in 345 children [597]

Manner (circumstances, under which the intracranial injuries were sustained		
Natural	6	2%
• Birth trauma		
• Haemorrhagic disease of the newborn		
• Hydrocephalus		
• Epilepsy		
• Haemophilia		
Uncertain	37	11%
Accidental	96	28%
Non-accidental	206	60%
Total	345	

Table 5.16 Age distribution of children with inflicted head injuries versus children with accidental injuries [597]

Age	Under 2 years	Above 2 years	Total
Accidental	75	21	96
Inflicted	180	26	206
Total	255	47	302

It is not possible to differentiate between accidental and non-accidental circumstances, merely on the basis of symptoms, found in children with intracranial injuries. According to Fortin and Stipanic, more than 50% of the symptomatic children with inflicted intracranial injuries initially show non-neurological symptoms, like respiratory dysfunction, hypotonia, nausea, vomiting, apnoea, and irritability [596]. Children may also present with clear neurological symptoms like decreased alertness, convulsions, and coma. This probably also accounts for children with accidental intracranial injuries.

It is also not possible to differentiate between accidental and non-accidental circumstances merely based on the presence of SDH and RH without further analysis of the nature and extent of the findings [593, 595].

According to Kelly et al. (2015), no pathognomonic diagnostic features exist concerning inflicted head trauma [597]. They compared in retrospect the findings in 345 children with head injuries either sustained in accidental or in non-accidental trauma (referred between 1991 and 2010) (Tables 5.15 and 5.16). The ages ranged from 6 days to 13 years, with a median of 7 months. Eighty-five percent of the children were below the age of 2 years and 15% were above that age. In children over 2 years the median age was 3 years with a middle 50% of 2–5 years. Children with inflicted head injuries were younger (median 5 months, middle 50% 2–12 months) compared to children with accidental head injuries (median 10 months, middle 50% 5–20 months). In children above the age of 2 years no differences in age range were found between inflicted and accidental head injuries (median 3 years, middle 50% 2–5 years). Characteristics of particular interest for inflicted head injuries in children under the age of 2 years were no history of trauma (88/98, 90%), no evidence of impact to the head (84/93, 90%), complex skull fractures with intracranial injury (22/28, 79%), subdural haemorrhage (160/179, 89%) and hypoxic-ischaemic injury (38/39, 97%). These characteristics did not differ significantly between children over the age of 2 years with accidental (21/47, 45%) and inflicted head injuries (26/47, 55%). Children over the age of 2 years, who were hospitalized because of inflicted head injuries are usually injured by mechanisms involving impact and should be considered at high risk of death. The mortality rate of inflicted head injuries was higher in children over the age of 2 years (10/26, 38%) than those under that age (19/180, 11%). Kelly et al. found that the probability of inflicted head injuries was similar regardless of socio-economic status or ethnicity.

In 2011 and 2013, the 'Houston Journal of Health Law & Policy' published two extensive analyzes by Narang et al. with regard to the 'Levels of Evidence' in the medical scientific literature on the evidential value of findings in inflicted head injuries and the admissibility of these scientific data in US courts in accordance with the Daubert criteria (Sect. 15.2) [598, 599].

In the second article, Narang et al. used the 2009 criteria of the Oxford Center for Evidence Based Medicine levels of evidence to support the weight of evidence of medical literature in establishing a diagnosis. Based on a very extensive literature review, Narang et al. provided an overview of the (in 2013 current and best) evidence for certain subjects: accidents/injuries due to accidental trauma, coagulation disorders, biomechanics, and hypoxia. In Table 5.17, a short overview of their findings is given. For the full text of this analysis, the reader is referred to the article. According to Narang et al. in children with head trauma, the emphasis in EBM is on the use of the current and best evidence available at the time of the evaluation. This was already stated in 1996 by Sackett, one of the founders of 'Evidence

Table 5.17 Overview of the levels of evidence of findings in head trauma [599]

Topic	Studies	Level
Subdural haematoma		
Trauma is the most common cause	Epidemiologic studies in young children, both prospective and retrospective, from multiple countries	3b
Non-accidental trauma is by far more common	Epidemiology and pathology studies in young children, both prospective and retrospective	2b
SDH being much more common in non-accidental trauma than in accidental trauma is a statistically significant conclusion	Numerous well-designed, prospective clinical studies	2b
Retinal haemorrhages		
Severe RH being much more common in non-accidental trauma than in accidental trauma is a statistically significant conclusion	Numerous well-designed, prospective clinical studies	2b
Severe RH carries a high specificity and positive predictive value for non-accidental trauma	Prospective, validating clinical studies	1b
	Systematic reviews	2a
Severe RH were not found in studies into the relation between RH and increased ICP	Prospective clinical studies and 'systemic reviews'	2b
Severe RH is rarely found in short-distance falls	Prospective and retrospective clinical studies Case descriptions show complicated short-distance falls	2b
Trauma history		
The absence of a trauma history, in the presence of traumatic injuries, holds a high specificity and positive predictive value for non-accidental trauma	Several well-designed, prospective clinical studies	2b
Short-distance falls		
Short falls occurring in objective settings, such as hospitals, have not resulted in subdural haematoma or death	Several consecutive case series.	3b
Severe injuries or death resulting from short falls are rare events	Well-designed, prospective studies and systematic reviews	2a
Clinical variables		
Clinical variables, such as apnoea and severe RH, demonstrate high positive predictive values for non-accidental trauma	Prospective, validating clinical studies	1b
	Systematic reviews	2a
Bleeding disorders		
Most bleeding disorders are rare. The more common bleeding disorders typically are mild, and intracranial haemorrhage resulting from bleeding disorders is a rare complication of the more severe rarer disease	Clinical studies	3b
	Symptom prevalence evidence	1b
Hypoxia		
Macroscopic SDH are not associated with hypoxia	Several well-designed radiology and pathology studies	2b
Severe RHs are not associated with hypoxia	Well-designed clinical studies and animal studies	2b
Adjunct hypotheses of hypoxia (such as 'dysphagia/choking', 'coughing', or 'dural immature vascular plexus') resulting in SDH and/or RH are supported by the lowest levels of evidence-based medicine		4 or 5
Evidence against adjunct hypotheses of hypoxia is much stronger		2b
Biomechanical studies		
Biomechanical studies have shown mixed results as to whether shaking can result in the estimated mechanical forces needed to cause SDH		
Biomechanical studies have shown that RHs can result from shaking		
Biomechanical studies have not shown that neck "failure" must result prior to the estimated forces required for SDH being achieved		

based' thinking in medicine: '*Evidence Based Medicine is the conscientious, explicit, and judicious use of the current, best evidence in making decisions about individual care*' [600].

5.6.4.3 Medical Conditions in the Differential Diagnosis of Physical Findings in Inflicted Head Trauma

There are many lists published of the findings in accidental trauma and in medical disorders in the differential diagnosis of the possible signs, symptoms, and physical findings in inflicted head trauma (Table 5.18). Some of these publications are well researched [601, 602]. Other publications do not differentiate between physical findings in adults and in children [577]. Finally, lists are found, which lack adequate supporting scientific evidence or even present non-existing medical conditions in the differential diagnosis [577, 603–605]. Despite the often missing evidence, most of these disorders can be excluded easily because of differences in clinical, laboratory, or radiological findings (Table 5.19, Fig. 5.76).

5.6.5 Intracranial Injuries and Skull Fractures

5.6.5.1 General Aspects of Intracranial Injuries in Children with Skull Fractures

As already stated in Sect. 5.3.5 calvarium fractures and intracranial injuries are only correlated to a limited degree.

Intracranial injuries can occur without the occurrence of a skull fracture. This applies to accidental as well as to non-accidental circumstances [26, 170, 184, 438, 606].

Table 5.18 Presenting signs, symptoms, and physical findings of inflicted head trauma

Possible presenting signs and physical findings	Possible physical findings
• Neurological symptoms – Decreased alertness – Convulsions – Coma • Non-neurological symptoms – Respiratory dysfunction – Hypotonia – Nausea and vomiting – Apnoea – Irritability	• Subdural haematoma • Retinal haemorrhages • Encephalopathy – Primary (traumatic) – Secondary (hypoxic-ischaemic) • Bruises • Fractures – Rib fractures – Classical metaphyseal lesions – Skull fractures – Other fractures

Calvarium fractures may be present without any intracranial injury, but can also be associated with several intracranial injuries, like epidural or subdural bleeding or contusion of the brain [437]. Dunning et al. calculated that a skull fracture has a relative risk of 6.13 (95% CI 3.35–11.2) for intracranial haemorrhage [607].

Demaerel et al. (2002) found that 45% of infants under the age of 2 years with intracranial injuries did not have a skull fracture and that 56% of children with a skull fracture did not have any intracranial injuries. Finally, they concluded that it is impossible to differentiate between accidental and non-accidental causes based on radiological findings [608].

According to Harwood-Nash et al. (1971), skull fractures with associated subdural haemorrhages are more often seen in older children than in infants [174]. However, the location of the skull fracture is not a good indicator for the location of the subdural haemorrhage. The series of Harwood-Nash showed that subdural haemorrhages were predominantly found contralateral to the fracture [174].

An epidural haematoma may directly result from fracturing of the calvarium. In a fracture of the temporal bone the medial meningeal artery can be damaged, which can lead to an epidural haemorrhage in the temporoparietal area. Epidural haemorrhages are nearly always of arterial origin. In a fracture of the occipital bone, however, the venous sinus may be damaged, leading to a venous epidural haemorrhage in the posterior cranial fossa [1].

Mogbo et al. (1998) did a retrospective study into the relation between skull fractures, visible on radiographs, and intracranial injury in 87 children under the age of 2 years old with a skull fracture [609]. In 67 children no neurological findings were found. In 32 of those children, a CT scan was performed to exclude intracranial injuries. In six children (19%) small focal haemorrhages were found around the fracture. This did not result in an intervention or change in policy. None of the children without neurological symptoms developed neurological complications at a later stage. In 20 of 87 children, acute neurological were found. They all had a CT scan, and in 16 of 20 children pathology was found. Three children had minor pathology, 13 children showed serious pathology which led to neurosurgical intervention in 9 (45%). In 15 children with acute neurological pathology further examination was performed within the scope of child protection proceedings. Based on these findings, 13 of them were placed into care. According to Mogbo et al., a CT scan is indicated in case of neurological symptoms and there is no indication for a CT scan based solely on the presence of a skull fracture because there is no direct correlation between skull fractures and clinically significant intracranial injuries.

This is in line with a prospective study by Lloyd et al. of 883 children (mean age 7 years) with head injury who either

Table 5.19 Overview of the differentiating findings in the most commonly mentioned medical conditions in the differential diagnosis of inflicted head

Medical condition	Differentiating findings
Coagulation disorders	• Clinical signs and symptoms, physical findings • Laboratory findings, specific for different coagulation disorders
• Vitamin K-deficiency	• Laboratory findings: at least 4 times prolonged PT and aPTT
• Immune-related thrombocytopenia	• Laboratory findings: extremely low number of thrombocytes
Metabolic disorders	• Clinical signs and symptoms • Physical findings • Laboratory findings • Neuroimaging (CT/MRI of the brain)
• Glutaric Aciduria type 1	• Part of the neonatal screening (in most countries) • First clinical signs and symptoms are usually above the age of 5–6 months • Specific findings on neuroimaging • Limited number of retinal haemorrhages, limited to the posterior pole
• Menkes syndrome	• First clinical signs and symptoms are usually above the age of several months • Specific findings on neuroimaging and on skeletal imaging • No retinal haemorrhages
Congenital malformations of intracranial vessels, e.g. aneurysm or arteriovenous malformation	• Clinical signs and symptoms • Specific findings on neuroimaging
Osteogenesis imperfecta (‘brittle bone disease’)	• Clinical signs and symptoms • Physical findings • Radiological findings • Laboratory findings
Infectious disorders	• Clinical signs and symptoms, e.g. fever • Physical findings, e.g. rash • Laboratory findings

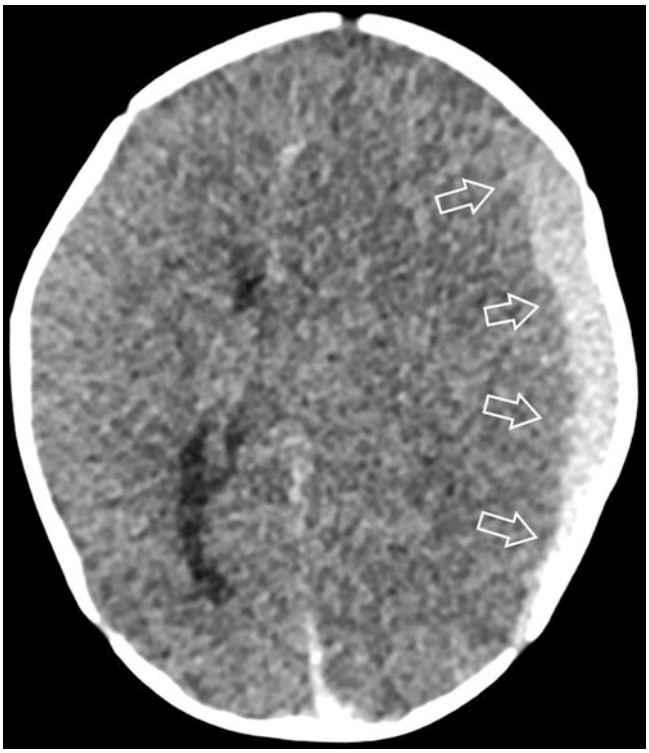


Fig. 5.76 Large acute subdural haemorrhage with midline shift. In this neonate vitamin K deficiency was the cause of the haemorrhage

had a skull fracture on radiographs or were admitted to the hospital [610]. The presence of neurological abnormalities had a sensitivity for identification of intracranial injury of 91% (21 of 23) and a negative predictive value of 97%. The corresponding values for skull fracture on radiography were 65% (15 of 23) and 83%. Four children died, of whom only one had a skull fracture. The authors conclude that a skull radiograph is not a reliable predictor of intracranial injury.

Gruskin and Schutzman (1999) performed a retrospective study into the predictors of complications in skull-/brain trauma in 278 infants under the age of 2 years, presenting at the emergency department of an academic hospital [611]. They concluded that clinical signs and symptoms were not suitable as predictors for skull fractures and/or intracranial injury in children <2 years. Also, they found three characteristics to identify children that are at low risk for complications:

- A fall of less than 1 m
- No neurological symptoms in the medical history
- No abnormalities of the scalp at physical examination

5.6.5.2 Intracranial Injuries in Children with Linear Skull Fractures

Schutzman and Greenes (2001) found intracranial injuries in approximately 15–30% of children with linear fractures of the calvarium [171].

Erlichman et al. (2010) evaluated the findings in 114 children with a diagnosis of a linear skull fracture, due to a minor head trauma, and compared these with the findings in a control group of 125 children without the diagnosis [612]. Twenty-five percent of the children with a linear fracture had an intracranial haemorrhage, compared to only 11% in the control group. All the intracranial haemorrhages were small. In none of the children a neurosurgical intervention was indicated.

Arrey et al. (2015) evaluated the findings in 326 children (median age 19 months; range 2 weeks to 15 years) with isolated, linear, and non-displaced skull fractures [613]. Exclusion criteria were an open or comminuted fracture, intracranial haemorrhage, more than 1 skull fracture, or pneumocephalus. Forty-five children had an altered mental status or loss of consciousness by history. There were no children with neurological deficits on examination, and no children were in need of a neurosurgical intervention.

Bressan et al. (2018) evaluated the findings in 6646 children under the age of 18 years with isolated, linear, and non-displaced skull fractures (i.e. without traumatic intracranial injury on neuroimaging) (587 studies of which 21 were included) [614]. Only one child needed an emergency neurosurgical intervention. No child died, 569 children had repeated neuroimaging. In six of these children, new evidence of an intracranial haemorrhage were found. In none of these children neurosurgery was needed. According to the authors, children with mental status changes, additional injuries, or possible non-accidental trauma may require observation.

5.6.5.3 Intracranial Injuries in Children with Depressed Skull Fractures

Intracranial injuries are found in approximately 30% of children with a depressed fracture [171, 391]. The deeper the fracture, the higher the chance that dura and brain tissue have been damaged. Besides intracranial haemorrhages, compression of the underlying brain tissue, laceration of the brain parenchyma and intraparenchymal bone fragments may occur in depressed fractures [391, 615].

References

1. Atabaki SM (2007) Pediatric head injury. Pediatr Rev 28:215–224
2. Hymel KP, Bandak FA, Partington MD, Winston K (1998) Abusive head trauma? A biomechanics-based approach. Child Maltreat 3:116–128
3. Ommaya AK, Gennarelli TA (1974) Cerebral concussion and traumatic unconsciousness. Correlation of experimental and clinical observations of blunt head injuries. Brain 97:633–654
4. Caffey J (1946) Multiple fractures in the long bones of infants suffering from chronic subdural hematoma. Am J Roentgenol Radium Therapy, Nucl Med 56:163–173
5. Cameron JM, Johnson HR, Camps FE (1966) The battered child syndrome. Med Sci Law 6:2–21

6. Cairns AM, Mok JY, Welbury RR (2005) Injuries to the head, face, mouth and neck in physically abused children in a community setting. Int J Paediatr Dent 15:310–318
7. Baetz K, Sledziewski W, Margetts D, Koren L, Levy M, Pepper R (1977) Recognition and management of the battered child syndrome. J Dent Assoc S Afr 32:13–18
8. Becker DB, Needleman HL, Kotelchuck M (1978) Child abuse and dentistry: orofacial trauma and its recognition by dentists. J Am Dent Assoc 97:24–28
9. da Fonseca MA, Feigal RJ, ten Bensel RW (1992) Dental aspects of 1248 cases of child maltreatment on file at a major county hospital. Pediatr Dent 14:152–157
10. Jessee SA, Rieger M (1996) A study of age-related variables among physically abused children. ASDC J Dent Child 63:275–280
11. Malecz RE (1979) Child abuse, its relationship to pedodontics: a survey. ASDC J Dent Child 46:193–194
12. Naidoo S (2000) A profile of the oro-facial injuries in child physical abuse at a children's hospital. Child Abuse Negl 24:521–534
13. Needleman HL (1986) Orofacial trauma in child abuse: types, prevalence, management, and the dental profession's involvement. Pediatr Dent 8:71–80
14. O'Neill JA Jr, Meacham WF, Griffin JP, Sawyers JL (1973) Patterns of injury in the battered child syndrome. J Trauma 13:332–339
15. Skinner AE, Castle RL (1969) 78 battered children: a retrospective study. National Society for the Prevention of Cruelty to Children, London
16. Tate RJ (1971) Facial injuries associated with the battered child syndrome. Br J Oral Surg 9:41–45
17. Jessee SA (1995) Physical manifestations of child abuse to the head, face and mouth: a hospital survey. ASDC J Dent Child 62:245–249
18. Sanger RG (1984) Oral facial injuries in physical abuse. In: Sanger R, Brose D (eds) Clinical management of child abuse and neglect. Quintessence, Chicago, pp 37–41
19. Bilo RAC, Oranje AP (2013) Afwijkingen in het hoofd-halsgebied [Traumatic lesions in the head and neck region]. In: van de Putte EM, Lukassen IMA, Russel-Kampschoer IM, Teeuw AH (eds) Medisch handboek kindermishandeling Bohn Stafleu van Loghum, Houten, pp 119–128
20. Vadiakas G, Roberts MW, Dilley DC (1991) Child abuse and neglect: ethical and legal issues for dentistry. J Mass Dent Soc 40:13–15
21. Harris CM (2013) Scalp anatomy. https://emedicine.medscape.com/article/834808-overview. Accessed 31 Aug 2021
22. Kim D, Taragin B (2009) Subgaleal hematoma presenting as a manifestation of Factor XIII deficiency. Pediatr Radiol 39:622–624
23. Lavelle JM, Shaw KN (1998) Evaluation of head injury in a pediatric emergency department: pretrauma and posttrauma system. Arch Pediatr Adolesc Med 152:1220–1224
24. Greenes DS, Schutzman SA (1997) Infants with isolated skull fracture: what are their clinical characteristics, and do they require hospitalization? Ann Emerg Med 30:253–259
25. Chan BS, Walker PJ, Cass DT (1989) Urban trauma: an analysis of 1,116 paediatric cases. J Trauma 29:1540–1547
26. DiMaio VJ, DiMaio D (2001) Trauma to the skull and brain: craniocerebral injuries. In: DiMaio VJ, DiMaio D (eds) Forensic pathology. CRC Press, London, pp 147–185
27. Batalis NI (2016) Forensic autopsy of blunt force trauma. https://emedicine.medscape.com/article/1680107-overview. Accessed 12 May 2021
28. Lancon JA, Haines DE, Parent AD (1998) Anatomy of the shaken baby syndrome. Anat Rec 253:13–18
29. Leung AK, Kao CP (1999) Extensive mongolian spots with involvement of the scalp. Pediatr Dermatol 16:371–372
30. Thakur BK, Kaplan AP (1996) Recurrent "unexplained" scalp swelling in an eighteen-month-old child: an atypical presentation of angioedema causing confusion with child abuse. J Pediatr 129:163–165
31. Mandavia DP, Newton EJ, Demetriades D (2003) Head injury. In: Mandavia DP, Newton EJ, Demetriades D (eds) Color atlas of emergency trauma. Cambridge University Press, Cambridge, pp 1–32
32. Bullock R, Graham DI (1997) Non-penetrating injuries of the head. In: Cooper GJ, Dudley HAF, Gann S, Gann DS, Little RA, Maynard RL (eds) Scientific foundations of trauma. Butterworth Heinemann, Oxford, pp 101–126
33. Lee AGF (2017) A scalp lesion in a newborn baby—case report. https://medicinetoday.com.au/dermatology-quiz/scalp-lesion-newborn-baby. Accessed 12 May 2021
34. Hughes CA, Harley EH, Milmoe G, Bala R, Martorella A (1999) Birth trauma in the head and neck. Arch Otolaryngol Head Neck Surg 125:193–199
35. Teng FY, Sayre JW (1997) Vacuum extraction: does duration predict scalp injury? Obstet Gynecol 89:281–285
36. Dessole S, Cosmi E, Balata A, Uras L, Caserta D, Capobianco G, Ambrosini G (2004) Accidental fetal lacerations during cesarean delivery: experience in an Italian level III university hospital. Am J Obstet Gynecol 191:1673–1677
37. Esposito C, Escolino M, Paternoster M, Buccelli C, Graziano V, Falco M, Alicchio F, Cerulo M, Settimi A, Savanelli A (2015) Fetal laceration during caesarean section and its medico-legal sequelae. Med Sci Law 55:97–101
38. Matsubara S, Usui R, Koike Y, Gomi A (2013) Birth injury after cesarean section at 24 weeks of gestation: a large scalp laceration. Arch Gynecol Obstet 287:617–618
39. Okaro JM, Anya SE (2004) Accidental incision of the fetus at caesarian section. Niger J Med 13:56–58
40. Mathur R, Marcus S (2011) Scalp laceration in a newborn due to cervical sutures. Arch Dis Child Fetal Neonatal Ed 96:F342
41. Mayr JM, Seebacher U, Schimpl G, Fiala F (1999) Highchair accidents. Acta Paediatr (Oslo, Norway : 1992) 88:319–322
42. Alias A, Krishnapillai R, Teng HW, Abd Latif AZ, Adnan JS (2005) Head injury from fan blades among children. Asian J Surg 28:168–170
43. Agrawal A, Jajoo SN, Joharapurkar SR (2008) Scalp avulsion injuries. JPMA 58:528
44. Borman H, Kocabalkan O, Ozgür F, Gürsu G (1997) A health hazard for female adolescent farmers: scalp avulsion. J Adolesc Health 20:2
45. Caldwell EH (1976) Complete scalp avulsion. Arch Surg 111:159–161
46. Güven E, Başaran K, Meyzin I, Keklik B, Emekli U (2011) Replantation of scalp avulsion following a go-kart accident: a case report. Ulus Travma Acil Cerrahi Derg = Turk J Trauma Emerg Surg 17:177–179
47. Livaoğlu M, Uraloglu M, Imamoğlu Y, Altun EM, Karaçal N (2016) Microsurgical replantation of two consecutive traumatic total scalp avulsions. J Craniofac Surg 27:e767–e768
48. Yin JW, Matsuo JM, Hsieh CH, Yeh MC, Liao WC, Jeng SF (2008) Replantation of total avulsed scalp with microsurgery: experience of eight cases and literature review. J Trauma 64:796–802
49. Ng ZY, Eberlin KR, Lin T, Masiakos PT, Cetrulo CL Jr (2017) Reconstruction of pediatric scalp avulsion injuries after dog bites. J Craniofac Surg 28:1282–1285
50. Mason AC, Zabel DD, Manders EK (2000) Occult craniocerebral injuries from dog bites in young children. Ann Plast Surg 45:531–534
51. Mitchell RB, Nañez G, Wagner JD, Kelly J (2003) Dog bites of the scalp, face, and neck in children. Laryngoscope 113:492–495

52. Atton AV, Tunnessen WW Jr (1990) Alopecia in children: the most common causes. Pediatr Rev 12:25–30
53. Castelo-Soccio L (2014) Diagnosis and management of alopecia in children. Pediatr Clin N Am 61:427–442
54. Cortés GA, Mardones VF, Zemelman DV (2015) Caracterización de las causas de alopecia infantil [Aetiology of childhood alopecia]. Rev Chil Pediatr 86:264–269
55. Cranwell W, Sinclair R (2018) Common causes of paediatric alopecia. Aust J Gen Pract 47:692–696
56. Phillips JH 3rd, Smith SL, Storer JS (1986) Hair loss. Common congenital and acquired causes. Postgrad Med 79:207–215
57. Hantash BM, Rashid RM, Schwartz RA (2010) Scarring alopecia. https://emedicine.medscape.com/article/1073559-overview. Accessed 12 May 2021
58. Bilo RAC, Oranje AP (2006) Physiological habits, self-mutilation and factitious disorders. In: Harper C, Oranje AP, Prose N (eds) Pediatric dermatology. Blackwell Scientific, Malden, pp 2096–2109
59. Hantash BM, Schwartz RA (2003) Traction alopecia in children. Cutis 71:18–20
60. Oranje AP, Peereboom-Wynia JD, De Raeymaecker DM (1986) Trichotillomania in childhood. J Am Acad Dermatol 15:614–619
61. Saraswat A (2005) Child abuse and trichotillomania. BMJ 330:83–84
62. Williams J (2003) Evaluation of signs and symptoms: bruises. In: Strachan Peterson M, Durfee M, Coulter K (eds) Child abuse and neglect: guidelines for identification, assessment and case management. Volcano Press, Volcano, pp 23–33
63. Seifert D, Püschel K (2006) Subgaleal hematoma in child abuse. Forensic Sci Int 157:131–133
64. Deridder CA, Berkowitz CD (2013) A toddler with vomiting, abdominal pain, and alopecia. Pediatr Emerg Care 29:1114–1115
65. Tedeschi C (1977) The wound—assessment by organ systems, I The head and spine. In: Tedeschi CG, Eckert WG, Tedeschi LG (eds) Forensic medicine. Saunders, Philadelphia, pp 29–75
66. Helfer RE, Slovis TL, Black M (1977) Injuries resulting when small children fall out of bed. Pediatrics 60:533–535
67. Lyons TJ, Oates RK (1993) Falling out of bed: a relatively benign occurrence. Pediatrics 92:125–127
68. Minns RA, Busutttil A (2005) Shaken baby syndrome: theoretical and evidential controversies. J R Coll Physicians Edinb 35:5–15
69. Nimityongskul P, Anderson LD (1987) The likelihood of injuries when children fall out of bed. J Pediatr Orthop 7:184–186
70. Tarantino CA, Dowd MD, Murdock TC (1999) Short vertical falls in infants. Pediatr Emerg Care 15:5–8
71. Warrington SA, Wright CM (2001) Accidents and resulting injuries in premobile infants: data from the ALSPAC study. Arch Dis Child 85:104–107
72. Peters ML, Starling SP, Barnes-Eley ML, Heisler KW (2008) The presence of bruising associated with fractures. Arch Pediatr Adolesc Med 162:877–881
73. Metz JB, Otjen JP, Perez FA, Done SL, Brown ECB, Wiester RT, Jenny C, Ganti S, Feldman KW (2020) Fracture-associated bruising and soft tissue swelling in young children with skull fractures: how sensitive are they to fracture presence? Pediatr Emerg Care. 37(12):e1392–e1396
74. Starling SP, Sirotnak AP, Heisler KW, Barnes-Eley ML (2007) Inflicted skeletal trauma: the relationship of perpetrators to their victims. Child Abuse Negl 31:993–999
75. Burns EC, Grool AM, Klassen TP, Correll R, Jarvis A, Joubert G, Bailey B, Chauvin-Kimoff L, Pusic M, McConnell D, Nijssen-Jordan C, Silver N, Taylor B, Osmond MH (2016) Scalp hematoma characteristics associated with intracranial injury in pediatric minor head injury. Acad Emerg Med Off J Soc Acad Emerg Med 23:576–583
76. Bilo RAC, Robben SGF, van Rijn RR (2010) Head. In: Bilo RAC, Robben SGF, van Rijn RR (eds) Forensic aspects of pediatric fractures; differentiating accidental trauma from child abuse. Springer, Cham, pp 15–48
77. Gebremariam A (1999) Subgaleal haemorrhage: risk factors and neurological and developmental outcome in survivors. Ann Trop Paediatr 19:45–50
78. Dutta S, Singh A, Narang A (2004) Subgaleal hematoma and seven exchange transfusions. Indian Pediatr 41:267–270
79. Vu TT, Guerrera MF, Hamburger EK, Klein BL (2004) Subgaleal hematoma from hair braiding: case report and literature review. Pediatr Emerg Care 20:821–823
80. Kuban K, Winston K, Bresnan M (1983) Childhood subgaleal hematoma following minor head trauma. Am J Dis Child 137:637–640
81. Edmondson SJ, Ramman S, Hachach-Haram N, Bisarya K, Fu B, Ong J, Akhavani M (2016) Hair today; Scalped tomorrow: massive subgaleal haematoma following sudden hair pulling in an adolescent in the absence of haematological abnormality or skull fracture. J Craniofac Surg 27:1261–1262
82. Kirkpatrick JS, Gower DJ, Chauvenet A, Kelly DL Jr (1986) Subgaleal hematoma in a child, without skull fracture. Dev Med Child Neurol 28:511–514
83. Levkoff AH, Macpherson RI, Wood BP (1992) Radiological case of the month. Unrecognized subaponeurotic hemorrhage. Am J Dis Child 146:833–834
84. Antón J, Pineda V, Martin C, Artigas J, Rivera J (1999) Posttraumatic subgaleal hematoma: a case report and review of the literature. Pediatr Emerg Care 15:347–349
85. Benaron DA (1993) Subgaleal hematoma causing hypovolemic shock during delivery after failed vacuum extraction: a case report. J Perinatol 13:228–231
86. Bofill JA, Martin JN (2008) Operative vaginal delivery. In: Gibbs RS, Danforth DN, Karlan BY, Haney AF (eds) Danforth's obstetrics and gynecology. Lippincott Williams Wilkins, Philadelphia, pp 462–490
87. Davis DJ (2001) Neonatal subgaleal hemorrhage: diagnosis and management. CMAJ 164:1452–1453
88. Laroia N (2008) Birth trauma. https://emedicine.medscape.com/article/980112-overview. Accessed 12 May 2021
89. Karcioglu ZA, Hoehn ME, Lin YP, Walsh J (2008) Ocular involvement after subgaleal hematoma. J AAPOS 12:521–523
90. Nichter LS, Bolton LL, Reinisch JF, Sloan GM (1988) Massive subgaleal hematoma resulting in skin compromise and airway obstruction. J Trauma 28:1681–1683
91. Takano I, Suzuki K, Sugiura Y, Suzuki R, Nagaishi M, Tanaka Y, Hyodo A (2015) A case of subgaleal hematoma with exophthalmos and diplopia. No Shinkei Geka 43:727–731
92. Barry J, Fridley J, Sayama C, Lam S (2015) Infected subgaleal hematoma following blunt head trauma in a child: case report and review of the literature. Pediatr Neurosurg 50:223–228
93. Onyeama CO, Lotke M, Edelstein B (2009) Subgaleal hematoma secondary to hair braiding in a 31-month-old child. Pediatr Emerg Care 25:40–41
94. Malek AM, Barnett FH, Schwartz MS, Scott RM (1997) Spontaneous rapid resolution of an epidural hematoma associated with an overlying skull fracture and subgaleal hematoma in a 17-month-old child. Pediatr Neurosurg 26:160–165
95. Neely JC 2nd, Jones BV, Crone KR (2008) Spontaneous extracranial decompression of epidural hematoma. Pediatr Radiol 38:316–318
96. Chida K, Yukawa H, Mase T, Endo H, Ogasawara K (2011) Spontaneous slow drainage of epidural hematoma into the subgaleal space through a skull fracture in an infant—case report. Neurol Med Chir 51:854–856

97. Yaka E, Pekdemir M, Kama A, Sarısoy HT, Yılmaz S (2014) Asymptomatic chronic epidural hematoma in a child as a result of extracranial decompression. J Emerg Med 46:482–485
98. Yamada SM, Tomita Y, Murakami H, Nakane M (2015) Delayed post-traumatic large subgaleal hematoma caused by diastasis of rhomboid skull suture on the transverse sinus. Childs Nerv Syst 31:621–624
99. Assad M, Spaight M, Sink D, Martin J (2018) Early recognition and management of fetal head trauma with massive subgaleal hemorrhage. J Neonatal-Perinatal Med 11:433–438
100. Shimokaze T, Itani Y, Shibasaki J (2011) Direct hyperbilirubinemia caused by severe subgaleal hemorrhage with ischemic liver injury. Pediatr Int 53:1082–1084
101. Ekéus C, Wrangsell K, Penttinen S, Åberg K (2018) Neonatal complications among 596 infants delivered by vacuum extraction (in relation to characteristics of the extraction). J Matern Fetal Neonatal Med 31:2402–2408
102. Santín-Amo JM, Gelabert-González M, Villa-Fernández JM, Castro-Bouzas D, Serramito-García R, García-Allut A (2011) Hematoma subgaleal crónico en un lactante. Presentación de un caso [Chronic subgaleal hematoma in a child. Case report]. Neurocirugia (Astur) 22:261–263
103. Fuijkschot J, Antonius T, Meijers PW, Vrancken S (2008) De subgaleale bloeding bij de neonatus; een potentieel levensbedreigende extracraniële bloeding [Neonatal subgaleal haemorrhage; a potential life-threatening extracranial haemorrhage]. Ned Tijdschr Geneeskd 152:96–100
104. Liu LY, Antaya RJ (2017) Neonatal subgaleal hematoma from trauma during vaginal delivery without instrument use. Pediatr Dermatol 34:e40–e41
105. Swanson AE, Veldman A, Wallace EM, Malhotra A (2012) Subgaleal hemorrhage: risk factors and outcomes. Acta Obstet Gynecol Scand 91:260–263
106. Thorup L, Koch KU (2013) Fatalt forløb ved neonatalt subgalealt hæmatom efter vakuumekstraktion [Neonatal subgaleal haemorrhage causing fatal course after vacuum-assisted extraction]. Ugeskr Laeger 175:34–35
107. Kilani RA, Wetmore J (2006) Neonatal subgaleal hematoma: presentation and outcome—radiological findings and factors associated with mortality. Am J Perinatol 23:41–48
108. Fareeduddin R, Schifrin BS (2008) Subgaleal hemorrhage after the use of a vacuum extractor during elective cesarean delivery: a case report. J Reprod Med 53:809–810
109. Smith A, Kandamany N, Okafor I, Robinson I, Foran A, McNamara R (2016) Delayed infant subaponeurotic (subgaleal) fluid collections: a case series of 11 infants. J Emerg Med 50:881–886
110. Baker JC, Smith KD, Gupta A (2016) Subgaleal hematoma from hair-combing. J Oral Maxillofac Surg 74:e89–e90
111. Falvo CE, San Filippo JA, Vartany A, Osborn EH (1981) Subgaleal hematoma from hair combing. Pediatrics 68:583–584
112. Palmer KM, Olan WJ, Vezina LG, Dubovsky EC (1998) Scalp hematomas in children after hair braiding. Emerg Radiol 5:176–179
113. Faber MM (1976) Massive subgaleal hemorrhage: a hazard of playground swings. Clin Pediatr (Phila) 15:384–385
114. Cooling DS, Viccellio P (1991) Massive subgaleal hematoma following minor head trauma. J Emerg Med 9(Suppl 1):33–35
115. Haidar-El-Atrache S, Agarwal R, Sivaswamy L (2017) An unusual cause of a scalp mass. J Pediatr 181:325–325.e321
116. De Oliveira SE (2008) Massive subgaleal hematoma. J Trauma 65:963
117. Kichari JR, Gielkens H (2013) Massive traumatic subgaleal haematoma. Emerg Med J 30:344
118. Ota FS (2002) Head trauma and hemorrhage. Case based pediatrics for medical students and residents. Department of Pediatrics, University of Hawaii John A. Burns School of Medicine
119. Hamlin H (1968) Subgaleal hematoma caused by hair-pull. JAMA 204:339
120. Shamji S, Jacoby JL (2015) Massive subgaleal hematoma and clinical suspicion of child abuse. The. J Am Osteopath Assoc 115:58. quiz 59-60
121. Yip CC, McCulley TJ, Kersten RC, Kulwin DR (2003) Proptosis after hair pulling. Ophthalmic Plast Reconstr Surg 19:154–155
122. Fujisawa H, Yonaha H, Oka Y, Uehara M, Nagata Y, Kajiwara K, Fujii M, Kato S, Akimura T, Suzuki M (2005) A marked exophthalmos and corneal ulceration caused by delayed massive expansion of a subgaleal hematoma. Childs Nerv Syst 21:489–492
123. Fox SM (2019) Subgaleal hematoma from hair braiding. https://pedemmorsels.com/subgaleal-hematoma-from-hair-braiding/. Accessed 31 Aug 2021
124. Raffini L, Tsarouhas N (2004) Subgaleal hematoma from hair braiding leads to the diagnosis of von Willebrand disease. Pediatr Emerg Care 20:316–318
125. Prakash S (1990) Bilateral proptosis from a subgaleal hematoma. J Neurosurg 72:835
126. Khare M, Kumar V, Marwah S, Nigam AS, Buxi G (2016) Dysfibrinogenemia with subgaleal hematoma: an unusual presentation. Indian J Hematol Blood Transfus 32:239–241
127. Sharma R, Raj R, Gupta LN (2014) Massive hemopericranium following trivial trauma in a child with hypofibrinogenemia: a case report and review of the literature. Indian J Neurosurg 3:166–168
128. Pomeranz AJ, Ruttum MS, Harris GJ (1995) Subgaleal hematoma with delayed proptosis and corneal ulceration. Ann Emerg Med 26:752–754
129. Jenkins TL, Zheng CX, Murchison AP, Bilyk JR (2017) Orbital compartment syndrome following post-traumatic subgaleal hematoma. Ophthalmic Plast Reconstr Surg 33:e33–e36
130. Alcover Bloch E, Jordán García I, Quintillá Martínez JM, Rodríguez Miguélez JM, Figueras Aloy J (1999) Hematoma subgaleal en un recién nacido afecto de hemofilia grave [Subgaleal hematoma in a newborn infant with severe hemophilia]. An Esp Pediatr 51:287–289
131. Balliu Badia PR, Alomar Ribas A, Ciria Calavia LM, Forner Sánchez N, Simonet Salas JM (1997) Hemorragia subgaleal como debut clínico de hemofilia A en un neonato [Subgaleal hemorrhage as the initial clinical sign of hemophilia A in a neonate]. An Esp Pediatr 47:421–423
132. Chia CC, Huang SC (2008) Neonatal coagulopathy presents as unusual and severe subgaleal hematomas after vacuum delivery. Taiwan J Obstet Gynecol 47:435–437
133. Cohen DL (1978) Neonatal subgaleal hemorrhage in hemophilia. J Pediatr 93:1022–1023
134. Radovanović T, Spasojević S, Stojanović V, Doronjski A (2016) Severe neonatal subgaleal hemorrhage as the first presentation of hemophilia A. Srp Arh Celok Lek 144:204–206
135. Talar T, Pacześniak U, Nowiczewski M, Kostrzewska M, Leszczyńska K, Gulczyńska E (2015) Subgaleal hematoma as a perinatal presentation of rare hematologic problems in newborns. Own experience. Dev Period Med 19:277–282
136. Guirgis MF, Segal WA, Lueder GT (2002) Subperiosteal orbital hemorrhage as initial manifestation of Christmas disease (factor IX deficiency). Am J Ophthalmol 133:584–585
137. Wetzel EA, Kingma PS (2012) Subgaleal hemorrhage in a neonate with factor X deficiency following a non-traumatic cesarean section. J Perinatol 32:304–305
138. Natarajan MS, Prabhu K, Braganza A, Chacko AG (2011) Posttraumatic subgaleal and orbital hematoma due to factor XIII deficiency. J Neurosurg Pediatr 7:213–217
139. Ryan CA, Gayle M (1992) Vitamin K deficiency, intracranial hemorrhage, and a subgaleal hematoma: a fatal combination. Pediatr Emerg Care 8:143–145

140. Borensztajn DM, Jansen S, Lopriore E, Boersma B (2010) Trombocytopenie bij twee pasgeborenen: onverwacht ernstige complicaties bij aterme neonaten [Thrombocytopenia in two newborn babies. Unexpected serious complications in full-term babies]. Ned Tijdschr Geneeskd 154:A1922
141. Winkelhorst D, Kamphuis MM, de Kloet LC, Zwaginga JJ, Oepkes D, Lopriore E (2016) Severe bleeding complications other than intracranial hemorrhage in neonatal alloimmune thrombocytopenia: a case series and review of the literature. Transfusion 56:1230–1235
142. Hutspardol S, Chuansamrit A, Soisamrong A (2010) Spontaneous subgaleal hemorrhage in a girl with impaired adrenaline-induced platelet aggregation. J Med Assoc Thai = Chotmaihet thangphaet 93:625–628
143. Stalder MW, Dorafshar AH, Redett RJ (2011) Calcified subgaleal hematoma with secondary cranial deformity in a patient with Kasabach-Merritt phenomenon. J Craniofac Surg 22:208–211
144. Modanlou H, Hutson S, Merritt AT (2016) Early blood transfusion and resolution of disseminated intravascular coagulation associated with massive subgaleal hemorrhage. Neonatal Netw 35:37–41
145. Dahdaleh NS, Lindley TE, Kirby PA, Oya H, Howard MA 3rd (2009) A "neurosurgical crisis" of sickle cell disease. J Neurosurg Pediatr 4:532–535
146. Al-Tonbary YA, Mansour A, Fouda A (2010) Acute myeloid leukemia presenting with a large subgaleal hematoma. Hematol Oncol Stem Cell Ther 3:51–52
147. Greco F, Fiumara A, Sorge G, Pavone L (2008) Subgaleal hematoma in a child with Sturge-Weber syndrome: to prevent stroke-like episodes, is treatment with aspirin advisable? Childs Nerv Syst 24:1479–1481
148. Nicholson L (2007) Caput succedaneum and cephalohematoma: the cs that leave bumps on the head. Neonatal Netw 26:277–281
149. Raines DA, Jain S (2019) Cephalohematoma. https://www.ncbi.nlm.nih.gov/books/NBK470192/?report=reader. Accessed 12 May 2021
150. Aguas J, Conde C, Fructuoso GG, Mondelo FJ, Ferrer E (1995) Giant cephalhematoma in a 15-year-old boy. Unilateral amaurosis as the main complication. Surg Neurol 43:363–366
151. Harouna YD, Gamatie Y (2000) Volumineux céphalhématome spontané chez un enfant de 13 ans [Spontaneous giant cephalohematoma in a 13-year-old child]. Med Trop (Mars) 60:369–371
152. Fujiwara K, Saito K, Ebina T (2002) Bilateral cephalhematomas in a juvenile—case report. Neurol Med Chir 42:547–549
153. Maruki C, Nakajima M, Tsunoda A, Ebato M, Ikeya F (2003) A case of giant expanding cephalhematoma: does the administration of blood coagulation factor XIII reverse symptoms? Surg Neurol 60:138–141. discussion 141
154. Murphy A, Gaillard F (2020) Orbital blowout fracture. https://radiopaedia.org/articles/orbital-blowout-fracture-1?lang=us. Accessed 31 Aug 2021
155. Daglioglu E, Okay O, Hatipoglu HG, Dalgic A, Ergungor F (2010) Spontaneous resolution of calcified cephalhematomas of infancy: report of two cases. Turk Neurosurg 20:96–99
156. Wong CS, Cheah FC (2012) Cephalhematoma infected by *Escherichia coli* presenting as an extensive scalp abscess. J Pediatr Surg 47:2336–2340
157. Krishnan P, Karthigeyan M, Salunke P (2017) Ossified cephalhematoma: an unusual cause of calvarial mass in infancy. J Pediatr Neurosci 12:64–66
158. Guclu B, Yalcinkaya U, Kazanci B, Adilay U, Ekici MA (2012) Diagnosis and treatment of ossified cephalhematoma. J Craniofac Surg 23:e505–e507
159. Tandon V, Garg K, Mahapatra AK (2013) 'Double skull' appearance due to calcifications of chronic subdural hematoma and cephalhematoma: a report of two cases. Turk Neurosurg 23:815–817
160. Yoon SD, Cho BM, Oh SM, Park SH (2013) Spontaneous resorption of calcified cephalhematoma in a 9-month-old child: case report. Childs Nerv Syst 29:517–519
161. Zakanj Z (2014) Prijelom lubanjskih kostiju i kefalhematom u novorodenceta—prikaz bolesnika [Skull fracture and cephalhematoma in a newborn—a case report]. Lijec Vjesn 136:335–338
162. Zimmermann P, Duppenthaler A (2016) Infected cephalhaematoma in a five-week-old infant—case report and review of the literature. BMC Infect Dis 16:636
163. Abdullah WZ, Ismail R, Nasir A, Mohamad N, Hassan R (2013) Developmental haemostasis for factor V and factor VIII levels in neonates: a case report of spontaneous cephalhaematoma. Fetal Pediatr Pathol 32:77–81
164. Salek M, Price J, Vaidya R, Richardson M (2018) Hemophilia A presenting with cephalohematoma in a newborn. Pediatrics 142:225
165. Tubbs RS, Bosmia AN, Cohen-Gadol AA (2012) The human calvaria: a review of embryology, anatomy, pathology, and molecular development. Childs Nerv Syst 28:23–31
166. McGrath A, Taylor RS (2019) Pediatric skull fractures. https://www.ncbi.nlm.nih.gov/books/NBK482218/. Accessed 31 Aug 2021
167. Wikipedia Contributors (2021) Neoteny in humans. https://en.wikipedia.org/w/index.php?title=Neoteny_in_humans&oldid=1038304628. Accessed 1 Sept 2021
168. Franken EA Jr, Smith JA (1975) Roentgenographic evaluation of infant and childhood trauma. Pediatr Clin N Am 22:301–315
169. Royal College of Paediatrics and Child Health (RCPCH) (2017) Child protection evidence—systematic review on fractures. https://www.rcpch.ac.uk/resources/child-protection-evidence--fractures. Accessed 31 Aug 2021
170. Ibrahim NG, Wood J, Margulies SS, Christian CW (2012) Influence of age and fall type on head injuries in infants and toddlers. Int J Dev Neurosci 30:201–206
171. Schutzman SA, Greenes DS (2001) Pediatric minor head trauma. Ann Emerg Med 37:65–74
172. Jennett B (1976) Some medicolegal aspects of the management of acute head injury. Br Med J 1:1383–1385
173. Webber RL, Folio J (1976) Radiographic detectability of occipital and temporal-parietal fractures induced in cadaver heads. J Trauma 16:115–124
174. Harwood-Nash DC, Hendrick EB, Hudson AR (1971) The significance of skull fractures in children. A study of 1,187 patients. Radiology 101:151–156
175. Bullock R, Teasdale GM (1990) Surgical management of traumatic intracranial haematomas. In: Braakmann R (ed) Handbook of clinical neurology. Elsevier Science, Cambridge
176. Ford LE, McLaurin RL (1963) Mechanisms of extradural hematomas. J Neurosurg 20:760–769
177. Weber W (1984) Experimentelle Untersuchungen zu Schadelbruchverletzungen des Sauglings [Experimental studies of skull fractures in infants]. Z Rechtsmed 92:87–94
178. Evans FG, Lissner HR (1956) Engineering aspects of fractures. Clin Orthop 8:310–322
179. King J, Diefendorf D, Apthorp J, Negrete VF, Carlson M (1988) Analysis of 429 fractures in 189 battered children. J Pediatr Orthop 8:585–589
180. Kleinman PK, Marks SC Jr, Richmond JM, Blackbourne BD (1995) Inflicted skeletal injury: a postmortem radiologic-histopathologic study in 31 infants. AJR Am J Roentgenol 165:647–650
181. Merten DF, Radlowski MA, Leonidas JC (1983) The abused child: a radiological reappraisal. Radiology 146:377–381
182. Ibrahim NG, Margulies SS (2010) Biomechanics of the toddler head during low-height falls: an anthropomorphic dummy analysis. J Neurosurg Pediatr 6:57–68

183. Gallagher SS, Finison K, Guyer B, Goodenough S (1984) The incidence of injuries among 87,000 Massachusetts children and adolescents: results of the 1980-81 Statewide Childhood Injury Prevention Program Surveillance System. Am J Public Health 74:1340–1347
184. Kleinman PK, Barnes PD (1998) Head trauma. In: Kleinman PK (ed) Diagnostic imaging of child abuse. Mosby, St Louis, pp 285–342
185. Kraus JF, Fife D, Cox P, Ramstein K, Conroy C (1986) Incidence, severity, and external causes of pediatric brain injury. Am J Dis Child 140:687–693
186. Garza-Mercado R (1982) Intrauterine depressed skull fractures of the newborn. Neurosurgery 10:694–697
187. Morgan JA, Marcus PS (2010) Prenatal diagnosis and management of intrauterine fracture. Obstet Gynecol Surv 65:249–259
188. Alexander E Jr, Davis CH Jr (1969) Intra-uterine fracture of the infant's skull. J Neurosurg 30:446–454
189. Dupuis O, Silveira R, Dupont C, Mottolese C, Kahn P, Dittmar A, Rudigoz RC (2005) Comparison of "instrument-associated" and "spontaneous" obstetric depressed skull fractures in a cohort of 68 neonates. Am J Obstet Gynecol 192:165–170
190. Beyers N, Moosa A, Bryce RL, Kent A (1978) Depressed skull fracture in the newborn. A report of 3 cases. S Afr Med J = Suid-Afrikaanse tydskrif vir geneeskunde 54:830–832
191. Preston D, Jackson S, Gandhi S (2015) Non-traumatic depressed skull fracture in a neonate or 'ping pong' fracture. BMJ Case Rep 2015:bcr2014207077
192. Brodhurst BE (1860) Cases of intra-uterine fracture with observations to show the analogy between fracture in utero and congenital distorsion. Med Chir Trans 43:115–126
193. Guha-Ray DK (1976) Intrauterine spontaneous depression of fetal skull: a case report and review of literature. J Reprod Med 16:321–324
194. Basaldella L, Marton E, Bekelis K, Longatti P (2011) Spontaneous resolution of atraumatic intrauterine ping-pong fractures in newborns delivered by cesarean section. J Child Neurol 26:1449–1451
195. Ilhan O, Bor M, Yukkaldiran P (2018) Spontaneous resolution of a 'ping-pong' fracture at birth. BMJ Case Rep 2018:bcr2018226264
196. Rugolotto S, Grippaldi E, Sidoti G, Padovani EM (2007) Intrauterine depressed skull fracture with spontaneous resolution. Pediatr Med Chir 29:47–49
197. Steinbok P (1989) Intrauterine depressed skull fracture. Pediatr Neurosci 15:317
198. Kilgarriff D, Brannick S, Daly E, Ring C (2021) NAI: never actually injured. BMJ Case Rep 14:e240302
199. Veeravagu A, Azad TD, Jiang B, Edwards MSB (2018) Spontaneous intrauterine depressed skull fractures: report of 2 cases requiring neurosurgical intervention and literature review. World Neurosurg 110:256–262
200. Fantacci C, Massimi L, Capozzi D, Romano V, Ferrara P, Chiaretti A (2015) 'Spontaneous' ping-pong fracture in newborns: case report and review of the literature. Signa Vitae 10:103–109
201. Brinton JH (1884) Report of two cases of intrauterine fracture, with remarks on this condition. Trans Am Surg Assoc 2:425–443
202. Lunney G (1886) Case report. Accident to a pregnant woman, resulting in fracture of the skull of the fetus. Med Rec NY 29:359–360
203. Potter EL (1952) Pathology of the fetus and the newborn. Year Book Medical, Chicago
204. Smith RR (1913) Intrauterine fracture: report of a case and a review of the literature. Surg Gynecol Obstet 17:346–349
205. Ben-Ari Y, Merlob P, Hirsch M, Reisner SH (1986) Congenital depression of the neonatal skull. Eur J Obstet Gynecol Reprod Biol 22:249–255
206. Härtl R, Ko K (1996) In utero skull fracture: case report. J Trauma 41:549–552
207. Loire M, Barat M, Mangyanda Kinkembo L, Lenhardt F, M'Buila C (2017) Spontaneous ping-pong parietal fracture in a newborn. Arch Dis Child Fetal Neonatal Ed 102:F160–f161
208. Tayeh C, Bali B, Milad N, Najjar M (2016) Congenital depression of the skull in a neonate. BMJ Case Rep 2016:bcr2016215437
209. Gallo P, Mazza C, Sala F (2010) Intrauterine head stab wound injury resulting in a growing skull fracture: a case report and literature review. Childs Nerv Syst 26:377–384
210. Moscote Salazar LR, Alcalá-Cerra G, Castellar Leones SM, Gutiérrez Paternina JJ (2012) Traumatismo encefalocraneano fetal por vía penetrante transvaginal [Transvaginal penetrating fetal head injury]. Arch Argent Pediatr 110:e99–e102
211. Tattoli L, Di Vella G, Solarino B (2017) A case of intrauterine lethal fetal injury after attempted suicide of the mother. Forensic Sci Int 280:e1–e5
212. Baker DP (1982) Trauma in the pregnant patient. Surg Clin North Am 62:275–289
213. Esposito TJ (1994) Trauma during pregnancy. Emerg Med Clin North Am 12:167–199
214. Rothenberger D, Quattlebaum FW, Perry JF Jr, Zabel J, Fischer RP (1978) Blunt maternal trauma: a review of 103 cases. J Trauma 18:173–179
215. Bowdler N, Faix RG, Elkins T (1987) Fetal skull fracture and brain injury after a maternal automobile accident. A case report. J Reprod Med 32:375–378
216. Evrard JR, Sturner WQ, Murray EJ (1989) Fetal skull fracture from an automobile accident. Am J Forensic Med Pathol 10:232–234
217. Breysem L, Cossey V, Mussen E, Demaerel P, Van de Voorde W, Smet M (2004) Fetal trauma: brain imaging in four neonates. Eur Radiol 14:1609–1614
218. Stafford PA, Biddinger PW, Zumwalt RE (1988) Lethal intrauterine fetal trauma. Am J Obstet Gynecol 159:485–489
219. Weigel B (1977) Intrauterine fetale Schädelverletzungen bei Unfällen in der Schwangerschaft [Intrauterine fetal skull injuries from an accident during pregnancy]. Zentralbl Gynakol 99:498–506
220. Sadro CT, Zins AM, Debiec K, Robinson J (2012) Case report: lethal fetal head injury and placental abruption in a pregnant trauma patient. Emerg Radiol 19:175–180
221. Avenarius S, Föhe K, Schultz H, Canzler E, Wood BP (1999) Radiological case of the month. Intrauterine stab wound to the head of a 29-week fetus. Arch Pediatr Adolesc Med 153:1103–1104
222. Schultz H, Bretschneider S, Lamme W, Minda R, Canzler E (1993) Intrauterine Messerstichverletzung des Kopfes bei einem Feten in der 29. Schwangerschaftswoche [Intrauterine stab injury with a knife in the head of a fetus in the 29th week of pregnancy]. Kinderarztl Prax 61:215–218
223. Grubb DK (1992) Nonsurgical management of penetrating uterine trauma in pregnancy: a case report. Am J Obstet Gynecol 166:583–584
224. Plotkin H (2004) Syndromes with congenital brittle bones. BMC Pediatr 4:16
225. Bar-Yosef O, Polak-Charcon S, Hoffman C, Feldman ZP, Frydman M, Kuint J (2008) Multiple congenital skull fractures as a presentation of Ehlers-Danlos syndrome type VIIC. Am J Med Genet A 146a:3054–3057
226. Solomons J, Coucke P, Symoens S, Cohen MC, Pope FM, Wagner BE, Sobey G, Black R, Cilliers D (2013) Dermatosparaxis (Ehlers-Danlos type VIIC): prenatal diagnosis following a previous pregnancy with unexpected skull fractures at delivery. Am J Med Genet A 161a:1122–1125
227. Rubin A (1964) Birth injuries: incidence, mechanisms and end results. Obstet Gynecol 23:218–221
228. Bhat BV, Kumar A, Oumachigui A (1994) Bone injuries during delivery. Indian J Pediatr 61:401–405

229. Camus M, Lefebvre G, Veron P, Darbois Y (1985) Traumatismes obstétricaux du nouveau-né. Enquête rétrospective à propos de 20409 naissances [Obstetrical injuries of the newborn infant. Retrospective study apropos of 20,409 births]. J Gynecol Obstet Biol Reprod (Paris) 14:1033–1043
230. Groenendaal F, Hukkelhoven C (2007) Botbreuken bij voldragen pasgeborenen [Fractures in full-term neonates]. Ned Tijdschr Geneeskd 151:424
231. Simonson C, Barlow P, Dehennin N, Sphel M, Toppet V, Murillo D, Rozenberg S (2007) Neonatal complications of vacuum-assisted delivery. Obstet Gynecol 109:626–633
232. Heise RH, Srivatsa PJ, Karsell PR (1996) Spontaneous intrauterine linear skull fracture: a rare complication of spontaneous vaginal delivery. Obstet Gynecol 87:851–854
233. Nadas S, Gudinchet F, Capasso P, Reinberg O (1993) Predisposing factors in obstetric fractures. Skelet Radiol 22:195–198
234. Vanhaesebrouck PJ, Poffyn A, Camaert J, De Baets F, Van de Velde E, Thiery M (1990) Leptomeningeal cyst and vacuum extraction. Acta Paediatr Scand 79:232–233
235. Hansen KN, Pedersen H, Petersen MB (1987) Growing skull fracture—rupture of coronal suture caused by vacuum extraction. Neuroradiology 29:502
236. Hickey K, McKenna P (1996) Skull fracture caused by vacuum extraction. Obstet Gynecol 88:671–673
237. Rupp W, Ropohl D, Bohnert M (2005) Zur Differenzialdiagnose von traumatischen Schädelbefunden bei Säuglingen: Residuen nach Vakuumextraktion [Differential diagnosis of traumatic skull findings in infants: residues after vacuum extraction]. Arch Kriminol 215:70–76
238. Alexander JM, Leveno KJ, Hauth J, Landon MB, Thom E, Spong CY, Varner MW, Moawad AH, Caritis SN, Harper M, Wapner RJ, Sorokin Y, Miodovnik M, O'Sullivan MJ, Sibai BM, Langer O, Gabbe SG (2006) Fetal injury associated with cesarean delivery. Obstet Gynecol 108:885–890
239. Gresham EL (1975) Birth trauma. Pediatr Clin N Am 22:317–328
240. Kendall N, Woloshin H (1952) Cephalhematoma associated with fracture of the skull. J Pediatr 41:125–132
241. Gonzalez Tortosa J, Martínez-Lage JF, Poza M (2004) Bitemporal head crush injuries: clinical and radiological features of a distinctive type of head injury. J Neurosurg 100:645–651
242. López-Guerrero AL, Martínez-Lage JF, González-Tortosa J, Almagro MJ, García-Martínez S, Reyes SB (2012) Pediatric crushing head injury: biomechanics and clinical features of an uncommon type of craniocerebral trauma. Childs Nerv Syst 28:2033–2040
243. Duhaime AC, Eppley M, Margulies S, Heher KL, Bartlett SP (1995) Crush injuries to the head in children. Neurosurgery 37:401–406. discussion 407
244. Partrick DA, Bensard DD, Moore EE, Partington MD, Karrer FM (1998) Driveway crush injuries in young children: a highly lethal, devastating, and potentially preventable event. J Pediatr Surg 33:1712–1715
245. Prasad MR, Ewing-Cobbs L, Baumgartner J (1999) Crush head injuries in infants and young children neurologic and neuropsychologic sequelae. J Child Neurol 14:496–501
246. Takeshi M, Okuchi K, Nishiguchi T, Seki T, Watanahe T, Ito S, Murao Y (2006) Clinical analysis of seven patients of crushing head injury. J Trauma 60:1245–1249
247. Brison RJ, Pickett W, Berg RL, Linneman J, Zentner J, Marlenga B (2006) Fatal agricultural injuries in preschool children: risks, injury patterns and strategies for prevention. CMAJ 174:1723–1726
248. Hajiaghamemar M, Lan IS, Christian CW, Coats B, Margulies SS (2019) Infant skull fracture risk for low height falls. Int J Legal Med 133:847–862
249. Offiah AC, Hall CM (2009) Skull. In: Offiah AC, Hall CM (eds) Radiological atlas of child abuse. Radcliff Publishing, Oxford, pp 17–44
250. Claydon SM (1996) Fatal extradural hemorrhage following a fall from a baby bouncer. Pediatr Emerg Care 12:432–434
251. Metz JB, Otjen JP, Perez FA, Done SL, Brown ECB, Wiester RT, Jenny C, Ganti S, Feldman KW (2020) Delays in care seeking for young children with accidental skull fractures are common. Acta Paediatr (Oslo, Norway: 1992) 110(6):1890–1894
252. Bechtel K, Stoessel K, Leventhal JM, Ogle E, Teague B, Lavietes S, Banyas B, Allen K, Dziura J, Duncan C (2004) Characteristics that distinguish accidental from abusive injury in hospitalized young children with head trauma. Pediatrics 114:165–168
253. Hughes J, Maguire S, Jones M, Theobald P, Kemp A (2016) Biomechanical characteristics of head injuries from falls in children younger than 48 months. Arch Dis Child 101:310–315
254. Johnson K, Fischer T, Chapman S, Wilson B (2005) Accidental head injuries in children under 5 years of age. Clin Radiol 60:464–468
255. Thomas G, Ward TJ, Guy R, Scot DJ (2005) Should children aged under 1 year presenting with a head injury have a routine skull X-ray. Clin Radiol 90:A41
256. Hobbs CJ (1984) Skull fracture and the diagnosis of abuse. Arch Dis Child 59:246–252
257. Weber W (1985) Zur biomechanischen Fragilität des Säuglingsschädels [Biomechanical fragility of the infant skull]. Z Rechtsmed 94:93–101
258. Samuel N, Jacob R, Eilon Y, Mashiach T, Shavit I (2015) Falls in young children with minor head injury: a prospective analysis of injury mechanisms. Brain Inj 29:946–950
259. Burrows P, Trefan L, Houston R, Hughes J, Pearson G, Edwards RJ, Hyde P, Maconochie I, Parslow RC, Kemp AM (2015) Head injury from falls in children younger than 6 years of age. Arch Dis Child 100:1032–1037
260. Plunkett J (2001) Fatal pediatric head injuries caused by short-distance falls. Am J Forensic Med Pathol 22:1–12
261. Spivack B (2001) Fatal pediatric head injuries caused by short-distance falls. Am J Forensic Med Pathol 22:332–336
262. 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
263. Levin AV (2001) Fatal pediatric head injuries caused by short distance falls. Am J Forensic Med Pathol 22:417–419
264. Schaber B, Hart AP, Armbrustmacher V, Hirsch CS (2002) Fatal pediatric head injuries caused by short distance falls. Am J Forensic Med Pathol 23:101–103. author reply 103-105
265. Lueder GT (2005) Retinal hemorrhages in accidental and nonaccidental injury. Pediatrics 115:192. author reply 192
266. Bechtel K, Stoessel K, Leventhal JM, Ogle E, Teague B, Lavietes S, Banyas B, Allen K, Dziura J, Duncan C (2005) Author reply to Lueder GT. Retinal hemorrhages in accidental and nonaccidental injury. Pediatrics 115:192
267. Wheeler DS, Shope TR (1997) Depressed skull fracture in a 7-month-old who fell from bed. Pediatrics 100:1033–1034
268. Thompson AK, Bertocci G, Rice W, Pierce MC (2011) Pediatric short-distance household falls: biomechanics and associated injury severity. Accid Anal Prev 43:143–150
269. Couper GW, Boddie DE, Eljamel MS, Kaar GF (2000) Unguarded electric plugs cause penetrating head injuries in children. J Accid Emerg Med 17:55
270. Tan MH, Choudhari KA (2003) Penetrating head injury from an electrical plug. Injury 34:950–953
271. López González A, Gutiérrez Marín A, Alvarez Garijo JA, Vila Mengual M (2006) Penetrating head injury in a paediatric patient caused by an electrical plug. Childs Nerv Syst 22:197–200
272. Kanagarajan A, Sgouros S (2007) Unusual penetrating craniocerebral injuries in children from mains plugs. Childs Nerv Syst 23:1181–1183

273. George M, Round J (2006) An Eiffel penetrating head injury. Arch Dis Child 91:416
274. Couper RT, Monkhouse W, Busutil M, Thompson P (1994) Stroller safety. Med J Aust 160:335–338
275. Lee AC, Fong D (1997) Epidural haematoma and stroller-associated injury. J Paediatr Child Health 33:446–447
276. Watson WL, Ozanne-Smith J (1993) The use of child safety restraints with nursery furniture. J Paediatr Child Health 29:228–232
277. Powell EC, Jovtis E, Tanz RR (2002) Incidence and description of stroller-related injuries to children. Pediatrics 110:e62
278. Fowler E, Kobe C, Roberts KJ, Collins CL, McKenzie LB (2016) Injuries associated with strollers and carriers among children in the United States, 1990 to 2010. Acad Pediatr 16:726–733
279. Vilke GM, Stepanski BM, Ray LU, Lutz MW, Murrin PA, Chan TC (2004) 9-1-1 responses for shopping cart and stroller injuries. Pediatr Emerg Care 20:660–663
280. Tripathi M, Tyebally A, Feng JX, Chong SL (2017) A review of stroller-related and pram-related injuries to children in Singapore. Inj Prev 23:60–63
281. Arnholz D, Hymel KP, Hay TC, Jenny C (1998) Bilateral pediatric skull fractures: accident or abuse? J Trauma 45:172–174
282. Centers for Disease Control and Prevention (CDC) (1992) Deaths associated with infant carriers—United States, 1986–1991. MMWR Morb Mortal Wkly Rep 41:271–272
283. Wickham T, Abrahamson E (2002) Head injuries in infants: the risks of bouncy chairs and car seats. Arch Dis Child 86:168–169
284. Greenberg RA, Bolte RG, Schunk JE (2009) Infant carrier-related falls: an unrecognized danger. Pediatr Emerg Care 25:66–68
285. Davis C, Brown S (2000) Penetrating intracranial trauma in an infant secondary to a modified baby bouncer. N Z Med J 113:406
286. Farmakakis T, Alexe DM, Nicolaidou P, Dessypris N, Petridou E (2004) Baby-bouncer-related injuries: an under-appreciated risk. Eur J Pediatr 163:42–43
287. Powell EC, Jovtis E, Tanz RR (2002) Incidence and description of high chair-related injuries to children. Ambul Pediatr 2:276–278
288. Kurinsky RM, Rochette LM, Smith GA (2014) Pediatric injuries associated with high chairs and chairs in the United States, 2003–2010. Clin Pediatr (Phila) 53:372–379
289. American Academy of Pediatrics (AAP) (2006) Shopping-cart-related injuries to children. Committee on Injury, Violence, and Poison Prevention, American Academy of Pediatrics. Pediatrics 118:825–827
290. Martin KJ, Chounthirath T, Xiang H, Smith GA (2014) Pediatric shopping-cart-related injuries treated in US emergency departments, 1990-2011. Clin Pediatr (Phila) 53:277–285
291. Smith GA, Dietrich AM, Garcia CT, Shields BJ (1996) Injuries to children related to shopping carts. Pediatrics 97:161–165
292. Selbst SM, Baker MD, Shames M (1990) Bunk bed injuries. Am J Dis Child 144:721–723
293. Macgregor DM (2000) Injuries associated with falls from beds. Inj Prev 6:291–292
294. Mayr JM, Seebacher U, Lawrenz K, Pesendorfer P, Berghold A, Baradaran S (2000) Bunk beds—a still underestimated risk for accidents in childhood? Eur J Pediatr 159:440–443
295. Belechri M, Petridou E, Trichopoulos D (2002) Bunk versus conventional beds: a comparative assessment of fall injury risk. J Epidemiol Community Health 56:413–417
296. Mack KA, Gilchrist J, Ballesteros MF (2007) Bunk bed-related injuries sustained by young children treated in emergency departments in the United States, 2001–2004, National Electronic Injury Surveillance System—All Injury Program. Inj Prev 13:137–140
297. D'Souza AL, Smith GA, McKenzie LB (2008) Bunk bed-related injuries among children and adolescents treated in emergency departments in the United States, 1990–2005. Pediatrics 121:e1696–e1702
298. McFaull SR, Fréchette M, Skinner R (2012) Emergency department surveillance of injuries associated with bunk beds: the Canadian Hospitals Injury Reporting and Prevention Program (CHIRPP), 1990–2009. Chronic Dis Inj Canada 33:38–46
299. Ono H, Sase T, Takasuna H, Tanaka Y (2019) Playground equipment-related head injuries requiring hospitalization in children. Pediatr Int 61:293–297
300. Illingworth CM, Jay A, Noble D, Collick M (1978) 225 skateboard injuries in children. Clin Pediatr (Phila) 17(781-782):788–789
301. Russell KW, Katz MG, Short SS, Scaife ER, Fenton SJ (2019) Longboard injuries treated at a level 1 pediatric trauma center. J Pediatr Surg 54:569–571
302. American Academy of Pediatrics (AAP) (2001) American Academy of Pediatrics: Falls from heights: windows, roofs, and balconies. Pediatrics 107:1188–1191
303. Stone KE, Lanphear BP, Pomerantz WJ, Khoury J (2000) Childhood injuries and deaths due to falls from windows. J Urban Health 77:26–33
304. Lallier M, Bouchard S, St-Vil D, Dupont J, Tucci M (1999) Falls from heights among children: a retrospective review. J Pediatr Surg 34:1060–1063
305. Mayer L, Meuli M, Lips U, Frey B (2006) The silent epidemic of falls from buildings: analysis of risk factors. Pediatr Surg Int 22:743–748
306. Musemeche CA, Barthel M, Cosentino C, Reynolds M (1991) Pediatric falls from heights. J Trauma 31:1347–1349
307. Vish NL, Powell EC, Wiltsek D, Sheehan KM (2005) Pediatric window falls: not just a problem for children in high rises. Inj Prev 11:300–303
308. Kim KA, Wang MY, Griffith PM, Summers S, Levy ML (2000) Analysis of pediatric head injury from falls. Neurosurg Focus 8:e3
309. Murray JA, Chen D, Velmahos GC, Alo K, Belzberg H, Asensio JA, Demetriades D, Berne TV (2000) Pediatric falls: is height a predictor of injury and outcome? Am Surg 66:863–865
310. Williams RA (1991) Injuries in infants and small children resulting from witnessed and corroborated free falls. J Trauma 31:1350–1352
311. Lehman D, Schonfeld N (1993) Falls from heights: a problem not just in the northeast. Pediatrics 92:121–124
312. Al B, Yildirim C, Coban S (2009) Falls from heights in and around the city of Batman. Ulus Travma Acil Cerrahi Derg = Turk J Trauma Emerg Surg 15:141–147
313. Meller JL, Shermeta DW (1987) Falls in urban children. A problem revisited. Am J Dis Child 141:1271–1275
314. Wang MY, Kim KA, Griffith PM, Summers S, McComb JG, Levy ML, Mahour GH (2001) Injuries from falls in the pediatric population: an analysis of 729 cases. J Pediatr Surg 36:1528–1534
315. Melo JR, Di Rocco F, Lemos-Júnior LP, Roujeau T, Thélot B, Sainte-Rose C, Meyer P, Zerah M (2009) Defenestration in children younger than 6 years old: mortality predictors in severe head trauma. Childs Nerv Syst 25:1077–1083
316. Shields BJ, Burkett E, Smith GA (2011) Epidemiology of balcony fall-related injuries, United States, 1990–2006. Am J Emerg Med 29:174–180
317. Kocak S, Dundar ZD, Yavuz K, Onal MA, Dikmetas C, Girisgin AS, Gul M, Cander B (2012) Etiologic factors in falls from height in pediatric cases. Eur J Trauma Emerg Surg 38:313–317
318. Pérez-Suárez E, Jiménez-García R, Iglesias-Bouzas M, Serrano A, Porto-Abad R, Casado-Flores J (2012) Caídas desde grandes alturas en Pediatría. Epidemiología y evolución de 54 pacientes [Falls from heights in Pediatrics. Epidemiology and evolution of 54 patients]. Med Intensiva 36:89–94
319. Albanese CT, Gardner MJ, Adkins MA, Schall L, Lynch JM (1997) Single rope tree swing injuries among children. Pediatrics 99:548–550

320. Randazzo C, Stolz U, Hodges NL, McKenzie LB (2009) Pediatric tree house-related injuries treated in emergency departments in the United States: 1990–2006. Acad Emerg Med Off J Soc Acad Emerg Med 16:235–242
321. Britton JW (2005) Kids can't fly: preventing fall injuries in children. WMJ 104:33–36
322. Joffe M, Ludwig S (1988) Stairway injuries in children. Pediatrics 82:457–461
323. Chiaviello CT, Christoph RA, Bond GR (1994) Stairway-related injuries in children. Pediatrics 94:679–681
324. Docherty E, Hassan A, Burke D (2010) Things that go bump … bump … bump: an analysis of injuries from falling down stairs in children based at Sheffield Children's Hospital. Emerg Med J 27:207–208
325. Pomerantz WJ, Gittelman MA, Hornung R, Husseinzadeh H (2012) Falls in children birth to 5 years: different mechanisms lead to different injuries. J Trauma Acute Care Surg 73:S254–S257
326. Zielinski AE, Rochette LM, Smith GA (2012) Stair-related injuries to young children treated in US emergency departments, 1999–2008. Pediatrics 129:721–727
327. Pennock AT, Gantsoudes GD, Forbes JL, Asaro AM, Mubarak SJ (2014) Stair falls: caregiver's "missed step" as a source of childhood fractures. J Child Orthop 8:77–81
328. Hibberd O, Nuttall D, Watson RE, Watkins WJ, Kemp AM, Maguire S (2017) Childhood bruising distribution observed from eight mechanisms of unintentional injury. Arch Dis Child 102:1103–1109
329. Lantz PE, Couture DE (2011) Fatal acute intracranial injury, subdural hematoma, and retinal hemorrhages caused by stairway fall. J Forensic Sci 56:1648–1653
330. Greeley CS (2012) Commentary on: Lantz PE, Couture DE. Fatal acute intracranial injury, subdural hematoma, and retinal hemorrhages caused by stairway fall. J Forensic Sci 2011;56(6):1648–53. J Forensic Sci 57:560–561. author reply 562
331. Royal College of Paediatrics and Child Health (RCPCH), Royal College of Ophthalmologists (RCO) (2013) Abusive head trauma and the eye in infancy. https://www.rcophth.ac.uk/wp-content/uploads/2014/12/2013-SCI-292-ABUSIVE-HEAD-TRAUMA-AND-THE-EYE-FINAL-at-June-2013.pdf. Accessed 31 Aug 2021
332. Sims A, Chounthirath T, Yang J, Hodges NL, Smith GA (2018) Infant walker-related injuries in the United States. Pediatrics 142:e20174332
333. Aziz A, McIntyre L, Khazen R (1985) Risks of baby walkers and options for prevention. Can Fam Physician 31:2147–2150
334. Chiaviello CT, Christoph RA, Bond GR (1994) Infant walker-related injuries: a prospective study of severity and incidence. Pediatrics 93:974–976
335. Gleadhill DN, Robson WJ, Cudmore RE, Turnock RR (1987) Baby walkers … time to take a stand? Arch Dis Child 62:491–494
336. Mayr JM, Gaisl M, Purtscher K, Noeres H, Schimpl G, Fasching G (1994) Baby walkers—an underestimated hazard for our children? Eur J Pediatr 153:531–534
337. Sabir H, Mayatepek E, Schaper J, Tibussek D (2008) Baby-walkers: an avoidable source of hazard. Lancet 372:2000
338. Trinkoff A, Parks PL (1993) Prevention strategies for infant walker-related injuries. Public Health Rep (Washington, DC: 1974) 108:784–788
339. American Academy of Pediatrics (AAP) (2001) Injuries associated with infant walkers. Pediatrics 108:790–792
340. Kavanagh CA, Banco L (1982) The infant walker. A previously unrecognized health hazard. Am J Dis Child 136:205–206
341. Wellman S, Paulson JA (1984) Baby walker-related injuries. Clin Pediatr (Phila) 23:98–99
342. Fazen LE 3rd, Felizberto PI (1982) Baby walker injuries. Pediatrics 70:106–109
343. Rieder MJ, Schwartz C, Newman J (1986) Patterns of walker use and walker injury. Pediatrics 78:488–493
344. Coats TJ, Allen M (1991) Baby walker related injuries—a continuing problem. Arch Emerg Med 8:52–55
345. Partington MD, Swanson JA, Meyer FB (1991) Head injury and the use of baby walkers: a continuing problem. Ann Emerg Med 20:652–654
346. Petridou E, Simou E, Skondras C, Pistevos G, Lagos P, Papoutsakis G (1996) Hazards of baby walkers in a European context. Injury Prev 2:118–120
347. Smith GA, Bowman MJ, Luria JW, Shields BJ (1997) Babywalker-related injuries continue despite warning labels and public education. Pediatrics 100:E1
348. Marnewick J, Dansey R, Morreau P, Hamill J (2011) Television tip-overs: the Starship Children's Hospital experience and literature review. Injury 42:534–538
349. Bol O, Cebiçci H, Koyuncu S, Şarlı B, Günay N (2016) A hidden household danger: television. Ulus Travma Acil Cerrahi Derg = Turk J Trauma Emerg Surg 22:265–268
350. Bernard PA, Johnston C, Curtis SE, King WD (1998) Toppled television sets cause significant pediatric morbidity and mortality. Pediatrics 102:E32
351. DiScala C, Barthel M, Sege R (2001) Outcomes from television sets toppling onto toddlers. Arch Pediatr Adolesc Med 155:145–148
352. Jea A, Ragheb J, Morrison G (2003) Television tipovers as a significant source of pediatric head injury. Pediatr Neurosurg 38:191–194
353. Ota FS, Maxson RT, Okada PJ (2006) Childhood injuries caused by falling televisions. Acad Emerg Med Off J Soc Acad Emerg Med 13:700–703
354. Scheidler MG, Shultz BL, Schall L, Vyas A, Barksdale EM Jr (2002) Falling televisions: The hidden danger for children. J Pediatr Surg 37:572–575
355. Yahya RR, Dirks P, Humphreys R, Rutka JT, Taylor M, Drake JM (2005) Children and television tipovers: a significant and preventable cause of long-term neurological deficits. J Neurosurg 103:219–222
356. Cusimano MD, Parker N (2016) Toppled television sets and head injuries in the pediatric population: a framework for prevention. J Neurosurg Pediatr 17:3–12
357. De Roo AC, Chounthirath T, Smith GA (2013) Television-related injuries to children in the United States, 1990–2011. Pediatrics 132:267–274
358. Eren B, Tas A, Guzey FK, Gulec I, Tufan A, Karacan M (2019) Television tip-over related head injuries: a particular type of child neglect. Turk Neurosurg 29:349–354
359. Safe Kids Worldwide (2018) TV and furniture tip-over prevention tips. https://www.safekids.org/sites/default/files/documents/tv_and_furniture_safety_tips_0.pdf. Accessed 31 Aug 2021
360. Befeler AR, Daniels DJ, Helms SA, Klimo P Jr, Boop F (2014) Head injuries following television-related accidents in the pediatric population. J Neurosurg Pediatr 14:414–417
361. Furyk J, Franklin RC, Costello D (2013) Ceiling fan head injury to children in an Australian tropical location. J Paediatr Child Health 49:480–485
362. O'Donnell C (2019) Pediatric ceiling fan-induced head injury. https://radiopaedia.org/cases/paediatric-ceiling-fan-induced-head-injury. Accessed 31 Aug 2021
363. Hoz SS, Dolachee AA, Abdali HA, Kasuya H (2019) An enemy hides in the ceiling; pediatric traumatic brain injury caused by metallic ceiling fan: case series and literature review. Br J Neurosurg 33:360–364
364. Brodeur AE, Monteleone JA (1994) Child maltreatment. A clinical guide and reference. GW Medical, St Louis, p 32

365. Merten DF, Carpenter BL (1990) Radiologic imaging of inflicted injury in the child abuse syndrome. Pediatr Clin N Am 37:815–837
366. Johnstone AJ, Zuberi SH, Scobie WG (1996) Skull fractures in children: a population study. J Accid Emerg Med 13:386–389
367. Loder RT, Bookout C (1991) Fracture patterns in battered children. J Orthop Trauma 5:428–433
368. Leventhal JM, Thomas SA, Rosenfield NS, Markowitz RI (1993) Fractures in young children. Distinguishing child abuse from unintentional injuries. Am J Dis Child 147:87–92
369. Reece RM (2001) Child abuse—medical diagnosis and management. Lippincott Williams & Wilkins, Philadelphia, pp 149–151
370. Merten DF, Osborne D, Radkowski MS, Leonidas JC (1984) Craniocerebral trauma in the child abuse syndrome: radiological observation. Pediatr Radiol 14:272–277
371. Pandya NK, Baldwin K, Wolfgruber H, Christian CW, Drummond DS, Hosalkar HS (2009) Child abuse and orthopaedic injury patterns: analysis at a level I pediatric trauma center. J Pediatr Orthop 29:618–625
372. Kleinman PK, Silvera VM (2015) Abusive head trauma: scalp, subscalp, and cranium. In: Kleinman PK (ed) Diagnostic imaging of child abuse. Cambridge University Press, Cambridge, pp 357–393
373. Hymel KP, Spivack BS (2001) The biomechanics of physical injury. In: Reece RM, Ludwig S (eds) Child abuse—medical diagnosis and management. Lippincott Williams en Wilkins, Philadelphia, pp 1–22
374. Worlock P, Stower M, Barbor P (1986) Patterns of fractures in accidental and non-accidental injury in children: a comparative study. BMJ 293:100–102
375. Kleinman PK, Spevak MR (1992) Soft tissue swelling and acute skull fractures. J Pediatr 121:737–739
376. Ros SP, Cetta F (1992) Are skull radiographs useful in the evaluation of asymptomatic infants following minor head injury? Pediatr Emerg Care 8:328–330
377. Meservy CJ, Towbin R, McLaurin RL, Myers PA, Ball W (1987) Radiographic characteristics of skull fractures resulting from child abuse. AJR Am J Roentgenol 149:173–175
378. Boal DK, Felman AH, Krugman RD (2001) Controversial aspects of child abuse: a roundtable discussion. 43rd annual meeting, Society for Pediatric Radiology. Pediatr Radiol 31:760–774
379. Fenton LZ, Sirotnak AP, Handler MH (2000) Parietal pseudofracture and spontaneous intracranial hemorrhage suggesting nonaccidental trauma: report of 2 cases. Pediatr Neurosurg 33:318–322
380. Graham DI (2000) Closed head injury. In: Purdue BN (ed) Mason JK. The pathology of trauma, Arnold, London, pp 191–210
381. Di Ieva A, Audigé L, Kellman RM, Shumrick KA, Ringl H, Prein J, Matula C (2014) The comprehensive AOCMF classification: skull Base and cranial vault fractures—level 2 and 3 tutorial. Craniomaxillofac Trauma Reconstr 7:S103–S113
382. Wiersema JM, Love JC, Derrick SM, Pinto DC, Donaruma-Kwoh M, Greeley CS (2014) Standardized descriptive method for the anthropological evaluation of pediatric skull fractures. J Forensic Sci 59:1487–1492
383. Donahue DJ, Sanford RA, Muhlbauer MS, Chadduck WM (1995) Cranial burst fracture in infants: acute recognition and management. Childs Nerv Syst 11:692–697
384. Sim SY, Kim HG, Yoon SH, Choi JW, Cho SM, Choi MS (2017) Reappraisal of pediatric diastatic skull fractures in the 3-dimensional CT era: clinical characteristics and comparison of diagnostic accuracy of simple skull X-ray, 2-dimensional CT, and 3-dimensional CT. World Neurosurg 108:399–406
385. Gurdjian ES, Webster JE, Lissner HR (1950) The mechanism of skull fracture. Radiology 54:313–339
386. O'Hara MA (2019) The infant with bilateral skull fractures: diagnostic considerations in consultation with a child abuse pediatrician. J Inj Violence Res 11:15–20
387. Hiss J, Kahana T (1995) The medicolegal implications of bilateral cranial fractures in infants. J Trauma 38:32–34
388. Ilbeygui R, Reiter C (2002) Synopsis und Atlas der Gerichtsmedizin. Facultas, Wien
389. Brittain C, Muthukumar P, Job S, Sanka S (2012) 'Ping pong' fracture in a term infant. BMJ Case Rep 2012:bcr0120125631
390. Mastrapa TL, Fernandez LA, Alvarez MD, Storrs BB, Flores-Urueta A (2007) Depressed skull fracture in Ping Pong: elevation with Medeva extractor. Childs Nerv Syst 23:787–790
391. Erşahin Y, Mutluer S, Mirzai H, Palali I (1996) Pediatric depressed skull fractures: analysis of 530 cases. Childs Nerv Syst 12:323–331
392. Luckett WH (1910) VII. Ping-Pong-Ball indentation of the skull without fracture. Ann Surg 51:518–519
393. Zia Z, Morris AM, Paw R (2007) Ping-pong fracture. Emerg Med J 24:731
394. Howship J (1816) Practical observations in surgery, and morbid anatomy. Longman, Hurst, Rees, Orme, and Brown, London
395. Bir SC, Kalakoti P, Notarianni C, Nanda A (2015) John Howship (1781-1841) and growing skull fracture: historical perspective. J Neurosurg Pediatr 16:472–476
396. Parmar RC, Bavdekar SB (2000) Images in radiology: type III growing skull fracture. J Postgrad Med 46:130–131
397. Lende RA, Erickson TC (1961) Growing skull fractures of childhood. J Neurosurg 18:479–489
398. DesChamps GT Jr, Blumenthal BI (1988) Radiologic seminar CCXLIX: growing skull fractures of childhood. J Miss State Med Assoc 29:16–17
399. Naim Ur R, Jamjoom Z, Jamjoom A, Murshid WR (1994) Growing skull fractures: classification and management. Br J Neurosurg 8:667–679
400. Ramamurthi B, Kalyanaraman S (1970) Rationale for surgery in growing fractures of the skull. J Neurosurg 32:427–430
401. Rinehart GC, Pittman T (1998) Growing skull fractures: strategies for repair and reconstruction. J Craniofac Surg 9:65–72
402. Sener RN (1995) Growing skull fracture in a patient with cerebral hemiatrophy. Pediatr Radiol 25:64–65
403. Kutlay M, Demircan N, Akin ON, Basekim C (1998) Untreated growing cranial fractures detected in late stage. Neurosurgery 43:72–76. discussion 76–77
404. Muhonen MG, Piper JG, Menezes AH (1995) Pathogenesis and treatment of growing skull fractures. Surg Neurol 43:367–372. discussion 372–363
405. Gupta SK, Reddy NM, Khosla VK, Mathuriya SN, Shama BS, Pathak A, Tewari MK, Kak VK (1997) Growing skull fractures: a clinical study of 41 patients. Acta Neurochir 139:928–932
406. Johnson DL, Helman T (1995) Enlarging skull fractures in children. Childs Nerv Syst 11:265–268
407. Pezzotta S, Silvani V, Gaetani P, Spanu G, Rondini G (1985) Growing skull fractures of childhood. Case report and review of 132 cases. J Neurosurg Sci 29:129–135
408. Iyer SG, Saxena P, Kumhar GD (2003) Growing skull fractures. Indian Pediatr 40:1194–1196
409. Arseni C, Ciurea AV (1981) Clinicotherapeutic aspects in the growing skull fracture. A review of the literature. Childs Brain 8:161–172
410. Lye RH, Occleshaw JV, Dutton J (1981) Growing fracture of the skull and the role of computerized tomography. Case report. J Neurosurg 55:470–472
411. Gruber FH (1969) Post-traumatic leptomeningeal cysts. Am J Roentgenol Radium Therapy, Nucl Med 105:305–307
412. Thompson JB, Mason TH, Haines GL, Cassidy RJ (1973) Surgical management of diastatic linear skull fractures in infants. J Neurosurg 39:493–497
413. Locatelli D, Messina AL, Bonfanti N, Pezzotta S, Gajno TM (1989) Growing fractures: an unusual complication of head injuries in pediatric patients. Neurochirurgia (Stuttg) 32:101–104

414. Buckingham MJ, Crone KR, Ball WS, Tomsick TA, Berger TS, Tew JM Jr (1988) Traumatic intracranial aneurysms in childhood: two cases and a review of the literature. Neurosurgery 22:398–408
415. Yamamoto M, Moore MH, Hanieh A (1998) Growing skull fracture after cranial vault reshaping in infancy. J Craniofac Surg 9:73–75
416. Moss SD, Walker ML, Ostergard S, Golembeski D (1990) Intrauterine growing skull fracture. Childs Nerv Syst 6:468–470
417. Hes R, de Jong TH, Paz y Geuze DH, Avezaat CJ (1997) Rapid evolution of a growing skull fracture after vacuum extraction in case of fetal hydrocephalus. Pediatr Neurosurg 26:269–274
418. Papaefthymiou G, Oberbauer R, Pendl G (1996) Craniocerebral birth trauma caused by vacuum extraction: a case of growing skull fracture as a perinatal complication. Childs Nerv Syst 12:117–120
419. Chung BS, Hagan L, Lammle M (2021) Radiological evaluation of the sutures of the skull. In: Turgut M, Shane Tubbs R, Turgut AT, Dumont AS (eds) The sutures of the skull. Springer, Cham, pp 161–170
420. Bang KE, Bordes SJ Jr, Shane Tubbs R (2021) Anatomy of the sutures of the calvaria. In: Turgut M, Shane Tubbs R, Turgut AT, Dumont AS (eds) The sutures of the skull. Springer, Cham, pp 43–52
421. Sanchez T, Stewart D, Walvick M, Swischuk L (2010) Skull fracture vs. accessory sutures: how can we tell the difference? Emerg Radiol 17:413–418
422. Quigley AJ, Stafrace S (2014) Skeletal survey normal variants, artefacts and commonly misinterpreted findings not to be confused with non-accidental injury. Pediatr Radiol 44:82–93. quiz 79–81
423. Idriz S, Patel JH, Ameli Renani S, Allan R, Vlahos I (2015) CT of normal developmental and variant anatomy of the pediatric skull: distinguishing trauma from normality. Radiographics 35:1585–1601
424. Choudhary AK, Jha B, Boal DK, Dias M (2010) Occipital sutures and its variations: the value of 3D-CT and how to differentiate it from fractures using 3D-CT? Surg Radiol Anat 32:807–816
425. Oakes PC, Tubbs RS (2021) Variation in the sutural anatomy of the skull. In: Turgut M, Tubbs RS, Turgut AT, Dumont AS (eds) The sutures of the skull. Springer, Cham, pp 97–113
426. Matsumura G, Uchiumi T, Kida K, Ichikawa R, Kodama G (1993) Developmental studies on the interparietal part of the human occipital squama. J Anat 182(Pt 2):197–204
427. Main C, Fairhurst JJ (2018) Normal variant mimicking a depressed skull fracture. J Forensic Radiol Imaging 12:40–42
428. Muroi A, Enomoto T, Ihara S, Ishikawa E, Inagaki T, Matsumura A (2021) Developmental changes in the occipital cranial sutures of children less than 2 years of age. Childs Nerv Syst 37:567–572
429. Nakahara K, Utsuki S, Shimizu S, Iida H, Miyasaka Y, Takagi H, Oka H, Fujii K (2006) Age dependence of fusion of primary occipital sutures: a radiographic study. Childs Nerv Syst 22:1457–1459
430. Franken EA Jr (1969) The midline occipital fissure: diagnosis of fracture versus anatomic variants. Radiology 93:1043–1046
431. Weir P, Suttner NJ, Flynn P, McAuley D (2006) Normal skull suture variant mimicking intentional injury. BMJ 332:1020–1021
432. Currarino G (1976) Normal variants and congenital anomalies in the region of the obelion. AJR Am J Roentgenol 127:487–494
433. Le Double A-F (1903) Traité des variations des os du crane de Ihomme et de leur signification au point de vue de l'anthropologie zoologique. Vigot Fréres, Paris
434. Eklund MJ, Carver KC, Stalcup ST, Riemer EC, Taylor MA, Hill JG (2016) Atypical accessory intraparietal sutures mimicking complex fractures in a neonate. Clin Imaging 40:806–809
435. Wiedijk JEF, Soerdjbalie-Maikoe V, Maat GJR, Maes A, van Rijn RR, de Boer HH (2016) An accessory skull suture mimicking a skull fracture. Forensic Sci Int 260:e11–e13
436. Sidpra J, Jeelani NUO, Ong J, Birch W, Mankad K (2021) Skull fractures in abusive head trauma: a single centre experience and review of the literature. Childs Nerv Syst 37:919–929
437. Adepoju A, Adamo MA (2017) Posttraumatic complications in pediatric skull fracture: dural sinus thrombosis, arterial dissection, and cerebrospinal fluid leakage. J Neurosurg Pediatr 20:598–603
438. Claudet I, Gurrera E, Honorat R, Rekhroukh H, Casasoprana A, Grouteau E (2013) Accidents domestiques par chute avant l'âge de la marche [Home falls in infants before walking acquisition]. Arch Pediatr 20:484–491
439. 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
440. Cameron JM (1978) Radiological pathological aspects of the battered child syndrome. In: Smith SM (ed) The maltreatment of children. University Park Press, Baltimore, pp 69–81
441. Kleinman PK, Coats B, Silvera VM (2015) Abusive head trauma. In: Kleinman PK (ed) Diagnostic imaging of child abuse. Cambridge University Press, Cambridge, pp 369–370
442. Harper NS, Eddleman S, Shukla K, Narcise MV, Padhye LJ, Peterson LJ, Murati MA, George CLS (2021) Radiologic assessment of skull fracture healing in young children. Pediatr Emerg Care 37:213–217
443. Hobbs CJ, Hanks HGI, Wynne JM (1993) Child abuse and neglect—a clinician's handbook. Churchill Livingstone, London
444. Makkat S, Vandevenne JE, Parizel PM, De Schepper AM (2001) Multiple growing fractures and cerebral venous anomaly after penetrating injuries: delayed diagnosis in a battered child. Pediatr Radiol 31:381–383
445. Miranda P, Vila M, Alvarez-Garijo JA, Perez-Nunez A (2007) Birth trauma and development of growing fracture after coronal suture disruption. Childs Nerv Syst 23:355–358
446. McLaurin RL (1983) Head injury. In: Warner TW (ed) Pediatric neurology. Harper & Row, New York, pp 507–548
447. Perheentupa U, Kinnunen I, Grénman R, Aitasalo K, Mäkitie AA (2010) Management and outcome of pediatric skull base fractures. Int J Pediatr Otorhinolaryngol 74:1245–1250
448. Battle WH (1890) Three lectures on some points relating to injuries to the head. Lancet 1:57–63
449. Qureshi NH, Harsh G (2008) Skull fracture. https://emedicine.medscape.com/article/248108-overview. Accessed 31 Aug 2021
450. Ropper AH, Brown RH (2005) Cranio-cerebral trauma. In: Ropper AH, Brown RH (eds) Adams and Victor's principles of neurology. McGraw-Hill, New York
451. Larner AJ (2006) A dictionary of neurological signs. Springer, Cham
452. Tolias C, Sgouros S (2008) Initial Evaluation and Management of CNS Injury. https://emedicine.medscape.com/article/434261--overview. Accessed 31 Aug 2021
453. Kaldewaij P, Vos PE (2002) Brilhematoom als teken van een voorste-schedelbasis-fractuur. Ned Tijdschr Geneeskd 5:11–12
454. Loonen MCB (1973) Trauma capitis en 'battered child syndrome'. In: Blauw G (ed) Kinderneurologie, capita selecta. Agon Elsevier, Amsterdam, pp 220–239
455. Gumus K (2007) A child with raccoon eyes masquerading as trauma. Int Ophthalmol 27:379–381
456. Alcalá-Galiano A, Arribas-García IJ, Martín-Pérez MA, Romance A, Montalvo-Moreno JJ, Juncos JM (2008) Pediatric facial fractures: children are not just small adults. Radiographics 28:441–461. quiz 618
457. Younes AA, Gottlieb W (2018) Pediatric mandible fractures. https://emedicine.medscape.com/article/872662-overview. Accessed 31 Aug 2021
458. Imahara SD, Hopper RA, Wang J, Rivara FP, Klein MB (2008) Patterns and outcomes of pediatric facial fractures in the United

States: a survey of the National Trauma Data Bank. J Am Coll Surg 207:710–716
459. Allred LJ, Crantford JC, Reynolds MF, David LR (2015) Analysis of pediatric maxillofacial fractures requiring operative treatment: characteristics, management, and outcomes. J Craniofac Surg 26:2368–2374
460. Oppenheimer AJ, Monson LA, Buchman SR (2013) Pediatric orbital fractures. Craniomaxillofac Trauma Reconstr 6:9–20
461. Enlow D (1982) Handbook of facial growth. WB Saunders, Philadelphia
462. McCoy FJ, Chandler RA, Crow ML (1966) Facial fractures in children. Plast Reconstr Surg 37:209–215
463. Hoppe IC, Kordahi AM, Lee ES, Granick MS (2015) Pediatric facial fractures: interpersonal violence as a mechanism of injury. J Craniofac Surg 26:1446–1449
464. Hoppe IC, Kordahi AM, Paik AM, Lee ES, Granick MS (2014) Examination of life-threatening injuries in 431 pediatric facial fractures at a level 1 trauma center. J Craniofac Surg 25:1825–1828
465. Mericli AF, DeCesare GE, Zuckerbraun NS, Kurland KS, Grunwaldt L, Vecchione L, Losee JE (2011) Pediatric craniofacial fractures due to violence: comparing violent and nonviolent mechanisms of injury. J Craniofac Surg 22:1342–1347
466. Owusu JA, Bellile E, Moyer JS, Sidman JD (2016) Patterns of pediatric mandible fractures in the United States. JAMA Facial Plast Surg 18:37–41
467. Gopalakrishnan V, Sahoo NK, Roy ID (2017) Mandibular fracture in a neonate. Int J Oral Maxillofac Surg 46:59–61
468. Kim TW, Seo EW, Song SI (2013) Open reduction and internal fixation of mandibular fracture in an 11-month-old infant: a case report. J Korean Assoc Oral Maxillofac Surg 39:90–93
469. Glazer M, Joshua BZ, Woldenberg Y, Bodner L (2011) Mandibular fractures in children: analysis of 61 cases and review of the literature. Int J Pediatr Otorhinolaryngol 75(1):62–4
470. Atanasov DT, Vuvakis VM (2000) Mandibular fractures in children. A retrospective study. Folia Med (Plovdiv) 42:65–70
471. Smith DM, Bykowski MR, Cray JJ, Naran S, Rottgers SA, Shakir S, Vecchione L, Schuster L, Losee JE (2013) 215 mandible fractures in 120 children: demographics, treatment, outcomes, and early growth data. Plast Reconstr Surg 131:1348–1358
472. Lustmann J, Milhem I (1994) Mandibular fractures in infants: review of the literature and report of seven cases. J Oral Maxillofac Surg 52:240–245. discussion 245–246
473. Schlievert R (2006) Infant mandibular fractures: are you considering child abuse? Pediatr Emerg Care 22:181–183
474. Vasconcelos BC, Lago CA, Nogueira RV, Gondim DG, Brito Filho A (2009) Mandibular fracture in a premature infant: a case report and review of the literature. J Oral Maxillofac Surg 67:218–222
475. Adekeye EO (1980) Pediatric fractures of the facial skeleton: a survey of 85 cases from Kaduna, Nigeria. J Oral Surg 38:355–358
476. Ahluwalia TP, Sandhu SS, Kapila BK (1983) Mandibular fracture in a 4-week old infant (a case report). J Indian Dent Assoc 55:269–271
477. Jaworski S, Dudkiewicz Z (1973) Złamanie porodowe zuchwy u noworodka [Mandibular fracture in the course of labor in a newborn infant]. Pediatr Pol 48:1501–1504
478. Maszkiewicz W, Irzyńska D (1983) Okołoporodowe uszkodzenie zuchwy u wcześniaka [Perinatal mandibular injury in a premature infant]. Wiad Lek 36:1217–1218
479. Scott EP, Rotondo CC (1946) Fracture with osteomyelitis of the mandible in a newborn infant. Am J Dis Child 72:411–414
480. Monks FT (1977) A fractured mandible in the new-born. Br J Oral Surg 14:270–272
481. Serel S, Can Z, Ersoy A, Sen Z (2005) Management of mandibular fracture using open reduction and internal fixation in a neonate: case report. J Oral Maxillofac Surg 63:396–399
482. Priest JH (1989) Treatment of a mandibular fracture in a neonate. J Oral Maxillofac Surg 47:77–81
483. York BV (1970) Management of mandibular fracture in 3-week-old infant: report of case. J Oral Surg 28:857–859
484. Chidzonga MM (1996) Mandibular fracture in a neonate: report of a case. J Oral Maxillofac Surg 54:1452–1454
485. Chidzonga MM (2006) Mandibular fracture in a neonate: report of a case. Int J Oral Maxillofac Surg 35:186–187
486. Knoche JW, LeBlanc KK, King TW, Knox BL (2012) An infant with a unilateral mandibular fracture: when to consider nonaccidental trauma. Clin Pediatr (Phila) 51:404–407
487. Letelier C, León M, Boesing L, Ortega RG, Pantoja R (2016) Infant multiple mandibular fracture: case report. Int J Med Surg Sci 3:82307
488. Siegel MB, Wetmore RF, Potsic WP, Handler SD, Tom LW (1991) Mandibular fractures in the pediatric patient. Arch Otolaryngol Head Neck Surg 117:533–536
489. Alberth M, Szilágyi Z, Póti S, Redl P (1997) Csecsemökorú gyermek állkapocstörése [Mandibular fracture in an infant]. Fogorv Sz 90:49–53
490. Chan YC, Au-Yeung KL (2017) A paediatric case of bilateral mandibular condyle fracture presenting with bloody otorrhoea following trauma. BMJ Case Rep bcr2016218995
491. Whittaker JD (2021) Zygomatic and nasal injury. https://www.rcemlearning.co.uk/reference/zygomatic-and-nasal--injury/#1571913770322-630e9407-0c42. Accessed 31 Aug 2021
492. Noyola-Frías MA, Toranzo FM, Reynoso ER (2011) Facial trauma in neonates: a case report. Revista ADM 68:303–6306
493. DeFazio MV, Fan KL, Avashia YJ, Danton GH, Thaller SR (2013) Fractures of the pediatric zygoma: a review of the clinical trends, management strategies, and outcomes associated with zygomatic fractures in children. J Craniofac Surg 24:1891–1897
494. Hussain K, Wijetunge DB, Grubnic S, Jackson IT (1994) A comprehensive analysis of craniofacial trauma. J Trauma 36:34–47
495. Sims AP (1985) Non-accidental injury in the child presenting as a suspected fracture of the zygomatic arch. Br Dent J 158:292–293
496. Jessee SA (1995) Orofacial manifestations of child abuse and neglect. Am Fam Physician 52:1829–1834
497. Kao R, Campiti VJ, Rabbani CC, Ting JY, Sim MW, Shipchandler TZ (2019) Pediatric midface fractures: outcomes and complications of 218 patients. Laryngoscope Investig Otolaryngol 4:597–601
498. Boyd K, Rizzuto PR (2017) What Is an orbital fracture? https://www.aao.org/eye-health/diseases/what-is-orbital-fracture. Accessed 31 Aug 2021
499. Hatton MP, Watkins LM, Rubin PA (2001) Orbital fractures in children. Ophthalmic Plast Reconstr Surg 17:174–179
500. Wolfsdorf J (2019) Eye injuries and eye socket fracture. https://www.nicklauschildrens.org/conditions/eye-injuries-and-eye-socket-fracture. Accessed 31 Aug 2021
501. Gerber B, Kiwanuka P, Dhariwal D (2013) Orbital fractures in children: a review of outcomes. Br J Oral Maxillofac Surg 51:789–793
502. Joseph JM, Glavas IP (2011) Orbital fractures: a review. Clin Ophthalmol 5:95–100
503. American Association for Pediatric Ophthalmology and Strabismus (AAPOS) (2020) Blowout fracture. https://aapos.org/glossary/blowout-fracture. Accessed 31 Aug 2021
504. Gonzalez MO, Durairaj VD (2010) Indirect orbital floor fractures: a meta-analysis. Middle East Afr J Ophthalmol 17:138–141
505. Langer PD (2020) Orbital floor fractures. https://eyewiki.org/w/index.php?title=Orbital_Floor_Fractures&redirect=no. Accessed 31 Aug 2021
506. Lash E (2019) Orbital floor blowout fracture. http://brownemblog.com/blog-1/2018/12/31/orbital-floor-blowout-fracture. Accessed 31 Aug 2021

507. Kroll AJ, Casten VG (1966) Diseases of the orbit. In: Liebman SD, Gellis SS (eds) The pediatrician's ophthalmology. Mosby, St. Louis, pp 231–250
508. Klenk G, Kovacs A (2003) Blow-out fracture of the orbital floor in early childhood. J Craniofac Surg 14:666–671
509. Grant JH 3rd, Patrinely JR, Weiss AH, Kierney PC, Gruss JS (2002) Trapdoor fracture of the orbit in a pediatric population. Plast Reconstr Surg 109:482–489. discussion 490–485
510. Valente L, Tieghi R, Elia G, Galiè M (2019) Orbital Fractures in Childhood. Ann Maxillofac Surg 9:403–406
511. Wang NC, Ma L, Wu SY, Yang FR, Tsai YJ (2010) Orbital blow-out fractures in children: characterization and surgical outcome. Chang Gung Med J 33:313–320
512. Egbert JE, May K, Kersten RC, Kulwin DR (2000) Pediatric orbital floor fracture : direct extraocular muscle involvement. Ophthalmology 107:1875–1879
513. Pelton RW (2020) Orbital roof fractures. https://eyewiki.aao.org/Orbital_Roof_fractures. Accessed 31 Aug 2021
514. Messinger A, Radkowski MA, Greenwald MJ, Pensler JM (1989) Orbital roof fractures in the pediatric population. Plast Reconstr Surg 84:213–216. discussion 217–218
515. Greenwald MJ, Boston D, Pensler JM, Radkowski MA (1989) Orbital roof fractures in childhood. Ophthalmology 96:491–496. discussion 496–497
516. Koltai PJ, Amjad I, Meyer D, Feustel PJ (1995) Orbital fractures in children. Arch Otolaryngol Head Neck Surg 121:1375–1379
517. Ng JD, Payner TD, Holck DE, Martin RT, Nunery WT (2004) Orbital trauma caused by bicycle hand brakes. Ophthalmic Plast Reconstr Surg 20:60–63
518. Steyn M (2011) Case report: forensic anthropological assessment in a suspected case of child abuse from South Africa. Forensic Sci Int 208:e6–e9
519. Bales CR, Randall P, Lehr HB (1972) Fractures of the facial bones in children. J Trauma 12:56–66
520. Losee JE, Afifi A, Jiang S, Smith D, Chao MT, Vecchione L, Hertle R, Davis J, Naran S, Hughes J, Paviglianiti J, Deleyiannis FW (2008) Pediatric orbital fractures: classification, management, and early follow-up. Plast Reconstr Surg 122:886–897
521. Barh A, Swaminathan M, Mukherjee B (2018) Orbital fractures in children: clinical features and management outcomes. J AAPOS 22:415.e411–415.e417
522. Brannan PA, Kersten RC, Kulwin DR (2006) Isolated medial orbital wall fractures with medial rectus muscle incarceration. Ophthalmic Plast Reconstr Surg 22:178–183
523. Criden MR, Ellis FJ (2007) Linear nondisplaced orbital fractures with muscle entrapment. J AAPOS 11:142–147
524. Kakizaki H, Zako M, Iwaki M, Mito H, Katori N (2005) Incarceration of the inferior oblique muscle branch of the oculomotor nerve in two cases of orbital floor trapdoor fracture. Jpn J Ophthalmol 49:246–252
525. Tse R, Allen L, Matic D (2007) The white-eyed medial blowout fracture. Plast Reconstr Surg 119:277–286
526. Cobb ARM. Orbital fractures. BMJ Best Pract, 2021 https://best-practice.bmj.com/topics/en-gb/1172
527. Hink EM, Wei LA, Durairaj VD (2014) Clinical features and treatment of pediatric orbit fractures. Ophthalmic Plast Reconstr Surg 30:124–131
528. Sirichai P, Anderson PJ (2015) Orbital fractures in children: 10 years' experience from a tertiary centre. Br J Oral Maxillofac Surg 53:938–942
529. Firriolo JM, Ontiveros NC, Pike CM, Taghinia AH, Rogers-Vizena CR, Ganor O, Greene AK, Meara JG, Labow BI (2017) Pediatric orbital floor fractures: clinical and radiological predictors of tissue entrapment and the effect of operative timing on ocular outcomes. J Craniofac Surg 28:1966–1971
530. Yang DJ, Kim YJ, Seo DW, Lee HJ, Park IJ, Sohn CH, Ryoo JM, Lee JS, Kim WY, Lim KS (2017) Characteristics of orbital wall fractures in preschool and school-aged children. Clin Exp Emerg Med 4:32–37
531. Borner U, Anschuetz L, Kaiser N, Rieke A, Dubach P, Caversaccio M (2019) Blunt nasal trauma in children: a frequent diagnostic challenge. Eur Arch Otorhinolaryngol 276:85–91
532. Zimmermann CE, Troulis MJ, Kaban LB (2006) Pediatric facial fractures: recent advances in prevention, diagnosis and management. Int J Oral Maxillofac Surg 35:2–13
533. Desrosiers AE 3rd, Thaller SR (2011) Pediatric nasal fractures: evaluation and management. J Craniofac Surg 22:1327–1329
534. Liu C, Legocki AT, Mader NS, Scott AR (2015) Nasal fractures in children and adolescents: mechanisms of injury and efficacy of closed reduction. Int J Pediatr Otorhinolaryngol 79:2238–2242
535. Bretschneider JH, Dunnebier EA (2007) Keel-, neus- en oorletsels. In: Kramer WLM, ten Duis HJS, Kimpen JLL, Leenen LPH, Patka P (eds) Handboek Kindertraumatologie. De Tijdstroom, Utrecht
536. Willging JP, Bower CM, Cotton RT (1992) Physical abuse of children. A retrospective review and an otolaryngology perspective. Arch Otolaryngol Head Neck Surg 118:584–590
537. Canty PA, Berkowitz RG (1996) Hematoma and abscess of the nasal septum in children. Arch Otolaryngol Head Neck Surg 122:1373–1376
538. Precious DS, Delaire J, Hoffman CD (1988) The effects of naso-maxillary injury on future facial growth. Oral Surg Oral Med Oral Pathol 66:525–530
539. Nathanson M (2000) The physically and emotionally abused child. In: Mason JK, Purdue BN (eds) The pathology of trauma. Arnold, London, pp 155–175
540. Christian CW, Mouden LD (2009) Maxillofacial, neck, and dental manifestations of child abuse. In: Reece RM, Christian CW (eds) Child abuse, medical diagnosis & management. American Academy of Pediatrics, Itasca, pp 189–208
541. Lam R (2016) Epidemiology and outcomes of traumatic dental injuries: a review of the literature. Aust Dent J 61(Suppl 1):4–20
542. Andreasen JO, Ravn JJ (1972) Epidemiology of traumatic dental injuries to primary and permanent teeth in a Danish population sample. Int J Oral Surg 1:235–239
543. McTigue DJ, Azadani E (2020) Evaluation and management of dental injuries in children. https://www.uptodate.com/contents/evaluation-and-management-of-dental-injuries-in-children. Accessed 31 Aug 2021
544. Wilson S, Smith GA, Preisch J, Casamassimo PS (1997) Epidemiology of dental trauma treated in an urban pediatric emergency department. Pediatr Emerg Care 13:12–15
545. Widmer R (1989) Tandtraumata. Mod Med:311–317
546. Faus-Damiá M, Alegre-Domingo T, Faus-Matoses I, Faus-Matoses V, Faus-Llácer VJ (2011) Traumatic dental injuries among schoolchildren in Valencia, Spain. Med Oral Patol Oral Cir Bucal 16:e292–e295
547. Royal College of paediatrics and Child Health (RCPCH) (2014) Oral injuries. https://www.rcpch.ac.uk/resources/child-protection-evidence-oral-injuries. Accessed 31 Aug 2021.
548. Glendor U (2009) Aetiology and risk factors related to traumatic dental injuries—a review of the literature. Dent Traumatol 25:19–31
549. Cagetti MG, Marcoli PA, Berengo M, Cascone P, Cordone L, Defabianis P, De Giglio O, Esposito N, Federici A, Laino A, Majorana A, Nardone M, Pinchi V, Pizzi S, Polimeni A, Privitera MG, Talarico V, Zampogna S (2019) Italian guidelines for the prevention and management of dental trauma in children. Ital J Pediatr 45:157

550. Paglia L (2018) Child abuse: awareness is the first step to action. Eur J Paediatr Dent 19:89
551. Blain SM, Winegarden T, Barber TK, Sognnaes RF (1979) Child abuse and neglect, II. Dentistry's role. J Dent Res 58:367
552. Kittle PE, Richardson DS, Parker JW (1981) Two child abuse/child neglect examinations for the dentist. ASDC J Dent Child 48:175–180
553. Wright JT, Thornton JB (1983) Osteogenesis imperfecta with dentinogenesis imperfecta: a mistaken case of child abuse. Pediatr Dent 5:207–209
554. Kaplan JM (1986) Pseudoabuse—the misdiagnosis of child abuse. J Forensic Sci 31:1420–1428
555. Edwards PC, Levering N, Wetzel E, Saini T (2008) Extirpation of the primary canine tooth follicles: a form of infant oral mutilation. J Am Dent Assoc 139:442–450
556. Carrotte PV (1990) An unusual case of child abuse. Br Dent J 168:444–445
557. Gantha SN, Chinta M, Kanumuri PK, Birra C (2017) Non-suicidal self-injury: an unexplored cause of dental trauma. BMJ Case Rep 2017:bcr2017219901
558. American Academy of Pediatric Dentistry (AAPD) (2020) Definition of dental neglect. The reference manual of pediatric dentistry. American Academy of Pediatric Dentistry, Chicago, IL, p 16
559. Harris JC, Elcock C, Sidebotham PD, Welbury RR (2009) Safeguarding children in dentistry: 2. Do paediatric dentists neglect child dental neglect? Br Dent J 206:465–470
560. Harris JC (2018) The mouth and maltreatment: safeguarding issues in child dental health. Arch Dis Child 103:722–729
561. Lourenço CB, Saintrain MV, Vieira AP (2013) Child, neglect and oral health. BMC Pediatr 13:188
562. British Dental Association (BDA) (2020) Dental neglect. https://bda.org/childprotection/Recognising/Pages/Dental-neglect.aspx. Accessed 31 Aug 2021
563. Ramazani N (2014) Child dental neglect: a short review. Int J High Risk Behav Addict 3:e21861
564. Greene PE, Chisick MC, Aaron GR (1994) A comparison of oral health status and need for dental care between abused/neglected children and nonabused/non-neglected children. Pediatr Dent 16:41–45
565. Hinchliffe J (2011) Forensic odontology, part 5. Child abuse issues. Br Dent J 210:423–428
566. Rawal SY, Claman LJ, Kalmar JR, Tatakis DN (2004) Traumatic lesions of the gingiva: a case series. J Periodontol 75:762–769
567. Cooper A, Floyd T, Barlow B, Niemirska M, Ludwig S, Seidl T, O'Neill J, Templeton J, Ziegler M, Ross A (1988) Major blunt abdominal trauma due to child abuse. J Trauma 28:1483–1487
568. Sinha CK, Lander A (2013) Trauma in children: abdomen and thorax. Surgery (Oxford) 31:123–129
569. Overly FL, Wills H, Valente JH (2014) 'Not just little adults'—a pediatric trauma primer. R I Med J (2013) 97:27–30
570. Milroy CM (2014) Blunt abdominal and thoracic injuries in children. In: Collins KA, Byard RW (eds) Forensic pathology of infancy and childhood. Springer, Berlin, pp 291–325
571. Araki T, Yokota H, Morita A (2017) Pediatric traumatic brain injury: characteristic features, diagnosis, and management. Neurol Med Chir 57:82–93
572. Yu YR, DeMello AS, Greeley CS, Cox CS, Naik-Mathuria BJ, Wesson DE (2018) Injury patterns of child abuse: experience of two Level 1 pediatric trauma centers. J Pediatr Surg 53:1028–1032
573. Hymel KP, Stoiko MA, Herman BE, Combs A, Harper NS, Lowen D, Deye KP, Homa K, Blackman JA (2010) Head injury depth as an indicator of causes and mechanisms. Pediatrics 125:712–720
574. Yoshino E, Yamaki T, Higuchi T, Horikawa Y, Hirakawa K (1985) Acute brain edema in fatal head injury: analysis by dynamic CT scanning. J Neurosurg 63:830–839
575. Willman KY, Bank DE, Senac M, Chadwick DL (1997) Restricting the time of injury in fatal inflicted head injuries. Child Abuse Negl 21:929–940
576. Marmarou A (2007) A review of progress in understanding the pathophysiology and treatment of brain edema. Neurosurg Focus 22:E1
577. David TJ (2008) Non-accidental head injury—the evidence. Pediatr Radiol 38:370–377
578. Rooks VJ, Eaton JP, Ruess L, Petermann GW, Keck-Wherley J, Pedersen RC (2008) Prevalence and evolution of intracranial hemorrhage in asymptomatic term infants. AJNR AmJ Neuroradiol 29:1082–1089
579. Holden KR, Titus MO, Van Tassel P (1999) Cranial magnetic resonance imaging examination of normal term neonates: a pilot study. J Child Neurol 14:708–710
580. Whitby EH, Griffiths PD, Rutter S, Smith MF, Sprigg A, Ohadike P, Davies NP, Rigby AS, Paley MN (2004) Frequency and natural history of subdural haemorrhages in babies and relation to obstetric factors. Lancet 363:846–851
581. Looney CB, Smith JK, Merck LH, Wolfe HM, Chescheir NC, Hamer RM, Gilmore JH (2007) Intracranial hemorrhage in asymptomatic neonates: prevalence on MR images and relationship to obstetric and neonatal risk factors. Radiology 242:535–541
582. Werner EF, Janevic TM, Illuzzi J, Funai EF, Savitz DA, Lipkind HS (2011) Mode of delivery in nulliparous women and neonatal intracranial injury. Obstet Gynecol 118:1239–1246
583. Sirgiovanni I, Avignone S, Groppo M, Bassi L, Passera S, Schiavolin P, Lista G, Cinnante C, Triulzi F, Fumagalli M, Mosca F (2014) Intracranial haemorrhage: an incidental finding at magnetic resonance imaging in a cohort of late preterm and term infants. Pediatr Radiol 44:289–296
584. Nikam RM, Kandula VV, Yue X, Krishnan V, Kumbhar SS, Averill LW, Paudyal B, Choudhary AK (2021) Birth-related subdural hemorrhage: prevalence and imaging morphology. Pediatr Radiol 51:939–946
585. Pollina J, Dias MS, Li V, Kachurek D, Arbesman M (2001) Cranial birth injuries in term newborn infants. Pediatr Neurosurg 35:113–119
586. Ou-Yang MC, Huang CB, Huang HC, Chung MY, Chen CC, Chen FS, Chao PH, Chen IL, Ou-Yang MH, Liu CA (2010) Clinical manifestations of symptomatic intracranial hemorrhage in term neonates: 18 years of experience in a medical center. Pediatr Neonatol 51:208–213
587. Perlman JM (2004) Brain injury in the term infant. Semin Perinatol 28:415–424
588. Parker LA (2005) Part 1: early recognition and treatment of birth trauma: injuries to the head and face. Adv Neonatal Care 5:288–297. quiz 298-300
589. Levin AV (1997) Ophthalmic presentations. ABC of child abuse. BMJ Publishing Group, London, pp 17–19
590. Laghmari M, Skiker H, Handor H, Mansouri B, Ouazzani Chahdi K, Lachkar R, Salhi Y, Cherkaoui O, Ouazzani Tnacheri B, Ibrahimy W, Alami H, Bezad R, Ahid S, Abouqal R, Daoudi R (2014) Hémorragies rétiniennes liées à l'accouchement chez le nouveau-né: fréquence et relation avec les facteurs maternels, néonataux et obstétricaux. Étude prospective de 2031 cas [Birth-related retinal hemorrhages in the newborn: incidence and relationship with maternal, obstetric and neonatal factors. Prospective study of 2,031 cases]. J Fr Ophtalmol 37:313–319
591. Emerson MV, Pieramici DJ, Stoessel KM, Berreen JP, Gariano RF (2001) Incidence and rate of disappearance of retinal hemorrhage in newborns. Ophthalmology 108:36–39
592. Watts P, Maguire S, Kwok T, Talabani B, Mann M, Wiener J, Lawson Z, Kemp A (2013) Newborn retinal hemorrhages: a systematic review. J AAPOS 17:70–78

593. The Royal College of Paediatrics and Child Health (RCPCH) and The Royal College of Ophthalmologists (ROC) (2013) Abusive head trauma and the eye in infancy. https://www.rcophth.ac.uk/wp-content/uploads/2014/12/2013-SCI-292-ABUSIVE-HEAD--TRAUMA-AND-THE-EYE-FINAL-at-June-2013.pdf. Accessed 4 May 2021
594. Levin AV (2010) Retinal hemorrhage in abusive head trauma. Pediatrics 126:961–970
595. Maguire SA, Watts PO, Shaw AD, Holden S, Taylor RH, Watkins WJ, Mann MK, Tempest V, Kemp AM (2013) Retinal haemorrhages and related findings in abusive and non-abusive head trauma: a systematic review. Eye (London, England) 27:28–36
596. Fortin G, Stipanicic A (2010) How to recognize and diagnose abusive head trauma in infants. Ann Phys Rehabil Med 53:693–710
597. Kelly P, John S, Vincent AL, Reed P (2015) Abusive head trauma and accidental head injury: a 20-year comparative study of referrals to a hospital child protection team. Arch Dis Child 100:1123–1130
598. Narang SK (2011) A daubert analysis of abusive head trauma/shaken baby syndrome. Houston J Health Law Policy 11:505–633
599. Narang SK, Melville JD, Greeley CS, Anderst JD, Carpenter SL, Spivack B (2013) A daubert analysis of abusive head trauma/shaken baby syndrome—part II: an examination of the differential diagnosis. Houston J Health Law Policy 13:203–327
600. Sackett DL, Rosenberg WM, Gray JA, Haynes RB, Richardson WS (1996) Evidence based medicine: what it is and what it isn't. BMJ 312:71–72
601. Frasier LD, Hinds TS, Luyet F (2016) Pediatric abusive head trauma. Volume 1: traumatic injuries. STM Learning, Saint Louis
602. Frasier LD, Hinds TS, Luyet FM (2016) Pediatric abusive head trauma. Volume 2: medical mimics pocket atlas. STM Learning, Saint Louis
603. Swedish Agency for Health Technology Assessment and Assessment of Scial Services (SBU) (2016) Traumatic shaking—the role of the triad in medical investigations of suspected traumatic shaking: a systematic review. Swedish Council on Health Technology Assessment (SBU), Stockholm
604. Bilo RAC, Banaschak S, Herrmann B, Karst WA, Kubat B, Nijs HGT, van Rijn RR, Sperhake J, Stray-Pedersen A (2017) Using the table in the Swedish review on shaken baby syndrome will not help courts deliver justice. Acta Paediatr (Oslo, Norway: 1992) 106:1043–1045
605. Bilo RAC (2018) The Swedish Agency for health technology-report about traumatic shaking: much ado about nothing? Forensic Sci Med Pathol 14:541–544
606. Stone EL, Davis LL (2019) State of the science: skull fracture and intracranial injury in children below age 2. J Emerg Nurs 45:545–550
607. Dunning J, Batchelor J, Stratford-Smith P, Teece S, Browne J, Sharpin C, Mackway-Jones K (2004) A meta-analysis of variables that predict significant intracranial injury in minor head trauma. Arch Dis Child 89:653–659
608. Demaerel P, Casteels I, Wilms G (2002) Cranial imaging in child abuse. Eur Radiol 12:849–857
609. Mogbo KI, Slovis TL, Canady AI, Allasio DJ, Arfken CL (1998) Appropriate imaging in children with skull fractures and suspicion of abuse. Radiology 208:521–524
610. Lloyd DA, Carty H, Patterson M, Butcher CK, Roe D (1997) Predictive value of skull radiography for intracranial injury in children with blunt head injury. Lancet 349:821–824
611. Gruskin KD, Schutzman SA (1999) Head trauma in children younger than 2 years: are there predictors for complications? Arch Pediatr Adolesc Med 153:15–20
612. Erlichman DB, Blumfield E, Rajpathak S, Weiss A (2010) Association between linear skull fractures and intracranial hemorrhage in children with minor head trauma. Pediatr Radiol 40:1375–1379
613. Arrey EN, Kerr ML, Fletcher S, Cox CS Jr, Sandberg DI (2015) Linear nondisplaced skull fractures in children: who should be observed or admitted? J Neurosurg Pediatr 16:703–708
614. Bressan S, Marchetto L, Lyons TW, Monuteaux MC, Freedman SB, Da Dalt L, Nigrovic LE (2018) A systematic review and meta-analysis of the management and outcomes of isolated skull fractures in children. Ann Emerg Med 71:714–724.e712
615. Steinbok P, Flodmark O, Martens D, Germann ET (1987) Management of simple depressed skull fractures in children. J Neurosurg 66:506–510

Spine

6

Rob A. C. Bilo, Simon G. F. Robben, Rick R. van Rijn, and Michelle Nagtegaal

Contents

R. A. C. Bilo (✉)
Veilig Thuis Rotterdam Rijnmond (Center for the Reporting of Child Abuse, Domestic Violence and Elder Abuse), Rotterdam, The Netherlands

S. G. F. Robben
Department of Radiology, Maastricht University Medical Center, Maastricht, The Netherlands
e-mail: s.robben@mumc.nl

R. R. van Rijn
Department of Radiology and Nuclear Medicine, Amsterdam UMC, University of Amsterdam, Amsterdam, The Netherlands

Department of Forensic Medicine, Netherlands Forensic Institute, The Hague, The Netherlands
e-mail: r.r.vanrijn@amsterdamumc.nl

M. Nagtegaal
Department of Forensic Medicine, Netherlands Forensic Institute, The Hague, The Netherlands
e-mail: m.nagtegaal@amsterdamumc.nl

6.1 General Aspects of Spinal Fractures

6.1.1 Clinical Presentation

Injuries to the spine can range from relatively mild ligament and muscle strains, to fractures, dislocations, or subluxation (= spondylolisthesis: the forward slippage of a vertebra on the one below) of the vertebrae with subsequent risk of damage to the spinal cord. Fractures and dislocations of the vertebrae can occur at all levels in the spine (cervical, thoracolumbar, and sacro-coccygeal—Fig. 6.1) and in all parts of a vertebra (Fig. 6.2). Spinal fractures and dislocations carry a risk of spinal cord injury, due to compression, by the fracture/dislocation itself or by the concomitant haematoma, or due to tearing of the cord.

Neurological symptoms are rarely seen in children with spinal fractures. Nevertheless, a full neurological examina-

R. A. C. Bilo et al. (eds.), *Forensic Aspects of Paediatric Fractures*, https://doi.org/10.1007/978-3-031-12041-1_6

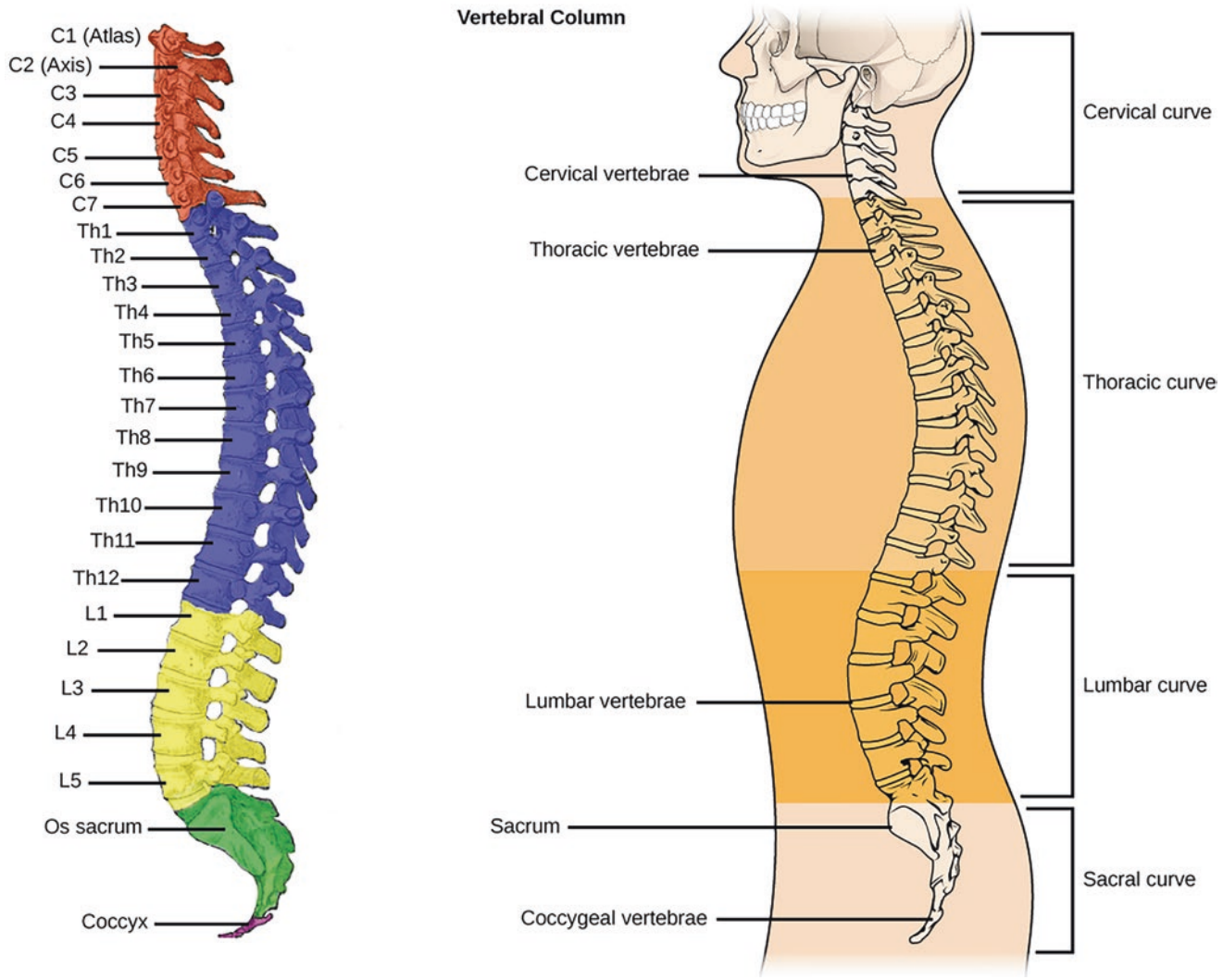


Fig. 6.1 Anatomic regions of the spine (Source: https://commons.wikimedia.org/wiki/File:Figure_38_01_07.jpg, licensed under the Creative Commons Attribution-Share Alike 3.0 Unported)

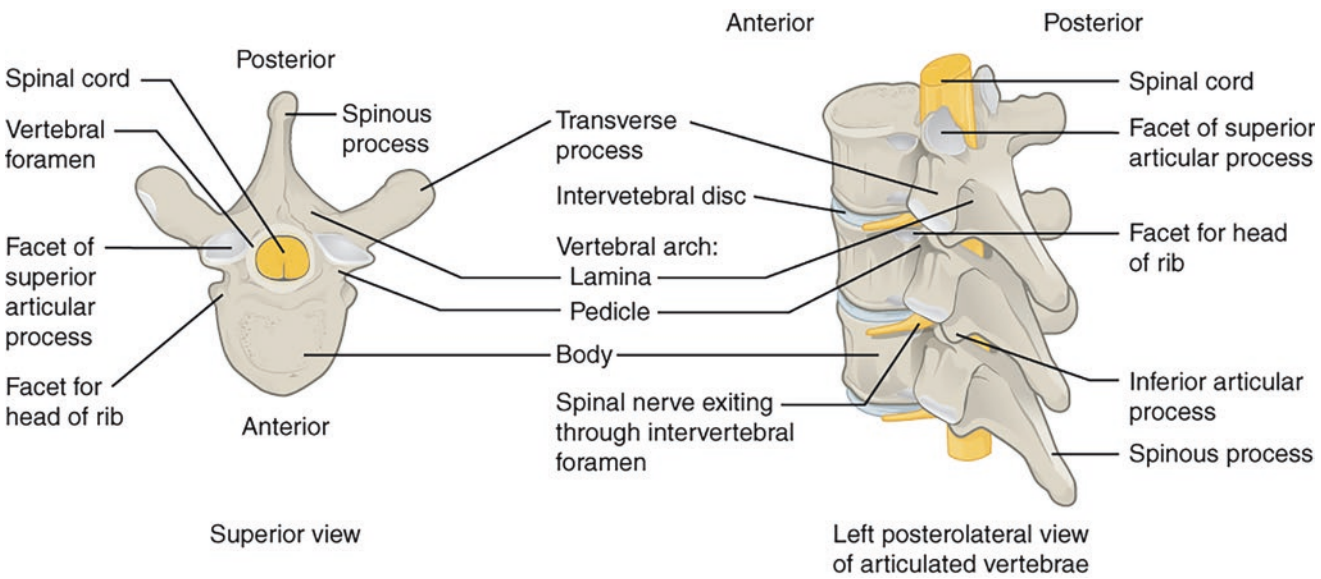


Fig. 6.2 Vertebral anatomy (Source: https://commons.wikimedia.org/wiki/File:718_Vertebra-en.svg, licensed under the Creative Commons Attribution-Share Alike 3.0 Unported)

tion should always be performed on every child that presents with a spinal fracture, because the consequences of neurological damage could be devastating [1]. In some children, only a slight kyphosis is found at physical examination, and sometimes also signs of compression or contusion of the spinal cord. However, a vertebral fracture in children is often a coincidental finding discovered in radiological imaging.

When a history of a trauma and clinical symptoms are lacking, spinal fractures can be missed [2]. For that reason some spinal injuries, regardless of whether these are accidental or non-accidental, will not be diagnosed in children. The absence of clinical signs and symptoms does not mean that long-term consequences will not occur over time. After years (sometimes serious) anomalies in spinal alignment or growth of the spine can develop [3]. In a systematic review by Parent et al., it was shown that children who sustain spinal injury before their adolescent growth spurt, have a higher risk of developing spinal deformities [4].

6.1.2 Epidemiology

Epidemiological findings of injuries to the spine, concerning incidence, age, Male-to-Female ratio, level of injuries, associated cord lesions, associated injuries, and mortality vary between various studies.

6.1.2.1 Age-Related Incidence and Male-to-Female Ratio

Spinal fractures are extremely rare in children and far less common than in adult patients [5, 6]. The exact incidence and prevalence of spinal fractures in children are unknown, but research has suggested that spinal fractures account for less than 1% of all fractures in children [7].

Piatt estimated, after evaluating the data from the US Kids' Inpatient Database (KID) and the National Trauma Data Bank (NTDB) registry for 2009, that in the United States the incidence of hospital admissions for spinal injury, irrespective of the circumstances (accidental or non-accidental), was 170 per one million and that the incidence of spinal cord injury was 24 per one million (age range from birth to 21 years of age) [8]. There was regional variance in incidence and adolescents predominated. Piatt also stated that true rates could be higher, because clinical manifestations are often lacking.

Based on the Canadian National Trauma Registry Reilly estimated an annual incidence of 1 child in 1,000,000 children under the age of 15 years. Reilly stated that the vast majority of spine and cord injuries, that are sustained in North America occur in patients between the age of 15 and 40 years and that in patients, aged 0–40 years with proven spinal injuries, only 10% is under the age of 15 years [9].

Spinal injury is more common in older children. Kim et al. studied the findings in 275 children with spinal injuries and found that they were more common in children, aged 12–16 years, with most injuries in children aged 15 and 16 years and with the highest risk in active adolescent boys [6]. Babu et al. found in a series of 84 children with spine injuries that 79% was 13 years of age or older [10]. There is an overall higher incidence of spinal injury in boys (61–86%) [11, 12]. Babu et al. even found a 6 to 1 male-to-female ratio [10].

6.1.2.2 Location and Age-Related Level of Fractures

Most studies on paediatric spinal injuries show a high incidence of cervical spine injury (56–100%), followed by the thoracolumbar region (1–34%) [4, 10, 13, 14]. Gopinathan et al. reviewed the literature concerning spine injuries in children and found that 60 to 80% of the paediatric spinal injuries, reported in the medical literature, were cervical spine injuries [15]. Saul and Dresing, however, found that the most common level was lower thoracic (TH7–8) and thoracolumbar (Th12-L1), while Özkan and Babu found that in paediatric patients fractures in the thoracolumbar region were quite rare [7, 10, 14].

Knox evaluated the findings in 206 children, aged 0–9 years, with spinal trauma and found that cervical spine injuries were more common in children under the age of 4 years than in children, aged 4–9 years old [13]. In patients with cervical spine injury, the upper cervical spine (52–68%) is more often involved than the lower cervical spine (25–28%). Both upper and lower cervical spine was injured in 7% [5, 11, 12]. Babu, however, found more injuries of the lower cervical spine (63%) than of the upper cervical spine (37%), probably because the mean age of these patients was higher (15 years) than in the other studies [10]. Patel evaluated the findings in 1098 patients with cervical spine injury and found that upper cervical spine injuries occurred in all age groups but that lower cervical spine injuries were more common in children above the age of 8 years [12]. Cirak evaluated 406 children with spinal injuries and found that in all age groups the upper cervical spine (C0-C4) was approximately three times more involved than the lower cervical spine (C5-C7) [5]. In their review, Gopinathan et al. found that the majority of cervical spine injuries in children occur between the skull and C4 and that in many cases C1 and C2 are involved. They found atlanto-axial injuries 2.5 times more often in children than in adults [16].

6.1.2.3 Cord Lesions and SCIWORA

Children can have spinal fractures with or without spinal cord injuries, but they can also have neurological symptoms due to spinal cord injuries without radiological abnormalities (SCIWORA) [6].

In a series of 1098 children with cervical spine injury 33% had neurological injury. Almost 1 in 4 children with neurological injuries had a complete spinal cord injury (8% of all patients with cervical spine injury) [12]. Complete spinal cord injuries were more common in lower cervical spine injuries [11]. It is regarded as a typical paediatric injury [17]. In paediatric trauma

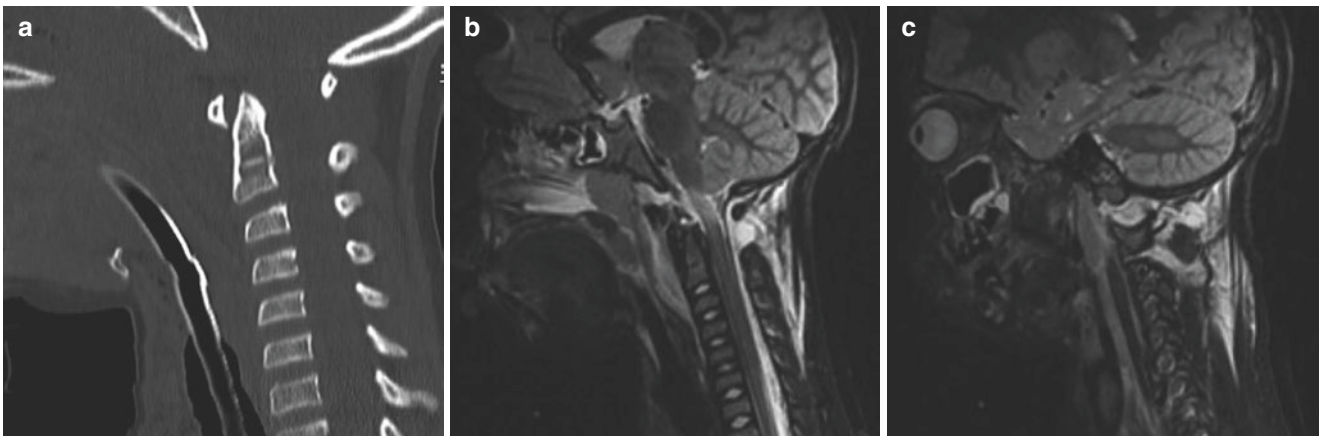


Fig. 6.3 (**a**) CT of the cervical spine shows atlanto-occipital dislocation with anterior displacement of the occiput in relation to the atlas. MRI shows (**b**) diffuse soft-tissue oedema and focal oedema of the cervical spine and (**c**) increased distance between the occiput and the body of C1

patients (spinal and non-spinal trauma) the incidence of SCIWORA is around 2%, [Reddy] but in children with spinal trauma the incidence rises to 6% [5, 6]. In children with cervical spinal trauma the incidence further rises to 17–38%. Pang and Pollack evaluated the findings in 55 children with SCIWORA [18]. Ten children (18%) had upper cervical cord lesions (C1-C4), 33 (60%) lower cervical cord lesions (C5-C8), and 12 (22%) thoracic cord lesions. They were the first to use the term SCIWORA for complete, severe partial, or (rarely) mild spinal cord injury in children without evidence of a vertebral fracture or dislocation on conventional radiographs (CR) or CT [18]. Pang and Pollack suggested that the mechanism of the cord injury in case of SCIWORA is probably related to the immature and elastic juvenile spine, which is more flexible and permits self-reducing but significant intersegmental displacements when subjected to flexion, extension, and/or distraction forces [19]. For that reason, the spinal cord would be vulnerable to injury even though the vertebral column is spared from disruption, and this vulnerability seems to be most evident in children under the age of 8 years. This is thought to be due to large head/trunk ratio, the immature and flexible vertebral spine combined with the relatively narrow spinal canal and the relatively poorer blood supply [19, 20].

In children under the age of 8 years, SCIWORA is more common than spinal cord injury with associated radiological spinal abnormalities [19, 20]. Pang and Pollack found complete transection or severe lesions in 22 of 55 children with SCIWORA (40%) and mild lesions in 33 (60%). In their study, the incidence of severe cord lesions was higher in children under the age of 8 years (21 of the 22 with severe lesions), whereas mild cord lesions were more present in children above that age (24 of the 33 with mild lesions) [19].

In current times with rapid technological developments and increasing use of modern CT scanners and especially MRI the diagnosis SCIWORA has become less relevant. A large portion of the children with injuries previously classified as SCIWORA turns out to have abnormalities detectable on MRI, such as injuries to the spinal cord, ligaments, muscles, or the vertebral endplate [21–23]. The high sensitivity for (intra)spinal pathology of MRI will limit the diagnosis SCIWORA to a smaller sample of patients, however, the complete absence of neuroimaging abnormalities remains clinically relevant in children with SCIWORA. The paediatric trauma guidelines of the Royal College of Radiology state that '*Where there are definitive neurological signs, the primary imaging modality should be MRI where possible*' [24].

6.1.2.4 Associated Injuries and Mortality

Kim et al. found associated injuries in 55% of 275 children with spinal trauma [6]. Traumatic brain injury is reported to be the most prevalent associated injury and was present in 29–37% of children [5, 6]. Fractures and dislocations were present in 27% of the patients [6]. Children above the age of 8 years have a higher risk for concomitant lesions [16]. Multiple fractures are more common in children of 13 years and older [7].

The overall mortality of spinal trauma is 3% [6]. In a study of 103 children with cervical spine injuries predictors of mortality included younger age, motor vehicle-related circumstances, C1 dislocations, injury severity score greater than 25, and associated closed head injuries [11]. The mortality rate in children with upper cervical spine injury was almost six times higher than in the group with lower cervical spine injury. Mortality was highest in children with atlanto-occipital dislocation (almost 50%) (Fig. 6.3a–c) [12].

6.2 Cause of Spinal Fractures

Spinal fractures in children with normal skeletal development are usually caused by a trauma with a high transfer of energy, such as. falls from height (Fig. 6.4a, b) or motor vehicle accidents (MVA). Rarely fractures have been described due to a trauma with a low transfer of energy. For example, a case report in an 37-year-old male describes the

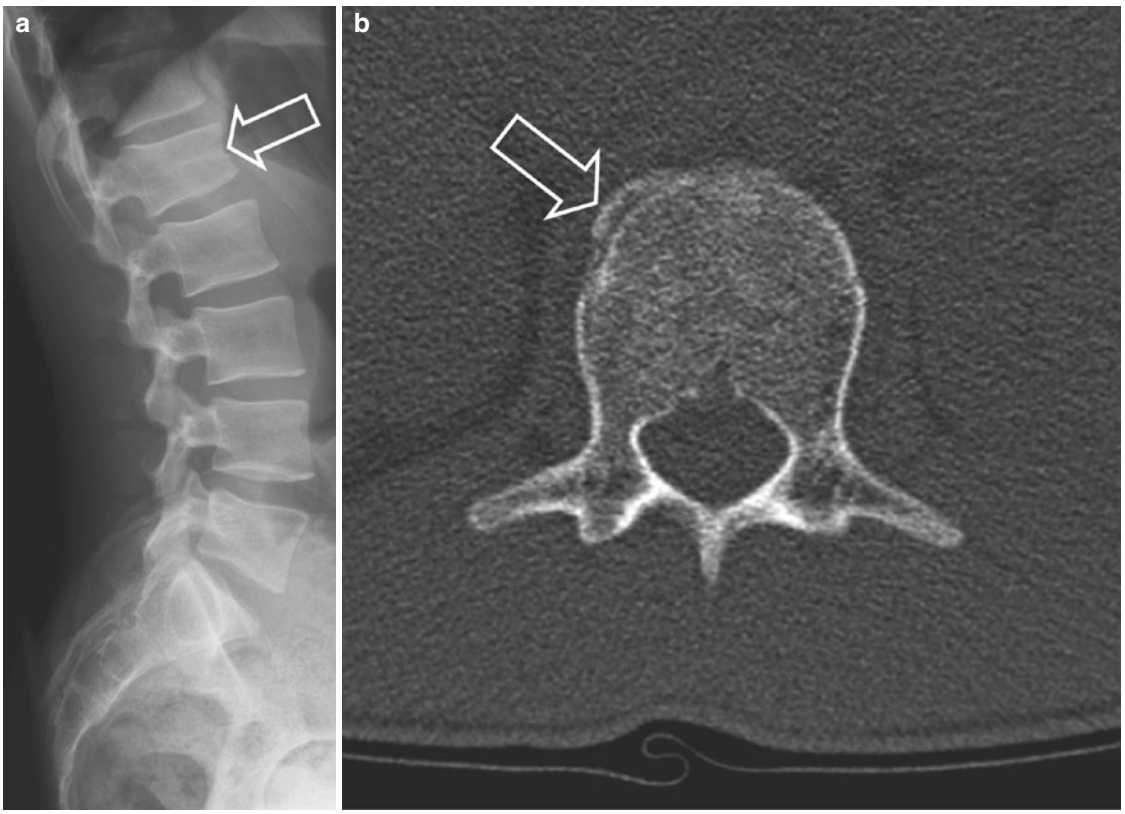


Fig. 6.4 (**a**) Accidental fracture of the first lumbar vertebra (*open arrow*) in a 15-year-old child (sports trauma from high jump). (**b**) CT scan of the lumbar spine shows a stabile fracture with only an anterior component (*open arrow*)

occurrence of an isolated transverse sacrum fracture due to a fall on the back onto a rough surface and one other case series describes spinal fractures in children in short distance falls [25, 26]. Fracturing due to low transfer of energy may also occur in patients with weakened bones, as is the case in osteogenesis imperfecta (Fig. 6.5) [27–29]. Spinal fractures in normally developed bones (but probably also in weakened bones) are thought to be caused by one of the following four injury mechanisms (Fig. 6.6a–d) [30, 31]:

(a) Flexion compression, resulting in a wedge deformity/fracture, due to simple compression of the anterior column with variable involvement of the middle and posterior column.
(b) Axial compression, resulting in a burst fracture in case of severe axial loading.
(c) Lateral flexion and rotation with or without a posterior-anteriorly directed force (translation), resulting in fracture dislocation (displacement in the horizontal plane).
(d) Flexion distraction, resulting in a horizontal fracture and separation of posterior elements (a so-called 'Chance' fracture, Fig. 6.7a, b). Distraction is the result of displacement in the vertical plane.

Most spinal fractures in paediatric patients are caused by hyperflexion and/or hyperextension combined with axial compression, regardless of the location of the fracture,

Fig. 6.5 Vertebrae plana in a child with osteogenesis imperfecta

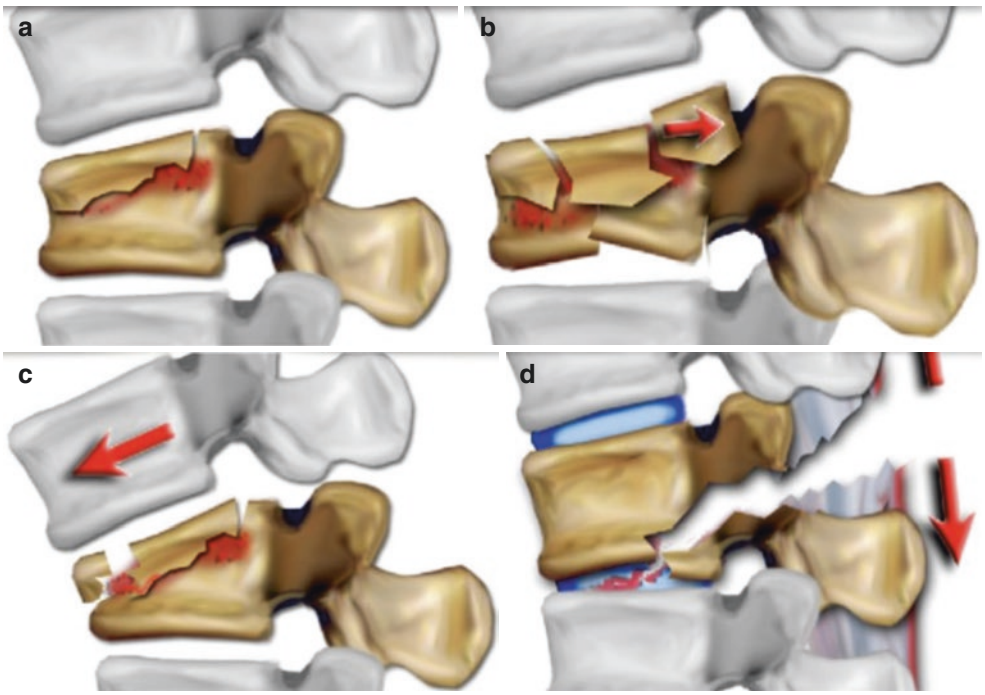


Fig. 6.6 (**a**) Fracture due to flexion-compression: wedge deformity, (**b**) due to axial-compression: burst fracture, (**c**) due to mainly lateral flexion and rotation: fracture dislocation, and (**d**) due to flexion-distraction: Chance fracture. (reprinted with permission from https://radiologyassistant.nl/neuroradiology/spine-thoracolumbar-injury [31])

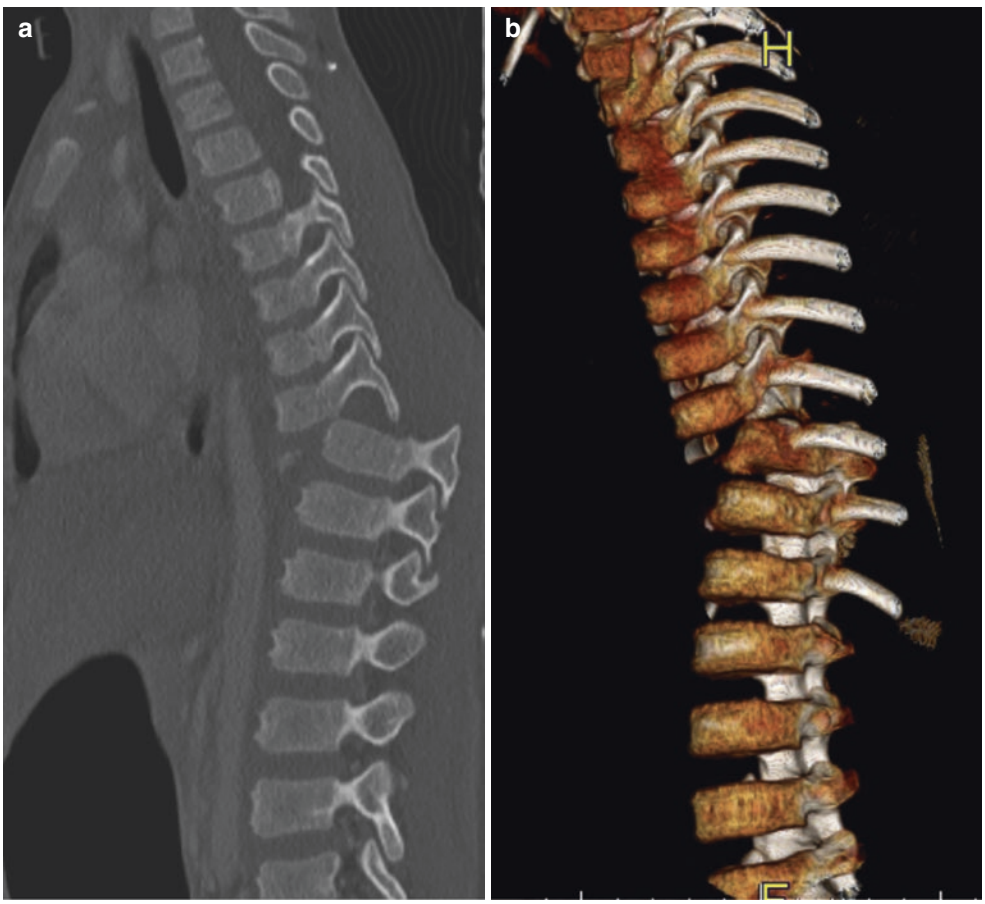


Fig. 6.7 A seven-year-old boy who sustained a seat belt injury in a car accident. Horizontal osseous and ligamentous flexion-distraction injury resulting in dislocation at level Th 9–10 (Chance fracture). Sagittal CT MPR (**a**) and 3D reconstruction (**b**)

whether it is in the vertebral body or in the vertebral arch [32, 33]. In addition, shearing forces or subluxation-dislocation (displacement of vertebrae relative to each other) can lead to fractures in paediatric patients [34].

A compression fracture of a vertebral body is caused by a combination of hyperflexion and axial loading (forces in the longitudinal direction of the vertebral column) with or without a rotating component [35]. These fractures are recognizable because of a diffuse loss of height. If the fractures are caused only by hyperflexion, loss of height will mainly occur at the anterior part of the vertebral body. Compression fractures of vertebral bodies can occur in all regions of the spine, but are most commonly found between the middle part of the thoracic spine (mid-thoracic) and the middle part of the lumbar spine (mid-lumbar). The severity of compression fractures of the vertebral body varies from a barely perceptible loss of vertebral body height to severe compression with clearly perceptible loss of height ([36]. Multiple, often consecutive, compression fractures (contiguous fractures) can occur, although vertebral bodies of normal height can be found between vertebral bodies with loss of height due to compression (non-contiguous fractures) [36, 37].

To assess the extent of spinal trauma not only the location and aspect of the fracture are of importance, but also the integrity of the posterior ligamentous complex and the neurological status of the patient. This has led to the introduction of the Thoracolumbar Injury Classification and Severity Scale (TLICS) (Table 6.1) [38, 39]. It has been shown in several studies that the TLICS can be applied to the paediatric population as well [40, 41]. Based on this classification the need for surgical treatment can be evaluated in an objective manner.

Table 6.1 Thoracolumbar injury classification and severity scale (adapted from Vaccaro et al.) [39]

Injury category	Point value
Injury morphology	
Compression	1
Burst	2
Translation or rotation	3
Distraction	4
PLC status	
Intact	0
Injury suspected or indeterminate	2
Injured	3
Neurological status	
Intact	0
Nerve root involvement	2
Incomplete spinal cord or conus medullaris injury	3
Complete spinal cord or conus medullaris injury	2
Cauda equina syndrome	3

PLC Posterior Ligament Complex
Score 0–3: Non-surgical treatment
Score 4: Surgeon can decide to perform surgery or not
Score > 4: Surgical treatment

6.3 Manner of Spinal Injuries

Spinal injuries and fractures can be sustained as a result of trauma before, during, and after birth. If sustained after birth, these fractures can occur in accidental and in non-accidental circumstances. In this section, we will deal with general aspects of accidental and non-accidental trauma after birth. A detailed discussion on spinal fractures before, during, or after birth is presented in Sects. 6.4 (cervical spine injuries), 6.5 (thoracolumbar spine injuries), and 6.6 (injuries of the sacrum and coccyx).

6.3.1 Trauma After Birth: Accidental Trauma

As previously stated, spinal fractures are rare in children. Most commonly they occur in accidental circumstances. Usually, these circumstances concern significant traumas such as motor vehicle accidents (reported in up to 56% of cases), sports- and play-related trauma, and falls from a considerable height [4, 6, 7, 14, 42–45]. With respect to SCIWORA, Carroll et al. performed a systematic review and identified 433 paediatric patients with SCIWORA [46]. Blunt trauma was the mechanism of injury in most patients. Irrespective of age, sports-related trauma was most common in almost 40%, followed by falls in about 24% and motor vehicle-related trauma in about 23.18%.

6.3.2 Trauma After Birth: Non-accidental Trauma

6.3.2.1 Clinical Aspects and Epidemiology

Non-accidental spinal injuries are rarely reported in medical literature and with the exception of a few studies are usually limited to case reports/series [1, 3, 42, 47–50]. Some authors suggest that spinal injuries seldom occur in non-accidental circumstances [1, 3, 51, 52]. Non-accidental circumstances, however, should always be considered and excluded, when a child presents with a spinal fracture, irrespective of the type of vertebral fracture. Especially when the medical history is blank or does not mention any severe trauma, bone disease or an earlier experienced osteomyelitis, or tuberculosis [53, 54].

Swischuk described seven children who sustained spinal fractures due to non-accidental circumstances [50]. One of these seven children also had associated spinal cord injury.

Knox et al. reported on 206 young paediatric patients (aged 0–9 years) with spinal injuries. Fifty-seven children (27.7%) were aged 0–3 years. In 19% of the children, aged 0 to 3 years, the spinal injuries were due to non-accidental trauma, while in all children accidental trauma (especially motor vehicle accidents), was most prevalent [13].

Table 6.2 Spinal fracture distribution based on 370 positive skeletal surveys (based on data reported by Kleinman and Barber) [58, 158]

Level	Number of fractures
Cervical spine	
1	0
2	1
3	0
4	0
5	0
6	0
7	0
Thoracic spine	
1	0
2	2
3	4
4	4
5	1
6	1
7	3
8	3
9	3
10	4
11	4
12	6
Lumbar spine	
1	2
2	2
3	1
4	1
5	0
Sacral spine	
1	0
2	0
3	1
4	2
5	2

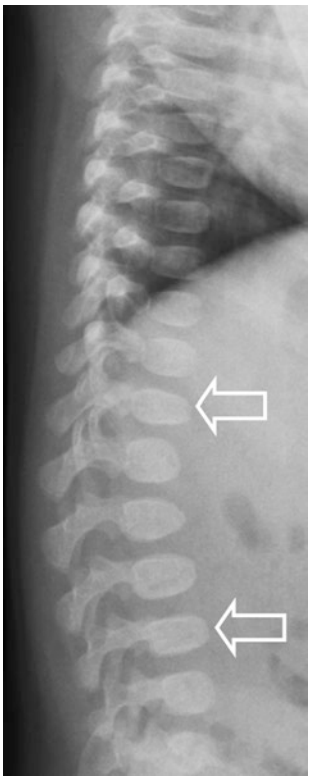


Fig. 6.8 A four-month-old child whose sib was admitted with inflicted traumatic brain injury. The skeletal survey shows fractures of the vertebral bodies at levels T12, L2, and L4

Kim et al. evaluated the findings in 275 children with spinal trauma. Of these children 32 were aged 4 years or younger. In only six cases, all aged under 2 years, the vertebral fractures were determined to be sustained in non-accidental circumstances [6].

In the older literature, the incidence of non-accidental spinal fractures is estimated to range between 0% and 3% in larger study populations [55, 56]. According to Bode and Newton, non-accidental injuries to the spine and the spinal cord occur in less than 1% of children with inflicted injuries [57]. Kleinman et al. reported on the yield of skeletal surveys with respect to the detection of hand, foot, and spine fractures in children (0–2 years) suspected of child abuse [58]. Out of 365 skeletal surveys, of which 62% (225/365) showed positive radiographic findings, 10 (2.7%) cases were positive for spinal fractures. The majority of fractures were found in the thoracic spine (Table 6.2).

Non-accidental spinal fractures most commonly are compression fractures of the vertebral bodies, due to extreme flexion or extension (in anterior-posterior direction), with or without a rotational component. This occurs particularly at the lower thoracic and higher lumbar level, for example due to shaking or direct-impact violence such as a blow by an object or a kick (Fig. 6.8). Often multiple compression fractures occur simultaneously (contiguous fractures). Sometimes they result from acute lateral flexion or from axial rotation. Lateral hyperflexion mainly leads to fractures at the thoracic level, and axial rotation mainly leads to fractures at the cervical level.

Kleinman considers fractures of the spinous process as a highly specific sign of non-accidental circumstances [54]. According to Kleinman, fractures and subluxations of the vertebral bodies have an average specificity, which means that in the absence of a plausible explanation there is a high risk the fracture is sustained in non-accidental circumstances. According to Brodeur and Monteleone, 50% of spinal injuries in children under the age of 1 year are sustained in non-accidental circumstances [53].

According to Piatt and Steinberg spinal cord injuries and vertebral fractures due to non-accidental circumstances have regularly been described as incidental findings but are rarely clinically apparent [49]. According to the authors, spinal cord injury without spinal fracture and without head injury is a rare presentation of inflicted injuries, and can escape recognition unless other (more or less) characteristic signs of inflicted injuries are detected.

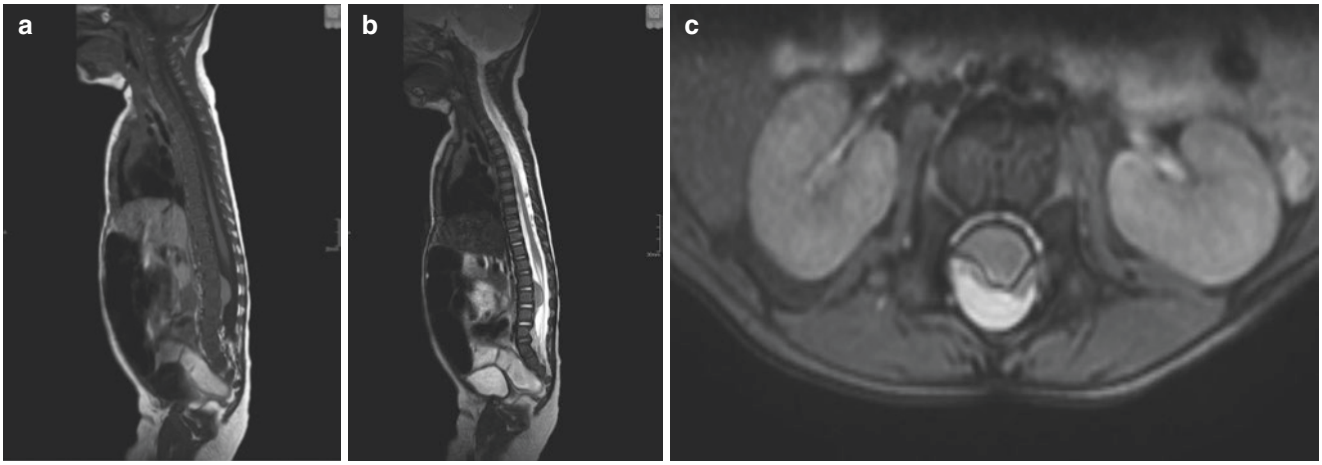


Fig. 6.9 Infant suspected to be the victim of inflicted traumatic brain injury. Sagittal T1 (**a**) and T2 (**b**) show a subdural haematoma at the level of L2–4. Axial T2-weighted imaging (**c**) shows the delineation by the denticulate ligaments

In 2010, Kemp et al. performed a systematic review of articles concerning the clinical and radiological characteristics of inflicted spinal injuries in paediatric patients under the age of 18 years (alive at presentation) [59]. They included 19 studies with a total of 25 children with inflicted spinal injuries. Twelve children (median age 5 months) had spinal injury solely at the cervical level. Ten of these children had musculoskeletal injuries such as Hangman's fractures, compression fractures, and dislocations. Six of these ten children had subsequent spinal cord injuries. In seven cases, the clinical signs of spinal injury were masked by respiratory symptoms and impaired levels of consciousness, six out of seven children were diagnosed with abusive head trauma. Thoracolumbar injuries were present in twelve children (median age 13.5 months). Eleven of these children had lesions at T11-L2 of which six had spinal cord injury. Nine children had fracture dislocations and three had compression fractures. Despite focal symptoms in all children, in eight cases spinal injuries were initially missed. One child had cervical, thoracic, and sacral injuries. Kemp et al. concluded that any clinical or radiological indication of spinal injury warranted an MRI and therefore in children undergoing brain MRI for a suspicion of abusive head trauma one should always consider to include a MRI of the spine (Fig. 6.9a–c) [59]. In the latest update of the imaging guideline of the Royal College of Radiology this has been incorporated as the standard (RCR 2019).

Jauregui et al. evaluated the findings in 22,192 patients under the age of 18 years with a diagnosis of vertebral column or spinal cord injury (2000–2012) [60]. One hundred sixteen children (0.5%) were diagnosed with abuse, most commonly physical abuse (75.9%). Non-accidental circumstances were more often present in children under the age of 2 years and in girls ($n = 76 < 2$ years and $n = 40 \geq 2$ years). Non-accidental vertebral fractures were more frequently located at the thoracic level (40.5%) and at the lumbar level (28.4%) compared to the cervical level (13.8%). There was an increased risk of thoracic and lumbar spinal fractures in the abused group compared to the non-abused group. Jauregui et al. concluded that the presence of a vertebral fracture in children below the age of 2 years without a plausible explanation should raise the suspicion of abuse.

6.3.2.2 Non-accidental Spinal Injuries and Associated Injuries

In most cases non-accidental spinal injuries are a coincidental finding in the screening or assessment for a suspicion of abuse (Fig. 6.10). Therefore, there are usually other findings that raise a suspicion of abuse, such as cutaneous lesions, other fractures, intracranial injuries, or injuries of internal organs [55, 61, 62]. These injuries may occur in the same moment, but repeated incidents of abuse are common.

Fractures of the upper extremities may occur at the same moment as vertebral compression fractures. For example, as reported by Akbarnia and Campbell, when a child is held over a hard surface and is slammed down with great force on its bottom [20]. In these cases, fractures of the upper extremities may be sustained in two ways. Firstly, when a child uses the fully stretched arms to break the trauma against the table, and secondly, when the maltreating person uses the arms as levers to lift the child up. Classic metaphyseal lesions and rib fractures are other common fractures in cases of non-accidental vertebral fractures [59].

Besides vertebral fractures, several other types of spinal injuries can be present in cases of abuse. Soto-Ares et al. evaluated the findings in 13 children (mean age 15.3 weeks) with suspected abusive head trauma [63]. Eight of these children had an MRI of the spine. In two of them a spinal epidural haematoma was found. No spinal fractures were reported.

Fig. 6.10 Multiple vertebral fractures at levels T11, T12, and L2 in an abused infant

Koumellis et al. (2009) examined the incidence of spinal pathology in 18 infants with abusive head trauma by using routinely MRI of the whole spine in addition to the brain. They found spinal subdural collections in eight of the 18 infants (44%). All were clinically occult. In six infants the collections were large and extended from the cervical to the sacral region. All eight infants with spinal subdural collections had associated supratentorial and infratentorial subdural haematomas. Two infants had a thoracic spine fracture, of whom one had a small epidural haematoma. Edelbauer et al. evaluated the findings (ultrasound, CT and/or MRI, and skeletal radiography) in six infants (mean age ± SD 3.3 ± 1.5 months) with suspected abusive head trauma and compared these findings with the findings in 12 healthy infants (mean age ± SD 2.5 ± 1.4 months) in whom an ultrasound of the spine was performed to exclude spinal dysraphism served as controls [64]. All infants with suspected abusive head trauma had intracranial subdural haematomas and spinal subdural haematomas. The size of the spinal subdural haematomas varied and extended from the cervical spine to the cauda equina. All spinal subdural haematomas were asymptomatic and detected by diagnostic ultrasound. There were no fractures visible on the plain X-rays of the spine in these infants. Spinal subdural haematomas were not observed in the control group. Choudhary et al. (2012) compared the occurrence of spinal subdural haemorrhages in children with abusive head trauma (n = 252, aged 0–2 years; 1997–2009) with the occurrence in children with well-documented accidental head injuries (n = 70, aged 0 to 2 years; 2003–2010) [65]. 67 of the 252 children with abusive head trauma (26.6%) had spinal imaging results of at least on spinal region, that could be evaluated. 31 children had spinal subdural haemorrhage (46%). Seven of the 29 children with cervical imaging had cervical spine subdural haemorrhages (24%). All children with spinal subdural haemorrhage also had intracranial supratentorial and posterior fossa subdural haemorrhages. In one of the 70 children with accidental head injuries an isolated spinal subdural haemorrhage was reported, at the thoracolumbar level. No cervical subdural haemorrhages were observed.

The occurrence of epidural haemorrhages in the spinal canal without the presence of fractures is very rare. Even if these haemorrhages are large, they may resolve spontaneously [66]. Rangwala et al. state that spinal epidural haematomas are much less common than spinal subdural haematomas in children with abusive head trauma [66]. They described the occurrence of a spinal epidural haematoma in a 2-year-old boy after a non-accidental trauma. The boy had no focal neurological deficits. Follow-up imaging studies showed spontaneous resolution of spinal epidural haematoma. Rabbitt et al. identified 76 children under the age of 5 years, who were evaluated for a suspicion of abusive head trauma with presence of MRI of the spine [67]. Forty-five children were diagnosed with spinal injuries (59%). Forty-seven children were eventually diagnosed with abusive head trauma of which 29 had an abnormal spinal MRI (62%). In 29 children abusive head trauma could not be confirmed, of which 16 children had an abnormal spinal MRI. Spinal injuries included ligament injury (n = 32, 42%), muscle oedema (n = 29, 38%), soft-tissue swelling (n = 24, 32%), spinal subdural haemorrhage (n = 12, 16%), spinal epidural haemorrhage (n = 6, 8%), and bony injury (n = 4, 5%). Spinal injuries were associated with more severe intracranial injuries. Spinal subdural haemorrhage was the only finding associated with a combination of retinal haemorrhages, non-contact head injuries (severe repetitive acceleration-deceleration/shaking), and a diagnosis of abusive head trauma. Spinal subdural haemorrhage was associated with other spine injuries but not with intracranial haemorrhage.

6.3.2.3 Spinal Injuries in Fatally Abused Children

According to several authors spinal injuries, spinal cord lesions, and vertebral fractures are, if a thorough post-mortem examination is performed, regularly found in infants and young children who have died of abusive head trauma [49, 68, 69]. Serinelli et al. found spinal injuries in 19 of the 51 cases of homicide due to child abuse of subjects <3 years old (37%), most commonly a subdural haemorrhage was reported [69]. Spinal injuries were mostly located in the thoracic area (n = 17, 33.3%), followed by the lumbosacral area (n = 14, 27.5%), and the cervical region (n = 13, 15.5%).

Kleinman and Marks evaluated the findings of four fatally abused infants and young children (aged 7–36 months) with a total of ten vertebral body fractures [70]. Three children died of inflicted intracranial injuries and one after abandonment. They found three pure vertebral body compression fractures, two superior end-plate fractures without compression deformity, and five anterosuperior end-plate fractures with associated compression deformity. Vertebral compression was generally mild (less than 25%).

Spinal injuries are not only found associated with serious and/or fatal abusive head trauma, but can also be found in children with other serious and/or fatal injuries due to abuse. Dudley and Garg described the findings in an initially unexplained death of a previously healthy 30-month-old child [71]. The foster father stated that he had found her unresponsive after a 2-h nap. She was transported to the hospital and died in the emergency room. Post-mortem skeletal survey, MRI of the brain, and retinal examinations were negative for injuries. The autopsy showed massive retroperitoneal haemorrhage, transections of the abdominal aorta and inferior vena cava, and complete tears of the anterior longitudinal ligament of the spine at C5-C6 and L1-L2 with diastases of the vertebral bodies at the corresponding intervertebral disc spaces. It was concluded that the autopsy findings matched a non-accidental trauma. The uncle admitted to punishing the girl by pinning her down by her flanks in the prone position and hyperextending her legs over her back, causing her feet to touch the back of her head.

6.4 Cervical Spine Injuries

6.4.1 Specific Aspects of Cervical Spine Injuries

Cervical spine injuries, including fractures, are rare in the paediatric population and occur in around 1.5% of injured children [12, 15, 72]. Cirak (406 children with spinal injuries) showed that in all age groups the upper CWK (C0-C4) was approximately 3 times more involved than the lower CWK (C5-C7) [5]. In a study of 206 young patients with spinal trauma (0–9 years) children younger than 4 years cervical spine injuries were more common compared to 4–9 year olds [13]. In a study of 1098 patients with cervical spine injury upper cervical spine injuries occurred in all age groups but lower CWK more common in >8 years of age [12].

They can be associated with significant disability and mortality [10, 73]. Although rare, cervical spine injuries are the most common spine injuries in the paediatric population [4, 5, 9, 10, 74]. Cervical spine injuries can be sustained before, during, and after birth.

The cervical spine is more vulnerable in younger children than in adolescents or adults [75, 76]. This higher vulnerability can be attributed to a combination of reasons: the relatively high weight of the head, the larger head compared to the whole body size, flatter vertebral bodies, lesser muscular development, and the ligamentous laxity [7, 75]. Besides that, the great flexibility of the neck in younger children allows the spinal cord to be damaged without a visible spinal fracture on CR or CT (SCIWORA—Sect. 6.1.2.3) [76]. Despite this higher vulnerability, considerable force is needed to fracture the cervical spine in children.

The cervical spine is difficult to assess in children using conventional radiography, especially for the untrained clinician/radiologist. This may result in underdiagnoses of fractures. Various authors reported on problems related to diagnosing fractures of the cervical spine.

Swischuk reported that prevertebral oedema seen on a radiograph may be the only indication that the spine had been damaged, since spontaneous reduction of the cervical spine after dislocation is common. It is important to note that in crying infants ‘pseudo prevertebral oedema’ can also be been seen (Fig. 6.11a, b).

Thomas et al. described a 9-week-old boy with a spinal cord injury due to a fracture of the C3 vertebral body [77]. The infant presented as a ‘floppy infant’ (hypotonia and no spontaneous arm movements). Routine radiographs of the cervical spine looked normal. However, the MRI revealed an injury with dislocation of part of the cartilaginous part of the body of the third cervical vertebra into the spinal canal. The child also had multiple fractures (rib, clavicle, ulna, and metaphyseal lesions).

Easter et al. stated that cervical spine injuries tend to occur in different locations compared to adults, and that these injuries are more difficult to identify based on history or physical examination because of lack of symptoms [78]. Additionally, the authors stated that radiographs will not identify all cervical injuries in children and that CT has higher sensitivity.

In adults the use of triage tools for imaging cervical spine trauma, in order to overcome overuse of imaging, has been advocated. For paediatric trauma patients Slaar et al. published a Cochrane review on triage tools for detecting cervi-

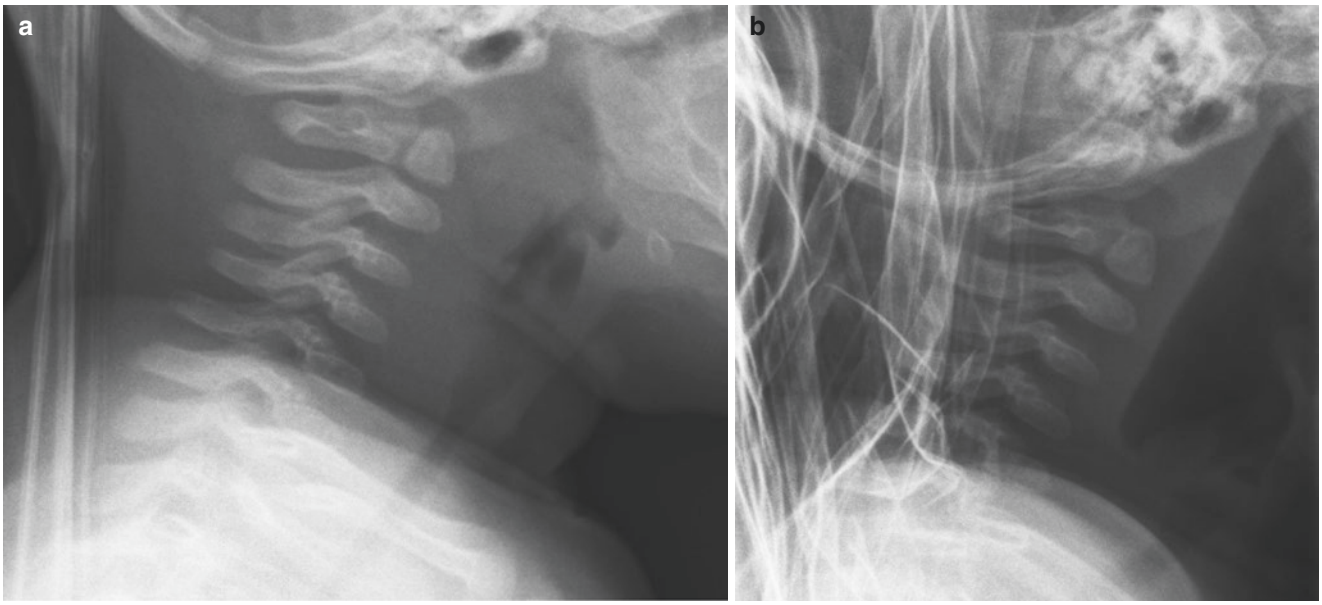


Fig. 6.11 (**a**) Lateral cervical spine radiograph in crying infant, note the prevertebral soft-tissue swelling. (**b**) Same child after consoling, note normalization of the prevertebral soft tissue

cal spine injury [79]. Their review showed that there are only a few studies assessing the diagnostic test accuracy of the two main triage tools, i.e. the National Emergency X-Radiography Utilization Study (NEXUS) criteria and the Canadian C-Spine Rule (CCR) in children. Currently, the evidence is insufficient to assess the accuracy of the CCR to detect cervical spine injury in children. The confidence interval of the sensitivity of the NEXUS criteria between the included studies showed a wide range with a total of four false negative test results. This means that if the NEXUS criteria are used in the paediatric setting cervical spine injury can still be missed.

Gopinathan et al. reviewed the literature concerning cervical spine injuries in children and found that the majority of cervical spine injuries in children occur between the skull and C4 and that in many cases C1 and C2 are involved [15]. Atlanto-axial injuries were found 2.5 times more often in children than in adults.

6.4.2 Manner of Cervical Spine Injuries

6.4.2.1 Trauma Before Birth

Cervical spine injuries due to trauma in utero have only very rarely been reported in the medical literature [80–83]. According to Hernandez-Marti et al. and Caird et al. these intrauterine injuries are caused by hyperextension of the foetal head and breech presentation in utero [80, 84].

Cervical spine injuries due to trauma in utero should be differentiated from findings that Blount et al. had described as spontaneous cervical and thoracic spinal epidural hematoma [85]. It should also be differentiated from atrophy of the cervical spinal cord due to an intrauterine ischaemic infarct in the C4-C6 region of the anterior spinal artery [86]. Currently, isolated cervical fractures that occurred in utero have not been described.

6.4.2.2 Trauma During Birth

Cervical spine injuries can be sustained during birth [87]. MacKinnon et al. evaluated 22 neonates with birth-related spinal cord injuries [88]. In 14 neonates the injuries were located above C4, in six neonates between C4 and Th4, and in two neonates in the thoracolumbar region. All 14 neonates with upper cervical spinal cord injuries were born head first and underwent attempted forceps rotations of the head, forceps delivery of the head or both. Two neonates had spinal subdural haemorrhages, one neonate had a fracture with dislocation. All six neonates with cervicothoracic spinal cord injuries had breech presentations.

Cervical spinal cord injury is most probably caused by hyperextension of the foetal head in utero and during delivery (Fig. 6.12) and with forceps-assisted breech vaginal deliveries, often with entrapment of the foetal head [84, 89]. Spinal cord injury can also occur in face presentation with hyperflexion of the cervical spine (Fig. 6.13) [89].

Other circumstances than position during birth have been described:

Menticoglou et al. evaluated the findings in 15 neonates with high cervical spinal cord birth injury and found that this injury was a rare but specific complication of forceps rotation of the head of 90 degrees or more from occipitoposterior or occipitotransverse position [90].

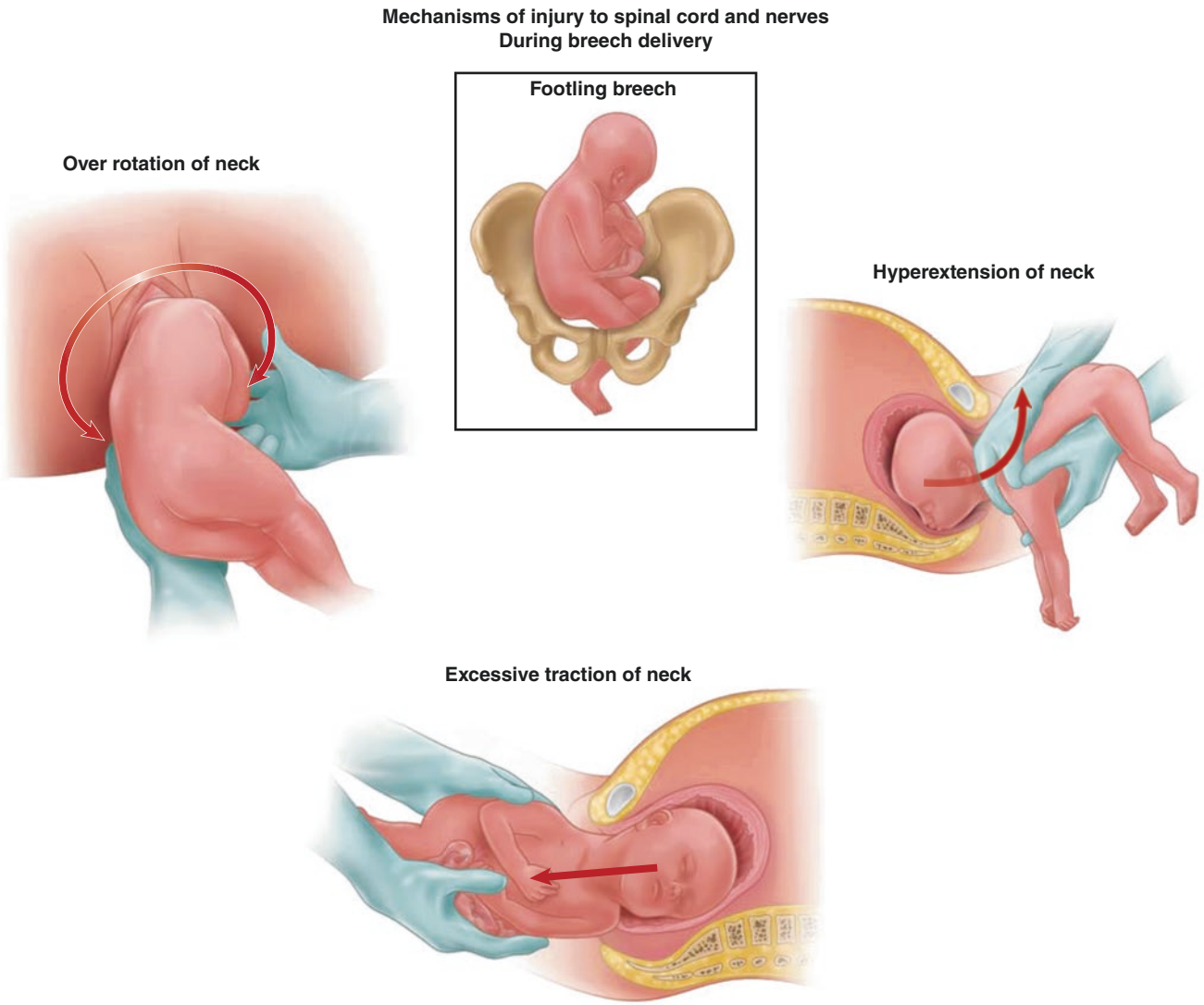


Fig. 6.12 Hyperextension of the cervical spine during breech delivery

Saleh et al. described a 2-week-old boy with spinal cord injury and C4-C5 spondyloptosis (with the body of C5 being completely anterior to the vertebral body of C4), due to trauma during birth [74]. Birth was complicated by previously undiagnosed abdominal dystocia, which required significant force applied to the boy's head and neck for extraction. The dystocia was caused by hydronephrosis (due to posterior urethral valves) and rupture of the left-sided kidney causing significant abdominal ascites.

Spinal cord injury may be complete or incomplete. Cervical spine injuries can be fatal, especially in case of a high cervical spine injury [91]. Forceful hyperextension of the neck can also result in injuries to the ligaments at the craniocervical junction [92].

Birth-related cervical spine injuries most commonly occur in the absence of bony injury [84]. Fractures and dislocations, due to birth trauma, have been reported, but are rare (Fig. 6.14a, b) [84, 87, 89, 93–95].

Stanley et al. described four infants with fracture dislocations of the cervical spine after traumatic delivery [93]. In three of the cases, the delivery was complicated by a shoulder dystocia and in the fourth case the delivery was a difficult breech delivery.

Caird et al. reported a neonate with spinal cord birth injury and a cervical fracture dislocation, following a complicated footling breech vaginal delivery (a footling breech delivery is a delivery in which one or both of the feet of the foetus are born first instead of the pelvis) [84].

Sheil et al. reported an eventually fatal birth trauma of a neonate with an enlarged abdomen due to a previously undiagnosed abdominal teratoma [94]. During vaginal delivery of the head, the remainder of the body lodged in the birth

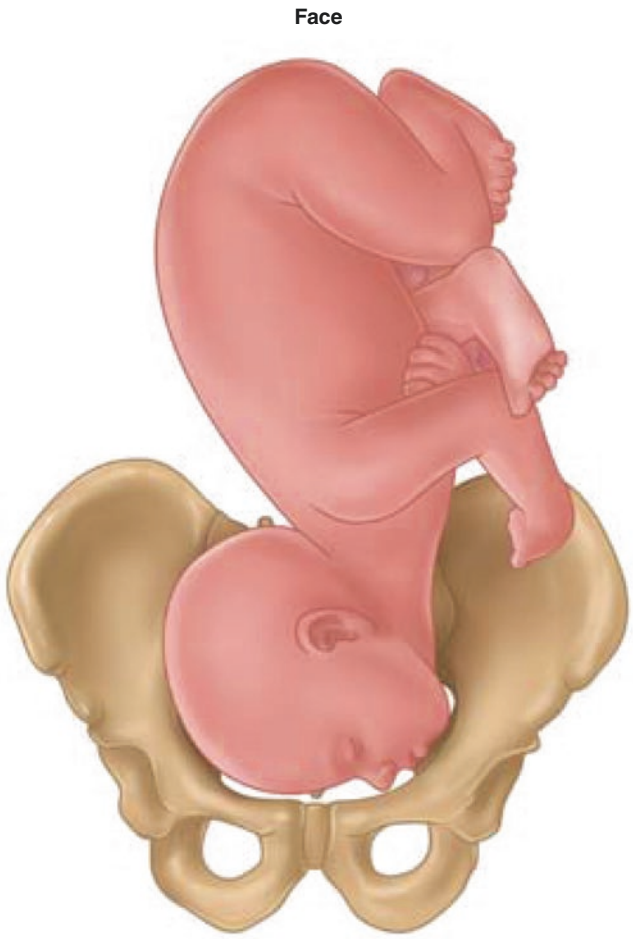


Fig. 6.13 Hyperextension of the cervical spine during face presentation

canal. After an episiotomy the midwife attempted to pull the head and the midwife reported a 'pop'. A caesarean section was performed and the neonate was delivered and diagnosed with a C5-C7 dislocation fracture and subtotal transection of the spinal cord. The spine was displaced posteriorly at the C5-C7 level, and the superior cervical spine was angled forward.

Often there are neurological sequelae and/or early child death. Vialle et al. evaluated the findings in nine children with birth-related spine injuries, of whom six had cervical spine injuries [89]. The pregnancy was uneventful in all cases. Three children with upper cervical spine injuries died before the age of 6 years. The six remaining children (including three children with non-cervical spine injuries) experienced no neurological improvement. Salek et al. reported on an infant who during birth sustained a C4–5 spondyloptosis with on MRI imaging findings in keeping with spinal cord transection at that level [95]. The cervical spine underwent open reduction and fusion using autologous rib grafts and a mini-absorbable plate, at the age of 4.5 years he was able to speak, walk with orthotics and use both his arms and hands.

6.4.2.3 Trauma After Birth: Accidental Circumstances

In children, most cervical spine injuries are sustained in significant accidental trauma such as motor vehicle accidents and falls [96, 97]. In young children, however, a relatively minor fall of less than 1.5 m can result in cervical spine injury [26]. In teenagers and adolescents, fractures occur mostly during participation in sports and traffic [97].

McGrory et al. reviewed the data of 143 paediatric patients, aged 2 months to 15 years, with cervical spine injuries [97]. Children under the age of 11 years had fewer injuries in total and were most often injured in falls. These children had less fractures than children between 11 and 15 years, but had more ligamentous injuries of the cephalic portion of the cervical spine with subluxations and dislocations of the upper cervical spine. They also had a high rate of mortality as a consequence of injury to the spinal cord. Children between 11 and 15 years of age had more injuries in total and were most often injured during sports and recreational activities. They had a higher male-to-female ratio. They were more frequently injured in the caudal portion of the cervical spine, and had a pattern of injury similar to that of adults.

Brown et al. analyzed the data of 103 consecutive paediatric patients with cervical spine injuries and found that the most common circumstances were motor vehicle-related trauma (52%), followed by sports-related trauma (27%) [73]. The cause was correlated to age; younger children sustained cervical spine injuries most commonly as a result of motor vehicle-related trauma and adolescents most commonly as a result of sporting activities. Sixty-eight percent of all children sustained injuries to C1 to C4; 25% to C5 to C7; and 7% to both. Thirty-eight percent of the children in their study group had SCIWORA at the cervical level. In their study group the type of cervical spine injury was related to the circumstances under which the injury was sustained. SCIWORA was present in 75% of children with a sports-related injury and in all children (n = 3) with injury as a result of non-accidental circumstances (child abuse). Cervical spine dislocations occurred most commonly in motor vehicle-related trauma (especially among pedestrians), while cervical spine fractures most commonly occurred in falls and dives.

Leonard et al. reported on cervical spine injuries in 540 children under the age of 16 years (5-year retrospective study) [72, 98]. They compared the findings in 3 age groups: under the age of 2 years (n = 27), 2–7 years old (n = 140), and 8–15 years old (n = 373). In the group of children under the age of 2 years and the group of 2–7 years old children cervical spine injuries were most commonly sustained in motor vehicle accidents (respectively in 56% and 37% of the children). Children in these age groups were more commonly injured in the axial region (occiput to C2) (respectively in 74% and 78% of the children). In the group of children, aged 8–15 years,

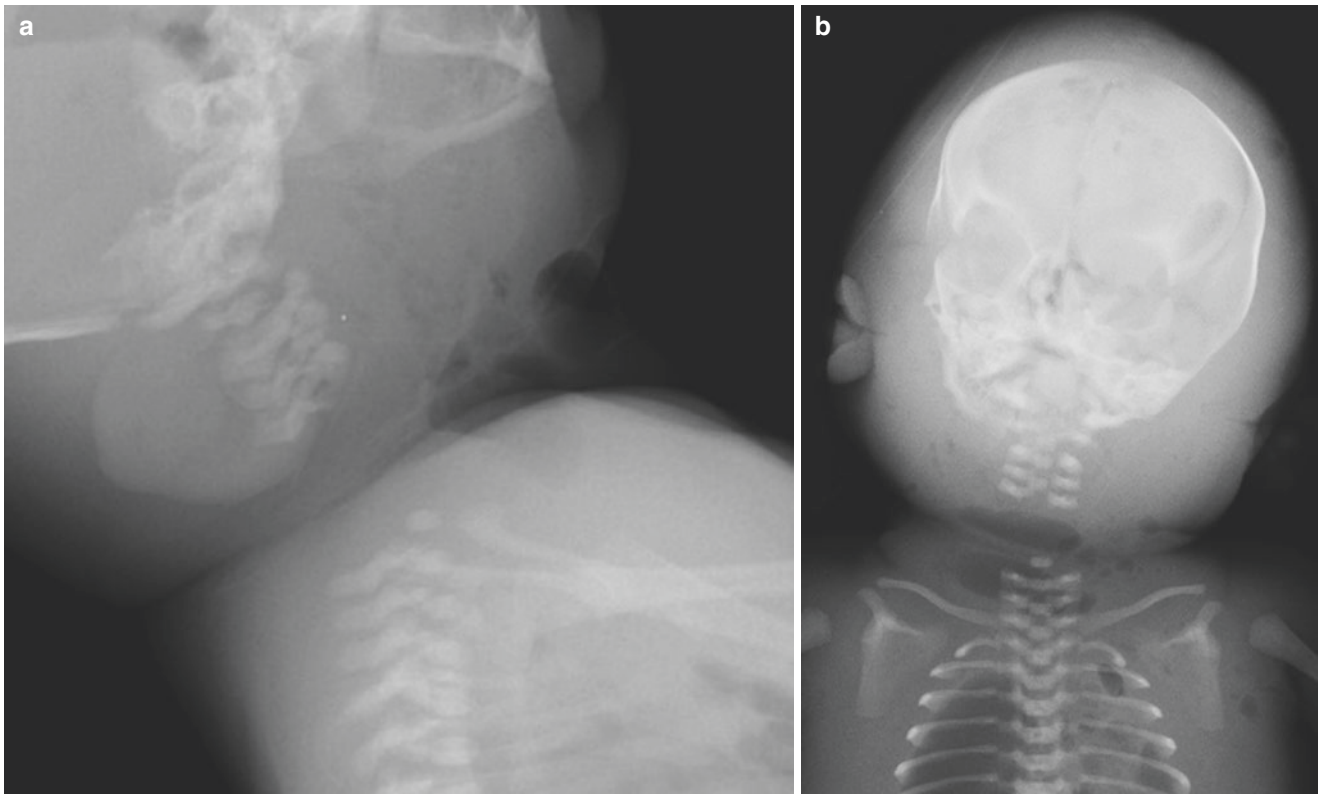


Fig. 6.14 Fatal cervical distraction fracture in two neonates as a result of birth trauma (**a**) lateral radiograph of the cervical spine shows a distraction of the cervical spine at the level of C4 with a bilateral fracture of the pedicles. (**b**) AP radiograph of the cervical spine shows a distraction of the cervical spine at the level of C3 and free air in the neck as a result of a transsection of the trachea. Also, note the oedema of the head

cervical spine injuries were sustained equally in sports-related trauma (23%) as in motor vehicle accidents (23%). Fifty-three percent of the injuries were located subaxially (C3 to C7). Neurological deficits were found in 21% of all children and 7% of the children died.

A specific type of cervical spine fracture is the so-called 'hangman's fracture' or traumatic spondylolisthesis of the axis [99, 100]. This type of fracture was first described by Wood-Jones in 1913 and was typically seen in judicial hanging, in which the body takes a fall [101]. In 1965 Schneider et al. introduced the term 'hangman's fracture [102]. Sumchai and Sternbach defined the hangman's fracture as a bilateral avulsion of the pedicles or their synchondroses from the C-2 vertebral body, frequently with anterior dislocation of C-2 or C-3 [103]. This fracture is caused in children as well as in adults from extreme hyperextension of the head in relation to the cervical spine. This may or may not result in subluxation of the second cervical vertebra in relation to the third. In adults, a hangman's fracture occurs mainly in sports or traffic accidents. In suicides, this type of fracture is not seen that often, since the height and subsequently the energy of the fall during hanging is insufficient to cause a fracture. In these cases, asphyxia is generally the cause of death. Irrespective of the circumstances (accidental or non-accidental) a hangman's fracture is rare in children. The fracture usually occurs in accidental circumstances in children over the age of 2 years [104, 105]. Only a few case reports of younger children are available.

Weiss and Kaufman describe a 12-month old, previously healthy girl, who struck her head while a passenger in an automobile involved in an accident. She had an abrasion of the left side of the forehead and was irritable. Initially, the radiographs were read as normal but because of persistent clinical findings repeated radiographs of the cervical spine were obtained and these showed a clear fracture through the neural arch of C2 [106].

Sumchai and Sternbach report a 7-week-old girl was cradled in the arms of an adult in the backseat of a car traveling at moderate speed when the driver lost control of the car. The car rolled over an embankment before coming to rest. The infant was observed to have been thrown backward onto the floor. The infant was diagnosed with a cranial contusion. Radiographic imaging was not performed. At home the child was irritable, especially when lifted by her arms. She lost the ability to support her head in prone position. A lump was noted in her neck and due to persisting irritability the child was re-evaluated. Radiographic imaging showed a hangman's fracture [103].

Parisi et al. report a 3-month-old infant with a subtle hangman's fracture after a motor vehicle accident. The child was not restrained and impacted against the windshield. Initial radiographic imaging was negative. Due to poor feeding and crying, especially during movements, the child was re-evaluated and diagnosed with a hangman's fracture [107]. The traumatic nature of the finding was confirmed by serial plain films and CT.

Finnegan and McDonald describe a 5-month-old girl, who sustained a fracture in a motor vehicle accident in which both parents were injured and the car was demolished [108]. The girl was observed in hospital overnight, but no injuries were found and she was sent home. The child was presented again 1 week later because the girl was no longer able to hold her head up when prone. She also was unusually irritable. No bruising of the head or neck was found during physical examination. The neurological examination also showed no abnormalities.

A hangman's fracture should not be confused with primary spondylolysis of the axis vertebra [107, 109, 110]. Williams et al. reported a congenital defect of C2 in a 2-year-old boy [111]. The defect was mistakenly diagnosed on plain film and CT as a hangman's fracture. Montalbano et al. mentioned a normal variant as a possible mistake [100]. According to them a normal anterolisthesis of C2, which can be seen in younger children, can mimic anterolisthesis as can be seen after traumatic spondylolisthesis. Even in case of findings suspected to be a hangman's fracture due to non-accidental circumstances one should always consider a possible alternative explanation. Van Rijn et al. reported on a 5-month-old infant who had been physically abused by one of her carers [112]. The girl showed defects at the C2–C3 level, which led to the suspicion of a 'hangman's fracture'. Physical examination did not show any soft-tissue swelling or haematomas. Neither were there any neurological defects. However, there was some local tenderness. In the end, it was concluded that the girl had a congenital defect of the arch of C2.

6.4.2.4 Trauma After Birth: Non-accidental Circumstances

Clinical Aspects and Epidemiology

Only occasionally reports are found in the medical literature concerning cervical spine injuries due to non-accidental circumstances, but these non-accidental injuries may be under-reported [50, 51, 113–117]. Cervical spine injuries can already occur in non-accidental circumstances at a very young age. In most children other (often severe) inflicted injuries are found:

Rooks et al. report on a 3-month-old child (part of a twin), who was brought to the emergency room because of fever and upper respiratory symptoms. The child was irritable but consolable and showed no abnormalities during physical examination. Chest radiographs showed multiple rib fractures. The child was admitted for further child abuse evaluation [117]. Skeletal survey showed 20 fractures such as a left distal radius and ulna fracture, multiple classic metaphyseal lesions, and a sternal depression fractures. The cervical spine series, including MRI, showed a mild spinal cord compression due to a C4-C5 anterior subluxation and a compression fracture of C5. The twin sister also received radiographic imaging and was diagnosed with a C5-C6 fracture dislocation with mild spinal cord compression. She also had multiple rib fractures and metaphyseal lesions.

Ghatan and Ellenbogen describe a 24-day-old girl who was a victim of violent shaking. She was diagnosed with a high cervical injury (atlanto-axial subluxation and dens fracture with compression of the upper cervical spinal cord), due to the violent shaking, which was admitted by the father [51]. The infant also had intracranial injuries (subdural haematoma, hypoxic-ischaemic brain damage), eight bilateral rib fractures and a hip dislocation, abdominal bruising, and a laceration of the liver.

Harmon et al. report a 10-week-old girl who presented to her paediatrician with wheezing and irritability. The paediatrician noticed the child was not moving her right arm. Multiple paraspinal bruises and scars were noted on the torso. Radiographic imaging showed bilateral posterior rib fractures, bilateral femoral fractures, and metaphyseal lesions of the right fibula and left femur. In addition, there was a large calcified mass in the prevertebral soft tissue. Cervical spine CT showed a precervical mass without osseous lesions. MRI showed a compression fracture at C5 with a heterogeneous precervical mass, biopsy and ultrasound confirmed the diagnosis of myositis ossificans circumscripta of the neck secondary to non-accidental trauma [118].

Holland et al. describe a 3-week-old infant was presented with diminished movement and irritability. The father reported that the child had fallen from his arms and struck her head on a changing table 2 days prior. Physical examination showed a lethargic child with bruising on the child's neck. Radiographic imaging showed a severe subaxial cervical fracture dislocation, bilateral rib fractures, and a distraction injury between C-5 and C-6 [115]. Further CT and MR imaging showed severe distraction at C5-C6 and a near-complete spinal cord transection resulting in quadriparesis. Follow-up MRI at the age of 2 years showed severe spinal cord injury with evidence of bilateral C5 nerve root avulsions.

Avulsion fractures of the spinous processes of the lower cervical and higher thoracic spine (called a clay-shoveler's fracture in adults) have been described as being caused by (inflicted) hyperflexion. This type of fracture is found predominantly in older children and adults [48]. Oral et al. described a 4-year-old disabled girl that had suffered an

avulsion fracture of the C2 and damage to the interspinal ligaments between C1 and C2 after the babysitter had thrown her on the bed from a distance of at most 50 cm (1–2 ft) [119]. The girl also had a bruise on her forehead.

Brown et al. reviewed the findings in 103 paediatric patients with cervical spine injuries and found that SCIWORA was present in all infants (n = 3) with non-accidental injuries [11]. In 2 patients cord symptoms were at both C1-C4 and C5–7 levels and in 1 patient at C5–7. All these patients had severe associated lesions caused by non-accidental trauma. One patient died and the 2 others survived with significant deficits.

Feldman et al. reported on 4 infants (aged 1, 2, 4, and 6 months) and a 15-month-old toddler with cervical spinal cord injury, sustained in non-accidental circumstances [114]. Evidence of associated spinal bony injury was often absent or unapparent until healing occurred. Four children had spinal cord injury without (or with minimal) radiological abnormality. All children showed other inflicted injuries, e.g. bruises, burns, intraoral injuries, subdural haematomas, fractures of skull, ribs, clavicula, and metaphyseal fractures.

When a 'hangman's fracture' is found in children, non-accidental circumstances should always be considered, especially when the patient's history does not offer a plausible explanation, concerning an accidental trauma with severe hyperextension [120–122]:

- Curphey et al. described the post-mortem findings in a 3-week-old girl [123]. The girl was found dead in her bed after her father reported to have picked her up from the bed when she started crying. The girl slipped from his hands and the father said he caught the baby by the neck and head with his hand and heard a cracking noise. Afterwards, he gave the girl her bottle and put her back to bed. Six hours later she was found dead. The girl had no visible injuries, except for a deformity of the left thigh with pronounced swelling, due to fracture of the left thigh with displacement of the fracture ends and formation of soft callus. Also, fractures of the right third rib and the left first rib with hard callus formation were found. The anterior aspect of the entire cervical spine was haemorrhagic. There was a recent fracture dislocation between C2 and C3 and a recent fracture separation between Th2 and Th3 with extensive interstitial haemorrhage. The father eventually admitted that he had squeezed and twisted the child's neck on the day of the girl's death. He had heard a snapping sound while doing this.
- McGrory and Fenichel described a 4-month-old infant with a hangman's fracture, due to repetitive rapid alternation of hyperextension and hyperflexion during shaking at the age of 4 weeks [124].
- Gille et al. reported an 11-month-old girl with a painful neck, due to a non-accidental fracture of the axis pedicles [120]. She also had an old humerus fracture.
- Kleinman and Shelton evaluated the findings in a 6-month-old boy who was admitted to the hospital with respiratory distress was diagnosed with 27 rib fractures (different ages) and multiple other injuries, including an old clavicular fracture and metaphyseal fractures, which were highly suspicious for non-accidental circumstances [121]. The C2-C3 subluxation was an unsuspected finding on the skeletal survey. According to the authors the subluxation could probably have been caused by hypertension of the cervical spine during shaking.
- Ranjith et al. reported a 23-month-old girl, who was admitted to hospital because of a 5-day history of irritability and general malaise [122]. According to her father, she was reluctant to move her neck. Imaging of the cervical spine showed a hangman's fracture of C2 with slight anterior subluxation of C2 on C3. The father stated that no trauma occurred. It was suspected that the fracture occurred in non-accidental circumstances.

Kleinman stated in a comment on the article of Ranjith et al. (2002) that the hangman's fracture might be more common that suggested in the medical literature and that a lateral view of the cervical spine should be included in the routine skeletal survey if non-accidental circumstances are suspected [116].

Non-accidental Cervical Spine Injuries and Associated Injuries

As already mentioned in the previous sections, non-accidental cervical spine injuries are often found in children with other inflicted injuries, such as other fractures, intracranial injuries, spinal injuries, and other systemic injuries [121, 123–126].

Baerg et al. described 53 children under the age of 36 months with inflicted head injuries (median age: 5 months; range: 1 to 35 months) [127]. Seven children (13.2%) died. Cervical spine injuries were identified in 8 children (15.1%): ligamentous injury (2), vertebral artery shear injury (1), atlanto-occipital dissociation (1), spinal cord injury with spinal cord epidural hematoma (2), and isolated spinal cord epidural hematoma (2). One child with cervical spine injury died.

Koumellis et al. (2009) evaluated spinal pathology in 18 infants with inflicted head injury. Cervical subdural hematomas were found in 3 infants (17%). In 2 of these infants the haematomas extended to the sacral region [128].

6.5 Thoracolumbar Spine Injuries

6.5.1 Specific Aspects of Thoracolumbar Spine Injuries

According to Daniels et al. thoracolumbar spine injuries in paediatric patients are caused by a trauma with a high transfer of energy, which may lead to compression fractures, burst fractures, flexion-distraction injuries (e.g. Chance fracture), fracture dislocation injuries, apophyseal fractures/ herniations, and spinous process and transverse process fractures (see also Sect. 6.2) [129]. The majority of thoracolumbar spinal fractures are caused by hyperflexion plus compression [32, 33]. Furthermore, they may also occur due to shearing forces or subluxation-dislocation. Shearing forces are involved in injuries sustained by the impact with a high transfer of energy, such as road traffic accidents in which the child is hit by a car [34].

6.5.2 Manner of Thoracolumbar Spine Injuries

6.5.2.1 Trauma Before Birth

There are no reports on sustained fractures of the thoracolumbar vertebrae, due to trauma before birth. Intrauterine sustained spinal cord injuries have been reported but are extremely rare [81, 85].

6.5.2.2 Trauma During Birth

Several reports are found in the medical literature concerning thoracolumbar spinal injuries (sometimes with fatal outcome), due to a trauma during birth:

Journeau et al. report on a neonate that was born at term by spontaneous vaginal delivery. Presentation was breech following a transverse position. The neonate showed a complete paralysis with a T3 level and was diagnosed with a Salter–Harris Type II fracture with an associated spine dislocation and complete transection of the spinal cord at T3-T4 was seen at radiography and confirmed by MRI [130]. The authors stated that this type of injury is rare and that it can occur in certain obstetric circumstances, like breech transverse presentations or large baby size. This is the only case which reported fractures.

The following cases report spinal trauma during birth, however all without fractures.

Ilagan et al.: Preterm neonate with a thoracic spinal cord transection (Th3-Th4) and paravertebral haemorrhage (according to the authors mimicking a catastrophic intracranial bleed), delivered by caesarean section because of breech presentation [131].

MacKinnon et al.: Twenty-two neonates with birth-related spinal cord injuries of which 6 had cervicothoracic and 2 had thoracolumbar spinal cord injuries. All these eight patients were breech deliveries. Four of the patients with cervicothoracic injury died [88].

Fotter et al. report on two neonates. The first neonate was manually rotated from a breech position after 38 weeks of gestation, but returned back to breech position. Onset of labour was spontaneous after 41 weeks of gestation, but a caesarean section was performed because of hyperextension of the head, a large biparietal diameter and maternal diabetes. The newborn showed flaccid paralysis of the trunk and limbs. The neonate was eventually diagnosed with a complete transection of the spinal cord. The second neonate was born after 40 weeks of gestation and vaginally delivered in double footling breech position. The delivery was reported to be difficult. The neonate was limp, pale, cyanosed, apnoeic, bradycardic, and all reflexes were absent. The neonate was diagnosed with a partial transection of the spinal cord [132].

Le Masne et al.: Two neonates with thoracic spinal cord injuries [133]. The first neonate was delivered by caesarean section, which was indicated because of breech presentation with hyperextension of the neck. After the section she rapidly developed acute respiratory distress and paraplegia. MRI showed spinal cord haemorrhage involving the cervical and upper thoracic cord with rupture of the cord. The patient died a few weeks later. The second neonate was delivered vaginally in breech presentation without any difficulty. She progressively developed tetraplegia evolving into spasticity. MRI showed stretching of cervical spinal cord.

Billingham et al.: Male neonate with an epidural haematoma at the level of T8 and distal cord oedema which extended to the conus medullaris after an atraumatic vaginal frank breech delivery [134]. According to the authors, the spontaneous occurrence of a spinal epidural hematoma could be related to an increase in the abdominal and/or thoracic pressure which they hypothesized resulted in an increased pressure in the anastomotic network of thin-walled and valve less vertebral venous plexus, during the frank breech delivery.

6.5.2.3 Trauma After Birth: Accidental Circumstances

Thoracolumbar spine injuries In children are rare and account for 2–5% of all paediatric spine injuries [129]. In children up to approximately 10 years old, thoracolumbar spinal injuries are most commonly sustained in motor vehicle accidents—either as pedestrian or as passenger in a car—or falls from a height (Fig. 6.15a, b) [32, 135–137]. Fractures in older children are usually caused by either sports or other recreational activities, traffic accidents, or suicide attempts [43–45, 138, 139].

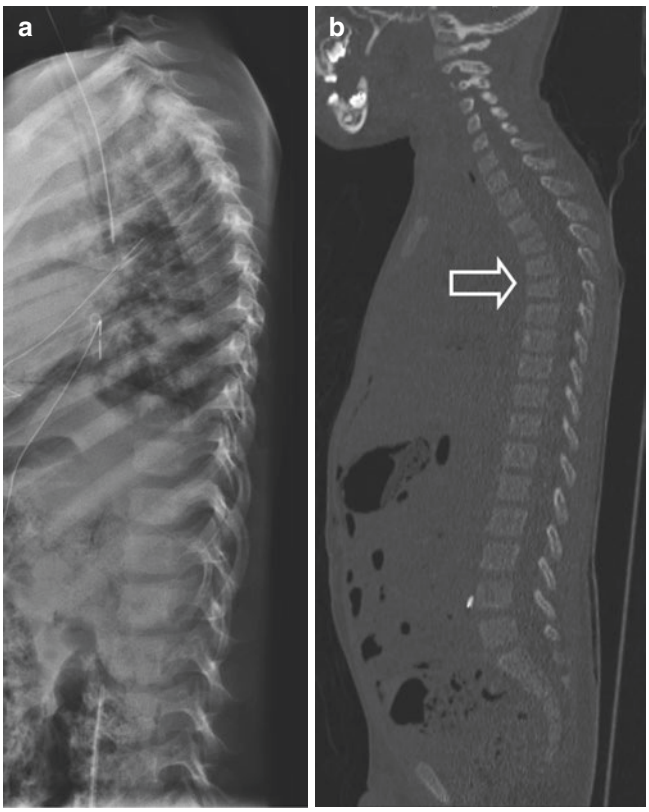


Fig. 6.15 A 3-year-old child who fell from the third floor. (**a**) spinal radiograph during trauma work-up shows thoracic vertebral fractures. (**b**) Post-mortem CT shows vertebral fractures of the fourth and fifth thoracic vertebra (arrow)

Babu et al. evaluated the findings in 90 children with thoracolumbar spinal injuries (mean age was 15.9 ± 3.2 years; range: 2–18 years) (male-to-female ratio 3:1) [140]. Sixty-four children (71.1%) sustained the injury due to fall from height (>10 feet/3 meters) and 18 children (20%) following a motor vehicle accident. The rest of the children sustained injury due to the fall of a heavy object on the neck or diving. The lumbar spine was the most common spinal level injured (n = 48, 53.3%), and fractures were the most common type of injury (n = 84, 93.3%). The most common associated injury was a long bone fracture.

Kraus et al. found that in 86 paediatric patients under the age of 16 years (average age 11.9 years) thoracolumbar spinal fractures were most commonly sustained during sporting activities (n = 46, 53%) and traffic accidents (n = 24, 28%) [141]. Fractures were most often located in the mid-thoracic (n = 40, 47%) and thoracolumbar spine (n = 35, 41%).

Franklin et al. did a retrospective review of 52 patients aged 0 to 18 years with a total of 191 thoracic and/or lumbar compression fractures who had both CT and MRI during the initial trauma evaluation [142]. Only ten patients (19%) had a single-level injury. Of 42 with multiple compression fractures, 34 (81%) had fractures in contiguous levels, and eight had non-contiguous injuries. T4 was the most commonly fractured vertebra. The mid-thoracic spine (T3-T6) and the thoracolumbar junction (T12-L1) were the most affected segment. Fewest fractures were seen at T8 and lower lumbar segments. Comparing CT and MRI, there was complete agreement in the number and distribution of fractures in 23 patients (44%). MRI identified additional levels of fracture in 15 patients (29%); 14 (27%) had fewer levels fractured on MRI than CT. Two patients (4%) with injuries identified on CT subsequently had normal MRI findings. Only one patient (2%) had fractures seen on MRI after a normal CT scan. Complete correlation between CT and MRI was seen in 59% (17/29) of patients aged 11 to 18 years, compared with 26% (6/23) of patients younger than 11.

6.5.2.4 Trauma After Birth: Non-accidental Circumstances

Non-accidental circumstances (child abuse, physical violence) should always be considered, irrespective of age, if a thoracolumbar spinal injury is found in a child, in particular when there is no plausible explanation.

Fractures and Fracture Dislocations

Most non-accidental fractures concern the vertebral body (Fig. 6.16) [20]. They result from a combination of extreme hyperflexion and hyperextension, such as in severe shaking or direct-impact forces. Anterior compression fractures resulting from hyperflexion in shaking are predominantly seen in the thoracolumbar junction and in the lower thoracic and higher lumbar spine [143].

The alternation of extreme flexion and extension during shaking may also cause avulsion fractures of the thoracic and lumbar spinous processes and damage to the interspinal ligaments in infants [48, 53, 143]. Ogden (1990) described multiple fractures of the spinous processes of the thoracolumbar spine due to shaking.

Fracture dislocations of thoracolumbar vertebrae, sometimes with cord compression, and kyphosis due to violence have been reported, but are probably rare (Ogden, 1990; Hobbs et al., 1993) [1–3, 50, 62, 124, 144]:

Cullen reported on five children with spinal injuries after probable maltreatment [1]:

- A 14-month-old boy with a fracture dislocation of the Th12 on L1 and a calvarium fracture. The boy also had a right-sided subdural haematoma and bruising on the right side of the head. There were no signs of spinal cord compression.
- A 16-month-old boy with an older compression fracture of the Th3 vertebral body (the compression fracture was already visible on films that were made 6 months earlier) and fractures of 2 ribs (left, anterior) and a healing fracture of the right femur.

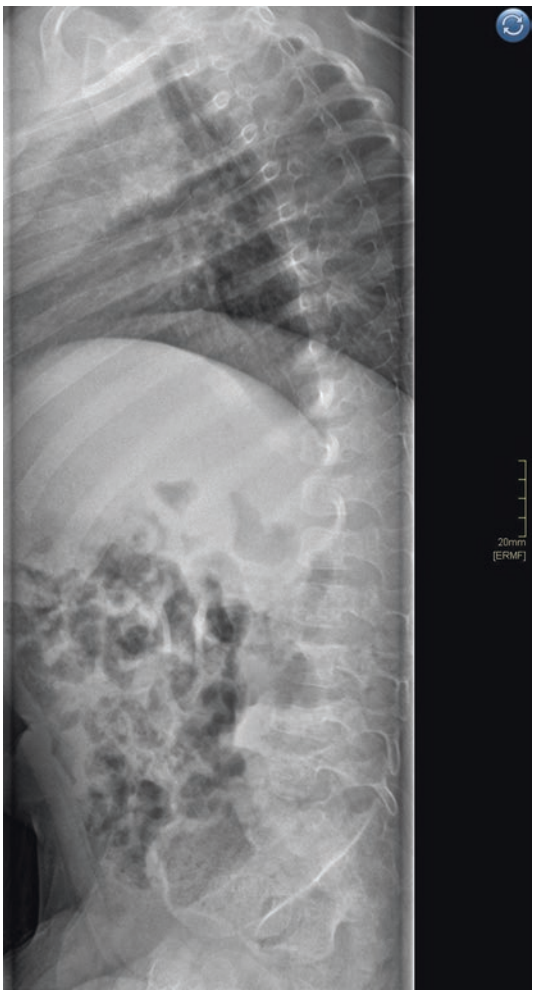


Fig. 6.16 A 11-month-old infant who was suspected to be a victim of child abuse. The radiograph of the spine, as part of the skeletal survey, shows a fracture of the fourth lumbar vertebra

- A 16-month-old girl with kyphosis at the thoracolumbar junction, due to a marked reduction in height of the bodies of Th12 and L1 with narrowing of the intervening disc space. The girl had no other bony injuries.
- A 19-month-old boy with a marked thoracolumbar kyphos, caused by a subluxation of Th11 on Th12, and a left parietal skull fracture, a metaphyseal lesion on the upper end of the left humerus, and recent fractures of the lower end of the left radius and ulna. He also had a scar over the right eyebrow, bruising of the right thigh and right ankle.
- A 36-month-old boy admitted to hospital because of failure to gain weight and retarded speech. Radiographs showed loss of the anterior height of L1 and L2, with disruption of their inferior surfaces and sclerosis anteriorly. The findings were thought to be from trauma. At the age of 24 months he was admitted to the hospital because of 'failure to thrive'. On admission, radiographs showed a recent fracture of the left parietal bone and old fractures of the right capitellum and lower end of the left humerus.

Most reports in the medical literature are single case reports

- Dickson and Leatherman evaluated the findings in a 33-month-old boy, who initially was diagnosed with a progressive dorsolumbar kyphosis (due to wedging of Th12), with no known previous trauma [3]. The injury was later found to probably be due to a non-accidental trauma, described by the authors as parental abuse. The boy also had rib fractures.
- Renard et al. reported a 13-month-old boy who was suddenly noticed to have a paraplegia and an significant kyphosis was diagnosed with a subluxation of Th12-L1 and compression of the spinal cord, initially with a complete paraplegia [144]. The boy made a full recovery from his paraplegia. According to the authors, the subluxation was due to extremely violent spanking.
- Diamond et al. evaluated a 12-month-old girl with a complete fracture dislocation Th12 on L1 [2]. The girl also had an abrasion on the forehead and bite marks on the hand. A hearing in family court confirmed the diagnosis of child abuse.
- Gabos et al. evaluated a 15-month-old girl, who initially was admitted to the hospital because of lethargy, petechiae around the head and neck, a palpable upper lumbar prominence for a week, and paraplegia [42]. The lumbar prominence and the paraplegia were caused by an isolated fracture dislocation (L1-L2) of the lumbar spine with severe spinal cord compression. The mother's boyfriend admitted to abusing the child.
- Tran et al. evaluated the findings in a 6-month-old boy with a thoracolumbar neurocentral synchondrosis fracture dislocation of Th12, associated with compression fractures of C4 and S4, sustained in non-accidental circumstances and most probably caused by massive hyperflexion and axial spinal loading [145]. The boy also had a parietal skull fracture, multiple rib fractures, metaphyseal lesions, and bilateral acromion fractures.
- Bode and Newton described an 8-month-old boy with a fracture dislocation at level Th12-L1 and a Th11-Th12 spinal cord contusion resulting from non-accidental trauma [57]. A 3-year-old child supposedly fell on the boy.
- Sieradzki and Sawark evaluated the findings in a 14-month-old boy with a fracture dislocation at level Th12-L1, due to non-accidental circumstances (thrown across a room and landing in jackknife position) [146]. The boy also had a healed fracture of the right forearm and bruising on the back.
- Lieberman et al. described a child with aortic disruption associated with L2-L3 fracture dislocation after reportedly drowning in a children's pool. The authors suggested the injuries were sustained in non-accidental circumstances, probably due to forced hyperextension [147].

- Webb et al. reported a 29-month-old who died after the child was found unresponsive at home after choking on baby wipes that were found in the mouth [148]. After autopsy it was concluded the child died of blunt force trauma to the back. Internal examination showed a complete fracture of L4 vertebral body and rupture of the anterior longitudinal ligament. The presence of granulation tissue and callus formation indicated prior healing injury with acute re-injury.

According to Levin et al. fractures of the thoracolumbar spine with spondylolisthesis are rarely seen in cases of child abuse [149]. They mentioned only six earlier reports in the medical literature. In a review of seven new cases (age from 6 months to 7 years), they found abnormalities that ranged from a subtle spondylolisthesis to overt dislocation of the vertebrae, generally at the L1-L2 level. In six children paravertebral calcifications were found. In two children, spondylolisthesis was the only confirmation found in imaging.

Carrion et al. reported on two girls (9 and 12 months old) who sustained a circumferential fracture of the growth plate at the thoracolumbar level due to non-accidental circumstances [150]. The 9-month-old girl had a Th11 subluxation with compression on the spinal cord. The 12-month-old girl had an anterolisthesis of Th12 on L1 with local compression of the spinal cord. Both had paraplegic symptoms due to fracture dislocation.

Brink et al. reported a 5-week-old boy with a distraction injury of the thoracic spine, spinal cord transection, and adjacent vascular injury due to non-accidental circumstances (physical abuse) [73].

Spinal Subdural and Epidural Haemorrhage

In recent years there has been more attention to thoracolumbar spinal subdural haemorrhage, and it has been shown to be more common in infants than once thought:

- Gruber and Rozzelle: A 4-month-old boy with a thoracolumbar spinal subdural haematoma (Th10-L4) and spinal cord compression (seen on MRI), due to non-accidental circumstances [151]. A CT scan of his head showed subdural and subarachnoid haemorrhaging. The boy also had retinal haemorrhages and a bruise on the left shoulder.
- Choudhary et al. compared the occurrence of spinal subdural haemorrhages in children with inflicted head injuries (n = 256, aged 0 to 2 years; 1997–2009) with the occurrence in children with well-documented accidental head injuries (n = 70, aged 0 to 2 years; 2003–2010) [65]. Sixty-seven of the 252 children with inflicted head injuries had spinal imaging results of at least one spinal region, that could be evaluated. Thirty-eight of the 67 children had undergone thoracolumbar imaging and 24 of these 38 had thoracolumbar spine subdural haemorrhages. Only 1 of the 70 children (a child with a displaced occipital fracture) with accidental head injuries had a thoracolumbar spine subdural haemorrhage at presentation.
- Koumellis et al. reported on 18 infants, age 1–12 months (mean age 3 months), with AHT of whom 8 (44%) had a subdural collections in the spine [128]. In none of the cases this lead to clinically detectable abnormalities.
- Hong et al.: A 5-month-old boy with bilateral intracranial subdural haematomas, sustained in non-accidental circumstances [152]. Imaging showed a spinal subdural haematoma extending from Th4 to L5 with cord compression.
- Edelbauer et al. presented data on the use of ultrasonography of the spine in 6 cases of non-accidental trauma (age 2.0–6.0 months (median 3 months). Besides the findings on CT scan or MRI in all cases the subdural haematomas were visible on ultrasound.

Thoracolumbar spine epidural haemorrhage is only rarely reported in children. Gosnold and Sivaloganathan reported the coincidental finding of a spinal epidural haematoma and microscopic haemorrhage into the spinal cord at the mid-thoracic level in a fatally abused child [47].

6.6 Injuries to the Sacrum and Coccyx

6.6.1 Specific Aspects of Injuries of the Sacrum and Coccyx

Injuries to the sacrum and coccyx are rare in the paediatric population. Sacral spine fractures account for about 0.16% of injuries in paediatric trauma patients [153]. Often these fractures are combined with associated fractures of the pelvis (Chap. 11) [25].

Transverse sacral spine fractures account for about 3% to 5% of all sacral fractures [25]. Isolated fracture dislocations of the sacrum are very uncommon [154, 155]. Sacrococcygeal fracture dislocation is an extremely rare injury in paediatric patients [154]. Most sacral fractures are not associated with neurologic injury [153]. Fractures of the sacrum are usually caused in otherwise healthy children by a trauma with a high transfer of energy.

6.6.2 Manner of Injuries of the Sacrum and Coccyx

6.6.2.1 Trauma Before and During Birth

No reports were found in the medical literature, concerning injuries of the sacrum and coccyx, due to trauma before or during birth.

6.6.2.2 Trauma After Birth: Accidental Circumstances

Hart et al. evaluated the data of 4876 cases of paediatric trauma at 1 children's hospital over a 7-year period [153]. Eight children (0.16%) had documented sacral fractures. All children sustained sacral fractures due to accidental circumstances, either motor vehicle accidents or fall from height.

Isik et al. reported a 4-year-old girl who was injured in a motor vehicle accident with bruising and tenderness in the abdominal and sacral region [155]. CT showed a dislocation at the third and fourth sacral vertebrae without any fractures.

Hamoud & Abbas reported a 12-year-old boy with severe pain at the area of his tailbone [154]. The boy could hardly walk and was unable to sit or lie on his back. CT showed an anterior fracture dislocation of the sacro-coccygeal joint. The fracture was due to a bad fall off his bicycle.

In 2017 Hamoud and Abbas presented 2 patients with fracture dislocations of the coccyx [156]. The first patient was a 19-year-old man with a complete posterior dislocation of the joint between the first and second coccygeal vertebrae. The man had slipped with his motorcycle on a wet road and had landed on his tailbone. The second patient was a girl aged 2 years and 4 months with a complete anterior dislocation of the first coccygeal vertebra, following a fall on the edge of a step, hitting her tailbone.

6.6.2.3 Trauma After Birth: Non-accidental Circumstances

Just like accidental injuries to the sacrum and/or the coccyx, non-accidental injuries to the sacrum and/or the coccyx are only very rarely reported:

Johnson et al. reported 3 girls, aged 5 months, 3 years, and 5 years with skeletal injuries associated with sexual abuse [157]. The 3-year-old girl had extensive soft-tissue injuries to the arms, legs, and perineum, but also sustained fractures of both pubic rami and the sacral side of the right sacroiliac joint (see also Chap. 11).

Tran et al. reported a 6-month-old boy who presented with a 5-day history of increasing respiratory distress. The father stated he fell with the child one month prior. Physical examination showed mild posterior scalp swelling. He was diagnosed with a thoracolumbar neurocentral synchondrosis fracture dislocation of Th12, associated with compression fractures of C4 and S4, multiple bilateral healing rib fractures, multiple classic metaphyseal lesions, bilateral acromial fractures, and a right linear parietal skull fracture. The injuries were thought to be sustained in non-accidental circumstances and probably caused by massive hyperflexion and axial spinal loading [145].

Barber et al. evaluated American College of Radiology standardized skeletal surveys and neuroimaging studies of 751 children (ages 0–4 years) [158]. One hundred forty-five children (19%) had a positive skeletal survey. Fourteen of these 145 children (2%) had a total of 22 definite spinal fractures, visible on the skeletal survey. Advanced imaging (CT, MRI, and bone scintigraphy) confirmed the fractures in 13 of the 14 children and demonstrated 12 additional spinal fractures. The children had 25 thoracic spine injuries, 6 lumbar spine injuries, and 8 sacral fractures on imaging (7 children had a spinal injury at multiple levels).

6.7 Differential Diagnosis

In the literature, several normal developmental variants are reported that were or easily could be falsely identified as radiological findings suggestive of non-accidental injuries [159]. If radiologists are not familiar with these normal variants inadvertently an incorrect diagnosis could be made.

The first, and most prevalent, finding that can be mistaken for a post-traumatic finding is a pseudosubluxation of C2 on C3 (Figs. 6.17a, b and 6.18) [159–164]. The diagnosis is made if the vertebral body of C2 shows anterior displacement compared to C3 while the posterior cervical line pass through, touches of lies 1 mm in front of the cortex of the posterior arch of C2 (Fig. 6.19) [162]. Shaw et al. evaluated a trauma population of 208 children, aged between 1 and 16 years who were admitted with polytrauma [162]. In their study population, 30 children (21.7%) showed a C2/C3 pseudosubluxation, there was no difference in the age or sex of the patients nor in the presence of an endotracheal tube, the severity of trauma (as scored by the injury severity score and revised trauma score), and the clinical outcome. The patients with a C2/C3 pseudosubluxation did have a significantly lower age, median age 6.5 years versus 9 years ($p = 0.0091$).

Aronica-Pollak et al. described a 4-month-old child with a radiologically reported fracture of the third lumbar vertebra, which was assumed to be a non-accidental fracture [165]. The child died 4 days after being hospitalized. Autopsy did not show a fracture, but a congenital defect in the construction of the vertebrae. The cause of death was determined to be Sudden Infant Death Syndrome (SIDS). Also, a congenital decreased height of a vertebral body may simulate an impression fracture. In those cases, additional MRI is helpful in making the diagnosis (Fig. 6.20a–d).

Van Rijn et al. described a 5-month-old infant who had been physically abused by one of her carers [112]. The girl showed defects at the C2–C3 level, which led to suspected 'hangman's fracture'. Physical examination did not show any soft-tissue swelling or haematomas. Neither were there any neurological defects; however, there was some local tenderness. In the end, it was concluded that the girl had a congenital defect of the arch of C2 (Fig. 6.21a–c). Hill et al. found an apparent C2 posterior arch defect in a child with Menkes disease and decided to evaluate cervical spine radio-

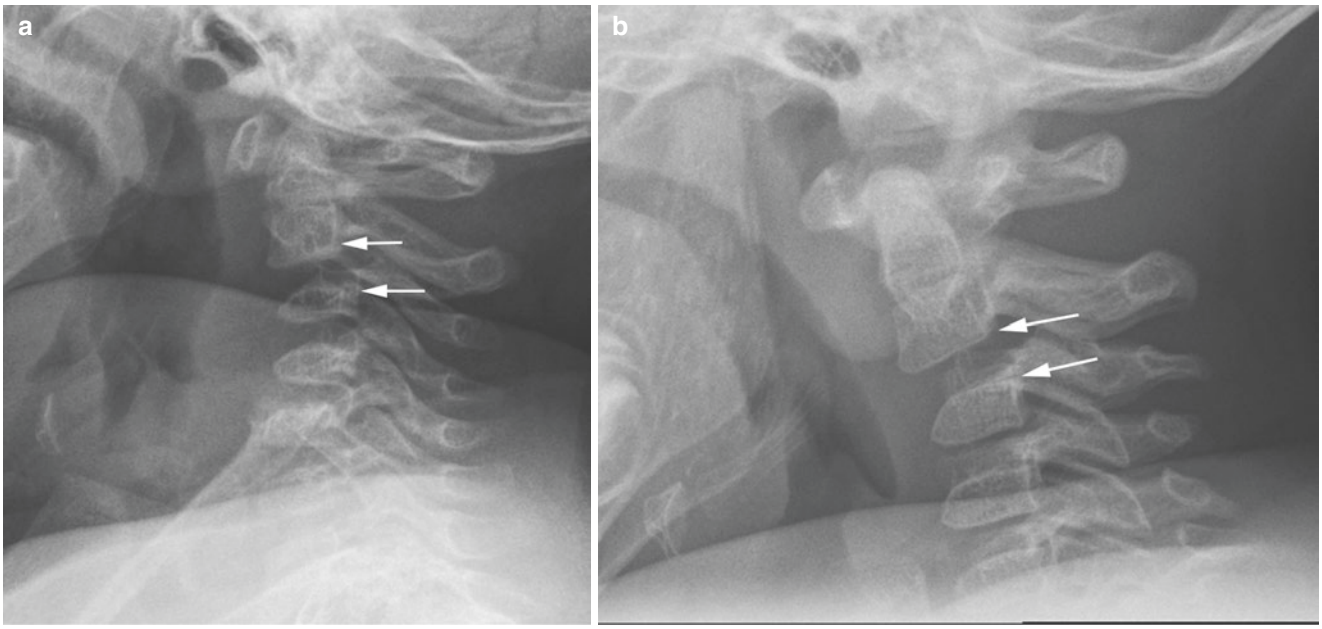


Fig. 6.17 C2/C3 pseudosubluxation in two 9-month-old infants (**a**, **b**) (arrow)

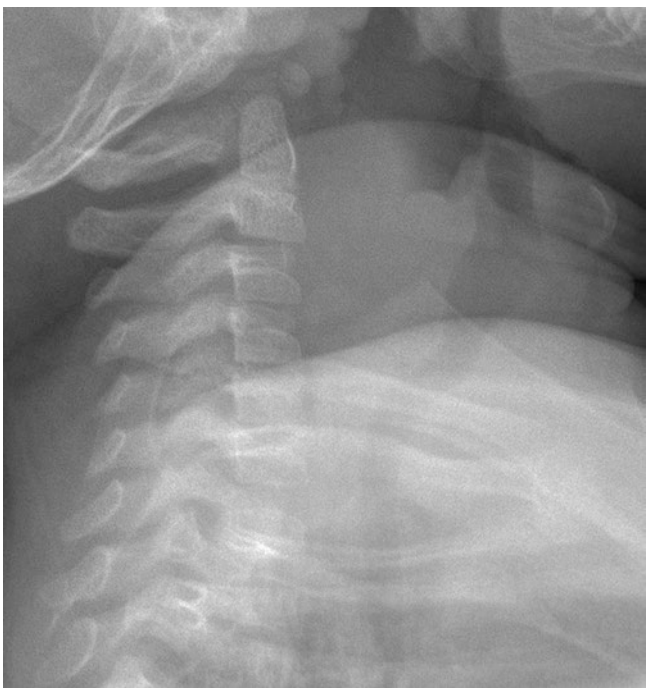


Fig. 6.18 C2/C3 pseudosubluxation in a 14 month old infant

graphs of 35 children with Menkes disease [166]. In four children they found apparent C2 posterior arch defects consistent with spondylolysis or incomplete/delayed ossification. The authors concluded that in a child with Menkes disease, suspicions of inflicted cervical spine injuries should be considered cautiously when a C2 posterior arch defect is found.

Changes occurring during normal development or resulting from infection in the vertebrae and intervertebral discs may also be a reason for confusion of these findings with spinal fractures [3, 20].

Doberentz et al. mentioned the presence in the infantile skeleton of vertical radiolucent bands running through a vertebral body, so-called coronal clefts of vertebrae (Fig. 6.22) [167]. These clefts are most commonly located in the lumbar spine and, according to the authors, should not be confused with spinal signs of child abuse. The authors also stated that coronal clefts show a completely different radiological appearance than usually are seen in compression fractures of the vertebral body. The exact meaning of coronal clefts is not known, but these clefts should be considered to be a normal variant, probably due to retarded ossification of the vertebral bodies in fetal development [167, 168]. Tanaka and Uhthoff concluded that coronal clefts should not be interpreted as a malformation, while, according to Doberentz et al., coronal clefts are found almost exclusively in foetuses with chromosomal aberrations or severe congenital malformations [168, 169]. Westvik and Lachman stated that clefts (most commonly coronal and sometimes sagittal clefts) were often observed in skeletal dysplasias, like atelosteogenesis, chondrodysplasia punctata, dyssegmental dysplasia, Kniest dysplasia, and short rib polydactyly syndrome [170].

Oestreich and Anton reported a normal radiographic finding that, according to them, may be mistaken for a non-accidental injury [171]. This can be seen in the posterior thoracolumbar spinous processes of young infants after the first week of life and it concerns a lucency paralleling the posterior margin of the ossified spinous process, which is

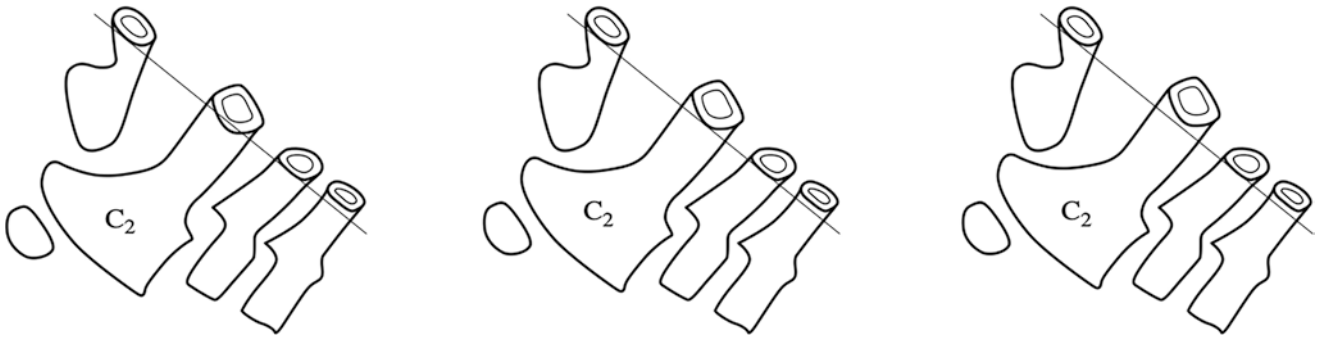


Fig. 6.19 Posterior Cervical Line. In physiological C2/C3 pseudosubluxation the posterior cervical line may (from left to right) pass through or touch or lie 1 mm in front of the cortex of the posterior arch of C2 (reprinted with permission from [162])

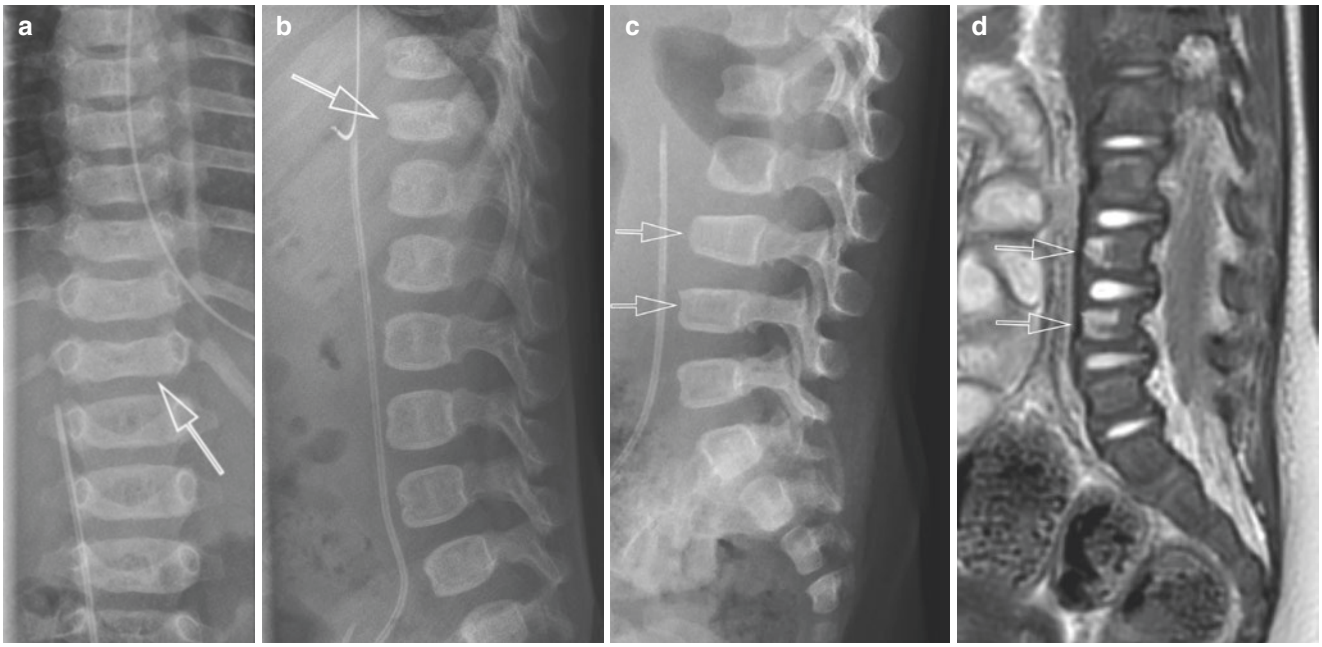


Fig. 6.20 Similarity between congenital decreased height and impression fractures of vertebral bodies. (**a**, **b**) A 4-month-old girl died from severe brain injury after a fall from the stairs at the hands of her father. Decreased height of the body of Th12 (arrows) was considered congenital because there were no abnormalities on post-mortem CT and MRI as well during autopsy. She also had segmentation anomalies on other levels. (**c**, **d**) Five-month-old boy who died under suspicious circumstances. Height loss of L3 and L4 (arrows in **c**) was considered post-traumatic because of oedema on T2-weighted MR images (arrows in **d**)

equivalent to the metaphyseal lucent bands seen normally after about a week of the child's age at the ends of long tubular bones. A similar lucency is seen just under the growth plate of vertebral bodies at that early age, giving the bone-in-bone appearance. Their index case was imaged at 3 weeks and at 5 weeks of age, with no evidence of periosteal reaction or endosteal callus in that follow-up study, confirming the lack of fractures.

Besides normal variants which could be mistaken for fractures, there are diseases that can lead to the occurrence of spinal fractures in children. Mostly these are oncological, such as Langerhans cell histiocytosis (Fig. 6.23a–c) or leukaemia, or endocrine disorders, such as hyperthyroidism or diabetes mellitus, where the structure of the vertebral body is compromised. These broader differential diagnoses are discussed in Chap. 14.

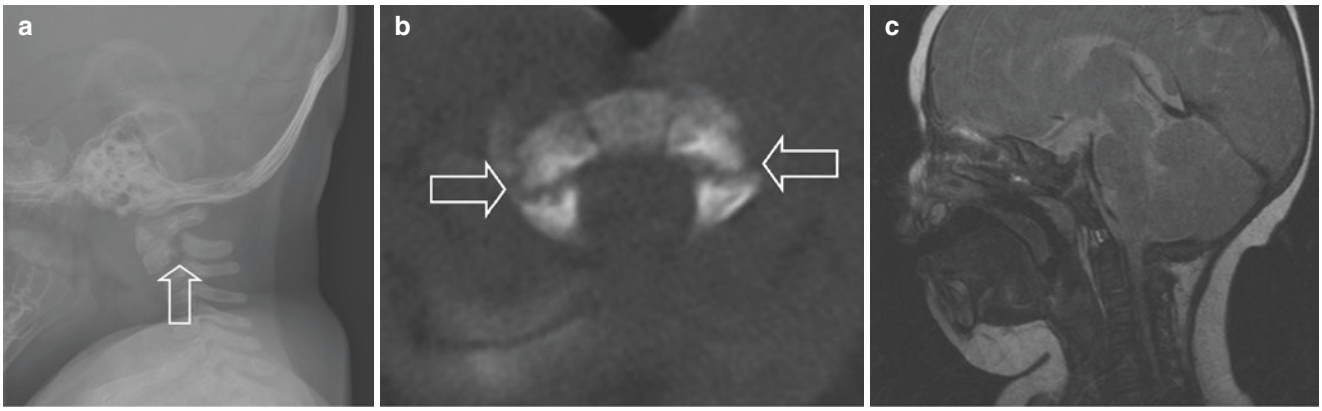


Fig. 6.21 (**a**) Radiological examination for suspected child abuse. The cervical spine view shows anterolisthesis (>3 mm) and a defect in the arch of C2 (open arrow) (Reprinted from [76]. With permission). (**b**) CT of the cervical spine shows a sclerotic margin of the arch defects (open arrow). (**c**) MRI of the cervical spine shows no signs of haematoma or bone oedema (sub-optimal quality due to movement artefacts) (reprinted with permission from [112])

Fig. 6.22 Coronal clefts of L3 and L4 in a 1-day-old child with multiple congenital abnormalities

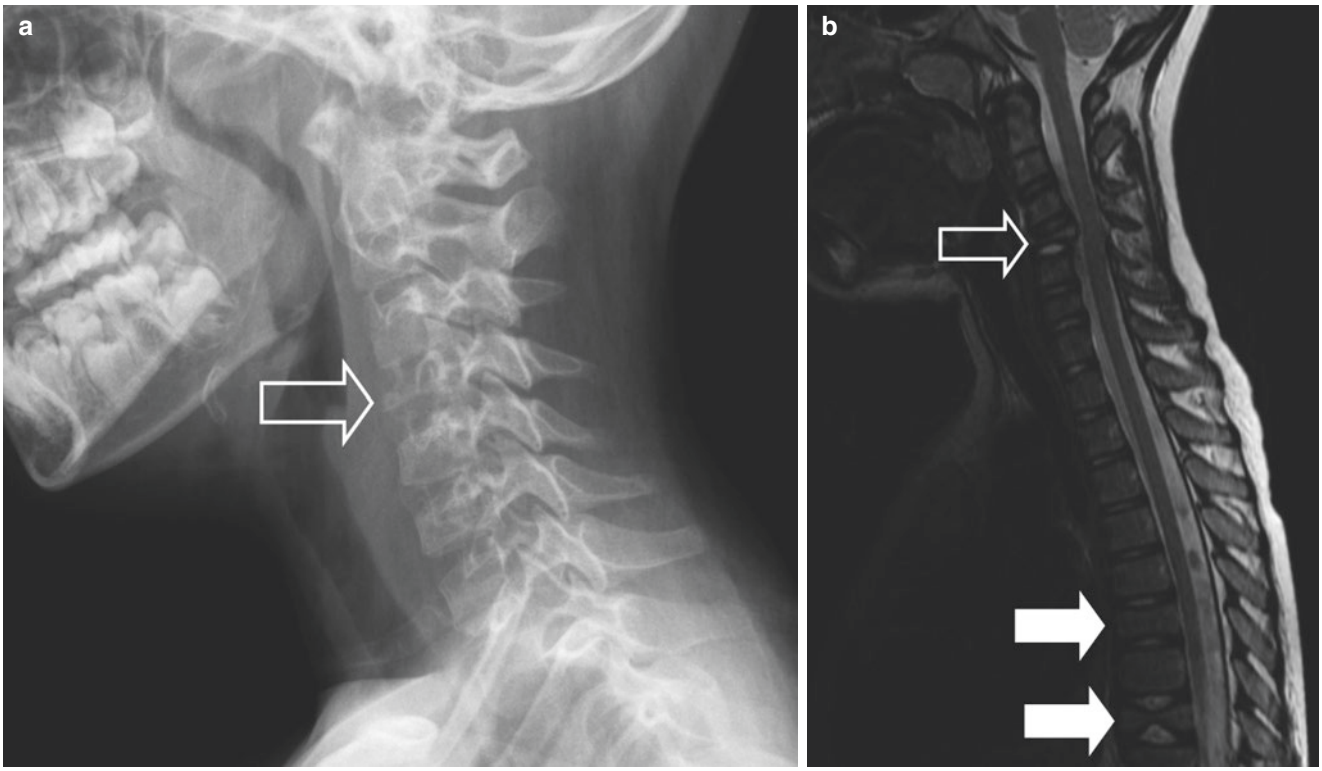


Fig. 6.23 (**a**) Girl with Langerhans cell histiocytosis. The conventional radiograph of the cervical spine shows a collapsed vertebral body at the level C4 and C6 (open arrow). (**b**) T2-weighted sagittal MRI view of the earlier found (CT and conventional radiograph) cervical collapse at the C4 level (open arrow) and C6. Collapsed vertebrae are seen at the thoracic level (arrows). All fractures are consistent with Langerhans cell histiocytosis

References

1. Cullen JC (1975) Spinal lesions in battered babies. J Bone Joint Surg Br 57:364–366
2. Diamond P, Hansen CM, Christofersen MR (1994) Child abuse presenting as a thoracolumbar spinal fracture dislocation: a case report. Pediatr Emerg Care 10:83–86
3. Dickson RA, Leatherman KD (1978) Spinal injuries in child abuse: case report. J Trauma 18:811–812
4. Parent S, Dimar J, Dekutoski M, Roy-Beaudry M (2010) Unique features of pediatric spinal cord injury. Spine (Phila Pa 1976) 35:S202–S208
5. Cirak B, Ziegfeld S, Knight VM, Chang D, Avellino AM, Paidas CN (2004) Spinal injuries in children. J Pediatr Surg 39:607–612
6. Kim C, Vassilyadi M, Forbes JK, Moroz NW, Camacho A, Moroz PJ (2016) Traumatic spinal injuries in children at a single level 1 pediatric trauma Centre: report of a 23-year experience. Can J Surg 59:205–212
7. Saul D, Dresing K (2018) Epidemiology of vertebral fractures in pediatric and adolescent patients. Pediatr Rep 10:7232
8. Piatt JH Jr (2015) Pediatric spinal injury in the US: epidemiology and disparities. J Neurosurg Pediatr 16:463–471
9. Reilly CW (2007) Pediatric spine trauma. J Bone Joint Surg Am 89(Suppl 1):98–107
10. Babu RA, Arivazhagan A, Devi BI, Bhat DI, Sampath S, Chandramouli BA (2016) Peculiarities and patterns of cervical spine injuries in children and adolescents: a retrospective series of 84 patients from a single institute. Pediatr Neurosurg 51:1–8
11. Brown RL, Brunn MA, Garcia VF (2001) Cervical spine injuries in children: a review of 103 patients treated consecutively at a level 1 pediatric trauma center. J Pediatr Surg 36:1107–1114
12. Patel JC, Tepas JJ 3rd, Mollitt DL, Pieper P (2001) Pediatric cervical spine injuries: defining the disease. J Pediatr Surg 36:373–376
13. Knox JB, Schneider JE, Cage JM, Wimberly RL, Riccio AI (2014) Spine trauma in very young children: a retrospective study of 206 patients presenting to a level 1 pediatric trauma center. J Pediatr Orthop 34:698–702
14. Özkan N, Wrede K, Ardeshiri A, Sariaslan Z, Stein KP, Dammann P, Müller O, Ringelstein A, Sure U, Sandalcioglu IE (2015) Management of traumatic spinal injuries in children and young adults. Childs Nerv Syst 31:1139–1148
15. Gopinathan NR, Viswanathan VK, Crawford AH (2018) Cervical spine evaluation in pediatric trauma: a review and an update of current concepts. Indian J Orthop 52:489–500
16. Mahan ST, Mooney DP, Karlin LI, Hresko MT (2009) Multiple level injuries in pediatric spinal trauma. J Trauma 67:537–542
17. Goergen S, Ditchfield M, Babl F, Oakley E, Rahman T, Johnson S (2015) Paediatric Cervical Spine Trauma. Royal Australian and New Zealand College of Radiologists, Education Modules for Appropriate Imaging Referrals
18. Pang D, Wilberger JE Jr (1982) Spinal cord injury without radiographic abnormalities in children. J Neurosurg 57:114–129
19. Pang D, Pollack IF (1989) Spinal cord injury without radiographic abnormality in children--the SCIWORA syndrome. J Trauma 29:654–664

20. Akbarnia BA, Campbell RM (1990) The role of the orthopedic surgeon in child abuse. In: Morrissy RT, Winter RB (eds) Lovell and Winter's pediatric Orthopaedics. Lippincott, Williams and Wilkins
21. Boese CK, Oppermann J, Siewe J, Eysel P, Scheyerer MJ, Lechler P (2015) Spinal cord injury without radiologic abnormality in children: a systematic review and meta-analysis. J Trauma Acute Care Surg 78:874–882
22. Pang D (2004) Spinal cord injury without radiographic abnormality in children, 2 decades later. Neurosurgery 55:1325–1342. discussion 1342–1323
23. Yucesoy K, Yuksel KZ (2008) SCIWORA in MRI era. Clin Neurol Neurosurg 110:429–433
24. The Royal College of Radiologists (RCR) and the Society and College of Radiographers (SCoR) (2017) The radiological investigation of suspected physical abuse in children. https://www.rcr.ac.uk/publication/radiological-investigation-suspected-physical-abuse-children. Accessed 05 July 2021
25. Kavalci C, Akdur G, Sayhan MB, Sogut O, Gökdemir MT (2011) Isolated transverse sacrum fracture: a case report. Emerg Med Int 2011:741570
26. Schwartz GR, Wright SW, Fein JA, Sugarman J, Pasternack J, Salhanick S (1997) Pediatric cervical spine injury sustained in falls from low heights. Ann Emerg Med 30:249–252
27. Ben Amor IM, Roughley P, Glorieux FH, Rauch F (2013) Skeletal clinical characteristics of osteogenesis imperfecta caused by haploinsufficiency mutations in COL1A1. J Bone Miner Res 28:2001–2007
28. Folkestad L, Hald JD, Ersbøll AK, Gram J, Hermann AP, Langdahl B, Abrahamsen B, Brixen K (2017) Fracture rates and fracture sites in patients with osteogenesis imperfecta: a Nationwide register-based cohort study. J Bone Miner Res 32:125–134
29. Sepúlveda AM, Terrazas CV, Sáez J, Reyes ML (2017) Fracturas vertebrales en niños con osteogénesis imperfecta tipo I [vertebral fractures in children with type I osteogenesis imperfecta]. Rev Chil Pediatr 88:348–353
30. Highsmith JM (2020) Types of spinal fractures. https://www.spineuniverse.com/conditions/spinal-fractures/types-spinal-fractures. Accessed 10 Aug 2021
31. West C, Roosendaal S, Bot J, Smithuis F (2015) Spine injury - Thoraco-lumbar injury classification and severity score (TLICS). https://radiologyassistant.nl/musculoskeletal/spine/tlics-classification-1. Accessed 10 Aug 2021
32. Horal J, Nachemson A, Scheller S (1972) Clinical and radiological long term follow-up of ventebral fractures in children. Acta Orthop Scand 43:491–503
33. Hubbard DD (1974) Injuries of the spine in children and adolescents. Clin Orthop Relat Res 100:56–65
34. Aufdermaur M (1974) Spinal injuries in juveniles. Necropsy findings in twelve cases. J Bone Joint Surg Br 56b:513–519
35. Offiah AC, Hall CM (2009) Flat bones and spine. In: Offiah AC, Hall CM (eds) Radiological atlas of child abuse. Radcliff Publishing, pp 77–79
36. Kleinman PK, Silvera VM (2015) Abusive craniocervical junction and spinal trauma. In: Kleinman PK (ed) Diagnostic imaging of child abuse. Cambridge University Press, pp 494–559
37. Royal College of Paediatrics and Child Health (RCPCH) (2019) Child protection evidence: systematic review on head and spinal injuries
38. Khurana B, Sheehan SE, Sodickson A, Bono CM, Harris MB (2013) Traumatic thoracolumbar spine injuries: what the spine surgeon wants to know. Radiographics 33:2031–2046
39. Vaccaro AR, Lehman RA Jr, Hurlbert RJ, Anderson PA, Harris M, Hedlund R, Harrop J, Dvorak M, Wood K, Fehlings MG, Fisher C, Zeiller SC, Anderson DG, Bono CM, Stock GH, Brown AK, Kuklo T, Oner FC (2005) A new classification of thoracolumbar injuries: the importance of injury morphology, the integrity of the posterior ligamentous complex, and neurologic status. Spine (Phila Pa 1976) 30:2325–2333
40. Savage JW, Moore TA, Arnold PM, Thakur N, Hsu WK, Patel AA, McCarthy K, Schroeder GD, Vaccaro AR, Dimar JR, Anderson PA (2015) The reliability and validity of the thoracolumbar injury classification system in pediatric spine trauma. Spine (Phila Pa 1976) 40:E1014–E1018
41. Sellin JN, Steele WJ 3rd, Simpson L, Huff WX, Lane BC, Chern JJ, Fulkerson DH, Sayama CM, Jea A (2016) Multicenter retrospective evaluation of the validity of the thoracolumbar injury classification and severity score system in children. J Neurosurg Pediatr 18:164–170
42. Gabos PG, Tuten HR, Leet A, Stanton RP (1998) Fracture-dislocation of the lumbar spine in an abused child. Pediatrics 101:473–477
43. Kewalramani LS, Tori JA (1980) Spinal cord trauma in children. Neurologic patterns, radiologic features, and pathomechanics of injury. Spine (Phila Pa 1976) 5:11–18
44. Paulson JA (1988) The epidemiology of injuries in adolescents. Pediatr Ann 17(84–86):89–96
45. Sneed RC, Stover SL, Fine PR (1986) Spinal cord injury associated with all-terrain vehicle accidents. Pediatrics 77:271–274
46. Carroll T, Smith CD, Liu X, Bonaventura B, Mann N, Liu J, Ebraheim NA (2015) Spinal cord injuries without radiologic abnormality in children: a systematic review. Spinal Cord 53:842–848
47. Gosnold JK, Sivaloganathan S (1980) Spinal cord damage in a case of non-accidental injury in children. Med Sci Law 20:54–57
48. Kleinman PK, Zito JL (1984) Avulsion of the spinous processes caused by infant abuse. Radiology 151:389–391
49. Piatt JH Jr, Steinberg M (1995) Isolated spinal cord injury as a presentation of child abuse. Pediatrics 96:780–782
50. Swischuk LE (1969) Spine and spinal cord trauma in the battered child syndrome. Radiology 92:733–738
51. Ghatan S, Ellenbogen RG (2002) Pediatric spine and spinal cord injury after inflicted trauma. Neurosurg Clin N Am 13:227–233
52. Vialle R, Mary P, Schmider L, le Pointe HD, Damsin JP, Filipe G (2006) Spinal fracture through the neurocentral synchondrosis in battered children: a report of three cases. Spine (Phila Pa 1976) 31:E345–E349
53. Brodeur AE, Monteleone JA (1994) Child maltreatment. A clinical guide and reference. GW Medical, p 32
54. Kleinman PK (2015) Diagnostic imaging of child abuse. Cambridge, Cambridge
55. Akbarnia B, Torg JS, Kirkpatrick J, Sussman S (1974) Manifestations of the battered-child syndrome. J Bone Joint Surg Am 56:1159–1166
56. Galleno H, Oppenheim WL (1982) The battered child syndrome revisited, vol 162. Clin Orthop Relat Res, pp 11–19
57. Bode KS, Newton PO (2007) Pediatric nonaccidental trauma thoracolumbar fracture-dislocation: posterior spinal fusion with pedicle screw fixation in an 8-month-old boy. Spine (Phila Pa 1976) 32:E388–E393
58. Kleinman PK, Morris NB, Makris J, Moles RL, Kleinman PL (2013) Yield of radiographic skeletal surveys for detection of hand, foot, and spine fractures in suspected child abuse. AJR Am J Roentgenol 200:641–644
59. Kemp AM, Joshi AH, Mann M, Tempest V, Liu A, Holden S, Maguire S (2010) What are the clinical and radiological characteristics of spinal injuries from physical abuse: a systematic review. Arch Dis Child 95:355–360
60. Jauregui JJ, Perfetti DC, Cautela FS, Frumberg DB, Naziri Q, Paulino CB (2019) Spine injuries in child abuse. J Pediatr Orthop 39:85–89

61. King J, Diefendorf D, Apthorp J, Negrete VF, Carlson M (1988) Analysis of 429 fractures in 189 battered children. J Pediatr Orthop 8:585–589
62. Kogutt MS, Swischuk LE, Fagan CJ (1974) Patterns of injury and significance of uncommon fractures in the battered child syndrome. Am J Roentgenol Radium Therapy, Nucl Med 121:143–149
63. Soto-Ares G, Denes M, Noulé N, Vinchon M, Pruvo JP, Gosset D (2003) Subdural hematomas in children: role of cerebral and spinal MRI in the diagnosis of child abuse [collections sous durales de l'enfant: place de l'IRM cerebrale et medullaire dans le diagnostic de maltraitance]. J Radiol 84:1757–1765
64. Edelbauer M, Maurer K, Gassner I (2012) Spinal subdural effusion - an additional sonographic sign of child abuse. Ultraschall Med 33:E339–e343
65. Choudhary AK, Bradford RK, Dias MS, Moore GJ, Boal DK (2012) Spinal subdural hemorrhage in abusive head trauma: a retrospective study. Radiology 262:216–223
66. Rangwala SD, Birk DM, Tobin MK, Hahn YS, Nikas DC (2017) Spontaneous resolution of spinal epidural hematoma resulting from domestic child abuse: case report. Pediatr Neurosurg 52:51–54
67. Rabbitt AL, Kelly TG, Yan K, Zhang J, Bretl DA, Quijano CV (2020) Characteristics associated with spine injury on magnetic resonance imaging in children evaluated for abusive head trauma. Pediatr Radiol 50:83–97
68. Callanan DL, McClain WD (2018) When a child dies unexpectedly: no truth be told. Am J Forensic Med Pathol 39:276–278
69. 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 62:107–118
70. Kleinman PK, Marks SC (1992) Vertebral body fractures in child abuse. Radiologic-histopathologic correlates. Invest Radiol 27:715–722
71. Dudley MH, Garg M (2014) Fatal child abuse presenting with multiple vertebral and vascular trauma. J Forensic Sci 59:386–389
72. Leonard JR, Jaffe DM, Kuppermann N, Olsen CS, Leonard JC (2014) Cervical spine injury patterns in children. Pediatrics 133:e1179–e1188
73. Brink FW, Gold DL, Adler B, Letson MM (2017) Distraction injury of the thoracic spine with spinal cord transection and vascular injury in a 5-week-old infant boy: a case of child physical abuse. Pediatr Emerg Care 33:192–197
74. Saleh S, Swanson KI, Bragg T (2018) Successful surgical repair and recovery in a 2-week-old infant after birth-related cervical fracture dislocation. J Neurosurg Pediatr 21:16–20
75. Booth TN (2012) Cervical spine evaluation in pediatric trauma. AJR Am J Roentgenol 198:W417–W425
76. Smart PJ, Hardy PJ, Buckley DM, Somers JM, Broderick NJ, Halliday KE, Williams L (2003) Cervical spine injuries to children under 11: should we use radiography more selectively in their initial assessment? Emerg Med J 20:225–227
77. Thomas NH, Robinson L, Evans A, Bullock P (1995) The floppy infant: a new manifestation of nonaccidental injury. Pediatr Neurosurg 23:188–191
78. Easter JS, Barkin R, Rosen CL, Ban K (2011) Cervical spine injuries in children, part I: mechanism of injury, clinical presentation, and imaging. J Emerg Med 41:142–150
79. Slaar A, Fockens MM, Wang J, Maas M, Wilson DJ, Goslings JC, Schep NW, van Rijn RR (2017) Triage tools for detecting cervical spine injury in pediatric trauma patients. Cochrane Database Syst Rev 12:Cd011686
80. Hernandez-Marti M, Dal Canto MC, Kidd JM (1984) Evidence of spinal cord injury in an infant delivered by cesarean section. A case report. Childs Brain 11:197–201
81. Kobayashi S, Kanda K, Yokochi K, Ohki S (2006) A case of spinal cord injury that occurred in utero. Pediatr Neurol 35:367–369
82. Scripcaru G, Ianovici N, Anghel M (1977) Fetal cervical spine injuries. Rev Med Chir Soc Med Nat Iasi 81:47–54
83. Weinberg L, Wyatt JP, Busuttil A (2001) Traumatic intrauterine fetal spinal fracture following seat belt use: a case report. J Trauma 51:1195–1196
84. Caird MS, Reddy S, Ganley TJ, Drummond DS (2005) Cervical spine fracture-dislocation birth injury: prevention, recognition, and implications for the orthopaedic surgeon. J Pediatr Orthop 25:484–486
85. Blount J, Doughty K, Tubbs RS, Wellons JC, Reddy A, Law C, Karle V, Oakes WJ (2004) In utero spontaneous cervical thoracic epidural hematoma imitating spinal cord birth injury. Pediatr Neurosurg 40:23–27
86. Roche-Herrero MC, Miranda-Cid C, Royo-Orejas A, Escudero-Lou RM (2004) Atrofia de la medula Espinal cervical tras infarto isquemico de origen prenatal [atrophy of the cervical spinal cord as a consequence of a prenatal ischemic infarct]. Rev Neurol 39:932–934
87. Shulman ST, Madden JD, Esterly JR, Shanklin DR (1971) Transection of spinal cord. A rare obstetrical complication of cephalic delivery. Arch Dis Child 46:291–294
88. MacKinnon JA, Perlman M, Kirpalani H, Rehan V, Sauve R, Kovacs L (1993) Spinal cord injury at birth: diagnostic and prognostic data in twenty-two patients. J Pediatr 122:431–437
89. Vialle R, Piétin-Vialle C, Ilharreborde B, Dauger S, Vinchon M, Glorion C (2007) Spinal cord injuries at birth: a multicenter review of nine cases. J Matern Fetal Neonatal Med 20:435–440
90. Menticoglou SM, Perlman M, Manning FA (1995) High cervical spinal cord injury in neonates delivered with forceps: report of 15 cases. Obstet Gynecol 86:589–594
91. Morgan C, Newell SJ (2001) Cervical spinal cord injury following cephalic presentation and delivery by caesarean section. Dev Med Child Neurol 43:274–276
92. Huisman T, Phelps T, Bosemani T, Tekes A, Poretti A (2015) Parturitional injury of the head and neck. J Neuroimaging 25:151–166
93. Stanley P, Duncan AW, Isaacson J, Isaacson AS (1985) Radiology of fracture-dislocation of the cervical spine during delivery. AJR Am J Roentgenol 145:621–625
94. Sheil AT, Collins KA (2007) Fatal birth trauma due to an undiagnosed abdominal teratoma: case report and review of the literature. Am J Forensic Med Pathol 28:121–127
95. Salek M, Price J, Vaidya R, Richardson M (2018) Hemophilia a presenting with cephalohematoma in a newborn. Pediatrics 142:225
96. Conry BG, Hall CM (1987) Cervical spine fractures and rear car seat restraints. Arch Dis Child 62:1267–1268
97. McGrory BJ, Klassen RA, Chao EY, Staeheli JW, Weaver AL (1993) Acute fractures and dislocations of the cervical spine in children and adolescents. J Bone Joint Surg Am 75:988–995
98. Leonard JC, Jaffe DM, Olsen CS, Kuppermann N (2015) Age-related differences in factors associated with cervical spine injuries in children. Acad Emerg Med 22:441–446
99. LeFever D, Whipple SG, Menger RP (2020) Hangman's fractures. https://www.ncbi.nlm.nih.gov/books/NBK519496/. Accessed 10 Aug 2021
100. Montalbano M, Fisahn C, Loukas M, Oskouian RJ, Chapman JR, Tubbs RS (2017) Pediatric Hangman's fracture: a comprehensive review. Pediatr Neurosurg 52:145–150
101. Wood-Jones F (1913) The ideal lesion produced by judicial hangings. Lancet 1:53–54
102. Schneider RC, Livingston KE, Cave AJ, Hamilton G (1965) "Hangman's fracture" of the cervical spine. J Neurosurg 22:141–154
103. Sumchai AP, Sternbach GL (1991) Hangman's fracture in a 7-week-old infant. Ann Emerg Med 20:86–89

104. Pizzutillo PD, Rocha EF, D'Astous J, Kling TF Jr, McCarthy RE (1986) Bilateral fracture of the pedicle of the second cervical vertebra in the young child. J Bone Joint Surg Am 68:892–896
105. Ruff SJ, Taylor TK (1986) Hangman's fracture in an infant. J Bone Joint Surg Br 68:702–703
106. Weiss MH, Kaufman B (1973) Hangman's fracture in an infant. Am J Dis Child 126:268–269
107. Parisi M, Lieberson R, Shatsky S (1991) Hangman's fracture or primary spondylolysis: a patient and a brief review. Pediatr Radiol 21:367–368
108. Finnegan MA, McDonald H (1982) Hangman's fracture in an infant. Can Med Assoc J 127:1001–1002
109. Currarino G (1989) Primary spondylolysis of the axis vertebra (C2) in three children, including one with pyknodysostosis. Pediatr Radiol 19:535–538
110. Mondschein J, Karasick D (1999) Spondylolysis of the axis vertebra: a rare anomaly simulating hangman's fracture. AJR Am J Roentgenol 172:556–557
111. Williams JP 3rd, Baker DH, Miller WA (1999) CT appearance of congenital defect resembling the Hangman's fracture. Pediatr Radiol 29:549–550
112. van Rijn RR, Kool DR, de Witt Hamer PC, Majoie CB (2005) An abused five-month-old girl: Hangman's fracture or congenital arch defect? J Emerg Med 29:61–65
113. 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
114. Feldman KW, Avellino AM, Sugar NF, Ellenbogen RG (2008) Cervical spinal cord injury in abused children. Pediatr Emerg Care 24:222–227
115. Holland CM, Kebriaei MA, Wrubel DM (2016) Posterior cervical spinal fusion in a 3-week-old infant with a severe subaxial distraction injury. J Neurosurg Pediatr 17:353–356
116. Kleinman PK (2004) Hangman's fracture caused by suspected child abuse. J Pediatr Orthop B 13:348; author reply 348
117. Rooks VJ, Sisler C, Burton B (1998) Cervical spine injury in child abuse: report of two cases. Pediatr Radiol 28:193–195
118. Harmon J, Rabe AJ, Nichol KK, Shiels WE (2012) Precervical myositis ossificans in an infant secondary to child abuse. Pediatr Radiol 42:881–885
119. Oral R, Rahhal R, Elshershari H, Menezes AH (2006) Intentional avulsion fracture of the second cervical vertebra in a hypotonic child. Pediatr Emerg Care 22:352–354
120. Gille P, Bonneville JF, François JY, Aubert D, Peltre G, Canal JP (1980) Fracture des pédicules de l'axis chez un nourrisson battu [fractures of axis pedicles in battered infant (author's transl)]. Chir Pediatr 21:343–344
121. Kleinman PK, Shelton YA (1997) Hangman's fracture in an abused infant: imaging features. Pediatr Radiol 27:776–777
122. Ranjith RK, Mullett JH, Burke TE (2002) Hangman's fracture caused by suspected child abuse. A case report. J Pediatr Orthop B 11:329–332
123. Curphey TJ, Noguchi TT, Kade H, Moore SM (1965) The battered child syndrome, responsibilities of the pathologist. Calif Med 102:102–104
124. McGrory BE, Fenichel GM (1977) Hangman's fracture subsequent to shaking in an infant. Ann Neurol 2:82
125. Governale LS, Brink FW, Pluto CP, Schunemann VA, Weber R, Rusin J, Fischer BA, Letson MM (2018) A retrospective study of cervical spine MRI findings in children with abusive head trauma. Pediatr Neurosurg 53:36–42
126. Katz JS, Oluigbo CO, Wilkinson CC, McNatt S, Handler MH (2010) Prevalence of cervical spine injury in infants with head trauma. J Neurosurg Pediatr 5:470–473
127. Baerg J, Thirumoorthi A, Vannix R, Taha A, Young A, Zouros A (2017) Cervical spine imaging for young children with inflicted trauma: expanding the injury pattern. J Pediatr Surg 52:816–821
128. Koumellis P, McConachie NS, Jaspan T (2009) Spinal subdural haematomas in children with non-accidental head injury. Arch Dis Child 94:216–219
129. Daniels AH, Sobel AD, Eberson CP (2013) Pediatric thoracolumbar spine trauma. J Am Acad Orthop Surg 21:707–716
130. Journeau P, Bourcheix LM, Wagner A, Padovani JP, Pouliquen JC (2001) Obstetric dislocation of the thoracic spine: case report and review of the literature. J Pediatr Orthop B 10:78–80
131. Ilagan NB, Liang KC, Piligian J, Poland R (1987) Thoracic spinal cord (T3-T4) transection in a breech-presenting, cesarean-section-delivered preterm infant. Am J Perinatol 4:233–234
132. Fotter R, Sorantin E, Schneider U, Ranner G, Fast C, Schober P (1994) Ultrasound diagnosis of birth-related spinal cord trauma: neonatal diagnosis and follow-up and correlation with MRI. Pediatr Radiol 24:241–244
133. Le Masne A, Rousseau S, Lequien P (1995) Traumatismes médullaires périnatalsPerinatal spinal cord injuries [Perinatal spinal cord injuries]. Arch Pediatr 2:1177–1181
134. Billingham C, Richardson R, Lilien L (2019) Breech delivery, rupture of Batson's plexus, T8 epidural hematoma, and paraplegia. J Neonatal Perinatal Med 12:325–331
135. Campbell J, Bonnett C (1975) Spinal cord injury in children. Clin Orthop Relat Res 112:114–123
136. Hadley MN, Zabramski JM, Browner CM, Rekate H, Sonntag VK (1988) Pediatric spinal trauma. Review of 122 cases of spinal cord and vertebral column injuries. J Neurosurg 68:18–24
137. Hegenbarth R, Ebel KD (1976) Roentgen findings in fractures of the vertebral column in childhood examination of 35 patients and its results. Pediatr Radiol 5:34–39
138. Kewalramani LS, Kraus JF, Sterling HM (1980) Acute spinal-cord lesions in a pediatric population: epidemiological and clinical features. Paraplegia 18:206–219
139. Shetty AP, Aiyer SN (2017) Pediatric thoracolumbar spinal injuries: a rare and unique clinical scenario. Neurol India 65:482–484
140. Babu RA, Arimappamagan A, Pruthi N, Bhat DI, Arvinda HR, Devi BI, Somanna S (2017) Pediatric thoracolumbar spinal injuries: the etiology and clinical spectrum of an uncommon entity in childhood. Neurol India 65:546–550
141. Kraus R, Stahl JP, Heiss C, Horas U, Dongowski N, Schnettler R (2013) Frakturen der Brust- und Lendenwirbelsäule im Wachstumsalter [fractures of the thoracic and lumbar spine in children and adolescents]. Unfallchirurg 116:435–441
142. Franklin DB 3rd, Hardaway AT, Sheffer BW, Spence DD, Kelly DM, Muhlbauer MS, Warner WC Jr, Sawyer JR (2019) The role of computed tomography and magnetic resonance imaging in the diagnosis of pediatric thoracolumbar compression fractures. J Pediatr Orthop 39:e520–e523
143. Merten DF, Carpenter BL (1990) Radiologic imaging of inflicted injury in the child abuse syndrome. Pediatr Clin N Am 37:815–837
144. Renard M, Tridon P, Kuhnast M, Renauld JM, Dollfus P (1978) Three unusual cases of spinal cord injury in childhood. Paraplegia 16:130–134
145. Tran B, Silvera M, Newton A, Kleinman PK (2007) Inflicted T12 fracture-dislocation: CT/MRI correlation and mechanistic implications. Pediatr Radiol 37:1171–1173
146. Sieradzki JP, Sarwark JF (2008) Thoracolumbar fracture-dislocation in child abuse: case report, closed reduction technique and review of the literature. Pediatr Neurosurg 44:253–257
147. Lieberman I, Chiasson D, Podichetty VK (2010) Aortic disruption associated with L2-L3 fracture-dislocation in a case of child abuse: a case report. J Bone Joint Surg Am 92:1670–1674
148. Webb M, Sherman SS, Sung L, Schmidt CJ, Hlavaty L (2020) Abusive pediatric thoracolumbar fracture due to forced hyperex-

tension: case report, biomechanical considerations, and review of the literature. J Forensic Sci 65:2023
149. Levin TL, Berdon WE, Cassell I, Blitman NM (2003) Thoracolumbar fracture with listhesis--an uncommon manifestation of child abuse. Pediatr Radiol 33:305–310
150. Carrion WV, Dormans JP, Drummond DS, Christofersen MR (1996) Circumferential growth plate fracture of the thoracolumbar spine from child abuse. J Pediatr Orthop 16:210–214
151. Gruber TJ, Rozzelle CJ (2008) Thoracolumbar spine subdural hematoma as a result of nonaccidental trauma in a 4-month-old infant. J Neurosurg Pediatr 2:139–142
152. Hong CS, Camara-Quintana J, Kundishora AJ, Diluna ML, Kahle KT (2019) Teaching NeuroImages: spinal subdural hematoma in pediatric nonaccidental trauma. Neurology 93:e522–e523
153. Hart DJ, Wang MY, Griffith P, Gordon McComb J (2004) Pediatric sacral fractures. Spine (Phila Pa 1976) 29:667–670
154. Hamoud K, Abbas J (2015) Fracture dislocation of the sacrococcygeal joint in a 12-year-old boy. A case report and literature review. Orthop Traumatol Surg Res 101:871–873
155. Isik M, Subasi M, Cebesoy O (2013) Uludag a (2013) isolated sacral dislocation in a 4-year-old child. BMJ Case Rep 2013:bcr2013200119
156. Hamoud K, Abbas J (2017) Fracture dislocations of the coccyx: a case series and literature review. J Clin Case Rep 7:10001005
157. Johnson K, Chapman S, Hall CM (2004) Skeletal injuries associated with sexual abuse. Pediatr Radiol 34:620–623
158. Barber I, Perez-Rossello JM, Wilson CR, Silvera MV, Kleinman PK (2013) Prevalence and relevance of pediatric spinal fractures in suspected child abuse. Pediatr Radiol 43:1507–1515
159. Adib O, Berthier E, Loisel D, Aubé C (2016) Pediatric cervical spine in emergency: radiographic features of normal anatomy, variants and pitfalls. Skelet Radiol 45:1607–1617
160. Condon L, Knight L (2005) Pseudosubluxation of the cervical spine. Br J Hosp Med (Lond) 66:586
161. Goñi Orayen C, Pérez Martínez A, Martín Martínez C, Asensio Llorente M, Peiro Ibáñez JL, de Diego SM (1992) Pseudosubluxación C2-C3 en la infancia: un error frecuente de diagnóstico clínico-radiológico [Pseudosubluxation of C2-C3 in childhood: a frequent clinico-radiological diagnostic error]. An Esp Pediatr 36:390–392
162. Shaw M, Burnett H, Wilson A, Chan O (1999) Pseudosubluxation of C2 on C3 in polytraumatized children--prevalence and significance. Clin Radiol 54:377–380
163. Verhestraeten B, Rüther W, Messler H (1988) Die Pseudosubluxation der oberen Halswirbelkörper--eine oft verkannte Normvariante im Kindesalter [Pseudodislocation of the upper cervical vertebrae--an often misdiagnosed normal variant in childhood]. Unfallchirurg 91:238–240
164. Vivas CR, Ramírez SM, Espinosa FJ, Sánchez AM, Peña CR (2010) Pseudosubluxación C2-C3 [Pseudosubluxation of C2-C3]. An Pediatr (Barc) 73:211–212
165. Aronica-Pollak PA, Stefan VH, McLemore J (2003) Coronal cleft vertebra initially suspected as an abusive fracture in an infant. J Forensic Sci 48:836–838
166. Hill SC, Dwyer AJ, Kaler SG (2012) Cervical spine anomalies in Menkes disease: a radiologic finding potentially confused with child abuse. Pediatr Radiol 42:1301–1304
167. Doberentz E, Madea B, Müller AM (2014) Coronal clefts in infants - rare differential diagnosis of traumatic injuries of vertebral bodies in battered children. Leg Med (Tokyo) 16:333–336
168. Tanaka T, Uhthoff HK (1983) Coronal cleft of vertebrae, a variant of normal enchondral ossification. Acta Orthop Scand 54:389–395
169. Doberentz E, Schumacher R, Gembruch U, Gasser JA, Müller AM (2013) Coronal vertebral clefts: a radiological indicator for chromosomal aberrations. Pediatr Dev Pathol 16:1–6
170. Westvik J, Lachman RS (1998) Coronal and sagittal clefts in skeletal dysplasias. Pediatr Radiol 28:764–770
171. Oestreich AE, Anton CG (2015) Normal spinous process metaphyseal-equivalent lucency simulating infant abuse fractures. Skelet Radiol 44:1519–1522

Ribs

7

Heike C. Terlingen, Rob A. C. Bilo, Marjo J. Affourtit, Simon G. F. Robben, and Rick R. van Rijn

Contents

H. C. Terlingen
Department of Forensic Medicine, Netherlands Forensic Institute, The Hague, The Netherlands
e-mail: h.terlingen@nfi.nl

R. A. C. Bilo
Veilig Thuis Rotterdam Rijnmond (Center for the Reporting of Child Abuse, Domestic Violence and Elder Abuse), Rotterdam, The Netherlands

M. J. Affourtit
Department of Paediatrics, Sophia Children's Hospital, Erasmus MC+, Rotterdam, The Netherlands
e-mail: m.affourtit@erasmusmc.nl

S. G. F. Robben (✉)
Department of Radiology, Maastricht University Medical Center, Maastricht, The Netherlands
e-mail: s.robben@mumc.nl

R. R. van Rijn
Department of Radiology and Nuclear Medicine, Amsterdam UMC, University of Amsterdam, Amsterdam, The Netherlands

Department of Forensic Medicine, Netherlands Forensic Institute, The Hague, The Netherlands
e-mail: r.r.vanrijn@amsterdamumc.nl

7.1 General Aspects of Rib Fractures

The thorax consists of the rib cage and the intrathoracic organs. The intrathoracic organs are the lungs and lower part of trachea, the heart and large vessels, like the aorta, pulmonary arteries and the vena cava, the oesophagus, the thymus, the thoracic lymph nodes, and the nerves, like the vagal nerve. The rib cage is the bony framework of the thoracic cavity and consists of twelve thoracic vertebrae, twelve pairs of ribs, and the sternum. The first seven ribs articulate anteriorly with the sternum at the sternocostal joints with their costal cartilages: the costochondral junction. The eighth, ninth, and tenth ribs articulate with their costal cartilages not with the sternum, but merge with the cartilage of the seventh rib. The eleventh and twelfth ribs are called floating ribs because they do not articulate directly or indirectly with the sternum. Each rib articulates posteriorly with the thoracic vertebrae at the costovertebral and costotransverse joints (Fig. 7.1) [1]. The arch of the rib can be divided into four regions (posterior, posterolateral, anterolateral, and anterior) and rib frac-

R. A. C. Bilo et al. (eds.), *Forensic Aspects of Paediatric Fractures*, https://doi.org/10.1007/978-3-031-12041-1_7

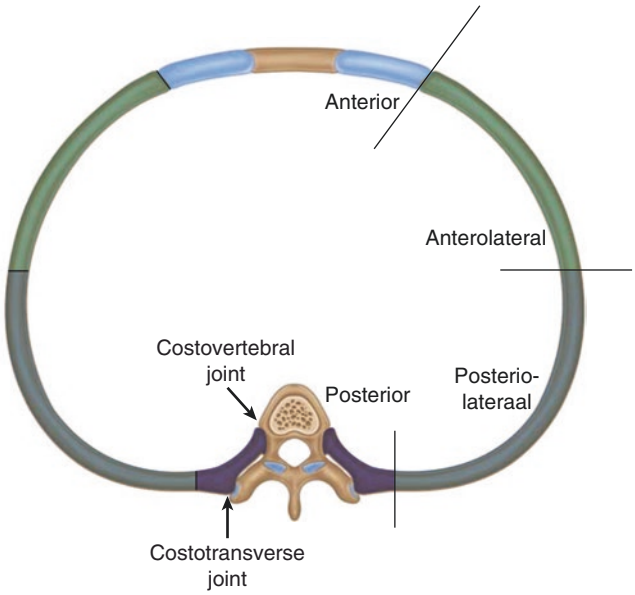


Fig. 7.1 Diagram of the four regions of the rib and the costovertebral joints

Table 7.1 Definition of fracture location and type [2]

Location of fracture	Definition
Posterior	Area from the lateral margin of the rib tubercle to the medial tip of the rib head
Posterolateral	Area from the lateral most point of the rib body to the lateral margin of the rib tubercle
Anterolateral and anterior	The anterior and anterolateral regions of the rib span from the most lateral point of the rib body to the sternal end. The interface of the anterior and anterolateral regions is the midpoint of this section of the rib
Type of fracture	*Definition*
Buckle	Incomplete fracture on the pleural surface of the rib
Transverse	Complete fracture with a vertical (superior-inferior) orientation
Oblique	Incomplete or complete fracture with a superolateral to inferomedial orientation or vice versa
Sternal end plate	Fracture of the sternal end plate or rim

tures can be classified into four types (sternal end, buckle, transverse, and oblique) (see Table 7.1 and Fig. 7.1) [2].

7.2 Cause of Rib Fractures

Around 85% of all chest injuries in paediatric patients, which are serious enough to warrant medical attention and/or treatment, are due to blunt force trauma, and around 15% are due to penetrating trauma [3, 4]. Blast injuries are very rare in paediatric patients.

Blunt force trauma can be due to 'low speed crush' blunt force trauma, in which the cage and intrathoracic organs are compressed, and in 'high speed impact' blunt force trauma, in which large amounts of kinetic energy, which are released during the impact, are transferred to the intrathoracic structures (pressure wave).

Not all children with thoracic trauma will have a combination of intrathoracic injuries, extrathoracic injuries, and rib fractures. Most children will have one injury, e.g. only rib fractures or intrathoracic injuries, or two injuries, e.g. a combination of rib fractures and bruising.

Rib fractures in a paediatric patient are usually due either to static loading (compression) or to dynamic impact loading (direct impact trauma to the ribs). In young children, rib fractures are usually caused by static loading (Sect. 7.2.1), while in mobile older children and in adolescents rib fractures are usually caused by dynamic impact loading (Sect. 7.2.2). In this section and in Sect. 7.3 (manner of rib fractures) the effects of static and dynamic impact loading will be dealt with. However, concerning the information in these sections it should be noted that still there is no clear understanding of what forces and mechanisms of injury are involved in the production of rib fractures, as was already stated in 2007 by Worn and Jones [5]. This is mainly due to the fact that in vivo experiments in children are impossible. The information in these sections reflects what is accepted as plausible in medical science.

Chest traumas, due to penetrating trauma, e.g. caused by bullets, knives or, other sharp objects, have been mainly reported in older children or in adolescents and only rarely reported in young paediatric patients (see Sect. 7.4) [6, 7]. Sometimes (more or less) blunt penetrating trauma may occur in paediatric patients, e.g. due to falls on railings or fence posts. In Sect. 7.4 cause and manner of penetrating trauma will be described.

7.2.1 Static Loading: Compression

In young children rib fractures are usually caused by static loading due to compression, resulting in deformation of the chest, e.g. when the chest is encircled by both hands and compressed [5]. Compression may result in deformation of the whole rib cage or of a part of the rib cage. The forces released during compression and deformation may have various effects on the rib cage and on the individual ribs. This may lead to fractures at different levels in or on different sides of the rib cage and to fractures in different locations in the rib arch (Table 7.2, Figs. 7.2 and 7.3). Rib fractures, due to compression, may be single or multiple. Often, they are found bilateral and multiple adjacent ribs are affected.

Fractures, due to compression, are most probably first sustained at the posterior side at the costovertebral junction,

Table 7.2 Overview of rib fractures, due to symmetrical and asymmetrical compression [5]

Compression	Mechanism	Fractures located at:
Symmetrical		
Anterior-posterior	Force exerted from the front (back on surface)—Sternum moved towards spine—Compression on the inside of the ribs and tension on the outside of the ribs	• Anterior, lateral and posterior costal arch.
		• Costochondral junction (possibly).
Anterior-posterior/ posterior-anterior	Force exerted from the front as well as the back—Sternum and spine moved towards each other—Leverage of rib on transverse process	• Transverse process (leverage).
		• Rib head (costovertebral joint).
Posterior-anterior	Force exerted from back (front on surface)—Spine moved towards sternum—Leverage of rib on transverse process	• Transverse process (leverage).
		• Rib head (costovertebral joint).
From the side	Force exerted from the side—Lateral costal arches moved towards each other	• Anterior, lateral, and posterior costal arch.
		• Costochondral junction (possibly).
	In the presence of leverage: (depends on the manner of holding and compressing)	• Transverse process (leverage).
		• Rib head (costovertebral joint).
Asymmetrical		
Combinations of anterior-posterior and sideways compression	Forces exerted to different degrees from the front, back and side—Oblique—Asymmetrical deformation of the ribcage in which the right or left anterior side is pushed in the direction of, respectively, the left or right posterior side	• Fractures are possible at all anatomical locations.

the location where the effect of mechanical forces is highest and where excessive leverage of the rib on the transverse process of the vertebra occurs. When compression increases, first lateral and then anterior fractures, will follow the posterior fractures [8].

In front-to-back compression the sternum is pushed inwards. This is also the case when the vertebrae are not pushed in the direction of the sternum. Hereby the costochondral junctions are pushed inwards, which may result in fractures at that location [9].

The finding of posterior or posterolateral fractures is particularly suggestive for compression as cause of a rib fracture. Kleinman and Schlesinger showed that considerable leverage from the posterior end of the rib against the vertebral transverse processes is necessary before a rib fracture is sustained at that location [10]. This effect occurs when the transverse processes are compressed in the direction of the sternum by moving the vertebra towards the sternum [11, 12]. This causes a fracture in the cortex of the posterior costal arch at the ventral side, possibly with complete cortical disruption (Fig. 7.4) [9, 10, 13, 14].

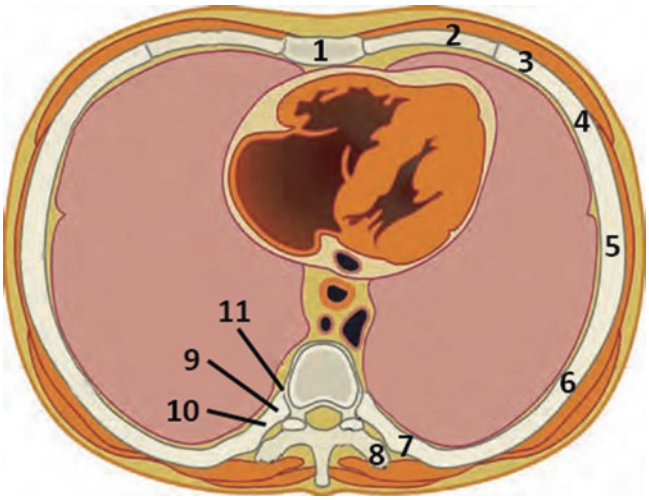


Fig. 7.2 Anatomy of the costal arch and possible locations for rib fractures in anterior-posterior and sideways compression. (1) Sternum. (2) Rib cartilage. (3) Sternal end of the rib. (4) Anterior costal arch. (5) Lateral costal arch. (6) Posterolateral costal arch. (7) Posterior costal arch (costal tubercle). (8) Tubercle of the transverse process of the vertebra. (9) Rib head. (10) Rib neck. (11) Costovertebral joint

In summary, compression may result in two kinds of fractures. First, fractures due to stress in the rib (costochondral, anterior, lateral, and posterior) and secondly due to leverage (around the vertebra at the costovertebral junction).

Compression does not necessarily leave externally visible bruising. It was found that compression fractures are rarely accompanied by bruising [15], although thumb and finger print bruises (thumb print at the anterior/upper part of the chest and fingerprints at the posterior side, often paravertebral) may be the only clinical signs for non-accidental compression fractures of the ribs (Fig. 7.5a, b) [16].

As far as is known from medical literature, rib fractures due to compression occur mainly in non-mobile children under the age of 2 years [17].

In the older literature, several other explanations for the occurrence of posterior rib fractures at the costovertebral junction can be found. Cameron and Rae assumed that posterior fractures could also be caused by lateral compression (side-to-side compression of the chest) during violent squeezing of the thorax [18]. Smith et al. assumed that posterior fractures also could be caused by dynamic impact loading: '*In our cases of costovertebral fractures, we suspect that the injury required a large amount of direct force, probably caused by the child being trodden upon or struck from*

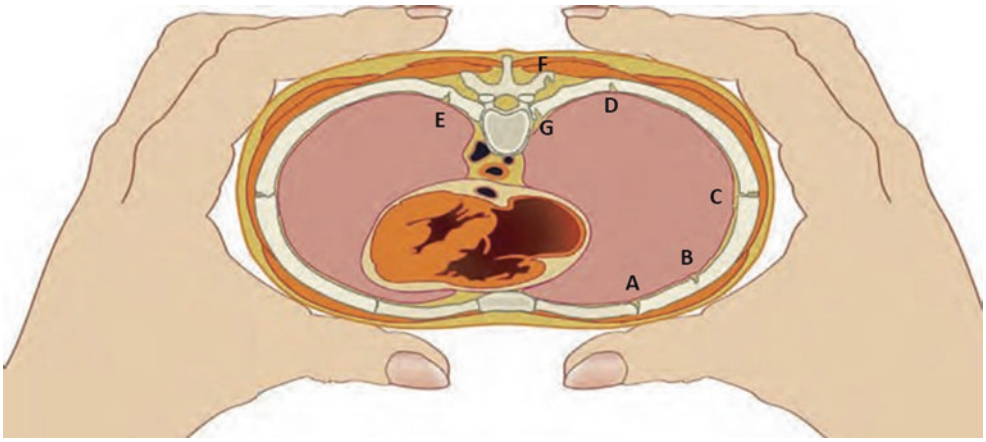


Fig. 7.3 Schematic representation of bimanual chest compression. Fractures locations at the: (**a**) Costochondral junction. (**b**) Anterior costal arch. (**c**) Lateral costal arch. (**d**) Posterolateral costal arch. (**e**) Posterior costal arch (costal tubercle). (**f**) Transverse process **(g)** Head of rib

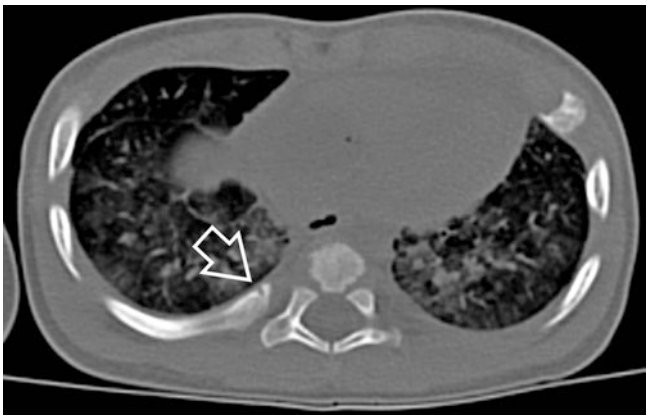


Fig. 7.4 A posterior rib fracture with disruption of the ventral cortex in an infant after bimanual resuscitation with vigorous thoracic compression

behind' [19]. According to Kleinman there is no scientific support for the views of Cameron and Rae and of Smith et al. [20]. However, in a publication by Bixby, which was coauthored by Kleinman, the hypothesis of Smith et al. is illustrated in a case report, concerning a 13-month-old boy who sustained multiple right-sided posterior and lateral rib fractures, including posterior fractures near the costovertebral junction [21]. The left-sided ribs were intact. The boy was sitting in a stroller, which was pushed across the street by his mother. The stroller was struck by an automobile at an unknown speed and the boy was ejected from the stroller and landed on the pavement approximately 2.5 metres away. He also sustained a minimally displaced and angulated distal right femur fracture. Bixby et al. concluded the abstract with: '*In this patient, the presumed mechanism of injury was consistent with the compressive forces that cause rib fractures in abused infants and young children. This case illustrates how a high-impact traumatic event may cause rib fractures that would otherwise point strongly to abuse'*.

Worn and Jones, just like Cameron and Rae, stated that violent lateral compression can lead to a situation in which the ribs are forced to fracture [5]. They also stated that lateral compression was (and still is) much less well-documented as cause of rib fractures, compared to anterior-posterior compression. Concerning dynamic impact loading, as described by Smith et al. [19], Worn and Jones are of the opinion that: '*Though unreported, it is reasonable to postulate that other scenarios such as direct blows (e.g. automotive accidents, resulting in a rapid lateral impact to the thorax, heavy objects falling or pressing on the lateral aspect of the chest, or a child falling onto a rigid surface) and manual assaults (e.g. slamming the infant sideways onto a solid surface, and violent squeezing) could also result in the required lateral compression'*.

The opinion of Worn and Jones, that lateral fractures could be caused by dynamic impact loading is supported by Bradley et al. and Blackburne, who, based on their research in piglets, concluded that complete lateral rib fractures in infants may in fact not be the result of pure compression and the biomechanical analysis of their findings suggests that blunt force trauma might offer a more credible explanation for lateral rib fractures in infants [22, 23].

7.2.2 Dynamic Impact Loading

In only a minority of cases, rib fractures in paediatric patients are caused by a direct impact to the chest. This is true, irrespective of the circumstances: accidental (e.g. a fall on an object or an accident) or non-accidental (e.g. a blow/punch or kick)(Sect. 7.3.3). Rib fractures can also be sustained by sudden deceleration, when a child hits a blunt object or wall at high speed (a.k.a. inertial loading).

Depending on the nature of the force applied, the fracture occurs at either the place of impact or the place where as a result of the impact the greatest stress is exerted on the rib(s) (Figs. 7.6 and 7.7). Often bruising will be found at the impact site [24].

Rib fractures due to dynamic impact loading are found mostly in mobile children above the age of 2 years, whereas

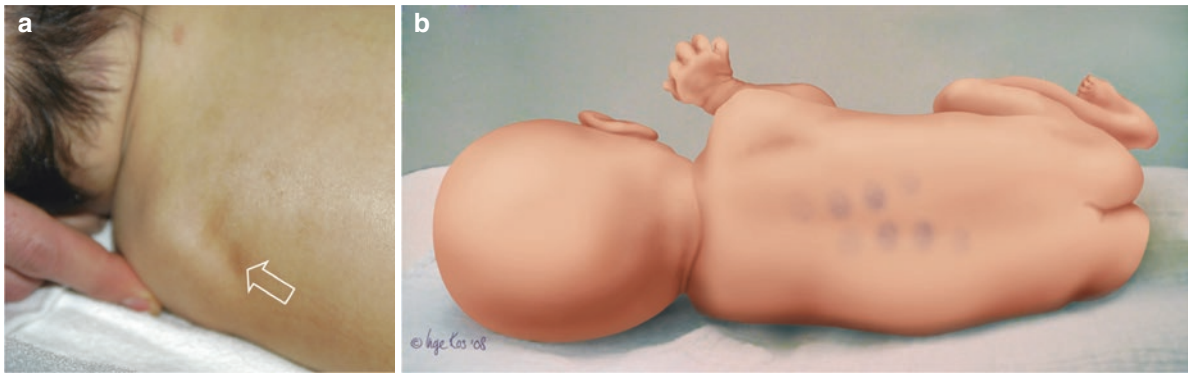


Fig. 7.5 (**a**) Fingerprint bruises (open arrow) on the back of an infant admitted to the intensive care unit with a Glasgow coma score of 3. (**b**) Graphic representation of fingerprint bruises on the back, resulting from compression of the chest

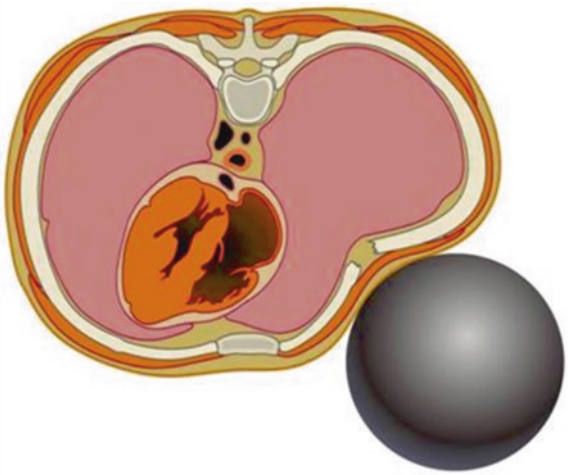


Fig. 7.6 Graphic representation of a rib fracture at the impact site after blunt chest trauma with deformation of a part of the rib arch and cage in the immediate vicinity of the fracture(s)

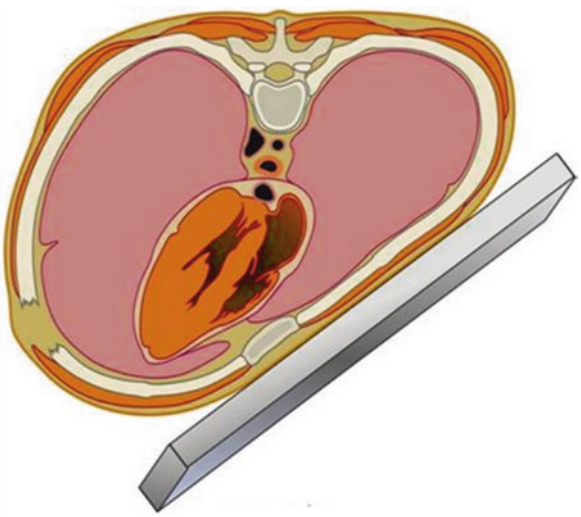


Fig. 7.7 Graphic representation of a rib fracture at the site of the highest stress after blunt chest trauma with deformation of a part of the rib arch and cage in the immediate vicinity of the impact site and on the other side

fractures due to static loading are predominantly found in younger often non-mobile children [17].

7.3 Manner of Rib Fractures

Rib fractures may occur intrauterine, during birth or after birth. Rib fractures that occur after birth can be due to accidental circumstances (e.g. a fall or motor vehicle accident), non-accidental circumstances (inflicted injuries), or iatrogenic (e.g. in resuscitation or connected to surgical procedures). Rib fractures are rarely seen due to circumstances like birth, resuscitations, and physiotherapy.

In addition to the foregoing, it should be noted that in children with bone disease rib fractures can occur with a lower load than in a child without bone disease. It should also be noted that the occurrence of rib fractures in these children is the result of a trauma, in which the loading of the rib(s) (= the transferred energy) exceeded the maximum load-bearing capacity of that bone, just like in children without a bone disease.

7.3.1 Trauma Before Birth

In the literature only a few case reports are found, in which the intrauterine occurrence of one or more than one rib fracture has been described (Fig. 7.8).

Van Mieghem et al. reported that intrauterine trauma due to the impact of the unrestrained foetus with the maternal pelvis or with extra maternal structures (seat belt, airbag, or steering wheel) is rare [25]. They found foetal injuries in less than 1% of the motor vehicle accidents, they evaluated, in which an unborn child is involved. Limb, rib, and skull fractures, and intracranial haemorrhages were the most common lesions.

More commonly it will concern foetuses with lethal forms of skeletal dysplasia's, resulting in an increased risk of intra-

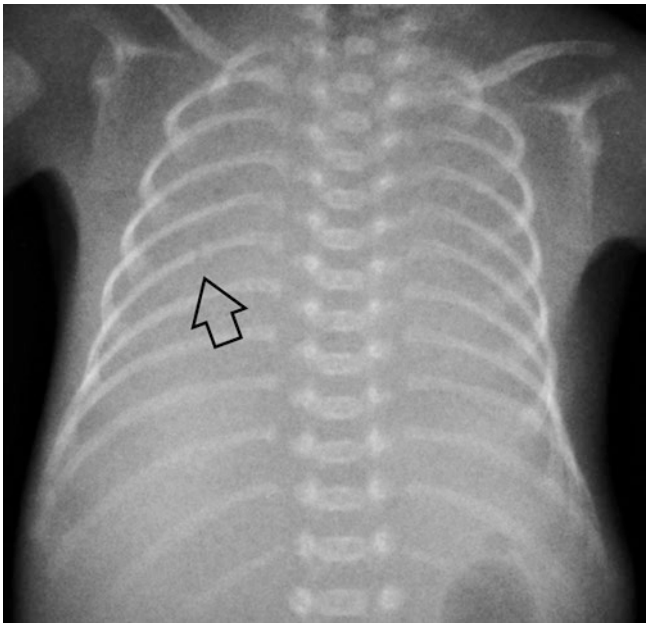


Fig. 7.8 Posterior rib fracture with mild callus formation (arrow) seen in a babygram of a foetus aborted at 23 weeks gestational age. The presence of callus excludes the termination of pregnancy procedure as a cause

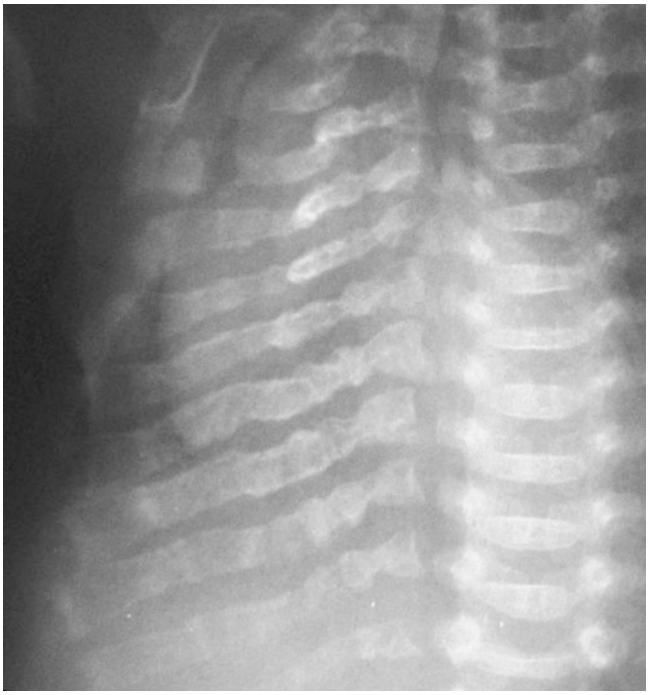


Fig. 7.9 Infant with osteogenesis imperfecta type IIA, born at 35 weeks gestational age. Prenatal ultrasonography demonstrated abnormal ribs and extremities. The infant died several hours after birth. Note the short thick ribs with continuous beading due to multiple fractures and generalized loss of height of the vertebral bodies due to collapse

uterine fractures. Usually there are multiple rib fractures, which often are already diagnosed before birth:

- Osteogenesis imperfecta type II (perinatal lethal osteogenesis imperfecta—OMIM #166210) (Figs. 7.9 and 14.13) [26–31].
- Lethal achondrogenesis type IA (Houston-Harris type—OMIM #200600) [27, 32, 33].
- Lethal osteopetrosis (marble bone disease, Albers-Schonberg disease—OMIM #259700) [34, 35].

7.3.2 Trauma During Birth

Skeletal lesions may occur in otherwise normal children as a result of a traumatic delivery, such as breech birth [36]. Clavicle fractures are the most prevalent and present in 1.7% to 3.5% of neonates [37].

During birth there is a great deal of force on the ribcage during the passage through the birth canal. Yet, it is rare to see rib fractures directly after delivery [38–40]. When they are present, they result either from trauma around the time of birth or in utero, from a rigid ribcage or from congenital disorders [40]. In a prospective study of 15,435 births, Rubin did not find any rib fractures [41]. In 43 children he found a clavicle fracture, in seven a humerus fracture and in one a skull fracture. In a retrospective study of over 20,000 births, Camus et al. also did not find any rib fractures [42]. Neither did Bhat et al. in their study of 34,946 live-born infants [43]. The literature only counts a limited number of case reports on rib fractures in newborn infants (Table 7.3).

It seems to be unlikely that rib fractures resulting from birth are present in normal-term birth and after a normal postpartum physical examination. All children described up to the present day had, as far as one can evaluate from case reports, almost always a high to very high birth weight. Shoulder dystocia is also regularly described as a risk factor. Only one child had a normal birth weight of 3300 g. The delivery of this child was complicated, because of shoulder dystocia [44]. Rib fractures have also been described as a result of complications during a breech or other traumatic delivery (Fig. 7.10) [36, 40, 45]. Various symptoms may indicate a traumatic birth: cephalic haematoma, bruising, swelling, and subcutaneous crepitus. A relatively common finding in birth-related rib fractures is the presence of ipsilateral clavicular fractures [40, 46–48].

Rib fractures can also occur during childbirth in children with a metabolic or genetic bone disease with an increased risk of fractures, such as severe osteogenesis imperfecta (OI) [38].

Table 7.3 Rib fractures resulting from delivery (Reprinted with permission [40].)

Author	Sex	Birth weight (g)	Dystocia	Delivery	Fracture location
Thomas [76]	Nk	5896	Nk	Forceps	Right posterior ribs 5–7
Rizzolo [44]	Nk	3300	+	Vacuum	5 ribs posterolateral[a]
Barry [189]	Nk	5020	+	Normal	5 ribs posterior[a]
Hartmann [190]	F	3912	–	Vacuum	Right posterior ribs 4–8
	M	4205	–	Vacuum	Right posterior ribs 6–8
Bulloch [46]	Nk	3946	+	Vacuum	Right posterior ribs 4–6 + right clavicle
Durani [47]	M	4309	–	Normal	Left posterior rib 7 + left clavicle
Ibanez [191]	M	3800	+	Vacuum	Right posterior ribs 7 and 8
Landman [48]	F	4400	–	Normal	Left posterior ribs 5–7 + left clavicle
	M	4500	–	Normal	Left posterior ribs 5–7
Van Rijn [40]	F	5070	+	McRoberts	Right posterior ribs 6 and 7 + right clavicle
	M	5020	+	Forceps	Left posterior ribs 4–6 + left clavicle
	M	4300	+	Normal	Left posterior ribs 5–8 + left clavicle
	F	5656	+	Normal	Right posterior ribs 4–9 + right clavicle
Jovanović [192]	M	4650	–	Normal	Left posterior ribs 3–7
Khan [193]	M	4040	+	Forceps	Left posterior ribs 5–7

Nk not known
[a] Number known, exact location unknown

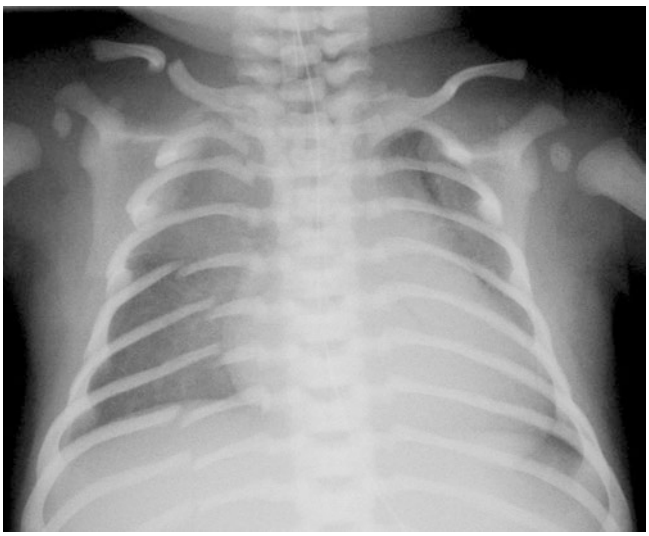


Fig. 7.10 Term neonate with a birthweight of 5.5 kg born after a traumatic vaginal delivery. Chest radiograph on day 3 shows multiple posterior rib fractures and an ipsilateral fracture of the clavicle (reprinted with permission from 'Birth-related mid-posterior rib fractures in neonates: a report of three cases (and a possible fourth case) and a review of the literature' [40]

7.3.3 Trauma After Birth: Accidental and Non-accidental Circumstances

7.3.3.1 Epidemiology of Rib Fractures, Due to Trauma After Birth

Rib fractures in adult and in paediatric patients are always due to thoracic trauma. Rib fractures are identified in approximately 10 to 20% of adult trauma victims [49, 50]. The prevalence of rib fractures in paediatric trauma patients is probably lower, and is estimated to be around 1 to 2% of all paediatric trauma patients, irrespective of the circumstances under which the fractures were sustained.

In young children, the circumstances of the trauma can be either non-accidental, most commonly due to compression (Sect. 7.2.1) or accidental, most commonly a severe blunt force trauma of the thorax, often as a pedestrian/cyclist in motor vehicle accidents (Sect. 7.2.2). If rib fractures are found in young children with congenital bone disorders, these are also always due to a trauma, although probably less force will be needed for the fracture to occur (Sect. 7.3.5.2). Rib fractures in older children and adults are most commonly sustained in accidental trauma, like motor vehicle accidents.

The first, and probably the most important reason for the low reported prevalence of rib fractures in young paediatric trauma patients compared to older paediatric trauma patients and adolescent or adult trauma patients, is the malleability of the thorax in paediatric patients, especially in young children. The higher malleability is due to the rib cage being more flexible and the costal cartilage being unossified. In young children therefore ribs are more likely to deform than to break, unless a certain level of loading of the ribs is exceeded [51, 52]. Due to this malleability, it can be ruled out that rib fractures arise during normal handling of children or during normal daily care [53].

A second reason why rib fractures in paediatric trauma patients are diagnosed less often, especially in young children, is because they can easily be missed. Often there are no conspicious clinical symptoms and complications which could be a reason for a referral for a radiological examination or for treatment. It is assumed that approximately 80% of rib

fractures in young children do not give any complaints [54]. Complaints are only seen in case of irritation of the pleura or in case of other injuries. Irritation of the pleura may cause pain and lead to noticeable crying. Very rarely complications may occur in paediatric patients, irrespective of the circumstances under which the rib fractures were sustained, e.g. rupturing of intercostal vessels or the occurrence of haemothorax, pneumothorax, bilateral chylothorax, pleural effusion, or subcutaneous emphysema (Sect. 7.3.3.2) [55]. If fractures of the lower ribs are found, one should be aware of simultaneous damage to the spleen, stomach, and/or bowels [17].

A third reason for the low reported prevalence is that fresh rib fractures can be missed on standard radiographs, because of the complex geometry of the ribs and overprojection of the spine. Therefore, if there is a suspicion of a fracture, or suspicion of abuse, additional oblique views are mandatory (Fig. 7.11a–c). In particular, fresh paravertebrally localized fractures are, unless there is dislocation of the fracture, not always visible on radiographs [53]. Occasionally, pleural thickening is the only sign that should raise concern for an occult rib fracture (Fig. 7.12a–c). In those cases, healing reactions of the bone may become visible over time. Therefore a second radiological examination is always indicated if non-accidental trauma is suspected (Fig. 7.12d). In deceased children, a post-mortem CT scan of the chest offers more accuracy in the acute phase than conventional chest radiography (Fig. 7.13a, b). The diagnostic accuracy of CT in living children, however, should be studied further [56].

The foregoing means that in an unknown number of young children rib fractures will never be diagnosed. Rib fractures are probably the most undiagnosed fractures, irrespective of the circumstances under which the fractures were sustained. Often when they are found, it is a coincidental finding in a child, that is examined for other reasons, or a finding within the scope of a full radiological examination in a young child in whom non-accidental trauma is suspected [57–59]. It has been established that when rib fractures are found in non-accidental trauma, they are rarely the only injury (Fig. 7.14a–f) [60].

Garcia et al. retrospectively analyzed the findings in 2080 children, aged between 0 and 14 years, who were admitted with a blunt or penetrating trauma to a Level 1 paediatric trauma centre between 1985 and 1988 [61]. Thoracic trauma was present in 104 children (5.9%). Of these children, 33 (1.6% of all admissions; 31.7% of the children with thoracic trauma) had rib fractures. The mean age of the children with rib fractures was 4.7 years. Nearly 60% of the children with rib fractures were 4 years old or younger. Rib fractures were most commonly (nearly 70%) sustained in traffic accidents as pedestrian or as crash occupant. Falls were identified in 9.1%. In the remaining cases the rib fractures were due to non-accidental trauma. Most of the children with non-accidental trauma were under the age of 3 years. Non-accidental trauma accounted for 63% of the injuries in children under the age of 3 years, while pedestrian injuries predominated among older children. Children with traffic accidents and with non-accidental trauma had more rib fractures than children involved in falls (mean number of rib fractures crash occupant 4.7, pedestrian 3.0, non-accidental trauma 4.6, and fall 1.3). Seventy percent of the children with two or more rib fractures had multisystem involvement. Six children had posterior rib fractures, of whom five were injured as pedestrians. Five children with posterior rib fractures also had additional intrathoracic injuries. Overall children with rib fractures were more severely injured than children without rib fractures. Trauma scores or mortality rates between children with posterior rib fractures and children with rib fractures in other locations were not significantly different. There were 14 deaths among 33 children with rib fractures, which constitutes a mortality rate of 42%. When compared to children without rib fractures, children with rib fractures had a higher mortality rate, but there was no statistically significant difference in morbidity between the 2 groups.

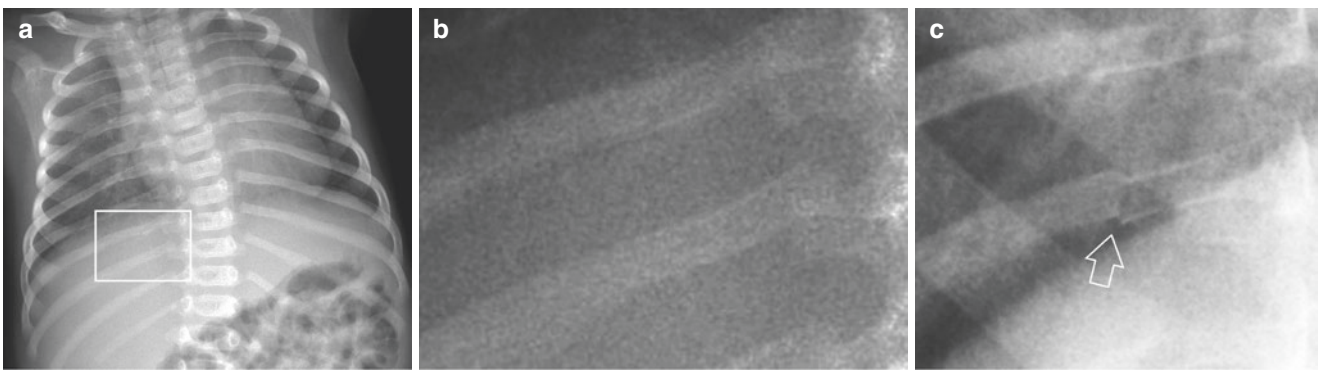


Fig. 7.11 An infant with unexplained bruises on the chest and a posterior rib fracture of the tenth rib on the right side. (**a**) AP chest radiograph does not show the fracture (inside of the box). (**b**) Detail of the box. (**c**) Only one of the oblique views clearly shows the fracture

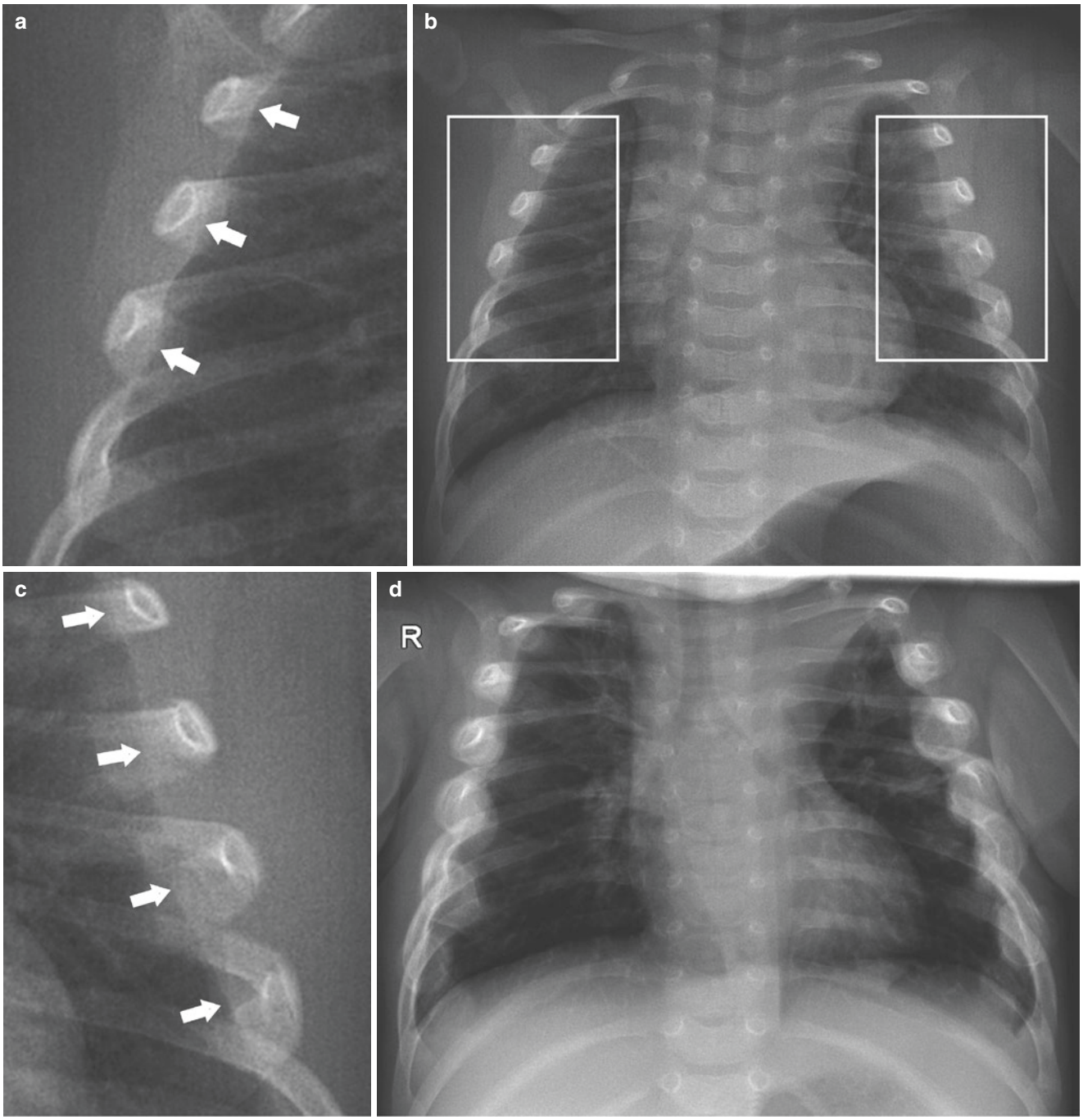


Fig. 7.12 An infant with multiple bruises of unknown origin. (**a**–**c**) Chest radiograph and detailed views show pleural thickening (arrows). (**d**) A second radiograph, 20 days later, shows massive bilateral periosteal new bone formation of multiple ribs. A skeletal survey also revealed metaphyseal avulsion fractures and fractures of the foot and tibia. The father confessed non-accidental injury

The risk of mortality increased with the number of ribs fractured. The mortality rate for the 18 children with both rib fractures and head injury was 71%.

Hedström et al. evaluated the fracture pattern in 10,203 fracture events, that had resulted in a total of 10,327 fractures, in paediatric patients under the age of 19 years [62]. The complete evaluation concerned the period from 1993 to 2007 in northern Sweden. In 2006 and 2007 in northern Sweden a total of 1551 fractures were registered, of which about 1% (n = 17) were rib fractures, irrespective of age of the paediatric patient or the circumstances under which the fractures were sustained. The exact prevalence of rib fractures in paediatric patients, however, is unknown, because of several reasons.

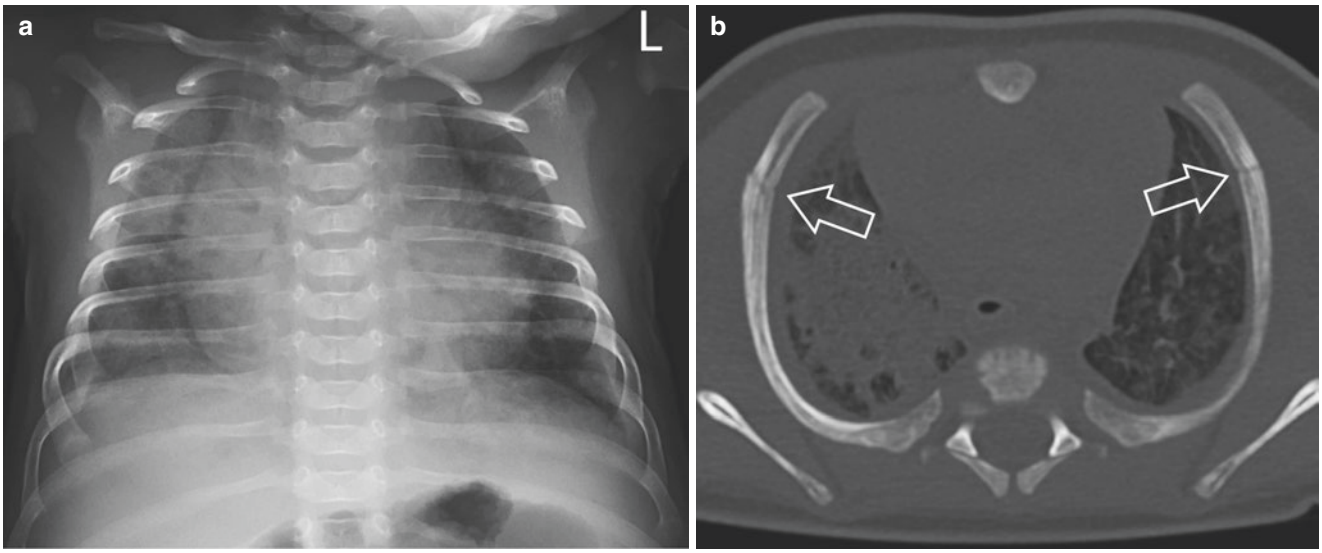


Fig. 7.13 Post-mortem examination of an infant. Cause of death was bronchiolitis with pneumonia after prolonged resuscitation (**a**). Chest radiograph shows pulmonary abnormalities and pleural thickening but no rib fractures (**b**). CT scan depicts subtle, but evident anterior rib fractures (arrows). In total 8 fractures were discovered on CT, believed to be caused by the resuscitation

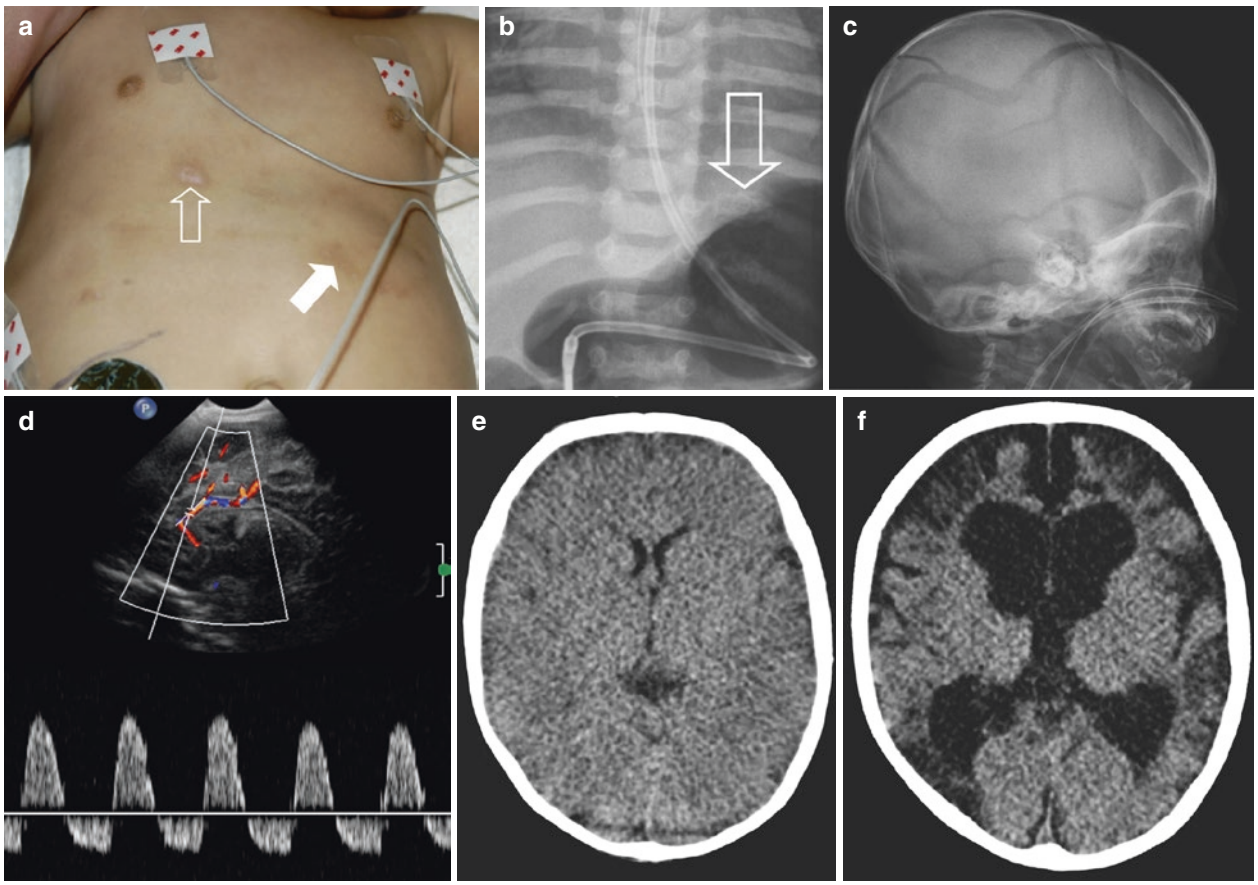


Fig. 7.14 (**a**) Infant admitted to the intensive care unit with a Glasgow coma score of 3. At physical examination skin lesions are found corresponding with burns, possibly a cigarette (open arrow) and a haematoma suspect of a bite injury (arrow). (**b**) Spinal view of the skeletal survey shows healing rib fractures (open arrow) on the left posterior side. (**c**) Skull shows a comminuted bilateral skull fracture (besides the physiologically open sutures). (**d**) Doppler ultrasound of the skull made at the paediatric intensive care unit shows a retrograde flow during diastole in the pericallosal artery. This is congruous with intracranial pressure in cerebral oedema. (**e**) CT at admittance shows an oedematous swollen brain with signs of hypoxic-ischaemic injury. (**f**) CT made 7 months after the first day of hospitalization shows severe focal and diffuse tissue loss

Table 7.4 Circumstances under which paediatric trauma victims had sustained rib fractures [51]

	n = 328	%
Accidental		
• Pedestrian hit by a car.	132	40.24
• Falls from height.	68	20.73
• Driver or passenger in a motor vehicle accident.	53	16.16
• Bicycle.	25	7.62
• Falls (non-height).	12	3.66
• Motorcycle.	1	0.3
Non-accidental		
• Assault.	9	2.74
Other (not further specified by the authors)	28	8.54

Table 7.5 Circumstances under which the 65 children under the age of 3 years had sustained rib fractures [63]

	n = 65	%
Non-accidental	47	72
Accidental	18	28
• Pedestrian hit by a car.	7	39
• Falls from height.	5	28
– Two- to four-story falls in 4 children.		
– Fall downstairs in adult's arms with associated crush injury,		
• Passenger in a motor vehicle accident.	4	22
• Television fallen on child.	1	
• Kicked by horse.	1	

Kessel et al. analyzed retrospectively the circumstances, under which 328 paediatric trauma victims (aged 0 to 15 years) had sustained rib fractures [51]. Most rib fractures were due to accidental circumstances. In only 2.74% ($n = 9$) the rib fractures were determined to be due to an assault (Table 7.4). Unfortunately, the authors did not age-related specify the circumstances. Also, the term 'assault' was not defined. Also in 8.54% the circumstances were not further specified by the authors.

Darling et al. reviewed the occurrence of rib fractures and associated injuries due to accidental and non-accidental trauma (period 2003 to 2010) in 65 children under the age of 3 years [63]. Children with bone demineralization were excluded. Of these children 47 (72%) (mean age 4 months) sustained non-accidental rib fractures and 18 (28%)(mean age almost 20 months) sustained accidental rib fractures (Table 7.5). Children with non-accidental trauma were younger than children with accidental trauma. Children with non-accidental trauma had more rib fractures than children with accidental trauma.

Skinner et al. analyzed the findings in 84 paediatric patients under the age of 18 years with severe blunt thoracic trauma [64]. In 63 patients (75%) the patient was a pedestrian hit by a car. Seventeen patients (20.2%) were involved in a motor vehicle accident as a passenger and 1 patient (1.2%) was involved as a driver. In 2 patients (2.4%) the trauma was due to a structural collapse or to a blunt force trauma (not further specified). In 1 patient (1.2%) the trauma was due to a fall. Rib fractures were found in 17 patients (20.2%). A flail chest was found in 2 patients (2.4%). Non-accidental trauma is mentioned in the text (*'Very few patients had injuries from structure collapse, assaults, motorcycle accidents or falls'*) no numbers are found in the article concerning non-accidental trauma.

Wegmann et al. retrospectively analyzed the findings in 248 infants (54% male, 46% female) under the age of 1 year (mean age of 7 months) who presented with fractures in an 11 years period (2001–2011) [65]. They described the location of the fractures, sites of the accident, circumstances, and mechanisms of injury, that lead to the fractures. Six patients (2%) were determined to have sustained fractures due to non-accidental trauma. These children sustained a total of 10 fractures (2 skull fractures, 4 proximal humeral fractures, 2 rib fractures, and 2 tibial fractures). No other children with rib fractures were identified. In none of the children, who fell from sitting/standing, fell from 50, 100, or 150 cm (20, 40, or 60 inches), fell from a stair or had an external impact, a rib fracture was diagnosed.

Naqvi et al. retrospectively reviewed the findings in all cases of moderate to severe trauma (abbreviated injury scale score of 2 and above in one or more body regions) of 213 children under the age of 16 years (mean age 7.8 years; standard deviation 5.2 years) in a major trauma centre in the United Kingdom (2012–2014) [66]. Blunt force trauma was present in almost all cases, except in 3 cases with penetrating trauma. Motor vehicle-related trauma was most common (46%), followed by falls under 2 metres (26.8%) and falls above 2 metres (12.2%). Non-accidental trauma was suspected in 3.6% ($n = 8$). Thoracic injuries were less common (16.9%) than head injuries (54%) or injuries to the extremities (39.9%), but children with thoracic injuries had the highest mortality rate (13.9%) with an overall mortality rate of 6.6%. None of the injuries to the thorax were isolated. All cases, in which non-accidental trauma was suspected, presented with either head injury or asphyxia-related brain injury. The authors did not describe the number of rib fractures and concomitant injuries in these children.

Hagedorn et al. reviewed the findings in 455 patients, aged 0 to 18 years, who were admitted (2004–2013) to a level I trauma institute following an all-terrain vehicle-related accident [67]. One or more than one thoracic injury was present in 102 patients (22%). The most common injuries were pulmonary contusion in 61%, pneumothorax in 45%, and rib fractures in 34% of the patients. The mean age of the patients with chest injuries was 11.5 years. Eight patients with chest injury died (8%) compared to 2 patients without chest injury (0.6%).

Ruest et al. evaluated retrospectively the findings in 7530 infants, aged between 0 and 2 years, with a total of 9720 radiographs of the thorax, to determine the prevalence of inci-

dental rib fractures identified by radiographs of the thorax obtained for indications unrelated to accidental or non-accidental trauma (study period 01 January 2011 to 31 October 2016) [68]. They identified incidental rib fractures on 5 chest radiographs, making the prevalence of chest radiographs with incidental rib fractures in their study population under 0.1%. The mean age of infants with incidental rib fractures was 3.6 months. According to the authors, the rib fractures were concerning for non-accidental trauma in 3 of the 5 infants. In 1 infant non-accidental trauma was confirmed. The fifth child had radiological findings consistent with osteopenia of prematurity. Ruest et al. concluded that the finding of incidental rib fractures is rare in children who are evaluated for indications unrelated to accidental or non-accidental trauma, but also were of the opinion that when rib fractures are found in the absence of a corresponding accidental trauma history and/or objective laboratory or radiological indications of metabolic bone disease, work-up for non-accidental trauma should be done. They also stated that alternative explanations for rib fractures occasionally used in a medico-legal context such as minor accidental trauma, undiagnosed medical conditions, and transient metabolic bone disturbances are unlikely to be the aetiology of incidental rib fractures.

Ruest et al. also reviewed chest radiographs of infants under the age of 2 years, which were made either for known accidental trauma (n = 226; mean age 10.8 months; SD 7.4 months) or for suspected non-accidental trauma (n = 51; mean age 5.3 months; SD 5.6 months) at a paediatric level 1 trauma centre [69]. Only one patient with a known accidental trauma had acute rib fractures on two chest radiographs, made 5 days apart. The rib fractures were located in the posterior part of the seventh and eighth rib and were associated with a small haemothorax. The infant was involved in a high-speed, rollover motor vehicle crash with an unstrained adult passenger landing on top of and subsequently crushing the infant. In the 'accidental' group no healing rib fractures were found. Despite a wide variety of injuries caused by minor to major trauma the presence of rib fractures in paediatric accidental trauma was uncommon. Even none of the four deceased children in this group had acute rib fractures identified on the first chest radiograph. In the group, in which non-accidental trauma was suspected, 10 of the 51 revealed rib fractures (9 of 10 showed only healing rib fractures and 1 showed acute rib fractures. After additional work-up (e.g. skeletal survey, laboratory evaluation, and head CT) and/or perpetrator confession, 38 of the 51 cases were determined to be consistent with non-accidental trauma and the remainder was characterized as accidental trauma, neglect, or normal variants (e.g. birthmarks). In 4 infants, in whom non-accidental trauma was suspected, the diagnosis remained unclear. The overall prevalence of rib fractures identified on chest radiographs in infants with a final diagnosis of non-accidental trauma was much higher.

7.3.3.2 Morbidity and Mortality Associated with Rib Fractures

Trauma is the leading cause of death in paediatric patients, under the age of 18 years. Although thoracic trauma accounts for only 5–12% of admissions to a trauma centre, it is second only to head injury as the most common cause of death [4]. According to Sharma, mortality is 5% for isolated thoracic trauma, approaches 20% in patients with concomitant abdominal injuries, and exceeds 30% in patients with concomitant head injuries [4]. Of the children with a blunt chest trauma, who die, approximately 15% will die directly from intrathoracic injuries, but in case of penetrating chest trauma, nearly 100% of the deaths result from the intrathoracic injuries [70]. In more than 50% of children with severe thoracic trauma, thoracic trauma is accompanied by injuries to other organ systems, due to the proportionately smaller size of the chest compared to the abdomen or the head in a young child. For that reason, a significant thoracic trauma should initially be treated as a multi-systemic trauma with a high risk of multi-organ involvement and an increased mortality (Fig. 7.15a, b) [4, 71].

In most of the adult trauma patients, the presence of rib fractures is associated with an increased morbidity and mortality, commonly due to the combined occurrence of rib fractures and injuries to the intra- and extrathoracic organs [49, 50]. In contrast children with a severe trauma to the chest more often have major intrathoracic injuries with minimal or no injury to the bony thorax [4]. As stated in Sect. 7.3.3.1 the rib cage in paediatric patients is much more flexible than the cage of adult patients. Therefore, the paediatric rib cage can absorb a larger amount of kinetic energy from an impact, with a higher transmission of the kinetic energy to the intrathoracic organs, resulting in a higher probability of serious multi-organ injuries and even death, compared to adult patients [4, 51].

Schweich and Fleisher reviewed the records of 21 children with rib fractures [72]. They found distinctive differences between children who sustained the fractures either in accidental or in non-accidental circumstances (Table 7.6). Sixteen children (76%) sustained fractures due to an accidental trauma, most commonly by pedestrian-motor vehicle accidents (age range 2 to 15 years) and 5 children (24%) sustained fractures due to non-accidental trauma (age range 3–7 months). Nineteen children fractured an average of 3.5 ribs, with a range of 1–8. Two children fractured 22 and 23 ribs. In the group of children with an accidental trauma, the number of rib fractures ranges from 1 to 8 ribs (median 2.5 ribs) and in the group of children with accidental trauma, the number ranged from 3 to 23 ribs (median 7 ribs). Neither an increased number of fractures nor first or second rib fractures were associated with more severe intrathoracic injuries. The five children with non-accidental rib fractures were young, had an unexplained history, and a paucity of multiple trauma.

Nakayama et al. reviewed the records of 105 children, aged 1 month to 17 years (mean age 7.6 years) with chest

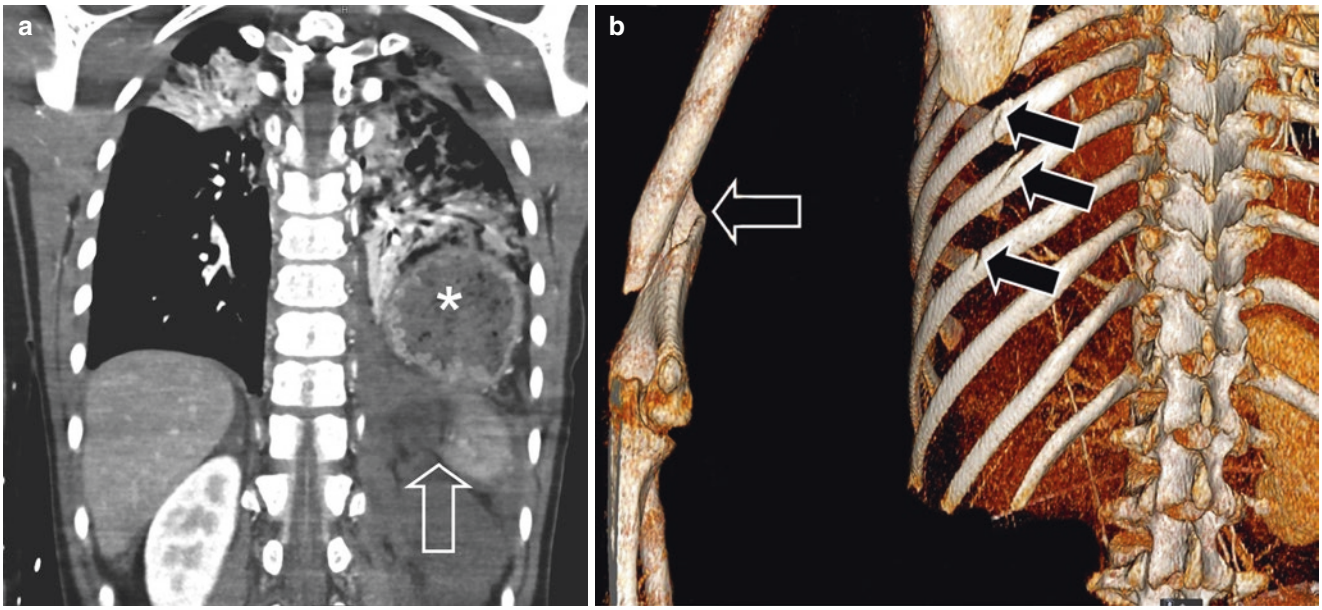


Fig. 7.15 (**a**, **b**) Teenage cyclist who was involved in a MVA. (**a**) Coronal image shows a, surgically proven, diaphragmatic hernia with the stomach displaced into the chest cavity (asterisk) and a splenic laceration (arrow). (**b**) Posterior 3D rendering shows an oblique distal humerus fracture (arrow) and three left-sided posterior rib fractures (black arrows)

Table 7.6 Differences between accidental and non-accidental trauma in 21 children with rib fractures

	Accidental (n = 16)	Non-accidental (n = 5)
Primary complaint	Severe trauma with adequate clinical history: Traffic accidents, fall from a height, shot wounds	Unexplained respiratory problems (usually no complaints)
Age child		
• Average	8 years and 7 months	3 months
• Range	2–15 years	0.5–7 months
Number of fractures		
• Average	3.3	11.8
• Range	1–8	3–23

injuries [73]. Nearly all injuries (97.1%) were due to blunt trauma, and more than 50% were traffic related. Rib fractures, commonly multiple, and pulmonary contusions occurred, respectively, 49.5% and 53.3% of the children, followed by pneumothorax in 37.1% and haemothorax in 13.3%. In 52% of the children with blunt trauma significant intrathoracic injuries occurred without rib fractures. Associated head, abdominal, and orthopaedic injuries were present in 68.6% of children.

Garcia et al. retrospectively analyzed the findings in 104 children, aged between 0 and 14 years, with a thoracic trauma, who were admitted to a Level 1 paediatric trauma centre (see also Sect. 7.3.3.1) [61]. Thirteen children (31.7%; mean age 4.7 years) had rib fractures. Nearly 60% of the children with rib fractures were 4 years old or younger. Seventy percent of the children with two or more rib fractures had multisystem involvement. Overall children with rib fractures were more severely injured than children without rib fractures. There were 14 deaths among the 33 children with rib fractures (42%). When compared to children without rib fractures, children with rib fractures had a higher mortality rate, but there was no statistically significant difference in morbidity between the two groups. The risk of mortality increased with the number of ribs fractured. The mortality rate for the 18 children with both rib fractures and head injury was 71%.

Kessel et al. compared retrospectively the findings in adult and paediatric trauma victims with rib fractures, irrespective of the circumstances under which the fractures were sustained: 6627 adults (aged 15 years and older) and 328 children (aged 0–15 years) [51]. Isolated rib fractures without associated injuries were found in 19 children (5.8%) and in 731 adults (11%). Adults had more often 4 or more fractured ribs than children. Children had significantly higher rates of associated brain injuries, abdominal solid organ (especially spleen and liver) injuries, pneumothorax/ haemothorax and lung contusions than adults. In case of associated extrathoracic injuries, mortality rate in both groups was around 6%. None of the children without extrathoracic injuries died, while 0.64% of the adults without extrathoracic injuries died.

According to Rosenberg et al. the number of rib fractures correlates with mortality in adult trauma patients [52]. This rate rises in adults sharply above six fractured ribs. The authors evaluated the data of 729,240 paediatric patients under the age of 21 years, of whom 19,442 had rib fractures, to see whether the number of rib fractures also correlates with the mortality rate in paediatric patients, just like in adult

trauma patients. The mortality rate increased from 1.79% in paediatric patients without rib fractures to 5.81% for one rib fracture and then nearly linearly increased to 8.23% for seven fractures. They also found that ventilator days also increased with increasing number of rib fractures.

Darling et al. reviewed the occurrence of rib fractures and associated injuries due to accidental and non-accidental trauma (period 2003 to 2010) in 65 children under the age of 3 years (see also Sect. 7.3.3.1) [63]. Children with bone demineralization were excluded. Of these children 47 (72%) (mean age 4 months) sustained non-accidental rib fractures and 18 (28%)(mean age almost 20 months) sustained accidental rib fractures (Table 7.5). Children with non-accidental trauma were younger than children with accidental trauma. Although children with non-accidental trauma had more rib fractures than children with accidental trauma, intrathoracic injuries as a whole and individual types of intrathoracic injuries (pneumothorax, pleural effusion, pulmonary contusion, pulmonary laceration) were more common in children with accidental trauma. Rates of other thoracic cage injuries (fractures of the clavicle, scapula, sternum and thoracic vertebrae) did not differ substantially between children with accidental or non-accidental trauma. Intracranial and intra-abdominal injuries and skull fractures were equally frequent, but fractures of the extremities were more common in children with non-accidental trauma. According to the authors mortality rates were not dissimilar between the non-accidental (8.5%) and accidental (11.1%) groups. Darling et al. explicitly stated that '*lack of intrathoracic injuries in abused children with rib fractures does not imply bone fragility*'.

Despite the fact that severe or fatal intrathoracic injuries usually are less common in children with non-accidental trauma than in children with accidental trauma, non-accidental trauma to the chest can lead to severe or fatal intrathoracic injuries. Cohle et al. reported 6 young children, aged 9 weeks to 2½ years (5 girls, 1 boy), with intentionally inflicted cardiac lacerations [74]. Rib fractures were found in 4 children, with at least two fractures in each child. According to Cohle et al. cardiac rupture from blunt force trauma most commonly is caused by compression of the heart between the sternum and vertebral column, but it may also occur from compression of the abdomen or legs, deceleration, blast injury, puncture of the heart by a fractured rib and rupture through a resolving contusion. The authors concluded that cardiac lacerations, as with other types of severe trauma acquired at home, are almost never sustained in accidental circumstances.

7.3.3.3 Accidental Versus Non-accidental Rib Fractures

In 2004 Williams and Connolly published an analysis of ten articles from the medical literature to arrive at a number of general conclusions on rib fractures in young children [53]. They summarized their conclusions in the following clinical bottom line:

- The likelihood that rib fractures are due to non-accidental trauma decreases as the child grows older.
- Rib fractures in children under the age of 3 years old are very suspect for non-accidental trauma.
- The absence of fractures on a radiograph does not exclude their presence (Fig. 7.16a–c). In particular, fresh paravertebral-localized fractures are not always (clearly) visible on radiographs, unless there is dislocation of the fracture (Fig. 7.17a, b).

The analysis of Williams and Connolly still is valid. Based on the data described in Sect. 7.3.3.1 the following conclusions can be drawn:

- Accidental rib fractures are rare in children under the age of 3 years. If they do occur, they are almost always the result of a severe trauma to the chest, e.g. an accident in which the child was hit by a car. Often the thoracic inju-

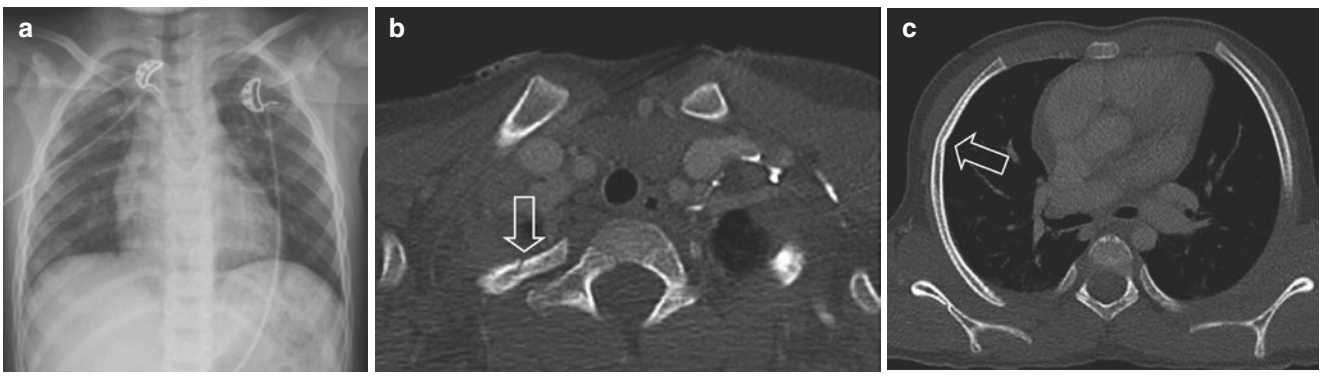


Fig. 7.16 (**a**–**c**) Child involved in a car accident, the car ran over the child. (**a**) Radiograph in the trauma bay shows no fractures. Chest CT shows a posterior fracture of the first rib (**b**) and anterolateral torus fracture of the fourth rib (**c**)

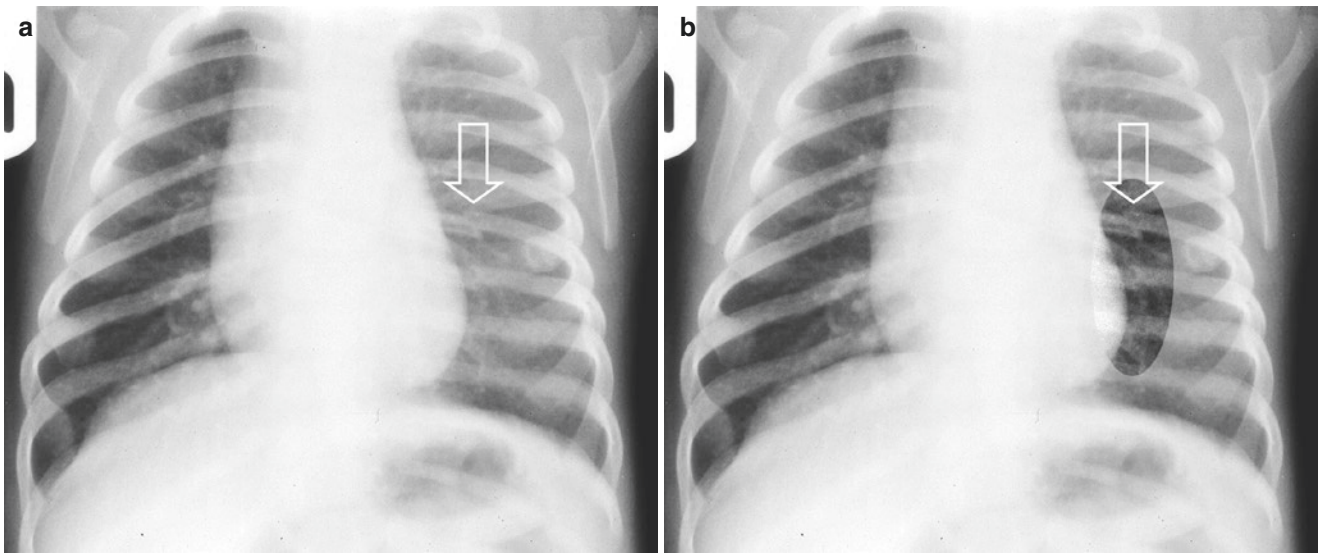


Fig. 7.17 (**a**) Fresh rib fractures on the left posterior side with slight dislocation of the fracture ends. (**b**) Using photo-enhancement software the fractures are made more visible

ries are accompanied by extensive other injuries, e.g. neurological injuries (Sect. 7.3.3.2) (Fig. 7.18a–j). Usually, this type of accident will have been observed by an independent outsider [15, 61].

- In children under the age of 3 years rib fractures usually occur in non-accidental trauma, except in children with a metabolic bone disease, in which a relatively minor trauma may result in rib fractures (Sect. 7.3.5.2) [61, 63, 75, 76].
- Children under the age of 3 years with non-accidental trauma usually have more rib fractures than children with accidental trauma [63].
- Intrathoracic injuries as a whole and individual types of intrathoracic injuries (pneumothorax, pleural effusion, pulmonary contusion, pulmonary laceration) are probably more common in children with accidental trauma [63].
- Lack of intrathoracic injuries in children with non-accidental rib fractures does not imply bone fragility [63].
- Simultaneous occurence of rib fractures and fractures of the extremities is probably more common in children with non-accidental trauma [63].
- Due to the malleability of the chest, one can exclude the possibility of rib fractures resulting from picking the child up in normal daily interactions and care. This, again, does probably not apply to children with a bone disease in whom fractures can occur with lesser loading than in children without a bone disease.
- In older children, adolescents, and adults, rib fractures are almost always sustained in accidental circumstances, such as motor vehicle accidents as pedestrian or crash occupant, or accidental household /playground trauma (e.g. falls from height or falls on an object) [51, 61]. Usually, this will involve a blunt force trauma, but a compressive force effect can also occur, e.g. in a situation where the paediatric or adult victim is run over.

Paine et al. performed a systematic review of the literature, published between 01 January 1990 and 30 June 2014, in which they included data for 1396 children 48 months or younger with rib fractures from ten publications [77]. Based on their review the authors concluded that '*The prevalence of suspected or confirmed abuse among children younger than 36 months presenting with rib fractures varied from 34% to 100% across the studies, reflecting heterogeneity of their study populations and methodologies. Excluding MVC and bone pathology as fracture etiologies produced abuse prevalence rates of 67–100% in children younger than 24 months and 91% in children younger than 12 months, demonstrating the high prevalence of abuse in young children with rib fractures*'. Table 7.7 presents an overview of the literature on accidental versus non-accidental rib fractures in children.

7.3.3.4 Non-accidental Rib Fractures

General Aspects of Non-accidental Rib Fractures

Rib fractures are probably the most common fracture resulting from non-accidental trauma (physical child abuse). Ninety percent of all non-accidental rib fractures are found in children under the age of 2 years [8, 54]. Of all fractures sustained in non-accidental trauma 5–27% are rib fractures [12, 78]. Rib fractures may be the only skeletal abnormality in about 30% of children who are radiologically evaluated because of a suspicion of non-accidental trauma (physically abused children) [79].

Rib fractures are probably even more prevalent in children with non-accidental trauma, but an unknown number

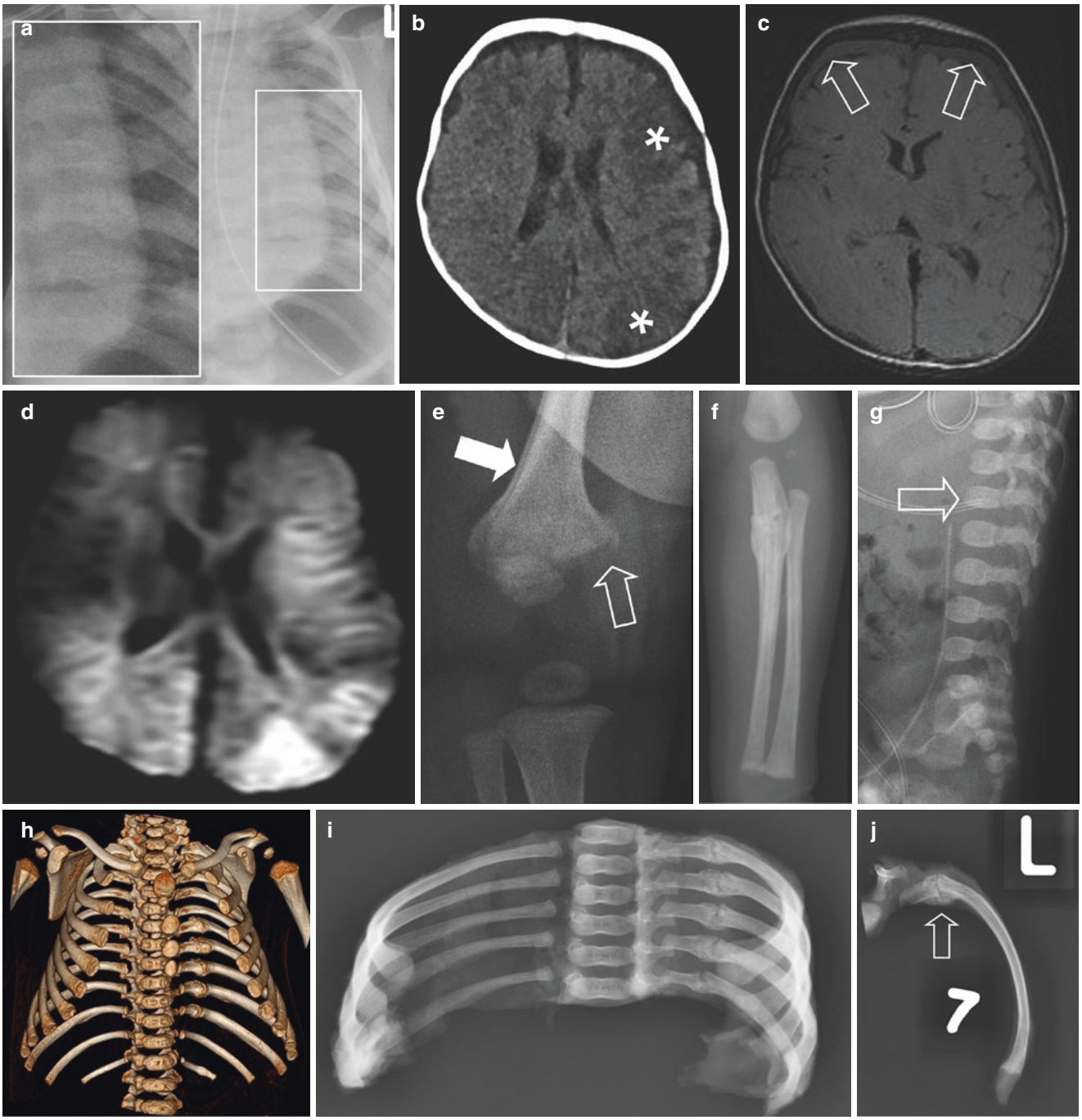


Fig. 7.18 Infant who was presented with a status epilepticus. (**a**) Chest radiograph shows healing posterior rib fractures of the seventh to tenth rib. (**b**) CT of the head at admission shows frontotemporal and occipital areas with decreased density (asterisk). (**c**) T1-weighted MRI shows bilateral subdural hematomas (arrow). (**d**) Diffusion-weighted MRI shows multiple bilateral areas of diffusion restriction in keeping with hypoxia. As a result of the severe brain trauma the child died and a legal autopsy was requested. As part of this process post-mortem radiology was performed. (**e**) Radiograph of the right knee shows a metaphyseal corner fracture of the distal femur (open arrow) and subperiosteal new bone formation (arrow). (**f**) Radiograph of the right lower arm shows a healing proximal ulnar fracture. (**g**) Radiograph of the spine shows a compression fracture of the first lumbar vertebral body. (**h**) 3D-CT of the chest shows healing posterior fractures of the sixth to tenth rib. (**i**) Specimen radiograph of a section of the chest. (**j**) Specimen of the seventh left rib shows a healing posterior rib fracture (arrow)

will not be diagnosed, since these fractures usually do not cause any complaints. In children that have died due to non-accidental trauma, one regularly finds fresh and healing or healed rib fractures. In autopsy cases, Kleinman et al. found radiographic evidence of 84 rib fractures (51%) in a total number of 164 fractures in 31 abused children [9].

Despite the fact that the statistical analysis of Barsness et al. concerning the positive predictive values (PPV) of non-

Table 7.7 Rib fractures accidental versus non-accidental overview of relevant literature

Author, year	Study period	Ages (months)	Total study population (Accidental/non-accidental)	N cases with rib fractures	Cases with accidental rib fractures (%)	Cases with non-accidental rib fractures (%)
Schweich, 1985 [72]	1980–1985	<216[a]	Selection based on presence of rib fracture	21	16 (76.2)	5 (23.8)
Bulloch, 2000 [46]	1994–1996	<12	Selection based on presence of rib fracture	39	7 (18)	32 (82.0)
Cadzow, 2000 [15]	1994–1998	<24	Selection based on presence of rib fracture	18	3 (16)	15 (83.3)
Barsness, 2003 [80]	6 year period	< 36	NR[b]	62	9 (17.7)	51 (82.3)
Pandya, 2009 [194]	1998–2007	≤48	1485 (985/500)	105	11 (10.5)	94 (89.5)
Ruest, 2021 [69]	2011–2016	< 24	264 (226/38)	12	2 (16.7)	10 (83.3)

[a] No specific data on young children
[b] Part of a larger study population of 3758 trauma evaluations in children

accidental rib fractures in young children is flawed, as it was calculated based on the number of rib fractures instead of the number of cases with rib fractures, the research nevertheless showed some interesting results [80, 81]. Barsness et al. identified in a 6-year period 62 children under the age of 3 years with a total of 316 rib fractures. They established that in 51 children (82%) the fractures were the result of non-accidental trauma (physical violence). Furthermore, their study showed that:

- In non-accidental trauma, multiple fractures were more prevalent than single fractures (Fig. 7.19a, b).
- Non-accidental trauma was likely when the fractures were located posterior and lateral (in 78% of children).
- Rib fractures (single or multiple) were the only skeletal signs of non-accidental trauma in 29% of their study population.

These data corresponded with the data found by Cadzow and Armstrong [15]. They also found that rib fractures are often found in children who also sustained, due to non-accidental trauma, fractures of (one of) the extremities or intracranial pathology (Figs. 7.20a–c and 7.21a–f).

Non-accidental circumstances should always be considered when [38, 75, 82]:

- Rib fractures are found outside of the perinatal period, although rib fractures have been reported sporadically in complicated deliveries (Sect. 7.3.2).
- There are no indications for bone disease.
- There is no adequate explanation for a trauma that caused the injuries, or when parents/carers provide no explanation at all.
- Multiple bilateral fractures are found particularly in the lower ribs on the posterior and lateral sides (combinations of fractures on the posterior and anterior sides can occur).
- Multiple fractures are found, and based on the healing process it can be established that the fractures differ in age (Figs. 7.22a, b and 7.23a, b).

Posterior rib fractures seem to be most prevalent between the fourth and ninth rib. Lateral rib fractures are most prevalent in the lower part of the chest. Damage to the anterior costochondral junction is usually found between the second and ninth rib [83].

Fractures can occur in different locations of one rib. When this happens to several ribs it can lead to a flail chest. Hereby the chest wall of the child moves in the opposite direction when breathing: at inspiration the chest wall moves inwards and at expiration outwards. At physical examination it will be found that the chest is no longer firm and feels less malleable. Gipson and Tobias report a 21-day-old infant with a flail chest resulting from non-accidental trauma [84]. They state that child abuse is the most likely explanation for a flail chest in infants when there are no clear indications of serious chest trauma or a metabolic disorders.

The reliability of the detection of rib fractures depends on the technique used. It seems justified to use detailed radiographs to establish these injuries in living and deceased children (Fig. 7.24a–c) [9].

Paine et al. did a systematic review to estimate the prevalence of non-accidental trauma in young children presenting with rib fractures and to identify specific characteristics that affect the probability that rib fractures are secondary to non-accidental trauma [77]. A total of 1396 children under the age of 48 months from 10 articles were included. The prevalence of rib fractures in children under the age of 12 months, due to non-accidental trauma, ranged from 67% to 82%. In children aged 12–23 months the prevalence was 29% and in children aged 24–35 months 28%. The location of a rib fracture was not associated with the likelihood of abuse.

Kriss et al. retrospectively reviewed the radiographs of 78 infants (aged 0–18 months) with rib fractures in whom non-

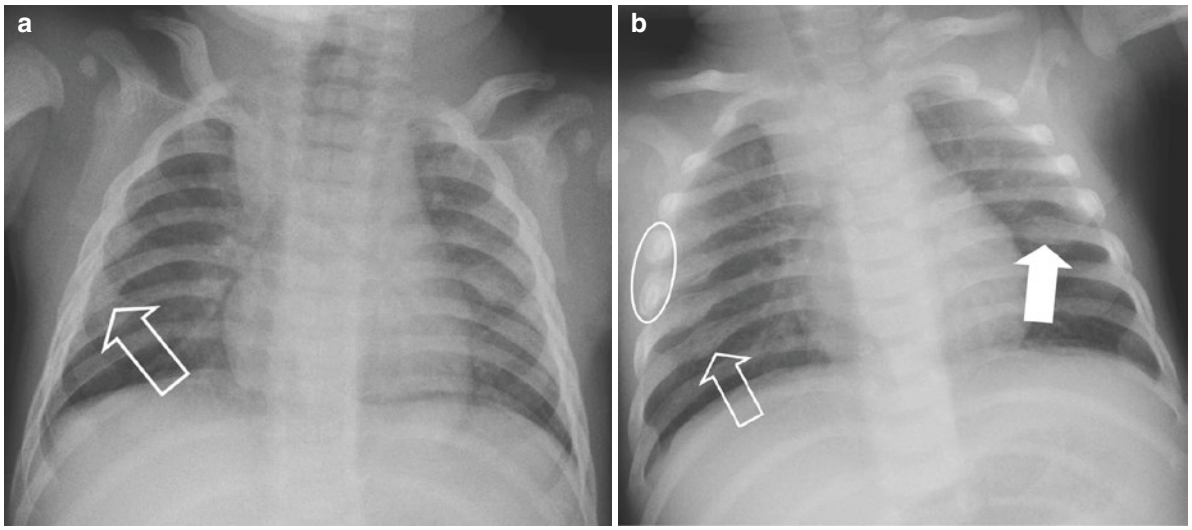


Fig. 7.19 Infant with multiple unexplained bruises. (**a**) Chest radiograph shows a healing posterolateral rib fracture of the fifth and sixth right rib (arrow). The skeletal survey also showed a fracture of the lower arm and metaphyseal corner fractures of the tibia. (**b**) Radiograph after 10 days shows a posterior rib fracture of the sixth left rib, which was not initially described in the referring hospital. On the right side multiple posterolateral rib fractures (open arrow) and two healing lateral rib fractures (oval)

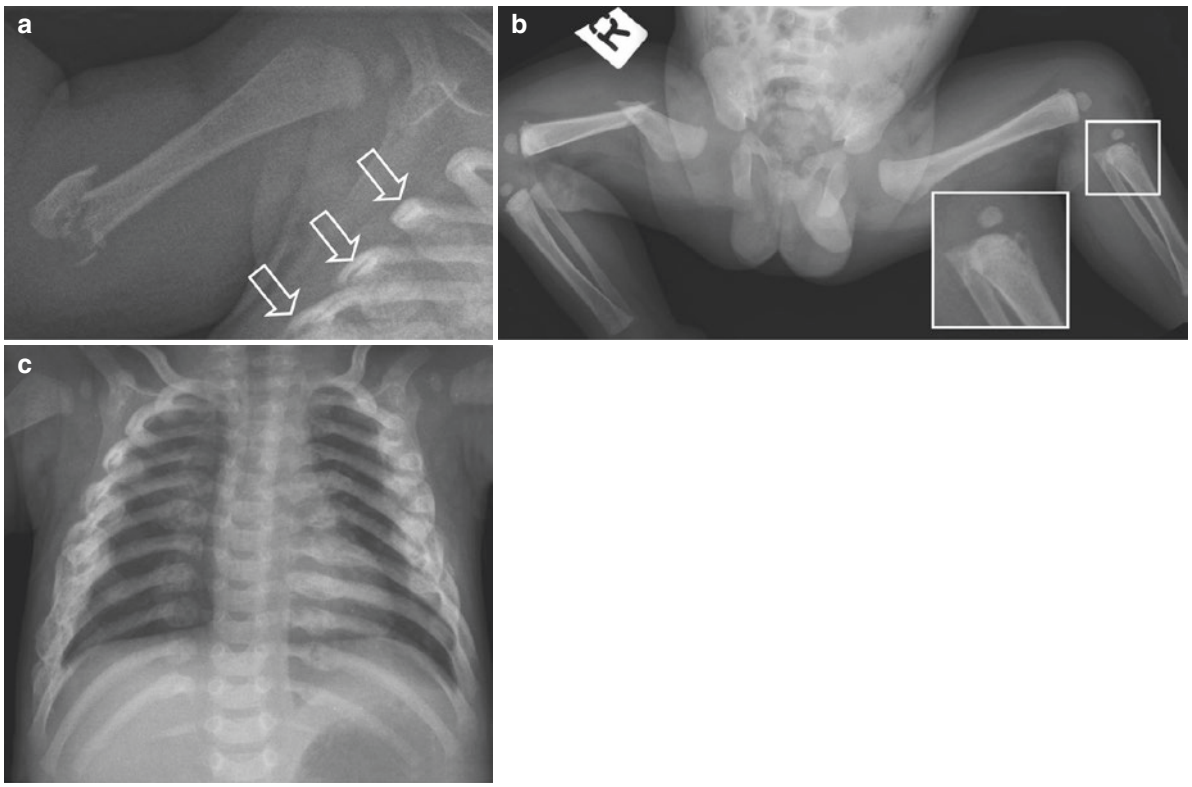


Fig. 7.20 (**a**) Right distal metaphyseal humerus fracture in an infant. The healing mid-axillar rib fractures are clearly visible on this view (open arrows), but were missed when reporting the humerus fracture. At the age of 3–4 weeks he had already been seen for a fracture of the left humerus. The physicians deemed the parent's statement that the child had clumsily been picked up plausible. (**b**) Six days after the visit for the distal metaphyseal humerus fracture, the child presented again at the emergency department, this time for a suspected femur fracture on the right. The radiograph shows a transverse mid-shaft femur fracture and a metaphyseal corner fracture of the proximal part of the left (see inset). These findings resulted in a full skeletal survey. (**c**) The chest radiograph showed more than 30 rib fractures originating from different points in time and showing different stages of healing

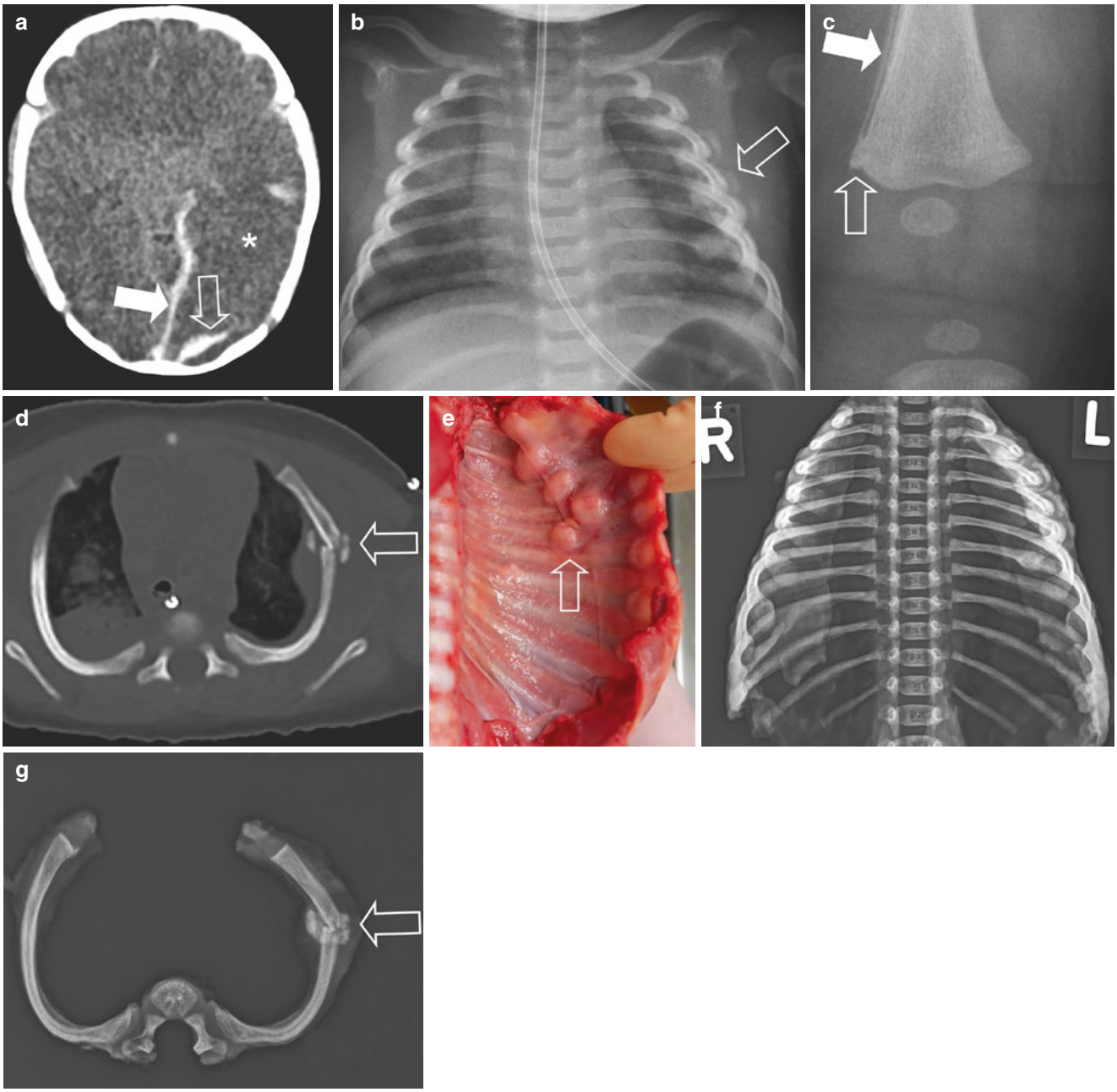


Fig. 7.21 Neonate who, according to the clinical history, became unwell while being bottle fed. At presentation a comatose neonate was seen. (**a**) CT showed a subdural hematoma along the falx and tentorium (arrow), a haemorrhagic contusion (open arrow), and diffuse oedema (asterisk). (**b**) Chest radiograph showed multiple healing lateral rib fractures on the left side (arrow). (**c**) A healing metaphyseal corner fracture of the distal right femur (open arrow) with subperiosteal new bone formation (arrow). (**d**) Chest CT showed clear callus formation around a lateral fracture of the third rib (arrow). The neonate died as a result of the sustained trauma and a legal autopsy was performed. (**e**) Photo of the left inside of the chest, after removal of the heart and lungs, shows multiple nodular changes due to callus formation (arrow). (**f**) Specimen radiograph of the thorax. (**g**) Specimen radiograph of the third rib showing a healing ri**b** fracture (arrow)

accidental trauma was confirmed [85]. They found a total of 360 rib fractures in 273 individual ribs involving 78 abused children. Sixty-three children (81%) had multiple rib fractures. They also found a significantly greater number of left-sided rib fractures (67%) than right-sided fractures. Fractures were most often identified in the posterior and lateral regions and mid-level of the ribcage (ribs 5 through 8). Fifty-four percent of subjects had other skeletal fractures; these non-rib fractures were also predominantly on the left side. In their conclusion they stated that *'further research is needed to understand whether factors such as perpetrator handedness are associated with these unequal distributions of fractures in abused children'*.

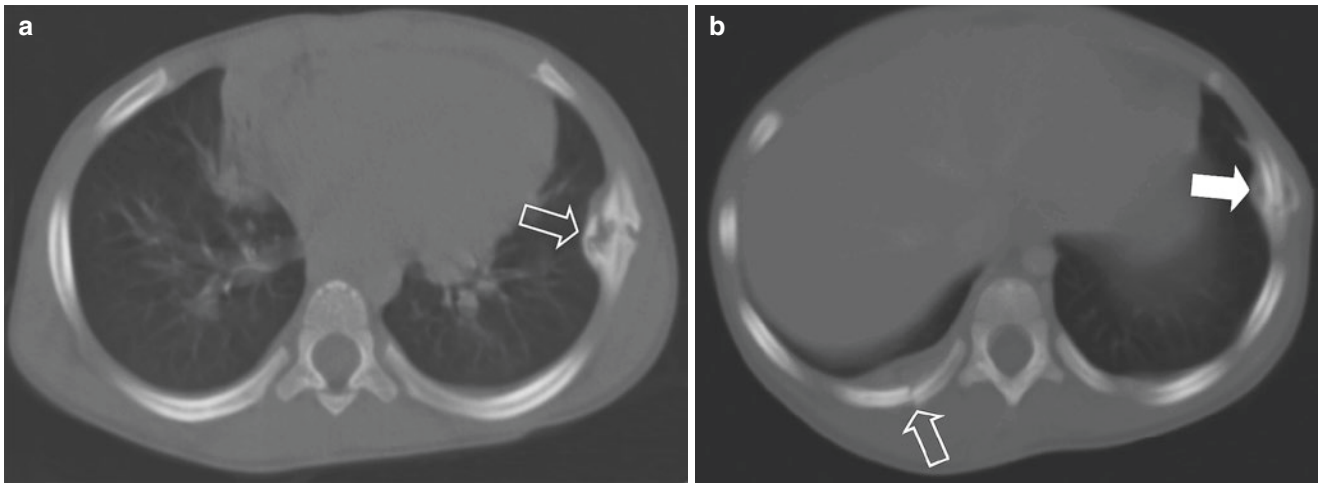


Fig. 7.22 (**a**) Child who, according to the clinical history, had allegedly fallen from a bunk bed. A chest CT (made at presentation in the trauma unit) shows a healing left lateral rib fracture. (**b**) Chest CT, a few slices lower than (**a**), shows a fresh right posterior rib fracture (open arrow). On the left lateral side a healing rib fracture can be seen (arrow)

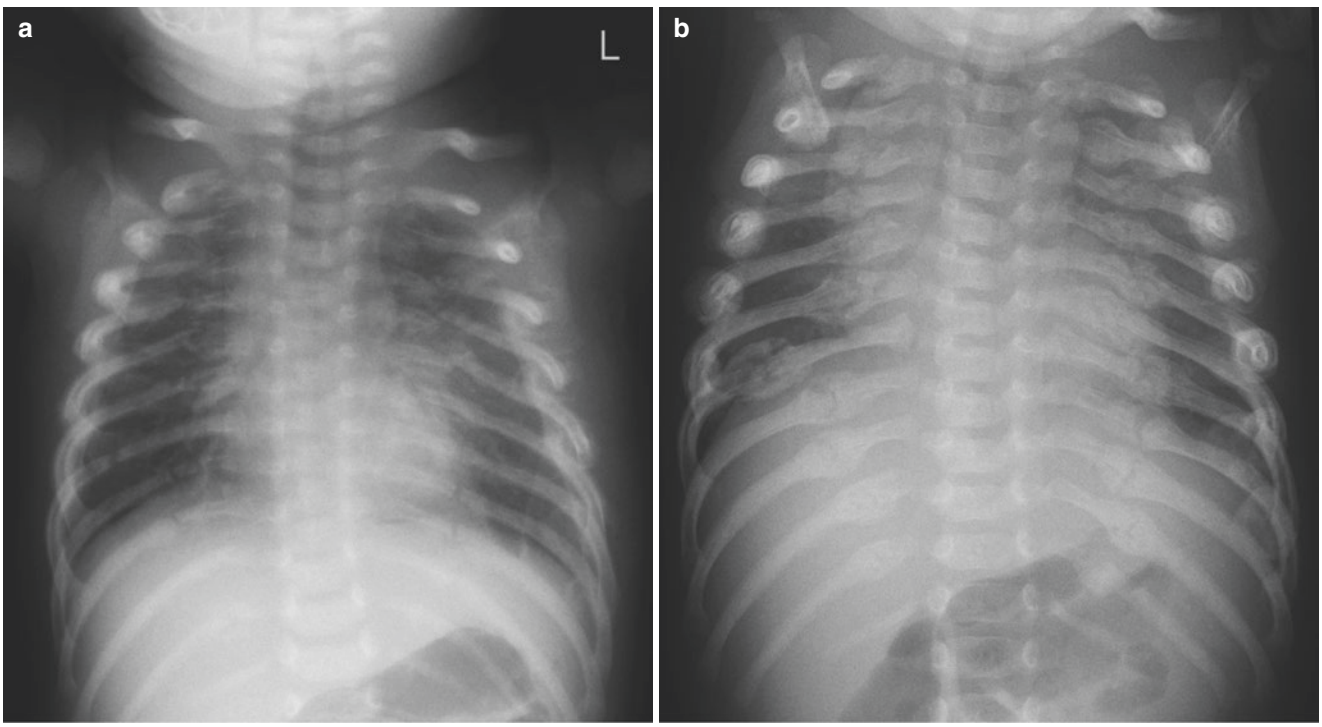


Fig. 7.23 Infant with multiple bruises. (**a**) Chest radiograph shows multiple healing lateral and fresh posterior rib fractures. (**b**) Repeat chest radiograph shows multiple healing rib fractures in nearly all ribs

Mitchell et al. did a systematic review to determine which children presenting with rib or long bone fractures should undergo a routine evaluation, concerning non-accidental trauma, based on age [86]. Fifteen articles were suitable for final analysis. In 77% of the children under the age of 3 years presenting with rib fractures, the rib fractures were due to non-accidental trauma. If in children under the age of 3 years motor vehicle accidents were excluded, non-accidental trauma was present in 96%. The authors strongly recommend routine evaluation for non-accidental trauma, including forensic paediatric consultation, for children with rib fractures under the age of 3 years.

Posterior Rib Fractures

Initially, it was thought that the vast majority of rib fractures, due to non-accidental trauma, were posterior fractures. This

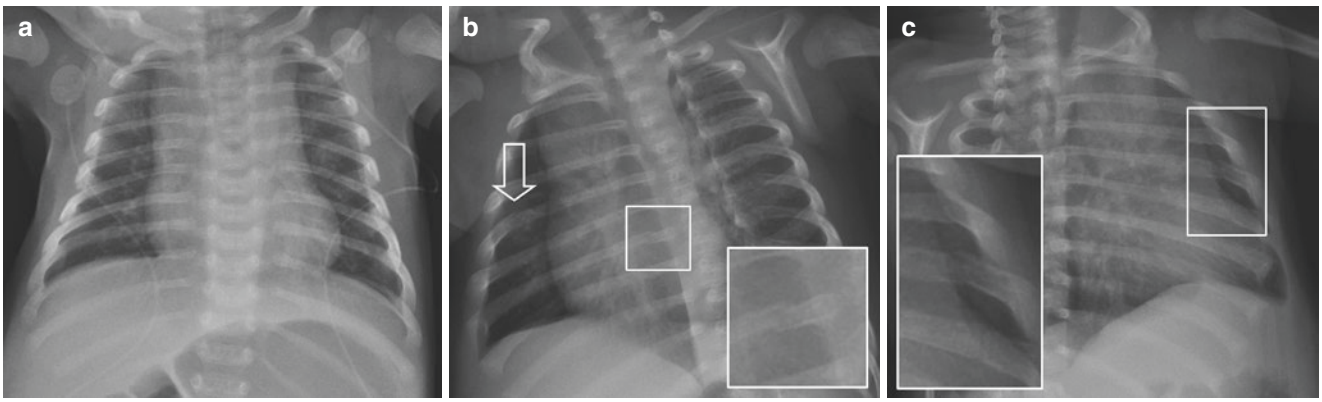


Fig. 7.24 Example of the value of right and left oblique radiographs. (**a**) AP chest radiograph shows a slight bilateral pleural thickening without clearly identifiable rib fractures. (**b**) Left oblique radiograph shows a lateral fifth rib fracture (arrow) and a posterior seventh rib fracture (inset) on the right side. (**c**) Right oblique radiograph shows lateral rib fractures of the fourth to sixth ribs on the left side (inset)

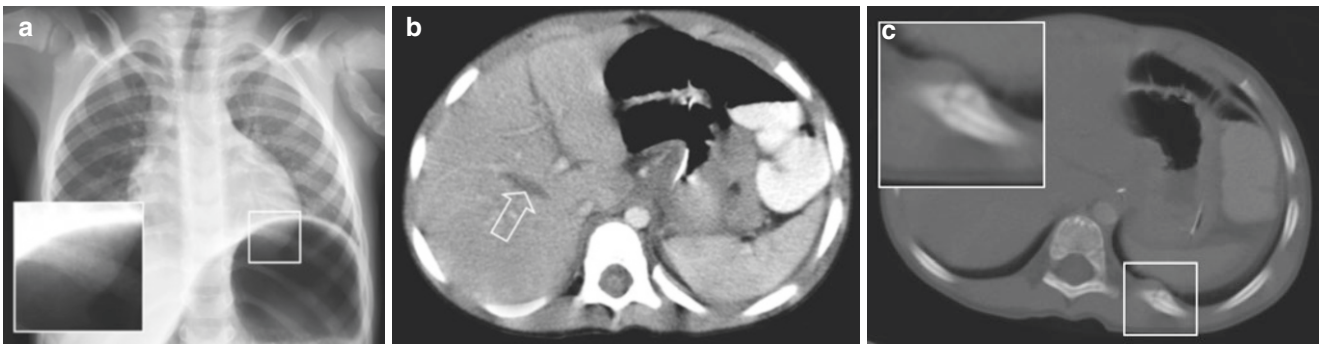


Fig. 7.25 (**a**) Posterior rib fracture with callus formation as an accidental find (see inset) in a child who had been admitted to hospital for severe abdominal pain. The stepfather persisted in his explanation that the boy had fallen when playing with the dog in the garden. (**b**) Abdominal CT scan showed a liver laceration (open arrow). (**c**) Abdominal CT scan confirms the presence of a posterior rib fracture with callus formation (see inset). At further investigation, it was found that both injuries, separated by a time interval, were the result of child abuse

is not correct, as was shown by Paine et al., they found that the location of a rib fracture was not associated with the likelihood of abuse [77]. Nevertheless, posterior rib fractures in young children are highly suggestive of non-accidental trauma, as described in the earlier sections.

Often, posterior fractures will not be visible until there is callus formation (Fig. 7.25a–c) [12]. Since in the acute phase the fracture can easily be overlooked, it is recommended to make a chest radiograph when non-accidental chest compression is suspected, at the first exanimation as well as at 2-week follow-up [13, 79, 87]. In case of enduring doubt regarding the presence of rib fractures in the acute phase despite oblique views and CT is inconclusive, one may consider performing bone scintigraphy (Chap. 3) [88].

Fractures of the First Rib

Fractures of the first rib are only very rarely reported in infants and toddlers (Fig. 7.26). Strouse and Owings evaluated 35 infants under the age of 2 years with rib fractures [89]. In 12 children the fractures were determined to be due to non-accidental trauma. Only in four children a fracture of the first rib was found: one neonate with congenital osteogenesis imperfecta and 3 infants with non-accidental trauma (in 1 infant bilateral). The authors identified two more children with first rib fracture from years prior to the study period. In four children, first-rib fractures were ‘isolated’, without fractures of adjacent bones. Strouse and Owings concluded that non-accidental trauma should be considered in infants with fractures of the first rib (uni- or bilateral), since it takes a severe trauma to fracture these ribs. Hereby one should think along the line of direct blunt force trauma (impact), compression, shaking, or acute axial loading (slamming). In a letter to the editor, in response to Strouse and Owings, Oestreich mentioned that he had seen a child with a first rib fracture, due to non-accidental trauma, only once [90]. He also described the radiological findings in a 7-month-old infant. Initially, it was thought that the child had a fracture of the left first rib, due to a non-accidental trauma.

In fact, the finding was a cervical rib synostosing with a process off the subjacent rib.

Melville et al. described 3 infants with 4 fractures of the first rib [91]. All fractures were due to non-accidental trauma. The authors did a literature search, but could not find one case of a fracture of the first rib in a healthy infant that was not the result of non-accidental circumstances. Just like Strouse and Owings, the authors state that a thorough medical and social evaluation should be done if a fracture of the first rib is found in an infant.

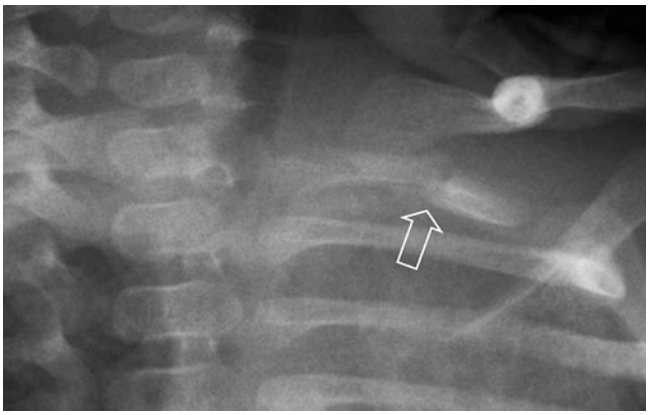


Fig. 7.26 An infant was evaluated for non-accidental injury because of haematomas on his back which were caused by forcefully holding/squeezing by his father during periods of persistent crying. The arrow indicates a healing fracture of the left first rib. Moreover, several bilateral old fractures at the costovertebral junction of other ribs were present (not shown)

In older children fractures of the first rib also are also uncommon [92]. According to Hamilton et al., the finding of a fracture of the first rib suggests a trauma with a high transfer of energy and resultant multisystem injuries. They described 33 children (0.27% of all paediatric trauma patients; mean age 10.9 ± 0.9 years) with either a first rib fracture or thoracic vascular injury owing to blunt trauma. 32 children had a first rib fracture. Most were due to motor vehicle accidents, followed by pedestrian versus motor vehicle, farming accidents, bicycle accidents and all-terrain vehicle accidents. In 2 children the circumstances were not described. Associated injuries concerned head injury and fractures of skull, facial bones, pelvis, lower extremity, spine and scapula. Also, injuries to intra-abdominal organs were found. Three children died.

In adolescents fractures of the first rib are mainly seen in motor vehicle accidents, falls and sporting activities, due to trauma with various severities [93]. A fracture of the first rib however may also occur due to a more or less trivial trauma. Lee et al. described a 13-years-old boy with a fracture of the left first rib, due to *'morning stretching with a yawn without sports activity'* [94]. According to Lee et al. non-accidental trauma was excluded.

Injuries to the Costochondral Junction

Injuries to the costochondral junction have been described to occur due to front-to-back compression, when the sternum and the costochondral junctions are pushed inwards, which may result in fractures at the costochondral junction (Fig. 7.27a, b) (Sect. 7.2.1) [5, 9]. This may occur during

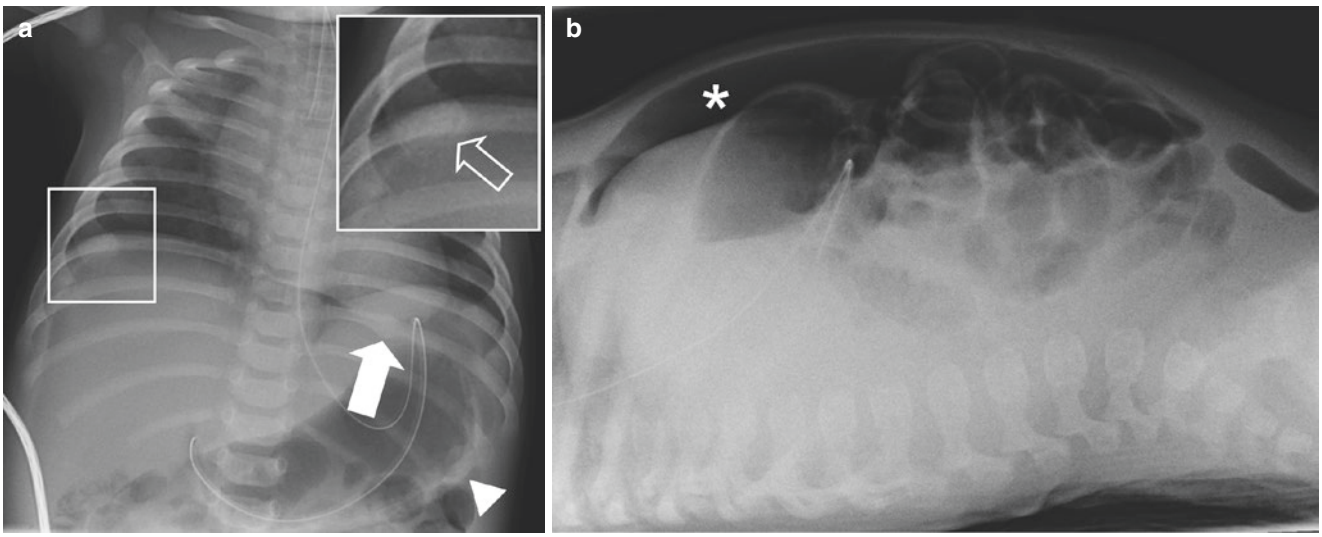


Fig. 7.27 According to the clinical history the father had tripped over the cat while holding the infant on the day before hospitalization. At admittance, the infant presented with tachycardia and decreased saturation (85%). (**a**) The chest radiograph at admittance showed anterior rib fractures on the right (see inset, open arrow), a healed posterior rib fracture on the left (arrow), and pneumoperitoneum, characterized by Rigler's sign (arrowhead) and 'continuous diaphragm' sign. (**b**) Cross table abdominal radiograph shows again the pneumoperitoneum (asterisk)

cardiopulmonary resuscitation (CPR) (Sect. 7.3.4.1.), although very rarely. In a systematic review Maguire et al. only found one child in whom a fracture at the costochondral junction could be explained by CPR [95].

In the literature only a limited number of articles is found in which the authors draw attention to injuries to the costochondral junction, due to non-accidental trauma (Fig. 7.28a, b).

Smeets et al. were the first to report a non-accidental costochondral dislocation of the lower ribs in a physically abused child, who also had fractures of skull, ribs, and long bones. An ultrasound showed the costochondral dislocation [96].

Ng and Hall describe three children under the age of 3 years (two boys, one girl; 7, 18, and 36 months of age), with fractures at the costochondral junctions of the sixth to ninth ribs [97]. According to Ng and Hall around 4% of rib fractures, due to non-accidental trauma, are located at the costochondral junction. The fractures were bilateral in two children and symmetrical in one. The fractures resembled 'bucket-handle' metaphyseal fractures, as can be found in the long bones and it was hard to visualize these fractures. They also stated that these fractures heal with minimal callus formation. The authors also stated that rib fractures at this location are often associated with major intra-abdominal blunt force trauma (in these cases severe physical abuse), such as rupture of the duodenum and spleen, ileal serosal tears, portal vein tear or transection of the pancreas.

Kleinman et al. evaluated the characteristics of rib fractures in 11 infants, who died due to non-accidental trauma [9]. These 11 infants had a total of 84 rib fractures. In 4 of the 11 children a total of 10 healing fractures at the costochondral junction were found. No acute fractures at this location were found. According to Kleinman et al., the fractures tended to involve the inner aspect of the costochondral interface with an associated osseous fragment.

Nimkin and Kleinman, just like Ng and Hall, stated that fractures at or around the costochondral junction are difficult to visualize and may occur more commonly than reported in the literature [98].

Weber et al. analyzed the post-mortem findings in 546 children, aged 7 to 365 days, presented as SUDI (Sudden Unexplained Death in Infancy), over a 10-year period (1996–2005) (see also Sect. 7.3.4.1) [99]. This included 94 forensic autopsies. Rib fractures were found in 24 infants (4%). In 4 children the fractures (mostly multiple) were located at the costochondral junction, in 2 children bilaterally located, and in 2 children only right sided. In 3 of these 4 children death was due to inflicted head injury and in 1 child due to inflicted asphyxia. No fractures at the costochondral junction were seen in apparent CPR-related cases.

Rib Fractures as Indication for Respiratory Obstruction by Chest Compression

Only a few case reports have been published concerning the risk of respiratory obstruction due to anterior-posterior compression of the chest in young children (also described as 'constrictive asphyxia') [100–103]. This form of static loading may impede respiration as well as oxygen uptake by mechanically closing the airways. The lack of oxygen will make the child disoriented. The face and neck will become cyanotic with petechiae and bruises on head, neck, and chest. There may also be subconjunctival and retinal haemorrhage. Other clinical signs may include tachypnoea, vomiting blood, and respiratory failure. However, these physical find-

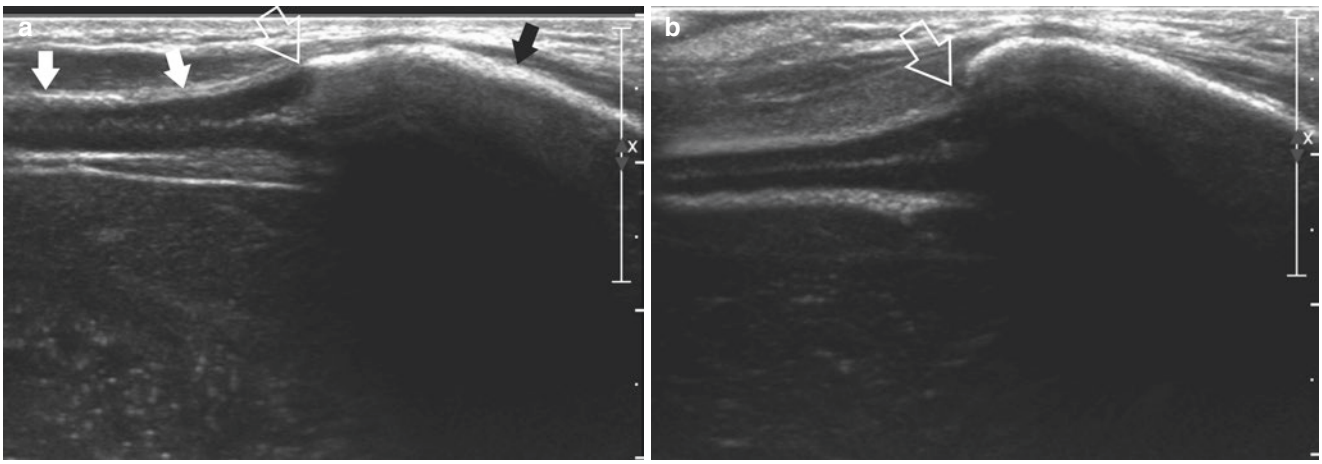


Fig. 7.28 Ultrasonography of costochondral dislocation in an abused infant. (**a**) Normal anatomy at the junction of the ventral osseous (black arrow) and cartilaginous rib (white arrows). The junction is indicated by the open arrow and shows a smooth transition from the bony rib to the cartilaginous rib. (**b**) Dislocation at the costochondral junction (open arrow): the cartilaginous part of the rib is depressed compared with the osseous part of the rib. The child also had bony rib fractures of different ages, metaphyseal avulsion fractures, and a skull fracture

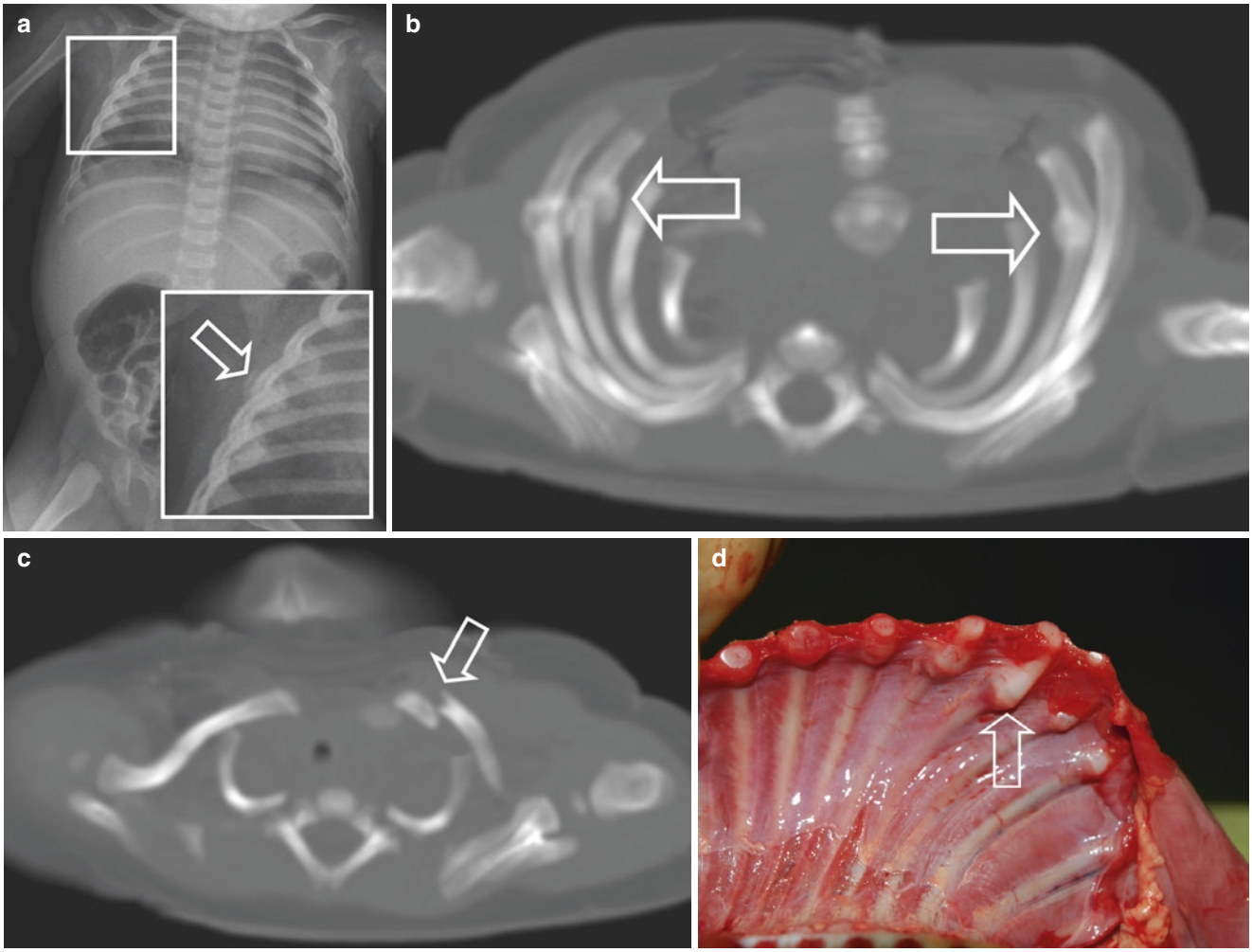


Fig. 7.29 (**a**) Post-mortem radiograph of an infant found dead in her crib. In compliance with the Dutch SIDS protocol a skeletal survey was done. The chest radiograph showed bilaterally localized lateral rib fractures which were, when considering the callus formation, not recently sustained (see inset, open arrow). (**b**) Bilateral-localized lateral rib fractures of an older date, visible on the total-body CT scan (MIP images, open arrow). (**c**) Left clavicle fracture (open arrow). This fracture was only visible on the CT scan and not on the radiograph, not even in retrospect. (**d**) Photograph at autopsy shows callus formation on the anterior side of the left chest wall (open arrow). On autopsy no brain pathology was found. Based on the clinical and forensic evaluation static chest compression leading to asphyxia was considered to be the cause of death

ings are not always present. The authors of the case reports, especially draw attention to the occurrence of rib fractures in this kind of static loading, and the potentially fatal course of this type of child abuse. They also state that rib fractures most probably are the most reliable indicator for the cause of this kind of asphyxia (Fig. 7.29a–d).

Boos described a situation in which the father confessed that he had pushed several times on the chest of his infant to make it stop crying [100]. He also pushed the legs of the infant against its chest. During the last incident at the age of 7 months, he wrapped the infant firmly into a sheet because of uncontrollable crying, after which he put the infant to bed. After a couple of hours the father found the infant dead (apnoeic and pulseless). The child had 14 old and new rib fractures (healing rib fractures of differing ages, anterolateral from the fourth to tenth rib on the left and fourth to seventh rib on the right; and new fractures of the eighth to tenth rib). The infant also had face and scalp haematomas. Death most probably was due to respiratory obstruction. In the discussion section Boos reported a second case (unknown sex, unknown age), with multiple rib fractures found during the autopsy. Initially no cause of death was determined. The father stated that he would hold the child tightly against his chest in order to stop his son's crying. One day after a squeezing episode the child was found dead in the morning. The father pleaded guilty to the murder of his child.

Gunther et al. presented the findings in 3 infants, aged 7 months (twin brothers), and 13 months, with multiple healing/healed and unhealed anterior and lateral rib fractures (including several refractures) due to inflicted anteroposterior manual chest compression and accompanied by haemorrhage into the viscera, and rupture of large vessels and solid organs [101]. One of the twin brothers died of abdominal trauma, with mesenteric scarring found during the autopsy.

Haemosiderin macrophages were present in his lungs. The boy had multiple new and healing rib anterior and lateral fractures, however, no posterior fractures or fractures at the costochondral junction were found. He also had fingertip bruising, bilaterally on the front of the thorax. The second twin brother survived, but was found to have multiple lateral rib fractures. There was no confession in these two cases. The third infant had new lateral rib fractures, located at the inner cortex and limited to the inner curvature. This infant also died of abdominal trauma. The infant had similar fingertip bruising on the front of the thorax as the first infant. In this case the perpetrator confessed to squeezing the infant.

Lauridson and Cromblin described the findings in a 4-month-old boy whose mother sought medical care because of poor feeding and low-grade temperature [102]. The infant had no external evidence of abuse, but chest radiographs showed multiple rib fractures (left fourth, fifth, sixth, and seventh ribs in the posterolateral region). There were no indications for abusive head trauma (no intracranial or retinal haemorrhages). No other fractures were found. The father admitted to the police, that he had, on multiple occasions, squeezed the infant's chest, as hard as he could (*'My arms would get a little achy'*). The father also stated that the infant turned blue. The father's squeezing behaviour resulted in asphyxia, due to restriction of respiration, resulting in unconsciousness, and in rib fractures.

Vester et al. presented two girls, 3 weeks and 2 months of age, who died under suspicious circumstances [103]. In both girls, non-accidental constrictive asphyxia, inflicted by their fathers, was established after extensive clinical, forensic medical, and legal investigations. In the first case, the father admitted that he sometimes sat on his infant while she was lying on the bed, and occasionally used to tighten his grip by pulling with his hands on the bottom of the bed, thus increasing the compressive forces. The girl had been in the hospital shortly at the age of 2 weeks because of dehydration after feeding difficulties. During that stay small bruises on her chest were seen, which were allegedly caused by rough cuddling by her older sister. Also, a bruise on her leg was seen, reportedly caused by her father. For reasons unknown, non-accidental trauma was not considered at that time. At the age of 3 weeks, the girl was readmitted to the hospital because of seizures. Upon arrival she was pale, drowsy, and hypothermic with a recorded temperature of 35.8 C, absent left-sided direct light reflex, and had bruises behind the left ear and on her right knee. She was subsequently admitted to the neonatal intensive care unit. A skeletal survey showed 'small hooks' on the right distal femoral metaphysis and 14 recent and older rib fractures. After initially denying a non-accidental trauma, the father confessed to the police, to periodically sitting on his daughter while she was lying on the bed in order to stop her from crying. The father reportedly sat on her the day prior to the final hospitalization, after which the baby was less responsive and had developed new chest bruises. He also confessed to shaking her in this period. In court, he was found guilty of multiple attempts of manslaughter.

The second girl died unexpectedly at the age of 2 months. The parents reported that on the day of her demise, her father checked in on her while she was lying in her bed crying. Approximately an hour later, her mother found her in her bed; pale, motionless, and without noticeable respiration. On the way to the hospital, her mother started resuscitation, which was continued by medical personnel in the emergency department. However, resuscitation efforts were unsuccessful and a judicial autopsy was requested. Post-mortem chest radiographs showed lateral rib fractures on both sides, all with callus formation. No other fractures were visible on the post-mortem skeletal survey. Post-mortem CT scans also revealed a fracture of the medial part of the left clavicle. On autopsy, a 3-mm bruise was seen on the chest, and a 10-mm bruise in the mouth. All costochondral junctions were thickened, and lateral rib fractures with callus formation of the right second to fifth ribs were found, along with a left third rib fracture which showed a fresh refracture. Furthermore, there was a fresh fracture of the left clavicle. Neuropathology indicated asphyxia without other neuropathological findings. Autopsy of the lungs showed signs of food aspiration. No other abnormalities were found. Although no definite cause of death was found, the pathologist ruled that the combination of rib fractures and food aspiration was indicative of asphyxia due to airway obstruction. After initially denying a non-accidental trauma, the father later admitted that in order to stop his daughter from crying, he would wrap her tightly in a blanket and hold her tightly against his chest, while encircling her with both arms until she would stop crying. On the evening of the fatal incident he squeezed her more tightly than normal, after which she went quiet and he put her back to bed. At that time he thought that she was still breathing, although she was very pale and had her eyes closed. In court the father was found guilty of abuse.

7.3.4 Trauma After Birth: Medical Procedures

7.3.4.1 Resuscitation and Rib Fractures

General Aspects of Resuscitation

> *External cardiopulmonary resuscitation is a potentially lifesaving intervention aimed at preserving the cerebral function of a person in cardiac arrest. However, certain injuries can be caused by the various techniques employed. Although these are seldom consequential, they may complicate the forensic evaluation of cases* [104].

Cardiopulmonary resuscitation (CPR) in itself can be physically traumatic due to the manual compression of the chest.

Also, orofacial injuries may occur due to the ventilation technique, whether done by mouth-to-mouth, by airbag-valve mask, by intubation, or by a combination of these methods.

If a person is accused of inflicting severe non-fatal, or fatal injuries to a child, that had to be resuscitated, he/she may claim that the injuries were caused by the attempts to resuscitate, either by him/herself, or by somebody else, e.g. medically trained personnel. These, according to the accused, CPR-related injuries may concern thoracic and abdominal structures and organs, e.g. rib fractures, visceral haemorrhages/intra-abdominal bleeding, rupture of large vessels and damage to solid organs [101, 105–107]. It may also concern superficial structures, like bruises or abrasions of the orofacial skin [108].

A statement about attempts to resuscitate should be carefully evaluated. Firstly, the evaluation should deal with the performed techniques and the skills of the persons, who performed the CPR. Secondly, the evaluation should be directed towards the probability of the injuries of the child, when compared to the used CPR technique.

If a child has been resuscitated and there are unexplained or suspicious findings in the head-neck region or elsewhere on the body (especially on the torso), one should always carefully ask the persons involved, who performed the resuscitation (medically trained or untrained person, including the parents), what was done (which methods was used) at which moment, where (at home, during transportation, in the emergency room, in the hospital), why and for how long. One should also ask all participants whether they noticed any injuries before the resuscitation was started and whether they were aware that they had induced injuries.

In case the child died, all equipment used during the resuscitation (e.g. airway and nasogastric tubes, intravenous lines, bone needles, cardiac resuscitation pads, and wound dressings) must be left in place to be assessed during the post-mortem examination. Also, any discarded items, including the mask, should accompany the body, so that the use of these items can be evaluated [108]. Both the comprehensive history of what happened during the resuscitation and the presence of the used devices may be helpful in differentiating resuscitation injuries from injuries, due to non-accidental trauma.

All injuries should be registered, including minor and therapeutically unimportant injuries, because they may have forensic significance. Preferably, pictures of all the injuries should be made as soon as possible after the death of the child, preferably by a forensic photographer.

If rib fractures are found in an autopsy and it is suspected that death of the child was due to non-accidental trauma, histological dating of the rib fractures (and other injuries) is advised to differentiate non-accidental rib fractures from CPR-related fractures, irrespective of a claim by the accused that the injuries (fatal and non-fatal), found during autopsy, were caused by attempts to resuscitate the child [101].

Two-Finger Cardiopulmonary Resuscitation and Two-Thumb-Encircling Hands Chest Compression

The traditional way of resuscitating neonates and infants is the ‘two-finger’ cardiac massage (‘two-finger’ infant cardiopulmonary resuscitation) (Fig. 7.30 A). Hereby anterior-posterior compression is applied, with the child supine on a flat solid surface, and the sternum is pressed towards the spine during cardiac massage. Pressure is exerted exclusively on

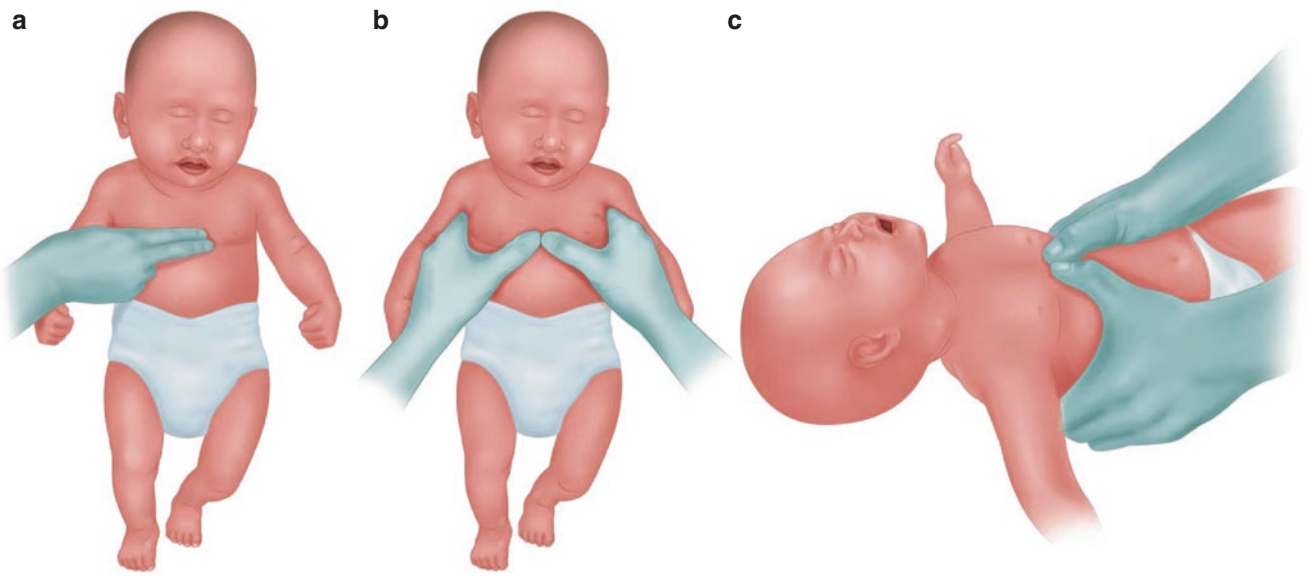


Fig. 7.30 (**a**) Two-fingers cardiopulmonary resuscitation (CPR) technique; (**b**) Two-thumbs encircling hands chest compression CPR technique, and (**c**) side view of two-thumbs encircling CPR technique

the front of the chest. In relation to the ribs and the costochondral junction, the sternum is moved inwards. At the same time, the spine and the ribs are more or less stationary on the flat solid surface. According to Chapman, this makes it impossible for leverage of the ribs on the vertebral processes to take place [16]. Even if the infant lies on a soft surface, such as a bed, it is unlikely that there will be posterior rib fractures, since the chest as a whole and the ribs and spine on the posterior side are pressed simultaneously into the soft surface below. Moreover, according to Worm and Jones, the forces exerted in two-finger cardiac massage are below the level of force required to cause rib fractures [5].

In the international guidelines of 2000 for the resuscitation of neonates and infants and in the revised version of these guidelines of 2006, the 'two-thumb-encircling hands chest compression' is considered to be an effective form of cardiac massage (Fig. 7.30 B & C) [109, 110]. In this manner of resuscitation, anterior-posterior compression is exerted while the ribcage of the infant is encircled by both hands (thumbs on the sternum and fingers on the back) and sternum and spine are compressed towards each other (bimanual anterior-posterior compressions) (Fig. 7.31). According to some authors, this way of encircling the chest is similar to the supposed manner in non-accidental compression [5, 111]. Worn and Jones stated that there are more risk factors for sustaining rib fractures in 'two-thumbs resuscitation'. There is a risk that the compressions during resuscitation are too deep (more than the recommended depth of one-third of the anterior-posterior diameter of the chest), that the compressions are too firm and/or that there is too much deformation of the ribs. Theoretically, this could increase the risk for fractures and, due to the posterior leverage, also the risk for posterior rib fractures. Kleinman and Schlesinger showed that in rabbits posterior rib fractures did not occur, due to digital sternal compression (two-finger CPR), but did occur, due to anteroposterior bimanual thoracic compression (two-thumbs CPR) [10]. They concluded that *'posterior rib fractures require excessive levering of the posterior ribs at the costotransverse process articulation'*.

Rib Fractures, Due to Resuscitation: Epidemiological Data

Feldman and Brewer probably were the first to evaluate the probability of the occurrence of rib fractures, due to CPR. They compared the findings in 113 children (41 children with non-accidental trauma, 50 patients who had to be resuscitated, and 22 patients with rib fractures) [75]. In total 29 of the 113 children had rib fractures. In 14 of these 29 children fractures were inflicted. In 4 children the fractures were sustained in traffic accidents. Rickets/osteoporosis was found in five children. In 5 children the fractures were sustained in surgical interventions. One child was diagnosed with osteogenesis imperfecta. In spite of prolonged resuscitation, no fractures could be attributed to CPR. CPR was performed by persons with completely different levels of expertise in this field (parents, emergency department personnel, other hospital personnel, and combinations of the aforementioned). Fractures, due to non-accidental trauma, were often multiple, of different ages, and spread over multiple adjacent ribs. Moreover, frequently these children also showed other physical and radiological signs of non-accidental trauma and/or neglect.

Spevak et al. performed a retrospective study of autopsy data and post-mortem radiographs of 91 infants under the age of 1 year (56 boys, 35 girls; age range from 26 hours to 8.5 months; mean age 2.4 months), who were resuscitated for other reasons than non-accidental trauma [112]. In none of the infants rib fractures were found. Spevak et al. concluded that rib fractures rarely occur in infants due to CPR and that when rib fractures are found in an otherwise normal child, non-accidental trauma should be considered.

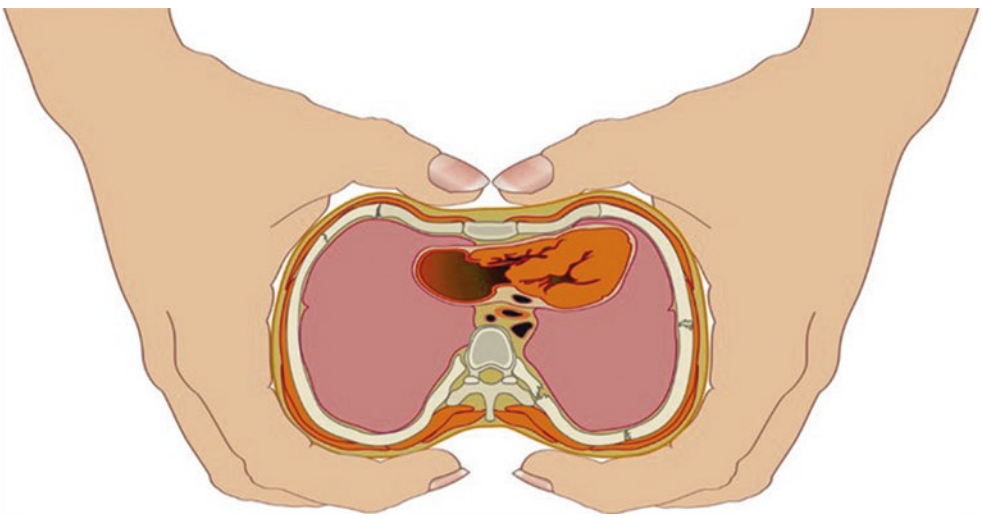


Fig. 7.31 Graphic representation of the 'two thumbs encircling hands chest compression', as advised in 2006 by the International Liaison Committee on Resuscitation (ILCOR) [110]

Betz and Liebhardt reviewed the autopsy reports of 233 children aged between 5 days and 7 years [113]. In 190 children death from natural causes was proclaimed and others died due to trauma. Ninety-four of the children who died of a natural cause had been resuscitated. Two of them had bilateral fractures in the mid-clavicular line. According to Betz and Liebhardt, these findings support the findings by other researchers that fractures of ventral parts of the ribs can occur during resuscitation. In 15 of the 43 trauma-related deaths, rib fractures were found, mainly of the posterior ends. According to Betz and Liebhardt, this shows that in case of posteriorly localized rib fractures in infants without metabolic bone diseases one should always consider non-accidental trauma. One of the most interesting findings in this study is that physicians (and not non-medical persons) caused more frequently or nearly exclusively notable injuries during resuscitation. This makes the regularly provided explanation for rib fractures in deceased children, e.g. that the fractures were the result of CPR by an inexperienced and panicking person quite improbable.

Bush et al. retrospectively analyzed type, number, and severity of unexpected complications of CPR (external cardiac compressions and ventilation) in 211 children under the age of 12 years (mean age 19 months) during an 8-year period (1988 through 1995) [114]. Children with historic or physical signs of prior trauma were excluded from the study. Cause of death was determined to be cod death in 56%, drowning in 8%, congenital cardiac defects in 7% and pneumonia in 4%. Average time of resuscitation was 45 min (ranging from 3 to 180 min) Fifteen children (7%) sustained injuries that were significant from a medical point of view and that could be considered to be due to the resuscitation: retroperitoneal haemorrhages in two children, pneumothorax in one child, pulmonary haemorrhage in one child, epicardial haematoma in one child and gastric perforation in one child. Although some children had been resuscitated for a protracted period of time by various persons with different levels of expertise, rib fractures were found in only one child, a 3-month-old infant, in whom CPR was performed by a lay-person (babysitter), 2 prehospital care providers and emergency department personnel for a total of 75 minutes. The infant had bilateral rib fractures, involving rib 8 and 9 at the sternochondral junction. The cause of cardiac arrest/death in this infant was determined to be Sudden Infant Death Syndrome. Bush et al. concluded that notable injury due to medical procedures is rare and is only seen in 3% of children. According to Bush et al. one should, irrespective of the resuscitation, always consider non-accidental trauma when injuries are found after CPR. Furthermore, they mention that the case reports found, only discuss rib fractures after prolonged and strenuous resuscitation.

Ryan et al. retrospectively analyzed the incidence, type, and pattern of injury related to resuscitation attempts in 346 children, aged 0 to 14 years, who died between 1994 and 1996 and underwent a full necropsy [115]. Children who were subject to recognized trauma before resuscitation or died because of a congenital abnormality were excluded. Finally, 204 children were included in the study. Resuscitation was performed in 75% of the children (n = 153). In 60% (n = 123) the CPR started before the ambulance arrived. In 65 children, who were resuscitated, only minor CPR-related injuries, such as superficial bruises and abrasions, were found. The likelihood of CPR-related injuries increased with the duration of the CPR. None of the children in this study had a rib fracture. According to Ryan et al., one should be cautious when attributing significant injuries to resuscitation attempts and alternative causes must be fully investigated.

Hoke and Chamberlain did a Medline search and literature review, concerning rib and sternal fractures in adults and children, resulting from conventional closed-chest compression (conventional CPR) in the treatment of cardiac arrest [116]. In adults, the reported incidence of rib fractures ranged from 13 to 97% and of sternal fractures from 1 to 43%. In children the reported incidence of rib fractures ranged from 0 to 2%. No reports on CPR-related sternal fractures in children were found. They also evaluated the findings in adults and children after active compression-decompression cardiopulmonary resuscitation (ACD-CPR). They found no convincing evidence that an increased complication rate would be associated with ACD-CPR. Hoke and Chamberlain stated that non-accidental trauma should always be considered when rib fractures are present after resuscitation in infants and toddlers, because skeletal chest injuries, due to manual CPR, are only rarely described. According to the authors rib or sternal fractures were unlikely to increase mortality, because they rarely cause severe internal organ damage. They also stated that the reasons for resuscitation (cardiac arrest) should always be carefully examined and that the child should be screened for further suspicious findings that could indicate non-accidental trauma [116]. Finally, they mentioned that it is rare for any rib fractures to occur during CPR in otherwise healthy children, irrespective of whether the CPR was performed by well-trained or untrained personnel. Various other studies before this review, but also later studies confirmed this proposition [75] [95, 112, 113].

In 2006, Maguire et al. published a comprehensive study of the literature on the prevalence of rib fractures in cardiopulmonary resuscitation (427 articles in various languages, published between 1950 and 1 October 2005) [95]. In the end they included six studies with the data of in total 923 children. Three resuscitated children had sustained an anterior-located rib fracture. Two fractures were mid-clavicular and one was located at the costochondral junction. The presence of multiple fractures had also been described. Resuscitation was carried out for different periods of time by trained medical personnel and by non-trained non-medical

personnel. Maguire et al. could not find one well-documented posterior-localized rib fracture that resulted from resuscitation. They remarked that in the studies they evaluated, the most modern and sensitive techniques for the detection of rib fractures in young children had not been applied.

The study of Dolinak underlines the conclusion of Maguire et al. regarding the diagnostics of rib fractures [117]. Dolinak evaluated the data of 70 consecutive autopsies in infants, aged between 2 weeks and 8 months, that had been resuscitated. Only children with no history or indications of injuries were included. In all children the parietal pleura of the thoracic cage was stripped and the ribs carefully examined for the presence of findings, characteristic of a fracture. Dolinak found recent subtle anterolateral-located rib fractures in 8 children (11%). Seven of the infants had more than one fracture, varying from 2 to 10 fractures. Five children had bilateral rib fractures. No lateral, posterolateral or posterior rib fractures were found. All rib fractures were subtle (often involving a merely 'bent defect' in the integrity of the rib) with little or no associated blood loss around the fracture line, and as such the fracture could easily have been missed, were it not for the removal of the pleura. The CPR method was not known in any of the resuscitated children with rib fractures. Dolinak stated that the anterolateral rib fractures he found in infants were the equivalent of rib fractures regularly found after resuscitation in adults. Dolinak, however, stated that these fractures cannot be considered to be specific for resuscitation attempts. Any other non-specific compressive force, pushing down on the chest might also lead to this type of rib fractures, irrespective of the circumstances, under which the compressive force occurred (accidental or non-accidental).

Røed et al. evaluated the findings in 261 medicolegal examinations of children, aged 0 to 4 years and 11 months (mean age 297.4 days; median age 144.5 days), in whom a full forensic autopsy was performed (1985–2004) [106]. Thirty-one children died of accidents, 12 children were homicide victims, 43 children died of natural causes and 175 children died, due to, what the authors called, an unknown cause of death (all certified as SIDS cases). CPR was performed in 119 children by health care workers and in 23 by parents or unskilled personnel. In 68 cases it was unknown, whether CPR was performed and in 51 no CPR was performed. The length of CPR in individual children was not known. Rib fractures were found in 6 children. In 3 of these children, CPR was performed by health care workers. No CPR was done on the other 3 children. The rib fractures were found in 3 victims of 'high energy traumatic accidents' and in 3 homicide victims. 5 children had either posterior or lateral fractures. One child, who was diagnosed with osteogenesis imperfecta, had 2 right-sided anterior fractures, found during the autopsy, but not seen on radiograph. No CPR was performed on this child. The child was involved in a car accident, in which the child was thrown around in the car. Røed et al. did not find any rib fractures, which could be attributed to CPR. According to Røed et al., all children with rib fractures had an injury pattern and an injury history that could explain both the rib fractures and their death.

Weber et al. analyzed the post-mortem findings in 546 children, aged 7 to 365 days, presented as SUDI (Sudden Unexplained Death in Infancy), over a 10-year period (1996–2005) [99]. This included 94 forensic autopsies. Rib fractures were found in 24 infants (4%). 15 of these 24 infants had healing rib fractures. Ten of these infants also had other findings suggestive of non-accidental trauma. Nine of the 24 infants had recent rib fractures with no surrounding tissue reaction histologically. In 7 of these 9 infants there were no other injuries and the rib fractures were interpreted to be CPR-related. All CPR-related rib fractures were situated in the anterolateral chest, in contrast to fractures due to non-accidental trauma, which were located in the anterolateral and/or posterior chest. Fractures at the anterior costochondral junction were also seen by Weber et al. in a minority of the non-accidental trauma cases, but fractures at this location were not seen in apparent CPR-related cases. Compared to healing rib fractures, which were detected on skeletal survey in 93%, recent rib fractures were only detected in 22% of skeletal surveys. Weber et al. confirmed the findings of Dolinak, that fresh rib fractures may be missed on skeletal survey, but can be reliably detected at post-mortem examination following stripping of the pleura and detailed examination of each rib [117]. Fresh anterolateral fractures, which may be multiple, contiguous and even bilateral, are highly likely to be related to resuscitation if there are no other associated injuries.

Matshes and Lew reviewed the findings in 546 children, under the age of 18 years, who had died from non-traumatic causes (with or without attempted resuscitation) and who were autopsied in the 10-year period from 1994 to 2003 [107]. Three hundred eighty-two children had a history of CPR (average age 4.17 years). In 248 of these 382 children, the CPR was performed by trained individuals only, in 133 children by both trained and untrained individuals, and in 1 child by untrained individuals only. There was no overlap between these 3 distinct groups. Matshes and Lew especially searched for CPR-related findings. In 19 children they found 22 findings that could be CPR-related: orofacial injuries, compatible with attempted endotracheal intubation in 15 children, focal pulmonary parenchymal haemorrhage in 4 children, anterior chest abrasions in 2 children, and prominent anterior mediastinal emphysema in 1 child. There were no significant hollow or solid thoraco-abdominal organ injuries. There were also no rib fractures. The remaining 164 children, who died from non-traumatic causes without attempted resuscitation served as a control group. In these children no injuries were found. Matshes and Lew stated that

the absence in their paediatric population of CPR-related rib fractures was in keeping with the findings in earlier research [75, 105, 112, 115]. They also reviewed 6 other studies, concerning a total of 1356 paediatric patients (including their own study) in which only 3 children with CPR-related rib fractures were described: 2 children with a fracture in the mid-clavicular line and 1 child with a fracture at the sternochondral junction [75, 105, 107, 112–114]. Matshes and Lew concluded that participation of non-medical or untrained individuals in CPR did not increase the likelihood of injury.

The earlier findings of Matshes and Lew, concerning the occurrence of rib fractures in infants from resuscitation attempts, were based upon data derived from infants undergoing traditional 'one-handed' CPR, and not from 'two-handed' CPR. In a follow-up study, concerning a period of 6 months, Matshes and Lew reported five unrelated, non-sequential cases of infant death with multiple acute antero-lateral rib arc fractures, which were determined to have occurred due to two-handed CPR (two-thumb-encircling hands chest compression), performed by trained medical personnel, and which could not be explained by another mechanism [118]. In all infants a detailed history of the mechanics of chest compressions was available. The infants died between the ages of 1 and 4 months. Metabolic and other bone diseases were excluded. All infants had at least 2 anterolateral rib fractures. Three infants had bilateral, multiple, and rib fractures. In all infants both the 1- and the 2-handed technique was used. Matshes and Lew concluded that the 2-handed CPR may result in rib fractures. An incorrect chest compression technique is one of the possible explanations, according to the authors. None of the infants had associated cutaneous injuries despite the technique used, or the degree of training of the performers of the CPR. Neither were there injuries to thoraco-abdominal organs. Histologic examination showed in all cases acute fractures, without signs of healing.

Reyes et al. reviewed the autopsy reports from 1997 to 2008 of 571 infants, 0 to 6 months, in whom CPR was performed prior to death [119]. They compared the findings in infants who were resuscitated in the period from 1997 to 2005 (pre-CPR revision) with the findings in infants in the period from 2006 to 2008 (post-CPR revision) (Table 7.8). They found 19 infants (3.3%), aged 0 to 179 days (mean age 78 days), in whom it was concluded that the rib fractures were CPR related. Fourteen infants became unresponsive at home, due to natural diseases (congenital heart disease, familial cardiomyopathy, metabolic disorder, complications of prematurity), infection, drowning, and undetermined causes in 10 infants. These 14 infants were all resuscitated by EMS personnel. Five infants were hospital in patients. All rib fractures were located in the anterior to lateral rib segments, most commonly in the fourth and fifth rib. The fractures were diagnosed in only 4 of the 15 infants who had post-mortem diagnostic imaging performed. In 16 infants the CPR duration was known and ranged from 21 to 260 minutes. The number of fractures varied from 1 to 11 and did not correlate with the duration of CPR. In the pre-revision period 1.3% of the infants ($n = 5$) had CPR-related rib fractures and in the post-revision period 7.9% ($n = 19$). According to the authors this is a significant increase ($p < 0.001$, Fisher-exact test). According to Reyes et al., their findings indicate an increase in CPR-related rib fractures after introducing two-handed CPR. They also stated that their findings reinforced the notion that anterior to antero-lateral rib fractures in pre-mobile infants may be a result of CPR and are not pathognomonic of non-accidental trauma.

Table 7.8 Infants under the age of 6 months who underwent CPR ($n = 571$; study period 1997–2008) [119]

	CPR-related rib fractures	No CPR-related rib fractures	Totals
1997–2005 (pre-CPR revision)	5	389	394
2006–2008 (post-CPR revision)	14	163	177
Totals	19	552	571

Franke et al. wanted to answer the question if it could be possible that posterior rib fractures in newborns and infants were caused by the 'two-thumbs' CPR technique, because this technique was, according to the authors, similar to the grip on an infant's thorax while shaking [111]. They reviewed the medical records of all infants, under the age of 12 months, who, in a period of 10 years, underwent CPR in their first year of life in three German Hospitals. Exclusion criteria were absence of medical records, no documentation of chest compressions, CPR outside the hospital, no radiograph after CPR, sternotomy, osteopenia, various other bone diseases, and accidental trauma. Children in whom non-accidental trauma was suspected, were also excluded. They included 80 neonates and infants (with a total of 546 chest radiographs; average number of radiographs 7) who had anterior-posterior chest radiographs after CPR. The mean duration of CPR was 11 min with a range from 1 to 180 min (median duration 3 min). 50 neonates underwent CPR immediately after birth. Forty-nine infants (61.2%) were premature, and 31 (38.8%) had a very low or an extremely low birthweight. In addition to the radiographs directly after CPR, 39 infants had a follow-up radiograph after at least 10 days. No rib fractures were identified in any infant. The results of this study suggest that rib fractures due to 'two-handed CPR' are uncommon. However, in 41 infants (51.2%), rib fractures could not definitively be excluded because of the limited quality of the Radiographs or unavailable follow-up radiographs. Despite the limitations of the study, Franke et al. concluded that their findings suggest that any rib fracture is uncommon after 'two thumbs CPR' and that there should be careful consideration

Table 7.9 Circumstances, under which 61 children sustained clinically not evident rib fractures [120]

Circumstances	Number
Physical abuse	20
Post-surgical (e.g. thoracotomy)	11
Accidental	
• Motor vehicle accident	1
• High distance fall	1
Medical procedures	
• Traumatic medical procedure (not otherwise specified)	1
• Presumed CPR	2
Bone disease	
• Metabolic bone disease of prematurity.	18
• Metabolic bone disease	1
Unknown	6
Total	61

of non-accidental trauma when these fractures are identified in a child without underlying bone disease or major trauma, regardless of whether CPR was performed and what technique used.

Cosway et al. evaluated the circumstances under which 61 children under the age of 2 years had sustained occult (defined by the authors as not clinically evident) rib fractures [120]. An overview of their findings is given in Table 7.9. According to the authors 2 children had acute fractures that were presumed to be secondary to cardiopulmonary resuscitation: one had adjacent anterior left-sided fourth and fifth rib fractures identified at post-mortem on specimen radiology and the other had posterolateral fractures of the sixth, seventh, and eighth ribs following bimanual chest compressions. CPR was accepted as explanation for the occurrence '*after exclusion of all other possible causal or contributory factors*'.

Ondruschka et al. evaluated the findings in 97 full autopsies of children under the age of 4 years (6-year period) [121]. A total of 40 cases were excluded: children who died due to trauma, including non-accidental trauma (n = 11), who died of natural causes, who were not resuscitated (n = 29), with known birth injuries (n = 1), with repeated CPR after initial return of spontaneous circulation (n = 3) and with prolonged survival after initial successful resuscitation of more than 24 hours (n = 2). The data of 57 children, who were resuscitated, were included in the study. The most common cause of cardiac arrest was SIDS. In 41.2% of the children, the resuscitation had started outside the hospital. Bystander CPR was performed in 43.1%. The mean duration of CPR was 50 min., ranging from 10 to 180 min. In no single case death was declared without at least partly professional CPR. 14 children (27.5%) had at least one CPR-related injury without preference to an age group. The injuries were relatively mild: bruising or abrasions of the skin (21.5%), bleeding in the airway or lung (5.9%, respectively 2%), bleeding in the heart (11.8%) and capsule bleeding in liver and spleen (both 2%). None of the recorded CPR-associated injuries were considered significant or life-threatening. One child, which was excluded from the study because of repeated episodes of CPR, had bilateral anterior rib fractures. The duration of CPR or the presence of bystander CPR did not correlate with the presence of any detected injury. It is not clear from the description of the study methods which CPR-technique had been used. The authors described, in general, that chest compressions in newborn and infants under the age of 1 ('*two thumbs encircling chest*') differ from chest compressions in children over 1 year of age ('*heel of hand*'). According to the authors in children with skeletal injuries and relevant injuries to the soft tissues and organs after CPR non-accidental trauma could be excluded.

In a large case study by Ruest et al. looking for rib fractures at chest radiographs obtained for medical reasons (7530 children, aged between 0 and 2 years, with a total of 9720 radiographs of the thorax), no acute rib fractures were found on the 43 chest radiographs associated with CPR [68].

There are two publications describing posterior rib fractures due to 'two-thumbs' CPR, the first is by Clouse and Lantz [122]. They presented this finding at a forensic annual meeting in 2008: '*Presented here are the gross, radiographic, and microscopic findings from four hospitalized neonates and infants, aged 1 day to 3 months, who died of natural causes but were noted to have posterior rib fractures at autopsy. Three cases showed evidence of acute fractures after terminal CPR attempts. In one case, remote fractures with callous formation were identified in an infant with multiple previous CPR episodes due to complications resulting from his premature birth. These infants and neonates spent the majority of their lives within the hospital. In all cases the infants had no history of abuse, no outward evidence of inflicted injury, and no additional internal injuries consistent with child abuse. It is imperative that the presence of posterior rib fractures in an infant not be ascribed impulsively to child abuse until a thorough investigation is conducted including assessment of resuscitative techniques*'. The description of the finding of posterior rib fractures in these four premature neonates and infants, who, according to the authors, died of natural causes and on whom 'two-thumbs' CPR was performed, can only be used as suggestive finding that in certain circumstances posterior rib fractures may occur due to 'two-thumbs' CPR. Unfortunately, the circumstances were not described by Clouse and Lantz. Before accepting the hypothesis of Clouse and Lantz, one would like to know why the children were born prematurely, which natural causes they died of, whether there was a bone disease with increased risk of the occurrence of fractures, e.g. metabolic bone disease of prematurity or osteogenesis imperfecta type II. In other words, because of the lacking data, a causal relationship between 'two-thumbs' CPR and posterior rib fractures cannot be established on the basis of the description

of the findings in these four premature neonates and infants with an unknown cause of death, who spent most of their live in hospital, despite that the authors stated that '*the infants had no history of abuse, no outward evidence of inflicted injury, and no additional internal injuries consistent with child abuse'*.

The second case report series was presented by Love et al. and deals with four cases of deceased children who died of a non-traumatic cause of death and who showed posterior rib fractures due to 2-thumb CPR [123]. According to the authors all four cases '*the child likely received or did receive 2-thumb CPR*' and all showed multiple rib fractures at several sites. It is noteworthy that in only one case radiography was performed and that the quality of the shown radiograph (almost constituting a babygram) is of sub-optimal quality. Based on the forensic autopsy report and relevant clinical findings the authors conclude that '*The cause of the posterior rib fractures in the presented cases is unknown; however, the most parsimonious explanation given the facts of each case is that they are secondary to CPR*'.

Rib Fractures, Due to Resuscitation: Concluding Remarks

CPR can be physically traumatic, irrespective of the technique that is used. Injuries are usually superficial, like bruises and abrasions, and never life threatening. Injuries to thoracic and abdominal structures are even much rarer than superficial injuries and are usually also not life-threatening. In most studies no association was found between the duration of CPR and the injuries that were found.

Concerning the occurrence of CPR-related rib fractures it is clear that two-finger CPR may, although very rare, lead to rib fractures. The rarity of rib fractures by using the two-finger CPR (manual chest compressions), even in children with increased risk of fractures or even in children with a lethal underlying bone disease, is underlined by a case report of Sewell and Steinberg [124]. They described the findings in a newborn girl with osteogenesis imperfecta type II. Chest radiographs were taken before and after the chest compressions. The radiographs were reviewed by several radiologists from 3 different hospitals and demonstrated no new radiographically visible rib fractures.

It is also clear that the position of the hands/fingers and the actions in two-thumb encircling hands chest compression seems similar to the position of the hands/fingers and the actions in the way, in which the chest is compressed in non-accidental squeezing.

There are indications that the incidence of rib fractures in two-thumbs CPR possibly is higher than in two-finger CPR. However, all rib fractures, due to two-thumbs CPR, that are reported in the medical literature, were located in the anterior and anterolateral segment, and only once in the posterolateral segment, except those that were reported by Clouse and Love with rib fractures in the posterior segment [122, 123].

A possible explanation for the absence of rib fractures in the posterior and posterolateral segment in two-thumbs CPR is that the person, who performs this technique, exerts pressure on the central part of the chest/the sternum, and uses his second to fifth fingers as a simple brace. The person, who violently squeezes the thorax compresses the thorax from anterior to posterior by pushing the sternum inwards, and from posterior to anterior by pushing the ribs in the posterior segment inwards, creating leverage of the ribs on the vertebral processes [16, 118]. The force that is used while squeezing the chest is illustrated in a case report by Lauridson and Cromblin [102]. The authors described a 4-month-old boy with multiple posterior rib fractures and without any external evidence of non-accidental trauma. The father admitted he hold the child between his arm and chest and repeatedly squeezed the child as hard as he could (so hard his arms got a little achy). The child turned purple and unconscious (probably due to cardiopulmonary effects of the squeezing: restriction of breathing and reduced cardiac output). The father thought he might have killed the child and blew into the child's mouth to get him to breathe. The child started breathing. Because of feeding difficulties the mother sought medical care. A chest radiograph revealed multiple healing rib fractures with callus formation. This case report endorses that violent squeezing concerns forces different from the force that is used in normally performed CPR.

There is one major caveat to the discussion on rib fractures resulting from CPR, to date, almost all studies have used conventional radiography. In an autopsy-based paper Dolinak describes a series of 70 consecutive autopsies in infants ranging in age from 2 weeks to 8 months, [117]. Of these infants, subtle anterolateral rib fractures were diagnosed in 8 (11%). With the increasing use of CT, especially in the post-mortem setting these subtle buckle fractures are increasingly diagnosed after CPR has been applied (Figs. 7.32a, b and 7.13a, b A & B). As these buckle fractures will heal without noticeable callus formation they will not be visible on radiography.

Other Injuries, Due to Resuscitation

Most studies concerning CPR-related injuries in children focus on major physical injuries, such as thoraco-abdominal injuries, which are potentially life threatening. However, major resuscitation injuries are rare [105, 107, 114, 125]. It is generally accepted that minor soft-tissue injuries are common in both adults and children [115, 125]. These CPR-related injuries, most commonly found in the orofacial region, often have no clinical relevance at all, but in the presence of rib fractures may be essential in a proper forensic evaluation in cases of suspected child abuse.

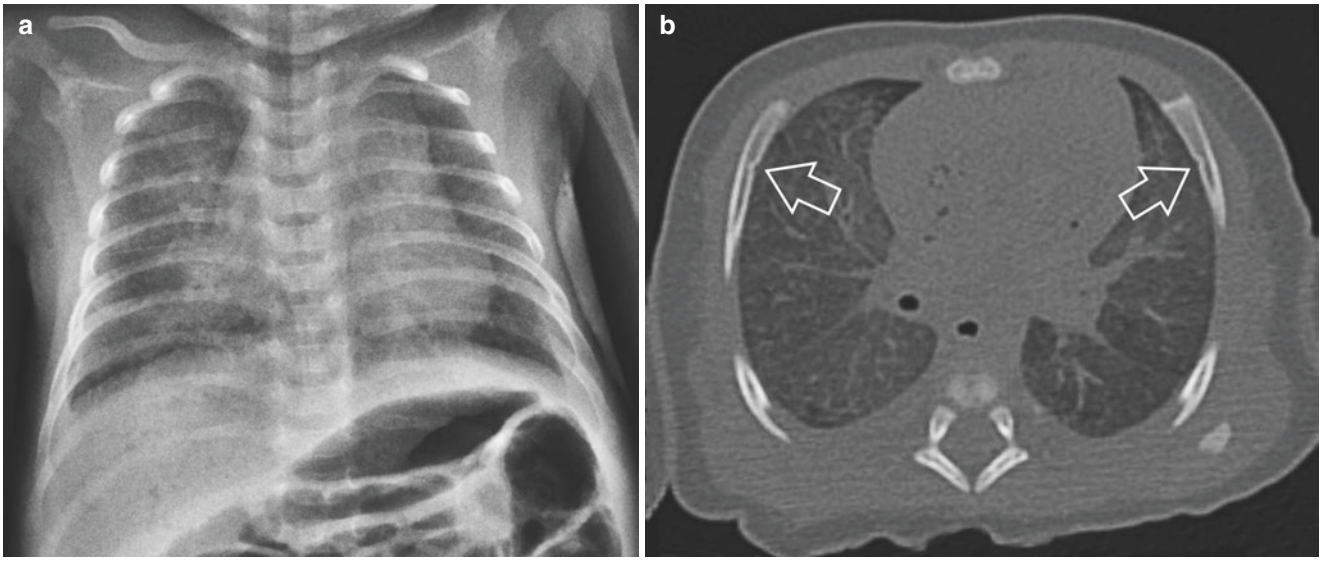


Fig. 7.32 Neonate who was found unresponsive in his bed, CPR was performed by caregivers and medical personnel. (**a**) Chest radiograph, as part of the skeletal survey, shows no sign of rib fractures. (**b**) Chest CT shows anterior buckling fractures of the fourth ribs (insets), thought to be resulting from CPR. Autopsy showed no signs of non-natural death

Table 7.10 Non-skeletal injuries due to resuscitation [114, 115, 195]

Resuscitation method	Findings
Airbag-valve ventilation (mask resuscitation)	Abrasions over the nasal bridge, undersurface of the nose, the lips, cheeks, and anterior chin surface
Mouth-to-mouth resuscitation	Tightly grouped fingernail imprints and abrasions around the nose, with loosely satellite fingernail imprints and abrasions on the cheeks, forehead, and chin.
Orotracheal intubation resuscitation	Contusion or laceration of the lips and compression of oral tissue against the teeth
Defibrillation	Burns and abrasions
Chest compression	Retroperitoneal haemorrhage, pneumothorax, pulmonary haemorrhage, epicardial hematoma, gastric perforation

Other, non-skeletal, injuries can consist of orotracheal lesions due to intubation, placement of a nasogastric tube, thoraco-abdominal organ trauma and skin lesions (Table 7.10) [108, 114, 126]. The skin lesions can consist of orofacial findings but there may be skin findings on the rest of the body, e.g. bruising of the anterior thoracic wall (or perhaps on both the anterior and posterior wall in vigorous 'two thumbs encircling hands chest compression'). The incidence of other injuries ranges, depending on the severity of findings and duration of resuscitation, from 7% to 62% [114, 115]. In a large retrospective post-mortem study in 546 children by Matshes et al. no relation between the development of CPR-related injuries and the level of training of the medical or non-medical resuscitators was found [107]. Matshes concluded that *'Injuries secondary to resuscitative efforts are infrequent or rare, pathophysiologically inconsequential and predominantly orofacial in location'* and that '*participation of nonmedical or untrained individuals in resuscitation did not increase the likelihood of injury*'. It can be concluded that whenever serious traumatic injuries are found after CPR, non-accidental trauma should be considered.

7.3.4.2 Physiotherapy, Chiropractic Care, and Rib Fractures

Only a few articles have been published in the medical literature, in which rib fractures in children due to chest physiotherapy or chiropractic care are reported.

Purohit et al. were the first to describe the occurrence of rib fractures in a male neonate, according to the authors, most probably due to chest physiotherapy, although they did not exclude the occurrence due to the stress of prolonged respiratory distress [127]. The boy, weighing 2.1 kg, was born at a gestational age of 32 weeks and was admitted to the neonatal intensive care unit, because of respiratory distress due to hyaline membrane disease. He received physiotherapy, starting at 7 days of age (percussion by hand or vibration with electric toothbrush) was started because of atelectasis of the right upper and middle lobes as well as an infiltrate in the remaining lung fields. It was stopped after a while, but started again at the age of 53 days because of recurrence of the atelectasis. At the age of 75 days, the chest radiograph showed healing fractures of the sixth and seventh rib on the right side and of the eighth rib on the left side. Radiographs of the rest of the skeleton showed no abnormalities. The fractures were seen during his stay at the hospital.

Chalumeau et al. described five boys, aged 1 to 6 months (median age 3 months), over a period of 4 years (1996–1999) when they received chest physiotherapy and who appeared to have sustained a rib fracture due to chest physiotherapy for bronchiolitis (4 children) or pneumonia (1 child) [128]. The median number of fractures was 4, with a range of 1 to 5. All fractures were located between the third and the eighth rib, in 4 children lateral and in 1 child posterior. In 4 children the fractures were unilateral and in 1 child bilateral. The authors estimated that the prevalence of rib fractures due to chest physiotherapy during the study period was 1:1000 in children admitted for bronchiolitis or pneumonia. They consider chest physiotherapy to be a potential but rare cause of rib fractures.

Gorincour et al. did a prospective study (May 2000 to May 2003) into rib fractures in children, treated for bronchiolitis [129]. They found in total six children, under the age of 2 years old with lateral rib fractures and possible remnants of rib fractures. The authors believe that in these children no plausible grounds for a suspicion of non-accidental trauma were present. The only possibility left was chest physiotherapy. Twelve of the 14 fractures were located in the lateral part of the chest from the fourth to the seventh rib. No fractures were found at the costochondral junctions. In 12 out of 14 lesions, only periosteal reactions were seen without a clearly visible fracture. According to the authors this was feasible since repeated chest physiotherapy causes subperiosteal haemorrhages rather than real fractures.

Chanelière et al. reported two children that had sustained lateral fractures of the third to sixth rib after physiotherapy for bronchiolitis [130]. The authors posed that, although rib fractures resulting from physiotherapy are rare, physicians should be aware of the possibility when confronted with rib fractures in a child that received chest physiotherapy.

Wilson et al. reported the presence of fractures of the left seventh and eighth ribs posteriorly in a 21-day-old infant who was treated by an chiropractor for colic 5 days before the fractures were diagnosed [131]. On day of presentation, the mother reported that she felt a crackling sensation when she touched the infant's back, while the patient was lying against her chest. On evaluation by the paediatrician, the finding of crepitus was confirmed and a chest radiograph was obtained, which revealed acute fractures of the left seventh and eighth ribs posteriorly. CT of the head, skeletal surveys and laboratory tests did not show any abnormalities. During the evaluation of the suspicion of non-accidental trauma no concerns were found. During the evaluation, the mother described that *'the chiropractor initially held the patient upside down by the hips, wrapping his hands around her hips and lower ribs. Next, he applied pressure along her spine with his fingertips. Finally he used a 'spring-activated device' on the patient's back while she was lying down on her mother's chest, in the same location the fractures were later found. The patient cried immediately after the procedure and then fell asleep. She continued to be fussy after the maneuver'*. The case was discussed with the chiropractor, who confirmed the mechanism of treatment that was described by the patient's mother.

7.3.4.3 Surgical Interventions and Rib Fractures

The occurrence of rib fractures due to surgical procedures has only been described in medical literature a few times.

Feldman and Brewer probably were the first to evaluate the probability of the occurrence of rib fractures, due to CPR. They compared the findings in 113 children (41 children with non-accidental trauma, 50 patients who had to be resuscitated, and 22 patients with rib fractures) (see also Sect. 7.3.4.1) [75]. In total 29 of the 113 children had rib fractures. In 5 of the 29 children the fractures were sustained in surgical interventions.

Cosway et al. evaluated the circumstances under which 61 children under the age of 2 years had sustained occult (defined by the authors as not clinically evident) rib fractures (see also Sect. 7.3.4.1) [120]. In 11 children it was determined that the occult rib fractures were due to surgical procedures.

7.3.5 Trauma after Birth: Medical Conditions

7.3.5.1 Rib Fractures in Preterm Children

Compared to term neonates, premature infants are at higher risk for fractures in day-to-day handling. Most often these are fractures of the long bones and ribs (Fig. 7.33). Rib fractures in premature infants usually will cause no clinical symptoms.

Helfer et al. described the findings in 4 infants (3 preterm and 1 term), who were presented to the physician with serious bony injury between the ages of 4 and 10 months [132]. In these children initially non-accidental trauma was suspected, because of the presence of multiple bone injuries. Finally, it became clear that the fractures most probably were due to parent- or caretaker-administered passive exercises (*'home-administered physical therapy'*). In 2 of the 4 children rib fractures were found. The rib fractures seen in these infants (1 fracture in 1 infant and 9 fractures in the second child) were thought to be the result of vigorous pinning of the infant to the table or floor as the helper held the chest. According to the authors, the amount of force inflicted by the caretakers of the 4 infants, as they demonstrated their techniques to the staff, was significantly greater than that which they were taught. In none of the infants rickets was present at the moment the fractures occurred. In two infants, however, rickets of prematurity occurred months earlier, but this was

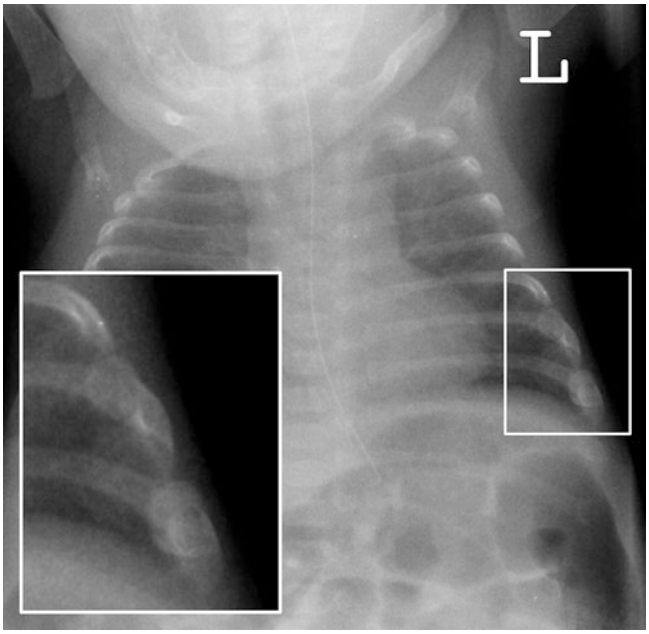


Fig. 7.33 Neonate born at 26 weeks and 2 days gestational age. Clinical follow-up chest radiograph several weeks later shows healing lateral rib fractures of the seventh and eighth left lateral ribs. As the child had not left the neonatal ICU the fractures were diagnosed as the result of prematurity-induced osteopenia

already healed and played no role in the occurrence of the fractures.

Prematurely born children and critically ill neonates also have an increased risk for sustaining rib fractures during resuscitation. These are nearly always located in the anterior or anterolateral segment [12].

Premature children also run an increased risk for rickets, which may include rib fractures. Keipert described a preterm infant, born at 26 weeks' gestation, who was found at the age of 14 weeks to have radiological changes consistent with rickets, and multiple healing fractures of the ribs [133]. Geggel et al. described the findings in 2 preterm infants with rickets in whom multiple rib fractures were found [134]. Dabezies and Warren evaluated the findings in 247 premature infants with a very low birth weight (weighing under 1500 gr) [135]. Rickets was diagnosed in 96 (39%) infants (mean age 50 days) and fractures were diagnosed in 26 (10.5%) infants (mean age 75 days). These 26 infants had a total of 98 fractures. Fractures of the ribs were found in 54 infants, of the radius in 13, of the humerus in 10, of the ulna in 8, of the femur in 5, of the metacarpal in 4, of the clavicle in 3 and finally of the fibula in 1 infant. Risk factors included hepatobiliary disease, total parenteral nutrition, diuretic therapy, physical therapy with passive motion and chest percussion therapy.

Smurthwaite et al. retrospectively reviewed the radiographs of 72 extremely low birth weight infants (1000 g or less) with a gestation range of 22–33 weeks, and who had survived 4 or more weeks [136]. Five of the 72 infants (7%) had radiologically apparent rib fractures. None involved posterior rib shafts. All infants with rib fractures died in the neonatal intensive care unit.

Lucas-Herald et al. evaluated the prevalence of rib fractures in 3318 preterm infants, who were born at less than 37 weeks of gestation [137]. One thousand four hundred forty-six infants had a total of 9386 chest radiographs. Less than 2% (n = 26) were identified as having a total of 62 rib fractures. The median gestational age at birth of these 26 infants was 26 weeks with a range of 23 to 34 weeks. A full skeletal survey was performed in 8 of 26 (31%). Investigations for non-accidental injury occurred in 4 of 26 (15%) cases. 18 infants were diagnosed with osteopathy of prematurity. In 5 children the circumstances were unknown and in 3 infants the rib fractures were determined to be due to non-accidental trauma. 27 (36%) of the 62 fractures were sited posteriorly. 15 infants with posterior rib fractures (53%) were diagnosed with osteopathy of prematurity. Risk factors, often more than 1 (total parenteral nutrition, diuretics, conjugated hyperbilirubinemia, low calcium/phosphate levels, chronic lung disease, high alkaline phosphatase levels, cystic fibrosis, and low serum vitamin D levels) were present in 23 of 26 (88%) infants. According to the authors, the evaluation of these fractures in infancy requires a detailed neonatal history irrespective of the site of rib fracture.

Wei et al. evaluated the findings in all babies admitted to a neonatal intensive care unit, in whom during their stay one or more fracture(s) were diagnosed [138]. The findings of 27 infants (median gestational age of 28 weeks and a range of 23.6 to 40.4 weeks; median birthweight of 920 g and a range of 485 to 4875 gr) with a total of 71 fractures were included. Rib fractures were most common: 17 of the 71 concerned anterior or lateral rib fractures and 28 posterior rib fractures (5 humeral, 3 ulnar, 4 radial, 8 femoral, 1 tibial, 4 clavicular and 1 skull). Preterm infants were at risk of developing multiple fractures. In preterm infants the fractures were associated with a variety of contributing factors (e.g. more preterm, lower birth weight, more commonly osteopenic, total parental feeding). The authors specifically stated that posterior rib fractures that have been viewed as a hallmark of NAI are relatively common in sick preterm infants in the neonatal intensive care unit. Posterior rib fractures were more commonly found in infants who were born more preterm (25.1 vs. 29.4 weeks) and who were requiring diuretics.

7.3.5.2 Rib Fractures in Children with Bone Disease

When a rib fracture is found in a neonate, one should always be aware of diseases that affect bone mineralization and may contribute to the occurrence of fractures in young children. Most descriptions of rib fractures in children with bone disease concern single case reports.

Osteogenesis Imperfecta and Rib Fractures

The most common bone disease in which an increased risk of rib fractures has been described, is osteogenesis imperfecta (OI) (see 14.3). OI-related rib fractures can occur before birth (Sect. 7.3.1), during birth (Sect. 7.3.2) and after birth, e.g. due to CPR (Sect. 7.3.4.1). Generally, this diagnosis will cause few problems in the most severe cases of OI, the foetus will die in utero, or (often) the infant will die shortly after birth (Sect. 14.3) [139].

Greeley et al. did a retrospective chart review of a series of 68 infants and children under 18 years of age who have the diagnosis of OI (any type) from a single institution [140]. Type 1 was diagnosed in 23 patients (34%), type 2 in 1 (2%), type 3 in 17 (25%), and type 4 in 24 (35%). In 3 patients (4%) the type was unknown. They found OI-related rib fractures in 15 patients (22%). Thirteen of these 15 patients (87%) were diagnosed prenatally or immediately after birth, 12 (80%) had blue sclera, 10 (67%) were noted to have osteopenia on radiograph and 5 (33%) had a family history of OI. The 2 (13%) paediatric patients with rib fractures who were not diagnosed prenatally or at birth were diagnosed at the ages of 14 and 43 months. Both were diagnosed with type 1, had a family history of OI and had blue sclera. In other words, no patients who were diagnosed with OI under the age of 1 year had rib fractures.

Pereira referred to the findings of Greeley et al. concerning OI-related rib fractures, but made one reservation: *'Rib fractures appearing in infancy would not be supported as being from OI, but at this point in time, much larger data collection is needed before saying that OI could be excluded in clinical practice based on if they appear in infancy'* [141]. According to Pereira symmetric rib fractures, specifically if they are posterior, medial, and bilateral, are more likely due to non-accidental trauma in children under the age of 3 years and are rarely seen in children with OI.

Rickets and Rib Fractures

Rickets-related rib fractures have been described in the medical literature (see also Sections 14.4 and 15.5.3). Chapman et al., e.g. found 3 lateral and 1 anterior-lateral rib fractures in 2 children of a total of 45 children, aged 2 to 24 months, with rickets, evident by radiograph [142]. Fracture types were transverse long bone shaft fractures and metaphyseal fractures. All infants and toddlers with fractures were mobile (two crawlings, four cruising, one walking). All fractures occurred exclusively in patients with severe, overtly evident rickets. In one of the children with rib fractures (11-month-old infant) radiology showed anterior rib end widening (rachitic rosary), right anterior lateral rib fractures, bilateral proximal humeral severe irregularity and disruption judged to represent metaphyseal fractures. None of the fractures were seen in a child with normal bone or mild rickets on radiograph.

Menkes Disease and Rib Fractures

There is only one case report in which the occurrence of one rib fracture was described in an infant with Menkes disease and in which it is suggested that the rib fracture was related to Menkes disease (see also Sects. 14.5.7).

Wacks et al. and Droms et al. reported the findings in a 5-month-old boy with bronchiolitis [59, 143]. The chest radiograph showed a healing posterior fracture of the right seventh rib, which led to a suspicion of non-accidental trauma. A complete skeletal survey was done, which showed bilateral irregularities to the distal radial and ulnar metaphyses consistent with healing metaphyseal fractures, as well as healing metaphyseal corner fractures of the right and left distal femurs. The survey also showed more than 5 wormian bones of the skull and an unusual pelvis configuration. No acute intracranial abnormalities, except the aforementioned wormian bones were seen on a CT scan of the head. On further examination, the boy had coarse hair with a pili torti appearance, diffuse pallor, hypotonia, pectus excavatum and an asymmetric sacral dimple. These findings, combined with the laboratory findings (low serum copper level and low serum ceruloplasmin level) led to Menkes disease as a diagnosis. Wacks et al. gave no definitive statement that all the skeletal findings, especially the healing rib fracture, were Menkes disease- or copper deficiency-related or that non-accidental trauma was definitively excluded. They only stated that: *'Even though a metabolic cause may be identified to explain injuries consistent with child abuse, it is important to consider child abuse in the differential until the full multidisciplinary workup has been completed'.*

Droms et al. stated: *'Although the effects of Menkes disease are pervasive, many of the initial clinical findings result from connective tissue abnormalities and can be misinterpreted as child abuse. These include subdural hematomas, cervical spine defects, rib fractures, and metaphyseal spurs of the long bone metaphyses. Other radiologic findings such as multiple wormian bones are thought to be normal anatomic variants or suggestive of bone dysplasias rather than child abuse'.*

Droms et al. further stated: '*Copper deficiency leads to connective tissue abnormalities and may result in subdural hematomas, wormian bones, cervical spine defects, rib fractures, and spurring of the long bone metaphyses. Several of these findings, including fractures and subdural hematomas, may be misinterpreted as child abuse'.*

Although the statement about rib fractures in children with Menkes disease is referenced by Droms et al. with articles by Cronin et al. and Dongkyu et al., the occurrence of Menkes disease- or copper deficiency-related rib fractures or even rib fractures, in general is not mentioned in these articles [144, 145]. The metaphyseal lesions in the case report by Wacks et al. and Droms et al. were initially determined to be metaphyseal corner fractures, but eventually were deter-

mined to be findings consistent with the metaphyseal findings in Menkes disease/copper deficiency. Already in 1987 Chapman stated: *'The differentiation of metaphyseal abnormalities caused by copper deficiency from metaphyseal fractures caused by child abuse and from normal developmental variants, such as cupping of the anterior ends of ribs and metaphyseal breaking, is not difficult for the experienced'* [146].

Drom et al. excluded that the rib fracture was due to a non-accidental trauma by stating that rib fractures belong to the specific findings in Menkes disease/copper deficiency and by stating: *'In any male infant in whom child abuse is suspected because of fractures or subdural hematomas, the hair should be examined for the coarseness and sparseness typically seen in Menkes disease'*. However, there is no evidence for that statement, concerning the rib fracture in this 5-month-old boy.

Metabolic Disturbances and Rib Fractures

Rib fractures have been described as congenital and acquired medical conditions with metabolic disturbances.

Rib fractures have been reported in neonates suffering from hyperparathyroidism/ hypercalciuric hypercalcaemia, due to an inborn error of metabolism. Nyweide et al. reported a 2-week-old infant, who presented with bilateral rib fractures, hypercalcemia and subperiosteal bone erosions [147]. Parathyroid hormone levels were elevated and urine calcium was low. Her parent's laboratory test results were normal. Gene sequencing revealed a new mutation of the calcium-sensing receptor gene, causing severe neonatal hyperparathyroidism, a variant of hypocalciuric hypercalcemia. According to Nyweide et al. this is a rare cause of neonatal hyperparathyroidism and non-abusive fractures.

Maternal factors may also play a role in the origin of rib fractures of their often premature neonates. Kaplan et al. presented the presence of diffuse osteopenia of the long bones and probable rib fractures in 10 premature infants, who were exposed in utero to large doses of MgSO4, due to prolonged maternal treatment with MgSO4 administration for preterm labour [148]. According to Kaplan et al. these infants have an increased risk of developing hypocalcaemia, osteopenia and fractures. The effects were most pronounced in infants who were products of multiple pregnancies.

In rare cases, serious malnutrition may cause metabolic disturbances, and as such increase the risk for fractures. This may lead to a suspicion of non-accidental trauma. Bilo et al. described a 7-month-old hospitalized baby with severe malnutrition, due to chronic malabsorption [149]. The malabsorption was caused by a 'short-bowel' syndrome, due to necrotizing enterocolitis. The infant had been hospitalized from birth on, and was totally dependent on parental feeding. Additional radiological examination showed an obvious case of rickets.

Robinson et al. described the findings in an 8-month-old boy with irritability and pain with movement [150]. He was unable to bear weight and had a poor head control. He had palpable clavicular bony lesions and point tenderness of the hips. Over the last 4 months the boy had lost several developmental milestones. He had decreased appetite and minimal weight gain. A skeletal survey showed multiple rib fractures, osteoporosis and rickets. Laboratory examination showed hypophosphatemia and an elevated serum aluminium level. Past medical history was positive for gastroesophageal reflux. He had been started on ranitidine and aluminium hydroxide at 2 months of age. The infant's formula contained elevated aluminium levels. Further investigation showed that ½ tablespoonful instead of ½ teaspoonful of antacid had been added to each 6-ounce formula bottle for the prior 6 months, while only 1 month of antacid therapy had been recommended. An objective causality assessment revealed a probable adverse drug event. According to the authors phosphate-binding substances like aluminium-containing antacids can bind large amounts of phosphorus, causing hypophosphatemia and metabolic bone disease.

Osteopetrosis and Rib Fractures

Autosomal dominant osteopetrosis (aka Albers-Schonberg disease, marble bone disease, osteosclerosis fragilis generalisata) is marked by increased bone density due to a defect in bone reabsorption by cells called osteoclasts. This leads to the accumulation of bone with defective architecture, making them brittle and susceptible to fracture [151].

Lethal osteopetrosis with multiple fractures, including rib fractures, in utero has been described by el Khazen et al. and Malinger et al. (see also Sect. 7.3.1) [34, 35].

Bodamer et al. described the presence of multiple fractures of the long bones due to osteopetrosis in a 3-month-old infant, although the finding of multiple fractures, according to Bodamer et al., is highly unusual [152].

Rib fractures have been described only very rarely in paediatric patients with osteopetrosis. Waguespack et al. retrospectively evaluated the data of 62 patients (all ages) with autosomal dominant osteopetrosis and fractures [153]. They found rib fractures in 8 patients (5%). Unfortunately, they did not differentiate between rib fractures in paediatric (under the age of 18 years) and adult patients. Only one case report concerning a young child with a possibly osteopetrosis-related rib fracture is found in the medical literature. Matrane et al. described the findings in a 2-year-old child, born after a consanguineous marriage, who was hospitalized because of mucocutaneous pallor, splenomegaly, failure to thrive and altered psychomotor development [154]. Radiological examination showed signs of osteopetrosis and a CT scan revealed a double fracture of the right coracoid process and the tenth right rib.

7.3.5.3 Rib Fractures in Serious Coughing Fits

Rib fractures have been described in adult and paediatric patients and may result from the forces released during prolonged and forceful coughing and therefore can be labelled as 'stress fractures' [155]. According to Sano et al. coughing-related rib fractures may occur in every age group regardless of the presence or absence of underlying disease [156]. Most commonly these are single rib fractures, which are subtle and non-displaced [156–158].

The first description of rib fractures due to coughing in adult patients, concerned the occurrence in pregnant women [159]. They may occur due to severe coughing or secondary to, e.g. airway infections, like tuberculosis, asthma, or irritation of the airways [155, 157, 160–166]. In paediatric patients coughing-related rib fractures are described in case of bouts of violent coughing, as can be seen in whooping cough.

The American Academy of Pediatrics (AAP) and the Centers for Disease Control (CDC) describe complications of whooping cough in adolescents and adults: fainting, sleep disturbances, urine incontinence and pneumonia [167, 168]. Both also mention rib fractures in teens and adolescents. The most critical course of whooping cough is seen in infants under the age of 6 months, especially in premature and non-vaccinated infants. The complications seen in infants under the age of 1 year are pneumonia in 22% of the infants, seizures in 2%, encephalopathy in less than 0.5%, and death. The incidence of fatal whooping cough is approximately 1% in infants under the age of 2 months and under 0.5% in infants between the ages of 2 and 11 months. The most prevalent complication and cause of whooping cough-related death is secondary bacterial pneumonia. Of the children that die, 85% is under the age of 3 months [168]. According to the CDC, the pressure in severe coughing fits in whooping cough can lead to pneumothorax, nose bleeds, subdural haemorrhages, hernias, and rectum prolapse. Neither the AAP nor the CDC mention rib fractures as a result of whooping cough in infants. The cases of rib fractures, due to coughing fits, that are reported in the medical literature, have only been described in adolescents and adults [169–172]. As in adult patients with cough-related fractures most commonly these are single rib fractures, although more fractures and symmetrical fractures have been reported. McNaughten et al. described the occurrence of rib fractures at the angle of the left fifth rib and anteriorly on the right sixth rib in a 13-year-old girl with a 3-day history of worsening paroxysmal cough and vomiting, due to pertussis [172]. The youngest child mentioned in the literature who had sustained a rib fracture (it concerned the first rib) due to whooping cough was an 11-year-old boy, who presented with a sudden onset of severe right-sided pleuritic chest pain with a 6-week history of a coughing illness and considerable weight loss [169].

Only very rarely other coughing disorders than whooping cough are reported in the medical literature. Allen and Aziz reported a 3-month-old girl with a ventricular septal defect who developed six unexplained posterior rib fractures while being in hospital because of paroxysmal coughing, due to H1N1 influenza [173]. As far as could be derived from the information given by the authors, they were of the opinion that the fractures could have been due to severe paroxysmal coughing. Chest physiotherapy was excluded because of the location of the fractures, which, according to the authors, mainly occur laterally in CPT (see also Sect. 7.3.4.2). Prematurity (Sect. 7.3.5.1), prolonged diuretic use, sub-optimal nutrition and bone density were mentioned by the authors as possible other contributing factors to the occurrence of the posteriorly located rib fractures in the child. According to the authors, non-accidental trauma was fully investigated and thought to be unlikely. They do not mention on what grounds non-accidental trauma was excluded. Unfortunately, the authors did also not mention how they thought that the location of the fractures could be explained, although posteriorly located rib fractures have been described regularly in preterm infants (see Sect. 7.3.5.1).

7.3.5.4 Bone Disease: Concluding Remarks

In infants and children rib fractures may, as shown above, be the result of underlying diseases or disorders. This implies that when faced with an infant or child with a rib fracture without a clear trauma history the attending clinician needs to consider not only non-accidental injury and thus should obtain a complete clinical history, including the perinatal period and family history. Based on this the appropriate ancillary tests should be ordered to rule out an underlying disease.

7.4 Penetrating Chest Trauma

7.4.1 General Aspects of Penetrating Chest Trauma

Penetrating chest trauma constitutes 9.5 to 15% of chest injuries in children, with most caused by gunshots, knife wounds, and injury from other sharp objects [4, 174]. Nearly 100% of the fatalities in children with penetrating chest trauma are due to the chest injury itself, while in fatalities in children with blunt force chest trauma approximately 15% of the deaths are due directly to intrathoracic injuries and almost half of the deaths to associated neurologic injury [4]. Rib fractures have been described in children as associated injuries in penetrating chest trauma.

Paediatric chest injuries can be sustained, due to accidental and non-accidental circumstances. As already stated in Sect. 7.2, chest trauma due to penetrating trauma, is mainly reported in older children or in adolescents and only rarely reported in young paediatric patients [4, 174]. Sometimes (more or less) blunt penetrating trauma may occur in paediatric patients, e.g. due to falls on railings or fence posts.

7.4.2 Penetrating Chest Trauma: Accidental Circumstances

Accidental penetrating chest trauma has been described in paediatric patients, of all ages up to the age of 18 years. In the medical literature several sharp objects, causing penetrating injuries, have been described. Examples are given in Table 7.11. The injuries in the case reports rarely were fatal, except for the case described by Murphy: a 12-year-old boy who was struck in his home in the left anterior chest by a single, sharp, slender fragment of glass blown from a window that shattered in a thunderstorm [175]. The boy immediately collapsed and was pronounced dead at the scene.

Boleken et al. evaluated the findings in 84 paediatric patients (mean age 10.3 ± 3.79 years; male-to-female ratio 6 to 1) with penetrating injuries to the thorax and abdomen (research period 2006 to 2012): 26 gunshot injuries and 58 stabbing injuries [176]. Thoracic injuries were present in 31 patients (mean age 9.77 ± 4.20 years), abdominal injuries in 43 (mean age 10.38 ± 3.71 years), and both thoracic and abdominal injuries were found in 10 (mean age 12 ± 2.26 years). According to the authors almost 70% of the patients were accidentally injured. In 11 of the 31 children with thoracic injuries, the injuries were caused by gunshots, while the injuries in the remaining 20 patients were caused by stabbing (11 knife injuries, 5 penetrating sharp device injuries, and 4 cases of falling on a sharp object). None of the children in the study had secondary complications or died due to the penetrating injury.

Table 7.11 Penetrating objects (accidental circumstances)

Author(s)	Sex	Age	Penetrating object	History
Riggle et al. [196]	Girl	15 months	Knitting needle	Sat unrestrained in car seat in the back of a car traveling approximately 5 miles per hour in a parking lot. Because of a sudden stop, she was thrown forward onto a bag of knitting supplies. A knitting needle was protruding from her right chest
Gettig et al. [197]	Girl	4 years	Knitting needle	Fell off the arm of a couch onto her mother's knitting bag
Kulaylat et al. [198]	Girl	2½ years	Nail from nail gun	The child reportedly picked up a lightweight nail gun and accidentally discharged the device against her body
O'Neill et al. [199]	Boy	4½ years	Cactus spine	Was playing in the yard when he accidentally fell, chest first, onto a 'barrel cactus' (echinopsis pasacana)
Papadopoulos et al. [200]	Boy	3 years	Leaf pin	Chest injury 1 week before. Fell while playing over a short palm tree (Yucca elephantipes)
Ramaswamy et al. [201]	Boy	12 years	Pencil	Involved in a rough tumble with his friends, suffered a penetrating injury to the posterior wall of his chest on the left side from a pencil in his coat pocket
Fisher et al. [202]	?	?	Pencil/pen	Fisher et al. described 14 children with injuries from 'penetrating' writing instruments: Head and neck (9), chest (1), bladder/perineum (2), and extremities (2)
Malla et al. [203]	Boy	10 years	Bamboo stake	Fall from an approximately 15-foot tall coconut tree, landing over an upright bamboo stake approximately 50 centimetre long, resulting in a trans-abdominal, trans-thoracic injury
Bawany et al. [204]	Girl	9 years	Metal rod	Was hit by a public transport bus and impaled by a metallic rod through the right axilla, and subsequently dragged for a distance
Linard et al. [205]	Boy	14 years	Needle	Sewing needle accidentally inserted through the chest wall and migrating spontaneously to the pericardium
McLaughlin et al. [206]	Boy	15 years	Bullet	Accidentally shot in the chest
DeCou et al. [207]	Boy	5 years	Air gun pellet	Accidentally shot by his brother with a pellet gun
	Boy	8 years	Air gun pellet	Accidentally shot by his brother with a pellet gun
	Boy	15 years	Air gun pellet	Accidentally shot by his brother with a pellet gun
Greenlees et al. [208]	Boy	16 years	Air gun pellet	Been shot in the chest with an air pellet gun while playing with a friend. Small entry wound in the right axilla
Murphy [175]	Boy	12 years	Shattered glass	Struck in his home in the left anterior chest by a single, sharp, slender fragment of glass blown from a window that shattered in a thunderstorm

7.4.3 Penetrating Chest Trauma: Non-accidental Circumstances

Non-accidental penetrating chest injuries may occur at home (often child abuse), outside the home (often physical assaults by peers) or in conflicts/wartime (often collateral damage). According to some authors, most of the non-accidental paediatric chest injuries, due to penetrating trauma, occur in conflicts and wartime, but are rarely seen in peacetime [177]. However, according to Cotton and Nance a dramatic increase in the incidence of penetrating thoracic injuries and resulting fatalities in children, especially due to violence outside the home, e.g. gang-related violence, took place in the United States at the end of the twentieth century and beginning of the twenty-first century [178].

7.4.3.1 Non-accidental Trauma at Home

Penetrating chest injuries due to non-accidental trauma at home are very rare. Only a few case reports are found. These concerned penetrating injuries due to sewing needles in young children.

Sola et al. reported a 3-month-old girl, who was brought to the emergency department because of ear pain [179]. It was noted that she had a raised erythematous area just inferior to the xyphoid process, which on palpation revealed a sharp metallic needle-like structure in the subcutaneous tissue. Radiographic evaluation of the subxyphoid mass showed a needle traversing posteriorly into the lower chest at the level of the diaphragm with proximity to the cardiac silhouette. A CT scan showed the presence of the needle within the pericardial sac. The medical history showed that the girl had been hospitalized in another hospital because of a closed head injury and an occipital fracture, due to a fall. On a skeletal survey there were no other fractures seen, except for the skull fracture. The investigation by the Child Protection Service determined that both injuries were inflicted.

Choudhary described the findings in a 3-year-old child [180]. A foreign body was detected on his chest radiograph while being evaluated for cough. Eventually, 3 sewing needles were seen on chest radiograph in the chest cavity. According to the authors '*In children, injuries caused by sewing needles usually occur accidentally except for some self-inflicted injuries caused by depressive behaviours or suicide attempts. Child abuse issues should be investigated in all such injury cases. In this case, the cause and timing of insertion of the needles into the chest cavity remain unknown. The possibility of child abuse or witchcraft rituals cannot be ruled out*'.

Deng et al. reported 2 girls, aged 2 and 5 years, in whom as an incidental finding sewing needles were found in the chest/lung, probably due to non-accidental trauma [181]. A chest Radiograph of the 5-year-old girl showed 2 needle-like foreign bodies on the right side of the chest close to the spine and posterior chest wall. A CT scan showed that one needle was partly situated in the chest wall and partly in the lung. The other was completely embedded in lung tissue. The parents and grandmother had no idea what had happened. There were no other signs or findings consistent with non-accidental trauma. A chest Radiograph of the 2-year-old girl showed a needle-like foreign body in the left upper part of the chest near the great arteries. The parents had no idea what had happened. There were no other signs or findings consistent with non-accidental trauma. Only a tiny little red dot-like papule was seen in the left armpit.

7.4.3.2 Non-accidental Trauma outside the Home

Inci et al. evaluated the findings in 94 children (mean age 11.51 ± 3.31 years; male-to-female ratio 5.25 to 1), who, during a 6-year period, were treated with penetrating chest injuries [182]. Stab wounds were found in 45 children, high-velocity gunshot wounds in 27, low-velocity gunshot wounds in 13, bomb (shrapnel) injuries in 7, and a shotgun wound in 1 child. One child had a penetrating chest injury with cervical trachea injury, due to a horse bite. This child died after surgery. According to the authors, the high rate of stab wounds might be related to a large number of children per family, the low educational level of the children and their parents, and the bad relations between parents and their children in the region of Turkey, where the research was done.

Sometimes a penetrating trauma of the chest is not noticed by the patient. Massad et al. described a penetrating intracardiac injury in a 16-year-old boy who presented with a retained 14-cm segment of an ice pick that went unnoticed by the patient for 4 days [183]. The ice pick had lacerated the anterior papillary muscle of the left ventricle causing avulsion of its tip and prolapse of the anterior leaflet of the mitral valve resulting in severe mitral regurgitation. The boy reported that he was assaulted by members of a street gang in a street fight. He did not recall any weapons or sharp objects and went home after the incident. However, he continued to have back pain, worsening by movement and associated with feverishness. Finally, his mother brought him to the hospital.

7.4.3.3 Non-accidental Trauma in Conflicts and Wartime

Coley et al. reported a 5-year-old Afghan girl with a life-threatening penetrating chest trauma, caused by a 5 mm x 3 mm metallic fragment from an explosive device [177]. The girl also had a rib fracture, due to the penetrating trauma.

Darwish et al. described the findings in 256 Syrian children, aged 12.8 ± 5 years, with paediatric chest injuries over a period of 12 years [184]. In 97 children the chest injuries

were due to blunt force trauma in falls, road traffic accidents, and assaults. In 156 children the injuries were caused by a penetrating trauma, mainly due to shrapnel, bullets and stab wounds. The authors saw a significant increase in the incidence of paediatric chest injuries, following the outbreak of the Syrian crisis, with a predominance of penetrating trauma. Rib fractures were found in 7 of the 159 children with penetrating trauma.

7.5 Differential Diagnosis

In this section normal variants and anatomical anomalies which may resemble acute or healing rib fractures will be discussed. These findings are only described in single case reports.

Knapp et al. reported the findings in a 4-month-old boy, who was brought to the hospital by his mother with tactile fever and coughing for 12 days [185]. The boy appeared to be well hydrated, well nourished, and in no distress. He had a respiratory rate of 21 breaths per minute, a heart rate of 100 beats per minute, and a temperature of 37 °C rectally. The only positive finding on physical examination was copious rhinorrhoea. In the ED, the patient was found to have an oxygen saturation of 100% on room air, and a chest radiograph was obtained. Initially, the chest radiograph was interpreted as being consistent with posterior fractures of the left fifth, sixth, seventh and eighth ribs. A social services consultation was requested, and a child abuse report was initiated. However, subsequent second reading of the film differed. The areas previously thought to represent rib fractures were noted to be views of the sternal ossification centres superimposed on the ribs because the film was not a true anteroposterior view. The repeat chest radiograph was normal.

McAloon and O'Neill described the findings in a 10-month-old febrile boy who was discharged from the hospital after initial assessment that included a chest radiograph [186]. Five days later, the A & E consultant received a radiologist's report describing possible healing rib fractures. A second radiology opinion agreed that appearances suggested old fractures of ribs 7, 8 and possibly 9 on the right and advised a skeletal survey. A child abuse report was initiated. The skeletal survey, however, was reported as normal. The suggestive findings noted 5 days earlier were not apparent on the second chest radiograph. Oblique views confirmed no rib fractures. It was then clarified to the parents and social services that there was no child protection issue. The suggestive findings were sternum ossification centres projecting over the ribs (Fig. 7.34).

Pasquale-Styles et al. described the findings in a 2½-month-old girl, who was brought to the hospital because she was found unresponsive by her father [187]. A family

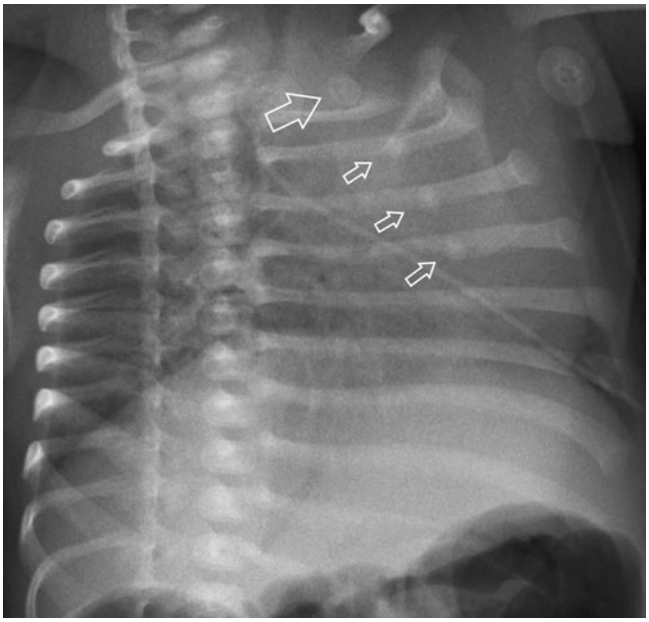


Fig. 7.34 Right oblique radiograph of the chest of a neonate, the case was referred for expert reading due to the presence of multiple rib fractures (small arrows). Closer inspection shows a sternal ossification centre cranial to the first left rib (large arrow). The other 'fractures' are in line and all are sternal ossification centres

member started CPR. A CT-scan of the head showed right tentorial subdural haematomas without associated mass effect and a small interhemispheric haematoma. She also had bilateral loss of grey-white differentiation in the cerebral hemispheres, which was interpreted as anoxic change. Fundoscopy showed multiple bilateral retinal haemorrhages. A skeletal survey on the second hospital day initially reported bilateral first rib fractures without associated scapular or clavicular fractures. The father was interrogated by the police and he admitted violently shaking his daughter out of frustration because of her crying, after which he had tossed her into a car seat. According to the father she was immediately unresponsive. The chest radiographs were repeated on day 14 and day 15. There was no change in the appearance of the apparent fracture sites seen at the anterolateral edges of the first ribs. There was no development of periosteal reaction or callus formation as would have been expected of a 2-week-old healing fracture. The girl died 24 days after being admitted to the hospital. Pre-autopsy full-body radiographs and autopsy examination revealed symmetric, bony nodules resembling calluses in the bilateral, anterolateral first ribs. These were consistent with pseudoarthrosis based on the lack of radiographic evidence of bone remodelling on the repeated radiographs 2 weeks later and again at the time of autopsy more than 3 weeks after injury, the lack of associated scapula and clavicle injuries that would correlate with direct trauma, and histologic

findings of abundant mature cartilage and fibrovascular tissue with minimal woven bone.

Bayramoglu et al. described a rare anatomical variant of the chest wall in a 15-year-old boy with a history of lymphoma [188]. During a radiological examination (positron-emission tomography/ computed tomography) bilateral multi-level posterior rib enlargements were found, which were initially identified as healing fractures. The authors, however, stated that the correct diagnosis would be multi-level posterior rib synchondroses with consecutive bridging. The authors concluded that this type of rib variant should be excluded in case of possible malignancies, possibility of fracture and suspected non-accidental trauma.

And finally, a variety of syndromal and non-syndromal rib anomalies may simulate rib fractures or healed rib fractures (Figs. 7.35, 7.36, 7.37, 7.38, and 7.39). Paediatric radiology expertise is necessary to recognize these costal variants. If these anomalies are not recognized, the subsequent skeletal survey will reveal clues for the proper diagnosis.

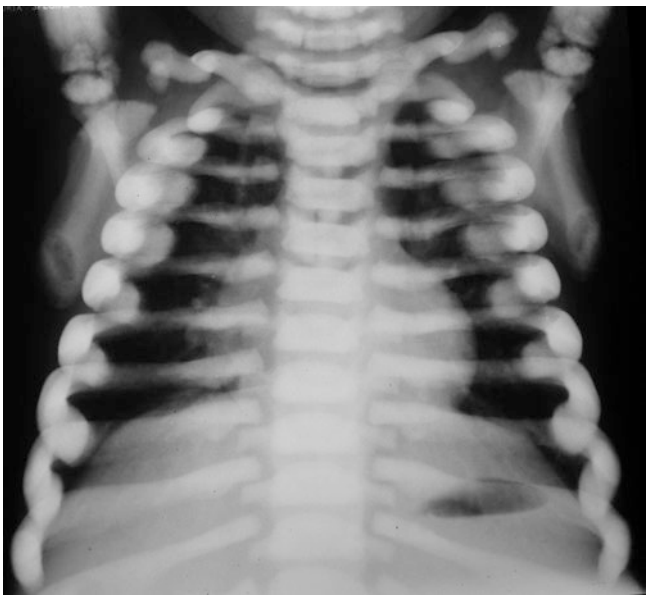


Fig. 7.36 Patient with infantile osteopetrosis simulating metaphyseal corner fractures of het humerus and extensive lateral and anterior rib fractures. All abnormalities are consistent with osteopetrosis

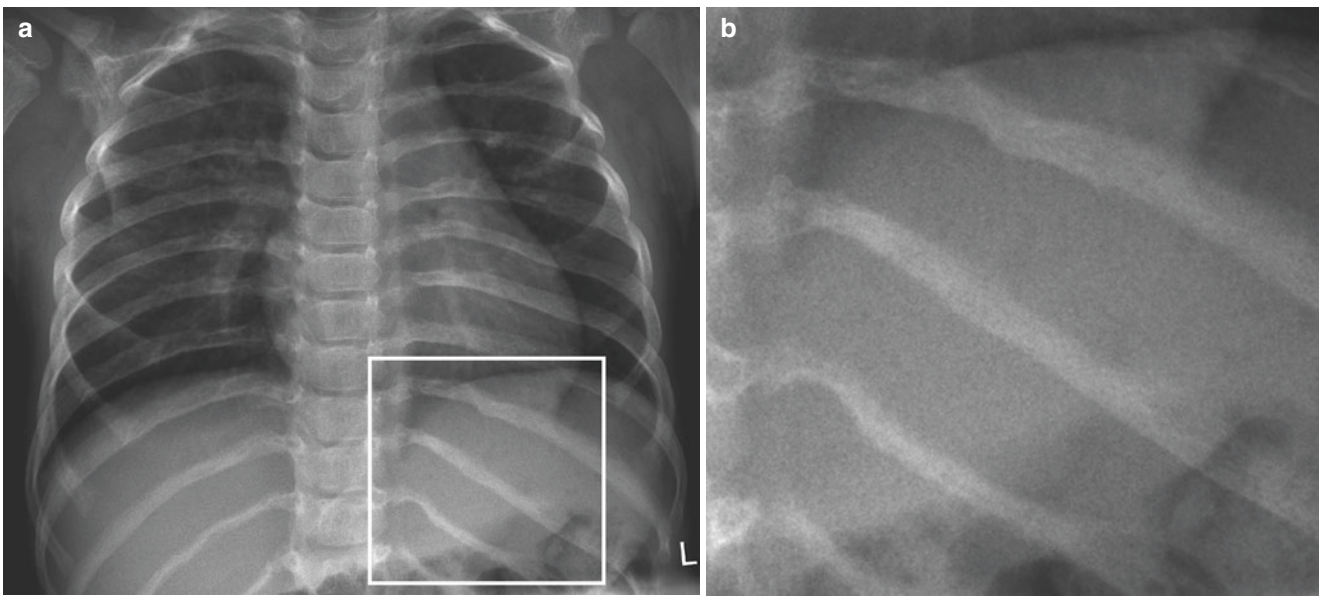


Fig. 7.35 This child had spine radiographs for thoracolumbar kyphosis. Abnormal ribs were identified and a chest radiograph (**a**) was performed showing irregular ribs with undulating contours suggestive of rib fractures (detail in **b**). A skeletal survey revealed skeletal abnormalities typical for Melnick Needles syndrome

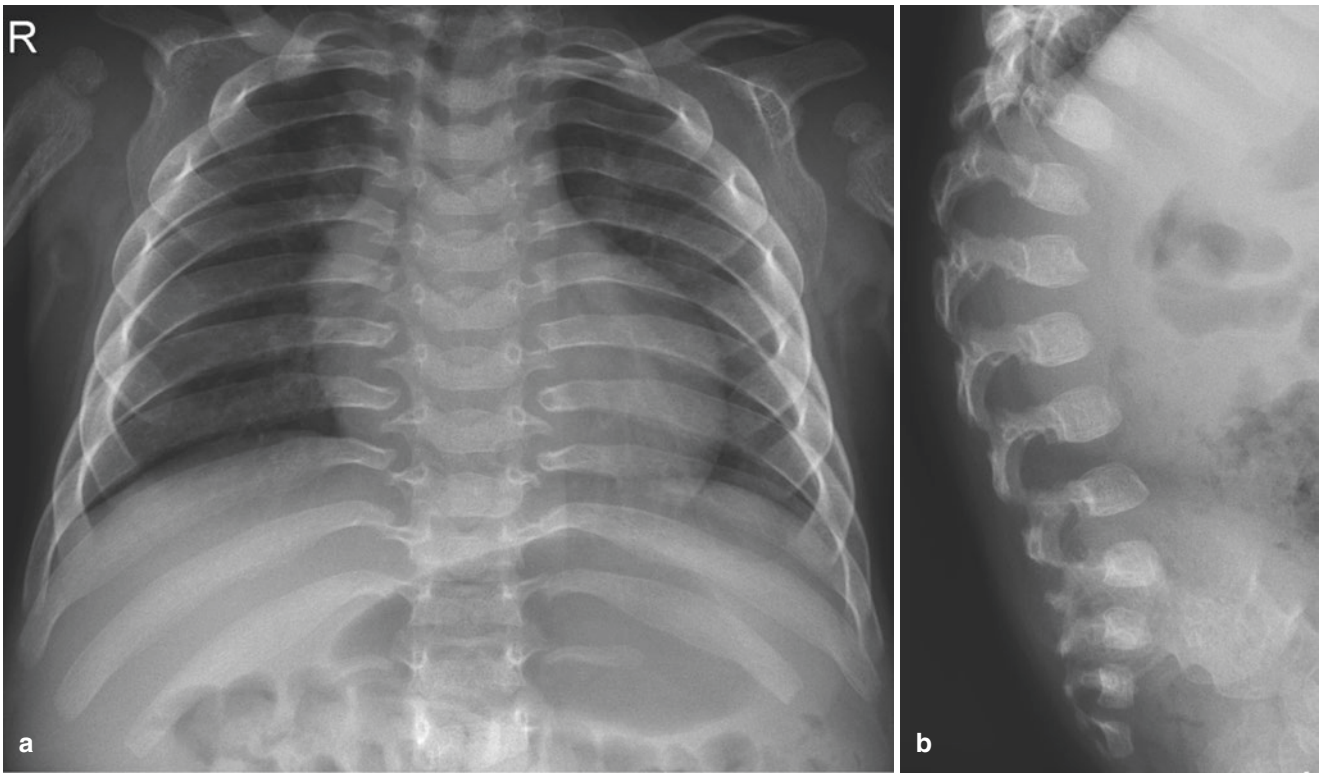


Fig. 7.37 Infant with thoracolumbar kyphosis and developmental delay. Radiographs reveal wide and slightly irregular ribs (**a**) and wedge-like deformities of the vertebral bodies of L1 and L2 (**b**). These findings resemble multiple healed rib fractures and impressed fractures of the lumbar spine but in fact are quite characteristic findings of dysostosis multiplex congenita. In this case, it was mucolipidosis type II

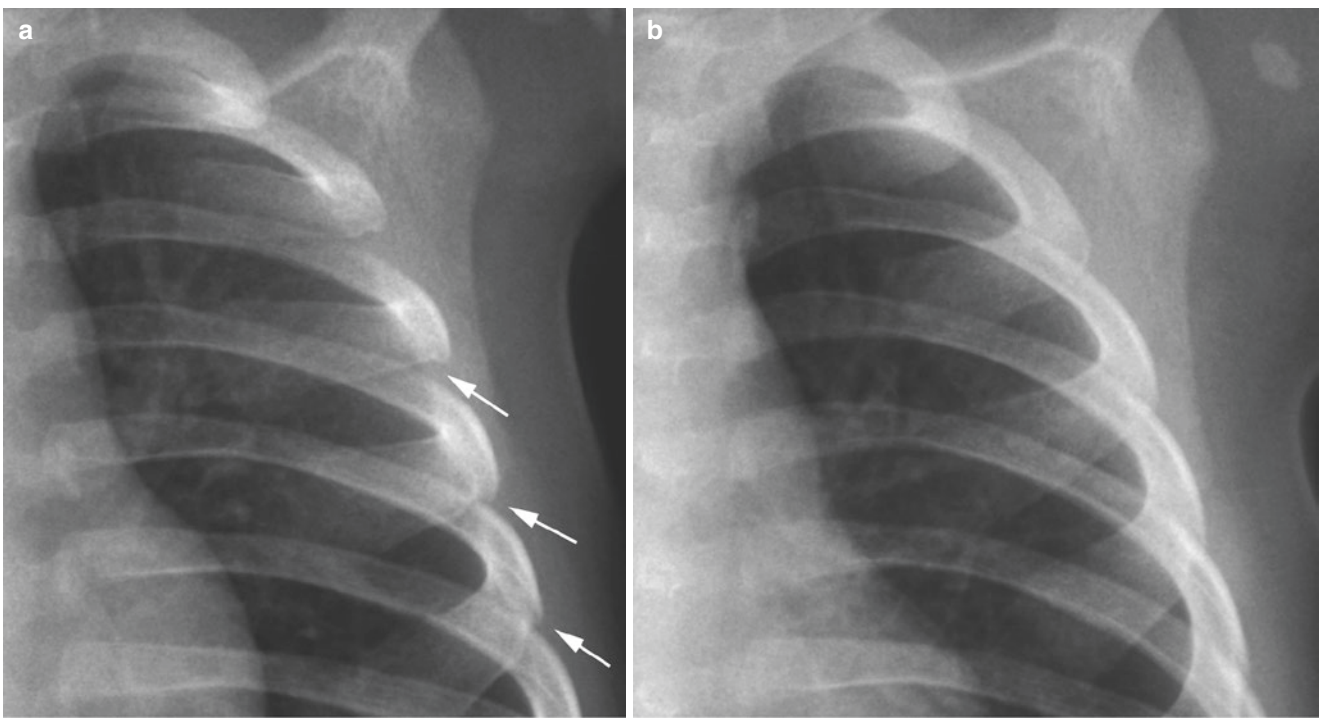


Fig. 7.38 (**a**) In a slightly lordotic view the subcostal grooves (housing the intercostal vessels) are visualized that might be interpreted as fractures (arrows). (**b**) A less lordotic view shows normal ribs

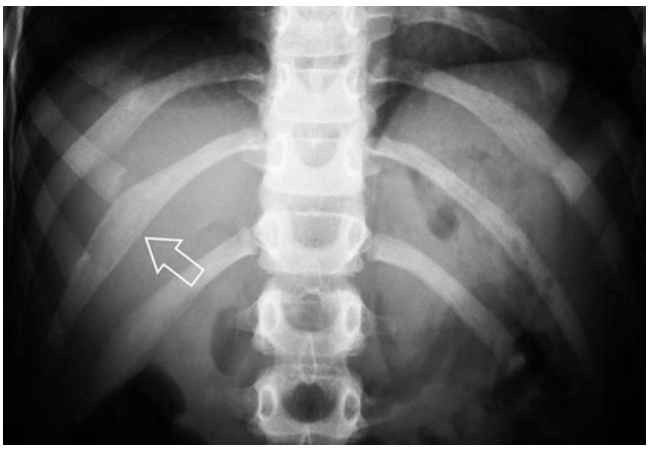


Fig. 7.39 Child with polyostotic fibrous dysplasia. Fusiform widening of the right 11th rib and ground glass appearance resembles a healed fracture (arrow). To a lesser extent also the tenth rib shows similar changes

References

1. Kuo K, Kim AM (2020) Rib Fracture. https://www.ncbi.nlm.nih.gov/books/NBK541020/. Accessed 02 Mar 2022
2. Love JC, Derrick SM, Wiersema JM, Pinto DC, Greeley C, Donaruma-Kwoh M, Bista B (2013) Novel classification system of rib fractures observed in infants. J Forensic Sci 58:330–335
3. Sinha CK, Lander A (2013) Trauma in children: abdomen and thorax. Surgery (Oxford) 31:123–129
4. Sharma MS (2016) Pediatric thoracic trauma. https://emedicine.medscape.com/article/905863-overview#showall. Accessed 02 Mar 2022
5. Worn MJ, Jones MD (2007) Rib fractures in infancy: establishing the mechanisms of cause from the injuries--a literature review. Med Sci Law 47:200–212
6. Bertelsen S, Howitz P (1972) Injuries of the trachea and bronchi. Thorax 27:188–194
7. Rasmussen OV, Brynitz S, Struve-Christensen E (1986) Thoracic injuries. A review of 93 cases. Scand J Thorac Cardiovasc Surg 20:71–74
8. Merten DF, Carpenter BL (1990) Radiologic imaging of inflicted injury in the child abuse syndrome. Pediatr Clin N Am 37:815–837
9. Kleinman PK, Marks SC Jr, Nimkin K, Rayder SM, Kessler SC (1996) Rib fractures in 31 abused infants: postmortem radiologic-histopathologic study. Radiology 200:807–810
10. Kleinman PK, Schlesinger AE (1997) Mechanical factors associated with posterior rib fractures: laboratory and case studies. Pediatr Radiol 27:87–91
11. Merten DF, Cooperman DR, Thompson GH (1992) Skeletal manifestations of child abuse. In: Reece RM (ed) Child abuse – medical diagnosis and management. Lea & Febiger, pp 23–53
12. Chapman S (1993) Recent advances in the radiology of child abuse. In: Hobbs CJ, Wynne JM (eds) Child abuse. Clinical Pediatrics, pp 211–234
13. Kleinman PK, Marks SC, Adams VI, Blackbourne BD (1988) Factors affecting visualization of posterior rib fractures in abused infants. AJR Am J Roentgenol 150:635–638
14. Kleinman PK, Marks SC, Spevak MR, Richmond JM (1992) Fractures of the rib head in abused infants. Radiology 185:119–123
15. 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
16. Chapman S (2004) Non-accidental injury. Imaging 16:161–173
17. Kleinman PK (2015) Diagnostic imaging of child abuse. Cambridge, Cambridge
18. Cameron JM, Rae LJ (1975) The radiological diagnosis. Differential diagnosis. In: Cameron JM, Rae LJ (eds) Atlas of the battered child syndrome. Churchill Livingstone, pp 20–50
19. Smith FW, Gilday DL, Ash JM, Green MD (1980) Unsuspected costo-vertebral fractures demonstrated by bone scanning in the child abuse syndrome. Pediatr Radiol 10:103–106
20. Kleinman PK (2015) Bony thoracic trauma. In: Kleinman PK (ed) Diagnostic imaging of child abuse. Cambridge University Press, pp 164–207
21. Bixby SD, Abo A, Kleinman PK (2011) High-impact trauma causing multiple posteromedial rib fractures in a child. Pediatr Emerg Care 27:218–219
22. Bradley AL, Swain MV, Neil Waddell J, Das R, Athens J, Kieser JA (2014) A comparison between rib fracture patterns in peri- and post-mortem compressive injury in a piglet model. J Mech Behav Biomed Mater 33:67–75
23. Blackburne WB (2015) Rib fractures in infants: retrospective survey of fractures and biomechanical study. Dunedin School of Medicine. University of Otago
24. Brodeur AE, Monteleone JA (1994) Child maltreatment. A clinical guide and reference. GW Medical:32
25. Van Mieghem T, Whittle WL, Farine D, Seaward G, D'Souza R (2013) Motor vehicle accidents in pregnancy: implications and management. J Obstet Gynaecol Can 35:303–304
26. Shapiro JR, Burn VE, Chipman SD, Jacobs JB, Schloo B, Reid L, Larsen N, Louis F (1989) Pulmonary hypoplasia and osteogenesis imperfecta type II with defective synthesis of alpha I(1) procollagen. Bone 10:165–171
27. Lee SH, Cho JY, Song MJ, Min JY, Han BH, Lee YH, Cho BJ, Kim SH (2002) Fetal musculoskeletal malformations with a poor outcome: ultrasonographic, pathologic, and radiographic findings. Korean J Radiol 3:113–124
28. Solovyov O, Goncharova YA, Zukin V (2010) Osteogenesis imperfecta, type II. https://sonoworld.com/TheFetus/page.aspx?id=2807. Accessed: 10 Aug 2021
29. Ayadi ID, Hamida EB, Rebeh RB, Chaouachi S, Marrakchi Z (2015) Perinatal lethal type II osteogenesis imperfecta: a case report. Pan Afr Med J 21:11
30. Buttiens A, Vanhoenacker FM, Van Hoywegen A, Y. Leroij (2017) Case 14505 Osteogenesis imperfecta type II. https://www.eurorad.org/case/14505. Accessed 02 Mar 2022
31. Doumit R, Tarabay H, Zafatayeff Hasbani S, El Helou N (2019) Imaging of prenatal fractures. https://epos.myesr.org/poster/esr/ecr2019/C-1529. Accessed 02 Mar 2022
32. Lauder I, Ellis HA, Ashcroft T, Burridge A (1976) Achondrogenesis type I. a familial subvariant? Arch Dis Child 51:550–557
33. Borochowitz Z, Lachman R, Adomian GE, Spear G, Jones K, Rimoin DL (1988) Achondrogenesis type I: delineation of further heterogeneity and identification of two distinct subgroups. J Pediatr 112:23–31
34. el Khazen N, Faverly D, Vamos E, Van Regemorter N, Flament-Durand J, Carton B, Cremer-Perlmutter N (1986) Lethal osteopetrosis with multiple fractures in utero. Am J Med Genet 23:811–819
35. Malinger G, Omoy A, El Shawwa R (1994) Osteopetrosis. https://sonoworld.com/TheFetus/page.aspx?id=366. Accessed: 10 Aug 2021
36. McClelland CQ, Heiple KG (1982) Fractures in the first year of life. A diagnostic dilemma. Am J Dis Child 136:26–29
37. Joseph PR, Rosenfeld W (1990) Clavicular fractures in neonates. Am J Dis Child 144:165–167
38. Hobbs CJ, Hanks HGI, Wynne JM (1993) Child abuse and neglect - a clinician's handbook. Churchill Livingstone

39. Bays J (2001) Conditions mistaken for child physical abuse. In: Reece RM, Ludwig S (eds) Child abuse, medical diagnosis and management. Lippincott Williams & Wilkins, pp 177–206
40. van Rijn RR, Bilo RA, Robben SG (2009) Birth-related mid-posterior rib fractures in neonates: a report of three cases (and a possible fourth case) and a review of the literature. Pediatr Radiol 39:30–34
41. Rubin A (1964) Birth injuries: incidence, mechanisms and end results. Obstet Gynecol 23:218–221
42. Camus M, Lefebvre G, Veron P, Darbois Y (1985) Traumatismes obstétricaux du nouveau-né. Enquête rétrospective à propos de 20409 naissances [Obstetrical injuries of the newborn infant. Retrospective study apropos of 20,409 births]. J GynecolObstetBiolReprod(Paris) 14:1033–1043
43. Bhat BV, Kumar A, Oumachigui A (1994) Bone injuries during delivery. Indian J Pediatr 61:401–405
44. Rizzolo PJ, Coleman PR (1989) Neonatal rib fracture: birth trauma or child abuse? J FamPract 29:561–563
45. Cumming WA (1979) Neonatal skeletal fractures. Birth trauma or child abuse? J Can Assoc Radiol 30:30–33
46. Bulloch B, Schubert CJ, Brophy PD, Johnson N, Reed MH, Shapiro RA (2000) Cause and clinical characteristics of rib fractures in infants. Pediatrics 105:E48
47. Durani Y, DePiero AD (2006) Images in emergency medicine. Fracture of left clavicle and left posterior rib due to birth trauma. Ann Emerg Med 47:210–215
48. Landman L, Homburg R, Sirota L, Dulizky F (1986) Ribfractures as a cause of immediate neonatal tachypnea. Eur J Pediatr 144:487–488
49. Pressley CM, Fry WR, Philp AS, Berry SD, Smith RS (2012) Predicting outcome of patients with chest wall injury. Am J Surg 204:910–913. discussion 913–914
50. Liebsch C, Seiffert T, Vlcek M, Beer M, Huber-Lang M, Wilke HJ (2019) Patterns of serial rib fractures after blunt chest trauma: an analysis of 380 cases. PLoS One 14:e0224105
51. Kessel B, Dagan J, Swaid F, Ashkenazi I, Olsha O, Peleg K, Givon A, Alfici R (2014) Rib fractures: comparison of associated injuries between pediatric and adult population. Am J Surg 208:831–834
52. Rosenberg G, Bryant AK, Davis KA, Schuster KM (2016) No breakpoint for mortality in pediatric rib fractures. J Trauma Acute Care Surg 80:427–432
53. Williams RL, Connolly PT (2004) In children undergoing chest radiography what is the specificity of rib fractures for non-accidental injury? Arch Dis Child 89:490–492
54. Merten DF, Radlowski MA, Leonidas JC (1983) The abused child: a radiological reappraisal. Radiology 146:377–381
55. Ichikawa Y, Sato A, Sato K, Nakamura K, Kitagawa N, Tanoue K, Shiro H (2015) Chylothorax associated with child abuse. Pediatr Int 57:1202–1204
56. Shelmerdine SC, Langan D, Hutchinson JC, Hickson M, Pawley K, Suich J, Palm L, Sebire NJ, Wade A, Arthurs OJ (2018) Chest radiographs versus CT for the detection of rib fractures in children (DRIFT): a diagnostic accuracy observational study. Lancet Child Adolesc Health 2:802–811
57. Magid N, Glass T (1990) A "hole in a rib" as a sign of child abuse. Pediatr Radiol 20:334–336
58. Blair L, Clauss E, Meredith M (2011) Child abuse: discovering the horrifying truth. JEMS 36:62–67; quiz 68
59. Wacks NP, Schoppel K, Sell PJ, Guggina T (2016) Opening Pandora's box: a chest radiograph in a 5-month-old with bronchiolitis. Hosp Pediatr 6:642–645
60. Worlock P, Stower M, Barbor P (1986) Patterns of fractures in accidental and non-accidental injury in children: a comparative study. BMJ 293:100–102
61. Garcia VF, Gotschall CS, Eichelberger MR, Bowman LM (1990) Rib fractures in children: a marker of severe trauma. J Trauma 30:695–700
62. Hedström EM, Svensson O, Bergström U, Michno P (2010) Epidemiology of fractures in children and adolescents. Acta Orthop 81:148–153
63. Darling SE, Done SL, Friedman SD, Feldman KW (2014) Frequency of intrathoracic injuries in children younger than 3 years with rib fractures. Pediatr Radiol 44:1230–1236
64. Skinner DL, den Hollander D, Laing GL, Rodseth RN, Muckart DJ (2015) Severe blunt thoracic trauma: differences between adults and children in a level I trauma Centre. South Afr Med J Suid-Afrikaanse tydskrif vir geneeskunde 105:47–51
65. Wegmann H, Orendi I, Singer G, Eberl R, Castellani C, Schalamon J, Till H (2016) The epidemiology of fractures in infants--Which accidents are preventable? Injury 47:188–191
66. Naqvi G, Johansson G, Yip G, Rehm A, Carrothers A, Stöhr K (2017) Mechanisms, patterns and outcomes of paediatric polytrauma in a UK major trauma Centre. Ann R Coll Surg Engl 99:39–45
67. Hagedorn KN, Johnston JH, Chinapuvvula NR, Beckmann NM, Cai C, Johnston SK (2019) Characterization of all-terrain vehicle-related chest injury patterns in children. Emerg Radiol 26:373–379
68. Ruest S, Kanaan G, Moore JL, Goldberg AP (2019) The prevalence of rib fractures incidentally identified by chest radiograph among infants and toddlers. J Pediatr 204:208–213
69. Ruest S, Kanaan G, Moore JL, Goldberg AP (2021) Pediatric rib fractures identified by chest radiograph: a comparison between accidental and nonaccidental trauma. Pediatr Emerg Care 37:e1409–e1415
70. Cooper A (1995) Thoracic injuries. Semin Pediatr Surg 4:109–115
71. Gutierrez IM, Ben-Ishay O, Mooney DP (2013) Pediatric thoracic and abdominal trauma. Minerva Chir 68:263–274
72. Schweich P, Fleisher G (1985) Rib fractures in children. Pediatr Emerg Care 1:187–189
73. Nakayama DK, Ramenofsky ML, Rowe MI (1989) Chest injuries in childhood. Ann Surg 210:770–775
74. Cohle SD, Hawley DA, Berg KK, Kiesel EL, Pless JE (1995) Homicidal cardiac lacerations in children. J Forensic Sci 40:212–218
75. Feldman KW, Brewer DK (1984) Child abuse, cardiopulmonary resuscitation, and rib fractures. Pediatrics 73:339–342
76. Thomas PS (1977) Rib fractures in infancy. Ann Radiol (Paris) 20:115–122
77. Paine CW, Fakeye O, Christian CW, Wood JN (2019) Prevalence of abuse among Young children with rib fractures: a systematic review. Pediatr Emerg Care 35:96–103
78. England SP, Sundberg S (1996) Management of common pediatric fractures. Pediatr Clin N Am 43:991–1012
79. Wootton-Gorges SL, Soares BP, Alazraki AL, Anupindi SA, Blount JP, Booth TN, Dempsey ME, Falcone RA Jr, Hayes LL, Kulkarni AV, Partap S, Rigsby CK, Ryan ME, Safdar NM, Trout AT, Widmann RF, Karmazyn BK, Palasis S (2017) ACR appropriateness criteria - suspected physical abuse-child. J Am College Radiol JACR 14:S338–s349
80. 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 54:1107–1110
81. Ricci LR (2004) Positive predictive value of rib fractures as an indicator of nonaccidental trauma in children. J Trauma 56:721; author reply 721-722
82. Kogutt MS, Swischuk LE, Fagan CJ (1974) Patterns of injury and significance of uncommon fractures in the battered child syndrome. Am J Roentgenol Radium Therapy, Nucl Med 121:143–149

83. Conway JJ, Collins M, Tanz RR (1993) The role of bone scintigraphy in detecting child abuse. Semin Nucl Med 23:321–333
84. Gipson CL, Tobias JD (2006) Flail chest in a neonate resulting from nonaccidental trauma. South Med J 99:536–538
85. Kriss S, Thompson A, Bertocci G, Currie M, Martich V (2020) Characteristics of rib fractures in young abused children. Pediatr Radiol 50:726–733
86. Mitchell IC, Norat BJ, Auerbach M, Bressler CJ, Como J, Escobar MA Jr, Flynn-O'Brien KT, Lindberg DM, Nickoles T, Rosado N, Weeks K, Maguire S (2020) Identifying maltreatment in infants and Young children presenting with fractures: does age matter? Acad Emerg Med *28*(1):5–18
87. Levitt CJ, Smith WL, Alexander RC (1992) Abusive head trauma. In: Reece RM (ed) Child abuse – medical diagnosis and management. Lea & Febiger, pp 1–23
88. Proisy M, Vivier PH, Morel B, Bruneau B, Sembely-Taveau C, Vacheresse S, Devillers A, Lecloirec J, Bodet-Milin C, Dubois M, Hamonic S, Bajeux E, Ganivet A, Adamsbaum C, Treguier C (2021) Whole-body MR imaging in suspected physical child abuse: comparison with skeletal survey and bone scintigraphy findings from the PEDIMA prospective multicentre study. Eur Radiol 31:8069
89. Strouse PJ, Owings CL (1995) Fractures of the first rib in child abuse. Radiology 197:763–765
90. Oestreich AE (1996) Cervical rib simulating fracture of the first rib in suspected child abuse. Radiology 199:582
91. Melville JD, Lukefahr JL, Clarke EA (2012) First rib fractures in abused infants: a report of three cases. Clin Pediatr (Phila) 51:426–430
92. Hamilton NA, Bucher BT, Keller MS (2011) The significance of first rib fractures in children. J Pediatr Surg 46:169–172
93. Funakoshi T, Furushima K, Kusano H, Itoh Y, Miyamoto A, Horiuchi Y, Sugawara M, Itoh Y (2019) First-rib stress fracture in overhead throwing athletes. J Bone Joint Surg Am 101:896–903
94. Lee SJ, Yie K, Chon SB (2012) Juvenile first rib fracture caused by morning stretching. J Emerg Med 43:e119–e121
95. Maguire S, Mann M, John N, Ellaway B, Sibert JR, Kemp AM (2006) Does cardiopulmonary resuscitation cause rib fractures in children? A systematic review. Child Abuse Negl 30:739–751
96. Smeets AJ, Robben SG, Meradji M (1990) Sonographically detected costo-chondral dislocation in an abused child. A new sonographic sign to the radiological spectrum of child abuse. Pediatr Radiol 20:566–567
97. Ng CS, Hall CM (1998) Costochondral junction fractures and intra-abdominal trauma in non-accidental injury (child abuse). Pediatr Radiol 28:671–676
98. Nimkin K, Kleinman PK (1997) Imaging of child abuse. Pediatr Clin N Am 44:615–635
99. Weber MA, Risdon RA, Offiah AC, Malone M, Sebire NJ (2009) Rib fractures identified at post-mortem examination in sudden unexpected deaths in infancy (SUDI). Forensic Sci Int 189:75–81
100. Boos SC (2000) Constrictive asphyxia: a recognizable form of fatal child abuse. Child Abuse Negl 24:1503–1507
101. Gunther WM, Symes SA, Berryman HE (2000) Characteristics of child abuse by anteroposterior manual compression versus cardiopulmonary resuscitation: case reports. Am J Forensic Med Pathol 21:5–10
102. Lauridson JR, Cromblin KL (2015) Prolonged abusive chest compressions in an infant: an occult form of child abuse: review of hemodynamics. Am J Forensic Med Pathol 36:274–275
103. Vester MEM, Bilo RAC, Nijs HGT, van Rijn RR (2018) Pediatric constrictive asphyxia a rare form of child abuse: a report of two cases. Forensic Sci Int 285:e17–e20
104. Olds K, Byard RW, Langlois NE (2015) Injuries associated with resuscitation - an overview. J Forensic Legal Med 33:39–43
105. Price EA, Rush LR, Perper JA, Bell MD (2000) Cardiopulmonary resuscitation-related injuries and homicidal blunt abdominal trauma in children. Am J Forensic Med Pathol 21:307–310
106. Røed U, Lilleng RK, Mæhle BO, Morild I (2008) Rib fractures and cardiopulmonary resuscitation in small children. Scand J For Sci 14:17–20
107. Matshes EW, Lew EO (2010) Do resuscitation-related injuries kill infants and children? Am J Forensic Med Pathol 31:178–185
108. Kaplan JA, Fossum RM (1994) Patterns of facial resuscitation injury in infancy. Am J Forensic Med Pathol 15:187–191
109. Niermeyer S, Kattwinkel J, Van Reempts P, Nadkarni V, Phillips B, Zideman D, Azzopardi D, Berg R, Boyle D, Boyle R, Burchfield D, Carlo W, Chameides L, Denson S, Fallat M, Gerardi M, Gunn A, Hazinski MF, Keenan W, Knaebel S, Milner A, Perlman J, Saugstad OD, Schleien C, Solimano A, Speer M, Toce S, Wiswell T, Zaritsky A (2000) International guidelines for neonatal resuscitation: an excerpt from the guidelines 2000 for cardiopulmonary resuscitation and emergency cardiovascular care: international consensus on science. Contributors and reviewers for the neonatal resuscitation guidelines. Pediatrics 106:E29
110. International Liaison Committee on Resuscitation (2006) The international liaison committee on resuscitation (ILCOR) consensus on science with treatment recommendations for pediatric and neonatal patients: pediatric basic and advanced life support. Pediatrics 117:e955–e977
111. Franke I, Pingen A, Schiffmann H, Vogel M, Vlajnic D, Ganschow R, Born M (2014) Cardiopulmonary resuscitation (CPR)-related posterior rib fractures in neonates and infants following recommended changes in CPR techniques. Child Abuse Negl 38:1267–1274
112. Spevak MR, Kleinman PK, Belanger PL, Richmond JM (1994) Cardiopulmonary resuscitation and rib fractures in infants. A postmortem radiologic-pathologic study. JAMA 272:617–618
113. Betz P, Liebhardt E (1994) Rib fractures in children - resuscitation or child abuse? Int J Legal Med 106:215–218
114. Bush CM, Jones JS, Cohle SD, Johnson H (1996) Pediatric injuries from cardiopulmonary resuscitation. Ann Emerg Med 28:40–44
115. Ryan MP, Young SJ, Wells DL (2003) Do resuscitation attempts in children who die, cause injury? Emerg Med J 20:10–12
116. Hoke RS, Chamberlain D (2004) Skeletal chest injuries secondary to cardiopulmonary resuscitation. Resuscitation 63:327–338
117. Dolinak D (2007) Rib fractures in infants due to cardiopulmonary resuscitation efforts. Am J Forensic Med Pathol 28:107–110
118. Matshes EW, Lew EO (2010) Two-handed cardiopulmonary resuscitation can cause rib fractures in infants. Am J Forensic Med Pathol 31:303
119. Reyes JA, Somers GR, Taylor GP, Chiasson DA (2011) Increased incidence of CPR-related rib fractures in infants--is it related to changes in CPR technique? Resuscitation 82:545–548
120. Cosway B, Mathura N, Mott A, Bredow M, Fraser J, Rawlinson A, Wei C, Thyagarajan MS, Harrison S, Kemp A (2015) Occult rib fractures: defining the cause. Child Abuse Rev 24:6–15
121. Ondruschka B, Baier C, Siekmeyer M, Buschmann C, Dreßler J, Bernhard M (2017) Cardiopulmonary resuscitation-associated injuries in still−/newborns, infants and toddlers in a German forensic collective. Forensic Sci Int 279:235–240
122. Clouse JR, Lantz PE (2008) Posterior rib fractures in infants associated with cardiopulmonary resuscitation. American Academy of forensic sciences, 60 th annual meeting
123. Love JC, Austin D, Giese KW, Roe SJ (2022) Cardiopulmonary resuscitation induced posterior rib fractures in nontraumatic pediatric deaths. Am J Forensic Med Pathol 43:55–59
124. Sewell RD, Steinberg MA (2000) Chest compressions in an infant with osteogenesis imperfecta type II: no new rib fractures. Pediatrics 106:E71

125. Plunkett J (2006) Resuscitation injuries complicating the interpretation of premortem trauma and natural disease in children. J Forensic Sci 51:127–130
126. Shkrum MJ, Ramsay DA (2007) Asphyxia. Forensic pathology of trauma - common problems for the pathologist. Humana Press, pp 65–180
127. Purohit DM, Caldwell C, Levkoff AH (1975) Letter: multiple rib fractures due to physiotherapy in a neonate with hyaline membrane disease. Am J Dis Child 129:1103–1104
128. Chalumeau M, Foix-L'Helias L, Scheinmann P, Zuani P, Gendrel D, Ducou-le-Pointe H (2002) Rib fractures after chest physiotherapy for bronchiolitis or pneumonia in infants. Pediatr Radiol 32:644–647
129. Gorincour G, Dubus JC, Petit P, Bourliere-Najean B, Devred P (2004) Rib periosteal reaction: did you think about chest physical therapy? Arch Dis Child 89:1078–1079
130. Chanelière C, Moreux N, Pracros JP, Bellon G, Reix P (2006) Fractures costales au cours des bronchiolites aiguës virales: à propos de 2 cas [Rib fractures after chest physiotherapy: a report of 2 cases]. Archives de pediatrie : organe officiel de la Societe francaise de pediatrie 13:1410–1412
131. Wilson PM, Greiner MV, Duma EM (2012) Posterior rib fractures in a young infant who received chiropractic care. Pediatrics 130:e1359–e1362
132. Helfer RE, Scheurer SL, Alexander R, Reed J, Slovis TL (1984) Trauma to the bones of small infants from passive exercise: a factor in the etiology of child abuse. J Pediatr 104:47–50
133. Keipert JA (1970) Rickets with multiple fractures in a premature infant. Med J Aust 1:672–675
134. Geggel RL, Pereira GR, Spackman TJ (1978) Fractured ribs: unusual presentation of rickets in premature infants. J Pediatr 93:680–682
135. Dabezies EJ, Warren PD (1997) Fractures in very low birth weight infants with rickets. Clin Orthop Relat Res 335:233–239
136. Smurthwaite D, Wright N, Russell S, Emmerson A, Mughal Z (2008) How common are rib fractures in extremely low birth weight preterm infants? Arch Dis Child Fetal Neonatal Ed Online first 6 August 2008
137. Lucas-Herald A, Butler S, Mactier H, McDevitt H, Young D, Ahmed SF (2012) Prevalence and characteristics of rib fractures in ex-preterm infants. Pediatrics 130:1116–1119
138. Wei C, Stevens J, Harrison S, Mott A, Warner J (2012) Fractures in a tertiary neonatal intensive care unit in Wales. Acta Paediatr (Oslo, Norway : 1992) 101:587–590
139. Cardenas N, Manrique TA, Catlin EA (1988) Flail chest in the newborn. A complication of osteogenesis imperfecta. Clin Pediatr (Phila) 27:161–162
140. Greeley CS, Donaruma-Kwoh M, Vettimattam M, Lobo C, Williard C, Mazur L (2013) Fractures at diagnosis in infants and children with osteogenesis imperfecta. J Pediatr Orthop 33:32–36
141. Pereira EM (2015) Clinical perspectives on osteogenesis imperfecta versus non-accidental injury. Am J Med Genet C Semin Med Genet 169:302–306
142. Chapman T, Sugar N, Done S, Marasigan J, Wambold N, Feldman K (2010) Fractures in infants and toddlers with rickets. Pediatr Radiol 40:1184–1189
143. Droms RJ, Rork JF, McLean R, Martin M, Belazarian L, Wiss K (2017) Menkes disease mimicking child abuse. Pediatr Dermatol 34:e132–e134
144. Cronin H, Fussell JN, Pride H, Bellino P (2012) Menkes syndrome presenting as possible child abuse. Cutis 90:170–172
145. Kim D, Choi J, Han KM, Lee BH, Choi JH, Yoo HW, Han YM (2015) Impaired osteogenesis in Menkes disease-derived induced pluripotent stem cells. Stem Cell Res Ther 6:160
146. Chapman S (1987) Child abuse or copper deficiency? A radiological view. Br Med J (Clinical research ed) 294:1370
147. Nyweide K, Feldman KW, Gunther DF, Done S, Lewis C, Van EC (2006) Hypocalciuric hypercalcemia presenting as neonatal rib fractures: a newly described mutation of the calcium-sensing receptor gene. Pediatr Emerg Care 22:722–724
148. Kaplan W, Haymond MW, McKay S, Karaviti LP (2006) Osteopenic effects of MgSO4 in multiple pregnancies. J Pediatr Endocrinol Metab 19:1225–1230
149. Bilo RAC, Robben SGF, van Rijn RR (2010) Rib fractures from other rare causes. In: Bilo RAC, Robben SGF, van Rijn RR (eds) Forensic aspects of paediatric fractures. Springer, pp 64–65
150. Robinson RF, Casavant MJ, Nahata MC, Mahan JD (2004) Metabolic bone disease after chronic antacid administration in an infant. Ann Pharmacother 38:265–268
151. National Organisation for Rare Disorders (NORD) (2018) Osteopetrosis. https://rarediseases.org/rare-diseases/osteopetrosis/. Accessed 02 Mar 2022
152. Bodamer OA, Bravermann RM, Craigen WJ (2001) Multiple fractures in a 3-month-old infant with severe infantile osteopetrosis. J Paediatr Child Health 37:520–522
153. Waguespack SG, Hui SL, Dimeglio LA, Econs MJ (2007) Autosomal dominant osteopetrosis: clinical severity and natural history of 94 subjects with a chloride channel 7 gene mutation. J Clin Endocrinol Metab 92:771–778
154. Matrane A, El Issami S, Bsiss MA (2016) Maladie des os de marbre : intérêt de l'imagerie hybride tomographie d'émission monophotonique/tomodensitométrie [Marble bone disease: The role of SPECT/CT hybrid imaging]. Archives de pediatrie : organe officiel de la Societe francaise de pediatrie 23:714–718
155. Connolly LP, Connolly SA (2004) Rib stress fractures. Clin Nucl Med 29:614–616
156. Sano A, Tashiro K, Fukuda T (2015) Cough-induced rib fractures. Asian Cardiovasc Thorac Ann 23:958–960
157. Mitchell JP (1951) Cough fracture. Br Med J 2:1492–1493
158. De Maeseneer M, De Mey J, Debaere C, Meysman M, Osteaux M (2000) Rib fractures induced by coughing: an unusual cause of acute chest pain. Am J Emerg Med 18:194–197
159. Baitner AC, Bernstein AD, Jazrawi AJ, Della Valle CJ, Jazrawi LM (2000) Spontaneous rib fracture during pregnancy. A case report and review of the literature. Bull Hosp Jt Dis 59:163–165
160. Cohen RC (1949) Cough fracture of ribs. Br Med J 1:133–135
161. Roberge RJ, Morgenstern MJ, Osborn H (1984) Cough fracture of the ribs. Am J Emerg Med 2:513–517
162. Oren V, Kozenitzky I, Babiacki A, Stern A (1988) Unusual cough related stress injuries. Eur J Nucl Med 14:108–111
163. Begley A, Wilson DS, Shaw J (1995) Cough fracture of the first rib. Injury 26:565–566
164. Kawahara H, Baba H, Wada M, Azuchi M, Ando M, Imura S (1997) Multiple rib fractures associated with severe coughing--a case report. Int Orthop 21:279–281
165. Litch JA, Tuggy M (1998) Cough induced stress fracture and arthropathy of the ribs at extreme altitude. Int J Sports Med 19:220–222
166. Hanak V, Hartman TE, Ryu JH (2005) Cough-induced rib fractures. Mayo Clinic Proc 80:879–882
167. American Academy of Pediatrics (AAP) (2018) AAP Red book online. Section 3. Summaries of infectious diseases: pertussis (whooping cough). http://redbook.solutions.aap.org/chapter.aspx?sectionid=189640156 & bookid=2205. Accessed 02 Mar 2022
168. Havers FP, Moro, P.L., Hariri S, Skoff T (2019) Chapter 16 Pertussis. Pink Book – epidemiology and prevention of vaccine-preventable diseases. https://www.cdc.gov/vaccines/pubs/pinkbook/pert.html. Accessed 23 Feb 2022

169. Prasad S, Baur LA (2001) Fracture of the first rib as a consequence of pertussis infection. J Paediatr Child Health 37:91–93
170. Rothstein E, Edwards K (2005) Health burden of pertussis in adolescents and adults. Pediatr Infect Dis J 24:S44–S47
171. Cortese MM, Baughman AL, Brown K, Srivastava P (2007) A "new age" in pertussis prevention new opportunities through adult vaccination. Am J Prev Med 32:177–185
172. McNaughten B, Thompson A, O'Donoghue D (2018) An unusual cause of chest pain in a teenage girl. Arch Dis Child Educ Pract Ed 103:152–154
173. Allen M, Aziz M (2014) Unexplained multiple rib fractures in a hospitalised child. Lessons from a serious case. Arch Dis Child 99:A175
174. van As AB, Manganyi R, Brooks A (2013) Treatment of thoracic trauma in children: literature review, red cross war memorial Children's hospital data analysis, and guidelines for management. Eur J Pediatr Surg 23:434–443
175. Murphy GK (1985) A single fatal penetrating chest wound from shattered wind-blown glass. Am J Forensic Med Pathol 6:332–335
176. Boleken ME, Cevik M, Yagiz B, Ter M, Dorterler ME, Aksoy TR (2013) The characteristics and outcomes of penetrating thoracic and abdominal trauma among children. Pediatr Surg Int 29:795–800
177. Coley E, Roach P, Macmillan AI, West AT, Johnston AM (2011) Penetrating paediatric thoracic injury. J R Army Med Corps 157:243–245
178. Cotton BA, Nance ML (2004) Penetrating trauma in children. Semin Pediatr Surg 13:87–97
179. Sola JE, Cateriano JH, Thompson WR, Neville HL (2008) Pediatric penetrating cardiac injury from abuse: a case report. Pediatr Surg Int 24:495–497
180. Choudhary S, Pujar Venkateshacharya S, Reddy C (2016) Sewing needle: a rare cause of intra-cardiac foreign body in a 3-year-old child. Cardiol Young 26:1425–1427
181. Deng X, Huang P, Wang J, Yi L, Liu J, Yang G (2019) Sewing needles in the lungs of children: two case reports. Medicine (Baltimore) 98:e15186
182. Inci I, Ozcelik C, Nizam O, Eren N, Ozgen G (1996) Penetrating chest injuries in children: a review of 94 cases. J Pediatr Surg 31:673–676
183. Massad MG, Khoury F, Evans A, Sirois C, Chaer R, Thomas Y, Snow NJ, Briller J, Geha AS (2002) Late presentation of retained intracardiac ice pick with papillary muscle injury. Ann Thorac Surg 73:1623–1626
184. Darwish B, Mahfouz MZ, Al-Nosairat S, Izzat MB (2018) Changing pattern and outcome of pediatric chest injuries in urban Syria. Asian Cardiovasc Thorac Ann 26:367–370
185. Knapp JF, Seidel JS, Schremmer R (2002) A 4-month-old infant with fever and cough. Pediatr Emerg Care 18:314–315
186. McAloon J, O'Neill C (2011) Ossification centres, not rib fractures. Arch Dis Child 96:284
187. Pasquale-Styles MA, Crowder CM, Fridie J, Milla SS (2014) Bilateral first rib anomalous articulations with pseudarthroses mimicking healing fractures in an infant with abusive head injury. J Forensic Sci 59:1668–1671
188. Bayramoglu Z, Yilmaz R, Caliskan E, Buyuksahin G, Bulut F, Aliyev S, Adaletli I (2018) A confounding rib variation: bilateral symmetric aberrant posterior rib articulations and bridgings. Surg Radiol Anat 40:63–65
189. Barry PW, Hocking MD (1993) Infant rib fracture--birth trauma or non-accidental injury. Arch Dis Child 68:250
190. Hartmann RW Jr (1997) Radiological case of the month. Rib fractures produced by birth trauma. Arch Pediatr AdolescMed 151:947–948
191. Ibanez G, Mora ND, Gado Rioja MA, del Herrera RC (2003) Fracturas costales obstétricas aisladas [isolated obstetric costal fractures]. An Pediatr (Barc) 58:612
192. Jovanović N, Ristovska N, Bogdanović Z, Petronijević M, Opalić J, Plećaš D (2013) Diagnosis and treatment of rib fracture during spontaneous vaginal delivery. Srp Arh Celok Lek 141:528–531
193. Khan NA, Lam V, Rickett A, Dickinson F (2016) Unforeseen rib fracture findings in infant chest radiographs: evidence of non-accidental injury or simply a case of birth trauma? BMJ case reports 2016
194. Pandya NK, Baldwin K, Wolfgruber H, Christian CW, Drummond DS, Hosalkar HS (2009) Child abuse and orthopaedic injury patterns: analysis at a level I pediatric trauma center. J Pediatr Orthop 29:618–625
195. Caplan MJ, Catanese CA (2010) Pediatric forensic pathology. In: Catanese CA (ed) Color atlas of forensic medicine and pathology. CRC Press, pp 147–188
196. Riggle A, Bollins J, Konda S, Aggarwal R, Beiswenger A (2010) Penetrating pediatric trauma owing to improper child safety seat use. J Pediatr Surg 45:245–248
197. Gettig K, Lawson KA, Garcia NM, Fox KA (2015) Penetrating knitting needle through the mediastinum in a child. J Trauma Nurs 22:132–135
198. Kulaylat AN, Chesnut CH 3rd, Patel S, Rocourt DV, Clark JB (2016) Penetrating cardiac nail gun injury in a child. Pediatr Emerg Care 32:536–537
199. O'Neill PJ, Sinha M, McArthur RA, Frechette A (2008) Penetrating cactus spine injury to the mediastinum of a child. J Pediatr Surg 43:e33–e35
200. Papadopoulos G, Kouerinis IA, Giannakopoulou A, Eleftherakis NG, Andreou N, Azariades MA (2010) Tropical plant needle causing recurrent cardiac tamponade in a pediatric patient. J Trauma 69:E35
201. Ramaswamy R, Dow G, Bassi S (2006) Pencil is mightier than the sword! Pediatr Neurosurg 42:168–170
202. Fisher SB, Clifton MS, Bhatia AM (2011) Pencils and pens: an under-recognized source of penetrating injuries in children. Am Surg 77:1076–1080
203. Malla G, Basnet B, Vohra R, Herrforth C, Adhikari S, Bhandari A (2014) Thoraco- abdominal impalement injury: a case report. BMC Emerg Med 14:7
204. Bawany FI, Khan MS, Khan A, Dar M (2013) Successful surgery of massive thoracic injury in a girl following chest trauma. JPMA The Journal of the Pakistan Medical Association 63:1571–1573
205. Linard C, Marques P, Bezon E, Delaperriere N, Germouty I, Fenoll B, de Vries P (2010) Corps étranger péricardique: une cause inhabituelle de douleur thoracique chez l'enfant [Pericardial foreign body: an unusual cause of chest pain in children]. Archives de pediatrie : organe officiel de la Societe francaise de pediatrie 17:1682–1684
206. McLaughlin RL, Analitis S, VanVleet S, Pederson R (2008) Right ventricular gunshot wound with retrograde embolization. J Trauma Nurs 15:123–125
207. DeCou JM, Abrams RS, Miller RS, Touloukian RJ, Gauderer MW (2000) Life-threatening air rifle injuries to the heart in three boys. J Pediatr Surg 35:785–787
208. Greenlees G, Govewalla P, Haqzad Y, Sharkey A, Cartwright N (2019) Penetration of the heart by an Airgun pellet: a case without significant effusion or Valvular injury. Ann Thorac Surg 108:e9–e10

Clavicle

8

Rob A. C. Bilo, Rick R. van Rijn,
Ingrid M. B. Russel-Kampschoer, and Simon G. F. Robben

Contents

8.1 General Aspects of Clavicle Fractures

The clavicle is situated at the front- and upper side of the ribcage, between the shoulder girdle and the sternum. For the greater part its course is clearly visible, just underneath the skin, and easily palpable along its full length.

Fractures of the clavicle are among the most frequently diagnosed fractures, in children as well as in adults. In children it is one of the most common fractures with an estimated incidence of 5–15% of childhood fractures [1, 2].

Depending on their location, fractures of the clavicle are categorized as: fractures of the middle third (mid-shaft) (Allman Group 1 fractures), the lateral or distal third (Allman Group 2 fractures), and the proximal or sternal third (Allman Group 3 fractures) (Fig. 8.1) [3]. Of all clavicle fractures, in children and adults, 76–85% are located in the middle third (shaft), 10–21% in the distal, and 3–5% in the proximal part [4–8]. In order to guide treatment, clavicle fractures of the lateral third of the clavicle can be classified according to the Neer classification (Fig. 8.2) [9, 10].

Young, non-mobile children with a fracture of the clavicle hardly ever show noticeable signs and symptoms. Sometimes the child moves the arm less on the affected side or cries during day-to-day care. More often it is found by chance because during day-to-day care the parents or during physical examination a physician notices a swelling on the clavicle due to new bone formation after 7–10 days (Figs. 8.3 and 8.4).

R. A. C. Bilo
Veilig Thuis Rotterdam Rijnmond (Center for the Reporting of Child Abuse, Domestic Violence and Elder Abuse), Rotterdam, The Netherlands

R. R. van Rijn
Department of Radiology and Nuclear Medicine, Amsterdam UMC, University of Amsterdam, Amsterdam, The Netherlands

Department of Forensic Medicine, Netherlands Forensic Institute, The Hague, The Netherlands

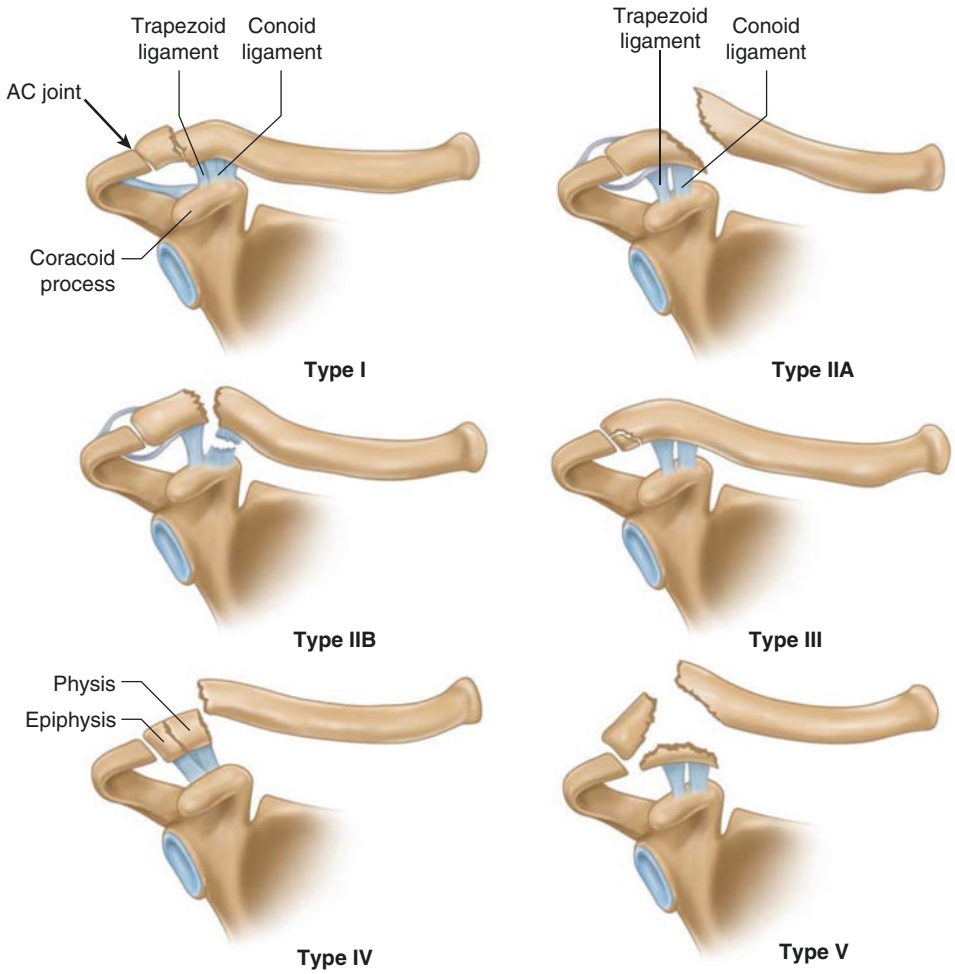
I. M. B. Russel-Kampschoer
Department of Paediatrics, University Medical Center Utrecht, Utrecht, The Netherlands

S. G. F. Robben (✉)
Department of Radiology, Maastricht University Medical Center, Maastricht, The Netherlands
e-mail: s.robben@mumc.nl

R. A. C. Bilo et al. (eds.), *Forensic Aspects of Paediatric Fractures*, https://doi.org/10.1007/978-3-031-12041-1_8

Fig. 8.1 Graphic representation of fractures of the clavicle. (**a**) Proximal; (**b**) mid-shaft; (**c**) distal

Fig. 8.2 Neer classification of clavicular fractures



It is easier to recognize signs and symptoms in older children. Children complain of moderate to severe pain around the fracture. They will stop using the affected side to relieve the pain caused by the fracture (i.e. they stop moving the arm). Most children with this type of fracture are inclined to 'look' towards the side of the fracture in order to relax the sternocleidomastoid muscle to avoid this muscle pulling at the broken bone [11].

Sometimes there is pain when pressure is exerted around the fracture. A haematoma or fluid collection may be visible near the fracture. There may be external evidence that the bone has lost its integrity, or one of the bony ends may protrude through the skin (a so-called compound fracture). In rare cases, reported in literature in up to 0.1–2% of all cases, the fracture will not heal and a pseudoarthrosis will form (Fig. 8.5a, b) [12, 13].

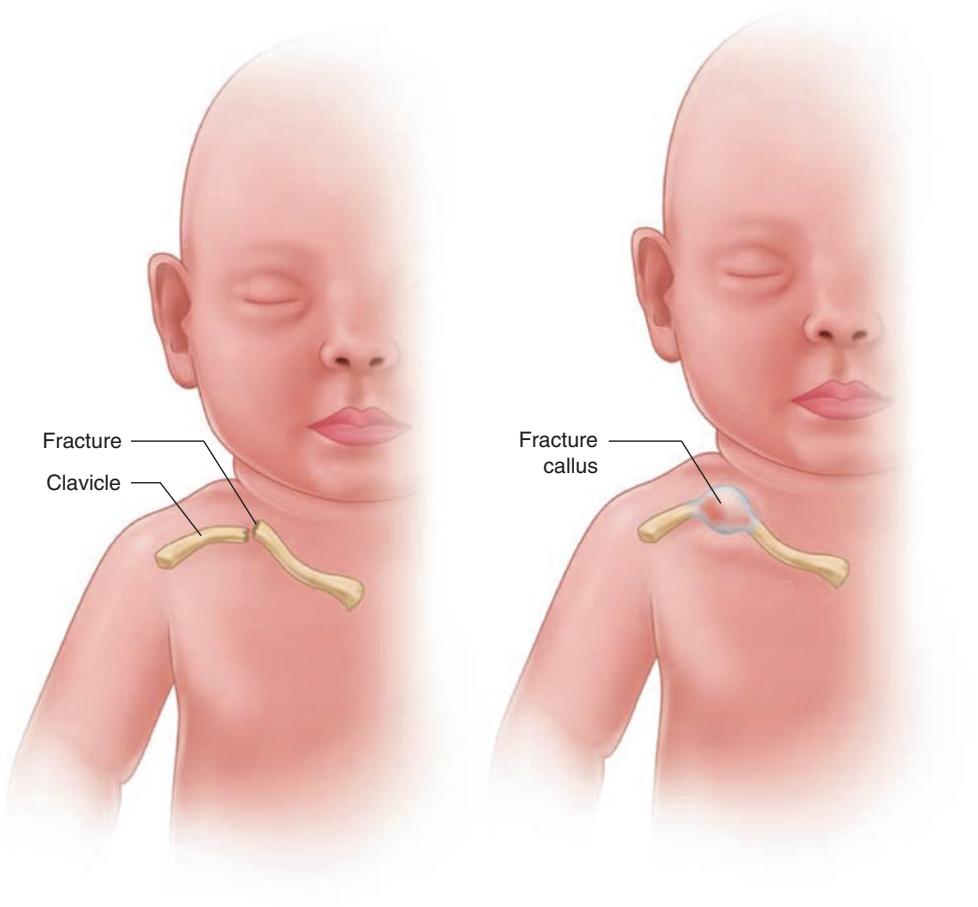


Fig. 8.3 Clavicle fracture in a young child, leading to callus formation after 7–10 days. On physical examination a lump is visible over the clavicle

Fig. 8.4 Healing clavicle fracture in a 6-week-old neonate resulting from birth trauma

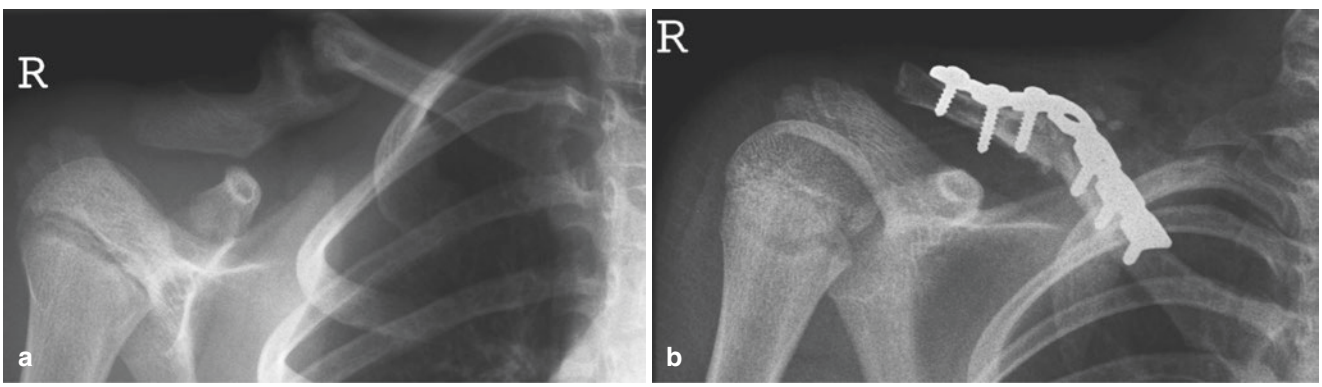


Fig. 8.5 (**a**) Several months after trauma a palpable lump persisted, radiography showed a post-traumatic pseudoarthrosis. (**b**) Post-surgical radiograph shows a well-aligned clavicle

8.2 Cause of Clavicle Fractures

All clavicle fractures result from trauma, either in normal bone or in weakened bone. The type of fracture depends on the cause (mechanism), the amount of energy that is transferred, and the bone strength. According to Stanley et al., fracturing of the clavicle is more likely when the energy that is transferred during the impact is absorbed quickly, than when it is absorbed more slowly [14].

Clavicle fractures in normal bone are almost always caused by a blunt force trauma with a high energy transfer (high energy trauma—impact trauma or compression/crushing trauma). Clavicle fractures may result from a direct or an indirect trauma [14]. Pathological fractures of the clavicle due to weakened bone are only rarely described in paediatric patients and will also be caused by direct and indirect trauma [15].

8.2.1 Direct Trauma with High Energy Transfer

Of all clavicle fractures 7% is caused by a direct impact or local compression on the clavicle, the point of the shoulder, or the sternum [16]. This may happen during sports activities, e.g. when the clavicle is hit, e.g. with a fist or a hockey stick or in contact sports when an opponent lands with his/her weight on top of the victim. It may also happen in motor vehicle accidents, e.g. with the use of three-point restraining seatbelts [17].

8.2.2 Indirect Trauma

Eighty-seven percent of clavicle fractures are caused by a fall onto the lateral shoulder, in which energy is transferred indirectly onto the clavicle and around 6% are caused by a fall on outstretched hand or arm (FOOSH), both causing a compressive force across the clavicle (Figs. 8.6a–c and 8.7) [14, 16]. The fracture occurs due to indirect transfer of energy via the scapula to the clavicle in both mechanisms. According to Stanley et al., it is not possible to deduce from the location of the fracture which of both mechanisms caused the fracture [14].

Stress fractures of the clavicle, although extremely rare, have been reported due to sporting activities [18–20]. Typical complaints of clavicle stress fractures are local pain over the clavicle sometimes associated with swelling and often with no definite history of trauma. Stress fractures of the clavicle may occur bilaterally [21].

Other rare causes of indirect trauma can be found in the medical literature. Kendrew et al. mentioned that very rarely, in adult patients the clavicle can fracture due to violent muscle contractions in seizures [16]. It is not known whether this can occur in paediatric patients.

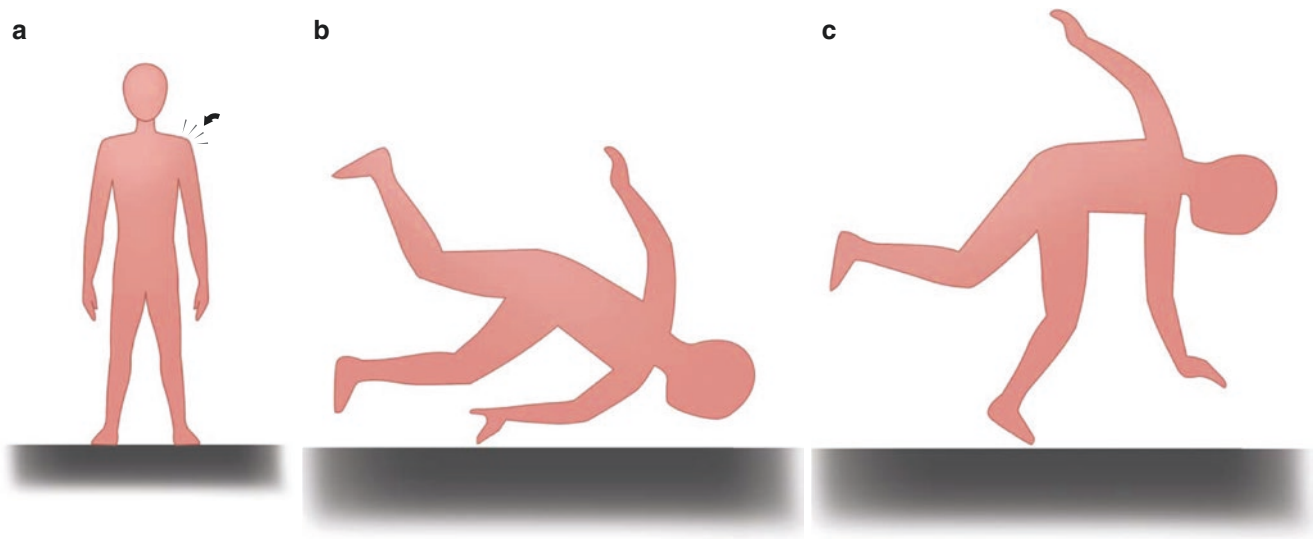


Fig. 8.6 Trauma mechanisms leading to clavicle fractures. (**a**) Direct impact on clavicle, (**b**) fall with impact on clavicle, and (**c**) fall on outstretched hand

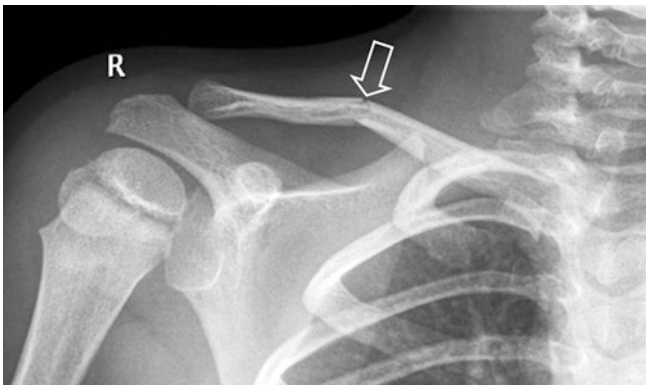


Fig. 8.7 Clavicle fracture (arrow) after fall on outstretched hand

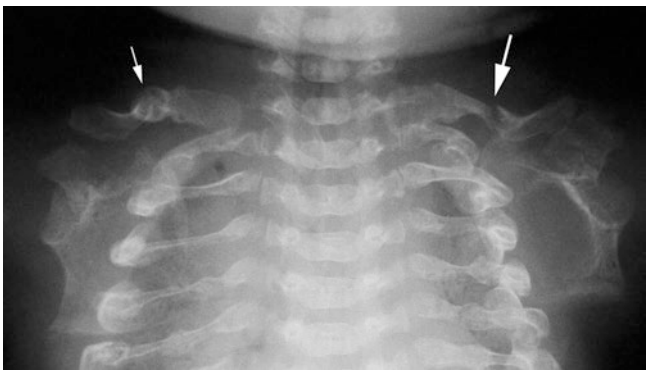


Fig. 8.8 A newborn with osteogenesis imperfecta type 2b. Recent clavicular fracture on the left (large arrow) and healed fracture on the right (small arrow)

8.3 Manner of Clavicle Fractures

Clavicle fractures may occur intra-uterine, during birth or after birth. Clavicle fractures that occur after birth can be due to accidental circumstances (e.g. a fall or motor vehicle accident) or non-accidental circumstances (inflicted injuries). However, it is impossible to distinguish between fractures occurring during or after birth or between accidental and non-accidental fractures, based on clinical symptoms and/or radiological characteristics [22].

8.3.1 Trauma Before Birth

In the literature only a few case reports are found, in which the intra-uterine occurrence of a clavicle fracture has been described [23–25]. Most of these case reports are associated with skeletal dysplasia syndromes including osteogenesis imperfecta and congenital metabolic disorders (Fig. 8.8). Samedi et al. reported a case of an isolated clavicle fracture in a term infant with normally mineralized bone, diagnosed postnatally, presumably due to an episode of a physical assault by her partner several weeks prior to delivery which included a heavy blow to her abdomen (Fig. 8.9) [25].

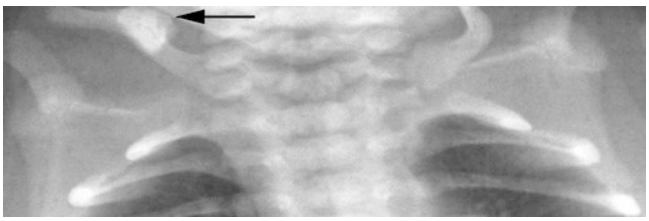


Fig. 8.9 Radiograph obtained 16 h after birth demonstrated a mild deformity of the middle third of the right clavicle with bony prominence and new bone formation (arrow) consistent with callus formation of healing clavicular fracture (courtesy of Dr. Veronica M Samedi, MD, University of Saskatchewan) [25]

8.3.2 Trauma During Birth

Clavicle fractures are the most prevalent fractures due to trauma during birth (Fig. 8.10). Frequently described risk factors are high birth weight, shoulder dystocia, above average gestational age and possibly vacuum extraction or use of forceps [26–29].

Nearly 90% of all fractures that occur during delivery are fractures of the clavicle [30–32]. In Fig. 8.11 the presumed mechanism for the occurrence of a clavicle fracture during birth is shown: the anterior shoulder is pressed against the mother's symphysis pubis during labour.

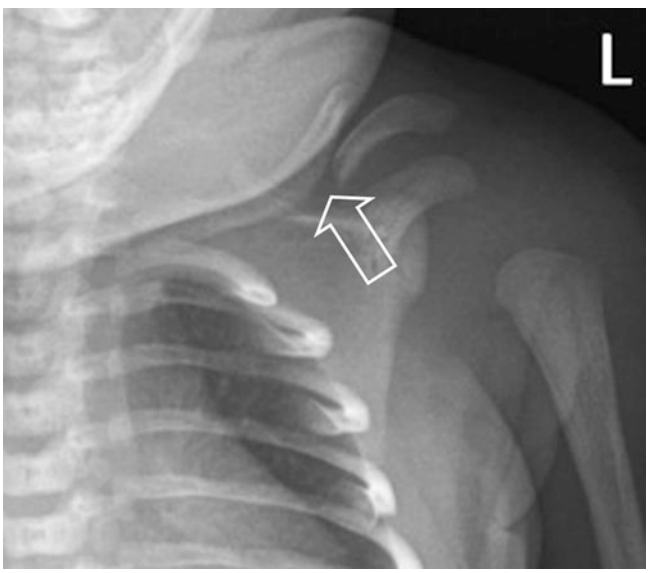


Fig. 8.10 Neonatal clavicle fracture resulting from birth trauma

Table 8.1 gives overview of the incidence of clavicle fractures in newborns in several studies. In a large nationwide study, based on data from 1997 to 2017, by Kekki et al. a total of 13,460 clavicle fractures were found in 1,203,434

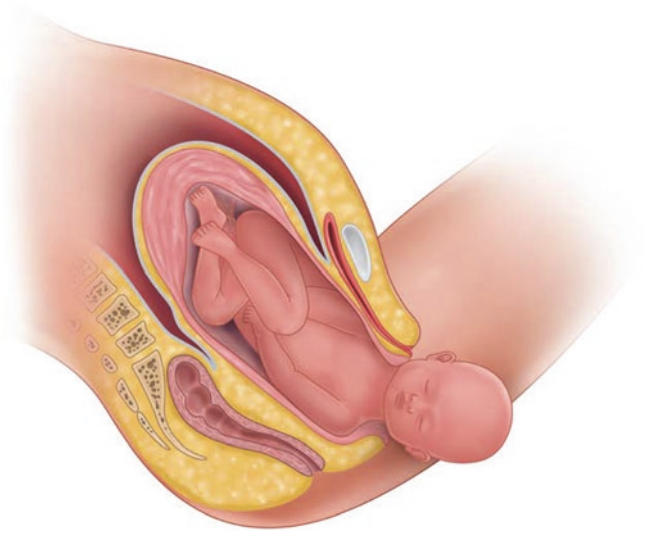


Fig. 8.11 The presumed mechanism for the occurrence of a clavicle fracture during birth

Table 8.1 Incidence of clavicle fractures in newborns

Author	N births	N clavicle fractures	Estimated incidence (%)
Rubin [32]	15,435	43	0.28
Camus et al. [80]	20,409	105	0.51
Oppenheim et al. [28]	21,632	58	0.27
Bhat et al. [81]	34,946	16	0.05
McBride et al. [38]	9106	43	0.47
Ahn et al. [36]	77,543	319	0.41
Choi et al. [34]	36,286	392	1.08
Casellas-García et al. [37]	23,508	155	0.66
Rehm et al. [82]	87,461	46	0.05
Kekki et al. [33]	1,203,434	13,460	1.12

live births [33]. In their study a significant decrease of the incidence of clavicle fractures was found, from 17.4 to 5.0 per 1000 live births. The risk of sustaining a clavicle fracture was much higher in a vaginal delivery than in a Caesarean section [34, 35]. Choi et al. found, in a sub-cohort study of 89,367 neonates, a clavicle fracture in 19 children out of a total of 36,286 children born by Caesarean section (0.05%) [34]. The most important risk factor for the occurrence of a clavicle fracture during Caesarean section was a high birthweight. If a clavicle fracture is found in a neonate, a brachial plexus injury should always be ruled out [28, 36–39].

Various authors point out that the occurrence of a clavicle fracture during birth is usually not predictable or preventable and rarely or never has any clinical consequences [27, 29, 36, 40]. According to Choi et al., this also applies to the occurrence during caesarean section [34].

The diagnosis of a birth trauma-related clavicle fracture can be delayed. Most neonates do not show any fracture-related symptoms. Almost half of the fractures were found only after meticulous and repeated examination and up to 15% is only diagnosed after a couple of weeks, as a result of callus formation, detected as a palpable mass by the parents or during a physical examination (Figs. 8.3 and 8.4) [36, 41]. A 'fresh' fracture may, even in retrospect, not be visible on imaging [2].

Table 8.2 First signs of healing in newborn clavicle fractures [42]

Signs of healing	First seen (days)	Peak period (days)
Periosteal reaction	7	11–42
Callus	11	12–61
Bridging	20	22–63
Remodelling	35	49–59

Differentiating between birth-related fractures and fractures due to a trauma after birth is possible to a certain extent, by analyzing the radiologically visible healing characteristics of the fracture. The healing process of clavicle fractures shows a predictable pattern (Table 8.2) [42, 43].

Walters et al. rarely found subperiosteal new bone formation in clavicle fractures before the seventh day after birth [43]. Subperiosteal new bone formation was usually present on the tenth day. Callus formation was rarely seen in clavicle fractures under 9 days of age, but was usually present after 15 days of age. Under normal circumstances, when a fracture is diagnosed later than 10 to 15 days after birth and in which there is no evidence of recovery, one can safely conclude that the fracture was not birth-related [4, 44].

8.3.3 Trauma After Birth: Accidental Circumstances

The incidence of clavicle fractures decreases with increasing age [45]. In mobile children of preschool age, clavicle fractures are a common occurrence (between 8% and 15% of all fractures in this age category) [6, 46, 47]. It is unusual to find accidental fractures of the medial or distal end of the clavicle in children less than 3 years of age, if these are encountered inflicted trauma should be considered [48]. In mobile children a clavicle fracture usually occurs in accidental circumstances (falls, sporting activities, motor vehicle accidents). Generally, it will be a mid-shaft fracture, mostly due to a fall on the shoulder or on the outstretched arm/hand (Fig. 8.12a, b) [6, 14, 49].

8.3.4 Trauma After Birth: Iatrogenic

In the literature a number of case reports are found, in which the occurrence of iatrogenic clavicle fractures, due to medical procedures or massage, is described. Sperry and Pfalzgraf described the occurrence of fractures in both clavicles and a fracture of the left upper arm (medial epicondyle) in a 9-month-old child [50]. The child was found unresponsive in the crib 5 h after the last feeding. The fractures were found

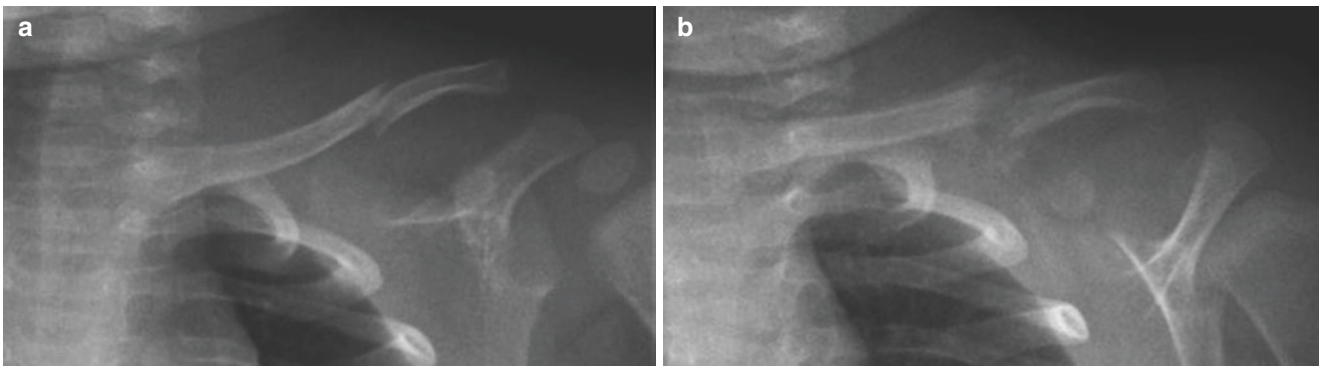


Fig. 8.12 (**a**) Non-displaced oblique mid-shaft clavicular fracture in a child after a fall from a chair. (**b**) Radiograph after 17 days shows soft callus formation

by post-mortem radiology. No external or internal abnormalities, consistent with injuries, were found during the autopsy. Some visceral and epicardial petechiae, as can be found in cot death, were found. The parents had no explanation for the occurrence of the fractures and an investigation by the police followed due to a suspicion of inflicted injury. During the investigation it appeared that the child had been treated by a non-licenced chiropractor 3 to 4 weeks before death because of alleged shoulder dislocations. According to the authors, this time interval corresponded to the histological dating of the fractures. The unusual location in both clavicles could also be explained in this way. The cause of death was determined to be a cot death.

Ibrahim described the occurrence of a fracture in the left clavicle of a 7-day-old newborn in the context of traditional treatment in Nigeria, whereby the hair is shaved and the uvula removed [51–53]. On day 10 after birth, the girl was seen in the Emergency Department of a hospital because of excessive crying, a swollen left shoulder and reduced movements of the left upper arm. Radiological examination revealed a fracture in the distal part of the clavicle. The authors reasoned that in this case the fracture could be attributable to excessive restrain during the procedure.

Mboutol-Mandavo et al. described the occurrence of fractures of the femur and clavicle in 2 children of 17 days and 1 month of age, respectively [54]. No evidence was found for an event in which the fractures could have occurred, or for osteogenesis imperfecta in the family. No other fractures were found that could indicate child abuse. The authors came to the conclusion that the injuries in both children were caused by a traditional form of African baby massage.

8.3.5 Trauma After Birth: Non-accidental Circumstances

In children most clavicle fractures, sustained after birth, are due to accidental circumstances. Inflicted clavicle fractures

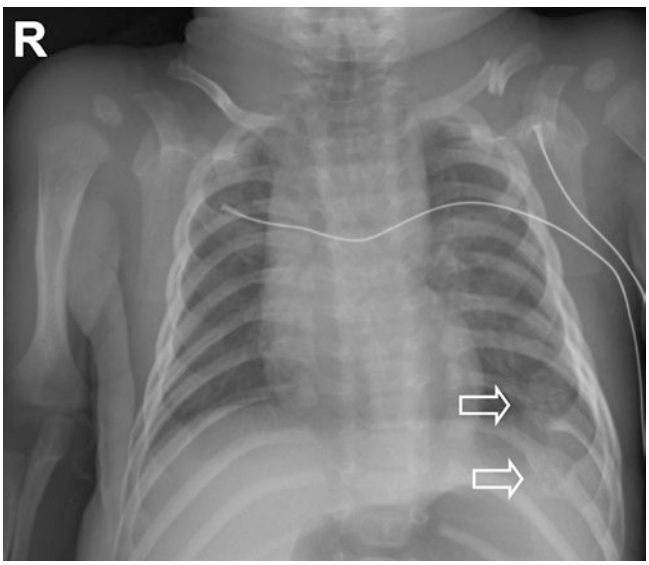


Fig. 8.13 Chest radiograph shows an acute clavicular fracture on the left in an infant. However, note the presence of healing posterior rib fractures (open arrows) indicating repetitive inflicted trauma

seem to be relatively rare in children (Fig. 8.13). Of all inflicted fractures in children, 2–7% are thought to be clavicle fractures [4, 55, 56].

Leventhal et al. evaluated the circumstances under which clavicle fractures were sustained in hospitalized children with fractures under the age of 3 years [57]. They found that the incidence of inflicted clavicle fractures decreased with increasing age. They also found that in children under 1 year of age 28.1% of the clavicle fractures were inflicted, in children between 1 and 2 years 16.7% and in children between 2 and 3 years 6%.

Pandya et al. found that clavicle fractures in children under the age of 48 months were more commonly sustained in non-accidental than in accidental circumstances, although clavicle fractures in children under the age of 18 months were sustained in non-accidental circumstances as often as in accidental circumstances [58].

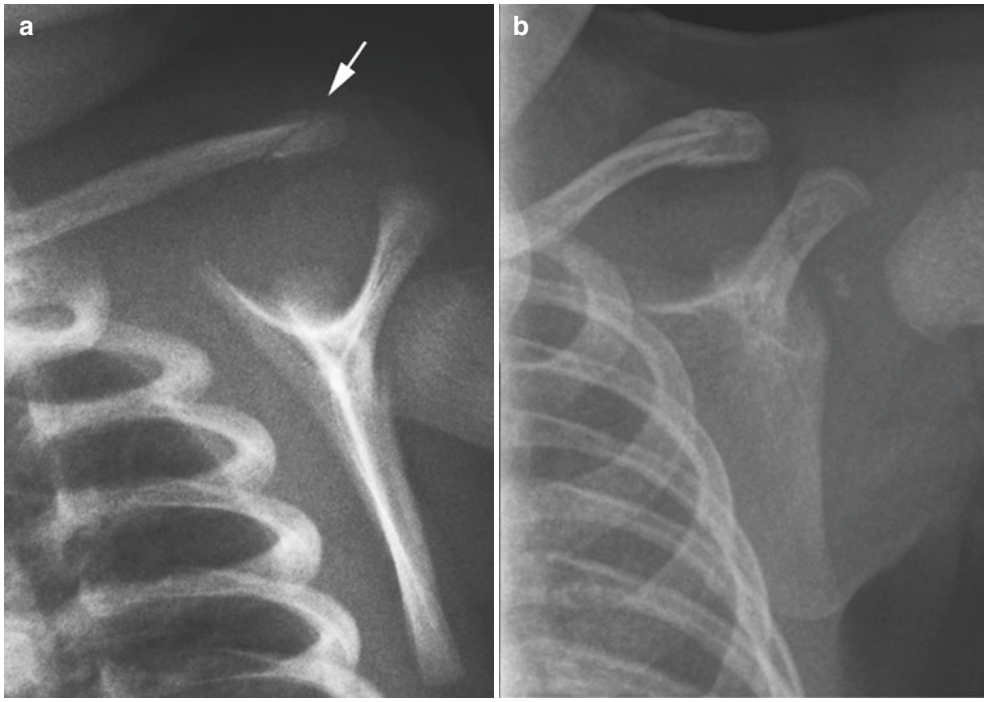


Fig. 8.14 (**a**) Non-accidental distal clavicular fracture (arrow) in a 2-week-old girl with a complex skull fracture, rib fractures, and CMLs of both tibiae. (**b**) Second skeletal survey shows callus formation

Worlock et al. found that clavicle fractures were inflicted in 18% of the children with fractures under the age of 18 months, compared to accidental fractures in 5% [59]. In the group of children between 18 and 60 months of age, clavicle fractures were found to be inflicted in 14% of the children, compared to 12% with accidental cause.

Barber et al. found bilateral clavicle fractures in 4 of 24 children, who sustained the clavicle fracture in non-accidental circumstances, and 10 of these 24 children had other fractures on the skeletal survey [60].

Inflicted clavicle fractures are caused by either a direct trauma (e.g. a direct blow on the clavicle or the shoulder) or by an indirect trauma (e.g. deliberate pushing or throwing of the child, leading to a fall on the shoulder or on an outstretched hand/arm or traction to the arm). A non-accidental blow on the clavicle or shoulder will usually cause a mid-shaft fracture, just as will happen in an accidental impact trauma, e.g. with an object.

Accidental fractures of the medial or distal end of the clavicle are rarely found in children less than 3 years old (Figs. 8.14a, b and 8.15) [48]. It is assumed that fracturing in these locations occurs through sudden traction to the arms, e.g. in violent shaking [4, 48, 61]. This type of fracture often occurs combined with injuries to the proximal humerus [62, 63]. The causing mechanism of these injuries can be compared to the mechanism that causes metaphyseal injuries of the long bones, which also can result from sudden traction to the arms and legs.

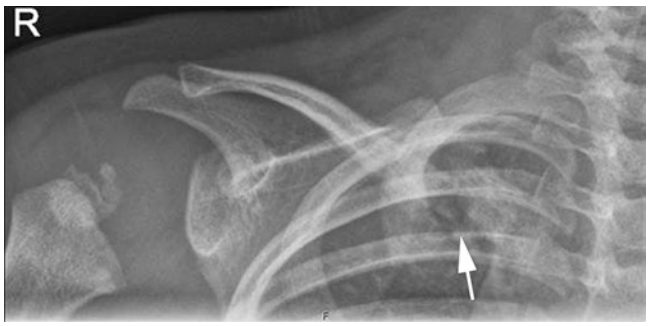


Fig. 8.15 Non-accidental medial clavicular fracture (arrow) in a 13-month-old infant who presented with a lump on the chest without a history trauma

It is not possible to differentiate in children between inflicted and accidental clavicle fractures based on the presence or absence of bruises near the fracture. Peters et al. evaluated the presence of bruising associated with fractures in children. They identified seven children with inflicted clavicle fractures, none of them had bruising near the fracture [64].

8.4 Differential Diagnosis of Clavicle Fractures

Congenital pseudoarthrosis of the clavicle (CPC) is, despite being a rare entity, often mentioned in the medical literature as a mimic of clavicle fractures [65–70]. It is more common in girls than in boys and most often the right clavicle is affected [70]. Although, it can be found bilaterally [68, 71]. The aetiology of CPC is debated with two potential explanations, the first is based on the fact that the clavicle is derived from two ossification centres that fuse in the 45th day of gestation. Failure to do so would lead to CPC [65]. The second explanation is that CPC is the result of pressure and pulsations of the bud of the subclavian artery on the developing clavicle, this would explain the higher prevalence on the right side [72]. It can be recognized, because of the presence of a painless swelling over the clavicle. Congenital pseudoarthrosis of the clavicle has been confused with a clavicle fracture, either due to birth trauma/accidental circumstances or non-accidental circumstances [70]. According to Walker et al. two distinct sections, each with a smooth and intact cortex, of the clavicle are seen on radiography [70]. In CPC there will be no radiographic evidence of subperiosteal new bone formation or callus formation. Most commonly, the pseudoarthrosis occurs at the junction of the middle and distal third of the clavicle [70].

There are other, in infancy rare, diseases that can mimic a (healing) clavicular fracture such as bone tumours (especially Ewing's sarcoma) and chronic recurrent multifocal osteomyelitis (CRMO) (Fig. 8.16a, b) [73–78]. These diseases can present with lamellated periosteal reaction, mimicking a healing fracture, without a history of trauma. Constriction of the clavicle may simulate a fracture in restrictive dermatitis (Fig. 8.17) [79]. However, the typical clinical features will confirm the diagnosis of restrictive dermatitis.

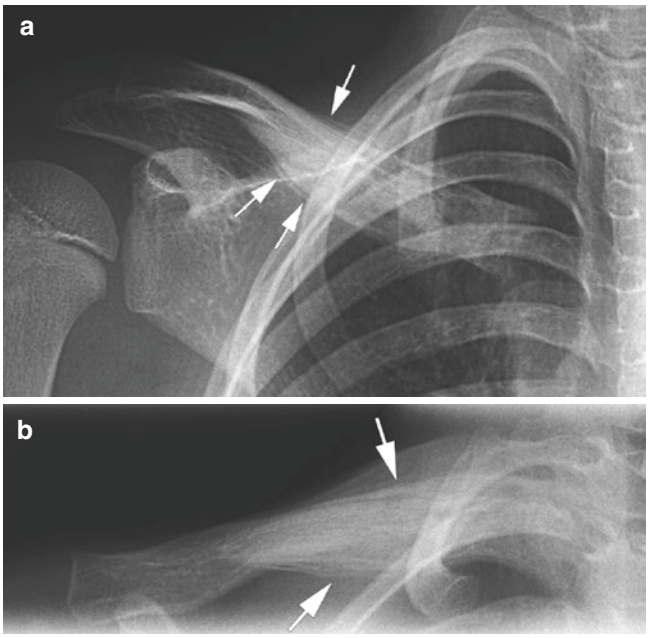


Fig. 8.16 AP (**a**) and caudo-cranial view (**b**) of the right clavicle with thickening and subperiosteal new bone formation (arrows) in a 7 year-old child with chronic recurrent multifocal osteomyelitis, simulating a healing fracture

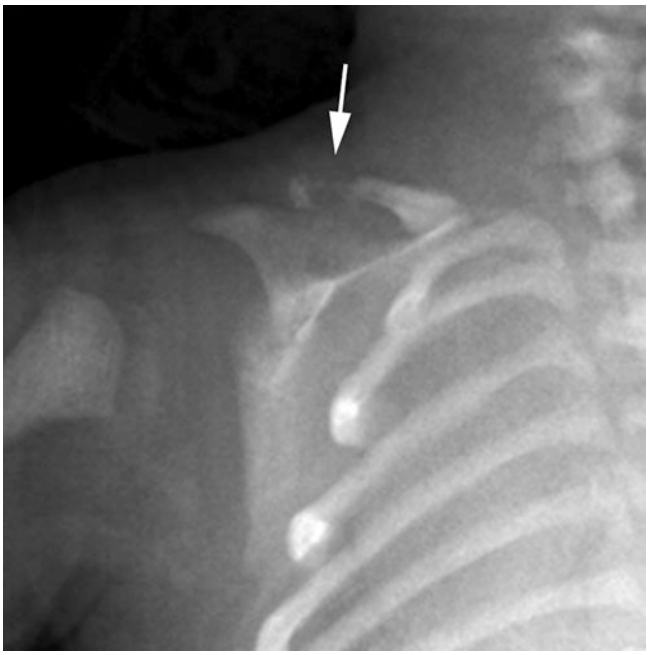


Fig. 8.17 A newborn with restrictive dermatitis. Severe stricture of distal clavicular (arrow) simulates a fracture

References

1. England SP, Sundberg S (1996) Management of common pediatric fractures. Pediatr Clin N Am 43:991–1012
2. Kaczor K, Pierce MC (2011) Abusive Fractures. In: Jenny C (ed) Child abuse and neglect: diagnosis, treatment and evidence. Elsevier Saunders, St. Louis
3. Allman FL Jr (1967) Fractures and ligamentous injuries of the clavicle and its articulation. J Bone Joint Surg Am 49:774–784
4. Kogutt MS, Swischuk LE, Fagan CJ (1974) Patterns of injury and significance of uncommon fractures in the battered child syndrome. Am J Roentgenol Radium Therapy, Nucl Med 121:143–149
5. Miller DS, Boswick JA Jr (1969) Lesions of the brachial plexus associated with fractures of the clavicle. Clin Orthop Relat Res 64:144–149
6. Nordqvist A, Petersson C (1994) The incidence of fractures of the clavicle. Clin Orthop Relat Res 1994:127–132
7. Pyper JB (1978) Non-union of fractures of the clavicle. Injury 9:268–270
8. Rowe CR (1968) An atlas of anatomy and treatment of midclavicular fractures. Clin Orthop Relat Res 58:29–42
9. Sambandam B, Gupta R, Kumar S, Maini L (2014) Fracture of distal end clavicle: a review. J Clin Orthop Trauma 5:65–73
10. Neer CS 2nd (1968) Fractures of the distal third of the clavicle. Clin Orthop Relat Res 58:43–50
11. Goddard NJ, Stabler J, Albert JS (1990) Atlanto-axial rotatory fixation and fracture of the clavicle. An association and a classification. J Bone Joint Surg Br 72:72–75
12. van der Werken C, Hoofwijk AG (1991) Fractures and pseudarthrosis of the clavicle. Ned Tijdschr Geneeskd 135:788–791
13. Neer CS (1960) Non-union of the clavicula. JAMA 172:1006–1011
14. Stanley D, Trowbridge EA, Norris SH (1988) The mechanism of clavicular fracture. A clinical and biomechanical analysis. J Bone Joint Surg Br 70:461–464
15. Vedantam R, Crawford AH, Kuwajima SS (1996) Aneurysmal bone cyst of the clavicle in a child. Br J Clin Pract 50:474–476
16. Kendrew JM, Wallace WA, Tibrewal S (2008) Clavicle fractures: synopsis of causation. https://www.gov.uk/government/publications/synopsis-of-causation-clavicle-fractures. Accessed 10 Aug 2021
17. Hayes CW, Conway WF, Walsh JW, Coppage L, Gervin AS (1991) Seat belt injuries: radiologic findings and clinical correlation. Radiographics 11:23–36
18. Roset-Llobet J, Saló-Orfila JM (1998) Sports-related stress fracture of the clavicle: a case report. Int Orthop 22:266–268
19. Fallon KE, Fricker PA (2001) Stress fracture of the clavicle in a young female gymnast. Br J Sports Med 35:448–449
20. Jones GL (2006) Upper extremity stress fractures. Clin Sports Med 25:159–174, xi
21. Fujioka H, Nishikawa T, Koyama S, Yamashita M, Takagi Y, Oi T, Tsunemi K, Tanaka J, Yoshiya S (2014) Stress fractures of bilateral clavicles in an adolescent gymnast. J Shoulder Elb Surg 23:e88–e90
22. Herndon WA (1983) Child abuse in a military population. J Pediatr Orthop 3:73–76
23. Freedman M, Gamble J, Lewis C (1982) Intrauterine fracture simulating a unilateral clavicular pseudarthrosis. J Can Assoc Radiol 33:37–38
24. Hawthorne ES (1903) Fractured clavicle with ossific union in utero. Lancet 162:315
25. Samedi A, Jorgenson K, Samedi V (2015) Isolated clavicular fracture as a result of trauma during pregnancy. In: 4th global congress for consensus in pediatrics and child health, Budapest, Hungary
26. Allen RH (2007) On the mechanical aspects of shoulder dystocia and birth injury. Clin Obstet Gynecol 50:607–623
27. Chez RA, Carlan S, Greenberg SL, Spellacy WN (1994) Fractured clavicle is an unavoidable event. Am J Obstet Gynecol 171:797–798
28. Oppenheim WL, Davis A, Growdon WA, Dorey FJ, Davlin LB (1990) Clavicle fractures in the newborn. Clin Orthop Relat Res 1990:176–180
29. Roberts SW, Hernandez C, Maberry MC, Adams MD, Leveno KJ, Wendel GD Jr (1995) Obstetric clavicular fracture: the enigma of normal birth. Obstet Gynecol 86:978–981
30. Farkas R, Levine S (1950) X-ray incidence of fractured clavicle in vertie presentation. Am J Obstet Gynecol 59:204–206
31. Cohen AW, Otto SR (1980) Obstetric clavicular fractures. A three-year analysis. J Reprod Med 25:119–122
32. Rubin A (1964) Birth injuries: incidence, mechanisms and end results. Obstet Gynecol 23:218–221
33. Kekki M, Salonen A, Tihtonen K, Mattila VM, Gissler M, Huttunen TT (2020) The incidence of birth injuries decreased in Finland between 1997 and 2017: a nationwide register study. Acta Paediatr (Oslo, Norway) 2020:1992
34. Choi HA, Lee YK, Ko SY, Shin SM (2017) Neonatal clavicle fracture in cesarean delivery: incidence and risk factors. J Matern Fetal Neonatal Med 30:1689–1692
35. Dolivet E, Delesalle C, Morello R, Blouet M, Bronfen C, Dreyfus M, Benoist G (2018) A case-control study about foetal trauma during caesarean delivery. J Gynecol Obstet Hum Reprod 47:325–329
36. Ahn ES, Jung MS, Lee YK, Ko SY, Shin SM, Hahn MH (2015) Neonatal clavicular fracture: recent 10 year study. Pediatr Int 57:60–63
37. Casellas-García G, Cavanilles-Walker JM, Albertí-Fitó G (2018) Clavicular fracture in the newborn: is fracture location a risk factor for obstetric brachial palsy? J Neonatal-Perinatal Med 11:61–64
38. McBride MT, Hennrikus WL, Mologne TS (1998) Newborn clavicle fractures. Orthopedics 21:317–319. discussion 319–320
39. Salonen IS, Uusitalo R (1990) Birth injuries: incidence and predisposing factors. Zeitschrift fur Kinderchirurgie: organ der Deutschen, der Schweizerischen und der Osterreichischen Gesellschaft fur Kinderchirurgie. Surg Infancy Childhood 45:133–135
40. Lurie S, Wand S, Golan A, Sadan O (2011) Risk factors for fractured clavicle in the newborn. J Obstet Gynaecol Res 37:1572–1574
41. Joseph PR, Rosenfeld W (1990) Clavicular fractures in neonates. Am J Dis Child 144:165–167
42. Fadell M, Miller A, Trefan L, Weinman J, Stewart J, Hayes K, Maguire S (2017) Radiological features of healing in newborn clavicular fractures. Eur Radiol 27:2180–2187
43. Walters MM, Forbes PW, Buonomo C, Kleinman PK (2014) Healing patterns of clavicular birth injuries as a guide to fracture dating in cases of possible infant abuse. Pediatr Radiol 44:1224–1229
44. Cumming WA (1979) Neonatal skeletal fractures. Birth trauma or child abuse? J Can Assoc Radiol 30:30–33
45. Jones E (1998) Skeletal growth and development as related to trauma. In: Green NE, Swiontkowski MF (eds) Skeletal trauma in children. Saunders
46. Landin LA (1983) Fracture patterns in children. Analysis of 8,682 fractures with special reference to incidence, etiology and secular changes in a Swedish urban population 1950–1979. Acta Orthop Scand Suppl 202:1–109
47. Lichtenberg RP (1954) A study of 2,532 fractures in children. Am J Surg 87:330–338
48. Merten DF, Cooperman DR, Thompson GH (1992) Skeletal manifestations of child abuse. In: Reece RM (ed) Child abuse – medical diagnosis and management. Lea & Febiger, pp 23–53
49. Kreisinger V (1927) Sur le traitement des fractures de la clavicule. Rev Chir 65:396–407
50. Sperry K, Pfalzgraf R (1990) Inadvertent clavicular fractures caused by "chiropractic" manipulations in an infant: an unusual form of pseudoabuse. J Forensic Sci 35:1211–1216
51. Prual A, Gamatie Y, Djakounda M, Huguet D (1994) Traditional uvulectomy in Niger: a public health problem? Soc Sci Med 39:1077–1082

52. Adoga AA, Nimkur TL (2011) The traditionally amputated uvula amongst Nigerians: still an ongoing practice. ISRN Otolaryngol 2011:704924
53. Ibrahim A (2013) Clavicular fracture following uvulectomy and traditional hair barbing: a case report. J Family Med Prim Care 2:390–392
54. Mboutol-Mandavo C, N'Dour O, Ouedraogo SF, Missengue-Bosseba R, Ndiaye D, Ngom G (2016) Fractures du nouveau-né et du nourrisson secondaires au massage traditionnel [Newborn and infant fractures secondary to traditional massage]. Arch Pediatr 23:963–965
55. 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? AJR Am J Roentgenol 197:W159–W163
56. Merten DF, Radlowski MA, Leonidas JC (1983) The abused child: a radiological reappraisal. Radiology 146:377–381
57. Leventhal JM, Martin KD, Asnes AG (2008) Incidence of fractures attributable to abuse in young hospitalized children: results from analysis of a United States database. Pediatrics 122:599–604
58. Pandya NK, Baldwin K, Wolfgruber H, Christian CW, Drummond DS, Hosalkar HS (2009) Child abuse and orthopaedic injury patterns: analysis at a level I pediatric trauma center. J Pediatr Orthop 29:618–625
59. Worlock P, Stower M, Barbor P (1986) Patterns of fractures in accidental and non-accidental injury in children: a comparative study. BMJ 293:100–102
60. Barber I, Perez-Rossello JM, Wilson CR, Kleinman PK (2015) The yield of high-detail radiographic skeletal surveys in suspected infant abuse. Pediatr Radiol 45:69–80
61. Merten DF, Carpenter BL (1990) Radiologic imaging of inflicted injury in the child abuse syndrome. Pediatr Clin N Am 37:815–837
62. Johnson CF (1990) Inflicted injury versus accidental injury. Pediatr Clin N Am 37:791–814
63. Launius GD, Silberstein MJ, Luisini A, Graviss ER (1998) Radiology of child abuse. In: Monteleone JA, Brodeur AE (eds) Child maltreatment – a clinical guide and reference. GW Medical, St. Louis, pp 31–58
64. Peters ML, Starling SP, Barnes-Eley ML, Heisler KW (2008) The presence of bruising associated with fractures. Arch Pediatr Adolesc Med 162:877–881
65. Currarino G, Herring JA (2009) Congenital pseudarthrosis of the clavicle. Pediatr Radiol 39:1343–1349
66. Chalfant JS, Sanchez TR (2019) Congenital clavicular pseudoarthosis-how to differentiate it from the more common clavicular fractures. Pediatr Emerg Care 35:e37–e39
67. de Figueiredo MJ, Dos Reis Braga S, Akkari M, Prado JC, Santili C (2012) Congenital pseudoarthrosis of the clavicle. Rev Bras Ortop 47:21–26
68. Price BD, Price CT (1996) Familial congenital pseudoarthrosis of the clavicle: case report and literature review. Iowa Orthop J 16:153–156
69. Sung TH, Man EM, Chan AT, Lee WK (2013) Congenital pseudarthrosis of the clavicle: a rare and challenging diagnosis. Hong Kong Med J 19:265–267
70. Walker BM, Vangipuram SD, Kalra K (2014) Congenital pseudoarthrosis of the clavicle: a diagnostic challenge. Glob Pediatr Health 1:2333794X14563384
71. Laliotis NA, Chrysanthou C, Anastasopoulos N (2020) Spontaneous union of bilateral congenital pseudoarthrosis of the clavicle, in a baby. J Clin Orthop Trauma 11:314–316
72. Lloyd-Roberts GC, Apley AG, Owen R (1975) Reflections upon the aetiology of congenital pseudarthrosis of the clavicle. With a note on cranio-cleido dysostosis. J Bone Joint Surg Br 57:24–29
73. Menashe SJ, Aboughalia H, Zhao Y, Ngo AV, Otjen JP, Thapa MM, Iyer RS (2020) The many faces of pediatric chronic recurrent multifocal osteomyelitis (CRMO): a practical location- and case-based approach to differentiate CRMO from its mimics. J Magn Reson Imaging 2020:e27299
74. Iyer RS, Thapa MM, Chew FS (2011) Chronic recurrent multifocal osteomyelitis: review. AJR Am J Roentgenol 196:S87–S91
75. d'Angelo P, de Horatio LT, Toma P, Ording Müller LS, Avenarius D, von Brandis E, Zadig P, Casazza I, Pardeo M, Pires-Marafon D, Capponi M, Insalaco A, Fabrizio B, Rosendahl K (2021) Chronic nonbacterial osteomyelitis – clinical and magnetic resonance imaging features. Pediatr Radiol 51:282–288
76. Sakran W, Lumelsky D, Schildkraut V, Smolkin V, Halevy R, Koren A (2003) Chronic recurrent multifocal osteomyelitis in infancy: a case report. Clin Pediatr (Phila) 42:741–744
77. Murphey MD, Senchak LT, Mambalam PK, Logie CI, Klassen-Fischer MK, Kransdorf MJ (2013) From the radiologic pathology archives: ewing sarcoma family of tumors: radiologic-pathologic correlation. Radiographics 33:803–831
78. Schaal MC, Gendler L, Ammann B, Eberhardt N, Janda A, Morbach H, Darge K, Girschick H, Beer M (2021) Imaging in non-bacterial osteomyelitis in children and adolescents: diagnosis, differential diagnosis and follow-up-an educational review based on a literature survey and own clinical experiences. Insights Imaging 12:113
79. Reed MH, Chudley AE, Kroeker M, Wilmot DM (1993) Restrictive dermopathy. Pediatr Radiol 23:617–619
80. Camus M, Lefebvre G, Veron P, Darbois Y (1985) Traumatismes obstétricaux du nouveau-né. Enquête rétrospective à propos de 20409 naissances [Obstetrical injuries of the newborn infant. Retrospective study apropos of 20,409 births]. J Gynecol Obstet Biol Reprod (Paris) 14:1033–1043
81. Bhat BV, Kumar A, Oumachigui A (1994) Bone injuries during delivery. Indian J Pediatr 61:401–405
82. Rehm A, Promod P, Ogilvy-Stuart A (2020) Neonatal birth fractures: a retrospective tertiary maternity hospital review. J Obstetr Gynaecol 40:485–490

Sternum

9

Rob A. C. Bilo, Simon G. F. Robben, and Rick R. van Rijn

Contents

9.1 General Aspects of Sternum Fractures

Sternum fractures in children are rare with an increasing incidence with age. Ozsoy and Tezcan reported on a series of 225,000 trauma cases, <18 years of age, presented to a paediatric trauma unit between January 2012 and January 2018 [1]. In their series only 10 sternum fractures were seen, mean patient age 11.8 years (range 3 to 18), most cases were the result of falls (60%). Schmitt et al. evaluated the data of 47,893 patients with sternum fractures (all inpatients in Germany from 2005 to 2012) and found that only in 1.83% these fractures were found before the age of 16 years, followed by a sudden increase in the frequency up to the age of 20 years [2]. This sudden increase was explained by the authors as a result of the increase of traffic accidents of this age group, compared to the group of minors under 16 years of age. In a retrospective study over a 16-year period in a level I trauma centre, Ramgopal et al. described an even lower incidence of 19 cases (0.07%) out of a total of 25,781 paediatric patients [3]. All cases were the result of high velocity trauma mechanisms and had significant comorbidity.

Hechter et al. identified 12 children with sternum fractures at a large paediatric hospital over an 11-year period: 4 children were younger than 3 years of age, 8 were above 3 years of age [4].

Rosenfeld et al. identified 3860 patients of 18 years and younger with sternum fractures [5]. Ninety percent of these patients were between the age of 12 and 18 years.

Chalphin and Mooney reported on a series of 65 children with sternal fractures, 46 (71%) were boys and the median age of their population was 11 years [6]. The three most common causes were motor vehicle accidents (12/65, 18%), sports (12/65, 18%), and trampoline (8/65 12%).

R. A. C. Bilo
Veilig Thuis Rotterdam Rijnmond (Center for the Reporting of Child Abuse, Domestic Violence and Elder Abuse), Rotterdam, The Netherlands

S. G. F. Robben (✉)
Department of Radiology, Maastricht University Medical Center, Maastricht, The Netherlands
e-mail: s.robben@mumc.nl

R. R. van Rijn
Department of Radiology and Nuclear Medicine, Amsterdam UMC, University of Amsterdam, Amsterdam, The Netherlands

Department of Forensic Medicine, Netherlands Forensic Institute, The Hague, The Netherlands

R. A. C. Bilo et al. (eds.), *Forensic Aspects of Paediatric Fractures*, https://doi.org/10.1007/978-3-031-12041-1_9

Table 9.1 Associated injuries (mainly described in adult patients) [7, 8]

Skeletal lesions	• Sternoclavicular joint dislocation • Rib fractures (isolated or serial) • Clavicular fractures • Scapula fractures • Spinal injuries (cervical, thoracic, lumbar) • Pelvic fracture
Intrathoracic lesions	• Mediastinal haemorrhage, pneumothorax, haemothorax • Pulmonary contusion • Cardiac contusion, cardiac tamponade • Rupture of the thoracic aorta
Other lesions	• Abdominal injury • Traumatic brain injury (concussion, intracranial haemorrhage)

Sternum fractures fall into two distinct categories: isolated sternum fractures without associated injury and polytrauma sternum fractures with associated extrasternal injury (Table 9.1) [5, 9]. One of the associated injuries are vertebral fractures, which if present can lead to an unstable thoracic cage [10]. Isolated sternum fractures can be considered to be a relatively mild injury with a very good outcome with conservative management and a very low mortality rate (<1%) [9, 11–14].

Moënne Bühlmann et al. evaluated the data of 79 paediatric patients aged 18 years and younger diagnosed with sternum fractures after trauma [15]. They found that associated lesions in this group of patients were rare (only in 3 of the 79 patients). However, Rosenfeld et al. state that the presence of a sternum fracture in a patient ≤18 years should always lead to a careful medical evaluation, because of the risk of associated injuries (Table 9.1) and of complications [5, 16]. Almost 40% of the patients in this study had to be admitted to an intensive care unit and 8% of the patients that were admitted to a hospital died.

Although most fractures will be diagnosed on either conventional radiography or nowadays more and more CT, a sternal fracture can be an incidental finding on ultrasonography. With the increasing use of point-of-care ultrasonography, especially emergency medicine physicians should be aware of this finding and the potential consequences [17–20].

9.2 Cause of Sternum Fractures

A fracture of the sternum is always due to a trauma. In adults direct trauma is the most common cause, while in the paediatric population indirect trauma is more common [21].

9.2.1 Direct Trauma

It is generally accepted in the medical literature that a considerable blunt force trauma on the sternum/anterior chest-wall (trauma due to high energy transfer and/or high velocity impact) or forceful compression of the sternum towards the vertebrae is needed to fracture the sternum [11, 22, 23]. A blunt force impact trauma may occur when a moving object hits the more or less stationary sternum or front of the chest (anterior chest-wall impact trauma) or when the moving body (sternum or front of the chest) hits a more or less stationary object (deceleration).

Ferguson et al. evaluated the data of 12 children (aged 5 to 12 years old) and found, contrary to what generally is accepted in the medical literature, that the sternum in children is commonly fractured by more minor blunt trauma than generally recognized in the literature [22]. This finding was largely confirmed by Moënne Bühlmann et al., they found that over 90% of traumatic sternum fractures in a group of 79 paediatric patients of 18 years and younger were caused by trauma with a low-energy transfer (low-energy trauma) [15].

9.2.2 Indirect Trauma

9.2.2.1 Distortion

The sternum can fracture when the thorax suddenly vigorously flexes, leading to distortion of the sternum due to severe hyperflexion-compression of the thoracic vertebrae, in the absence of any impact trauma or compression [22]. According to Ferguson et al., all children with a sternum fracture after an indirect trauma with hyperflexion-compression should have a careful examination of the spine [22].

9.2.2.2 Fatigue Fractures in Normal Bone: Stress Fractures

Stress fractures of the sternum are extremely rare. This type of sternum fracture results from repetitive muscular action to the sternum and has been described in young gymnasts and in body builders [24–26]. Hassan et al. hypothesized that the cause (mechanism) of this type of stress fracture in gymnasts is repeated distortion of the sternum due to sudden forward flexion of the thoracic spine and violent protractions of the shoulders during tumbles, repeatedly stressing the sternum via the clavicles and leading to an uneven distribution of forces across the scalene muscles and pectoralis major [24]. These rare fractures may be underdiagnosed [25].

Stress fracture of the sternum is a rare injury and can occur in young athletes due to repeated stress. A case of a 14-year-old boy is reported who sustained fracture of the sternum without any history of significant trauma when he simply tried to lift his whole body over his arms and felt pain in front of the chest.

9.2.2.3 Fatigue Fractures in Weakened Bone: Insufficiency Fractures

Stress fractures due to insufficiency are also very rare and mainly described in patients, e.g. in eating disorders, patients on long-term steroid treatment, patients with underlying diseases, like osteoporosis, osteopenia, chronic obstructive pulmonary disease, rheumatoid arthritis, systemic lupus erythematosus or multiple myeloma, and postmenopausal women [27–30].

Only a few case reports on paediatric stress fractures due to insufficiency are found in the medical literature. Mitchell and Elliott described this type in an adolescent with cystic fibrosis due to osteoporotic changes [31]. Latzin et al. described a sternum fracture in a 16-year-old girl with cystic fibrosis with severe pulmonary disease complicated by osteoporotic fractures of the sternum and the sixth and seventh thoracic vertebral bodies [32]. Olmos et al. described the occurrence of a sternum fracture due to osteoporotic changes in one patient with anorexia nervosa in addition to another case report in the medical literature on a sternum fracture in another patient with anorexia nervosa [33]. Korovessis et al. described what they described as a 'spontaneous' fracture of the sternum in a 2-year-old girl, as a complication of treatment in a Boston brace for a progressive rigid thoracolumbar kyphoscoliosis [34]. Also patients with osteogenesis imperfecta are at a higher risk of sternal fractures after a relatively mild trauma (Fig. 9.1).

Fig. 9.1 Eight-year-old boy with osteogenesis imperfecta after a relatively mild hyperflexion injury resulting in a fracture of the manubrium (arrow)

9.3 Manner of Sternum Fractures

As stated in Sect. 9.2 fractures of the sternum are always the result of trauma. To the best of our knowledge the occurrence of intrauterine sternum fractures or sternum fractures due to birth is not mentioned in the medical literature.

9.3.1 Trauma After Birth: Accidental Circumstances

Sternal fractures in children are relatively rare, and isolated sternal fractures are even rarer. However, there have been multiple case reports describing such isolated fractures (Table 9.2). Rostan described an isolated sternum fracture in a 12-year-old soccer player [35]. Perez and Coddington described a sternum fracture in a 7-year-old boy who fell from monkey bars and striking his chest on the monkey bar [36]. Pérez-Martínez described, in a Spanish article, a case of a 6-year-old girl who fell on a trampoline [37]. DeFriend and Franklin reported two children who sustained a sternum fracture due to an indirect trauma; hyperflexion of the sternum due to a fall of a swing [38]. Ferguson et al. evaluated the data of 12 children (aged 5–12 years old), in 7 children

Table 9.2 Isolated sternum fractures in children

Author	Year	Age[a]	Sex	Trauma mechanism
Rostan [35]	1981	12	Male	Soccer trauma
Perez [36]	1983	7	Male	Fall from monkey bar
Pérez-Martínez [37]	1996	6	Female	Fall on trampoline
DeFriend [38]	2001	8	Female	Fall from swing
		7	Female	Fall from swing
Ferguson [22]	2003	12	Female	Fall from bike
		11	Female	Slipped on street
		10	Female	Fall from bike
		10	Female	Fall on bouncy castle
		5	Female	Fall from trampoline
		10	Female	Fall from bike
		7	Male	Fall from tree
		11	Female	Fall from trampoline
		10	Male	Fall from trampoline
		11	Male	Slipped in bath
		11	Male	Fall from bike
		10	Female	Fall from gym bar
Fichtel [39]	2016	8	Female	Fall in merry-go-round
		14	Female	Slipped
		5	Male	Fall from swing or hit by swing
		10	Male	Fall from parallel bars
Korhonen [40]	2017	10	Male	Fall from trampoline
		11	Male	Fall from trampoline
Fukuhara [18]	2018	5	Male	Hit by door
Sesia [41]	2018	8	Male	Fall from trampoline
Binder [42]	2020	10	Male	Knee impact

[a]Age in years

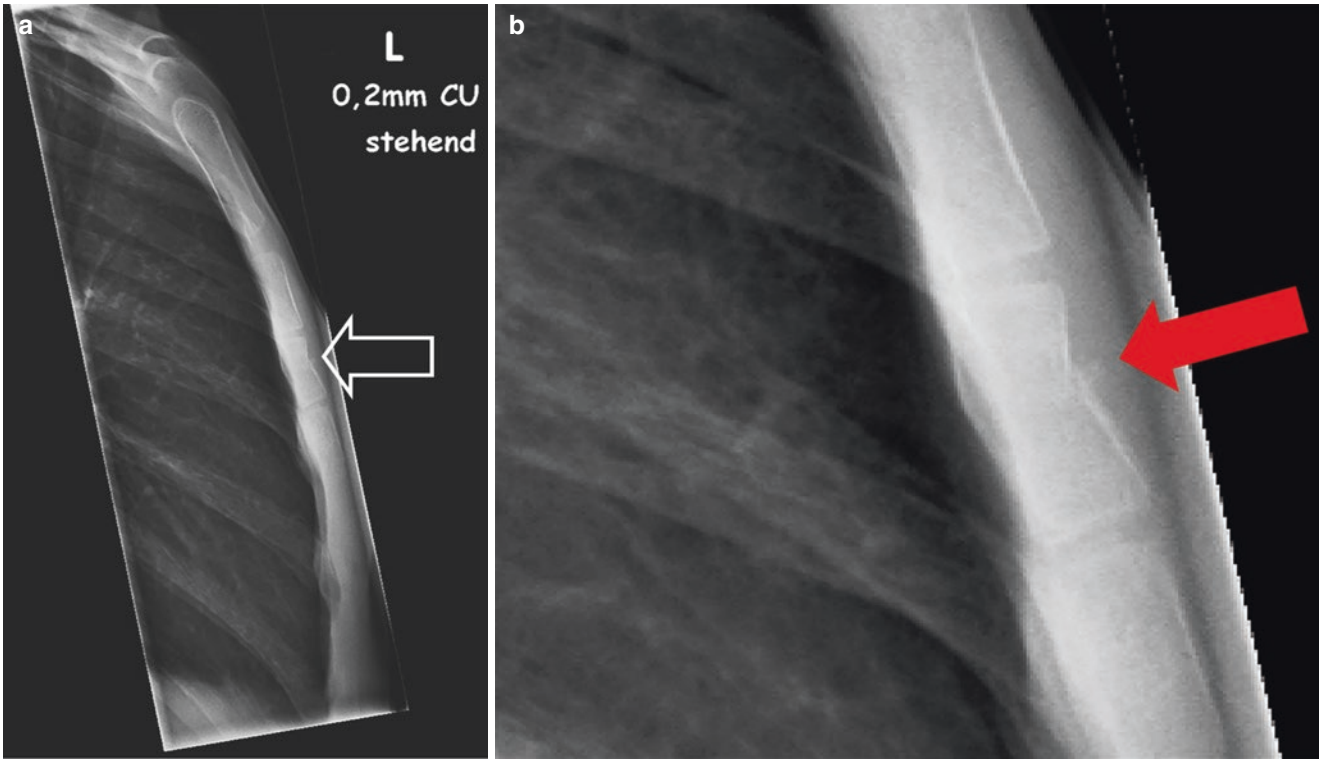


Fig. 9.2 (**a**) Ten-year-old boy who was hit over the sternum resulting in a sternum fracture (arrow). (**b**) Detailed view of the fracture (red arrow) (Courtesy of Professor S. Banaschak, Institute of Legal Medicine/University Hospital of Cologne, Cologne, Germany)

the sternum fracture was caused by an accidental direct blow on the chest (4× falling of a bicycle, 2× falling forwards, 1× fall from trampoline) and in 5 children the fracture was caused by an accidental indirect trauma (fall on the upper back or head, causing hyperflexion of the thoracic spine) [22]. Fichtel et al. reported on four cases and stated that isolated sternum fractures in children often are due to typical age-related traumatic incidents, like described in the article of Ferguson et al. [22, 39]. In a publication on trampoline-related injuries, Korhonen et al. describe two boys who sustained isolated sternum fractures after a fall from a trampoline [40]. Fukuhara et al. describe a case of a 5-year-old boy who presented with precordial pain five days after he was hit by a door in the chest [18]. CT of the chest at the day of trauma showed no fracture, but on day 5 the authors diagnosed a fracture using point-of-care ultrasonography. The image provided in the article, the fact that is known that CT is superior in detecting fractures, and the presence of an abscess should raise the possibility of the presence of a self-limiting sternal tumour of childhood [14]. Sesia et al. describe a case of an 8-year-old boy with a sternum fracture after a trampoline-related fall [41]. The fracture was slightly displaced and using a vacuum bell, which was applied for 6 weeks, stable reduction and consolidation was achieved. Binder et al. describe a case of a 10-year-old boy who was presented with pain over the sternum and a radiograph showed a fracture of the sternum (Fig. 9.2a, b) [42]. A thorough clinical history revealed that the complaints have started after his 12-year-old cousin hit him with a knee against the chest. In older literature several other cases have been described, although details of these cases are limited [43–48]. Based on the literature Table 9.3 provides an overview of the cause (mechanism) and manner of accidental isolated sternum fractures.

9.3.2 Trauma After Birth: Medical and Paramedical Procedures

Chest compression during resuscitation in adults is regularly mentioned in the medical literature as a cause of fractures of the sternum [53–55]. The incidence of CPR-related sternum fractures in adults seems to vary depending on the used method. Mechanical chest compressions seem to cause more CPR-related sternum fractures than manual compressions. Hoke and Chamberlain found an incidence varying from 1 to 43% in manual compression and of 0–93% in mechanical compression [54]. Friberg et al. found sternum fractures in 80% of the adult patients with mechanical chest compressions, compared to 38% with mechanical compressions [53]. Koster et al. compared the AutoPulse, LUCAS, and manual resuscitation in a prospective randomized clinical trial in which they included 337 patients [56]. They found sternal

Table 9.3 Overview of cause and manner (accidental) of sternum fractures [2, 7, 22, 24–26, 38, 49–52]

Cause (mechanism)		Manner: accidental, e.g.	Often fractures of
Direct trauma	Deceleration	Motor vehicle collision with • Impact of chest on steering wheel, mostly in cars without airbags • Use of seat belt	Body or manubrium
	Blunt impact on the anterior chest	Car vs. pedestrian collision with direct impact to the anterior chest Contact sports with direct blow to the anterior chest Direct blow to the anterior chest (not specified)	Body or manubrium
Indirect trauma	Distortion of the sternum due to severe hyperflexion-compression of the thoracic vertebrae	Fall on the upper back with severe bending of the thoracic spine Fall from swing, trampoline, gym bar	The upper and middle body of the sternum Eventually resulting in a thoracic spine wedge fracture
	Overuse stress	Repetitive upper body building exercises without a clear trauma Repetitive overstretching during gymnastics	
	Insufficiency stress	Patients with severe thoracic kyphosis, osteoporosis, long-term steroid therapy Postmenopausal women and elderly patients	

fractures on CT scans in respectively 3 out of 103 (2.9%), 7 out of 108 (6.5%), and 5 out of 126 (4.0%) patients. As far as could be derived from the medical literature, fractures of the sternum as a result of chest compressions during resuscitation have never been reported in children [54, 57].

9.3.3 Trauma After Birth: Non-accidental Circumstances

According to the literature, a sternum fracture is considered to carry a high specificity for an inflicted injury/child abuse, especially when no accident is mentioned in the clinical history [58–62]. Hechter et al. however disagree with this statement [4]. They are of the opinion that, although sternum fractures may be rare, they are not very specific for inflicted injuries (child abuse). They performed a retrospective study over a period of 11 years in which they found 12 children with sternum fractures (four children were 2 years or younger, the others 3 years or older). In two children, both below the age of 2, they suspected that the fracture was inflicted (child abuse). Ferguson et al. and Moënne Bühlmann et al. also found that sternum fractures could result from a trauma with a low-energy transfer (see Sect. 9.2.1) [15, 22].

A sternum fracture can be sustained in non-accidental circumstances due to a direct trauma (direct punch or blow to the anterior part of the chest, forceful compression of the chest) or by an indirect trauma with distortion of the sternum due to severe hyperflexion-compression of the thoracic vertebrae (Fig. 9.3a, b).

In young patients that have a skeletal survey for suspicion of non-accidental injury, the small ossification centres of the sternum may simulate healing fractures on the oblique chest radiographs (Fig. 9.4a, b).

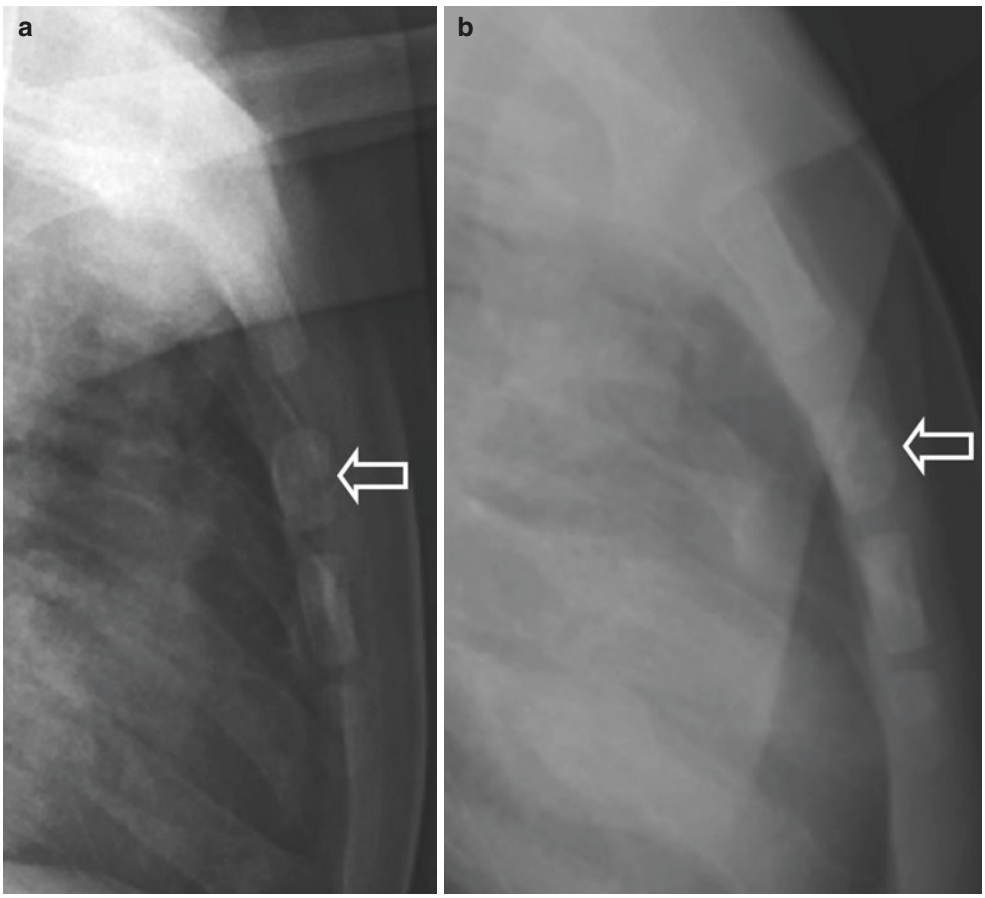


Fig. 9.3 (**a**) Sternum fracture (arrow) in an abused child with multiple other fractures. (**b**) After 14 days the fracture shows healing of the fracture (arrow) (Courtesy of Professor A. Offiah, Sheffield University, United Kingdom)

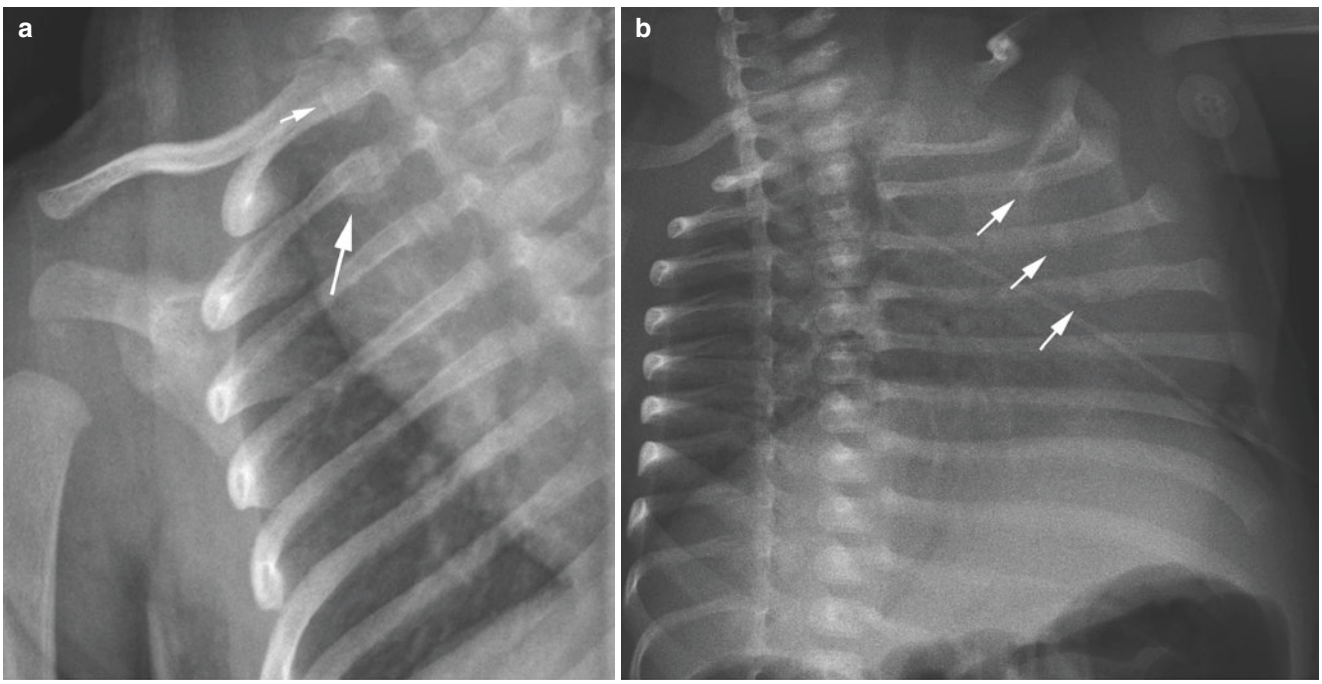


Fig. 9.4 Sternal ossification centres simulating healing rib fractures. (**a**) Seven-week-old boy who had a skeletal survey because his twin brother was suspected of non-accidental injury. The manubrium is well recognized (small arrow) but the second sternal ossification centre is simulating a healing fracture of the second rib (arrow). (**b**) Four-week-old girl suspect for non-accidental injury. The sternal ossification centres simulate multiple healing fractures on the oblique chest radiograph (arrow)

9.4 Differential Diagnosis of Sternum Fractures

9.4.1 Sternal Segment Dislocations

Sternal segment dislocations are extremely rare in the paediatric population. There are only a few cases reported in the medical literature [63–66]. Sternal segment dislocation can be misdiagnosed as a sternum fracture [64]. The most common location of sternal segment dislocation is in the manubriosternal joint, but dislocations have also been described in other sternal segments (Fig. 9.5) [64, 67]. The clinical signs and symptoms of sternal segment dislocations are similar to those of a fracture.

Most dislocations are caused by direct trauma to the chest, but also indirect trauma and non-traumatic causes, e.g. due to osteomyelitis, have also been reported. Wada et al. presented 3 children with sternal segment dislocations: a 4-year-old boy with a direct trauma (blow to the chest), a 3-year-old boy with an indirect trauma, and a 10-year-old boy with osteomyelitis [67]. Murray et al. described a 3-year-old girl with a traumatic sternal segment dislocation after a direct trauma due to a fall on a pole while playing in the park [63]. Nakagawa et al. described a 10-year-old boy who had a sternal segment dislocation after bending backward, while playing dodge ball [64]. He was initially misdiagnosed as having a sternum fracture. The authors were of the opinion that the forces that result in sternal segment dislocation are not strong enough to produce internal injuries. Pawar et al. described a case in a 19-month-old boy who suffered a sternal segment dislocation after a presumed fall from the bed [65].

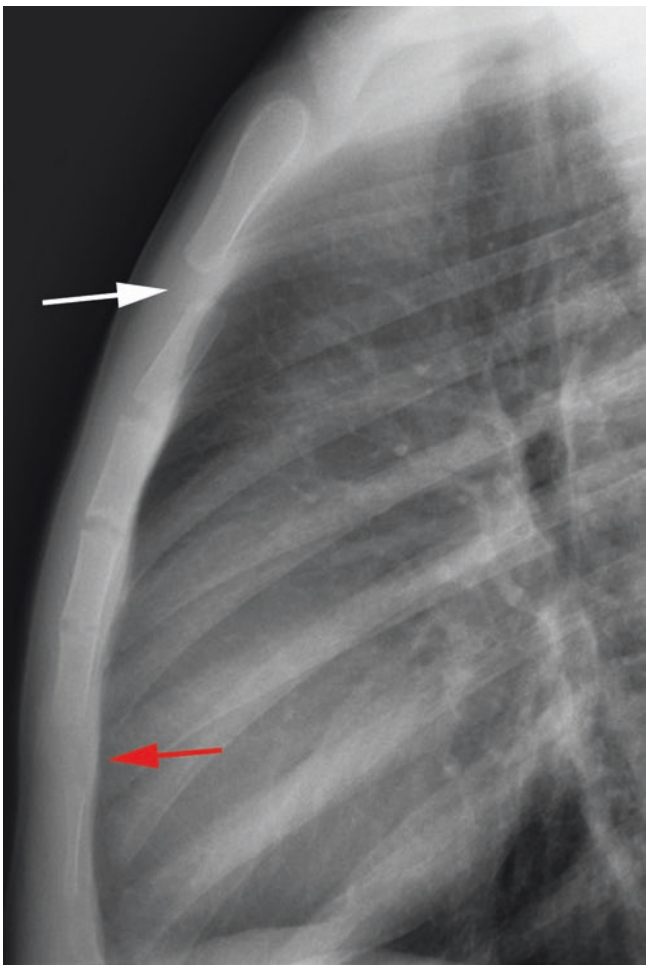


Fig. 9.5 Ten-year-old boy who landed on his buttocks during trampoline jumping which caused high thoracic hyperflexion (i.e. chin on sternum) resulting in chondral fracture dislocation between the manubrium and the second ossification centre of the sternum (arrow). Also note the distraction between the distal ossification centre and the rest of the sternum

There are two types of manubriosternal joint dislocation [68]:

- Type I: backward dislocation of the body caused by a direct force acting on it, for example by direct compression injury to the anterior chest.
- Type II (most common), due to severe hyperflexion-compression of the thoracic vertebrae: indirect forces transmitted to the sternum through the clavicles, the chin, or the upper two ribs causing backward displacement of the manubrium. Norotte et al. described this type in a 14-year-old boy, occurring during an exercise in parallel bars without any fall [69]. Nijs and Broos described the same severe hyperflexion-compression in a 9-year-old gymnast who felt a sharp pain and a sudden click at the manubriosternal joint while stopping his backward swing and initiating his forward movement on the parallel bars [70].

If inflicted, the most likely cause will be a direct punch or blow to the chest, although forceful compression of or an indirect trauma to the chest may also result in a non-accidental dislocation.

9.4.2 Self-limiting Sternal Tumour of Childhood

Sometimes an infant will present with a swelling of unknown origin on the sternum. Most often this will lead to a suspicion of a tumour, especially if it is growing, however to the untrained eye or if other findings suspected for child abuse are present it could also raise the question of the presence of a fracture.

A relatively rare, but easy to diagnose, cause for a sternal swelling is the self-limiting sternal tumour of childhood (SELSTOC) [14, 71–77]. A SELSTOC is a benign aseptic inflammatory process that is located at the level of the sternum. The imaging method of choice is ultrasound which in general will show a quite classic finding of a hypo-echoic

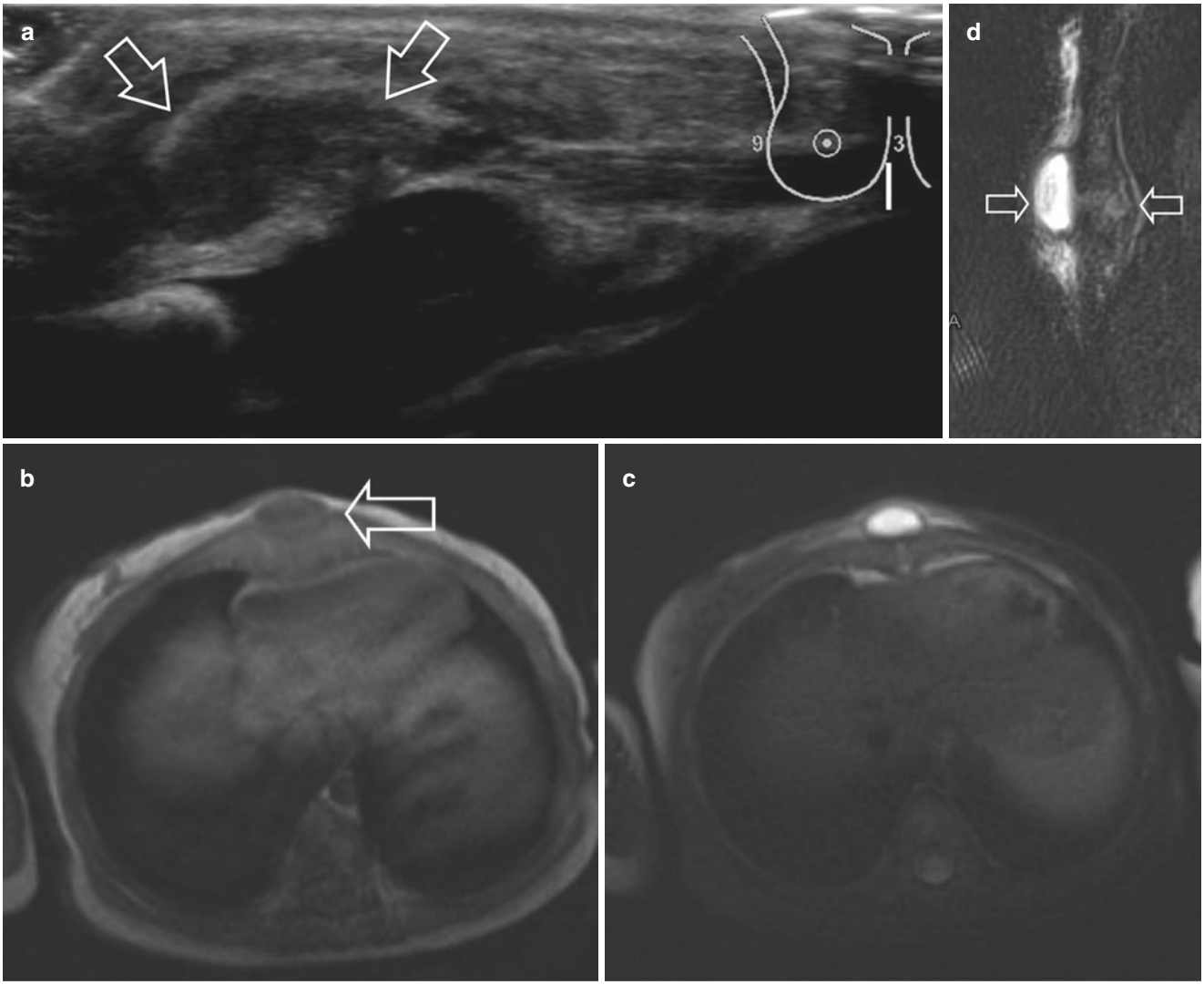


Fig. 9.6 Self-limiting sternal tumour of childhood in a 10-month-old infant who presented with a painless swelling of the sternum. (**a**) Ultrasound shows a hypo-echoic mass (arrows). An outside MRI shows the typical finding of a dumbbell-shaped mass. (**b**) On T1 weighted imaging the mass has a low signal intensity (arrow), (**c**) on T2 weighted imaging it has a high signal intensity. (**d**) On sagittal T2 weighted imaging the dumbbell shape is visible (arrows)

well-circumscribed lesion that extends into the cartilage between the sternal bone centres (Fig. 9.6a). The lesions are often dumbbell-shaped extending posteriorly to the sternum. Although the findings on ultrasound are diagnostic sometimes MRI will be performed, if this is done this will show a lesion with a high signal intensity on T2-weighted images and low/intermediate signal intensity on T1-weighted images (Fig 9.6b). CT is not indicated in these cases.

In the largest published series to date, Winkel et al. presented a series of 14 infants and children (10 boys, median age 16 months, range 7–50 months) [14]. In seven cases there was pain at presentation and a raised temperature in five cases. In eight cases, a succesful wait-and-see policy was initiated leading to full resolution of the mass. In the other cases surgical incision/excision was performed, often in the outside referring hospital. Based on their findings, and supported by other publications, a SELSTOC should be seen as a 'do not touch' lesion.

References

1. Ozsoy IE, Tezcan MA (2019) A rare injury in children: sternum fractures. J Coll Physicians and Surg Pak 29:993–995
2. Schmitt S, Krinner S, Langenbach A, Hennig FF, Schulz-Drost S (2018) Analysis on the age distribution of sternum fractures. Thorac Cardiovasc Surg 66:670–677
3. Ramgopal S, Shaffiey SA, Conti KA (2019) Pediatric sternal fractures from a level 1 trauma center. J Pediatr Surg 54:1628–1631
4. Hechter S, Huyer D, Manson D (2002) Sternal fractures as a manifestation of abusive injury in children. Pediatr Radiol 32:902–906

5. Rosenfeld EH, Lau P, Shah SR, Naik-Mathuria B, Wesson DE, Wakeman DS, Vogel AM (2019) Sternal fractures in children: an analysis of the National Trauma Data Bank. J Pediatr Surg 54:980–983
6. Chalphin AV, Mooney DP (2020) Pediatric sternal fractures: a single center retrospective review. J Pediatr Surg 55:1224–1227
7. Recinos G, Inaba K, Dubose J, Barmparas G, Teixeira PG, Talving P, Plurad D, Green D, Demetriades D (2009) Epidemiology of sternal fractures. Am Surg 75:401–404
8. Scheyerer MJ, Zimmermann SM, Bouaicha S, Simmen HP, Wanner GA, Werner CM (2013) Location of sternal fractures as a possible marker for associated injuries. Emerg Med Int 2013:407589
9. Odell DD, Peleg K, Givon A, Radomislensky I, Makey I, Decamp MM, Whyte R, Gangadharan SP, Berger RL (2013) Sternal fracture: isolated lesion versus polytrauma from associated extrasternal injuries--analysis of 1,867 cases. J Trauma Acute Care Surg 75:448–452
10. Morgenstern M, von Rüden C, Callsen H, Friederichs J, Hungerer S, Bühren V, Woltmann A, Hierholzer C (2016) The unstable thoracic cage injury: the concomitant sternal fracture indicates a severe thoracic spine fracture. Injury 47:2465–2472
11. Jeyam M, Wallace WA, Tibrewal S (2008) Sternal fractures: synopsis of causation. https://www.gov.uk/government/publications/synopsis-of-causation-sternal-fractures. Accessed 2 Aug 2020
12. Bar I, Friedman T, Rudis E, Shargal Y, Friedman M, Elami A (2003) Isolated sternal fracture–a benign condition? Isr Med Assoc J 5:105–106
13. Sadaba JR, Oswal D, Munsch CM (2000) Management of isolated sternal fractures: determining the risk of blunt cardiac injury. Ann R Coll Surg Engl 82:162–166
14. te Winkel ML, Lequin MH, de Bruyn JR, van de Ven CP, de Krijger RR, Pieters R, van den Heuvel-Eibrink MM (2010) Self-limiting sternal tumors of childhood (SELSTOC). Pediatr Blood Cancer 55:81–84
15. Moënne Bühlmann K, Araneda Castiglioni D, Ortega Flores X, Pérez Sánchez C, Escaffi Johnson J, Pérez Matta M, Godoy Lenz J (2019) Clinical and radiological study of sternal fractures in pediatrics. Radiologia 61:234–238
16. Jayle CP, Corbi PJ, Franco S, Menu PM (2005) Destructive sternitis 3 years after blunt chest trauma. Ann Thorac Surg 80:348
17. Kozaci N, Avcı M, Ararat E, Pinarbasili T, Ozkaya M, Etli I, Donertas E, Karakoyun OF (2019) Comparison of ultrasonography and computed tomography in the determination of traumatic thoracic injuries. Am J Emerg Med 37:864–868
18. Fukuhara S, Sameshima T, Matsuo H, Ohashi T (2019) Sternal fracture complicated by a subcutaneous abscess in a 5-year-old boy and diagnosed using point-of-care ultrasound. J Emerg Med 56:536–539
19. You JS, Chung YE, Kim D, Park S, Chung SP (2010) Role of sonography in the emergency room to diagnose sternal fractures. J Clin Ultrasound 38:135–137
20. Jin W, Yang DM, Kim HC, Ryu KN (2006) Diagnostic values of sonography for assessment of sternal fractures compared with conventional radiography and bone scans. J Ultrasound Med 25:1263–1268. quiz 1269–1270
21. Hackl S, Berninger MT, Erichsen C, Lang M, Woltmann A (2018) Sternumfrakturen – Rippenfrakturen. Orthopädie Unfallchirurgie 13:571–592
22. Ferguson LP, Wilkinson AG, Beattie TF (2003) Fracture of the sternum in children. Emerg Med J 20:518–520
23. Livingston DH, Haurer CJ (2004) Trauma to the chest wall and lung. In: Moore EE, Feliciano DV, Mattox KL (eds) Trauma. McGraw-Hill, Philadelphia, pp 507–537
24. Hassan I, Ramagole DA, Janse van Rensburg DC, Grant RC (2010) Sternal fracture in a gymnast: a case report and literature review. SAJSM 2:50–51
25. Hill PF, Chatterji S, DeMello WF, Gibbons JR (1997) Stress fracture of the sternum: an unusual injury? Injury 28:359–361
26. Swarup S, Bonomally K, Ansari MZ (1999) Fracture of the sternum–an unusual case. Eur J Emerg Med 6:71–72
27. Abrahamsen S, Madsen CF (2014) Atraumatic sternum fracture. BMJ Case Reports. Published online 17 Oct 2014. https://doi.org/10.1136/bcr-2014-206683
28. Lin KH, Ponampalam R (2006) Sternum insufficiency fracture presenting as acute chest pain: a case report and review of the literature. Eur J Emerg Med 13:122–124
29. Reuling EM, Jakma TS, Schnater JM, Westerweel PE (2015) Spontaneous sternal fracture due to multiple myeloma requiring extensive surgical repair. BMJ Case Reports. Published online 30 Nov 2015. https://doi.org/10.1136/bcr-2015-211498
30. Sarbay I, Dogan H (2018) A rare cause of chest pain: spontaneous sternum fracture. J Emerg Crit Care Med 2. https://doi.org/10.21037/jeccm.2018.06.03
31. Mitchell EA, Elliott RB (1980) Spontaneous fracture of the sternum in a youth with cystic fibrosis. J Pediatr 97:789–790
32. Latzin P, Griese M, Hermanns V, Kammer B (2005) Sternal fracture with fatal outcome in cystic fibrosis. Thorax 60:616
33. Olmos JM, Pesquera C, Amado JA, Riancho JA, González-Macías J (1990) Pathologic fracture of the sternum in a patient with anorexia nervosa. Revista Clinica Espanola 186:23–25
34. Korovessis P, Sdougos G, Dimas T (1994) Spontaneous fracture of the sternum in a child being treated in a Boston brace for kyphoscoliosis. A case report and review of the literature. Eur Spine J 3:112–114
35. Rostan A (1988) A partial fracture of the sternum in a 10-year-old soccer player. Schweizerische Zeitschrift fur Sportmedizin 36:126
36. Perez FL Jr, Coddington RC (1983) A fracture of the sternum in a child. J Pediatr Orthop 3:513–515
37. Pérez-Martínez A, Marco-Macián A, Gonzálvez-Piñera J, Agustí-Buztke B, Solera Santos G, Goñi-Orayen C, Moya-Marchante M (1996) Cortical fracture of the sternum in a child: an infrequent case. Cirugia Pediatrica 9:130–131
38. DeFriend DE, Franklin K (2001) Isolated sternal fracture–a swing-related injury in two children. Pediatr Radiol 31:200–202
39. Fichtel I, Fernandez FF, Wirth T (2016) Sternal fracture in growing children: a rare and often overlooked fracture? Documentation of four cases. Unfallchirurg 119:570–574
40. Korhonen L, Salokorpi N, Suo-Palosaari M, Pesälä J, Serlo W, Sinikumpu JJ (2018) Severe trampoline injuries: incidence and risk factors in children and adolescents. Eur J Pediatr Surg 28:529–533
41. Sesia SB, Heinrich DM, Kocher GJ, Haecker FM (2018) Treatment of isolated sternal fracture with a vacuum bell in an 8-year-old boy. Interact Cardiovasc Thorac Surg 26:888–889
42. Binder S, Arpe V, Rothschild MA, Banaschak S (2020) Sternal fractures are (almost) only caused by resuscitation, right? And in children? Rechtsmedizin 30:194–197
43. Fortýn K, Kucík J (1970) Fractures of the sternum in children. Rozhledy v chirurgii: mesicnik Ceskoslovenske chirurgicke spolecnosti 49:625–631
44. Kläber V (1979) Sternal fracture in a 12-year-old. Zentralblatt fur Chirurgie 104:244–245
45. Schulte HD (1970) Sternal body fracture in a four year old child. Zentralblatt fur Chirurgie 95:892
46. Unrein HD (1978) Juvenile sternal fractures. Padiatrie und Grenzgebiete 17:199–202
47. Chi YL (1979) Fracture of sternum in children: report of 3 cases (author's transl). Zhonghua wai ke za zhi [Chinese journal of surgery] 17:495
48. Koteles G, Wein G, Szirmay Z (1962) Sternum fractures in childhood. Der Chirurg; Zeitschrift fur alle Gebiete der operativen Medizen 33:373–374

49. Horikawa A, Miyakoshi N, Kodama H, Shimada Y (2007) Insufficiency fracture of the sternum simulating myocardial infarction: case report and review of the literature. Tohoku J Exp Med 211:89–93
50. Loder RT, Schultz W, Sabatino M (2014) Fractures from trampolines: results from a national database, 2002 to 2011. J Pediatr Orthop 34:683–690
51. Martin SL, Stewart RM (1999) Chest and thorax injuries. In: Schenck RC (ed) Athletic training and sports medicine. American Academy of Orthopaedic Surgeons, pp 355–378
52. Robertsen K, Kristensen O, Vejen L (1996) Manubrium sterni stress fracture: an unusual complication of non-contact sport. Br J Sports Med 30:176–177
53. Friberg N, Schmidbauer S, Walther C, Englund E (2019) Skeletal and soft tissue injuries after manual and mechanical chest compressions. Eur Heart J 5:259–265
54. Hoke RS, Chamberlain D (2004) Skeletal chest injuries secondary to cardiopulmonary resuscitation. Resuscitation 63:327–338
55. Lardi C, Egger C, Larribau R, Niquille M, Mangin P, Fracasso T (2015) Traumatic injuries after mechanical cardiopulmonary resuscitation (LUCAS2): a forensic autopsy study. Int J Legal Med 129:1035–1042
56. Koster RW, Beenen LF, van der Boom EB, Spijkerboer AM, Tepaske R, van der Wal AC, Beesems SG, Tijssen JG (2017) Safety of mechanical chest compression devices AutoPulse and LUCAS in cardiac arrest: a randomized clinical trial for non-inferiority. Eur Heart J 38:3006–3013
57. Boz B, Erdur B, Acar K, Ergin A, Türkçüer I, Ergin N (2008) Frequency of skeletal chest injuries associated with cardiopulmonary resuscitation: forensic autopsy. Turkish J Trauma Emerg Surg 14:216–220
58. Bullock DP, Koval KJ, Moen KY, Carney BT, Spratt KF (2009) Hospitalized cases of child abuse in America: who, what, when, and where. J Pediatr Orthop 29:231–237
59. Dwek JR (2011) The radiographic approach to child abuse. Clin Orthop Relat Res 469:776–789
60. Hobbs CJ, Hanks HGI, Wynne JM (1993) Child abuse and neglect – a clinician's handbook. Churchill Livingstone
61. Kleinman PK (1998) Skelet trauma: general considerations. In: Kleinman PK (ed) Diagnostic imaging of child abuse. Mosby, pp 8–25
62. Kogutt MS, Swischuk LE, Fagan CJ (1974) Patterns of injury and significance of uncommon fractures in the battered child syndrome. Am J Roentgenol Radium Therapy, Nucl Med 121:143–149
63. Murray N, Rypens F, Trudel JS, Cantin MA, Miron MC (2017) Traumatic sternal segment dislocation in a 3-year-old girl: sonographic findings. J Clin Ultrasound 45:45–49
64. Nakagawa T, Tsuboi T, Wada A, Nito M, Aruga N, Oiwa K, Masuda R, Iwazaki M (2015) Sternal segment dislocation in a child treated by conservative observation. Tokai J Exp Clin Med 40:27–28
65. Pawar RV, Blacksin MF (2007) Traumatic sternal segment dislocation in a 19-month-old. Emerg Radiol 14:435–437
66. Kusaba A, Saito S (2003) Apophyseal dislocation of the body of the sternum in a child: a case report. J Orthop Trauma 17:126–128
67. Wada A, Fujii T, Takamura K, Yanagida H, Matsuura A, Katayama A (2002) Sternal segment dislocation in children. J Pediatr Orthop 22:729–731
68. Soysal O, Akdemir OC, Ziyade S, Ugurlucan M (2012) Management of sternal segment dislocation in a child with closed reduction. Case Rep Med 2012:676873
69. Norotte G, Peres E, Vanderweyen A, Razafindralasitra P (1997) Segmental sternal dislocation in children. Apropos of a surgically treated case. Revue de chirurgie orthopedique et reparatrice de l'appareil moteur 83:283–285
70. Nijs S, Broos PL (2005) Sterno-manubrial dislocation in a 9-year-old gymnast. Acta Chir Belg 105:422–424
71. Ilivitzki A, Sweed Y, Beck N, Militianu D (2013) Sternal pseudotumor of childhood: don't touch the lesion. J Ultrasound Med 32:2199–2203
72. Adri D, Kreindel T (2019) Self limiting sternal tumors of childhood: two case reports. Radiologia (Engl Ed) 61:167–170
73. Moreira BL, Marchiori E (2020) Self-limiting sternal tumor of childhood: a "do not touch" lesion. J Pediatr 221:260–261
74. Alonso Sánchez J, Gallego Herrero C, García Prieto J, Cruz-Conde MC, Casado Pérez C, Rasero Ponferrada M, Coca Robinot D (2021) Self-limiting sternal tumors of childhood (SELSTOC): a diagnostic challenge. Radiologia (Engl Ed) 63:400–405
75. Fuente-Lucas G, Planells-Alduvin MC, Tallón-Guerola P, Alcalá-Minagorre P (2021) Self limiting sternal tumors of childhood in a 7 months old infant. An Pediatr (Barc) 95:57–59
76. Westerveld G, Laven RJA (2021) [A child with a sternal tumour]. Nederlands tijdschrift voor geneeskunde 165
77. Yamane A, Yasui D, Ichikawa T (2021) A case of self-limiting sternal tumor of childhood. Radiol Case Rep 16:602–603

Scapula

10

Rob A. C. Bilo, Simon G. F. Robben, and Rick R. van Rijn

Contents

10.1 General Aspects of Scapula Fractures

The shoulder joint is composed of the scapula, clavicle, and the head of the humerus. The scapula is part of three joints: the acromioclavicular joint (acromion and clavicula), the glenohumeral joint (glenoid and proximal humerus), and the scapulothoracic joint (anterior scapula and posterior thorax) (Fig. 10.1) [1]. In this constellation, the scapula provides a stable base for movements of the humerus [2].

According to the literature scapula fractures are rare, representing a small percentage of all fractures in adult patients [3, 4]. Based on a 10-year data set, 2002–2012, obtained from the US national trauma data bank Tatro et al. calculated an incidence in a patient population of 1,091,391 patients to be 1.74% [5]. The incidence in children is not known, but is probably even lower than in adults [1]. The low incidence is due to the unique anatomy, the mobility on the chest wall, and the protective nestling in many layers of muscles (supraspinatus, infraspinatus, and subscapularis) and connective tissue [2, 6–9]. However, scapular fractures can also easily be missed on standard radiographs, which partly may be an explanation for the low estimated incidence. Computed tomographic scans, which allow for three-dimensional reconstructions, are considered the gold standard for the diagnosis of and pre-treatment assessment of scapular fractures [10–13]. Because of the (real or supposed) low incidence, only limited evidence-based data is known about paediatric scapula fractures. Most of the known data are derived from adult studies. For the assessment of associated soft-tissue trauma, MRI imaging is indicated [14].

Clinical signs and symptoms include numbness or weakness of shoulder and arm, (severe) pain at rest and while moving, (severe) local swelling/bruising and local abrasions.

Scapula fractures can be classified as isolated scapula fractures without associated injury and hardly, if any, signifi-

R. A. C. Bilo
Veilig Thuis Rotterdam Rijnmond (Center for the Reporting of Child Abuse, Domestic Violence and Elder Abuse), Rotterdam, The Netherlands

S. G. F. Robben
Department of Radiology, Maastricht University Medical Center, Maastricht, The Netherlands

R. R. van Rijn (✉)
Department of Radiology and Nuclear Medicine, Amsterdam UMC, University of Amsterdam, Amsterdam, The Netherlands

Department of Forensic Medicine, Netherlands Forensic Institute, The Hague, The Netherlands
e-mail: r.r.vanrijn@amsterdamumc.nl

R. A. C. Bilo et al. (eds.), *Forensic Aspects of Paediatric Fractures*, https://doi.org/10.1007/978-3-031-12041-1_10

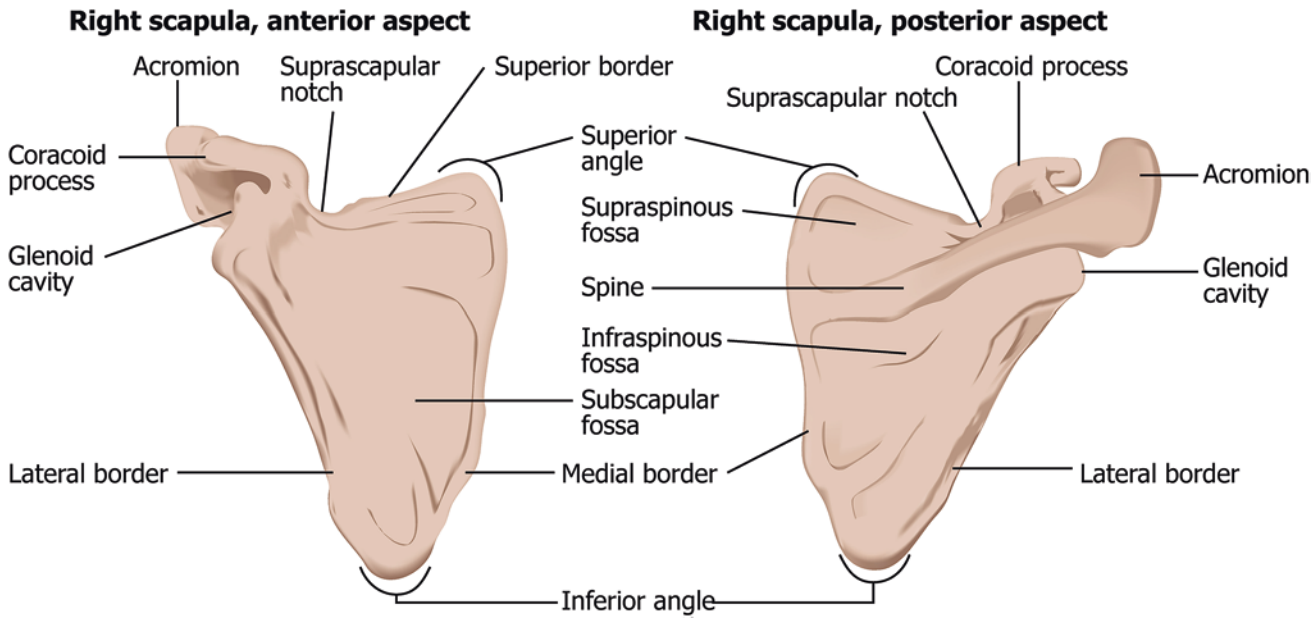


Fig. 10.1 Anatomy of the scapula. (Rendering by A.J. Loeve. Based on a figure by Matteo Mancuso, licenced under the Creative commons 3.0 licence) [62]

Table 10.1 Glenoid cavity fractures [15]

Type	
I	Fractures of either the anterior (Ia) or posterior (Ib) glenoid rim
II–IV	Transverse glenoid fractures with fracture line
II	Exiting inferiorly through the lateral scapular border
III	Superior through or near the notch
IV	Medially through the medial scapular border
V	More than one fracture line. Type V fractures are a combination of the other types
VI	Fractures with severe comminution

Table 10.2 Associated injuries [1, 2, 20]

Skeletal injuries	Spine fractures Skull fractures Rib fractures (Ipsilateral) clavicle fracture Upper extremity fractures
Thoracic injuries	Pulmonary injury Pulmonary contusion Pneumothorax, haemothorax Tracheobronchial rupture
Other injuries	Intracranial haemorrhage Brachial plexus injury Spinal cord injuries Subclavian vessel injury Axillary vessel injury Ruptured viscera

cant threat to life and polytrauma scapula fractures with associated injuries, which will determine the patient's overall outcome. Fractures can occur in one part of several parts of the scapula [1]. The most common fracture in children (and in adults) is the scapular body (45% of all scapula fractures), followed by the glenoid neck (25%), the glenoid cavity (10%), the acromion process (8%), the coracoid process (7%), and finally the scapular spine (5%).

Glenoid neck fractures are classified according to the degree of displacement and angulation. In type I the displacement is less than 1 cm and the angulation less than 40°, in type II more than 1 cm and more than 40° [15]. Glenoid cavity fractures are classified depending on location and degree of comminution (Ideberg classification) (Table 10.1) [15].

Acromion fractures are classified according to the amount of displacement. Type I fractures are non-displaced or minimally displaced, type II fractures are displaced but do not reduce the subacromial space, and type III fractures are displaced and do reduce the subacromial space [15].

The presence of a scapula fracture generally indicates a severe, high-energetic and not seldom life-threatening trauma, involving the upper posterior chest, in which adults as well as children often suffer damage to vital organs as well (Table 10.2) [8, 9, 16–18]. Therefore a complete head-to-toe examination is always indicated, if a scapula fracture is found in a child [1].

According to Weatherford there is a 2–5% associated mortality rate in adults, usually due to pulmonary or intracranial injuries [19]. Shannon et al. found no higher mortality rates in their paediatric patient population with scapula fractures due to high-energy motorized vehicle accidents, but they did find that in their population scapula fractures were

associated with significant morbidity (intracranial haemorrhage, skull fractures, thoracic injury, upper extremity fractures, and spine fractures) compared to control patients [20].

Some extremely rare scapula fracture variants, with or without complications, have been described in paediatric case reports by:

- Blue et al.: fracture of the scapular body in a 13-year-old boy, after being struck by a dump truck, while riding a bicycle [21]. A fragment of the fracture penetrated the thoracic cavity, resulting in a pneumothorax.
- Bowen and Miller: angulated, greenstick fracture of the scapula mimicking scapular winging in a skeletally immature 12-year-old boy [22].
- Park et al.: acromial apophysiolysis in a 14-year-old boy [23].
- Shin et al.: greenstick fracture of the scapular body in a 6-year-old boy, after being struck by a dump truck [24]. The fracture fragment impaled the parenchyma of the left lung, resulting in a pneumothorax.
- Alaia et al.: growth plate injury at the base of the coracoid process in seven males and one female (mean age 15 years): five athletes, two patients with neuromuscular disorders, and 1 subject after a fall [25].
- Miller et al.: bowing type fracture of the scapular tip in a 4-year-old boy [26].

In severe trauma, a scapulothoracic dissociation/traumatic disruption of the scapulothoracic articulation with complete separation of the scapula from the posterior chest wall can occur [1, 27]. This condition is uncommon in children, but has been described in young children, always secondary to major accidental shoulder trauma, sometimes leading to complete upper extremity amputation [28–31].

10.2 Cause of Scapula Fractures

10.2.1 Direct Trauma with High-energy Transfer

Scapula fractures are usually caused by a blunt force trauma (direct blow) on the back or side with a high-energy transfer directly to the scapula during the impact. If the blunt force is spread over a large part of the scapula, crushing of the scapula may happen [4]. A high-energy transfer during impact is considered to be necessary to cause a scapula fracture because only the dorsal aspect of the scapular spine and acromion are situated subcutaneously and the remainder of the scapula is lying deeper and is well protected against a trauma with low energy transfer during the impact [1]. Acromion fractures are usually due to a direct blow to the lateral shoulder [15].

10.2.2 Indirect Trauma

Indirect trauma with axial transmission of the load through the arm to the scapula through falling on the elbow or an outstretched hand, is also described as cause [2, 32]. In this indirect trauma, the humeral head will impact on the glenoid cavity and glenoid rim. Indirect trauma also includes stress fractures caused by overuse (repetitive use) and insufficiency [33–36].

According to Rush glenoid fractures are not only often caused by a direct blow to the lateral shoulder, but also occur due to an indirect trauma, caused by a fall onto a flexed elbow with the humeral head being driven into the glenoid [15]. Whether an anterior or posterior rim fracture occurs, depends on the position of the arm.

10.2.3 Traction Trauma

Traction trauma due to pulling by muscles or ligaments is also known to cause of scapula fractures. In adults scapula fractures due to traction have been described as a result of divergently orientated muscles simultaneously contracting in different directions during electrical shocks or seizures [4].

According to Rush, coracoid fractures are usually an avulsion injury resulting from pull of the acromioclavicular ligaments or the conjoint tendon. Scapulothoracic dissociation is often caused by severe traction [15].

In neonates, fractures of the acromion have been described due to agonist/antagonist contraction of muscles around the shoulder joint. Kalideen and Satyapal prospectively followed 171 neonates with neonatal tetanus, a potentially fatal infection which most often occurs through cutting of the umbilical cord using non-sterile techniques or applying non-sterile traditional remedies to the umbilical cord stump, and found avulsion fractures of the acromion (usually bilateral) in 10 new-borns with severe neonatal tetanus [37, 38]. According to Kalideen and Satyapal, neonatal tetanus will lead to muscle hypertonia and contractions of agonist/antagonist muscles in the shoulder, sometimes resulting in avulsion fractures of the acromion [38].

Coote et al. reported bilateral acromial fractures in an infant with malignant osteopetrosis [39]. The fractures were found one day after the infant had two witnessed seizures. Jacoby et al. described the occurrence of bilateral acromial fractures in a neonate with epileptic encephalopathy [40].

10.2.4 Cause of Scapula Fractures on Specific Locations

Forward and Wallace presented an overview of causes of scapula fractures on specific locations in adults [2]:

- Body or spine fractures are typically caused by a direct blow with high-energy transfer.
- Acromion fractures are usually caused by a direct downward impact to the point of the shoulder.
- Neck fractures are mostly caused by a force applied to the shoulder from the front or from the back.
- Glenoid rim fractures are mostly caused by a load transmitted along the humerus after a fall onto the flexed elbow or the outstretched hand.
- Stellate glenoid fractures are usually caused by a direct blow to the lateral aspect of the shoulder.
- Coracoid process fractures are usually caused by a direct blow or an avulsion.

The data of Forward and Wallace can probably be used in older children and teenagers. There are no data known on the cause of scapula fractures on specific locations in younger children.

10.3 Manner of Scapula Fractures

The occurrence of intrauterine scapula fractures or scapula fractures due to birth is not mentioned in the medical literature.

10.3.1 Trauma After Birth: Accidental Circumstances

Accidental scapula fractures rarely occur in children under the age of 2 years old. In older children, these fractures usually result from a clearly identifiable severe blunt trauma with a high-energy transfer directly to the scapula.

Probably the most common accidental circumstances in adults and in children are traffic accidents, e.g. pedestrian or cyclist versus motor vehicle collisions or high-speed motorcycle of motor vehicle collisions (direct trauma).

Other known circumstances are accidental falls on the shoulder from a significant height or falls on the flexed elbow or an outstretched hand during daily activities (indirect trauma).

Scapula fractures can also occur during sports, due to:

- Direct trauma, e.g. a direct blow with a hockey stick or baseball bat or a body check against the scapula [41, 42]
- Direct and indirect trauma, e.g. a fall directly on the shoulder or on the flexed elbow/outstretched hand, while horseback riding, mountain biking, or skiing [43]
- Overuse (repetitive use) while playing softball/baseball

Crushing of the scapula results from spreading of the blunt force over a larger part of the scapula and can occur in, e.g. traffic accidents with overriding of the trunk, railroad accidents, or forestry accidents [4].

Moon et al. described a rare case of an 11-year-old girl with fractures of the acromion, clavicle, and first rib on the left and contralateral fractures of the first and second ribs. Initially the circumstances stayed unclear, until eventually it was discovered that these fractures were stress fractures caused by a nervous tic consisting of repetitive, vigorous shrugging and translation of the shoulders [36].

10.3.2 Trauma After Birth: Medical and Paramedical Procedures

Scapula fractures have been described as very rare complication of cardiopulmonary resuscitation in adults [44, 45]. As far as could be derived from the medical literature, scapula fractures have never been reported in children as a result of chest compressions during resuscitation.

10.3.3 Trauma After Birth: Non-accidental Circumstances

Several authors consider scapula fractures in a young child to be highly specific for non-accidental circumstances (inflicted injury) [46–52].

Because accidental scapula fractures in children are rare and result from severe direct trauma such as a motor vehicle accident or fall from a great height, any scapula fracture should raise a high suspicion for non-accidental circumstances, if a history of high-energy trauma is lacking (Fig. 10.2).

Inflicted scapula fractures may result from direct trauma or from traction/indirect trauma. A direct blow to the scapula

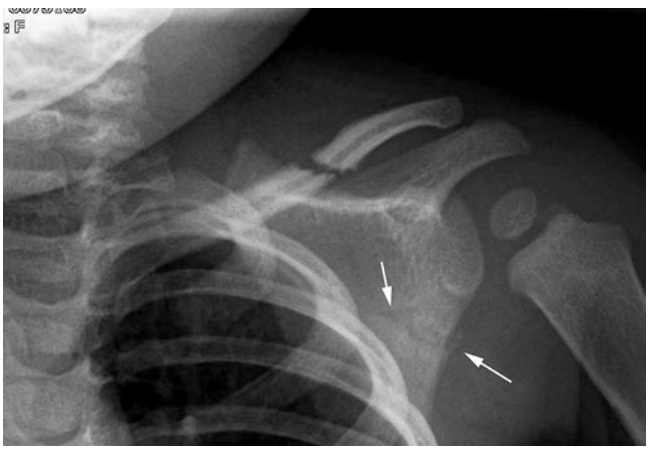


Fig. 10.2 One-year-old boy with a clavicular fracture and a healing fracture of the scapular body. According to the parents he fell from the couch on outstretched hands 2 days ago

may lead to a non-specific linear of 'star burst' fracture [51, 53]. This may happen in a fight or in a physical assault. Fractures of the glenoid fossa or the corpus resulting from direct-impact violence are very rare inflicted injuries [54]. A fracture of the glenoid fossa is usually the result of an indirect trauma due to a fall on the upper arm.

Non-accidental avulsion fractures of the acromion process or, less commonly, fractures of the coracoid process are probably due to violent arm traction or shaking. The acromion is the most prevalent location for inflicted injuries due to traction; either a fracture is found or there may be a dislocation of the acromioclavicular joint (Fig. 10.3). Fragmentation of the acromion, avulsion fractures of the acromion and, less frequently, fractures of the coracoid process or other parts of the scapula may be found after traction trauma. This happens, for example when a child is shaken, when the arms are pulled with a great deal of force, or when the arm is turned onto the back with brute force [51, 55]. When an inflicted acromion fracture is suspected in a child, one should always be aware of other associated fractures such as clavicle, glenoid fossa, coracoid, proximal humerus, or the upper ribs (Figs. 10.4 and 10.5).

Fig. 10.3 Bucket-handle fracture of the acromion (arrow) in a 3-month-old infant who presented with multiple bruises. Skeletal survey detected over 20 traumatic skeletal lesions among which rib fractures of different ages and metaphyseal corner fractures of tibia and femur. Also note the metaphyseal corner fracture of the proximal humerus

Fig. 10.4 One-month-old girl with a tibial shaft fracture without a history of trauma. Skeletal survey revealed multiple metaphyseal corner fractures of the arms and legs (including inferior angle of right scapula) and right-sided acromial fracture and distal clavicular fracture. No rib fractures were present

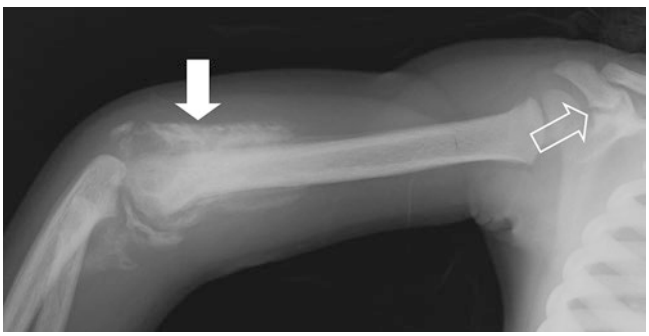


Fig. 10.5 Humerus fracture with extensive callus formation (arrow) and acromion fracture (open arrow) in abused child

If there is a suspicion that the fracture was inflicted a complete age appropriate 'non-accidental injury' medical workup is indicated.

10.4 Differential Diagnosis of Scapula Fractures

In the differential diagnosis of scapular fractures in children (especially of the acromion), one should be aware of the possible presence of an accessory ossifying nuclei at the end of the acromion, the so-called os acromiale, since this may mimic a fracture (Fig. 10.6) [56–59]. The acromion is built up of three elements, from posterior to anterior: the metacromion, the mesacromion, and the preacromion (Fig. 10.7). These elements fuse to become one acromial bone which subsequently fuses with the scapular spine. Fusion should in general be completed between the ages of 15–18 years, although fusion can be delayed to a later age or may even never occur [59, 60]. Kleinman and Spevak described the presence of an os acromiale in 10 out of 78 infants, who died of sudden infant death syndrome [58]. In six children the finding was bilaterally located and in four unilaterally. Currarino and Prescott described the finding in six children, 2–19 months of age [56]. In four children the finding was diagnosed as a fracture and in two as an anatomical variant. The presence in adults has been reported to be around 3% equally distributed between sexes [61].

Compared to an os acromiale a genuine fracture will show a sharply defined edge compared to the regular bone. Also, when healing there are signs of callus formation in a fracture, which will not be seen in an accessory ossifying nucleus.

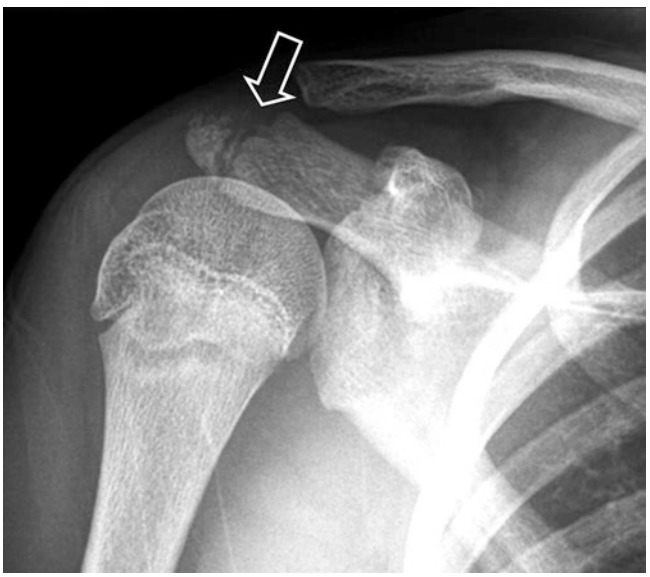


Fig. 10.6 Os acromiale (arrow) as a normal variant in a healthy child

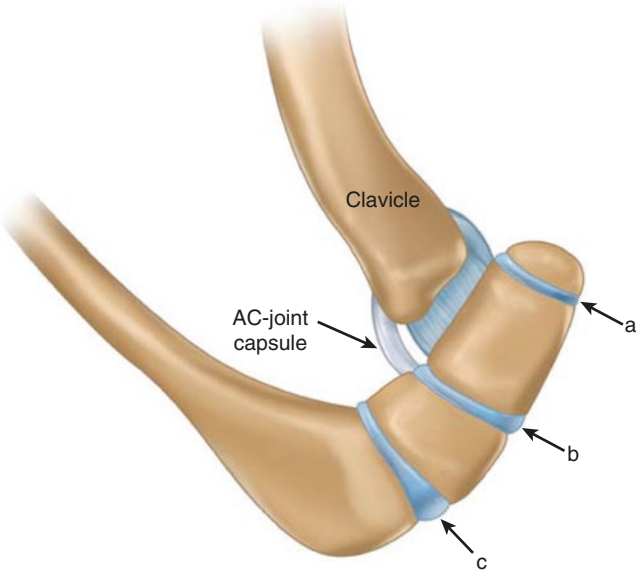


Fig. 10.7 The types of os acromiale: (**a**) the space between the os pre-acromiale and the acromion, (**b**) the space between the os meso-acromiale and the acromion, and (**c**) the space between the os meta-acromiale and the acromion

References

1. Schwartz BS, Pensy R, Eglseder A, Abzug JM (2014) AC dislocations, SC dislocations, and scapula fractures. In: Abzug JM, Kozin SH, Zlotolow DA (eds) The pediatric upper extremity. Springer, pp 1277–1298
2. Forward DP, Wallace WA (2008) Scapula fractures: synopsis of causation. Accessed 10 Aug 2021
3. Voleti PB, Namdari S, Mehta S (2012) Fractures of the scapula. Adv Orthop 2012:903850
4. Wiedemann E, Euler E, Pfeifer K (2000) Scapular fractures. In: Wulker N, Mansat M, Fu FH (eds) Shoulder surgery, an illustrated textbook. Martin Dunitz, pp 504–510
5. Tatro JM, Schroder LK, Molitor BA, Parker ED, Cole PA (2019) Injury mechanism, epidemiology, and hospital trends of scapula fractures: a 10-year retrospective study of the National Trauma Data Bank. Injury 50:376–381
6. Ada JR, Miller ME (1991) Scapular fractures. Analysis of 113 cases. Clin Orthop Relat Res:174–180
7. Imatani RJ (1975) Fractures of the scapula: a review of 53 fractures. J Trauma 15:473–478
8. McGinnis M, Denton JR (1989) Fractures of the scapula: a retrospective study of 40 fractured scapulae. J Trauma 29:1488–1493
9. Wilber MC, Evans EB (1977) Fractures of the scapula. An analysis of forty cases and a review of the literature. J Bone Joint Surg Am 59:358–362
10. Berritto D, Pinto A, Russo A, Urraro F, Laporta A, Belfiore MP, Grassi R (2018) Scapular fractures: a common diagnostic pitfall. Acta Biomed 89:102–110
11. Ramponi D, White T (2015) Fractures of the scapula. Adv Emerg Nurs J 37:157–161
12. Haapamaki VV, Kiuru MJ, Koskinen SK (2004) Multidetector CT in shoulder fractures. Emerg Radiol 11:89–94
13. Ng GP, Cole WG (1994) Three-dimensional CT reconstruction of the scapula in the management of a child with a displaced intra-articular fracture of the glenoid. Injury 25:679–680

14. Roedl JB, Morrison WB, Ciccotti MG, Zoga AC (2015) Acromial apophysiolysis: superior shoulder pain and acromial nonfusion in the young throwing athlete. Radiology 274:201–209
15. Rush J Scapula fractures. https://posna.org/Physician-Education/Study-Guide/Scapula-Fractures. Accessed 29 July 2020
16. Livingston DH, Hauser CJ (2003) Trauma to the chest wall and lung. In: Moore EE, Feliciano DV, Mattox KL (eds) Trauma. McGraw-Hill
17. McGahan JP, Rab GT, Dublin A (1980) Fractures of the scapula. J Trauma 20:880–883
18. Thompson DA, Flynn TC, Miller PW, Fischer RP (1985) The significance of scapular fractures. J Trauma 25:974–977
19. Weatherford B (2016) Scapula fractures. https://www.orthobullets.com/trauma/1013/scapula-fractures. Accessed 16 July 2020
20. Shannon SF, Hernandez NM, Sems SA, Larson AN, Milbrandt TA (2019) High-energy pediatric scapula fractures and their associated injuries. J Pediatr Orthop 39:377–381
21. Blue JM, Anglen JO, Helikson MA (1997) Fracture of the scapula with intrathoracic penetration. A case report. J Bone Joint Surg Am 79:1076–1078
22. Bowen TR, Miller F (2006) Greenstick fracture of the scapula: a cause of scapular winging. J Orthop Trauma 20:147–149
23. Park KJ, Kim YM, Kim DS, Choi ES, Shon HC, Jeong JJ (2015) Avulsion fracture of the acromial physis in a 14-year-old boy: a case report. Arch Orthop Trauma Surg 135:223–225
24. Shin SJ, Wang SI, Kim JR (2016) Lung injury caused by greenstick fracture of the scapular body in a 6-year-old boy. Skelet Radiol 45:555–558
25. Alaia EF, Rosenberg ZS, Rossi I, Zember J, Roedl JB, Pinkney L, Steinbach LS (2017) Growth plate injury at the base of the coracoid: MRI features. Skelet Radiol 46:1507–1512
26. Miller C, Grainger AJ, Phillips RS, Sabouni MY, Kraft JK (2018) Bowing fracture of the inferior angle of the scapula, a difficult diagnosis. Pediatr Radiol 48:146–149
27. Morris CS, Lloyd T (1990) Case report 642: Traumatic scapulothoracic dissociation in a child. Skelet Radiol 19:607–608
28. An HS, Vonderbrink JP, Ebraheim NA, Shiple F, Jackson WT (1988) Open scapulothoracic dissociation with intact neurovascular status in a child. J Orthop Trauma 2:36–38
29. Lovejoy J, Ganey TM, Ogden JA (2009) Scapulothoracic dissociation secondary to major shoulder trauma. J Pediatr Orthop B 18:131–134
30. Nettrour LF, Krufky EL, Mueller RE, Raycroft JF (1972) Locked scapula: intrathoracic dislocation of the inferior angle. A case report. J Bone Joint Surg Am 54:413–416
31. Oreck SL, Burgess A, Levine AM (1984) Traumatic lateral displacement of the scapula: a radiographic sign of neurovascular disruption. J Bone Joint Surg Am 66:758–763
32. Goss TP, Owens BD (2006) Fractures of the scapula: diagnosis and treatment. In: Iannotti JP, Williams GR (eds) Disorders of the shoulder: diagnosis and management. Lippincott Williams & Wilkins, pp 794–795
33. Donovan M, Attia MW (2018) An unusual cause of an isolated scapula fracture. JAAPA 31:26–28
34. Hart RA, Diamandakis V, El-Khoury G, Buckwalter JA (1995) A stress fracture of the scapular body in a child. Iowa Orthop J 15:228–232
35. Marcano AI, Samitier G, Wright TW, Farmer KW (2014) Stress fracture of second rib and scapular spine in a female softball player. Curr Sports Med Rep 13:314–318
36. Moon BS, Price CT, Campbell JB (1998) Upper extremity and rib stress fractures in a child. Skelet Radiol 27:403–405
37. World Health Organisation (WHO) (2021) Vaccine-preventable diseases – neonatal tetanus. https://www.who.int/immunization/monitoring_surveillance/burden/vpd/WHO_SurveillanceVaccinePreventable_14_NeonatalTetanus_R1.pdf. Accessed 10 Aug 2021
38. Kalideen JM, Satyapal KS (1994) Fractures of the acromion in tetanus neonatorum [corrected]. Clin Radiol 49:563–565
39. Coote JM, Steward CG, Grier DJ (2000) Bilateral acromial fractures in an infant with malignant osteopetrosis. Clin Radiol 55:70–72
40. Jacoby J, Nicholls AJ, Clarke NM, Fairhurst J (2011) Bilateral acromial fractures in a neonate with epileptic encephalopathy. Pediatr Radiol 41:788–789
41. Echlin PS, Plomaritis ST, Peck DM, Skopelja EN (2006) Subscapularis avulsion fractures in 2 pediatric ice hockey players. Am J Orthop (Belle Mead NJ) 35:281–284
42. Kaminsky SB, Pierce VD (2002) Nonunion of a scapula body fracture in a high school football player. Am J Orthop (Belle Mead NJ) 31:456–457
43. Banerjee AK, Field S (1985) An unusual scapular fracture caused by a water skiing accident. Br J Radiol 58:465–467
44. Abramowitz Y, Aviram G, Roth A (2010) Scapular facture following cardiopulmonary resuscitation. Resuscitation 81:498–499
45. Kam AC, Kam PC (1994) Scapular and proximal humeral head fractures. An unusual complication of cardiopulmonary resuscitation. Anaesthesia 49:1055–1057
46. Bullock DP, Koval KJ, Moen KY, Carney BT, Spratt KF (2009) Hospitalized cases of child abuse in America: who, what, when, and where. J Pediatr Orthop 29:231–237
47. Dwek JR (2011) The radiographic approach to child abuse. Clin Orthop Relat Res 469:776–789
48. Hobbs CJ, Hanks HGI, Wynne JM (1993) Child abuse and neglect – a clinician's handbook. Churchill Livingstone
49. Jayakumar P, Barry M, Ramachandran M (2010) Orthopaedic aspects of paediatric non-accidental injury. J Bone Joint Surg Br 92:189–195
50. Kleinman PK (1998) Skelet trauma: general considerations. In: Kleinman PK (ed) Diagnostic imaging of child abuse. Mosby, pp 8–25
51. Kogutt MS, Swischuk LE, Fagan CJ (1974) Patterns of injury and significance of uncommon fractures in the battered child syndrome. Am J Roentgenol Radium Therapy, Nucl Med 121:143–149
52. Love JC, Sanchez LA (2009) Recognition of skeletal fractures in infants: an autopsy technique. J Forensic Sci 54:1443–1446
53. Swischuk LE (1992) Radiographic signs of skeletal trauma. In: Ludwig S, Kornberg AE (eds) Child abuse – a medical reference. Churchill Livingstone, pp 151–174
54. Merten DF, Radlowski MA, Leonidas JC (1983) The abused child: a radiological reappraisal. Radiology 146:377–381
55. Kleinman PK (2015) Diagnostic imaging of child abuse. Cambridge, Cambridge
56. Currarino G, Prescott P (1994) Fractures of the acromion in young children and a description of a variant in acromial ossification which may mimic a fracture. Pediatr Radiol 24:251–255
57. Keats TE, Anderson MW (2012) Atlas of normal roentgen variants which may simulate disease. Saunders
58. Kleinman PK, Spevak MR (1991) Variations in acromial ossification simulating infant abuse in victims of sudden infant death syndrome. Radiology 180:185–187
59. You T, Frostick S, Zhang WT, Yin Q (2019) Os acromiale: reviews and current perspectives. Orthop Surg 11:738–744
60. Barbier O, Block D, Dezaly C, Sirveaux F, Mole D (2013) Os acromiale, a cause of shoulder pain, not to be overlooked. Orthop Traumatol Surg Res 99:465–472
61. Rovesta C, Marongiu MC, Corradini A, Torricelli P, Ligabue G (2017) Os acromiale: frequency and a review of 726 shoulder MRI. Musculoskelet Surg 101:201–205
62. Mancuso M (2020) Evaluation and robotic simulation of the glenohumeral joint. Faculté des sciences et techniques de l'ingénieur. École polytechnique fédérale de Lausanne, Lausanne

Pelvis

11

Rob A. C. Bilo, Simon G. F. Robben,
Ingrid M. B. Russel-Kampschoer, and Rick R. van Rijn

Contents

11.1 General Aspects of Pelvic Fractures

The pelvis consists of the two paired hip bones (pubic bone, ischial bone, and iliac bone), connected to each other anteriorly by the pubic symphysis and posteriorly by the os sacrum/coccyx (Fig. 11.1). The pelvis supports the spine and the weight of the upper body, while sitting and standing, it also transfers the weight of the upper body to the lower limbs while standing, and it protects the organs in the pelvis [1].

Pelvic ring fractures in skeletally immature paediatric and adolescent patients are uncommon due to the relative malleability of the pelvis and they account for about 0.3–4% of all fractures in children [2]. According to Nodzo et al., paediatric pelvic fractures account for approximately 2.4–5.5% of annual admissions at large level I trauma centres [3]. Of all paediatric pelvic fractures acetabular fractures account for 1–15% of cases [3–5].

Pelvic fractures can be classified according to Torode and Zieg [6] (Fig. 11.2a–d) or by using the more elaborate modified Tile AO Müller classification (AO stands for Arbeitsgemeinschaft für Osteosynthesefragen) (Table 11.1) [8]. Fractures of the acetabulum are not included in these classifications. Classification of fractures is mostly used to either guide protocol-based medical treatment or for research purposes, in a forensic setting a verbal description is more likely useful.

Although pelvic fractures are relatively rare in children, the finding of a pelvic fracture in a child may indicate serious other injuries. Simple ring fractures (in children mostly due

R. A. C. Bilo
Veilig Thuis Rotterdam Rijnmond (Center for the Reporting of Child Abuse, Domestic Violence and Elder Abuse), Rotterdam, The Netherlands

S. G. F. Robben
Department of Radiology, Maastricht University Medical Center, Maastricht, The Netherlands

I. M. B. Russel-Kampschoer
Department of Paediatrics, University Medical Center Utrecht, Utrecht, The Netherlands

R. R. van Rijn (✉)
Department of Radiology and Nuclear Medicine, Amsterdam UMC, University of Amsterdam, Amsterdam, The Netherlands

Department of Forensic Medicine, Netherlands Forensic Institute, The Hague, The Netherlands
e-mail: r.r.vanrijn@amsterdamumc.nl

R. A. C. Bilo et al. (eds.), *Forensic Aspects of Paediatric Fractures*, https://doi.org/10.1007/978-3-031-12041-1_11

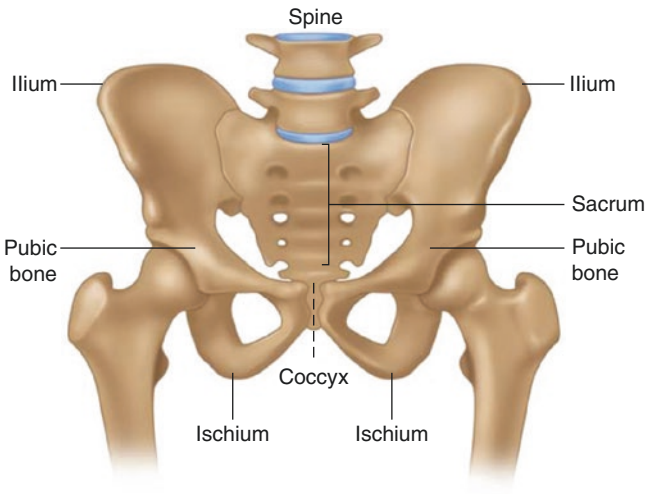


Fig. 11.1 Normal anatomy of the pelvis

to high-energy transfer in pedestrian versus motor vehicle collisions) (Torode and Zieg type 3) are the most common fractures in children (almost 50% of all pelvis fractures in children) [9]. Leonard et al. (2011) found that in their cohort of cases 82% sustained one or more associated injuries (head injuries represented 25% and orthopaedic/skeletal injuries 33% of all associated injuries) [9]. They are of the opinion that this type of fracture is a reliable marker for severe trauma. Sink and Flynn stated that 58–87% of children with pelvic ring fractures will have associated injuries due to the high-energy trauma that caused the pelvis fracture (Table 11.2) [11]. About 20% of paediatric polytrauma victims will have pelvic ring injuries.

Complications may occur (Table 11.3). Mortality is reported in paediatric and adolescent patients with pelvis fractures in up to 25% with an average of 6.4% [12]. The mortality rate in children with pelvis fractures, however, is much lower

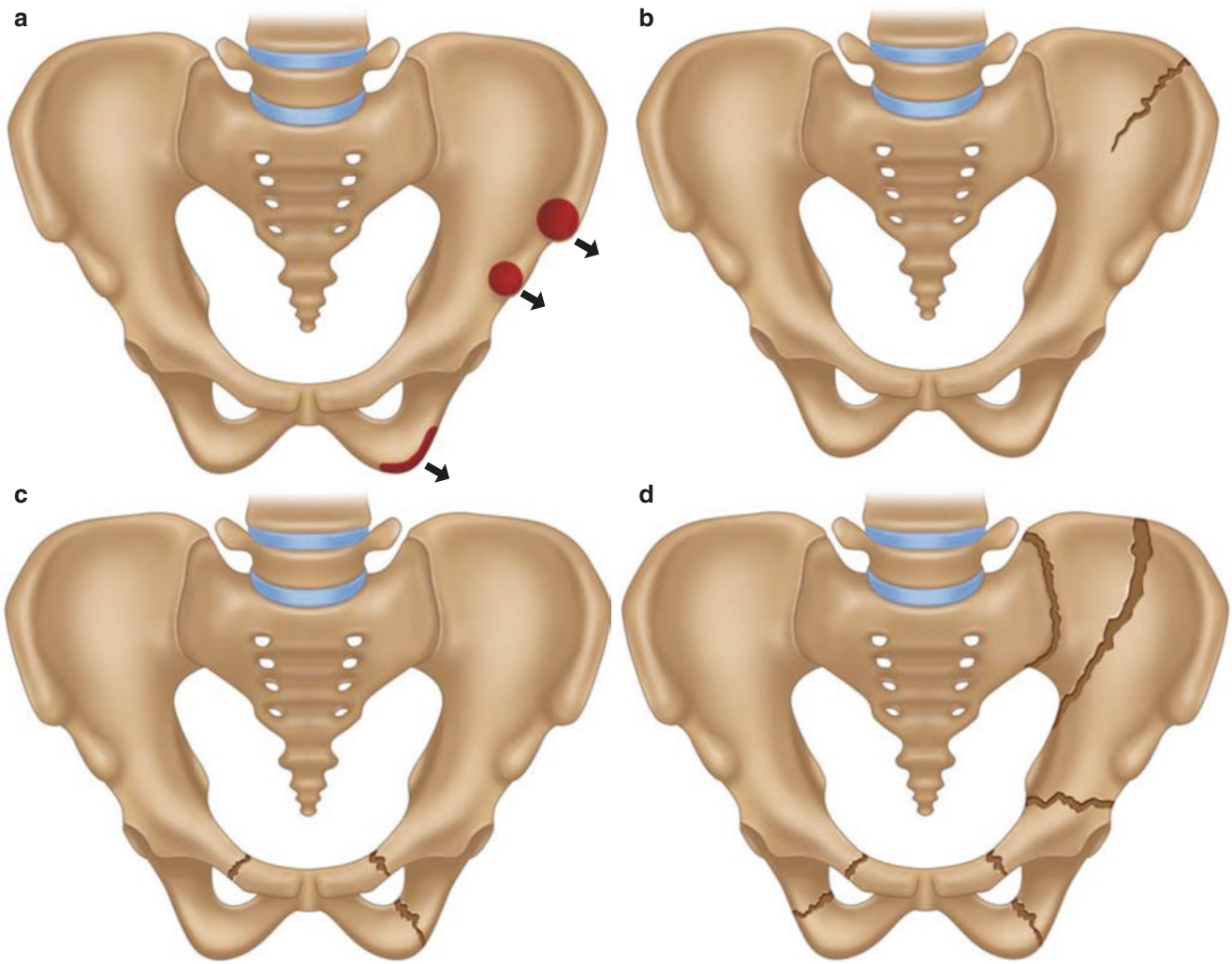


Fig. 11.2 Torode and Zieg classification of pelvis fractures, (**a**) Type 1 avulsion fracture, (**b**) Type 2 iliac wing fracture, (**c**) Type 3 simple ring fracture (stable), and (**d**) Type 4 ring disruption fracture (unstable)

Table 11.1 Modified Tile AO Müller classification [7]

Type	Description
Type A: Stable—posterior arch is intact	A1: fracture does not involve the pelvic ring (avulsion fracture or fracture of the iliac wing)
	A1.1: iliac spine
	A1.2: iliac crest
	A1.3: ischial tuberosity
	A2: stable or minimally displaced fracture of the pelvic ring - A2.1: iliac wing fractures - A2.2: unilateral fracture of anterior arch - A2.3: bifocal fracture of anterior arch
	A3: transverse fracture of the sacrum - A3.1: sacrococcygeal dislocation - A3.2: sacrum undisplaced - A3.3: sacrum displaced
Type B: Rotationally unstable, vertically stable—incomplete disruption of the posterior arch	B1: open book injury (external rotation)
	B1.1: sacroiliac joint, anterior disruption
	B1.2: sacral fracture

Table 11.2 Additional injuries, associated with high-energy transfer trauma [5, 9, 10]

CNS and visceral injuries	• In more than 50 percent of children with pelvic fractures • Probably due to the higher energy required to fracture the more elastic pelvis in children
Urogenital injuries (e.g. bladder or urethral rupture)	• Especially in a fracture of the ring with segmental instability (Torode type IV)
Fractures and dislocations	• Femoral head fractures/dislocations, associated with acetabular fractures
Life-threatening haemorrhage	Rarely

Table 11.3 Potential complications of pelvic fractures [4, 5, 10]

Leg length discrepancy
Growth disturbance of the acetabulum due to injury of the triradiate cartilage, resulting in acetabular dysplasia, hip subluxation, or hip joint incongruity
Gait disturbance
Persistent low back pain
Osteonecrosis of the femoral head after acetabular fractures associated with hip dislocation
Myositis ossificans
Neurologic deficits secondary to sciatic, femoral, and/or lumbosacral plexus nerve injuries
Injuries to blood vessels inside the pelvis

than that in adults [13]. If a child with a pelvis fracture dies, this usually will be due to associated head or visceral injury in polytrauma and only rarely due to exsanguination from damaged blood vessels inside the pelvis [5, 10].

11.2 Cause of Pelvic Fractures

All pelvic fractures result from trauma, either in normal bone or in weakened bone. The type of fracture depends on the cause (mechanism), the amount of energy that is transferred, and the bone strength. Pelvic fractures in normal bone are almost always caused by a blunt force trauma with a high transfer of energy (high-energy trauma—impact trauma or compression/crushing trauma), although some types of pelvic fractures (apophyseal avulsion fractures, stress fractures, and insufficiency fractures) are caused by a trauma with a lower energy transfer (overload in normal or weakened bone) [5].

11.2.1 Direct Trauma with High-Energy Transfer

There is no literature available on paediatric direct trauma. All data available are based on adults and included here. According to the Young–Burgess classification of pelvic fractures in adults, there are four major types of blunt force trauma with possible high-energy transfer, resulting in specific fractures of the pelvis (Table 11.4 and Fig. 11.3) [14–16]. Acetabulum fractures are not included in this classification.

11.2.2 Indirect Trauma with Low(er) Energy Transfer: Overuse Fractures

There are two types of overuse fractures of the pelvis: apophyseal avulsion fractures, due to traction (a.k.a. apophyseal stress injuries), and fatigue fractures, due to overload either in normal (stress fractures) or in weakened (insufficiency fractures) bone [17]. This type of fractures is the result of a mismatch between on the one hand the burden of activity on growing bone and cartilage and on the other hand their intrinsic biomechanical properties and the intrinsic ability of the bone to repair itself [18, 19].

11.2.2.1 Apophyseal Avulsion Fractures

Apophyseal avulsion fractures are more common in skeletally immature children and adolescents and are commonly recognized overuse fractures of the pelvis in this group of patients [17]. According to Jaimes et al., the cartilage of the growth plate of the epiphyses and apophyses is the weakest structure of the developing skeleton [18]. According to Kjelling, the apophysis and growth plate are most vulnerable at times of growth acceleration [20]. McKinney and Roth found that these fractures are almost exclusively diagnosed in patients between 14 and 25 years of age [21]. Calderazzi et al. found a mean age of 14.5 years [22].

Table 11.4 Young–Burgess classification of pelvic fractures [14–16]

Type	Mechanism: a force that is	Radiological findings/injuries
Anterior-posterior compression	Directed from anterior to posterior	'Open book' fracture: diastasis of the pubic symphysis or vertical fracture of the pubic rami
• Type I		Diastasis of the symphysis under 2.5 cm No significant posterior ring injury
• Type II		Diastasis of the symphysis over 2.5 cm Diastasis of the anterior sacroiliac joint Disruption of sacrospinous and sacrotuberous ligaments
• Type III		Disruption of anterior and posterior sacroiliac ligaments (sacroiliac dislocation) Disruption of sacrospinous and sacrotuberous ligaments Associated with vascular injury
Lateral compression	Directed from lateral to medial	Transverse fracture of the pubic rami
• Type I		Compression fractures of the pubic rami (superior pubic ramus and inferior pubic ramus) and ipsilateral anterior sacral ala No ligament disruption
• Type II		Rami fracture and ipsilateral posterior ilium fracture dislocation ('crescent' fracture) Rupture of the posterior sacroiliac ligament
• Type III		Ipsilateral lateral compression and contralateral anterior-posterior compression ('windswept' pelvis)
Vertical shear	Directed in posterior and superior direction (significant axial loading, delivered over one hemipelvis or both hemipelves)	Vertical fracture of the pubic rami Vertical/superior hemipelvis displacement, most often through sacroiliac joint, sometimes through iliac wing or os sacrum A.k.a. Malgaigne fracture
Complex	A combination of any of the three primary mechanisms, e.g. • Lateral and anterior-posterior compression • Lateral compression and vertical shear	Complex fracture pattern, often massive injuries

Avulsion fractures of the pelvis occur in a trauma with a lower energy transfer, compared to the energy transfer in the fractures [10, 17, 20, 23]. Because of the thick surrounding periosteum the fractures will not become widely displaced [23].

According to Shah et al. and Calderazzi et al., apophyseal avulsion injuries most often occur due to forceful and repetitive either concentric or eccentric muscle contractions (Table 11.5), but may also result from excessive passive stretching/lengthening acting on not yet ossified growth plates, or from activities requiring running, kicking, or rapid directional change [22, 24]. They also found, based on a review, that avulsion fractures of the anterior inferior iliac spine were the most common injury (46%), followed by anterior superior iliac spine avulsion (32%), ischial tuberosity avulsion (12%), and iliac crest avulsion (11%) (Fig. 11.4a, b).

11.2.2.2 Fatigue Fractures in Normal Bone: Stress Fractures

A stress fracture is a fracture that occurs in a bone after the bone has been subjected to repeated (often cyclic) tensile or compressive stresses, rather than one sudden impact trauma. None of these stresses would individually be large enough to cause a fracture in a person without an underlying disorder with increased bone fragility, but the intrinsic ability of the bone to repair itself is exceeded by the repetitive character of the loading [19, 26].

Stress fractures in a pelvic bone are rare, compared to more common stress fractures, e.g. of the lower extremities (tibia, metatarsal bones) and occur as a result of weight bearing activities that put repetitive stress on the pelvic bones, usually due to sport, e.g. long-distance running, sprinting, jumping, or (ballet) dancing. In adults it often occurs when a person quickly increases the duration and intensity of a physical activity without gradually building up endurance or when a person changes training conditions [27, 28]. Occasionally they may occur due to repetitive kicking in sports such as football or soccer [28]. It may also occur in persons with a lack of physical activities, who suddenly start training.

Stress fractures of a pelvic bone in children are usually located in the sacrum and in the pubis, but can be found in other bones of the pelvis [18]. According to Portela and Santos, there usually is no history of a preceding trauma [29]. The clinical presentation is vague pain in the abdomen, low back pain, and buttock tenderness. Due to the weight bearing function of the sacrum, the pain intensifies with physical activities and reduces with rest [18, 29]. According to Jaimes et al., stress injuries of pelvis usually are seen in young runners and, less frequently, in adolescents, participating in volleyball, ballet, or gymnastics [18].

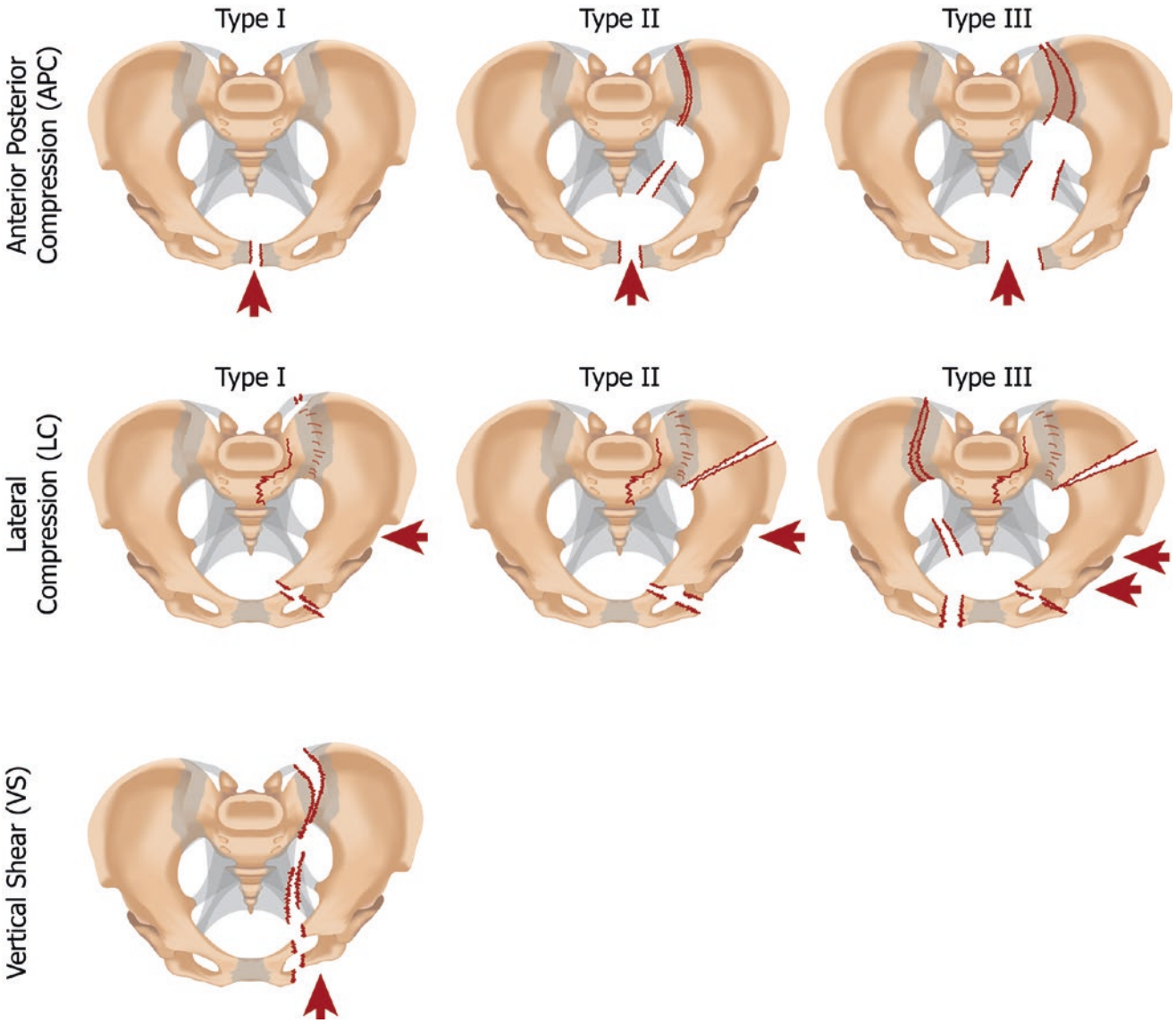


Fig. 11.3 Young–Burgess classification of pelvis fractures (rendering by A.J. Loeve, adapted from the figure released into the public domain by Alton and Gee [14])

Table 11.5 Overview of mechanisms of the most common avulsion injuries of the pelvis [17, 18, 22, 24, 25]

Location	Origin of muscles and avulsion mechanism
Anterior inferior iliac spine	• Origin of the m. rectus femoris • Eccentric contraction of the m. rectus femoris, most commonly due to forceful extension of the hip while the knee is flexed (often seen in sprinters and athletes involved in kicking)
Anterior superior iliac spine	• Origin of the m. sartorius and tensor fascia lata • Sudden forceful contraction of the m. sartorius with the hip in extension and the knee flexed (most common in running athletes, but may also occur with kicking)
Ischial tuberosity	• Origin of the hamstrings • Forceful flexion of the hip with the knee extended • Eccentric overload of the hamstring muscles with forceful contraction against resistance, which places strain at the hamstring origins
Iliac crest	• Very rare • Origin of anterior abdominal wall muscles • Usually a quick change of direction.
Corpus and pubic symphysis	• Uncommon • Origin of m. gracilis and other thigh adductors • Usually from chronic overuse • Acute avulsions are rare, but may occur in young athletes usually with forceful contraction against resistance. Usually seen within the spectrum of athletic pubalgia

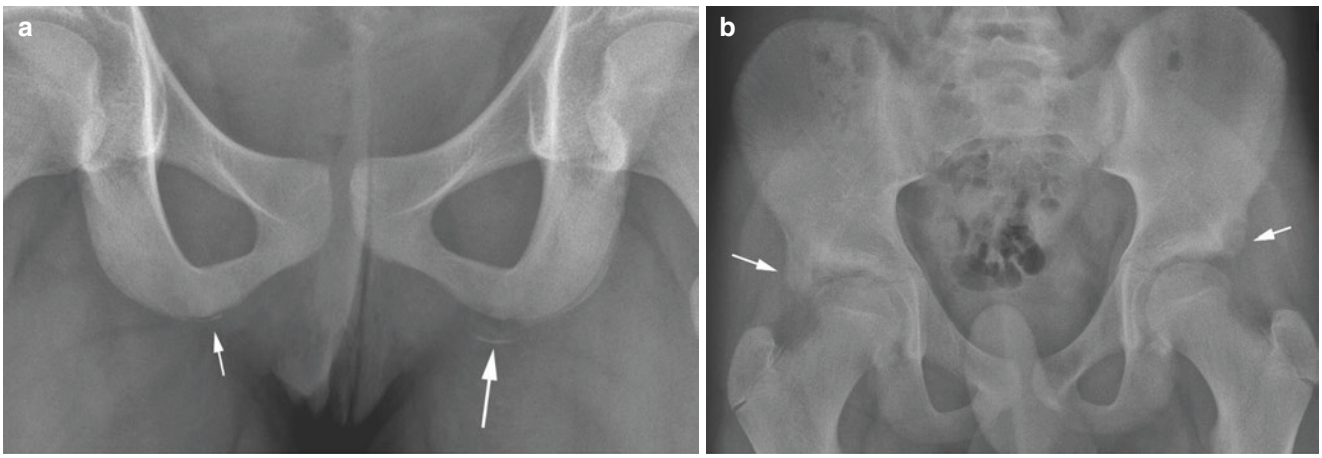


Fig. 11.4 Acute and chronic avulsion lesions; (**a**) 15-year female gymnast experienced a sudden pain during execution of a split. An avulsion of the apophysis of the ischial tuberosity is seen on the left (large arrow) compared to the normal apophysis (small arrow). (**b**) 14-year-old soccer player with chronic pain in both groins. The excessive bone formation at the anterior inferior iliac spine (arrows) are caused by chronic repetitive strain. The right side is more affected

Table 11.6 Overview of stress fractures of the pelvis in children and young adolescents under the age of 16 years

Author(s)	Anatomical location	Sex and age	Manner
Devas [30]	Ischiopubic junction	4 boys, 1 girl (5 to 8 years)	
Grier et al. [31]	Sacrum	Girl (14 years)	Runner, no history of trauma, insidious onset of pain
		Boy (9 years)	No history of trauma, gradual onset of pain
Lambert and Fligner [32]	Iliac crest	Boy (15 years)	Swinging a baseball bat
Rajah et al. [33]	Sacrum	Girl (11 years)	Aerobic exercise
Haasbeek and Green [34]	Sacral ala	2 female teenagers	Athletics
Martin et al. [35]	Sacrum	Girl (9 years)	
Lam and Moulton [36]	Sacrum	Boy (10 years)	School physical education
Pereira et al. [37]	Iliac crest	Girl (15 years) Girl (14 years)	Gymnastics class Dance class
Patterson et al. [38]	Sacrum	Adolescent (15 years)	No history of athletic participation or trauma
Kenawey et al. [39]	U-shaped sacral fracture with iliac crest apophyseal avulsion	Boy (8 years)	
Mortati et al. [40]	Iliac crest	Girl (11 years)	Running
Coursier et al. [41]	Sacral ala	Boy (11 years)	No report of trauma/intensive physical exercise
Coulier [42]		Boy (15 years)	Soccer player
Casabianca et al. [43]	Iliac crest	Boy (16 years)	Competitive sprinter
Portela and Santos [29]	Sacrum	Boy (10 years)	Soccer player

Stress fractures of a pelvic bone are only rarely described in children and young adolescents under the age of 16 years (see Table 11.6).

11.2.2.3 Fatigue Fractures in Weakened Bone: Insufficiency Fractures

An insufficiency fracture is a fracture that occurs when the strength of a bone is reduced to a level that stresses, that normally would not fracture a healthy bone, will break the weakened bone. Medical conditions that cause reduced bone strength usually are generalized throughout the skeleton, but may be more localized, e.g. due to demineralization of one limb, resulting from disuse [26].

Insufficiency fractures of the pelvis are usually found in elderly patients with, e.g. osteoporosis or osteomalacia. Fractures in elderly patients may occur as a result of everyday movements and routine activities as descending stairs or as a result of a more or less minor trauma like a fall from standing [44].

Insufficiency fractures, however, may also occur in paediatric patients [45, 46]. Insufficiency fractures in paediatric patients due to medical conditions, e.g. osteogenesis imperfecta, are extensively described in Chap. 15.

Maugars and Prost described the occurrence of pelvic fractures in young women with anorexia nervosa, due to bone loss, both of cortical and trabecular bone [46]. They

stated that the fractures are similar to those observed in postmenopausal osteoporosis, and the mechanism of bone loss in anorexia nervosa is similar to that in patients with postmenopausal osteoporosis, but may be favoured by other factors as alcohol intake or drug abuse [46]. According to them, fractures may occur in adolescents with long-standing anorexia nervosa, without weight loss. Some authors suggest a common mechanism relating the amenorrhoea observed in women training for high performance sports and that in anorexia nervosa.

Shelat and El-Khoury mentioned the higher incidence of sacral stress fractures in female athletes (especially in runners) and stated that these stress fractures are due to the combination of caloric imbalance, hormonal dysregulation, and impaired bone health [17]. Sacral stress fractures show characteristics of both fatigue (due to overuse) and insufficiency (due to bone loss).

11.3 Manner of Pelvic Fractures

Injuring and fracturing of the pelvis are always due to a trauma. Birth is not mentioned in the medical literature as a risk for the development of a fracture of the pelvis of the neonate.

11.3.1 Trauma After Birth: Accidental Circumstances

The majority of pelvic fractures in adults and children occur in accidental circumstances and are typically due to high-energy trauma, such as impact trauma or compression/crushing due to traffic accidents (e.g. pedestrian versus motor vehicle, motor vehicle versus motor vehicle, overrunning by motor vehicle) or falls from significant heights. Often serious associated injuries are found (Tables 11.2 and 11.3). Vertical shear fractures occur most commonly after a fall or jump from a height with impact onto the lower extremities [47].

According to Gänsslen et al. in children the organs in the pelvis are not well protected and often sustain injury in the absence of pelvic fractures [2]. In fact the pelvis in children is to a certain extent protected against fracturing by its relative malleability. This is particularly the case in extreme compressive forces, as can occur when a child is run over by a vehicle. Not only intrapelvic organs are not well protected, but also the genitals and anus may get injured:

- Boos et al. reported the occurrence of anogenital injuries in four children, who were run over by a slow-moving motor vehicle, in which the wheel of the vehicle passed longitudinally over the child's torso [48]. Two children had perianal lacerations and two had hymenal lacerations.
- Gabriel et al. reported the occurrence of isolated vaginal lacerations with genital bleeding in a 5-year-old girl after a vehicle rolled over her pelvis [49].

11.3.2 Trauma After Birth: Sporting Activities

11.3.2.1 Apophyseal Avulsion Fractures

Apophyseal avulsion injuries of the pelvis in paediatric and adolescent patients have been mainly described in adolescents, due to sporting activities, and may account for 10 to 24% of sporting injuries in children and adolescents (Fig. 11.4) [50]. The most common activities are soccer, running, and ballet dancing, although these injuries have also been described in football, baseball, lacrosse players, jumping, and track [17, 23, 50, 51]. Murray described six football players aged between 12 and 15 years of age with pelvic avulsion injuries (mean age 13.8 years): 5× avulsion injury of the anterior inferior iliac spine (AIIS), 1× avulsion injury of the ischial tuberosity (IT) [52]. In five patients the avulsion occurred while kicking and in one patient while sprinting.

11.3.2.2 Fatigue Fractures in Normal Bone: Stress Fractures

Stress fractures in a pelvic bone usually occur as a result of sporting activities with excessive weight bearing, that put repetitive stress on the pelvic bones, e.g. long-distance running, sprinting, jumping, or (ballet) dancing. It often affects people who quickly increase the duration and intensity of a physical activity without gradually building up endurance. It may also occur after a change in training conditions (such as surface, footwear or technique changes, etc.) [27, 28, 53]. Occasionally this type of fracture may occur due to repetitive kicking in sports such as football or soccer.

11.3.2.3 Trauma After Birth: Non-accidental Circumstances

Pelvic fractures are extremely rare in case of non-accidental circumstances [54]. Inflicted fractures have been reported in all parts of the pelvis [55].

It takes a great deal of force to cause a pelvic fracture, and for that reason non-accidental circumstances should be considered when the clinical history does not mention an accidental and severe high-energy trauma [56]. Moreover, if an inflicted pelvis fracture is found in a child, it is not rare to find other inflicted injuries, such as severe intracranial or visceral injuries or other fractures (Figs. 11.5a, b and 11.6). If a femur fracture is suspected to be inflicted, it is essential that the pelvis is meticulously examined for the presence of fractures on the side of the femur fracture [57, 58]. However, sometimes the only sign of inflicted injuries may be a periosteal reaction.

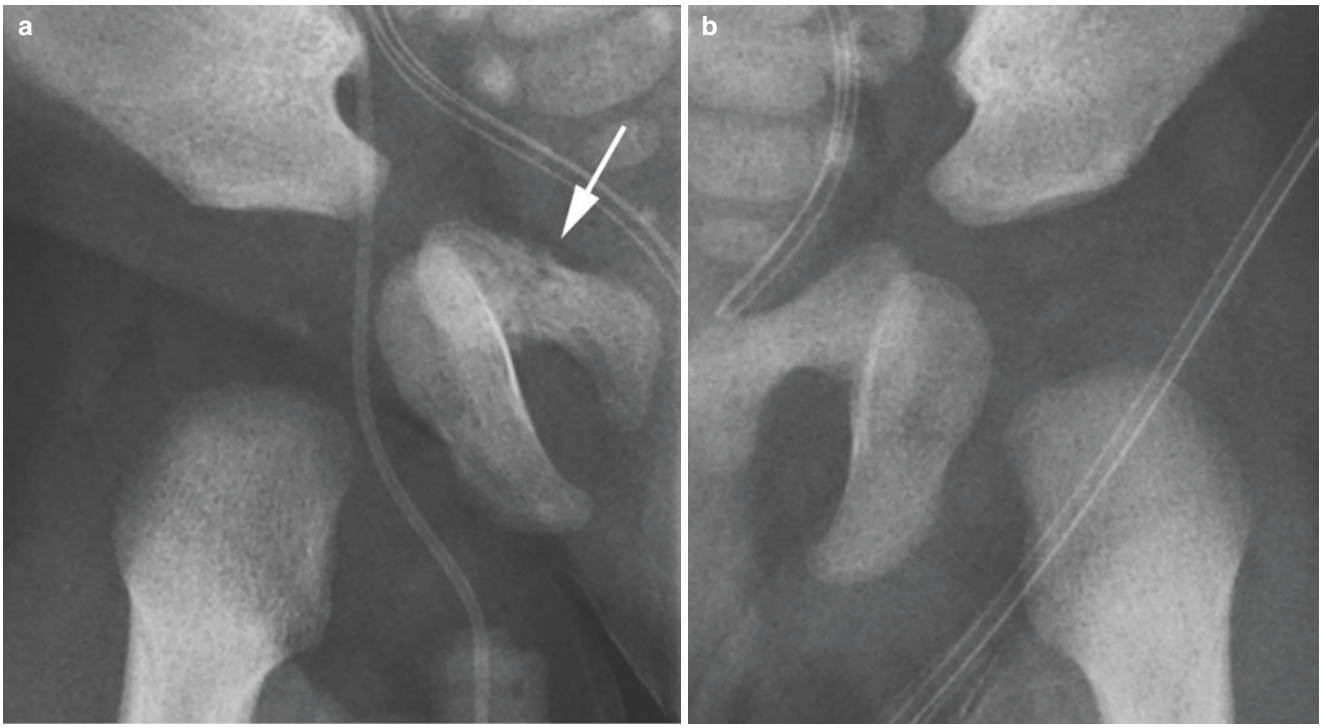


Fig. 11.5 Four-month old infant. Post-mortem images after fatal child abuse. The child also had multiple healed rib fractures, a healing ulna fracture, healing epiphysiolysis distal right femur, subdural haemorrhages, and severe hypoxic-ischemic brain injury. (**a**) A healing fracture is seen in the right pubic bone (arrow). (**b**) Normal left side for comparison

Fig. 11.6 Subtle avulsion fracture (bucket handle-like) of the ischial tuberosity in a 4-month-old infant (arrows). He also had rib fractures, skull fractures, and several CMLs of both legs

Perez-Rosello et al. described, based on the medical literature, two different patterns of age-specific pelvis injuries in children [59]:

- In infants, injuries of the pelvis tend to be subtle and typically located at the superior pubic ramus (Fig. 11.5).
- In toddlers and older children, injuries tend to be gross and varied in location. The injuries do result from massive blunt trauma, in extremely rare cases from sexual abuse or both (Fig. 11.7).

As stated before, inflicted pelvic injuries are rare and consequently rarely reported in the medical literature and if published only as case reports:

- Nazer et al. reported on a 15-month-old girl admitted with a history of convulsions and loss of consciousness [58]. The child had previous head and limb injuries resulting in bilateral subdural hematomas and fractures at different stages of healing of femur and pelvis.
- Ablin et al. reported on three children with inflicted pelvic injuries [60]. Two children had pelvic fractures. One child had a heterotopic ossification (i.e. extra-skeletal bone that is identical biologically and histologically to normal cortical and cancellous bone [61]) of the pelvis and thighs due to extensive bruising in the pubic, genital, buttock, and thigh areas, resulting from physical and sexual abuse.
- Tan and Gelfand reported on a 5-year-old boy multiple bruises with a lordotic posture and an abnormal gait, who

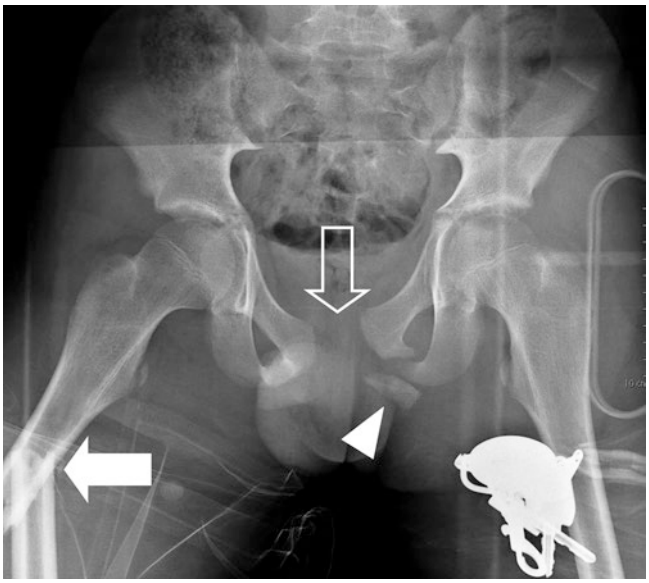


Fig. 11.7 Child after high-energy trauma. Diastasis of symphysis pubis (open arrow), fracture of left pubic bone and right femur

had inflicted rib and pelvic fractures [62]. These fractures were initially not seen on conventional radiology and CT-scans, but finally diagnosed on bone scintigraphy.

- Prendergast et al. reported on an almost 4-year-old girl who arrived in the hospital in cardiorespiratory arrest [63]. CPR was performed without success. A skeletal survey showed multiple bilateral humeral fractures in various stages of healing and a pelvic ring fracture (near the left superior pubic ramus and anterior part of the acetabulum). During autopsy numerous abrasions and bruises were noticed. The child also had scars on the head, trunk, and extremities. There were lacerations and haemorrhages of the external genitalia and vagina. There was haemorrhage in the retroperitoneal space, in the right atrial wall, and in the muscles of buttocks and extremities. The cause of death was determined to be a laceration of the ileum with associated peritonitis.
- Starling et al. reported on two male infants with initially unexplained pelvic fractures, which turned out to be inflicted [56].
- Johnson et al. reported on two children that sustained pelvic fractures and one child with a fracture of the femoral shaft related to sexual abuse [64]. A 3-year-old girl had suffered extensive injuries to the soft tissue of the arms, legs, and perineum. Moreover, she had fractures of both pubic arches and the sacral side of the right sacroiliac joint. A 5-year-old girl had presented with acute abdominal complaints and pneumoperitoneum due to a rectum rupture from sexual abuse; she also had an old healed fracture of the pubic arch with damage to the pubic symphysis. The last girl, 5 months old, had sustained a tear of the hymen and a fracture of the femoral shaft without dislocation.
- Sawyer et al. described heterotopic ossification of the hip after an inflicted injury in a 3-year-old child [65]. They concluded that non-accidental circumstances should be considered in the differential diagnosis in children with heterotopic ossification.
- Bixby et al. found an ischial apophyseal fracture in a previously healthy 4-month-old infant next to a comminuted left femur fracture and four vertebral compression deformities [54]. The fractures were determined to be inflicted.

11.4 Paediatric Versus Adult Pelvic Fractures

Paediatric pelvic fractures differ from pelvic fractures in adults, concerning aetiology, fracture type, and associated injuries, although the same types of trauma and circumstances have been described in paediatric and adolescent patients [2, 5, 9, 12, 66–68].

11.4.1 High-energy Trauma: Pelvis Ring Fractures

The pelvic ring in skeletally immature paediatric patients is more elastic and more resistant to fracturing than in skeletally mature paediatric and adult patients. Fractures in skeletally immature paediatric patients also tend to be more stable than in adults [2, 68].

The foregoing is due to the special anatomy of the immature paediatric pelvis [2, 5, 10]. The immature pelvis consists of more cartilage, which is able to absorb more energy than bone. The periosteum of the pelvis ring in the immature skeleton is thicker, which prevents to a certain extent bony displacement within the pelvis ring during stress. It also creates a periosteal ‘splint’ around dislocations (symphyseal, sacroiliac). The symphysis pubis and the sacroiliac joint are wider, thicker, and more elastic, which also leads to a higher capacity to absorb transferred energy.

According to Kruppa et al., skeletally mature children are more likely to sustain more complex injury patterns, to have a higher rate of associated injuries and to have higher injury severity scores than skeletally immature children [67]. They determined the skeleton of the pelvis to be mature when triradiate cartilage (the ‘Y’-shaped synchondrosis between the ilium, ischium, and pubis which forms the acetabulum) was closed.

The vast majority of paediatric and adult pelvic fractures are the result of high-energy trauma, in which the patient has been

struck by a car or was injured as a passenger in a motor vehicle [2]. Demetriades et al. found that adults were twice as likely as children to suffer pelvic fractures in traffic accidents (motor vehicle collisions or motor vehicle-pedestrian collision) [66]. Adults were also seven times more likely to suffer pelvic fractures as children in falls from heights above 4.5 m (15 ft.).

There seems to be a higher incidence of pelvis injuries due to lateral compression in paediatric patients than in adults, while in adults anterior-posterior compression injuries seem to be more common [5, 10]. According to several authors, the majority of accidental paediatric pelvic fractures are the result of pedestrians struck by motor vehicles, in which the pedestrian is more likely to be struck on the side of the body, causing a lateral compression injury, while most adults and adolescents with pelvis fractures, due to motor vehicle accidents, are drivers or front-seat passengers who are susceptible to anterior-posterior pelvis injuries. Paediatric passengers however are also more likely to sustain lateral compression injuries during motor vehicle accidents [4, 66].

Saglam et al. stated that, due to the greater elasticity, healing capacity, and re-modelling in paediatric patients, the prognosis of pelvic fractures is better in these patients than in adult patients [68].

11.4.2 High-energy Trauma: Acetabulum Fractures

As stated before, Kruppa et al. determined the skeleton of the pelvis to be mature when the triradiate cartilage was closed [67]. Closure of the triradiate cartilage occurs at an approximate bone age of 12 years in girls and 14 years in boys [69]. According to Swensen and Otsuka, this closure marks the moment at which the pelvic bones become stronger than the pelvic ligaments [10]. In the immature pelvis with a still open triradiate cartilage fractures most commonly occur in the pubic rami and iliac wings. Acetabular fractures, diastasis of the pubic symphysis, and separation of the sacroiliac joints are most commonly seen in skeletally mature patients [10, 70].

According to Holden et al., fractures of the triradiate cartilage may lead to growth disturbance of the acetabulum, resulting in acetabular dysplasia, hip subluxation, or hip joint incongruity [4]. Other complications include osteonecrosis of the femoral head in acetabular fractures associated with hip dislocation, myositis ossificans, and neurologic deficits secondary to sciatic, femoral, and/or lumbosacral plexus nerve injuries.

11.4.3 Lower Energy Trauma: Apophyseal Avulsion Fractures

Swensen and Otsuka state that the epiphyseal and apophyseal regions of the growing pelvis predispose skeletally immature children and adolescents to unique injuries and sequelae [10].

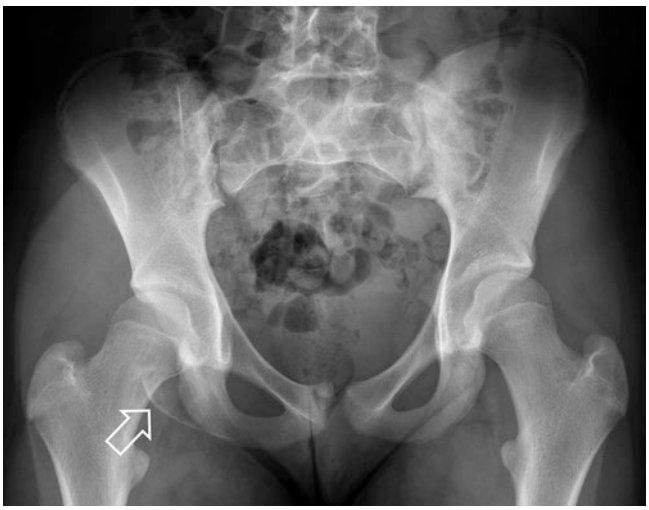


Fig. 11.8 Avulsion of the inferior ramus of right pubic bone (arrow), this is the insertion of the thigh adductors and gracilis muscle

Apophyseal avulsion fractures are more common in skeletally immature children and adolescents. The tendons in the skeletally immature patients are stronger than the cartilaginous growth centres in these patients and excessive traction on the muscle-tendon-bone unit (sudden, violent muscle contraction or excessive repetitive action) will result in apophyseal avulsion fractures (Fig. 11.8) [20, 23].

According to Swensen and Otsuka, fractures through the epiphyseal and apophyseal growth centres may result in growth arrest, leg length discrepancy, and deformity [10].

11.5 Differential Diagnosis of Pelvic Fractures

As stated before, pelvic fractures are extremely rare in children, especially in infants. Therefore normal variants, several medical conditions, e.g. Legg–Calvé–Perthes disease, slipped capital femoral epiphysis, soft-tissue injuries, tumours, infections, and inflammatory processes, and inflicted injuries should be part of the differential diagnosis:

- Rajah et al. pointed out that fatigue fractures of the sacrum may mimic primary bone tumours or infection [33].
- Perez-Rosello et al. analyzed the data of 14 infants (aged 1.5–7 months of age) with findings, suspected to be pelvis fractures [59]. In four infants the findings were classified as normal variants, in three children as fractures and in seven children the findings were described as indeterminate. In seven children it was concluded that, because of the presence of inflicted injuries (e.g. subdural haematoma, retinal haemorrhages, rib fractures, classic metaph-

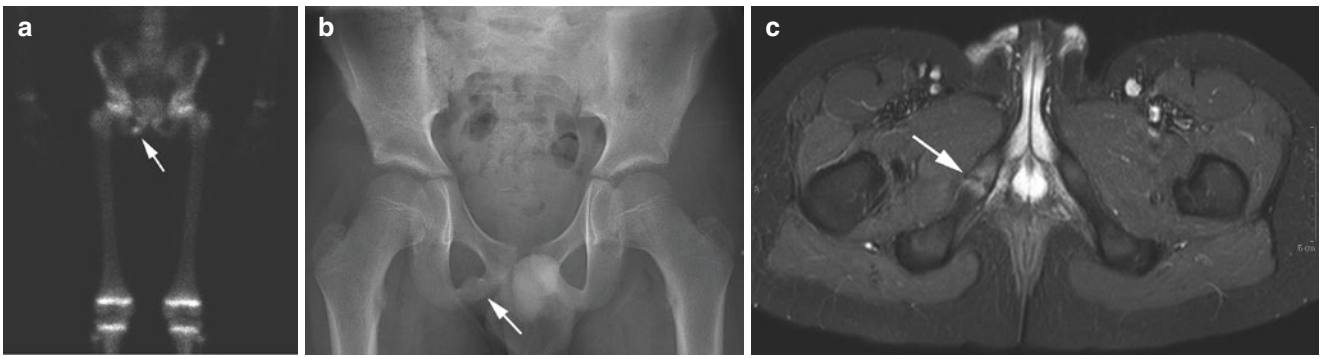


Fig. 11.9 Normal ischiopubic synchondrosis simulating pathology in a 9-year-old child during staging for Ewing sarcoma of a rib. There were no pelvic complaints. (**a**) Bone scan showed a hot spot (arrow) in the right pelvis. A pelvic radiograph (**b**) and T2-weighted MR image with fat saturation (**c**) demonstrate an ischiopubic synchondrosis on the right side (arrow) with oedema but without signs of fracture or metastasis. Asymmetric ischiopubic synchondroses are well-known normal variants and may show slight oedema and even enhancement after gadolinium

yseal lesions, long bone fractures), the child had been abused. In three of the children with inflicted injuries, the pelvic findings were determined to be normal variants, in three other children of this group as fractures, and in one child as indeterminate. They concluded that fractures of the superior pubic ramus and developmental variants can be difficult to differentiate radiographically.

- Ombregt stated that during the healing phase of an avulsion fracture the abundant reactive ossification in the soft tissues clinically and radiographically may be mistaken for a neoplasia [23].
- Portela and Santos stated that a stress fracture of the sacrum in a child should be differentiated of a wide range of pathologies like sacroiliitis, pars interarticularis fracture, osteoid osteoma, bone infection, or a soft-tissue malignancy [29].
- According to Kiel and Kaiser, stress fractures of the pelvis do not have specific clinical symptoms and for that reason can mimic other causes of groin and hip pain, for example adductor strain, osteitis pubis, or sacroiliitis [71].
- Herneth et al. reviewed pelvic MRIs of 28 children (4–16 years) who were scanned for reasons other than bone disorders in order to evaluate the MRI features of the ischiopubic synchondrosis [72]. They found signal alterations (hyperintense on T2-fatsat) in 89%, fusiform swelling in 68%, signal alteration of adjacent soft tissues in 57%, and gadolinium enhancement in 83%. They state that these findings are nonspecific and may be due to mechanical stress at this temporary joint but may resemble tumour, infection, or trauma (Fig. 11.9).

References

1. Roy B, Wallace A (2008) Pelvic fractures: synopsis of causation. https://www.gov.uk/government/publications/synopsis-of-causation-pelvic-fractures. Accessed 15 Aug 2021
2. Gänsslen A, Heidari N, Weinberg AM (2013) Fractures of the pelvis in children: a review of the literature. Eur J Orthop Surg Traumatol 23:847–861
3. Nodzo SR, Hohman DW, Galpin RD (2012) Bilateral acetabular fractures in an adolescent after low-energy trauma. Pediatr Emerg Care 28:568–569
4. Holden CP, Holman J, Herman MJ (2007) Pediatric pelvic fractures. J Am Acad Orthop Surg 15:172–177
5. Shaath K, Andras L (2019) Pelvis fractures – pediatric. https://www.orthobullets.com/pediatrics/3000/pelvis-fractures%2D%2Dpediatric. Accessed 10 Aug 2021
6. Torode I, Zieg D (1985) Pelvic fractures in children. J Pediatr Orthop 5:76–84
7. Müller E (1995) Comprehensive classification of pelvis and acetabulum fractures. Maurice E. Müller Foundation, Bern, Switzerland
8. Tile M (1996) Acute pelvic fractures: I. causation and classification. J Am Acad Orthop Surg 4:143–151
9. Leonard M, Ibrahim M, McKenna P, Boran S, McCormack D (2011) Paediatric pelvic ring fractures and associated injuries. Injury 42:1027–1030
10. Swensen SJ, Otsuka NY Pelvic fractures. https://posna.org/Physician-Education/Study-Guide/Pelvic-Fractures. Accessed 10 Aug 2021
11. Sink EL, Flynn JM (2014) Thoracolumbar spine and lower extremity fractures. In: Weinstein SL, Flynn JM (eds) Lovell and Winter's pediatric orthopaedics. Lippincott Williams & Wilkins, Philadelphia, pp 1776–1778
12. Hermans E, Cornelisse ST, Biert J, Tan E, Edwards MJR (2017) Paediatric pelvic fractures: how do they differ from adults? J Child Orthop 11:49–56
13. DeFrancesco CJ, Sankar WN (2017) Traumatic pelvic fractures in children and adolescents. Semin Pediatr Surg 26:27–35
14. Alton TB, Gee AO (2014) Classifications in brief: young and burgess classification of pelvic ring injuries. Clin Orthop Relat Res 472:2338–2342
15. Burgess AR, Eastridge BJ, Young JW, Ellison TS, Ellison PS Jr, Poka A, Bathon GH, Brumback RJ (1990) Pelvic ring disruptions: effective classification system and treatment protocols. J Trauma 30:848–856
16. Young JW, Burgess AR, Brumback RJ, Poka A (1986) Pelvic fractures: value of plain radiography in early assessment and management. Radiology 160:445–451
17. Shelat NH, El-Khoury GY (2016) Pediatric stress fractures: a pictorial essay. Iowa Orthop J 36:138–146
18. Jaimes C, Jimenez M, Shabshin N, Laor T, Jaramillo D (2012) Taking the stress out of evaluating stress injuries in children. Radiographics 32:537–555

19. Sanderlin BW, Raspa RF (2003) Common stress fractures. Am Fam Physician 68:1527–1532
20. Kjellin I (2008) Apophyseal avulsion injury of the pelvis. http://radsource.us/apophyseal-avulsion-injury-of-the-pelvis/. Accessed 10 Aug 2021
21. McKinney BI, Nelson C, Carrion W (2009) Apophyseal avulsion fractures of the hip and pelvis. Orthopedics 32:42
22. Calderazzi F, Nosenzo A, Galavotti C, Menozzi M, Pogliacomi F, Ceccarelli F (2018) Apophyseal avulsion fractures of the pelvis. A review. Acta Biomed 89:470–476
23. Ombregt L (2013) Hip disorders in children: avulsion fractures about the hip. In: Ombregt L (ed) A system of orthopaedic medicine. Churchill Livingstone, pp e256–e261
24. Shah R, Shelat N, El-Khoury GY, Bennett DL (2016) Avulsion injuries of the pelvis. J Am Osteopath Coll Radiol 5:5–11
25. Bedoya MA, Jaramillo D, Chauvin NA (2015) Overuse injuries in children. Top Magn Reson Imaging 24:67–81
26. DeWeber K (2017) Overview of stress fractures. https://www.uptodate.com/contents/overview-of-stress-fractures. Accessed 10 Aug 2021
27. NYU Langone health types of hip & pelvic fractures. https://nyulangone.org/conditions/hip-pelvic-fractures-in-adults/types. Accessed 10 Aug 2021
28. PhysioAdvisor.com Pelvic stress fracture. https://www.physioadvisor.com.au/injuries/hip-groin/pelvic-stress-fracture/. Accessed 10 Aug 2021
29. Portela R, Santos M (2017) Fatigue fractures of the sacrum on children: case report. J Orthopedics Rheumatol 4:3
30. Devas MB (1963) Stress fractures in children. J Bone Joint Surg Br 45:528–541
31. Grier D, Wardell S, Sarwark J, Poznanski AK (1993) Fatigue fractures of the sacrum in children: two case reports and a review of the literature. Skelet Radiol 22:515–518
32. Lambert MJ, Fligner DJ (1993) Avulsion of the iliac crest apophysis: a rare fracture in adolescent athletes. Ann Emerg Med 22:1218–1220
33. Rajah R, Davies AM, Carter SR (1993) Fatigue fracture of the sacrum in a child. Pediatr Radiol 23:145–146
34. Haasbeek JF, Green NE (1994) Adolescent stress fractures of the sacrum: two case reports. J Pediatr Orthop 14:336–338
35. Martin J, Brandser EA, Shin MJ, Buckwalter JA (1995) Fatigue fracture of the sacrum in a child. Can Assoc Radiol J 46:468–470
36. Lam KS, Moulton A (2001) Stress fracture of the sacrum in a child. Ann Rheum Dis 60:87–88
37. Pereira GJC, da Rosa PH, Cruz M (2002) Indirect avulsion of the iliac crest epiphysis. A rare lesion. Acta Ortop Bras 2:58–61
38. Patterson SP, Daffner RH, Sciulli RL, Schneck-Jacob SL (2004) Fatigue fracture of the sacrum in an adolescent. Pediatr Radiol 34:633–635
39. Kenawey M, Addosooki A (2014) U-shaped sacral fracture with iliac crest apophyseal avulsion in a young child. J Pediatr Orthop 34:e6–e11
40. Mortati RB, Borghi Mortati L, Silva Teixeira M, Itiro Takano M, Armelin Borger R (2014) Avulsion fracture of the iliac crest in a child. Rev Bras Ortop 49:309–312
41. Coursier R, Degisors S, Lespessailles E, Toumi H (2015) Sacral ala stress fracture in a child. J Clin Case Rep 5:1–2
42. Coulier B (2015) Acute avulsion of the iliac crest apophysis in an adolescent indoor soccer. J Belg Soc Radiol 99:20–24
43. Casabianca L, Rousseau R, Loriaut P, Massein A, Mirouse G, Gerometta A, Khiami F (2015) Iliac crest avulsion fracture in a young sprinter. Case Rep Orthop 2015:302503
44. Dunbar RP, Lowe JA (2016) "Pelvic fractures." OrthoInfo. Retrieved 22-06-2020, from https://orthoinfo.aaos.org/en/diseases--conditions/pelvic-fractures/
45. Hacking C, Harvey H (2019) Pelvic fractures. https://radiopaedia.org/articles/pelvic-fractures. Accessed 10 Aug 2021
46. Maugars Y, Prost A (1994) Ostéoporose de l'anorexie mentale [Osteoporosis in anorexia nervosa]. Presse Med 23:156–158
47. Storch B (2017) Pelvic vertical shear fractures. https://coreem.net/core/pelvic-vertical-shear-fractures/. Accessed 10 Aug 2021
48. Boos SC, Rosas AJ, Boyle C, McCann J (2003) Anogenital injuries in child pedestrians run over by low-speed motor vehicles: four cases with findings that mimic child sexual abuse. Pediatrics 112:e77–e84
49. Gabriel NM, Clayton M, Starling SP (2009) Vaginal laceration as a result of blunt vehicular trauma. J Pediatr Adolesc Gynecol 22:e166–e168
50. Boyd KT, Peirce NS, Batt ME (1997) Common hip injuries in sport. Sports Med 24:273–288
51. Stevens MA, El-Khoury GY, Kathol MH, Brandser EA, Chow S (1999) Imaging features of avulsion injuries. Radiographics 19:655–672
52. Murray E (2019) An interesting case series describing a spate of pelvic avulsion injury in a cohort of elite adolescent footballers. Phys Ther Sport 35:7–11
53. Lowth M, Cox J (2017) Pelvic fractures. https://patient.info/bones-joints-muscles/pelvic-fractures-leaflet. Accessed 10 Aug 2021
54. Bixby SD, Wilson CR, Barber I, Kleinman PK (2014) Ischial apophyseal fracture in an abused infant. Pediatr Radiol 44:1175–1178
55. Merten DF, Cooperman DR, Thompson GH (1992) Skeletal manifestations of child abuse. In: Reece RM (ed) Child abuse – medical diagnosis and management. Lea & Febiger, pp 23–53
56. Starling SP, Heller RM, Jenny C (2002) Pelvic fractures in infants as a sign of physical abuse. Child Abuse Negl 26:475–480
57. Brodeur AE, Monteleone JA (1994) Child maltreatment. A clinical guide and reference. GW Medial Publishing, p 32
58. Nazer H, Daradkeh T, Mohamed S, Shamayleh AQ, Marei O (1988) A diagnostic dilemma in Jordan: two child abuse case studies. Child Abuse Negl 12:593–599
59. Perez-Rossello JM, Connolly SA, Newton AW, Thomason M, Jenny C, Sugar NF, Kleinman PK (2008) Pubic ramus radiolucencies in infants: the good, the bad, and the indeterminate. AJR Am J Roentgenol 190:1481–1486
60. Ablin DS, Greenspan A, Reinhart MA (1992) Pelvic injuries in child abuse. Pediatr Radiol 22:454–457
61. Palmer W, Bancroft L, Bonar F, Choi JA, Cotten A, Griffith JF, Robinson P, Pfirrmann CWA (2020) Glossary of terms for musculoskeletal radiology. Skelet Radiol 49(Suppl 1):1–33
62. Tan TX, Gelfand MJ (1997) Battered child syndrome. Uncommon pelvic fractures detected by bone scintigraphy. Clin Nucl Med 22:321–322
63. Prendergast NC, deRoux SJ, Adsay NV (1998) Non-accidental pediatric pelvic fracture: a case report. Pediatr Radiol 28:344–346
64. Johnson K, Chapman S, Hall CM (2004) Skeletal injuries associated with sexual abuse. Pediatr Radiol 34:620–623
65. Sawyer JR, Kapoor M, Gonzales MH, Warner WC Jr, Canale ST, Beaty JH (2009) Heterotopic ossification of the hip after non-accidental injury in a child: case report. J Pediatr Orthop 29:865–867
66. Demetriades D, Karaiskakis M, Velmahos GC, Alo K, Murray J, Chan L (2003) Pelvic fractures in pediatric and adult trauma

patients: are they different injuries? J Trauma 54:1146–1151. discussion 1151
67. Kruppa CG, Khoriaty JD, Sietsema DL, Dudda M, Schildhauer TA, Jones CB (2018) Does skeletal maturity affect pediatric pelvic injury patterns, associated injuries and treatment intervention? Injury 49:1562–1567
68. Saglam Y, Dikmen G, Bademler S, Aksoy M, Dikici F (2015) Analysis of the cause, classification, treatment, outcome and associated injuries of pediatric pelvic ring fractures. Ulus Travma Acil Cerrahi Derg 21:392–396
69. Dimeglio A (2001) Growth in pediatric orthopaedics. J Pediatr Orthop 21:549–555
70. Amorosa LF, Kloen P, Helfet DL (2014) High-energy pediatric pelvic and acetabular fractures. Orthop Clin North Am 45:483–500
71. Kiel J, Kaiser K (2019) Stress reaction and fractures. https://www.ncbi.nlm.nih.gov/books/NBK507835/. Accessed 10 Aug 2021
72. Herneth AM, Trattnig S, Bader TR, Ba-Ssalamah A, Ponhold W, Wandl-Vergesslich K, Steinbach LS (2000) MR imaging of the ischiopubic synchondrosis. Magn Reson Imaging 18:519–524

Extremities

12

Rob A. C. Bilo, Selena de Vries, Michelle Nagtegaal, Simon G. F. Robben, and Rick R. van Rijn

Contents

R. A. C. Bilo
Veilig Thuis Rotterdam Rijnmond (Center for the Reporting of Child Abuse, Domestic Violence and Elder Abuse), Rotterdam, The Netherlands

S. de Vries · M. Nagtegaal
Department of Forensic Medicine, Netherlands Forensic Institute, The Hague, The Netherlands

S. G. F. Robben
Department of Radiology, Maastricht University Medical Center, Maastricht, The Netherlands

R. R. van Rijn (✉)
Department of Forensic Medicine, Netherlands Forensic Institute, The Hague, The Netherlands

Department of Radiology and Nuclear Medicine, Amsterdam UMC, University of Amsterdam, Amsterdam, The Netherlands
e-mail: r.r.vanrijn@amsterdamumc.nl

R. A. C. Bilo et al. (eds.), *Forensic Aspects of Paediatric Fractures*, https://doi.org/10.1007/978-3-031-12041-1_12

12.1 General Aspects of Fractures of the Extremities

The bones of the extremities can be categorized according to their shape in long and short bones. Long bones are found in the arms (humerus, radius, ulna), fingers (metacarpals, phalanges), legs (femur, tibia, fibula), and toes (metatarsals, phalanges). The carpals of the wrists and the tarsals of the ankles are short bones (see Sect. 2.4.1.2). Irrespective of their anatomical location, long bones always consist of (Figs. 12.1 and 12.2a–c):

- Physis: Growth plate.
- Diaphysis: The shaft; the medulla-containing tubular middle part of a long bone.
- Epiphysis: The wider parts at both ends of a long bone.
- Metaphysis: A narrow area between the diaphysis and the epiphysis.

From an anatomical, physiological, and biomechanical perspective, the skeleton, especially of the extremities, of young children differs from the adult skeleton. Depending on

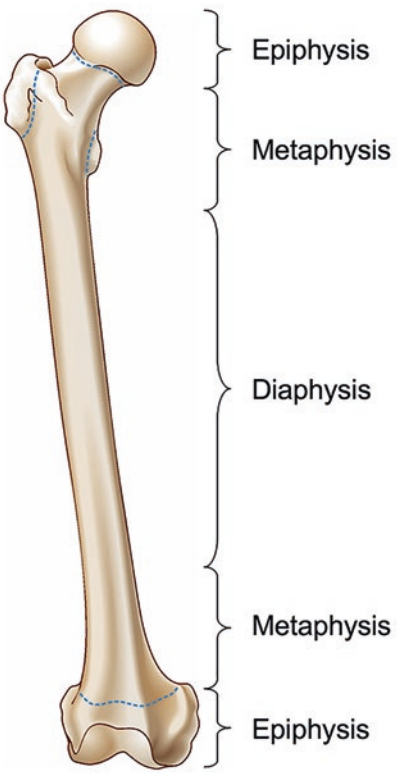


Fig. 12.1 Schematic representation of the anatomy of the long bones

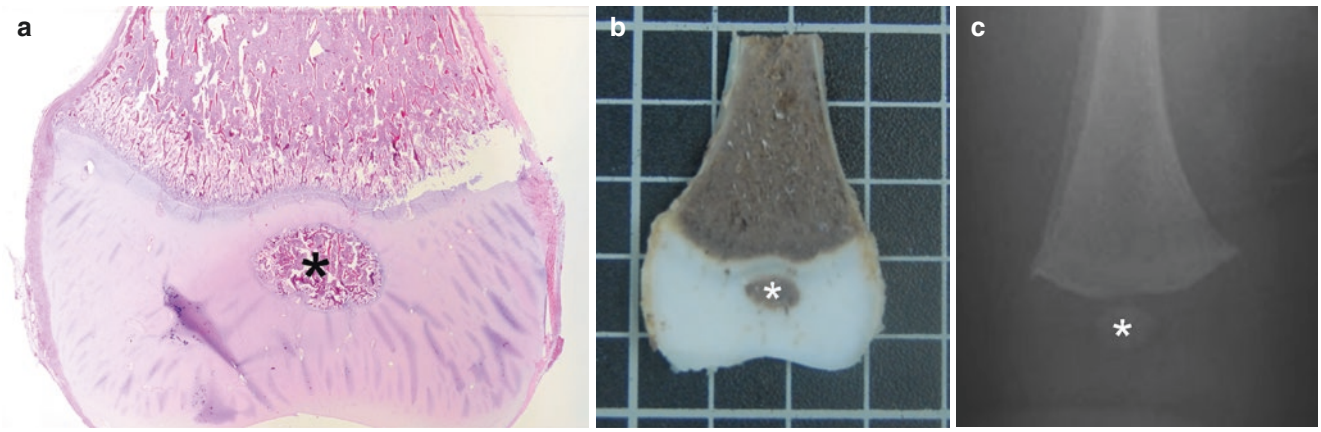


Fig. 12.2 (**a**) Histological section of the distal femur of a 3-month-old neonate, which shows ossification of the distal epiphysis of the femur (asterisk). (**b**) Corresponding specimen photo of the distal femur, showing ossification of the epiphysis of the distal femur. (**c**) Corresponding radiograph of the distal femur, showing ossification of the epiphysis of the distal femur

the characteristics of the force of impact, specific fractures will occur in children in specific parts of the long and short bones of the extremities.

12.2 Fractures of the Diaphysis

12.2.1 Cause of Fractures of the Diaphysis

A number of aspects should be considered in the analysis of what long bones are exposed to in either daily life or under the impact of force. These concern:

- The force or combination of forces exerted on the bone in day-to-day use and when under the impact of force: the load bearing of the bone ('load').
- The force of the bone to resist this load ('stress').
- The changes in shape or size of tissue in reaction to this stress ('stretch/strain').

When a fracture is sustained, the three pure forms (load, stress, strain) seldom occur just by themselves, but nearly always a combination of the three is seen (Table 12.1). Three pure forms of strain can be distinguished: compression, tension, and shearing:

- Compression: Compression is defined as a perpendicular force that affects a surface in such a manner that it compresses the object. Bone has great resistance to this kind of force. When a fracture is caused by compression, it is usually because the compression is not quite along the central axis of the bone [1]. In such cases, compression will cause the bone to bow, which results in tension on one side, which ultimately determines the nature of the fracture.
- Tension: Tension is defined as a perpendicular force that affects a surface in such a manner that it pulls an object apart. Bone is less resistant to tension than to compression. In tension the bone is stretched out like a spring: it becomes longer and thinner. Tension exerted on a bone for a limited period of time does not necessarily lead to a fracture. In normal cases it will fully recover; however, as soon as the limit of the elasticity of the bone is exceeded, damage is inflicted. This damage is not necessarily visible on radiographs. Only in cases with prolonged or stronger tension, a fracture will become visible. The fracture line will follow the contours of the weakest areas of the bone, which sometimes causes the fracture to have a zigzag line.
- Shearing: Shearing is physically equal to compression and tension, but the force is exerted in such a manner that the tissue is distorted and deformed. Bone is not very resistant to shearing.

Furthermore, various combinations may be seen, such as bowing and torque:

- Bowing: Bowing is caused by a force that causes tension on one side (the convex side) and compression on the opposite side (the concave side). In bowing, the cortex on the tension side will usually be damaged first. When this happens, and the loading stops, it will result in a so-called 'greenstick fracture' (Fig. 12.3). When the loading does not stop, the fracture will spread. The most classical expression of this type of loading is the transverse fracture. Depending on the type of bone and the additional forces exerted, other types of fractures may occur. In immature bone, the bone may also yield on the compression side first, which may lead to a buckle fracture (torus fracture) of the compression side (Fig. 12.4).

Table 12.1 Biomechanical aspects of shaft fractures

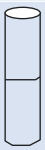
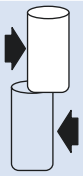
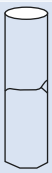
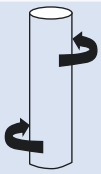
Force/ combination of forces	Fracture type	
 Compression	 Compression	• Oblique fracture
	 Compression and bowing	• Transverse fracture, possibly with lose fragments on the compression side • Greenstick fracture • Torus fracture • Bowing fracture
 Tension	 Tension/shearing	• Transverse fracture, possibly with a zig-zag pattern
 Shearing		• Metaphyseal corner fracture
 Bowing	 Bowing	• Transverse fracture, possibly with lose fragments on the compression side • Greenstick fracture • Torus fracture • Bowing fracture
	 Bowing and compression	
 Torsion	 Torsion	• Spiral fractures

Fig. 12.3 Sixteen-year-old boy who had a painful wrist after romping around with his brother. The lateral side of the distal ulna shows a greenstick fracture (open arrow)

- Torque: Torque is the result of forces rotating an object along the longitudinal axis, when the other side is stationary or turned in the opposite direction. When the torque forces are directed to the left, it will cause a spiral fracture that turns to the right, and the other way around (Fig. 12.5).

The growing bone in children reacts differently to subjected forces than the fully developed bone in adults. The presence of larger and more extensive Haversian canals together with increased elasticity make the child's bone more malleable than adult bone. Consequently, immature bone (in particular the diaphysis of the long bones) can deform more during bending than adult bone without breaking. Finally, the periosteum in children is thicker, stronger, and less firmly attached to the (diaphyseal) bone. It is less frequently torn after trauma and, likewise, can act as a stabilizing factor in case of a fracture. This means that in children specific types of fracture of the shaft are found that are typical for growing bone. This concerns in particular the so-called incomplete fractures:

Fig. 12.4 Three-year-old boy with a torus fracture of metatarsal I of the right foot after taking a jump (open arrow)

Fig. 12.5 Spiral fracture of the tibia in an infant as a result of a spoke wheel injury

- 'Buckle' fracture or torus fracture (damage to the cortex at the compression side of the bend): In axial compression of a bone that has very limited ability to bow, a child can sustain a torus fracture at the shaft-metaphyseal transition (Fig. 12.6a, b). These fractures are stable by nature and when immobilized will heal within 2–3 weeks.
- 'Greenstick' fracture (damage to the cortex at the tension side of the bend): This type of fracture can occur when the bone is bowed past the fracture limit at the tension site. It concerns an incomplete fracture on the tension side of the bone and plastic deformation with an intact cortex and intact periosteum at the compression side. In these cases, the force that caused the damage to the cortex on the tension side is insufficient to cause a complete fracture (Fig. 12.7).
- 'Bowing' fractures: In very young children there can be such deformation of the bone that it bows beyond its yield point, the point beyond which deformation becomes permanent (plastic) and no longer spontaneously recoverable (elastic). In these cases, there is no radiologically visible damage in the cortex, neither to the tension nor to the compression side. The fracture will only be visible by the bowing of the diaphyseal segment (Fig. 12.8a, b). These fractures can be very subtle and sometimes comparison with the contralateral bone is helpful. Bowing fractures are common in the forearm.

12.2.2 Manner of Fractures of the Diaphysis

In their original publication from 1962 on 'The battered child syndrome', Kempe et al. stated that the child's extremities are '*the handles for rough handling*' [2]. This may lead to fractures, in particular of the long bones of the extremities. However, in mobile children fractures of arms and legs are also frequently sustained in accidental circumstances. Sometimes their location is an indicator of non-accidental trauma. In other cases, the clinical history and the level of

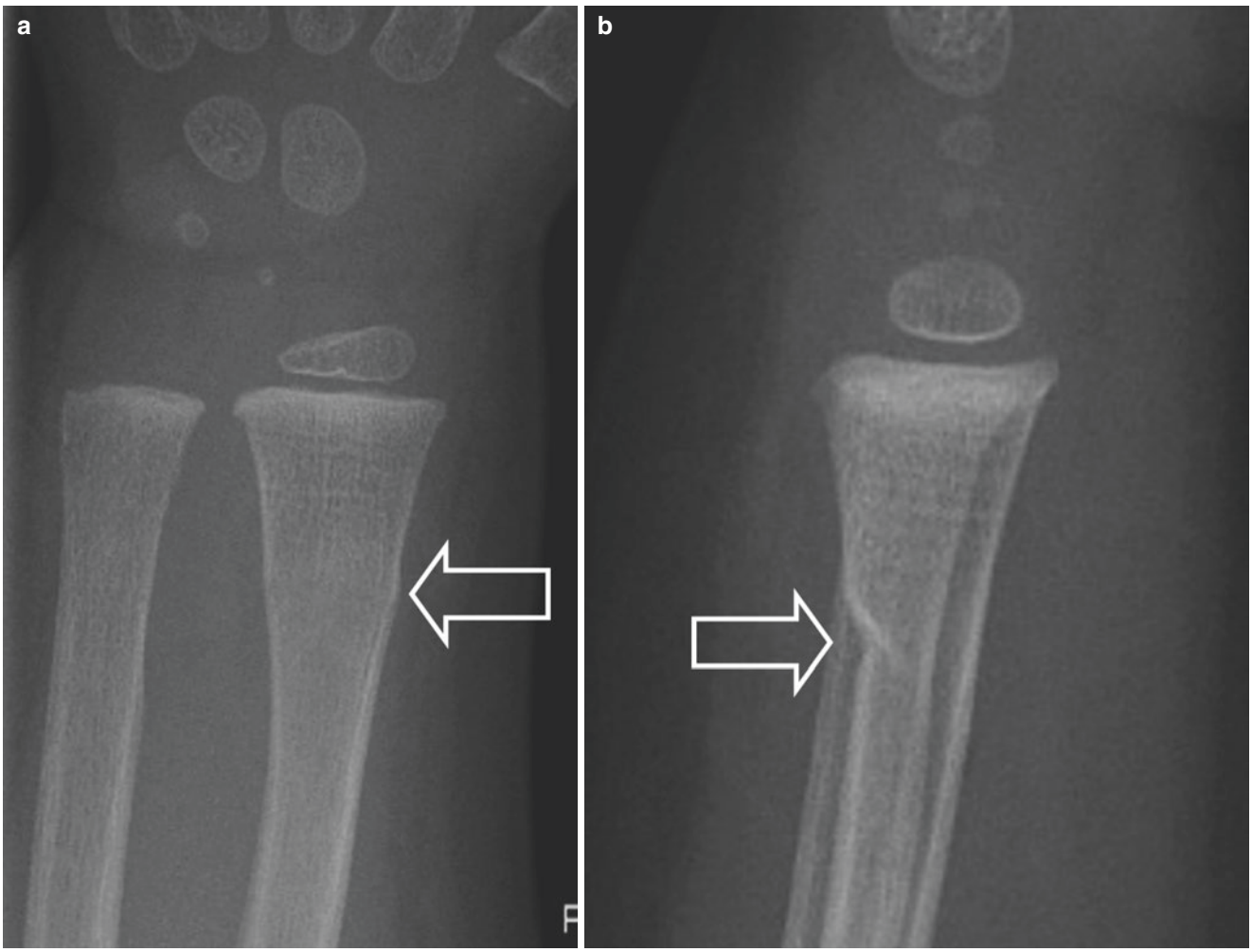


Fig. 12.6 Two-year-old infant who presented with a painful arm after a fall from a chair. (**a**) AP radiograph shows an irregularity of the cortex of the distal radius (arrow). (**b**) on the lateral radiograph shows a clear torus fracture of the distal radius (arrow)

Fig. 12.7 Six-year-old child who sustained a fall onto an outstretched hand (FOOSH)

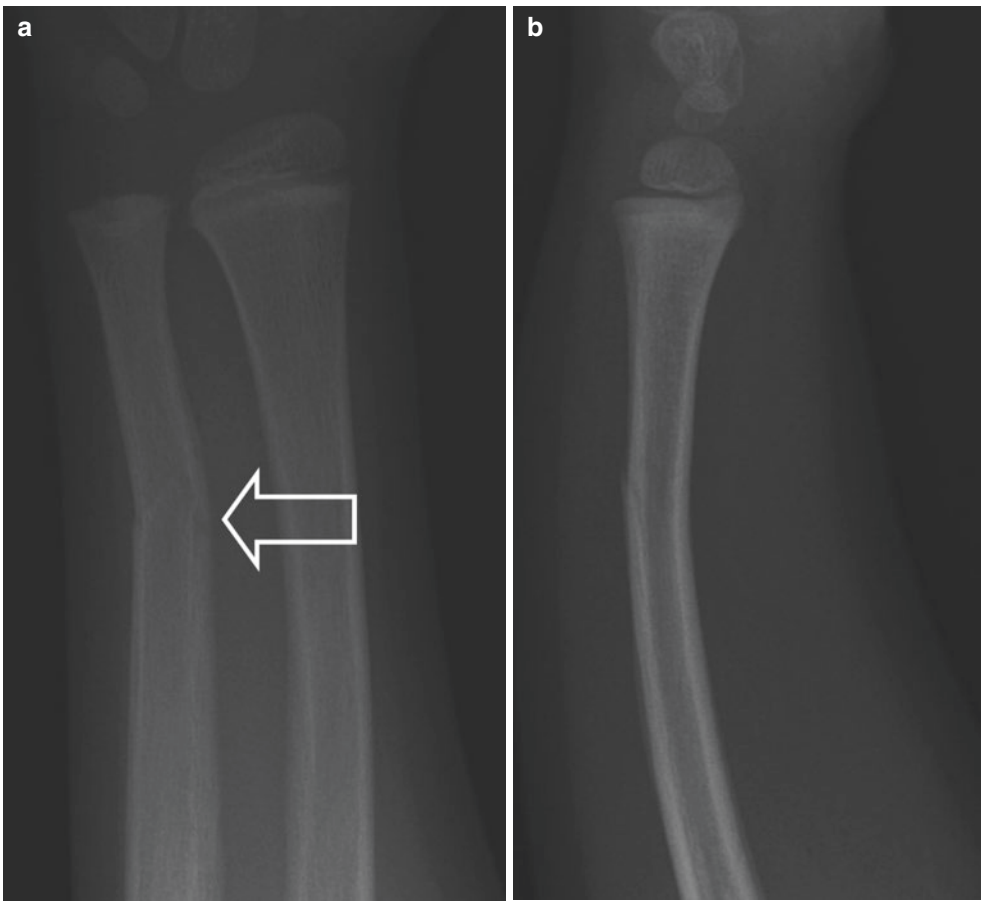


Fig. 12.8 Five-year-old child who sustained a fall onto an outstretched hand (FOOSH). (**a**) There is a transverse fracture of the ulna (arrow). (**b**) The lateral radiograph shows a bowing fracture of the radius

development of the child could help differentiate between accidental and inflicted extremity fractures. The reader is referred to Sects. 12.4–12.9, concerning the more specific aspects of fractures of certain long bones.

12.3 Fractures of the Metaphysis and Epiphysis

12.3.1 Introduction

The most important difference between the still developing skeleton of a child and the fully grown adult skeleton is the presence of growth plates (physeal plates) in the long bones. These growth plates are responsible for the longitudinal growth of a bone in the skeleton of young children by enchondral bone formation, whereas growth in width originates in the periosteum by membranous bone formation.

The epiphyses determine the size and form of the joint ends. Typical long bones, like the humerus, radius, ulna, femur, tibia, and fibula, have two epiphyses (one at both ends) whereas some of the smaller long bones only have one epiphysis.

Growth plates consist of cartilage. This cartilage is among the weakest parts of the child's skeleton, especially of the long bones. Due to this weakness growth plates are less resistant to forces exerted to the extremities, compared to the joint capsules, tendons, and ligaments [3]. The growth plates also are the most vulnerable places in the growing skeleton when the joint is subjected to force. This vulnerability will remain as long as ligaments and tendons are more resistant to forces than bone. The damage may consist of a fully or partially torn-off metaphysis (resulting in a metaphyseal corner fracture). When the fully grown skeleton is subjected to the same forces, it more likely results in damage to the ligaments around the joint.

Because growth plates are unique for children, all fractures that have some relation to the growth plate are also unique for children. Amongst these fractures are Salter-Harris fractures and the epiphyseal transitional fractures (triplane fractures and Tillaux fractures). All growth plate-related fractures are at risk for premature focal closure of the growth plate.

12.3.2 Metaphyseal Corner Fracture

12.3.2.1 Introduction

The Metaphyseal Corner Fracture (MCF, a.k.a. Classic Metaphyseal Lesion) is a planar fracture through the most immature portion of the metaphysis in the region of the trabecular transition zone (from primary to secondary spongiosa), disrupting the delicate trabeculae composed of central calcified cartilage cores covered by thin layers of newly formed bone [4]. The fracture line passes peripherally to undercut the subperiosteal bone collar [5, 6]. The resultant fracture fragment is made up of subperiosteal bone collar, physis, and a layer of the primary spongiosa of the metaphysis [4, 5]. MCFs can be extensive diffuse (micro) fractures that extend over the entire metaphysis separating a complete rim-like fragment, or localized (incomplete) injuries separating only a portion of this disk [7, 8].

The term classic metaphyseal lesion in fact is a misnomer, because it is a genuine fracture and not only an unspecified lesion. Thompson et al. stated that 'A classic metaphyseal lesion is a unique type of fracture with specific morphologic characteristics. Therefore, we suggest using the term "classic metaphyseal fracture" in lieu of classic metaphyseal lesion to improve precision of terminology' [9].

When these fractures are present over the full circumference of the bone, the radiographs will show a detached metaphyseal rim that is smaller in the centre and wider at the edges (a so-called 'bucket-handle fracture, Fig. 12.9a). Sometimes the radiographs only show the wider edge (the so-called 'corner fracture'; Fig. 12.9b). The 'corner fracture' and 'bucket-handle fracture' are simply different radiographic projections of the same fracture. For consistency we will speak of Metaphyseal Corner Fracture throughout this chapter.

Metaphyseal corner fractures are almost exclusively seen in children less than 2 years of age. The fracture may be seen in just one bone or around one joint. Hereby should be mentioned that in a MCF of the proximal tibial metaphysis there is often an associated avulsion fracture of the femur (distal metaphysis). Metaphyseal corner fractures are found most frequently in the distal femur and the proximal and distal tibia (Figs. 12.10a and 12.11a, b), making the tibial metaphysis the most prevalent location for MCFs in infants (Fig. 12.12) [5, 10, 11]. There seems to be a tendency for MCFs in the distal tibia to favour the medial

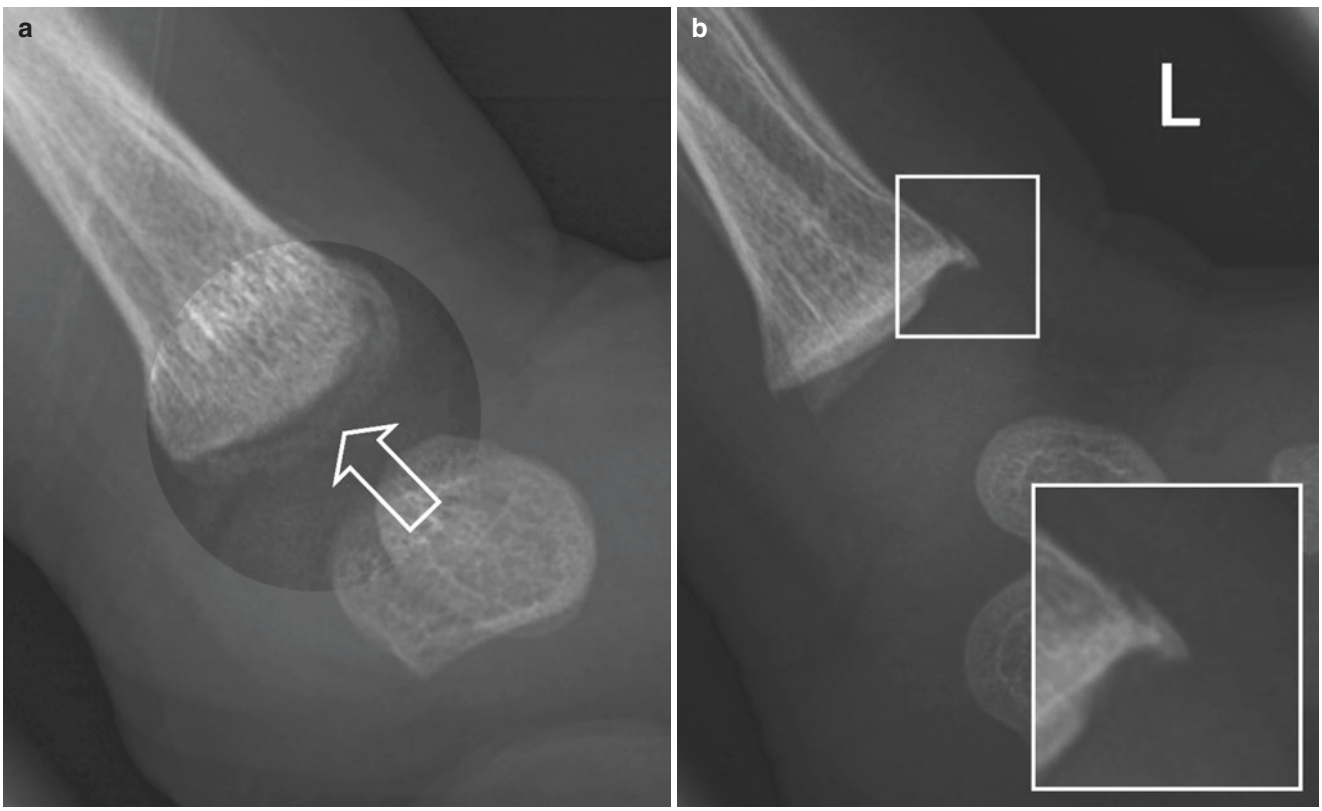


Fig. 12.9 (**a**) Two-month-old girl who died when 'co-sleeping'. Radiological examination within the scope of the Dutch cot-death protocol shows a bucket-handle fracture of the distal left tibia (open arrow). (**b**) Radiograph of the same tibia from a different angle shows a corner fracture (see inset)

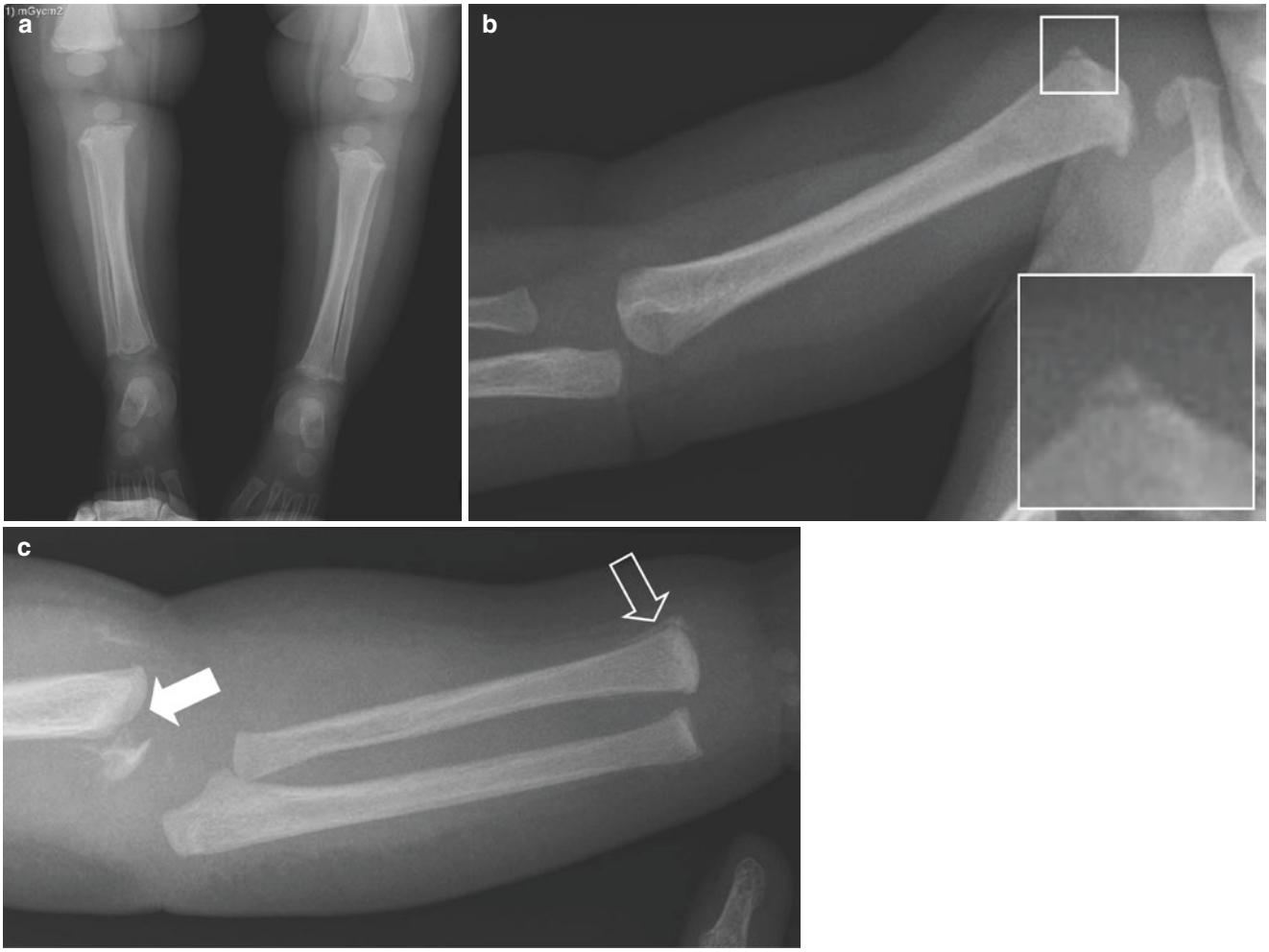


Fig. 12.10 (**a**) Severely abused 4-month-old girl. The skeletal survey shows healing metaphyseal corner fractures of the distal femurs and the proximal and distal tibias. Reactive sub-periosteal new-bone formation is visible along the greater part of the right tibia shaft. (**b**) Metaphyseal corner fracture of the right proximal humerus (see inset). (**c**) Metaphyseal corner fracture of the left distal radius (open arrow) and a distal metaphyseal humerus fracture (arrow)

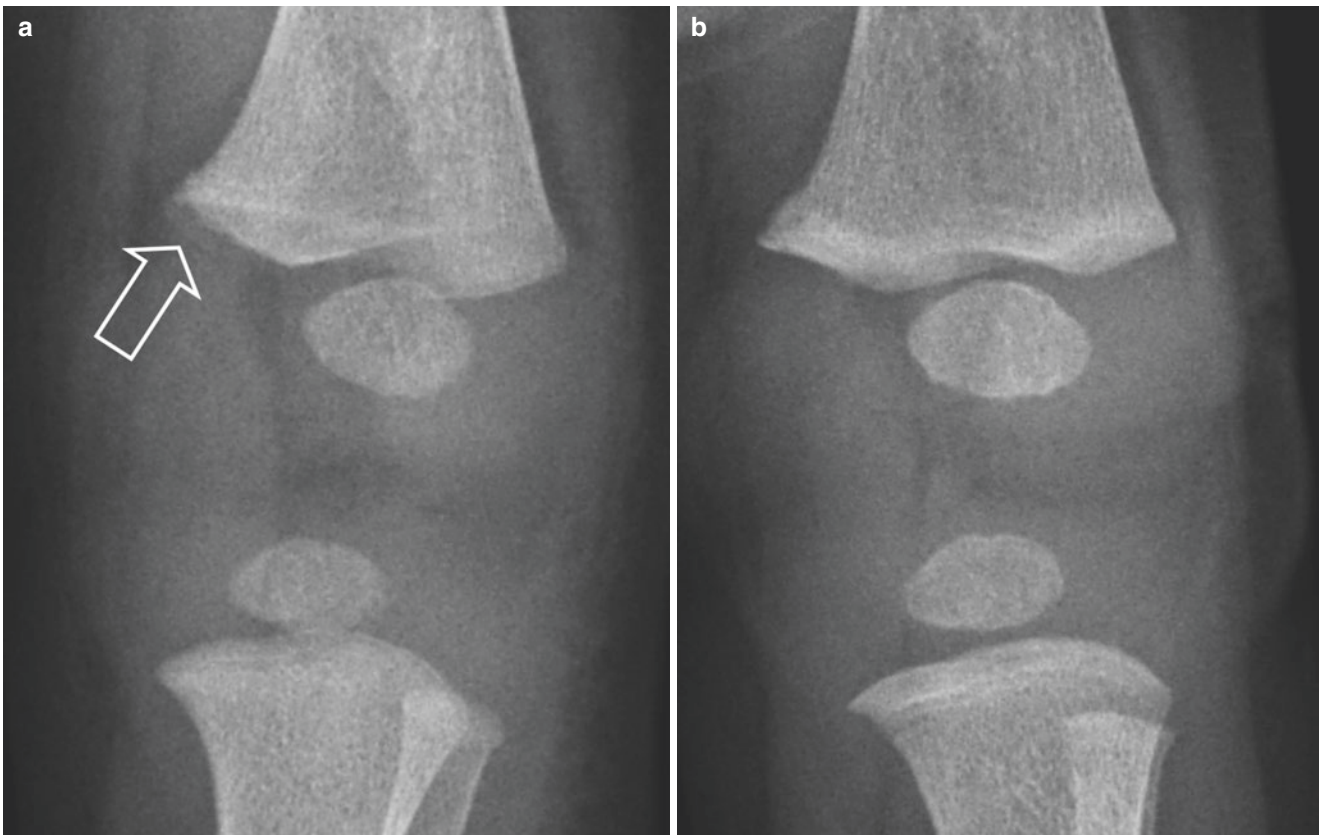


Fig. 12.11 (**a**) Four-month-old infant, the skeletal survey showed an irregularity at the medial side of the distal femur metaphysis (arrow). (**b**) Follow-up radiograph after 14 days shows local sclerosis in keeping with a healing fracture

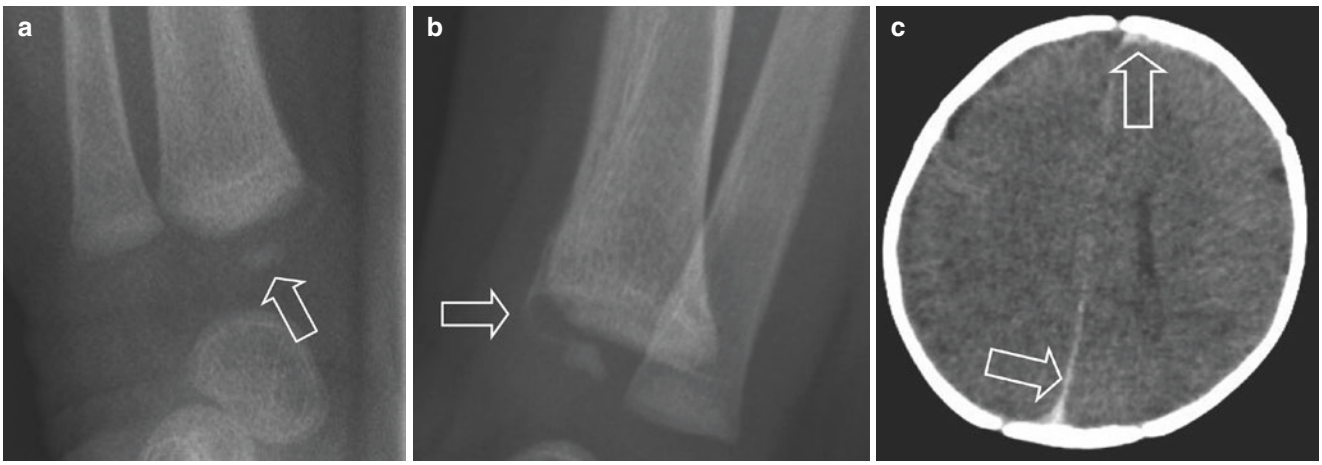


Fig. 12.12 Previously healthy two-week-old neonate who was admitted because of convulsions. (**a**) AP radiograph of the ankle shows a metaphyseal corner fracture of the medial tibial metaphysis (arrow). (**b**) Lateral radiograph shows the MCF located on the anterior side (arrow). (**c**) CT of the head shows a subdural hematoma (arrows)

Fig. 12.13 Medial metaphyseal corner fracture of the distal tibia metaphysis (arrow)

margin of the metaphysis (Fig. 12.13) [12, 13]. After the lower extremity locations the proximal humerus is most affected location (Fig. 12.10b) Fractures to the elbow and wrist have been reported less frequently (Figs. 12.10c and 12.14a, b) [7, 13–18]. The long-term consequences of MCFs appear to be minimal or even absent [19].

The rate of healing is variable. Therefore, it is not possible to give a precise timing of injury based on radiographic findings [20–22]. Because the periosteum does not have to be disrupted, commonly no healing features are seen at all (Fig. 12.15a, b) [21]. If there is significant displacement and periosteal stripping then sclerosis and subperiosteal new bone formation (SPNBF) may be present [23] (Fig. 12.16a, b). Another feature of healing MCFs is small cartilaginous protrusions from the growth plate into the metaphysis (Fig. 12.17). Based on extensive experience with follow-up skeletal surveys, Kleinman suggests that most healing MCFs become radiographically inconspicuous at 4 weeks and completely healed at 6 weeks [24]. Tsai et al. found subperiosteal new bone formation in an estimated prevalence of 34% on single point-in-time frontal radiographs of distal tibial MCFs [22]. When employing additional radiographs (both initial lateral view and follow-up skeletal survey) detection increased to

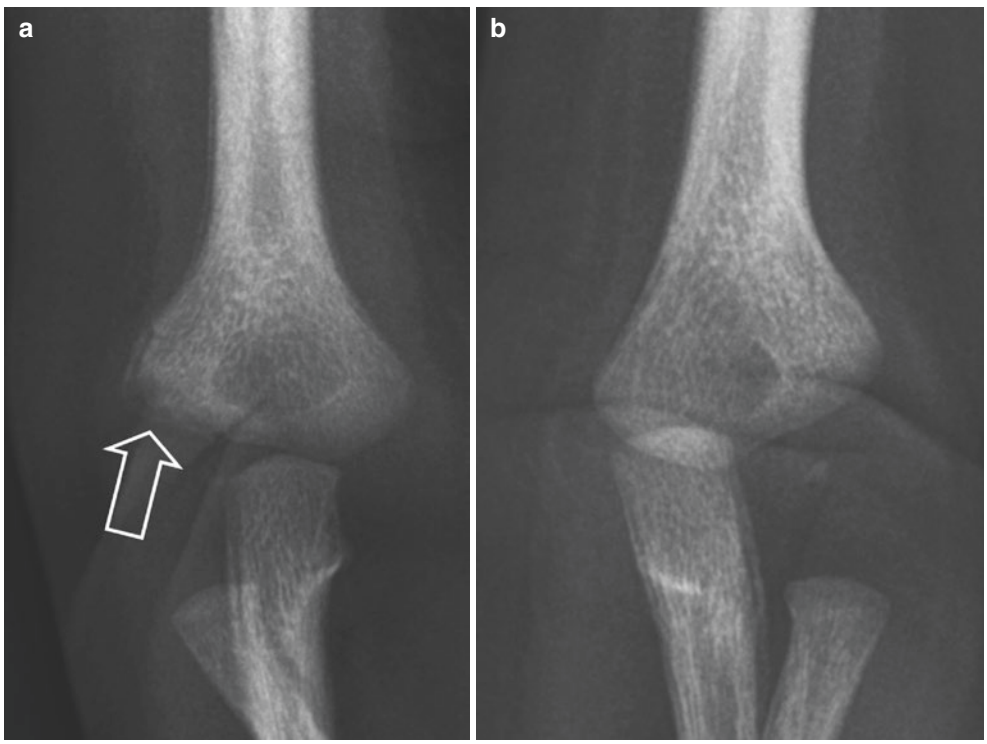


Fig. 12.14 (**a**) Extended metaphyseal corner fracture of the distal humerus in a 3-month-old infant (arrow). (**b**) After one month there is complete healing, note the presence of a healing fracture of the proximal ulna

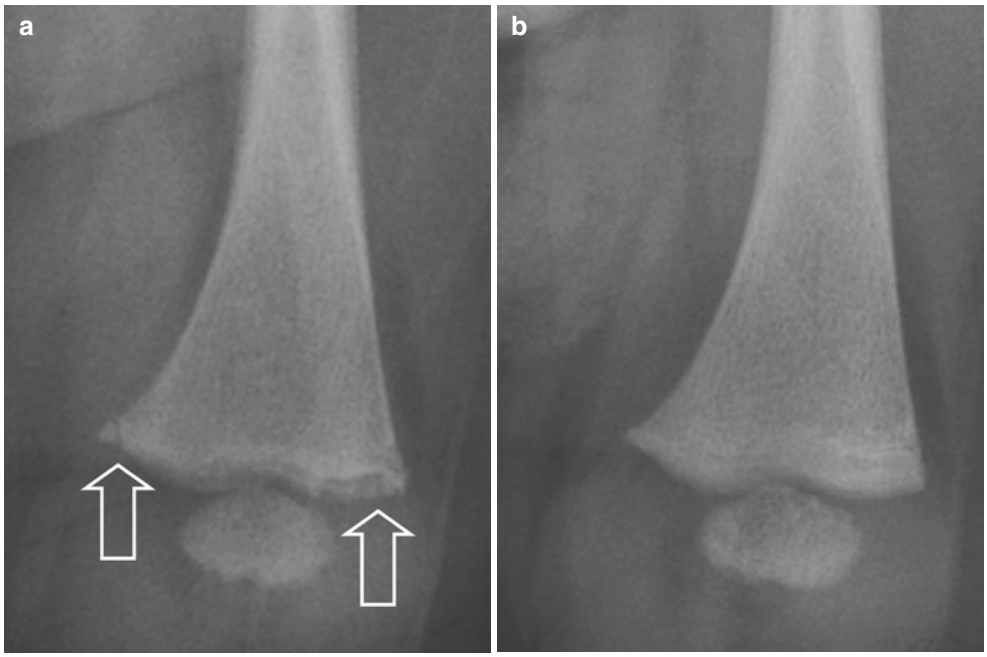


Fig. 12.15 (**a**) Metaphyseal corner fracture or the distal femur (arrows). (**b**) Radiograph after 18 days shows a normalized distal femur without signs of a healing fracture

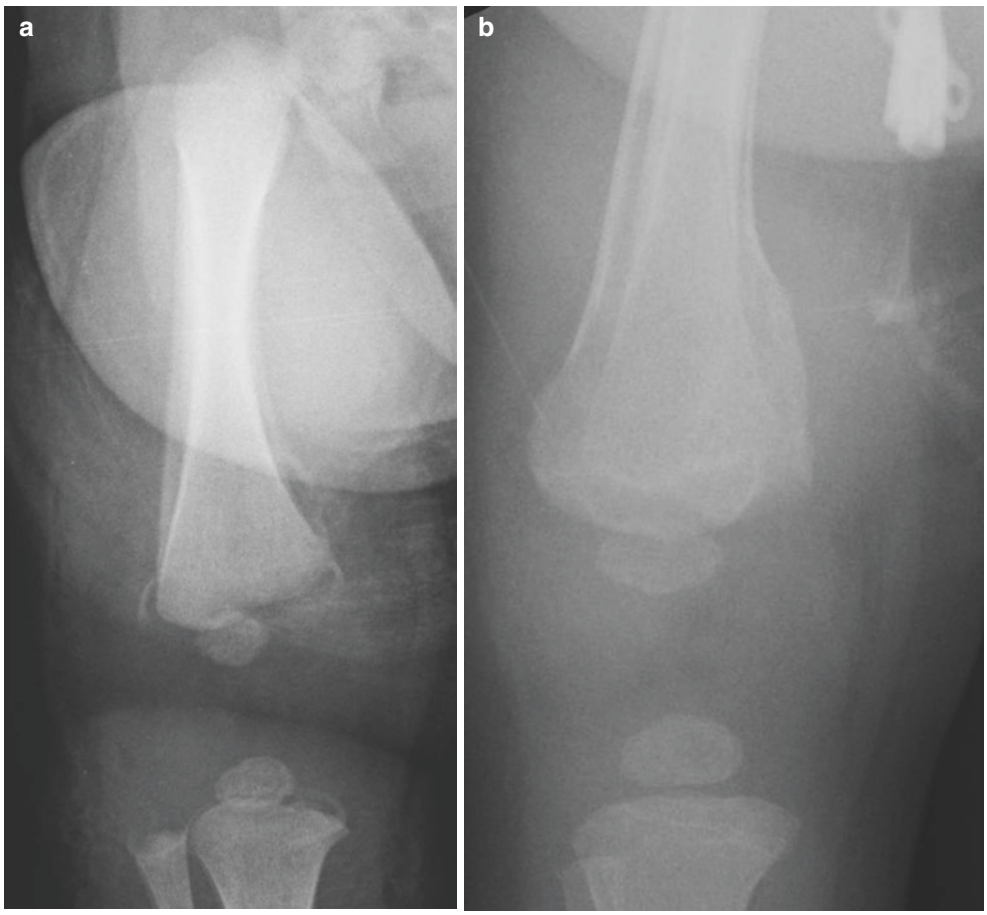


Fig. 12.16 (**a**) Radiograph of the right leg shows metaphyseal corner fractures of the distal femur and proximal tibia. (**b**) Follow-up skeletal survey shows abundant subperiosteal new bone formation along the shaft of the femur and a fully healed proximal tibia

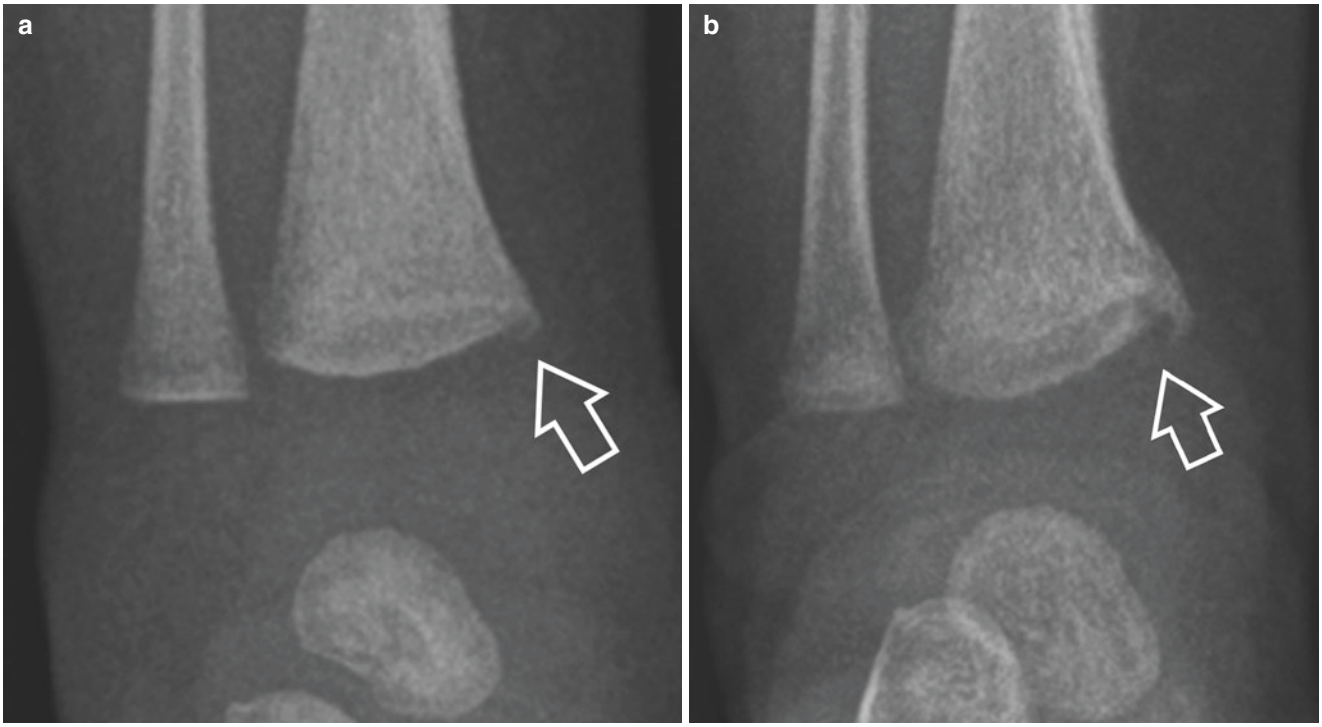


Fig. 12.17 (**a**) Radiograph of the distal tibia of a 2-week-old infant, who also had skull, rib, and clavicle fractures shows a metaphyseal corner fracture (arrow). (**b**) Radiograph after 3 weeks shows a cartilaginous protrusion from the growth plate into the metaphysis (arrow)

71%, but still 29% of distal tibial MCFs failed to demonstrate periosteal reaction. Karmazyn et al. saw subperiosteal new bone formation in only 47% (16/34) of the cases, according to the authors probably due to a higher percentage of acute MCFs in their initial series [20]. In the retrospective cohort study by Barber et al. skeletal surveys and follow-up skeletal surveys of 567 infants and children showed 124 MCFs in 50 cases [10]. On the initial skeletal survey 72 (58%) of the MCFs were healing, including 12 fractures evident only on follow-up survey. Unfortunately, the article did not describe which radiographic healing features were seen.

These studies underscore that, if non-accidental trauma is suspected, the follow-up skeletal survey has important additional value in the detection, confirmation, and dating of fractures. This applies in particular to the determination of MCFs, as others pointed out before [25–29].

Other radiological modalities such as whole-body MRI and PET images have not shown sufficiently successful in the detection of MCFs [30–32]. Proisy et al. compared the skeletal survey to whole-body MRI and bone scintigraphy [32]. In their study they found a total of 29 MCF in 13 children on the skeletal survey. Fifteen (51.7%) of these cases were detected by whole-body MRI and nine (31%) were detected by bone scintigraphy. It is suggested that ultrasound may help determine the presence of MCFs whenever radiographs are equivocal [33, 34]. A study on distal tibias of foetal piglets and a study on bone specimens from five fatally abused infants with MCFs both used 3D high-resolution micro-CT coupled with histopathology to depict the region of the chondro-osseous junction (the region of the trabecular transition zone) [4, 35]. The authors concluded that 'High-resolution CT coupled with histopathology provides elucidation of the morphology of the MCF' for now the use of micro-CT should be considered for the research domain only. High quality-thin-sliced volume CT scanning is required to make 3D- and multiplanar reconstructions diagnostic.

More research is necessary to determine the diagnostic value of ultrasound and 3D high-resolution computed tomography in the diagnosis of MCF [33, 34].

12.3.2.2 Cause of Metaphyseal Corner Fractures

Metaphyseal corner fractures are thought to be caused by torsional and traction shear strains applied across the metaphysis, for example when an infant's extremity is pulled or twisted or if the child is 'shaken' (Fig. 12.18) [24, 36]. Metaphyseal corner fractures were also experimentally produced in immature porcine pelvic limbs through application of controlled varus and valgus bending [9, 37].

Adamsbaum et al. performed a 15-year retrospective study, based on more than 500 cases from French courts, where they selected all children with at least one MCF, this yielded a study population of 67 children with a median age of 3.4 months [11]. Of these children 44 (66%) had multiple

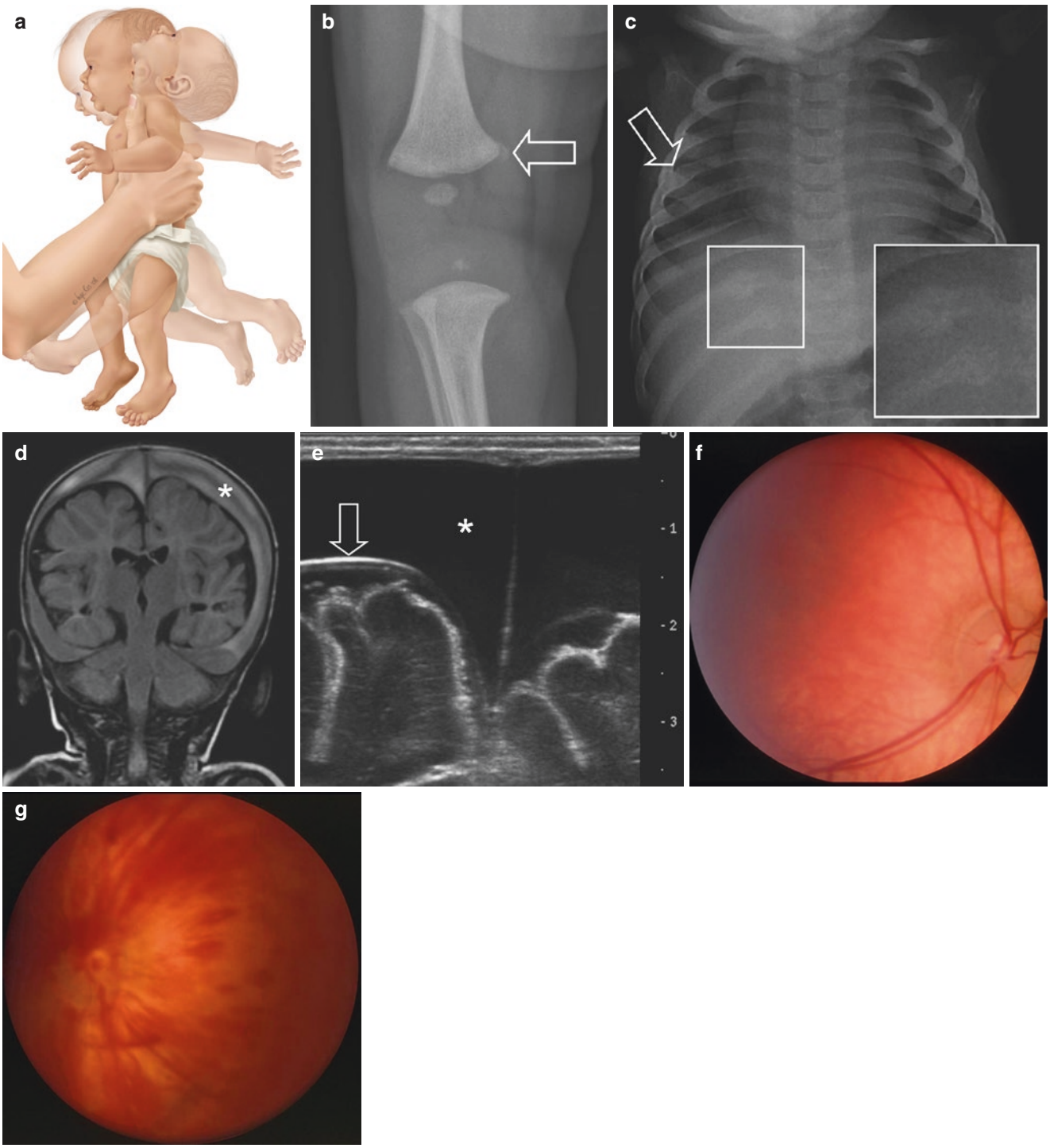


Fig. 12.18 (**a**) Graphic representation of a shaking incident. (**b**) Two-month-old boy with inflicted traumatic brain injury. The radiograph of the skeletal survey shows a metaphyseal corner fracture of the right distal femur (open arrow). (**c**) Four-month-old girl with inflicted traumatic brain injury. The skeletal survey shows a healing posterior fracture of the 9th right rib (see inset) Furthermore, there is an already healed rib fracture visible of the 5th right rib (open arrow). (**d**) MRI (T2 FLAIR) of this girl (**c**) shows a bilateral subdural haematoma (asterisk). (**e**) Cranial ultrasonography of this girl (**c**) shows the bilateral subdural haematoma (asterisk). Displacement of the arachnoid membrane (open arrow) is distinctly visible. (**f**) Normal view of the retina of a normal right eye at fundoscopy. (**g**) Diffuse retinal bleed in a left eye at fundoscopy resulting from inflicted skull/brain injury

MCFs. In 27 cases there was a confessing perpetrator and they described that their actions were abusive, violent, and intentional. With respect to the abuse they stated that they exerted *'excessive stress on the joints defined as "indirect skeletal forces" with "torsion, traction, violent compression (or crushing), and forced movements (crossing the arms, folding the legs up over the abdomen, separating the thighs'*. Diapering was the most common reported circumstance in which violent handling was described by male perpetrators only (44%), followed by dressing/undressing (30%).

12.3.2.3 Manner of Metaphyseal Corner Fractures

Metaphyseal corner fractures can be sustained due to birth trauma and due to trauma after birth. If sustained after birth the fracture may occur due to accidental and non-accidental trauma. Metaphyseal corner fractures may also occur due to medical procedures.

Despite the fact that the fracture may occur due to several circumstances, it is a highly specific fracture for non-accidental trauma in young children, characteristically seen in infants [8, 23, 38–41]. Caffey was the first to describe a '*metaphyseal fragment partially or completely separated from the end of the shaft*' [38].

Metaphyseal corner fractures are rarely, if ever, a reason for seeking medical consultation. They are usually found as occult findings on skeletal surveys. Metaphyseal corner fractures can be found in approximately 30% of children under the age of 12 months of whom a skeletal survey was made because of suspected non-accidental trauma [41]. Metaphyseal corner fractures are commonly encountered in infants with high-risk factors for non-accidental trauma (significant intracranial injury, retinal haemorrhages, and skeletal injuries) and are rare in infants with skull fractures associated with falls, but no other risk factors [42].

Kleinman et al. evaluated 31 deceased infants for the presence and distribution of fractures [43]. They found a total of 165 fractures of which there were 72 (44%) long bone fractures, of these the most commonly encountered were metaphyseal corner fractures with 64 fractures (89% of all long bone fractures and 39% of the total amount of fractures. In a large cohort of 2,890 infants evaluated for physical abuse there were 119 (4.1%) with MCFs and of them 84% had at least one non-MCF fractures identified [44].

Although the MCF is commonly seen in high-risk for non-accidental trauma cases, in some cases the occurrence due to medical procedures and accidental circumstances should be considered (Fig. 12.19a, b).

MCFs have been reported after vaginal breech delivery and Caesarean section both with or without attempted external cephalic version (Fig. 12.20a, b): Sieswerda-Hoogendoorn et al. report a case of a term neonate who was born via vaginal breech delivery after an unsuccessful external cephalic version (ECV). After birth the baby was admitted to the neonatal intensive care unit (NICU), where irritability was noted during diaper changing and a MCF of the right distal femur was diagnosed on day 6 of life [45].

There are two other case reports reporting a MCF after ECV. In the first case ECV was performed on a 36-year-old primipara woman [46]. The external version was followed by an emergency Caesarean section because of blood-stained cervical discharge. After birth a swollen right leg with diminished movement was seen. This was also recorded on photographs made in hospital. The parents returned one week after discharge and a radiograph then showed a MCF of the distal right femur.

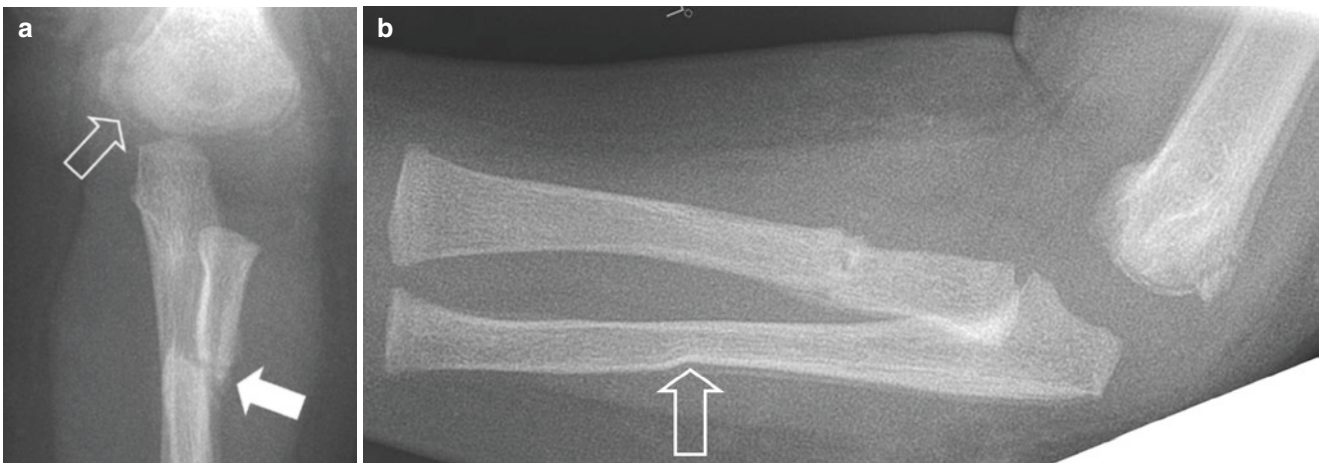


Fig. 12.19 Two-month-old infant who was attacked by a pit bull terrier. (**a**) AP radiograph after two weeks shows a transverse proximal radius fracture with signs of healing (arrow) and a metaphyseal corner fracture of the distal humerus with callus formation and subperiosteal new bone formation (open arrow). (**b**) Lateral radiograph obtained at the same time also shows a mid-diaphyseal torus fracture of the ulna (arrow)

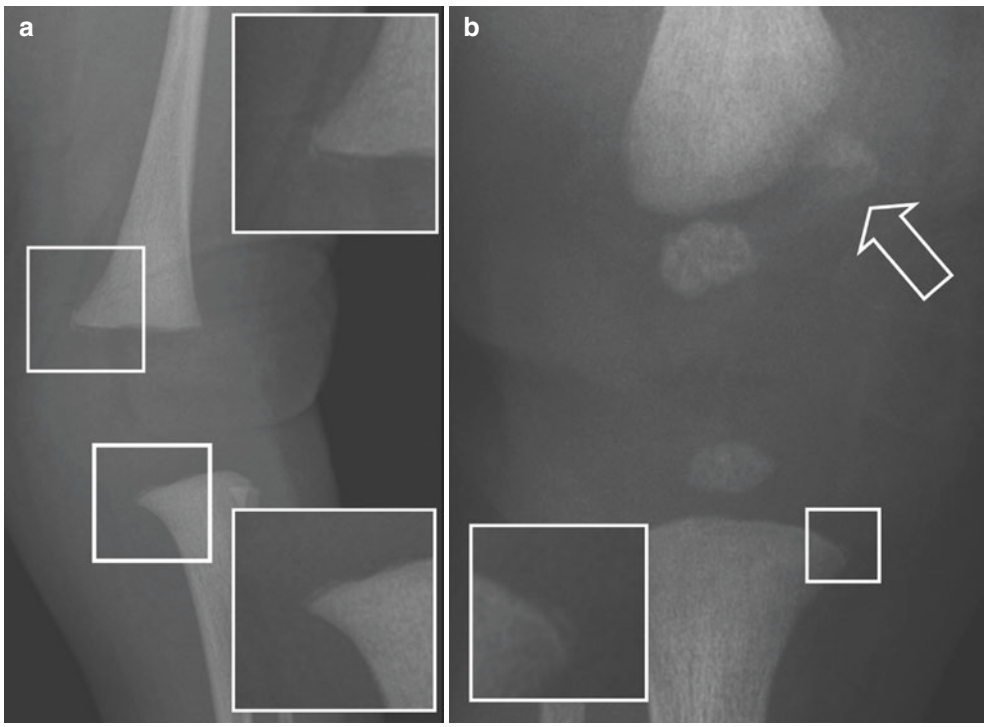


Fig. 12.20 (**a**) Term neonate, born at 39 weeks. Physical examination shows abnormal alignment of the left knee after uncomplicated delivery. A radiograph of the knee shows a metaphyseal corner fracture of the distal femur and the proximal tibia (see inset). (**b**) Term neonate after birth shows a swollen right knee after a complicated breech delivery. A radiograph of the knee shows a metaphyseal corner fracture of the proximal tibia (see inset) and a Salter–Harris type II fracture of the distal femur

In the second case, ECV was performed on a 29-year-old primipara, although successful an emergency caesarean section had to be performed due to foetal stress [47]. After birth, the neonate was fussy and showed decreased movement of the left leg, a radiograph three hours after birth showed a corner fracture of the distal femur and a bucket-handle fracture of the proximal tibia. However, in both cases it is questionable whether the MCF was caused by the version or by the emergency caesarean section.

In a retrospective analysis over a period of 22 years, O' Connell and Donohue identified three neonates born by elective Caesarean section (two breech and one cephalic presentation) with a MCF of a distal femur [48]. Lee et al. reported a MCF of distal tibia following a difficult footling breech delivery [49]. The authors state that the traction and torque placed on the legs during this difficult delivery were a potential mechanism for this injury. Finally, Buonuomo et al. describe a neonate with multiple fractures, among which a metaphyseal fracture of the femur, ultimately resulting in the diagnosis infantile myofibromatosis [50].

MCFs have been described to occur due to medical procedures:

- Grayev et al. reported the occurrence in serial casting treatment of clubfeet in 7 infants, who were considered not to be victims of child abuse. One child was abused, in this case the skeletal survey also showed 24 rib fractures [51].
- Burrell et al. reported a 20-day-old infant with a diagnosis of congenital vertical talus who sustained a metaphyseal corner fracture of the distal tibia during manipulation in preparation for intravenous line placement [52]. The event was independently witnessed, including an audible 'pop' at the time of the fracture. Prior X-rays showed normal bones.
- Della Grotta et al. suggested physical therapy with massage, passive range of motion, and positioning techniques of the lower extremities as the circumstances under which a MCF of the right proximal tibia (in combination with a shaft fracture of the right femur) occurred in an infant with a myelomeningocele, hypertonic lower extremities that lacked sensation, as well as bilateral flexion contractures of the knees and club feet [53]. The child remained in the hospital at the time when these fractures occurred and a child abuse evaluation was negative which made the authors conclude that accidental trauma secondary to physical therapy was the likely aetiology of the MCF.

Two cases of motor vehicle collision-related extremity MCF are described by Culotta et al. although they acknowledge that an alternative clinical consideration for each of the babies is that they had the misfortune to suffer both MVC and physical abuse while with their caregivers [54]. In both of the cases, the caregiver reported that the infant was restrained in a rear-facing car seat behind the driver's seat at the moment of the car accident.

12.3.2.4 Differential Diagnosis

There are several radiological normal variants that may be mistaken for MCFs [55–57]:

- Step-off, an almost 90 degrees angulation in the cortex (Fig. 12.21).
- Beak, seen in medial projection of the proximal humerus and proximal tibia (Fig. 12.22).
- Spur, a discrete longitudinal projection of bone that is continuous with the cortex and extends beyond the metaphyseal margin(Figs. 12.23, 12.24, and 12.25).
- Metaphyseal fragmentation occurs in children of 15 months and older occasionally encountered in healthy children of 15 months and older with physiologic bowing (Fig. 12.26) [58].

Lesions that can have some similarity to the appearance of MCF can be found in diseases such as rickets, osteomyelitis, congenital syphilis, and spondylometaphyseal dysplasia 'corner fracture type' and Menke's disease (see Chap. 14).

12.3.3 Salter–Harris Fractures

12.3.3.1 Introduction

Trauma during childhood may result in typical fractures with involvement of the growth plate, the so-called Salter–Harris fractures. Salter and Harris described five types of fractures (Table 12.2). These fractures are seen in approximately 18–30% of all trauma-related long bone fractures in children [59].

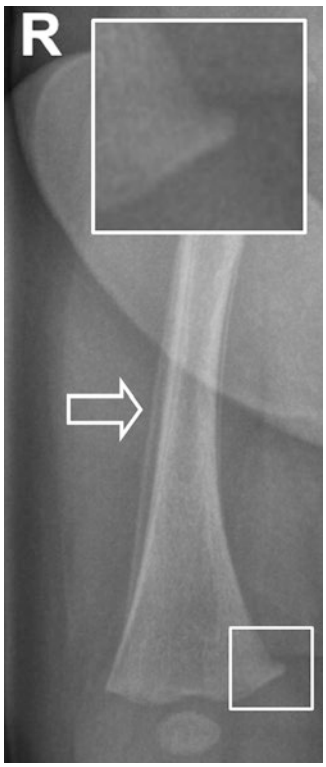


Fig. 12.21 The medial side of the distal femur metaphysis shows a physiological step-off (inset). Note that there is also physiological subperiosteal new bone formation

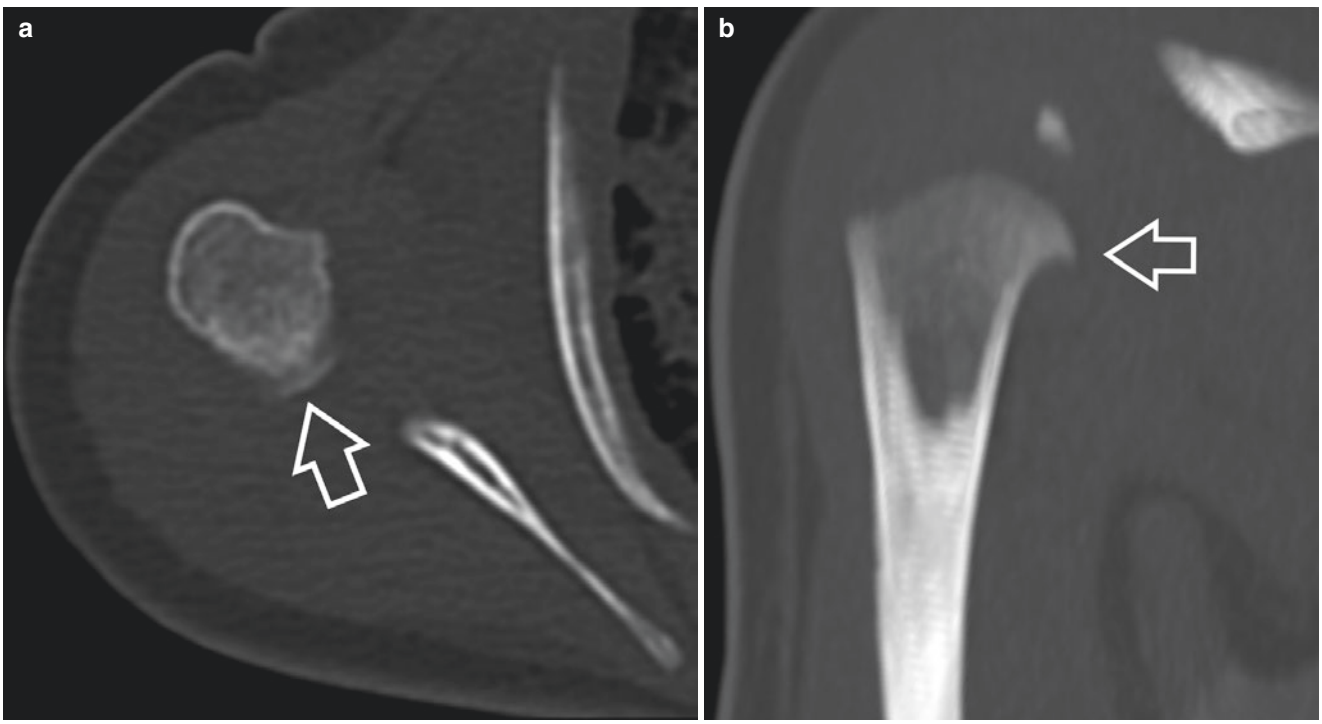


Fig. 12.22 (**a**) Pseudoavulsion fracture of the proximal humerus on native axial image (arrow). (**b**) Coronal reconstruction shows that the pseudoavulsion is caused by breaking of the humerus (arrow)

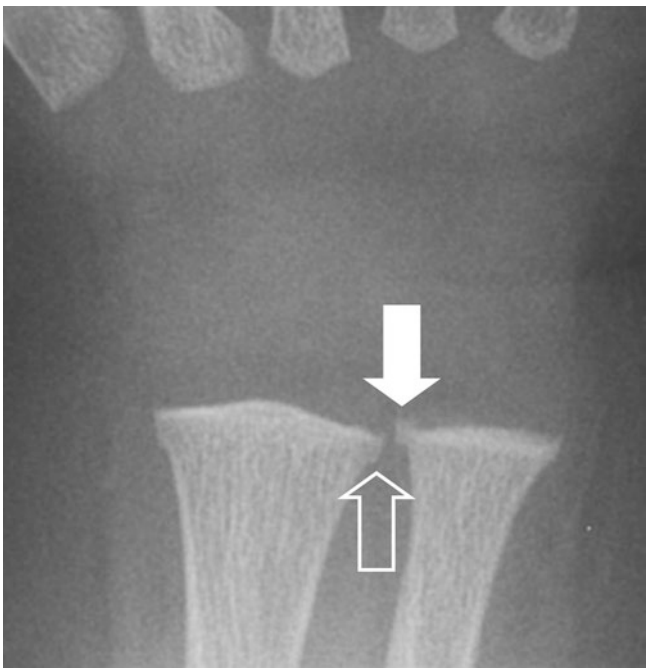


Fig. 12.23 Radiograph of the wrist shows a spur on the lateral side of the distal ulna (arrow) and a step-off on the medial side of the distal radius (open arrow)

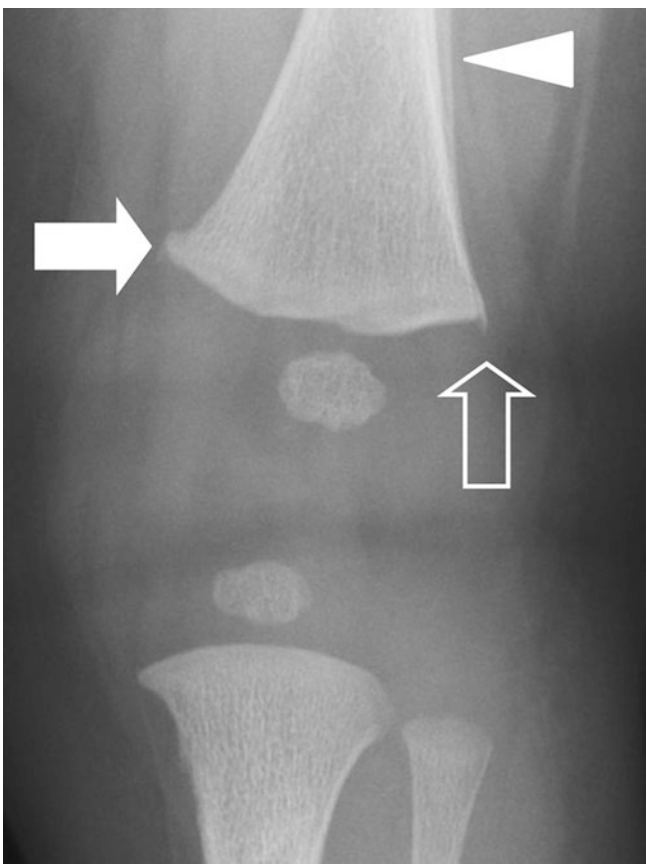


Fig. 12.24 Radiograph of the knee shows a spur on the lateral side of the distal femur (open arrow), a step-off of the medial side of the distal femur (arrow), and physiological sub-periosteal new bone formation along the diaphysis (arrowhead)

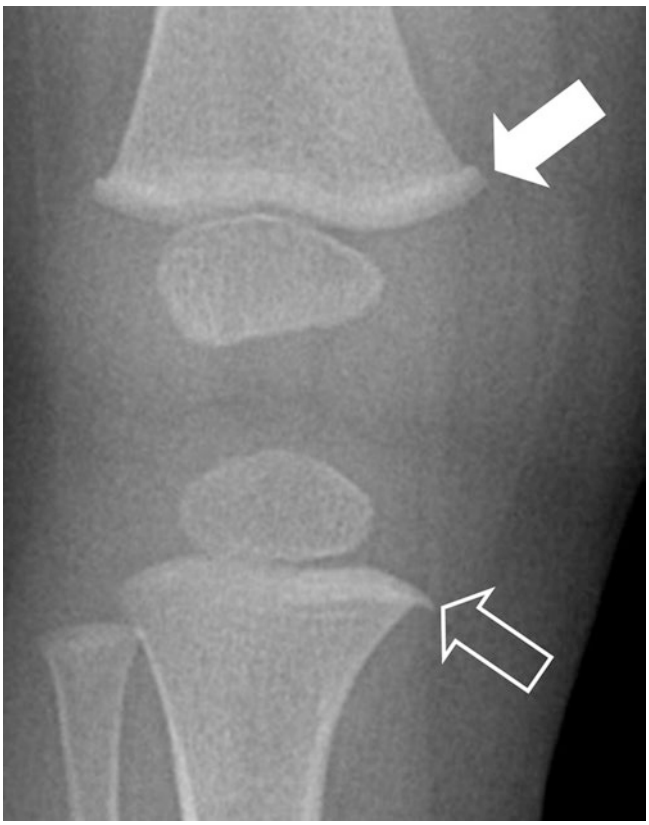


Fig. 12.25 More than one normal variant can be seen in a single child. The lateral distal femur metaphysis shows a step-off (arrow) and the lateral proximal tibia metaphysis shows a spur (open arrow)

Fig. 12.26 Metaphyseal fragmentation in a 1-year 9 months-old child with tibia vara

Table 12.2 Classification of meta-epiphyseal fractures according to Salter–Harris

Type			Mnemonic	
I		In type I the fracture line 'follows' the growth plate, separating epiphysis, and metaphysis. The growth plate is still attached to the epiphysis. Usually, there is no damage to the growth plate. Type I is seen in particular in young children. Relative incidence is 8.5%. The mechanism involved is shearing. Dislocation is only seen when the periosteum has been damaged. The healing process is quick (usually within 2–3 weeks).	**S**	**S**traight across
II		Type II runs through the metaphysis and (in part) the growth plate along the metaphyseal transition zone. It is the most common type (relative incidence 73%), generally in children >10 years of age. Type II heals fast. As in type I, the mechanism involved is a shearing force or avulsion due to an angular force. This type of fracture usually heals quickly.	**A**	**A**bove
III		Type III runs through the epiphysis and (in part) the growth plate. Although the growth zone has been damaged, hardly any growth disturbance is seen after a type III fracture. is rarely seen, and then mostly to the lower legs. Type III is quite rare (6.5%) and often seen at the lower legs in children in whom the growth plate is partially fused.	**L**	**L**ow or be**L**ow

(continued)

Table 12.2 (continued)

Type			Mnemonic	
IV		Type IV runs across the epiphysis, growth plate, and metaphysis. The relative incidence is 12%. The risk for focal physeal arrest is substantial and treatment is typically surgical rather than conservative. Focal physeal arrest may lead to deformation of the joint as a result of the bony bridging of the growth plate which may impede local growth.	**T**	**T**hrough
V		Type V is a compression fracture of the growth plate due to axial loading. This type is commonly seen in the knee and ankle. This type is rare (<1%) and usually occult on initial imaging. The risk for focal physeal arrest is high.	**ER**	**ER**asure of growth plate

12.3.3.2 Cause of Salter–Harris Fractures

The Salter–Harris (SH) classification is based on the mechanism of injury (cause) and the relationship of the fracture line to the growing cells of the growth plate and is correlated with the prognosis concerning growth disturbance [3, 60–62].

According to Salter and Harris types I–IV are the result of shearing/avulsion forces while type V results from compression of the growth plate. Vertically oriented splitting compression force across the epiphysis, physis, and adjacent metaphysis can cause type IV SH fractures [62, 63].

Because of the lack of epiphyseal ossification in infants, Salter–Harris type II fractures may be missed on conventional radiographs or may appear as dislocations [64, 65]. MRI or ultrasonography may then be of help in diagnosing SH fractures in this age group (Fig. 12.27a, b) [66].

12.3.3.3 Manner of Salter–Harris Fractures

In childhood and adolescence, Salter–Harris fractures mostly are sustained due to accidental trauma. These fractures occur most frequently in young adolescents, aged 11–15 years, except for the humeral physeal fractures (occurring almost twice as often in the distal humerus) which also have peak incidences at ages 4–6 years [67]. The most common sites are the phalanges of the fingers and the distal radius [59, 67]. Salter–Harris type II fractures are the most prevalent (Fig. 12.28) [67]. Humeral and femoral Salter–Harris fractures are also found as birth injuries due to force of labour and obstetric manipulation [68–74].

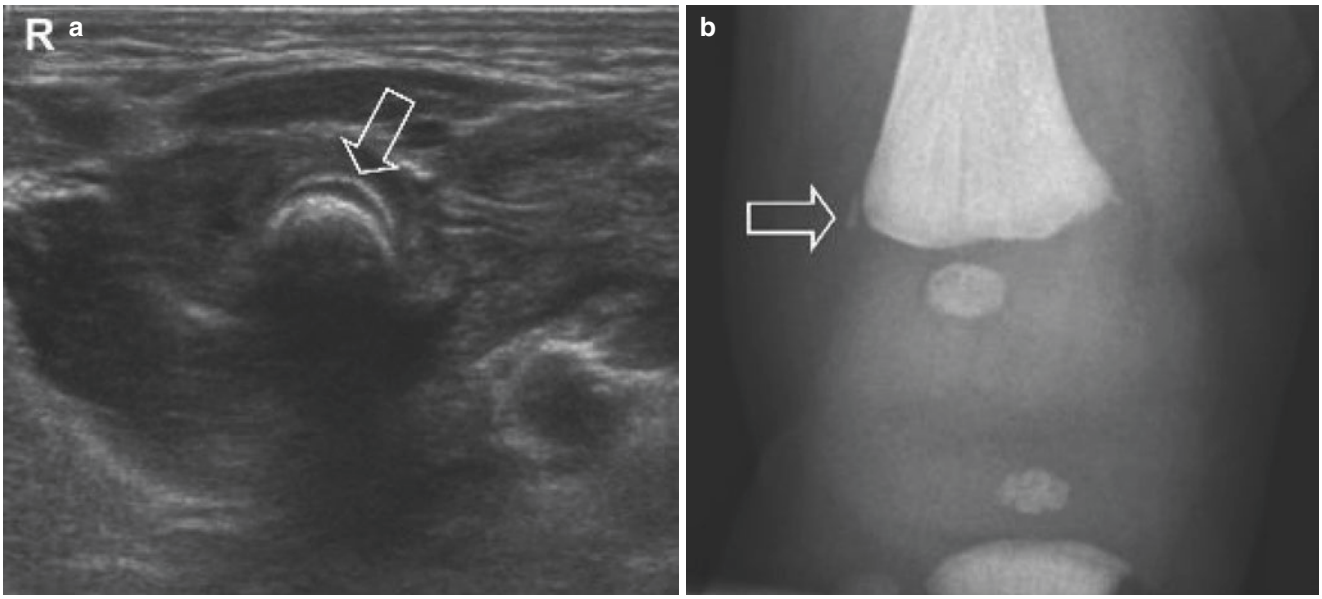


Fig. 12.27 Term neonate born after a complicated breech delivery after premature rupture of membranes. After birth the child was noted to have a slightly swollen knee. (**a**) Ultrasonography of the knee showed a metaphyseal corner fracture (arrow) (**b**) AP radiograph confirms the presence of this metaphyseal corner fracture (arrow)

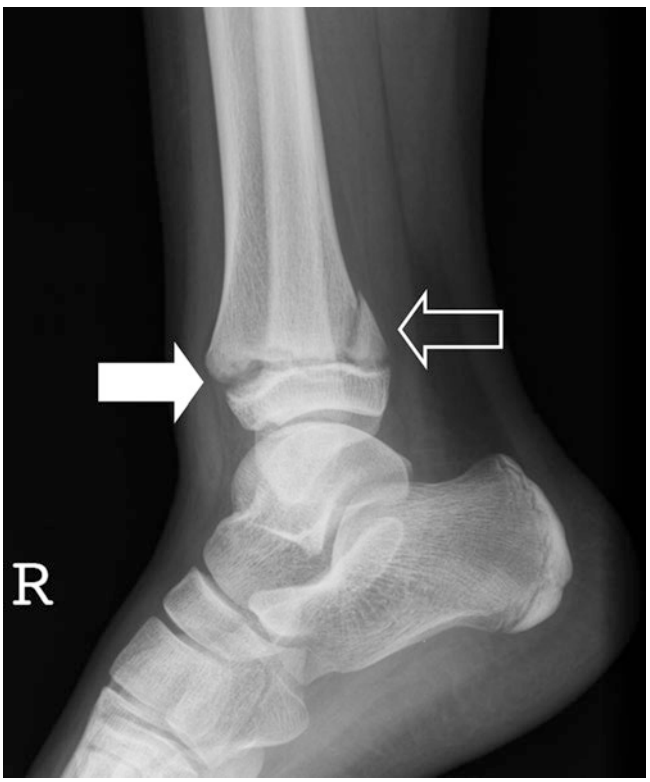


Fig. 12.28 Twelve-year-old girl (with unknown trauma) with a Salter–Harris type II fracture of the distal tibia (open arrow). The fracture through the growth plate can be identified by the anterior diastasis (arrow)

Literature on physeal fractures in children with non-accidental trauma is scarce. A few case reports and case series mention physeal fractures in the proximal femur and the distal humerus with proven non-accidental circumstances [65, 75, 76].

Barber performed a large cohort study to define the yield of rigorously performed skeletal surveys in infants with suspected physical abuse [10]. In this study 13 of the 313 (4%) infants diagnosed with a fracture had at least one Salter–Harris fracture (all type II), most frequently of the distal humerus. All SH-fractures were evident on the initial survey and four of the children had other positive findings on the skeletal survey.

12.3.4 Epiphyseal Transitional Fractures

Epiphyseal transitional fractures typically occur in the distal tibia during the 18-month period of closure of the growth plate aged between 12 and 15 years. Closure of the distal tibial growth plate starts centrally and medially before progressing laterally. This partial closure leaves the ankle vulnerable to these types of fractures, especially during external rotation.

The triplane fracture configuration consists of:

(1) A fracture line along the coronal plane through the posterior metaphysis.

(2) A fracture line along the sagittal plane through the epiphysis.
(3) A fracture line along the transverse plane through the growth plate.

The fracture may consist of 2–4 fragments. The triplane fracture appears as a Salter–Harris type II on lateral radiographs and as a Salter-Harris type III on AP radiographs. CT has a definite impact on fracture classification, displacement, and treatment [77]. A gap of 2 mm or more is considered by some authors as the threshold between conservative and surgical treatment. Triplane fractures account for 5–15% of all ankle fractures in adolescents [78]. Growth arrest, although usually insignificant, may occur in 7–21%.

The Tillaux fracture is a SH-type III involving the anterolateral aspect of the distal tibial epiphysis which is seen in adolescents in whom the medial section of the distal tibia metaphysis has started to close. As a result, only the anterolateral part of the growth plate is open and vulnerable to injury and consequently, the Tillaux fracture is seen in adolescents aged between 12 and 15 years. Lateral triplane fractures, just like Tillaux fractures, are caused by supination, combined with external rotation (twisting), while medial triplane fractures are caused by adduction [78]. Epiphyseal transitional fractures have only been reported due to accidental trauma.

12.4 Humerus

12.4.1 General Aspects of Humerus Fractures

Humerus fractures are most frequently seen in children under the age of 3 years and above the age of 12 years [79]. This is irrespective of the circumstances under which the fractures were sustained.

Fractures of the humerus have been described to occur before (very rare), during, or after birth. If sustained after birth, one should differentiate between accidental and non-accidental circumstances. According to Caviglia et al., one should be aware that the circumstances, under which humerus fractures are sustained vary between age groups [79].

12.4.2 Fractures of the Proximal Humerus

12.4.2.1 General Aspects of Fractures of the Proximal Humerus

Fractures of the proximal humerus account for 2–5% of all fractures in paediatric patients [80–86]. Proximal humeral fractures are 3–4 times more likely to occur in boys than girls and are most common in adolescents with a peak age of 15 years [87]. Chae et al. evaluated the findings in 41 children with proximal humerus fractures (aged 1 month to 15 years; mean age 8.6 years) and found that 56% of this type of fracture occurred in girls and 44% in boys [88].

Table 12.3 Neer-Horowitz classification [86].

Type I	Minimally displaced (<5 mm)
Type II	Displaced <1/3 of shaft width
Type III	Displaced greater than 1/3 and less than 2/3 of shaft width
Type IV	Displaced greater than 2/3 of shaft width

Proximal humerus fractures include fractures of the proximal metaphysis or the proximal physis [84, 89].

Metaphyseal fractures account for about 70% of the fractures of the proximal humerus in paediatric patients and can be described using the Neer-Horowitz classification based on the amount of angulation and displacement at the fracture site (Table 12.3) [89–91]. Fractures at this location typically occur in children aged 5–12 years [87].

Fractures of the physis account for around 30% of proximal humerus fractures and are categorized according to the Salter–Harris classification [91]. SH-type I is most common in children under the age of 5 years, while SH type II is most common in children above the age of 12 years. According to Popkin et al. SH types III and IV are rare in paediatric patients and are usually associated with high-energy trauma [87].

12.4.2.2 Cause and Manner of Fractures of the Proximal Humerus

Fractures of the proximal humerus may occur during and after birth.

During Birth

Birth-related fractures can be found in all parts of the humerus with midshaft fractures being the most common [92, 93].

The risk of fractures of the proximal humerus is highest in large infants during vaginal delivery (although proximal humerus fractures have also been reported in smaller infants) or during breech delivery (irrespective of the size of the child) [94, 95]. Other known risk factors are labour dystocia and macrosomia (birth weight above 4.5 kg) [87].

Birth-related growth plate injuries have also been described. Varghese et al. presented two neonates with humeral growth plate fractures (Salter–Harris type 1), one at the proximal humerus and one at the distal humerus [96]. Jones et al. described a premature neonate, delivered by caesarean section due to malpresentation, with a Salter–Harris II fracture of the proximal humerus [97].

Popkin et al. stated that a proximal humerus fracture that is diagnosed in the first week of life is considered to be birth-related if no history of a trauma after birth is known: '*During the descent down the birth canal, the infant's arm can be placed in a variety of compromised positions that can result in a physeal fracture of the proximal humerus*'. According to Popkin et al. birth-related fractures of the proximal humerus are classic physeal separations or SH-type I injuries [87].

After Birth: Accidental and Non-accidental Circumstances

After birth fractures of the proximal humerus can occur due to direct or indirect trauma in either accidental or non-accidental circumstances.

In children under the age of 3 years one should always consider non-accidental circumstances [87, 91]. There are no radiographic findings on imaging of the shoulder that are suggestive of non-accidental circumstances in case of a proximal fracture. The incidence of proximal humerus fractures due to non-accidental circumstances is not known. Chae et al. evaluated the findings in 41 children with proximal humerus fractures (aged 1 month to 15 years). In only 1 child (age not specified) the circumstances were determined to be non-accidental.

In older children proximal humerus fractures usually are sustained in accidental circumstances due to a moderate-energy trauma, associated with falls, motor vehicle crashes, or sports participation [87].

Fractures may be due to a direct trauma (blunt force trauma) to the shoulder/proximal arm, usually to the posterior shoulder. This can be a direct blow/strike to the shoulder/proximal arm or a fall on the posterolateral part of the shoulder. They may also occur due to an indirect trauma, like a fall (backwards) on an outstretched hand, with the arm in abduction and external rotation, the hand in dorsiflexion, and the elbow in hyperextension. These fractures often occur during traffic accidents, sporting activities (contact sports, like hockey and soccer, horseback riding, gymnastics) and during play (Fig. 12.29) [81, 87, 98]. Popkin et al. also mentioned the occurrence of overuse injuries due to repetitive throwing in baseball (little league shoulder, due to overthrowing, mainly in baseball players aged 11–14 years). Another example given by Popkin et al. is the occurrence of an avulsion fracture of the lesser tuberosity in throwing athletes aged 12–15 years and in fly fishermen [87].

Fractures at this location may result from complications of underlying diseases such as tumours, metabolic diseases, and secondary neuropathies [81, 91, 99–101]. Proximal humerus fractures have also been described resulting from aneurysmal and unicameral bone cysts and can be associated with complications of radiation therapy [87].

Fig. 12.29 Subcapital humerus fracture in a 4-year-old girl who fell from a swing

12.4.3 Fractures of the Humerus Shaft

12.4.3.1 General Aspects of Humeral Shaft Fractures

The reported incidence of humeral shaft fractures in paediatric patients under the age of 16 years varies greatly. According to Caviglia et al. humeral shaft fractures account for around 0.75%, according to Marengo et al. for around 2–5% and according to Shrader for up to 10% of all fractures in paediatric patients [79, 84, 102]. Shaft fractures account for approximately 20% of all humerus fractures in children with an estimated incidence of 12–30 per 100.000 paediatric patients per year [103]. Shaft fractures seem to be more common in children under the age of 3 years and above the age of 12 years [79, 103]. Boys to girls ratio is around 2:1 [85].

Shaft fractures are described by [79, 104]:

- Anatomical location: proximal, middle, or distal third part of the shaft
- Fracture pattern: spiral, oblique, transverse, or comminuted
- Degree of displacement and angulation
- Presence of soft tissue damage: open or closed fracture

12.4.3.2 Cause and Manner of Humeral Shaft Fractures

Fractures of the humeral shaft have been described to occur before (very rare), during, or after birth. If sustained after birth, one should differentiate between accidental and non-accidental circumstances. According to Caviglia et al. one should be aware that the circumstances, under which

humeral shaft fractures are sustained vary between age groups [79]. In neonates, e.g. shaft fractures are seen mainly within the scope of birth trauma in macrosomic babies (Figs. 12.30 and 12.31a, b), while in children under the age of 3 years, non-accidental trauma should always be considered. In children above the age of 10 years, shaft fractures are often the result of direct or indirect blunt force accidental trauma (Fig. 12.29) [79].

Before Birth

Prenatally acquired shaft fractures are probably very rare and almost always found in children with severe congenital bone disease, e.g. osteogenesis imperfecta type II (see Chap. 14) [105]. One of the first reports on bilateral intrauterine humeral shaft fractures was by Barker in 1857 [106]. Barker described the findings in a newborn, who died within minutes after birth. The girl had numerous long bone fractures (humerus, radius, ulna, femur, tibia, fibula) and extensive skull abnormalities (absent frontal bone, absent parietal bone, abnormal occipital bone) (Fig. 12.32). Although no diagnosis was given in the article, one may suspect that the child today would probably be diagnosed with perinatal lethal type II osteogenesis imperfecta. Barker referred to several other case reports, in which numerous prenatally acquired fractures, including humerus fractures, had been described. In a follow-up article Barker stated: *'P.S. It may be worthy of remark that the lady, Mrs. L., who in September, 1856, gave birth to the foetus, the subject of the foregoing remarks, was yesterday (September 27th) delivered of a healthy and well formed female child at the full period'* [106].

Fig. 12.30 Birth-related humerus fracture after a complicated delivery due to shoulder dystocia

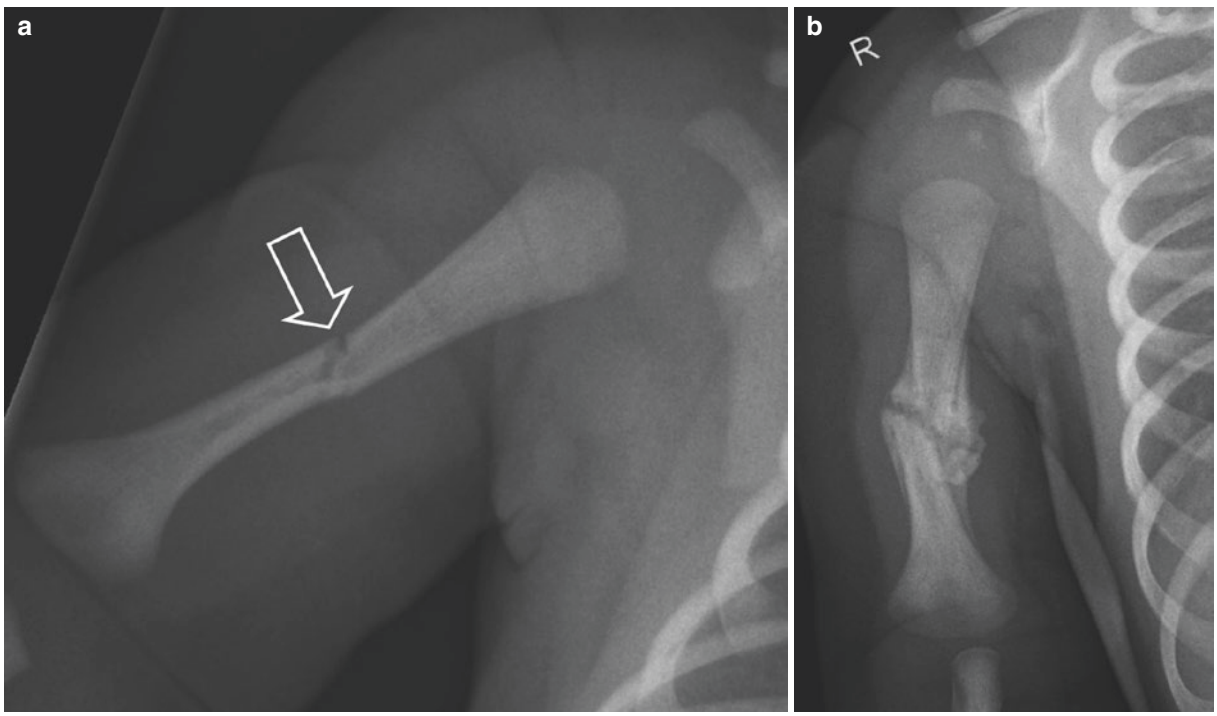


Fig. 12.31 Neonate born at 40 weeks gestational age, birthweight 4060 g (P89), vaginal delivery was complicated by a shoulder dystocia. During delivery the gynaecologist heard a 'crack'. (**a**) Radiography showed a mid-diaphyseal humerus fracture (arrow). (**b**) Follow-up radiography shows callus formation around the fracture

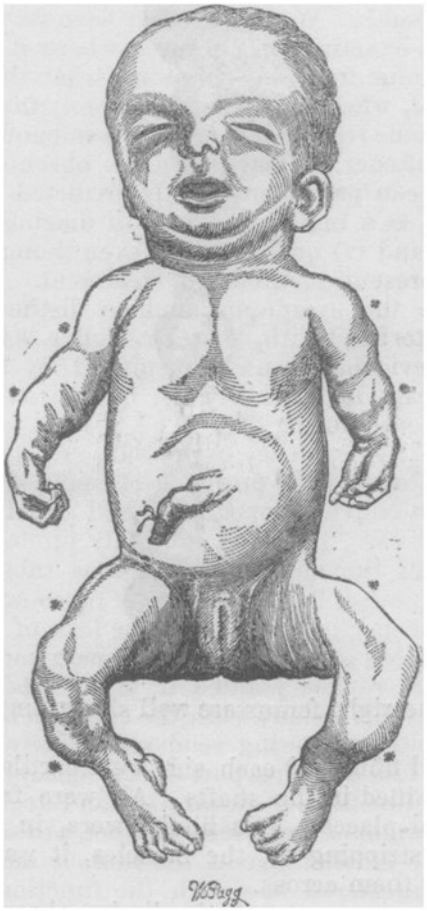


Fig. 12.32 Drawing of the newborn, who died within minutes after birth, described by Barker [106]. The girl had numerous long bone fractures

Fig. 12.33 Humerus shaft fracture in a one-day-old neonate after an uncomplicated vaginal delivery

During Birth

Birth-related humeral fractures can be found in all parts of the humerus (shaft, proximal, and distal humerus) with midshaft fractures being the most common [107, 108]. Shaft fractures may occur due to trauma during vaginal birth (Fig. 12.33), but also, although less common, in caesarean section (sometimes even bilaterally), due to limb extraction [92, 93, 108]. Shaft fractures are caused by hyperextension or rotation of the arm during passage through the birth canal [109–111]. Shaft fractures are often seen as birth trauma in infants due to obstetric manoeuvres during a breech delivery [112], but may also occur in vaginal delivery with shoulder dystocia [113–115].

The typical birth-related shaft fracture is a complete, transverse midshaft fracture (Fig. 12.34) [116].

Humeral (shaft) fractures are the second most common birth-related long bone fractures in the neonate after clavicular fractures [116]. Their exact incidence is not known and is estimated to be up to 0.6/1000 live births (Table 12.4) [107, 117–121]. Von Heideken et al. found that the occurrence of a birth-related shaft fracture was associated with maternal obesity, labour dystocia, shoulder dystocia, vacuum-assisted delivery, male sex, multiple birth, breech, preterm, large-for-gestational age, birth weight over 4000 g, and injury of brachial plexus [117]. A bone fragility diagnosis was recorded in 1% of the neonates birth-related humerus shaft fractures.

After Birth: Accidental or Non-accidental Circumstances

Transverse shaft fractures are caused by a direct trauma, a blunt force trauma, directly impacting the shaft (e.g. a blow). The more energy is transferred during the impact (the harder the blow hits the upper arm), the more likely the fracture is to be comminuted [122]. Spiral/oblique fractures are caused by an indirect trauma from a fall or another incident with humeral rotation or twisting, as may happen in arm wrestling (Fig. 12.34) [116, 122].

Concerning the occurrence of humeral shaft fractures after birth, Von Heideken et al. found an incidence after birth of 0.073 per 1000 children under the age of 1 year (142 children in a nationwide study in 1,855,267 infants under the age of 12 months). In 56% of these children falls were reported. In 14% of the shaft fractures were determined to have occurred in non-accidental circumstances. This concerned mainly children under the age of 6 months.

Fig. 12.34 One-month-old infant girl who, according to the parents, had fallen from a bed. The spiral fracture of the humerus does not correspond with the trauma description

Table 12.4 Incidence of birth-related humerus shaft fractures

Author	*N*	Incidence per 1000
Rubin [329]	15,435	0.45
Madsen [107]	105,119	0.36
Bhat [121]	34,946	0.20
Basha [120]	34,519	0.05
Suleiman [119]	5,030	0.60
Rehm [118]	87,461	0.15
Von Heideken [117]	1,855,267	0.10

Some authors state that humerus fractures (including fractures at locations other than the shaft) are the most common fractures in young children under the age of 3 years due to non-accidental trauma [96, 123]. Loder and Bookout found that shaft fractures were the second most common long bone fracture due to non-accidental trauma after tibial fractures [39]. Others report that most humerus fractures (46–81%, five different studies), especially in young children outside the neonatal period and under the age of 3 years occur due to non-accidental trauma. In children under the age of 15 months, the reported percentage of non-accidental trauma ranges from 67% to 100% [40, 124–127]. The most common locations in non-accidental trauma are midshaft and metaphyseal [96, 123].

Williams and Hardcastle published a 'best evidence topic report' on the relation between humeral shaft fractures and non-accidental trauma in children [128]. Their study comprised 44 articles, of which two were able to provide an indication regarding the formulated query: '*What is the specificity of an isolated proximal humerus fracture in children who are suspected of being abused*' [123, 129]. Their analysis provided the following clinical bottom line: '*Although a humerus fracture cannot be seen as pathognomonic for child abuse, such a fracture in a young child should always be followed up with a closer look into its origin*' (Fig. 12.35). Williams and Hardcastle maintain that both included studies tried to define the specificity of the various types of humerus fracture in relation to child abuse, but that in both studies there was no 'golden standard'. Consequently, it is feasible that in both studies children have been overlooked or that it was falsely concluded that non-accidental trauma was involved. Yet, it appears that the incidence of non-accidental trauma in this type of fracture is high. In particular in children under the age of 3 years, spiral and oblique fractures were more often the result of non-accidental trauma than of anything else.

Shaw et al. did a retrospective study on 34 children under the age of 3 years with a humeral shaft fracture [129]. The authors excluded children with a humerus fracture at a different places (supracondylar, epicondylar, condylar, proximal epiphyseal, and metaphyseal). From a revision of the clinical data and data from the county child protective services the authors established whether or not the child had probably sustained the fracture in non-accidental circumstances (defined as probable child abuse). Cases were reviewed independently by four physicians and were classified as probable abuse (non-accidental trauma), probable not abuse, and indeterminate. Shaw et al. concluded that most fractures of the humerus shaft were accidental. After evaluation, only 18% were classified as 'probable abuse'. Neither age nor fracture pattern was conclusive in differentiating between accidental and non-accidental circumstances. The history and findings other than the fracture itself were critical in establishing the circumstances. Based on these findings, the authors concluded that six factors can be essential in establishing whether non-accidental trauma should be suspected:

1. The presence of simultaneous or older injuries (Fig. 12.36).
2. Delay in seeking medical treatment.
3. Differences in or contradicting stories regarding the incident.
4. The child is accompanied by a person other than the one present at the incident.

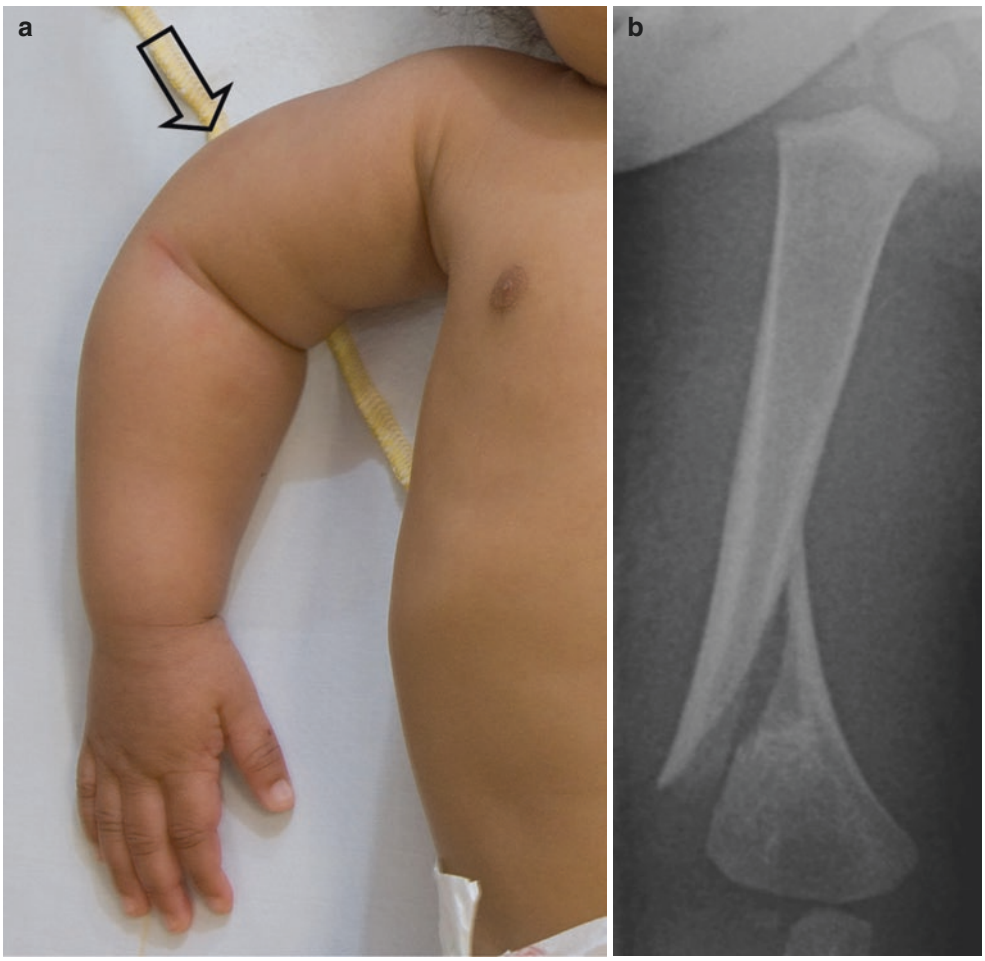


Fig. 12.35 (**a**) Seven-month-old girl suspected of being abused. Slightly abnormal alignment of the upper arm is visible (open arrow). (**b**) Radiograph shows a distal oblique humerus fracture

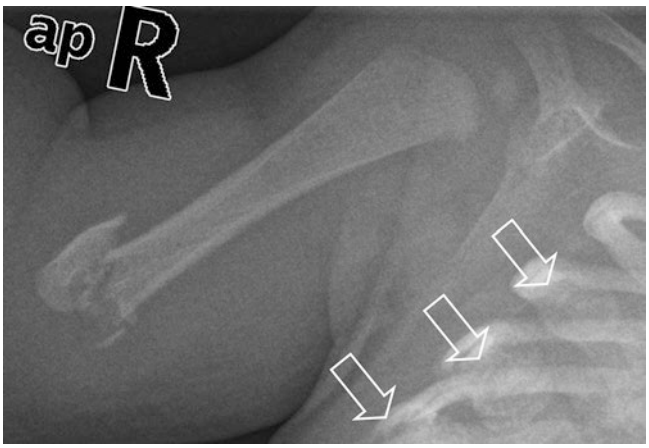


Fig. 12.36 Right distal metaphyseal humerus fracture in a 3-month-old boy. The mid-axillar rib fractures on the right are clearly visible in this view (open arrows)

5. The lack of metabolic or genetic bone diseases.
6. The parent shows lack of involvement or unusual behaviour.

Pandya et al. performed a large retrospective study, in children under the age of 4 years, in an urban level I paediatric trauma centre [130]. In the period 1998–2007, a total of 1485 children, 500 non-accidental (377 <18 months), and 985 accidental (425 <18 months) cases, were included. In the non-accidental group there were 43 (8.6%) humerus fractures and in the accidental group 102 (10.3%, p=0.28). In the under 18 months group this was, respectively, 37 (9.8%) versus 19 (4.5%, p<0.001). Based on their findings the authors concluded that in the under 18 months group '*the odds of a humerus fracture (2.3 times) were found to be significantly higher in the child abuse group than in the control group*'. For the whole study population the odds ratio for abuse was 0.8 (95% CI: 0.6–1.2).

In another study from the same group the authors compared 36 children (representing 39 humerus fractures) under the age of 4 years in whom the cause of the fracture was recorded as abuse with 95 children with an accidental humerus fracture [131]. Based on univariate logistic regression the authors showed that the odds of being a victim of non-accidental injury in children with an age below 18 months was 31.54 times greater, in children with any additional fractures or injuries to another body system it was 65.1 times greater, and in children with physical and/or radiographic evidence of prior injuries it was 131.60 times greater.

Fig. 12.37 Graphic representation of the mechanism as described by Hymel and Jenny

If non-accidental trauma cannot be ruled out in a child younger than 2 years with a humerus fracture a skeletal survey should be performed according to the guideline of the Royal College of Radiology and the Society and College of Radiographers (See Chap. 3) [29].

In older children shaft fractures usually occur in accidental circumstances, due to either indirect trauma, like a fall on an outstretched hand, or direct trauma, usually with a high transfer of energy, e.g. a direct blow to the upper arm, e.g. in traffic accidents, falls or sporting activities [116]. Accidental shaft fractures occur more frequently in children that have been victims of a serious accident [132]. In contact sports (martial arts), there is also a possibility of direct trauma, due to a direct blow (e.g. a karate blow) or indirect trauma, due to falling in a judo throw. Other circumstances are skateboarding, mountain biking, downhill skiing, and trampoline jumping [133, 134].

After Birth: The 'Hymel manoeuvre'

A rare trauma mechanism that can lead to fractures in the humerus shaft in non-mobile infants is the so-called 'Hymel manoeuvre' (Figs. 12.37, 12.38, and 12.39) [135].

Hymel and Jenny presented two cases, one of which was videotaped, in which a parent unintentionally fractured the humerus of their infant by turning the infant from a prone to a supine position. In the second case, the father gave a similar clinical history and subsequent evaluation ruled out non-accidental injury.

In 2014, Somers et al. published a paper describing 7 infants (aged 4–7 months) who were presented with only a humerus shaft fracture [136]. In none of the cases, a videotape was available as a source of evidence, but the clinical histories were independently obtained as part of court hearings. In three cases the parents stated that they witnessed that their child tried to roll from prone to supine, where he/she was initially obstructed from doing so by the dependent arm. After several attempts, the infant gained sufficient momentum to roll over and as a result the dependent arm got overloaded and broke. In the other 4 cases, the infant was placed in a prone position and found in a supine position. The

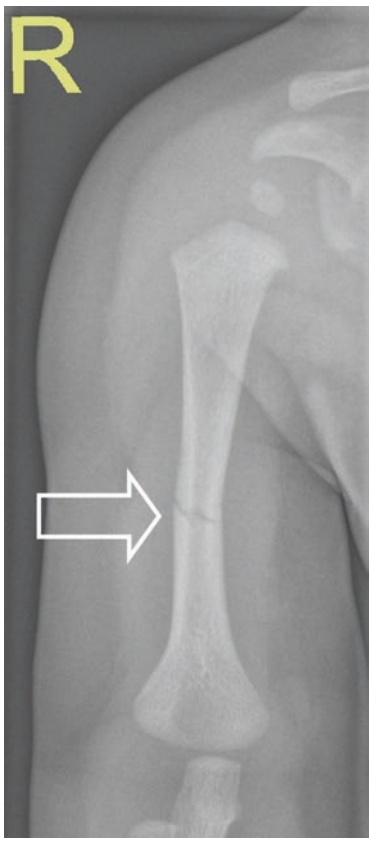


Fig. 12.38 Two-month-old infant, father turned the infant from prone to supine position holding the right arm. During this manoeuvre he heard a 'crack' and the infant started to cry. Radiography showed an oblique fracture of the left humerus. A skeletal survey, CT of the head, and a follow-up survey all were negative. The reported history is in keeping with the publication by Hymel and Jenny [135]

authors agree that, given the lack of an actual video of the event, they cannot state with certainty that this is a valid trauma mechanism. However, given the Hymel video, one should at least consider this as a potential trauma mechanism which of course can only be in the differential diagnosis if all other evaluations and examinations are negative.

In 2020, Altai et al. casted doubt on this proposed mechanism by performing a CT-based finite element study [137].

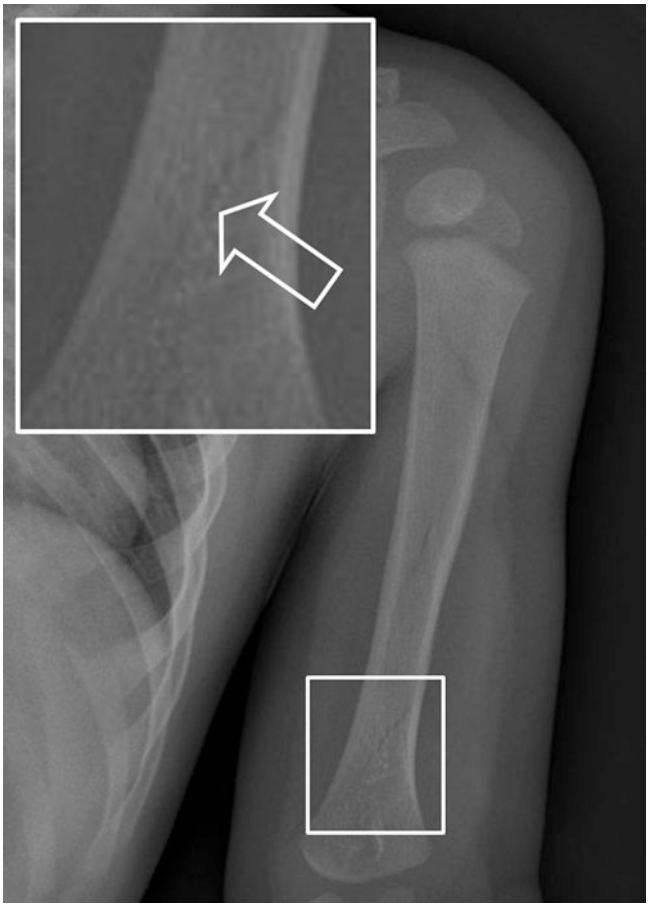


Fig. 12.39 Three-month-old infant who was positioned in a prone position on a duvet. He was found on his back, crying and upon inspection did not use his arm. Radiography showed a fracture of the right humerus (arrow). A skeletal survey, CT of the head, and a follow-up survey all were negative. After extensive evaluation it was concluded that the reported history was in keeping with the publication by Somers et al. [136]

According to their data, the highest predicted strain is around 20% of the predicted elastic limit of humerus during an infant rolling over.

After Birth: Underlying Medical Conditions

Shaft fractures may also occur as a complication of underlying medical conditions, e.g. unicameral benign bone cysts or other benign lesions (pathological fracture) [79, 116]. Pathological fractures may also occur in disorders with increased bone fragility, like osteogenesis imperfecta, fibrous dysplasia, scurvy, and osteopetrosis [134]. Von Heideken et al. found among infants with birth-related humerus shaft fractures, that 1% had a bone fragility diagnosis (n = 2; osteogenesis imperfecta and rickets/vitamin D deficiency). Among children with a later humeral shaft fracture 6% had a bone fragility disorder (n = 8; 7× osteogenesis imperfecta and 1× rickets/vitamin D deficiency).

One should consider a pathological shaft fracture if the fracture occurs after a mild trauma. Pathological fractures occur most commonly in children aged between 3 and 12 years [104].

12.4.4 Fractures of the Distal Humerus

12.4.4.1 General Aspects of Fractures of the Distal Humerus

Fractures of the distal humerus account for over 10% of all paediatric fractures [138]. Several fracture patterns of the distal humerus can be found, which include [138, 139]:

- Supracondylar fractures
- Lateral condyle fractures
- Medial condyle fractures
- Medial epicondyle fractures
- Transphyseal fractures
- Capitellum fractures

When evaluating the distal humerus, the ossification centres of the epiphysis should be taken into account. These ossify according to a set order (Fig. 12.40 and Table 12.5) [140]. Although CRITOE (see Fig. 12.40 and Table 12.5) is generally applicable variation does occur, in a study by Goodwin et al. in 212 of 221 children (96%) the order was according to the general rule [141]. In seven cases, the inter-

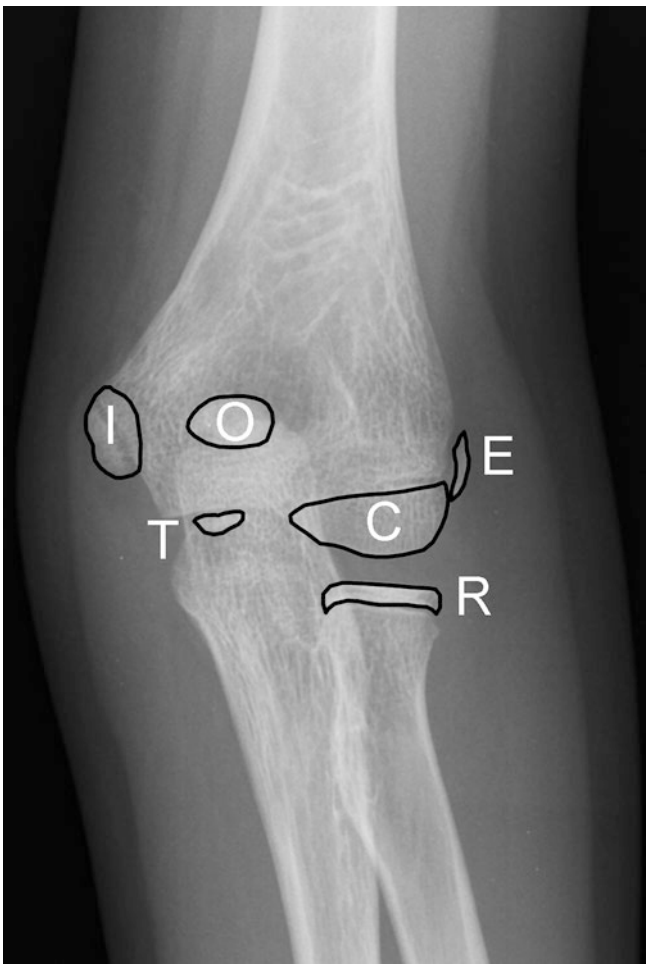


Fig. 12.40 Ossification of the elbow follows a set sequence that is described in the acronym CRITOE (see Table 12.5)

Table 12.5 Ossification sequence of the elbow [140, 466]

	Age of appearance (year, mean (5th—90th percentile))	
Structure	Girls	Boys
Capitellum[a]	1–11 months	2–26 months
Radius head	4.2 (1.6–6.8)	5.9 (3.3–8.5)
Internal epicondyle	4.2 (1.3–7.1)	6.8 (3.9–9.7)
Trochlea	8.4 (5.7–11.1)	9.7 (7.0–12.5)
Olecranon	8.3 (6.0–10.6	9.9 (7.6–12.2)
External epicondyle	9.4 (7.3–11.6)	11.2 (8.0–14.4)

[a] Based on UpToDate. Others based on Patel et al.

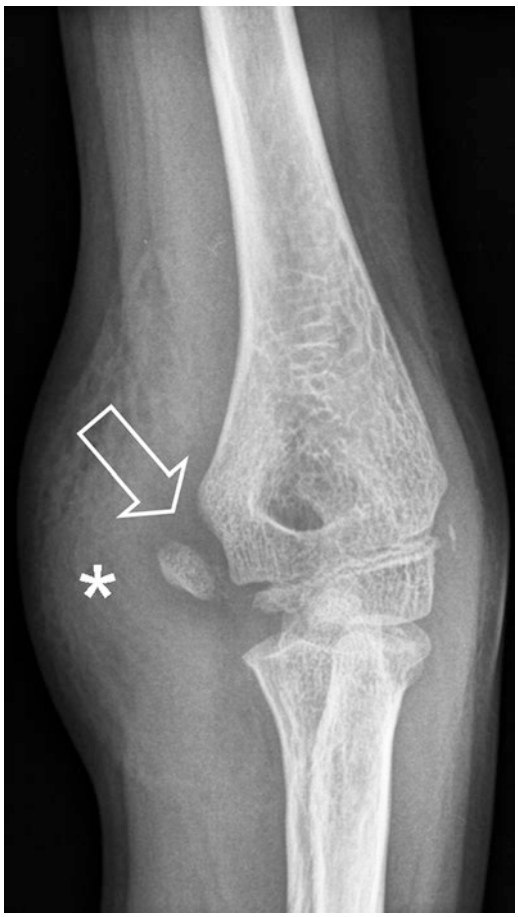


Fig. 12.41 Avulsion fracture of the medial epicondyle of the humerus (open arrow) in a 9-year-old girl who had fallen from a skateboard. There is considerable soft-tissue swelling present (asterisk)

nal epicondyle was visible before the radial head and in two cases the olecranon was visible before the trochlear centre.

Avulsion fractures of growth centres have been found after accidental trauma (Fig. 12.41).

With respect to metaphyseal corner fractures of the distal humerus the reader is referred to Sect. 12.3.2.

12.4.4.2 Supracondylar Fractures: Epidemiology, Cause, and Manner

Supracondylar fractures are common fractures in paediatric patients. In some studies, the incidence is estimated at

Fig. 12.42 Ten-year-old girl with a supracondylar humerus fracture after a fall

10–16% of all paediatric fractures [138, 142]. Supracondylar fractures are the most common fractures of the distal humerus/elbow fractures and account for 60–75% of all distal humerus fractures/elbow fractures [138, 139]. This type of fracture is much more common in skeletally immature children aged between 3 and 7 years than in adults, with a peak age between 5 and 7 years [142–144]. Ninety percent of all subcondylar fractures are found in children under the age of 10 years and are more common in boys than in girls [145].

Over 95% of supracondylar fractures are of the extension type due to a fall on the outstretched hand, e.g. a fall from a moderate height, like from a bed or a monkey bar [145]. In the extension type the distal part is displaced posteriorly.

Flexion-type fractures are uncommon and account for less than 5% of all supracondylar fractures. Flexion-type fractures are much more common in older adult patients and are caused by direct impact on the flexed elbow, e.g. in a fall directly on the flexed elbow [139, 146]. In this type, the distal part of the fractures is displaced posteriorly.

In children over the age of 18 months, supracondylar fractures and dislocations most commonly occur in accidental circumstances and are only rarely reported due to non-accidental circumstances (Fig. 12.42) [123, 130, 131, 147–149]. In younger children and certainly in non-mobile/non-ambulatory children non-accidental circumstances should always be excluded [123, 149].

Thomas et al. evaluated the medical records and radiographs of 39 children under the age of 3 years with either humeral or femoral fractures [125]. Fourteen of them had humeral fractures. In 11 children the circumstances were determined to be non-accidental. In 3 children, all 3 with supracondylar fractures the circumstances were determined to be accidental (fall from a tricycle, a rocking horse, or a fall downstairs). Humerus fractures other than supracondylar fractures were all found to be due to abuse. The clinical history usually shows that the (mobile) child fell on the extended arm (hand in dorsiflexion and elbow in hyperextension) or directly on the bent elbow [125, 147].

Strait et al. evaluated retrospectively the findings in 124 children under the age of 3 years with humerus fractures, concerning the circumstances of the occurrence (inflicted, undetermined, not-inflicted) [123]. In 9 out of 25 children under the age of 15 months (36%) the fractures were determined to be inflicted and in only 1 of 99 children over the age of 15 months (73%). Non-accidental circumstances were excluded in 91 of 124 children (73%). In 23 of 124 children the circumstances were undetermined (18.5%). Ten children under the age of 15 months had supracondylar fractures. In 2 of these 10 children the circumstances were determined to be non-accidental. In these children the clinical history and the moment that medical help was sought were conclusive [150]. Twelve children had spiral/oblique fractures. In 7 of them (58%) the circumstances were determined to be non-accidental. According to Strait et al. non-accidental circumstances should be considered in children under the age of 15 months with humeral fractures, including children with supracondylar fractures.

Rosado et al. evaluated the findings in 97 children under the age of 18 months with a total of 100 humerus fractures [149]. The most common fracture location was the distal humerus (65%) and the most common fracture type was supracondylar (48%). Child Protection evaluated 44 children (45%) and determined that in 24 of these children, with a total of 25 humerus fractures, the fractures were sustained in non-accidental circumstances (25% of the total study population). The most common fracture location, in children determined to have been sustained in non-accidental circumstances, was the distal humerus (50%) and the most common types were transverse and oblique (25% each). However, transverse and oblique fractures were also seen in patients whose injuries were determined to have been due to accidental circumstances. Children with non-accidental fractures were younger and non-ambulatory than children with accidental fractures. Children with non-accidental fractures also had more often additional injuries, suspected to be inflicted.

Rinaldi and Hennrikus reviewed the findings in 75 children with displaced supracondylar elbow fractures: 42 boys, 33 girls with an average age of 6 years (range: 1 year 4 months to 12 years 4 months; 70 children older than 3 years) [151]. Forty-seven percent of the fractures occurred at home. Only one child (the youngest child in the evaluated population), aged 1 year and 4 months was reported because of suspected non-accidental circumstances. The child supposedly was injured from a fall at home. The evaluation did not confirm the suspicion of non-accidental circumstances. Rinaldi and Hennrikus concluded that paediatric supracondylar elbow fractures in their study only occurred due to accidental falls while children were at play. The mechanisms involved were fall on the outstretched hand and hyperextension of the elbow.

12.4.4.3 Fractures of the Lateral Condyle: Epidemiology, Cause, and Manner

Fractures of the lateral condyle are the second most common fractures of the distal humerus and account for up to 20% of all paediatric elbow fractures [138, 139, 152]. This fracture is most commonly found in children aged between 4 and 10 years with a peak at 6 years of age, these fractures are most commonly a Salter–Harris-type IV fracture [139, 152].

This fracture occurs after a fall on an outstretched hand (FOOSH) (Fig. 12.43). Two theories exist concerning the cause of the fracture (mechanism) [152–154]:

Fig. 12.43 Fourteen-month-old child who was seen in the emergency department after a fall onto an outstretched hand (FOOSH). AP radiograph of the left elbow shows an avulsion fracture of the lateral condyle (arrow)

- A push-off mechanism: This theory postulates that the fracture is the result of a force directed upward and outward along the radius. If the radial head impacts the distal humerus, this may cause the fracture of the lateral condyle. This typically occurs due to a fall on an outstretched hand (axial loading).
- A pull-off mechanism: This theory postulates that the pull of the tendons of the extensor carpi radialis longus, extensor carpi radialis brevis, and brachioradialis, that are attached to the lateral condyle, may result in an avulsion fracture of the lateral humeral condyle.

According to Tewjani et al., the most likely cause is a combination of push- and pull-off mechanisms [153]. Reports on fractures of the lateral condyle only describe the occurrence due to accidental circumstances. No case reports (series or single) were found concerning the occurrence of fractures of the lateral condyle due to non-accidental circumstances, although Kleinman states that fractures of the lateral condyle may occur due to non-accidental circumstances and Offiah and Hall are of the opinion that a fracture at this location has a medium specificity concerning non-accidental circumstances [155].

12.4.4.4 Fractures of the Medial Condyle: Epidemiology, Cause, and Manner

According to Walsh '*medial condyle fractures involve a fracture line that extends through and separates the medial metaphysis and epicondyle from the rest of the humerus; by definition, the fracture line must involve the trochlear articular surface. Medial condyle fractures must be distinguished from medial epicondyle fractures that involve the medial column but are extraarticular'* [156]. Isolated fractures of the medial condyle are very rare and probably account for less than 1–2% of all distal humerus fractures [156, 157]. This type of fracture is most common in children, aged 7–14 years [156]. Concerning the cause of a fracture of the medial condyle (mechanism) 3 theories exist [156, 157]:

- A fall on the palm of an outstretched arm, with the elbow forced into valgus (axial loading).
- A fall on the point of the elbow (apex of the flexed elbow), with the olecranon driving the medial condyle proximally and medially (direct impact).
- An avulsion fracture, due to violent contraction of the flexor and pronator muscles that attach to the medial epicondyle, such as that which occurs in arm wrestling.

Because this type of fracture is so rare, hardly any (if any) epidemiological data concerning the circumstances of the occurrence in paediatric patients are known. Kleinman reports an unusual SH-type III medial condylar fracture of the distal humerus in a 23-month-old girl, which was determined to be inflicted [158].

12.4.4.5 Fractures of the Medial Epicondyle: Epidemiology, Cause, and Manner

As stated in Sect. 12.4.4.4 fractures of the medial epicondyle should be distinguished from fracture of the medial condyle. Fractures of the medial epicondyle are much more common than fractures of the medial condyle and account for 10–20% of all elbow fractures in children and adolescents [139]. In up to 60% of these fractures are associated with elbow dislocation [159]. Bauer et al. described the simultaneous occurrence of bilateral elbow dislocation with bilateral medial epicondyle fractures in a 13-year-old female gymnast (trampoline gymnastics) with hyperlaxity [160].

Most occur in paediatric patients aged between 11 and 14 years (peak ages 11 and 12 years) [156]. According to Smithuis 80% of these fractures occur in boys with a peak age in early adolescence [139].

Walsh mentions three theories concerning the cause of fractures of the medial epicondyle:

- A direct blow on the posterior medial aspect of the epicondyle.
- An avulsion mechanism, due to activity of the flexor muscles of the forearm. This may occur when a child falls on the extended arm and hyperextends wrist and fingers, placing more stress on the forearm flexors. This avulsion mechanism may also in arm-wrestling and throwing a baseball ('little league elbow').
- An avulsion mechanism in which, due to dislocation of the elbow, the ulnar collateral ligament (UCL) provides an avulsion force that causes the medial epicondyle to fail.

Irrespective of the theory in a fracture of the medial epicondyle the apophyseal fragment is partially or completely separated from the rest of the humerus.

Most of the fractures of the medial epicondyle will occur in older children and adolescents, due to accidental circumstances or sporting activities [161]. No reports were found concerning the occurrence due to non-accidental circumstances. Sperry and Pfalzgraf described the occurrence of healing symmetrical clavicular fractures and a healing left medial humeral epicondyle fracture in a 9-month-old child [162]. The child was found unresponsive in his crib, five hours after his last feeding. During the autopsy no physical signs, suggesting non-accidental trauma, were found. Only a few visceral pleural and epicardial petechiae were found, which were determined to be consistent with the sudden infant death syndrome (SIDS). The healing fractures were found on post-mortem total body radiographs. The parents had no explanation for these injuries and denied causing any harm to the child. The case was reported to the police and the district attorney's office as suspected non-accidental trauma. During the investigation the parents stated that the child had undergone *'chiropractic'* manipulations by an unlicensed

therapist, between three and four weeks prior to death, to correct supposed *'shoulder dislocations'*. This time interval correlated with the histologic age of the injuries, and the history explained their unusual bilateral location and appearance.

12.4.4.6 Transphyseal Fractures: Epidemiology, Cause, and Manner

Transphyseal fractures (a.k.a. transcondylar fractures or distal humerus physeal separation) are fractures through the distal humeral physis, in which the entire distal humeral epiphysis is separated from the metaphysis. According to Shore the physis is biomechanically the weakest location in distal humerus in skeletally immature children [163]. This type of fracture most commonly occurs in children under the age of 3 years [164, 165].

Transphyseal fractures can be classified according to the Salter–Harris classification (see Sect. 12.3.2) [165]:

- SH-type I (pure physeal injury) is seen most commonly in children under the age of 3 years.
- SH-type II (metaphyseal fragment attached to distal fragment) is most common in children over the age of 3 years.
- SH-types III and IV (intra-articular extension) occur, but are rare.

Transphyseal fractures have been described to occur during and after birth [64, 165, 166]:

- In vaginal delivery fractures may happen due to the force of labour or due to obstetric manoeuvres. Shoulder dystocia and traumatic delivery are known risk factors. It may also occur during a caesarean section, due to excessive traction. Usually, birth-related transphyseal fractures are recognized before the age of 2 weeks.
- Transphyseal fractures have also been described to occur after birth, either in accidental or in non-accidental circumstances.

Gigante et al. described 5 cases of birth-related distal humeral growth plate fractures [69]. During a 30-month follow-up 4 cases showed an excellent clinical and radiological outcome with conservative treatment, in one case surgical intervention with stabilization using a K-wire was needed. On conventional radiographs the diagnosis may, due to the non-ossified epiphysis, be difficult (Fig. 12.44a, c). In several case reports the use of ultrasound in the diagnosis is propagated as a problem-solving modality (Fig. 12.44b) [68, 167, 168]. Although birth-related humerus fractures are usually diagnosed during the first day of life delayed presentation of several days is not uncommon and thus a delayed presentation should not rule out birth trauma [169].

Supakil et al. reviewed the findings in 16 children with transphyseal fractures (distal humeral epiphyseal separation) under the age of 36 months (mean age 8.6 months; 10 boys, 6 girls) [166]. In 10 (63%) children, one or more additional humeral fractures were found (bucket-handle fractures in 5 and condylar avulsion fractures in 6 children). Six children (38%) were under the age of 2 weeks. In these children, the fracture was determined to be secondary to birth trauma. In 4 children the fracture occurred in a vaginal delivery (breech delivery with footling presentation 1×, nuchal cord wrapped around right upper extremity 1×, shoulder dystocia 1× after uncomplicated). In two children the fractures occurred in an uncomplicated caesarean section. In 4 of the 10 children above the age of 14 days (3 boys, aged 3.3 months, 8.1 months, 2.3 years; 1 girl; aged 3.3 months), the fracture was determined to have occurred in non-accidental circumstances. In the remaining 6 children the fracture occurred due to accidental circumstances (sibling jumping on child

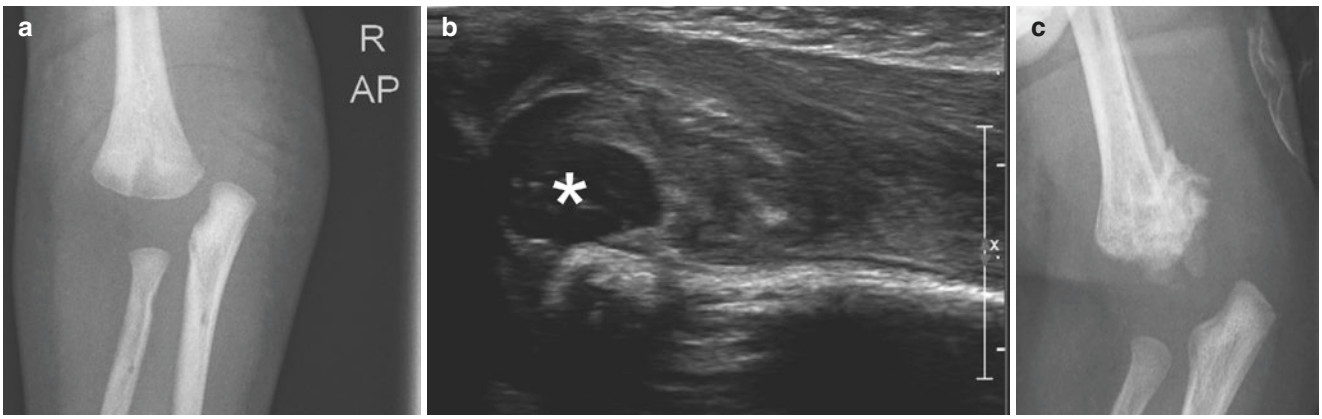


Fig. 12.44 Neonate born after an uneventful vaginal delivery. Two days after birth the nurse noted a lack of motion of the left arm. (**a**) Radiography showed an abnormal relation in the elbow, which was suspect for a humerus epiphysiolysis. (**b**) Ultrasonography clearly shows the displaced epiphysis (asterisk). (**c**) Radiography on day 22 of life shows callus formation along the distal humerus metaphysis. Long-term follow-up showed a normal development of the elbow joint

twice, falls downstairs twice, falls from chair twice). In the same publication the occurrence of a transphyseal fracture in a 1-month-old neonate was reported. Because a relevant obstetrical history of a history of a preceding trauma was lacking, it was suspected that the fracture occurred due to non-accidental circumstances.

The supposed mechanism in non-accidental trauma is a rotational force (twisting) and in accidental trauma a fall on outstretched hand with an extended elbow [165]. According to Shore in infants extension forces may more likely cause a transphyseal fracture, while in older children comparable forces may more likely cause a supracondylar fracture [163].

12.4.4.7 Capitellum Fracture: Epidemiology, Cause, and Manner

Capitellum fractures are very rare in children and adolescents, accounting for less than 1% of all elbow fractures. Murthy et al. evaluated 32 paediatric patients with capitellum fractures (22 boys, 10 girls) [170]. Although Ertl states that capitellum fractures do not occur in children under the age of 10 years, the mean age in the series of Murthy et al. was 11.8 years, with an age range of 6–16 years [170, 171]. Fractures at this location result from a low-energy fall on outstretched hand (axial compression with the elbow in a semi-flexed position) or a fall or blow directly onto the elbow (direct impact) [171, 172]. No data are found concerning the occurrence of capitellum fractures due to non-accidental circumstances.

12.5 Radius and Ulna

12.5.1 General Aspects of Fractures of the Radius and Ulna

In children, fractures of the forearm are probably the most common fractures of the long bones, with an estimation of up to 40% to 50% of all paediatric fractures [81, 147, 173–175]. This is irrespective of type (complete, incomplete, or plastic deformation) or location (proximal, middle, or distal third) of the fractures. Forearm fractures are more common in boys than in girls [176].

Distal radius (and ulna) fractures are the most common forearm fractures in children under the age of 16 years and account for around 75% of all forearm fractures and 20–25% of all paediatric fractures (Fig. 12.45) [40, 81, 82, 174, 177–179]. Distal forearm fractures may be incomplete fractures (buckle/torus fractures and greenstick fractures), complete fractures (metaphyseal corner fractures or Salter-Harris fractures), or bowing fractures [61].

Fractures of the distal radial and ulnar growth plate are often Salter–Harris I or II fractures (Fig. 12.46a, b). Although distal forearm fractures (excluding MCF) may occur at any age, they are predominantly seen during the growth spurt in puberty with peak ages in girls between 10 and 12 years and in boys between 12 and 14 years [180, 181]. Distal forearm fractures are 2–3 times more common in boys than in girls [181].

Fig. 12.45 Distal fracture of the radius and ulna in a 6-year-old girl after a fall

Fractures of the forearm shaft (transverse, oblique, or spiral) are the third most common fracture in children, after distal radius and supracondylar humerus fractures, and may account for around 15% of all paediatric fractures [177]. In other studies, lower percentages are mentioned, namely 3–6% of all paediatric fractures and around 20% of forearm fractures [82, 176]. Midshaft fractures are more frequently seen in young children [182].

Isolated fractures of the ulnar shaft are rare in children. A Parry or ‘nightstick’ fracture is a specific type of isolated fracture of the ulnar shaft which fulfils the following criteria: absence of radial involvement, transverse fracture line, distal from midshaft, and minimal displacement [183].

Olecranon fractures are uncommon in childhood and account for around 4% of paediatric elbow fractures (Fig. 12.47a, b). Olecranon fractures have been found in children between 5 and 17 years of age with a boy to girl ratio of 3.5:1 [176, 184, 185].

Radial head and neck fractures account for around 1% of all paediatric fractures and for 5–10% of all elbow fractures. The median age of children with these fractures is 9–10 years of age and the boys to girls ratio is 1:1 [186].

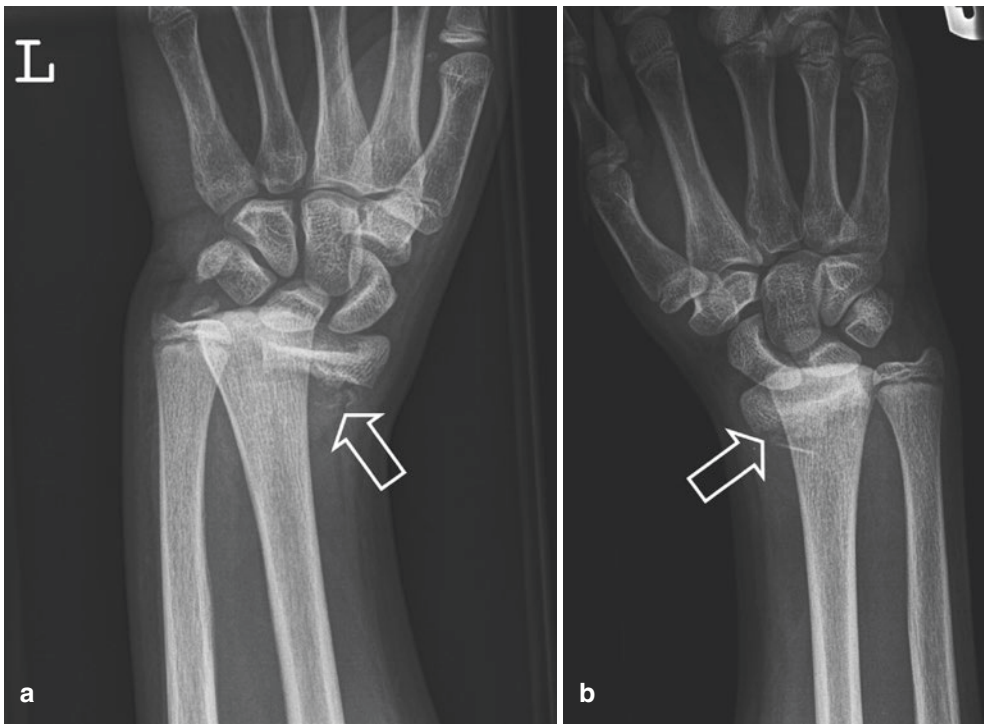


Fig. 12.46 Bilateral Salter–Harris type II fracture (**a** and **b**) of the distal radius in a 13-year-old boy after a fall while skating

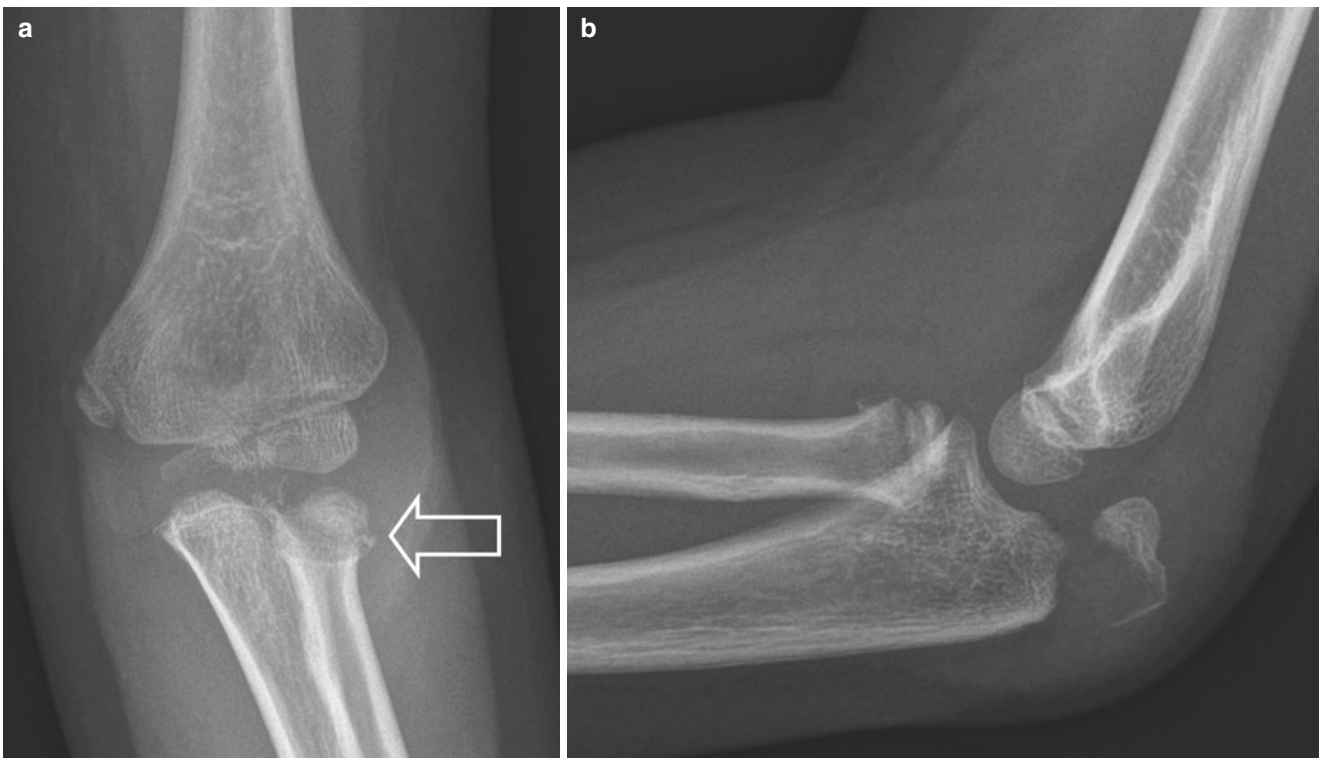


Fig. 12.47 Five-year-old child who presented with pain in the elbow and limited range of motion after a fall onto an outstretched hand (FOOSH). (**a**) AP radiograph shows a fracture of the proximal radius head (arrow). (**b**) Lateral radiograph shows a fracture of the olecranon

Fig. 12.48 Monteggia fracture, with the characteristic midshaft fracture of the ulna and dislocation of the head of the radius (open arrow), in a 2-year-old child after a fall from the couch (Courtesy of J. Davis, fellow Chadwick Center for Children and Families, San Diego, CA, USA)

Galeazzi fractures are distal radius fractures at the distal metaphyseal–diaphyseal junction with concomitant disruption of the distal radio-ulnar joint. This type is rare, compared to other distal forearm fractures, ranging from 0.3% to 3% of the distal radius fractures, with a peak incidence between 9 and 13 years [61, 187, 188].

A Monteggia's fracture is a proximal ulna fracture or plastic deformation of the ulna with an associated radial head dislocation (Fig. 12.48). Just like the Galeazzi fracture, this type is rare and may account for 0.4% of all forearm fractures. The peak incidence is between 4 and 10 years [187, 189–191]. Combined Monteggia and Galeazzi fractures have been described to occur [192, 193].

Bowing fractures due to plastic deformation have been described to occur in all long bones (Sect. 12.2.1). The radius and ulna, followed by the fibula, are the most commonly affected bones [194].

The clinical signs of a forearm fracture, and in particular of a shaft fracture, are pain, pain at pressure, swelling, crunching (crepitus), restricted movement in wrist and hand, and possibly an aberrant alignment or the arm. This is seen mainly in complete fractures. In 'bowing' fractures and minimal greenstick fractures an aberrant position is seen (may be minimal), there is sensitivity when touched and restricted movement of the lower arm. Pain and swelling may be minimal, while crepitus may not be present at all [195].

12.5.2 Cause of Fractures of the Radius and Ulna

In children fractures of the forearm most commonly are caused by an indirect trauma, usually in a fall on the outstretched hand (FOOSH) (Table 12.6). This may happen

Table 12.6 Cause of fractures of the forearm [61, 176, 181, 186, 188, 190, 467]

Forearm	Usually, fall on outstretched hand (FOOSH)—fall from a height, sporting event, or playground equipment injury
Distal radius (and ulna) fractures	Usually fall on an outstretched hand, extended at wrist, often during sports or play
Radius and ulnar shaft fractures	• Direct trauma (direct blow to the forearm) • Indirect trauma – Motor vehicle accidents – Falls from height—axial loading to forearm through hand
Isolated ulnar shaft fracture—Parry fracture—'nightstick' fracture	• Direct trauma (direct blow to the forearm—a nightstick is a police baton) • Indirect trauma (fall)
Olecranon fracture	Indirect trauma (most common)—fall onto outstretched arm with: • Elbow in flexion (most common): triceps and brachialis tensioning causes a transverse olecranon fracture • Elbow in extension: varus/valgus bending forces through the olecranon causes longitudinal fracture lines. – Varus may lead to associated radial head dislocation – Valgus may lead to an associated fracture of the radial neck Direct trauma (least common)—direct blow to the elbow: • Shear force creates anterior tension failure with anterior displacement of the distal fracture and intact posterior periosteum
Radial head and neck fractures	• Usually associated with an extension and valgus loading injury of the elbow • Elbow dislocation
Galeazzi fracture	Axial loading in combination with extremes of forearm rotation (pronation or supination): • Pronation produces an apex dorsal radial fracture with the distal ulna displaced dorsally • Supination produces an apex volar radial fracture with the distal ulna displaced volarly
Monteggia fracture	Fall on outstretched hand

when a child tries to break his/her fall by outstretching the arm and hand. As soon as the child lands on the extended arm, the main deforming force is transferred to the radius. Consequently, a fracture in the forearm may occur first in the radius, and then in the ulna. Often these are incomplete fractures, either greenstick or torus fractures. Fractures of the forearm are less commonly caused by a direct blow to the forearm, which is perpendicular to the forearm.

12.5.3 Manner of Fractures of the Radius and Ulna

Fractures of the forearm (radius and/or ulna), including MCF, have been described to occur before, during, or after birth. If sustained after birth, forearm fractures can occur due to accidental and non-accidental circumstances.

12.5.3.1 Before Birth

Fractures of the forearm, occurring in utero, have only very rarely been reported in the medical literature.

Onimus et al. reported the case of a patient (second pregnancy, first birth) who had been involved in a motor vehicle accident (frontal impact) when she was 7 months pregnant [196]. She was driving and wearing her seatbelt. Because of recurring metrorrhagia and uterine contractions in the period after the accident, a caesarean section was done. The neonate showed angulation of the left forearm. On X-ray a fracture line was seen with a callous already formed.

In 2 neonates the forearm fractures were related to the presence of an amniotic constriction band of the forearm:

- Ho et al. described increased swelling to the right forearm distal to a congenital fibrous band in a 1-day-old neonate who was born at 28 3/7 weeks of gestational age [197]. The band was associated with underlying mid-shaft fractures of the right arm with pseudoarthrosis.
- Angelis et al. described a 2-day-old preterm male, who was born at 31 weeks by caesarean section [198]. His left hand was swollen due to a constriction band with severe swelling and vascular compromise of the hand. Radiography showed a displaced fracture of the radius and ulna at the level of the band on the distal third of the forearm with pseudoarthrosis.

12.5.3.2 During Birth

In Sect. 2.6.2 and Table 2.9 an overview is given of fractures, that were sustained during birth. Only 1 dislocation of the elbow was reported by Bhat et al. [121]. No fractures of the forearm due to birth trauma are mentioned in the epidemiological studies, shown in Sect. 2.6.2. Only one case report concerning a birth-related radius fracture was found. Thompson et al. described a spiral fracture of the radius in a neonate after a complicated delivery due to shoulder dystocia. The child had a birth weight of 4,610 g [199]. The physical examination after the delivery showed bilateral cephalhaematomas, bruising of the face and forehead, a markedly oedematous left upper arm, and bruising of the right forearm. On X-ray, a spiral fracture of the right radius and a fracture of the left midhumeral shaft were seen.

12.5.3.3 After Birth: Accidental Circumstances

In mobile children, fractures of the radius and ulna are usually the result of accidental trauma, most commonly due to a fall on the outstretched hand (FOOSH) [81, 82]. Ryan et al. reviewed the findings in 929 paediatric patients, aged 0–17 years (mean age 8.4 years; male to female ratio 2:1) with isolated forearm fractures (2003–2006) [200]. They classified the circumstances as major trauma (motor vehicle collision; pedestrian or bicyclist struck by a moving vehicle; fall greater than patient height), minor trauma (Fall less than patient height; fall equal to patient height; other mechanisms not meeting criteria for major trauma severity classification), and unknown (unable to determine severity of trauma from documentation). Most fractures were determined to have occurred due to a minor trauma (58%), followed by unknown trauma (36.2%) and major trauma (5.8%). In the group of children aged 0–4 years (n = 150; 16%) falls from furniture were most common, in the 5–9 years group (n = 410; 44%) falls from monkey bars and in the 10–17 years group (n = 369; 40%) injuries due to organized sporting activities. Most fractures were fall-related (83%), while only 10% of the forearm fractures were caused by a direct blow to the forearm.

Other circumstances of forearm fractures, described in the medical literature are falls from high chairs (Sect. 13.3.5.5) [201] from shopping carts (Sect. 13.3.5.10) [202], and with baby-walkers (Sect. 13.3.5.8) [203].

12.5.3.4 After Birth: Non-accidental Circumstances

Fractures of the forearm occur frequently in non-accidental trauma. In several studies describing a series of children with non-accidental fractures, the radius and/or ulna belong to the most commonly affected bones:

- Worlock et al. compared the findings in 35 children (28 children under the age of 18 months; 7 children between 19 and 60 months; 0 children above the age of 60 months) with non-accidental fractures to the findings in 826 children (19 children under the age of 18 months; 97 children between 19 and 60 months; 710 children above the age of 60 months) with accidental fractures [40]. Worlock et al. found non-accidental forearm fractures only in children under the age of 5 years. They found metaphyseal corner

fractures of the forearm only in infants under the age of 18 months. Other non-accidental fractures in this group were 4 fractures of the shaft: 2 greenstick fractures, 1 transverse fracture, and 1 healing fracture (periosteal reaction). In the toddlers (19–60 months) 2 shaft fractures (1 oblique fracture and 1 healing fracture) and one distal fracture (greenstick fracture) were found.

- Leventhal et al. evaluated the findings in 215 children under the age of 3 years with a total of 253 fractures [124]. The fractures were sustained in 24.2% in non-accidental circumstances and in 67.4% in accidental circumstances. In 8.4% the circumstances were not known. Concerning forearm fractures (n = 17) they found that 4 (23%) were due to non-accidental trauma and 12 (71%) to accidental trauma. In one child the circumstances were unknown. According to the authors non-accidental trauma should be suspected in a child under the age of 1 year with a fracture of the radius and/or ulna.
- Loder et al. reviewed the findings in 1794 patients under the age of 20 years with injuries due to non-accidental trauma [204]. They found a total of 1053 fractures, of which 83 were fractures of radius and/or ulna. Of these 51 were found in children under the age of 1 year, 17 between 1 and 2 years, 13 between 3 and 12 years, and 2 between 13 and 20 years.
- Van As et al. evaluated the physical findings in 1,037 children between 1 month and 13 years (median age 16.5 months, average age 44.8 months; male to female ratio 2:1) with injuries due to non-accidental trauma [205]. Of these children 121 had a total of 149 fractures (21 had multiple fractures). 15 children had fractures of the radius and/or ulna.
- Pandya et al. did a large retrospective study, in children under the age of 4 years, in an urban level I paediatric trauma centre [130]. In the period 1998–2007 a total of 1485 children, 500 non-accidental (377 <18 months) and 985 accidental (425 <18 months) cases, were included. In the non-accidental group there were 23 (4.6%) radius and/or ulna fractures and in the accidental group 7 (0.7%) (p<0.001). In the under 18 months group this was, respectively, 19 (5.0%) versus 3 (0.7%). In the over 18 months group this was, respectively, 4 (3.3%) versus 4 (0.7%). Based on their findings the authors concluded that radius and/or ulna fractures were more common in the non-accidental group than in the accidental group, irrespective of the age of the child (p<0.001).
- Ryznar et al. evaluated the findings in 135 children under the age of 18 months with a total of 216 forearm fractures. Most fractures were torus fractures (57%), followed by transverse fractures (26%), irrespective of the circumstances (accidental or non-accidental). Children whose only forearm fracture was a metaphyseal corner fracture were excluded from the study. Forty-seven (35%) children were evaluated by child protection teams. In 11 children (23%) it was concluded that the fractures were sustained in non-accidental circumstances. Children with non-accidental fractures were significantly younger than children with accidental fractures (7 months versus 12 months; $p < 0.0001$). Next to age, additional injuries, and an absent or inconsistent explanation were found more often in the children with non-accidental forearm fractures. The most common causing mechanism in accidental fractures was a fall (82%). Ryznar et al. also concluded that no particular type of forearm fracture was specific for non-accidental trauma.
- Hermans et al analyzed the findings in 36 paediatric patients, between 2 and 16 years (mean age 8.9 years; range between 2.3 and 15.4 years) with isolated fractures of the ulna (a.k.a. 'nightstick' fracture) [183]. Only in 6 patients the fracture was caused by a direct trauma (2× kicked by another kid; 1× kicked by pony; 1× other kid fell on arm; 2× other kid stepped on arm). No association between the occurrence of an isolated ulna fracture and non-accidental trauma could be established by the authors.

Based on the findings in the foregoing literature one can conclude the following concerning fractures of the forearm:

- The younger the child, the more likely a forearm fracture is sustained in non-accidental circumstances.
- Metaphyseal corner fractures of the forearm most commonly occur in infants under the age of 18 months and are highly suggestive of non-accidental circumstances.
- Except for metaphyseal corner fractures, differentiation between accidental and non-accidental circumstances is not possible, based on the type of forearm fracture.
- In children under the age of 5 years radius and/or ulna fractures seem to be more commonly sustained in non-accidental than in accidental circumstances.
- In forearm fractures one should always consider non-accidental trauma in non-mobile children/children who do not (yet) walk.
- One should also consider non-accidental circumstances if the explanation of how the fracture occurred does not match the known trauma mechanism(s) (inconsistent history) or when other physical findings are found, which are suggestive of non-accidental circumstances (concomitant injuries).

12.5.4 'Nursemaid's Elbow'

12.5.4.1 General Aspects of the 'Nursemaid's elbow'

'Nursemaid's elbow', or pulled elbow syndrome, is the popular name for what initially was known as radius head subluxation (RHS), but at the moment as annular ligament

displacement (ALD) [206]. It is one of the most common paediatric joint injuries, which accounts for over 20% of upper extremity orthopaedic injuries in children [207]. The recurrence rate is about 25% [208]. Bilateral occurrence of the nursemaid's elbow has been described but is very rare [209, 210]. A nursemaid's elbow typically occurs in children between 1 and 4 years of age [211]. The youngest child, reported in the medical literature is an infant of 2 months, with several reports of children under the age of 6 months [208, 212, 213]. Nursemaid's elbow is also seen in older children, although seldom in children over the age of 7 years [212]. In older children subluxation is prevented by a thicker and stronger distal attachment of the annular ligament [214].

Rudloe et al. evaluated the findings in 3170 children (median age 2.1 years; 50% between 1.5 and 2.8 years) with a nursemaid's elbow. Girls to boys ratio was 3:2 [215]. In almost 60% of the children the left arm was involved.

Vitello et al. reviewed the findings in 1,228 children under the age of 6 years (mean age 28.6 months; 44% between 18 and 29 months), who visited an emergency department and were diagnosed with a nursemaid's elbow [211]. 137 children visited the ED more than 1 time (up to 7 times in one child) because of a nursemaid's elbow. Girls to boys ratio was 3:2. Most of the included children were over the 75th percentile for weight and more than one quarter were over the 95th percentile in each gender. 60% of the subluxations were found in the left elbow.

The radiological examination, which is usually not required due to the obvious clinical history, generally shows no dislocation. When the radiograph is taken, positioning of the arm by the radiographer will usually reduce the nursemaid's elbow.

12.5.4.2 Cause and Manner of the 'Nursemaid's elbow'

> *'Imagine a parent innocently swinging around a toddler ... a yank on an outstretched arm to keep a preschooler from falling ... a caregiver attempting to move a reluctant child by dragging the child by the hand ... a helping hand to lift a young child up over the curb or a high step. None of these activities is ever intended to hurt a child, yet the result of these specific activities send many children with anxious parents and caregivers to emergency departments and unscheduled pediatrician appointments each year'* [216].

Nursemaid's elbow is caused by traction along the longitudinal axis of the arm or a sudden pull of the extended pronated arm. Initially, it was assumed that due to the traction/pulling the radial head moves out of the weak annular ligament and capitellum, resulting in slipping over and subluxation of the radial head into the supinator muscle and annular ligament [212, 217]. According to Browner, however, it is not a subluxation of the radius head that is responsible for the nursemaid's elbow, but a displacement of the annular ligament (Fig. 12.49)

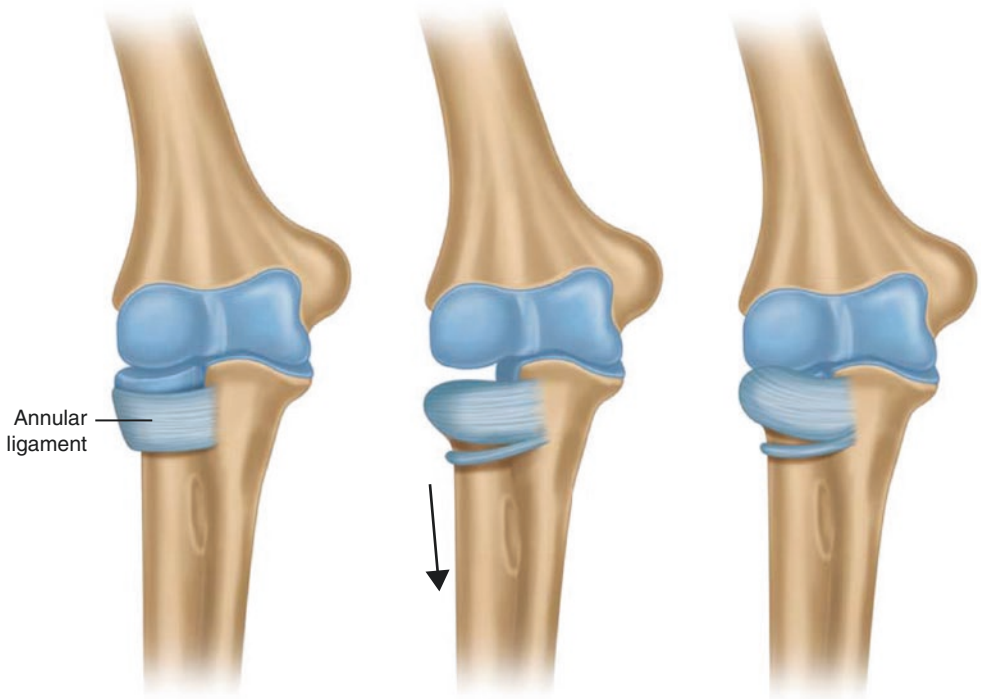


Fig. 12.49 Subluxation of the annular ligament due to a sudden longitudinal traction

[206]. The traction/pulling causes the annular ligament to slip over the head of the radius and come to rest in the radial–humeral joint between the radius and capitellum, where it becomes entrapped.

It is not clear how much force is needed to cause a nursemaid's elbow. According to the Pediatric Society of North America (POSNA) only very little force is needed to pull the bones of the elbow partially out of place which explains why, according to POSNA, the nursemaid's elbow is so common. Others, however, are of the opinion that it needs pulling the child's arm with great force to cause a nursemaid's elbow [212]. Concerning the causing mechanisms, it does not matter whether a nursemaid's elbow is the result of a subluxation of the radius head or of a displacement of the annular ligament.

Rudloe et al. analyzed the causing mechanisms in 3,170 children (median age 2.1 years, with 50% of the children between 1.5 and 2.8 years) [215]. They found that the nursemaid's elbow in 63% was due to traction and in 17% due to non-traction. In 19% the causing mechanism was unknown or undocumented. In the traction group (n = 2011) several traction/pulling mechanisms were identified:

- Lifting the child by one arm or both arms (28.3%)
- 'Wrestling' or 'roughhousing' (12.3%)
- Swinging the child by one arm or both arms (9.2%)
- Placing the child into and out of a seat (4.3%)

Male caregivers were more likely to be involved when a child was swung by the arms, lifted or 'wrestled'/'roughhousing' with. Mechanisms more common for female caregivers included the child pulling away from the parent, tripping (the child tumbling while being held by the hand or wrist), and getting the child dressed (e.g. pulling a child's arm through a coat sleeve with too much force).

In the non-traction group (n = 547) the majority occurred during a fall. In 30 children the nursemaid's elbow occurred due to rolling over in the bed, while the arm was caught under the body. Most of these 30 children were under the age of 1 year and 22 of them were girls.

Li et al. analyzed the findings in 69 children (median age 2.4 years; 50% between 1.5 and 3.6 years) with nursemaid's elbow due to non-axial traction mechanisms [218]. The most commonly reported causing mechanisms were falls (57%), direct hits to the elbow (16%), and rolling over (7%).

The predominance of the nursemaid's elbow in the left arm can most probably be explained by the fact that most adults are right-handed holding the child's left hand or wrist [207].

'Rolling over' is probably the most common mechanism in infants under the age of 1 year. The 'rolling over' mechanism was first described by Newman, who reported 4 infants under the age of 6 months with the 'rolling over' mechanism [213]. In 3 of these 4 infants it was not a spontaneous 'rolling over', but a forced 'rolling over', in which the child was rolled over by another (once an older sister and two times the mother). Newman also reported 1 infant under the age of 6 months who had been lifted by the arm and another infant whose arm was pulled by a sibling.

Newman stated about the 'rolling over' mechanism in infants: '*Although child abuse should always be kept in mind when there is unusual trauma in the young baby, none of the instances described was thought to represent abuse'*. In other words, knowing the causing mechanism of a nursemaid's elbow (e.g. traction/pulling or rolling over) does not imply knowing whether the nursemaid's elbow was sustained due to accidental or non-accidental circumstances.

12.6 Fractures of the Hand

12.6.1 General Aspects of Fractures of the Hand

Approximately one-fifth of all hand injuries in children are fractures [219]. Fractures of the hand are common injuries in children and adolescents and account for 15% of all paediatric fractures and for 2.3% of all paediatric ER visits [220, 221]. Boys sustain hand fractures more often than girls in an almost 3:1 ratio [220]. The incidence of hand fractures is low in infants, but increases with age.

Vadivelu et al. found that hand fractures occurred in toddlers in 34 per 100.000 per year, while hand fractures in children aged 11–18 years increased up to 663 per 100.000 per year [222].

Kreutz-Rodrigues et al. did a review of frequency and pattern of paediatric hand fractures in a 27-year period [223]. The data of 4356 hand fractures in patients under the age of 18 years (mean age 12.2 years) (categorized in 3 age groups: 0–5, 6–11, and 12–17 years) were evaluated. Most hand fractures were found in the 12–17 years group (n = 2775, 64%), followed by the 6–11 years group (n = 1347, 31%) and finally the group of children under the age of 5 years (n = 234, 5%). Most hand fractures were found in the proximal/middle phalanx (48%), followed by metacarpal (33%), distal phalangeal (12%), and intra-articular metacarpophalangeal/proximal interphalangeal/distal interphalangeal joints (7%). Proximal/middle phalangeal fractures were the most common in all age groups.

Chung and Spilson found that in children, aged 5–14 years, the overall incidence of hand fractures was 546 per 100,000 per year [224]. The incidence of carpal fractures was 131 per 100.000 per year, of metacarpal fractures 250 per 100.000 per year, and of phalangeal fractures 165.6 per 100.000 per year. According to Chung and Spilson the highest incidence of phalangeal fractures occurs in the 0–4 year age group (around 0.2% of children in that age group.

Metacarpal fractures and carpal fractures occur slightly more rarely, at a rate of approximately 0.1% of children overall.

12.6.2 Cause and Manner of Fractures of the Hand

Fractures of the hand are usually caused by a direct impact, either by being hit with an object or by punching, or by crushing with a heavy object. Fractures may also occur due to hyperextension or hyperflexion [225].

Fractures of the hand can be sustained in accidental and in non-accidental circumstances.

In young children accidental crush injuries to the digits are common, e.g. after getting stuck in the door (Fig. 12.50) [226]. In older children, hand fractures usually are sustained in accidental circumstances, e.g. in sports and play-related trauma (Figs. 12.51, 12.52a–c and 12.53) [220, 226]. In adolescents fractures of the hands are often described to occur in non-accidental circumstances, e.g. in fights, and can be the result of self-defence and/or fighting back [220].

Although the hand is the second most frequently injured part of the body in older children and adolescents, the number of studies on fractures of the hands in children, especially in association with non-accidental trauma, is limited compared to other bony injuries [224, 227–232].

According to Johnson et al. in the paediatric population the hands can be considered to be a target organ in case of non-accidental trauma (child abuse) [233]. They evaluated the findings in 944 reports of non-accidental trauma. Injuries involving the hands were found in 94 children. Of the 94 children 18 (20%) were admitted to hospital, because of burn injuries (17), apnoea (2—once combined with a bite mark and once with bruising), fractures (2), bruising (2), crushing injury (1—fingers slammed in a door). In 19 children (2%) the hands were the only location with inflicted injuries: burns (8), bruises (2), human bite marks (2), erythema (2), fractures (2), swelling (2), and laceration (1). The fractures were found in a 5-year-old girl after being hit on the hand and in an 11-year-old girl with an unknown history. Despite the fact that the hand may be a target organ, hand fractures seem to be relatively rare compared to other hand injuries (Table 12.7). Of the 'hand only' group 5 children were admitted to hospital.

Fig. 12.50 Four-year-old child whose finger got caught in the door opening. Radiography shows a crush fracture of the distal phalanx of the second digit (inset)

Fig. 12.51 Fracture of the proximal phalanx (open arrow) of a 4-year-old girl who had a television topple on her hand

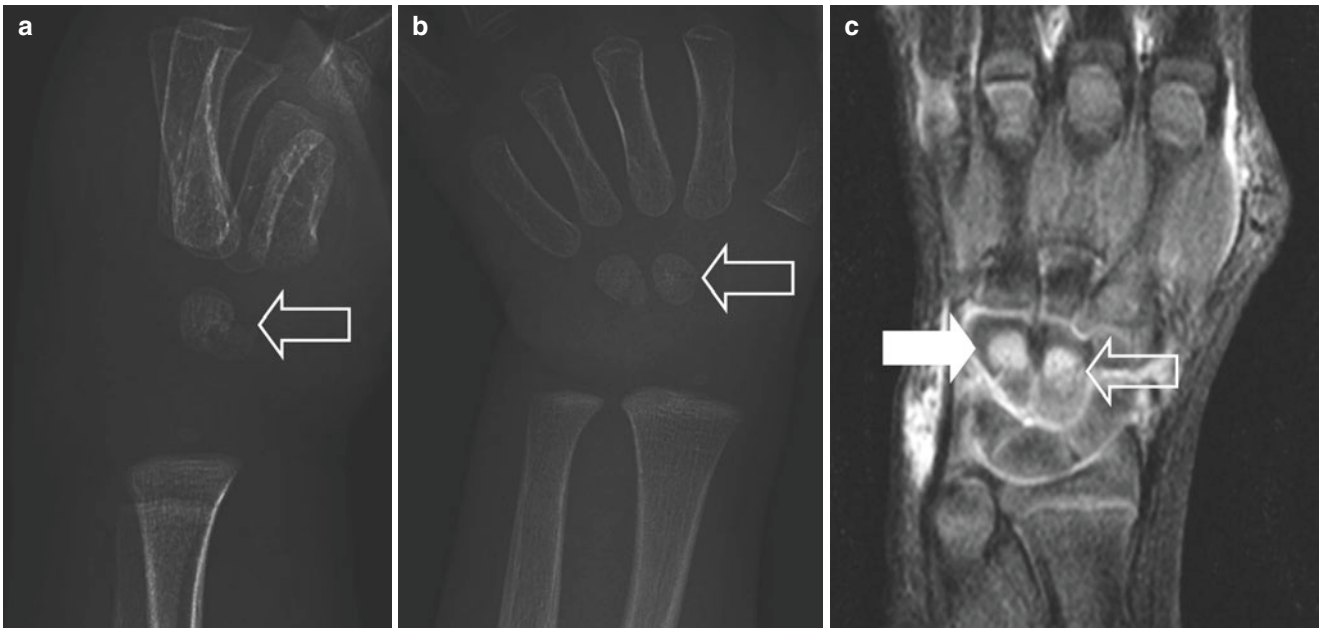


Fig. 12.52 (**a**) Two-year-old girl who had a drawer fall on her hand while playing. Radiological examination revealed a fracture of the capitate bone (Reprinted with permission [465]) (**b**) Postero-anterior view of the hand shows the fracture of the capitate bone. (**c**) Coronal STIR-weighted MRI shows bone oedema at the location of earlier-mentioned capitate fracture (open arrow); however, also of the hamate bone (arrow)

Fig. 12.53 Five-year-old child who sustained a trauma, a heavy object fell on his hand, while playing in the schoolyard. Radiograph shows an intra-articular communitive fracture of the head of the proximal phalanx of the 5th finger (inset)

Table 12.7 Inflicted injuries to the hand [233]

	Hand only	Hand and other body areas	Total	%
Abrasions	0	11	11	12
Bruises	2	29	31	33
Burns	8	16	24	25
Other (incl. fractures)	9 (2)	19	28	30
Total	19	75	94	100

Unexplained or unexplainable fractures of the hands in children under the age of one year are thought to have a strong association with non-accidental trauma (Figs. 12.54a–e, 12.55a, b and 12.56a, b) [10, 234–236].

Nimkin et al. evaluated 11 infants under the age of 10 months with fractures of hands and feet, due to non-accidental trauma [235]. A total of 22 fractures were noted. Six infants had a total of 15 fractures of the hands (6× metacarpal and 9× proximal phalangeal fractures). The authors found predominantly torus fractures, which according to the authors are consistent with forced hyperflexion. Seven infants had three or more additional fractures of long bones of the upper and lower extremities, and seven infants had additional fractures of the ipsilateral extremity. Only one child showed clinical symptoms.

Despite the supposed strong association with non-accidental trauma Pandya et al. reported in a comparative study an OR of 0.3 to find these fractures due to accidental

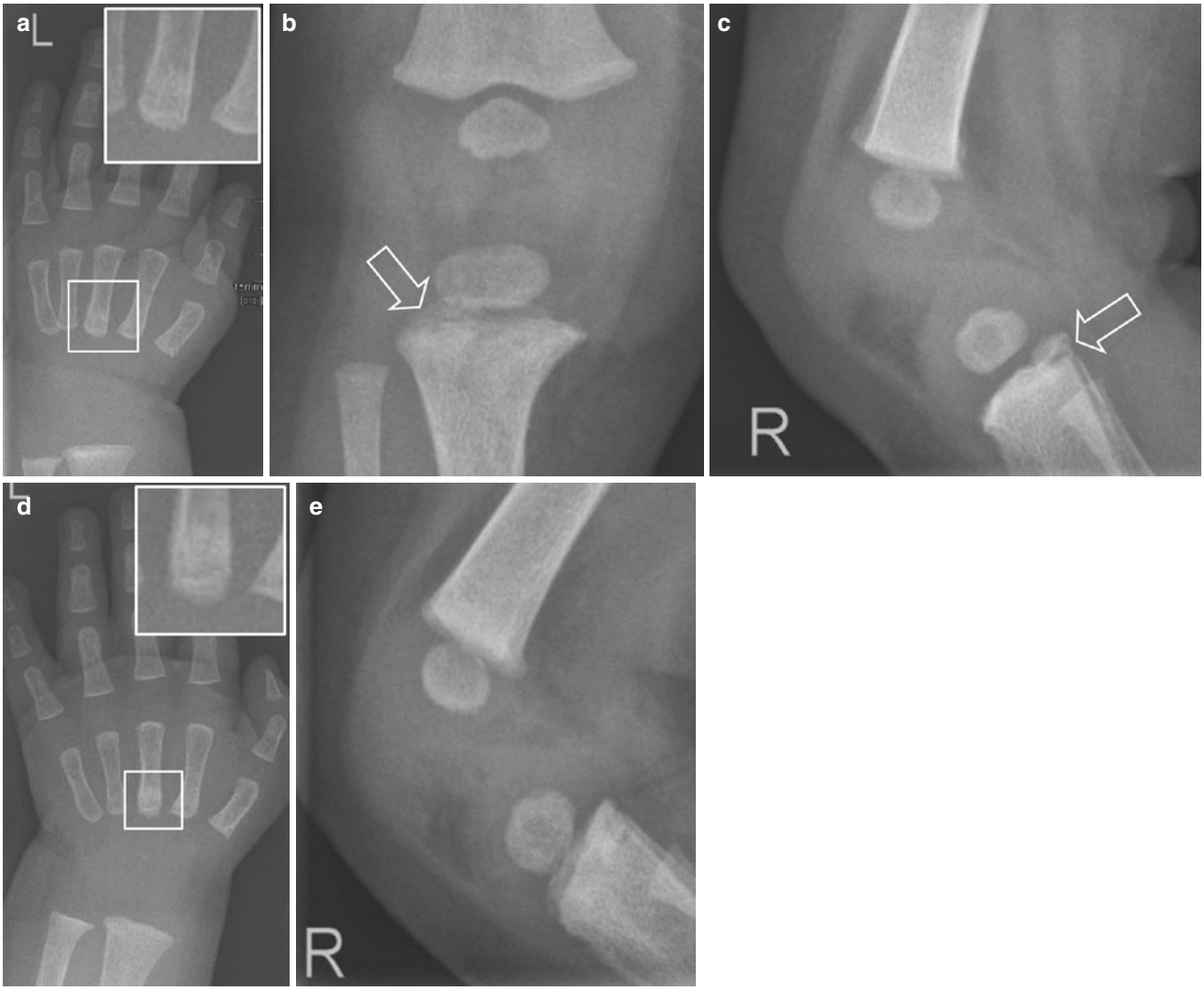


Fig. 12.54 Four-month-old sibling of an abused index case. (**a**) Radiograph of the left hand shows slight irregularity and subperiosteal new bone formation at the base of MC-III (inset). (**b**) AP radiograph of the right knee shows a metaphyseal corner fracture of the proximal tibia (arrow). (**c**) Lateral radiograph shows the posterior location of the fracture (arrow). Repeat skeletal survey shows (**d**) a healing fracture of the base of MC-III (inset) and (**e**) a healed metaphyseal corner fracture of the proximal tibia

circumstances compared to non-accidental circumstances in infants younger than 18 months old and an OR of 0.5 in children older than 18 months [130]. The inclusion by the authors of children aged 0–18 months means the inclusion of both pre-mobile and mobile children in the same group. The findings in pre-mobile and mobile children were not split. Because of this it is not possible to draw definitive conclusions concerning the circumstances under which hand fractures were sustained in the pre-mobile group.

Hand fractures have only rarely been reported in studies, concerning infants and children who underwent a skeletal survey for the evaluation of suspected non-accidental trauma [10, 236–238]. Barber et al. reported on a study on 567 children, of whom 313 suffered a total of 1,029 fractures [10]. Seven (2.2%) children had fractures of the hand. In the study by Kleinman et al. 225 out of 365 children had one or more fractures on the skeletal survey. Five (2.2%) children had a total of 10 fractures of the hand [236]. Karmazyn et al. studied 930 children of whom 317 had a total of 899 fractures. One infant had 6 (0.7%) fractures of the hand [237]. In the, by far largest, the study of Lindberg et al. out of 2890 children 1208 had one or more fractures. Fifteen of them (1.1%) had a total of 20 fractures of the hand [238]. In this study, there were 7 children with either a fracture to the hand or foot, but it was not possible to discriminate as the report spoke of, e.g. a digital fracture.

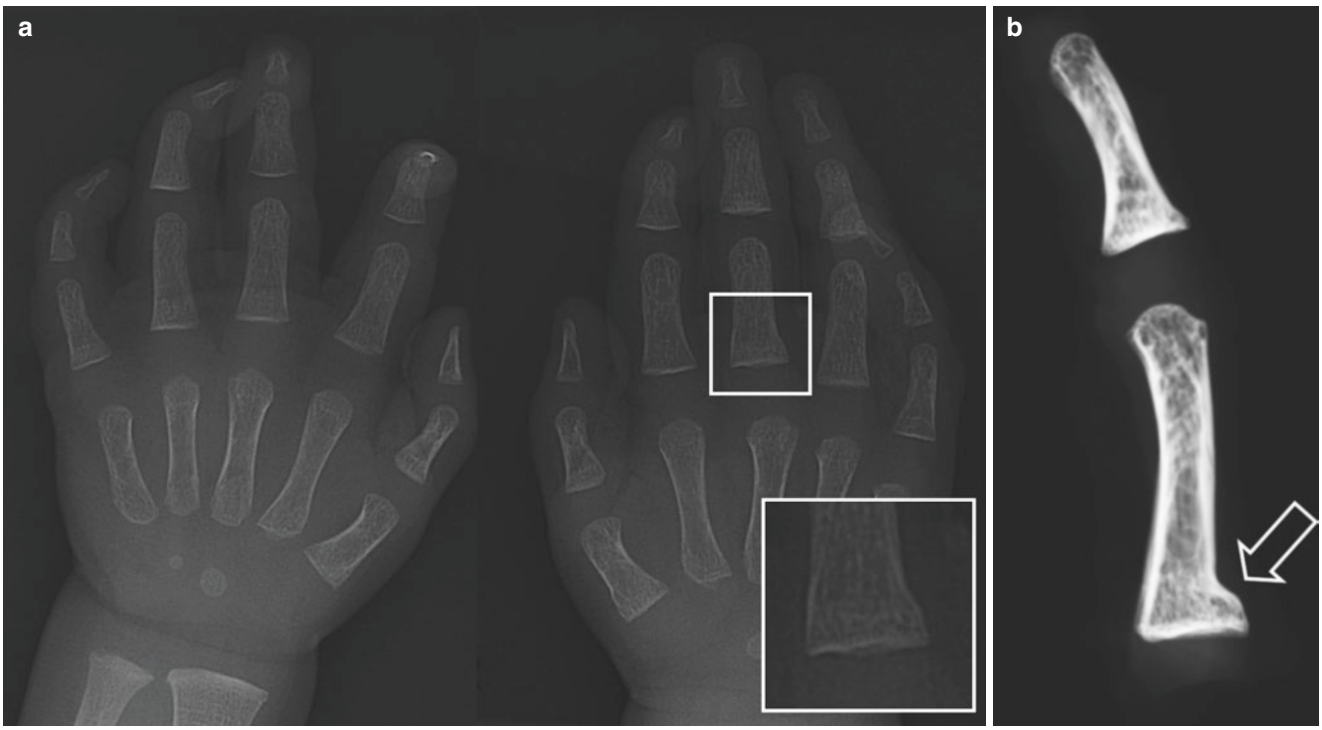


Fig. 12.55 (**a**) One-and-a-half month old girl was found dead in her crib. Radiological examination of the hands revealed a torus fracture at the base of the proximal phalanx of the third finger of the right hand (see inset). (**b**) Radiograph of the finger, sampled at autopsy. The radiograph has been taken with a mammography system, because of its high resolution

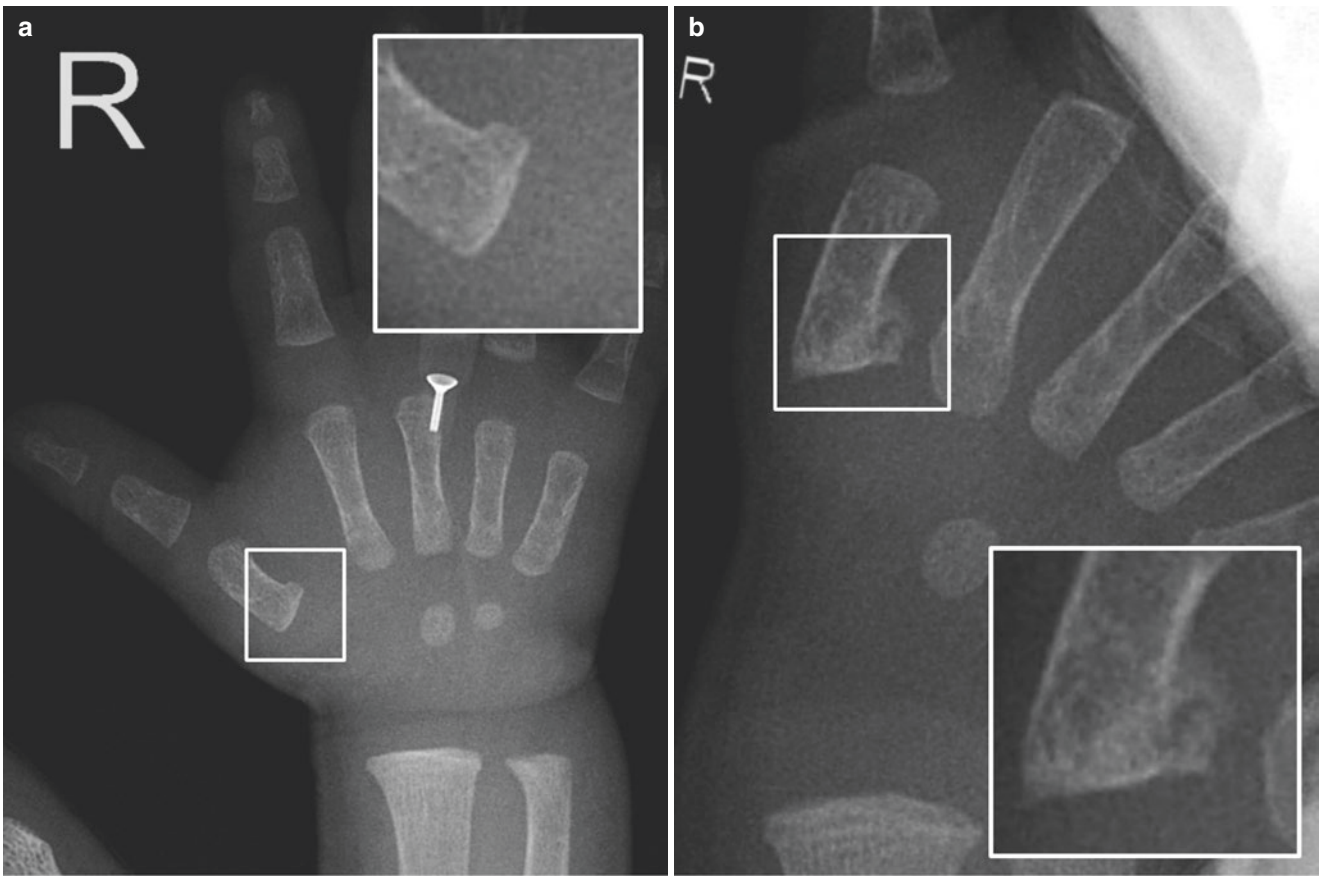


Fig. 12.56 Four-month-old infant suspected of being a victim of child abuse. (**a**) Radiograph of the right hand shows a buckle fracture of the base of MC-I (inset). (**b**) Radiograph after 2 weeks clearly shows callus formation (inset)

12.7 Femur

12.7.1 General Aspects of Femur Fractures

The femur is the largest and strongest bone in the body. The proximal part of the femur (caput femoris—head of the femur) articulates with the acetabulum of the pelvis to form the hip joint and the collum femoris (neck of the femur) connects the caput with the shaft. The distal end of the femur is characterized by the presence of the medial and lateral condyles, which articulate with the tibia and patella to form the knee joint.

Fractures of the femur are relatively rare in paediatric patients and account for around 2% of all paediatric fractures. Fractures of the shaft are much more common than fractures of the proximal or distal femur. The first peak of occurrence is found in the first 2–3 years of life and the second peak is in adolescence [239].

Buess et al. analyzed the findings in 100 consecutive children (0–18 years; mean age 7.3 months) with femoral fractures [240]. Boys to girls ratio was 1.85:1. Only one patient was older than 16 years, a severely handicapped spastic child with cerebral palsy with a pathological fracture. Buess et al. found 3 peaks: 0–4 years, 6–10 years, and 13–15 years. Fractures in the youngest children most likely occurred due to accidental falls (usually low energy trauma). Traffic accidents (high energy trauma) were seen mainly in the group of school children, whereas sports-related fractures were seen mainly in adolescents. Pathological fractures were found in 8 children: spastic cerebral palsy in four children, achondroplasia, bone cyst, poliomyelitis, and posttraumatic osteoporosis each in one child. In two children in the youngest group the femur was due to non-accidental trauma.

Brown and Fisher evaluated the occurrence of femur fractures in 2753 children under the age of 6 years by using the '1997 Kids' Inpatient Database' [241]. They did not differentiate between fractures of the proximal femur, shaft, and distal femur. They found that the occurrence of femur fractures was highest in the first year (especially during the 3rd month of life, slightly decreasing between the 4th and 11th months) and between the ages of 20–40 months. In the children under 1 year of age the boys to girls ratio was 1:1, whereas in the older children more boys than girls sustained femur fractures. According to Brown and Fisher their findings suggest that an infant has as great a chance of sustaining a femur fracture due to non-accidental trauma as an older child dominantly does due to accidental trauma because of their increasing motoric abilities, e.g. climbing, and their increasing mobility.

Loder et al. evaluated the characteristics of femur fractures in 9963 children and adolescents under the age of 18 years: 1076 fractures between 0 and 2 years (11%), 2119 between 2 and 5 years (21%), 3237 between 6 and 12 years (33%) and 3528 between 13 and 18 years (35%) [242]. Boy to girl ratio was almost 2.5:1. Of the 9963 fractures, 9458 were closed. Of the closed fractures 70% were shaft fractures, 12% were proximal and 18% were distal fractures. Shaft fractures occurred in 2493 (81%) of 3096 closed fractures in children under the age of 6 years compared to 3940 (65%) of 6080 in children aged 6–18 years ($P< 0.001$). Fractures of the proximal femur occurred twice as often in the 2 older age groups (aged 6–18 years) compared with younger children under the age of 6 years. The fewest fractures of the distal femur were found in the 2–5 years old group. Open fractures were found in 505 cases. 70% of the open fractures occurred in the adolescent group. Fractures in younger children most likely occur due to accidental falls (usually low energy trauma), whereas those in older children most commonly occur as a result of motor vehicle accidents (high energy trauma). Approximately 2% of all children sustained the fracture (location not further specified) due to non-accidental trauma. Nearly all of these children were under the age of 2 years. In these age group 15% of all femoral fractures were due to non-accidental trauma.

Petković et al. evaluated the findings in 143 children (average age 8.6 years) with femur fractures [243]. Sixty-five percent were shaft fractures, 21% were fractures of the proximal fracture and 14% of the distal fracture. Boys to girls ratio was 3.2:1. The fractures occurred during play and sport activities in 67 children and in traffic accidents in 64 children. Pathological fractures were found in 12 children.

Baldwin et al. compared the findings in 70 children with non-accidental femur fractures to 139 children with accidental femur fractures [244]. Children with accidental femur fractures more often had shaft fractures (46% versus 66%), children with non-accidental femur fractures more often had distal femur fractures (37% versus 20%). No difference was found between both groups concerning proximal femur fractures. They identified 3 risk factors that could be helpful in differentiating non-accidental from accidental femur fractures (Fig. 12.57):

- Age younger than 18 months.
- Physical and/or radiographic evidence of prior trauma.
- History suspicious for non-accidental trauma.

Volkman reviewed the findings in 228 children under 2 years of age with a total of 235 femoral fractures (including 6 bilateral fractures and 1 case with 2 fractures in the same limb). Volkman found that the overall percentage of non-accidental femur fractures was 10.9% (25 of 228 children). In children under the age of 6 months, 25.8% (15 of 58 infants) of the fractures were the result of non-accidental trauma, under the age of 1 year, 16.4% (20 of 122 infants) and over 1 year, 4.7% (5 of 106 children). The percentage of suspicious or indeterminate cases was 25.8% (59 of 228

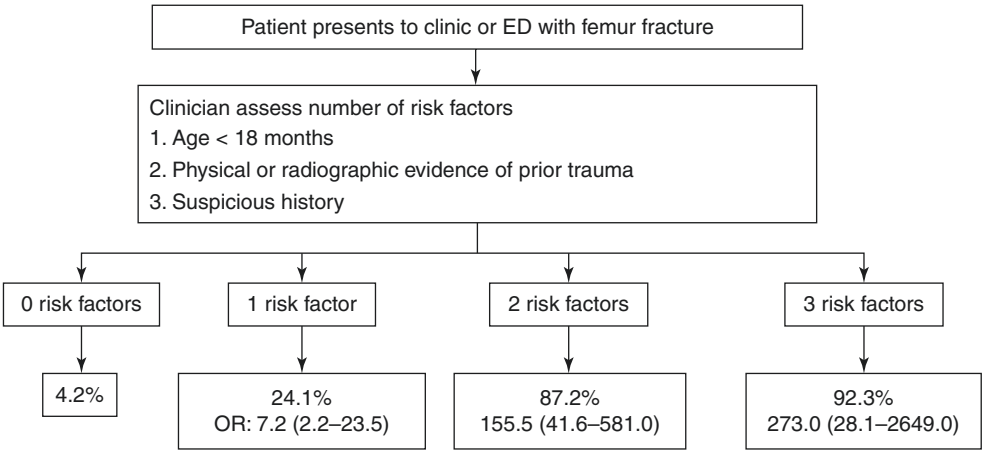


Fig. 12.57 Algorithm for determining whether a femur fracture stems from abuse or accidental trauma as presented by Baldwin et al. [244]. Percentages refer to the risk of an abusive femur fracture, the Odds Ratio is the result of a multiple logistic regression model with a number of risk factors (risk factors: age younger than 18 months, physical and/or radiologic evidence of prior trauma, and suspicious history)

cases) and of accidental cases 74.2% (169 of 228 cases). Proximal femur fractures were found in 36 children, midshaft fractures in 134 children, distal femur fractures in 65 children:

- Of the proximal fractures 6 were the result of non-accidental trauma, and 26 of accidental trauma. In four children the circumstances were not known.
- Of the midshaft fractures 15 were the result of non-accidental trauma, and 97 of accidental trauma. In 22 children the circumstances were not known.
- Of the distal femur fractures 10 were the result of non-accidental trauma, and 46 of accidental trauma. In nine children the circumstances were not known.

Volkman concluded that several factors could help differentiate between non-accidental and accidental femur fracture, namely age under 12 months, non-ambulatory status, delayed presentation, concurrent injuries, bilateral fractures, and unknown/inconsistent history of mechanism of injury.

Engström et al. evaluated the occurrence of femur fractures in Swedish paediatric patients under the age of 16 years (n = 709) [245]. Most fractures were located in the shaft (64%), followed by the distal femur (27%) and the proximal femur (9%). Boys to girls ratio was almost 2:1. Most fractures were observed in boys aged 2–3 years and in adolescent boys, while in girls the fractures were evenly distributed. In younger children the fractures were most commonly sustained in falls, whereas in adolescents traffic-related accidents were the most common.

Rokaya et al. evaluated the findings in 104 children (mean age 5.5 years; boys to girls ratio 1.6:1) with femur fractures [246]. 65.3% were fractures of the shaft, 18.2% of the proximal femur and 16.3% of the distal femur. Most occurred due to accidental falls from varying heights (ladder, rooftop, cliff, horse, bicycle) or during sporting activities. In 4 children the fracture occurred in non-accidental circumstances (physical assault).

Valaikaite et al. reviewed the findings in 348 children with a total of 353 femur fractures [247]. The mean age was 7.5 years, ranging from 0 to 15 years. 37 children were under the age of 1 year, 112 between 1 and 5 years of age, 125 between 6 and 11 years, and 74 between 12 and 15 years. Except for children under the age of 1 year, most fractures occurred in male patients (69%), with boys to girls ratio of 2.2:1. In the group of children under 1 year of age 68% were girls (girls to boys 2.2 to 1). Fractures of the shaft were most common in all ages (72.2%), followed by fractures of the distal femur (17.9%) and the proximal femur (8.2%). In 1.7% the location was not reported in the medical records. Femoral fractures were mainly due to low-energy trauma in neonates and infants, to road accidents and low-energy trauma in preschool children, to sports accidents (especially skiing) in school-age children, and to road traffic accidents in teenagers. 94.9% were closed fractures. Pathological fractures were found in 29 cases (13× shaft, 6× distal femur, 10× proximal femur).

12.7.2 Fractures of the Proximal Femur

12.7.2.1 General Aspects of Fractures of the Proximal Femur

Fractures of the proximal femur are uncommon in children. They account for less than 1% of all paediatric fractures [239, 248–250]. Boy to girl ratio is 2.5:1 [251].

Fractures of the proximal femur and hip fractures are sometimes used as synonyms [252]. This can be confusing because hip fractures can also be defined as fractures in 1 or more of the bones, that form the hip joint (proximal femur and pelvis, especially the acetabulum—see also Chap. 11).

Proximal femur fractures are classified as transepiphyseal, transcervical, cervicotrochanteric, and intertrochanteric fractures (Fig. 12.58) [248, 250, 252, 253]:

- Transcervical fractures (Delbet type II) are fractures through the mid-portion of the femoral neck. This is the most common type in children and adolescents, account-

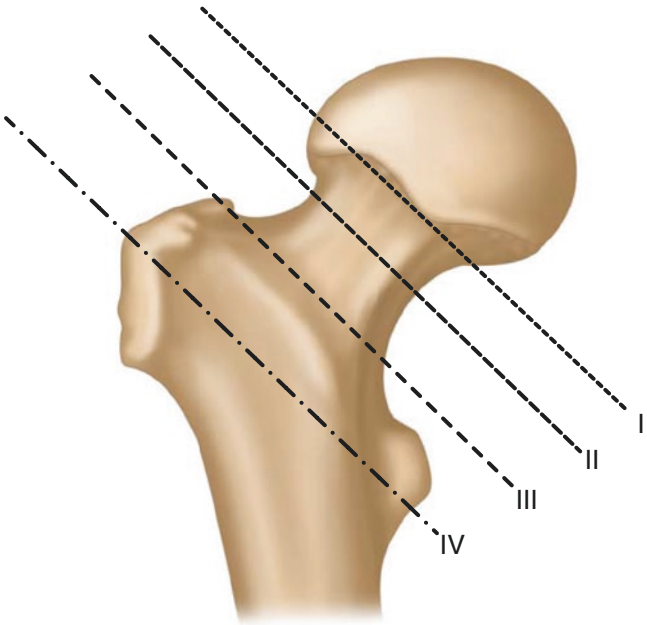


Fig. 12.58 Femoral neck classification according to Delbet

ing for 40–50% of fractures of the proximal femur in paediatric patients. Miller identified three peak ages for type II fractures: 2–4 years of age, 8 and 9 years of age, and 12 and 13 years of age [248, 254].

- Cervicotrochanteric fractures (Delbet type III) are fractures through the base of the femoral neck and are found in 25–35%.
- Intertrochanteric fractures (Delbet type IV) are fractures between the greater and the lesser trochanters and account for 6–15%.
- Transepiphyseal fractures (Delbet type I) are fractures through the proximal femoral physis, representing a Salter–Harris type I of the proximal femur. This type may occur with or without dislocation of the femoral head. This is the least common type (less than 10%). However, in infants and young children under the age of 2 years and in children aged 5–10 years this type is more common than in children between 2 and 5 years and above 10 years.

Due to the vascular anatomy and the active growth plate, specifically in skeletally immature young children, fractures of the proximal femur can be associated with severe complications such as premature physeal closure, coxa vara or valga, avascular osteonecrosis, mal- or non-union, limb shortening, and arthritic changes [253, 255–259].

12.7.2.2 Cause of Fractures of the Proximal Femur in Normal Bone

Because of the high bone mineral density the proximal femur in children is very strong (except for the physis). A severe high-energy trauma will be necessary to fracture the proximal femur [259, 260]. Up to 90% of all fractures of the proximal femur are caused by a high-energy trauma [248, 250]. In children 30–85% of the proximal femur fractures are associated with other often major injuries, reflecting the severity of the trauma [248]. Associated injuries are head and/or facial injuries, abdominal injuries (splenic lacerations, retroperitoneal haemorrhage), intra-pelvic visceral injuries, perineal injury, and other skeletal injuries, e.g. pelvic ring or acetabular fractures, hip dislocation, or other femur fractures [248, 251, 256, 261, 262].

Several mechanisms, resulting in a high-energy trauma to the proximal femur, can be deduced from the literature. These may result from direct trauma, e.g. due to a direct blow to the hip, or from indirect trauma, e.g. due to axial loading, hyperabduction, or torsion (see Sect. 12.7.2.3). According to Ogden et al. birth-related fractures of the proximal femur in neonates most probably are due to a combination of hyperextension, abduction, and rotation during forceful traction [70].

If it is suspected that a fracture of the proximal femur resulted from a low-energy trauma, (e.g. a fall from standing height or a twisting mechanism) one must consider the presence of an underlying disorder with weakened bone, e.g. metabolic bone diseases, benign and malignant bone tumours, or non-accidental trauma (see also Sect. 12.7.3.3.4) [250, 251, 263].

12.7.2.3 Manner of Fractures of the Proximal Femur

Before Birth

No reports concerning proximal femur fractures, sustained before birth, were found.

During Birth

Birth-related injuries of the proximal femur are probably rare, and have only been described in single case reports or small series of cases.

The majority of these studies focus on proximal femoral epiphysiolysis of the femur (= Delbet I, transepiphyseal fracture) [264–271] [70, 248, 252]. This most commonly occurs due to a difficult and traumatic breech delivery and is caused by a combination of hyperextension, abduction, and rotation during forceful traction of the leg [70]. ‘Large for date’ neonate probably are more at risk [272]. Clinically it is usually diagnosed soon after birth as the neonate tends to keep the extremity limp in a position of flexion, abduction, and external rotation. Clinically passive motion is painful and swelling can be present.

After Birth: Accidental or Non-accidental Trauma

After birth fractures of the proximal femur may occur in accidental and in non-accidental circumstances. There are

two peaks of occurrence: children under the age of 2–3 years, and older children above the age of 11 years and in adolescents [251, 253].

Non-accidental fractures of the proximal femur are less common than non-accidental fractures of the shaft and the distal femur. According to Baldwin et al. diaphyseal femur fractures are found more often in children with accidental trauma than in children with non-accidental trauma, while distal femur fractures were more common in children with non-accidental trauma than in children with accidental trauma [244]. They found no difference in the occurrence of proximal femur fractures between both groups.

In children under the age of 2–3 years non-accidental circumstances should always be considered in case of fractures of the proximal femur, especially if a history of a plausible accident is lacking or if the described accident can be considered to be a minor trauma with insufficient force to explain the fracture [75, 244, 252, 273–275].

- Jones et al. reported two girls with hip problems [76]. The first girl was seen at the age of 9 months at a well-child check-up and the mother stated that she was not rolling over or bearing weight on her legs, which she did before. A radiograph showed that the femoral head was seated in a normal acetabulum, but that the neck was displaced cranially and laterally. A skeletal survey showed healing metaphyseal corner fractures of the proximal humerus bilaterally, a healing metaphyseal corner fracture of the right distal humerus and a metaphyseal corner fracture of the left proximal tibia. Several rib fractures were also found. Her mother's boyfriend eventually admitted that he forcefully pulled the arms backward in anger and that he caused the hip injury during a frustrated diapering attempt by abducting the child's thigh and then pushing the shaft towards her acetabulum. The second girl was seen at the age of 2 months. It was suspected that she had a displaced hip. On X-ray, it was seen that her left proximal femur was displaced cranially and laterally. There were signs of early callus. At the age of 4 months proximal and distal parts of both femurs had periosteal layering. There were also healing rib fractures visible and healing fractures of the distal right radius and healing metaphyseal corner fractures of the distal right and left femur, both proximal tibias and possibly the distal tibias. No indication was found for a primary skeletal abnormality. Jones et al. noted that this type of injury can look identical to epiphyseal separations due to birth trauma.
- According to Beaty non-accidental trauma should always be excluded if a Delbet type 1 fracture is found in an infant [248].
- Gholve et al. described a 3-year-old girl with a femoral neck fracture, which occurred due to non-accidental circumstances [275]. The authors stated that femoral neck fractures usually are sustained in a high-energy trauma or, less common due to pathological conditions (Sect. 12.7.3.3.4). However, the possibility of non-accidental circumstances should be considered if there is no indication of one of these.
- Pastor et al. presented a 5-month-old boy with a 4-day history of diagnosed upper respiratory illness and a new 2-day history of decreased left hip motion and pain [274]. The decreased movement and pain were caused by a fracture of the femoral neck. A full skeletal survey indicated a metaphyseal corner fracture of the right distal femur and a periosteal reaction of the right fibula shaft. The boy also had evidence of fractures of the left distal femur and the left proximal fibula in various stages of healing. Externally visible injuries were not described.
- Kembhavi and James described the findings in a 4-year-old girl with bilateral intertrochanteric fractures [273]. Initially, it was thought that the fractures were due to a fall from height. However, the child had multiple fractures in different stages of healing: relatively minimal vertebral wedge compression fractures, combined with older fractures (right-sided supracondylar humerus fracture, left proximal ulna fracture, and right-sided proximal tibial metaphyseal fracture with physeal injury). It was concluded that the fractures were sustained due to non-accidental circumstances. The child had no externally visible injuries.
- Shalaby-Rana et al. reported eight children, aged 2.5–26 months (mean age 10 months) with a total of 10 fractures of the proximal femur physis [75]. All children showed lateral displacement of the proximal femur. Two children, aged 3 and 8 months, had bilateral fractures of the proximal femur. In seven of the eight children non-accidental trauma was confirmed. Six of them, aged 2.5–10 months, had other fractures, most commonly rib fractures or metaphyseal corner fractures. In two children no other fractures were found. One child, aged 13 months, was disciplined by his father by slapping his thigh. Eighteen days before presentation the father had punched the child's left thigh, after which the child stopped bearing weight. In one child, aged 26 months, it was concluded that the child was medically neglected, because the parents did not seek medical care until 2 weeks after the child stopped bearing weight.

In children above the age of 11 years and in adolescents up to 90% of all proximal femur fractures occur in accidental circumstances, usually a high energy trauma with a high-energy transfer, like motor vehicle accidents, falls from great heights, or high-impact sports trauma [250, 259, 260]. Transcervical fractures (Delbet type II fractures) most commonly occur due to severe trauma with a high-energy transfer/high-velocity accidents involving a direct impact, such as

motor vehicle accidents, or pedestrian-vehicle accidents. Delbet type II fractures may also occur due to falls from height [248, 253]. Three peak ages can be identified for type II fractures: 2–4 years of age, 8 and 9 years of age, and 12 and 13 years of age [248].

Cervicotrochanteric fractures (Delbet type III) and intertrochanteric fractures (Delbet type IV) also demand trauma with a high energy transfer [248].

According to Beaty, a severe trauma is needed to sustain a trans(epi)physeal fractures (Delbet type I fracture). This may occur during birth (see Sect. 12.7.2.3.2) and after birth due to an accidental trauma with a high-energy trauma or in seizures [248, 276]. Two peak ages have been described: infants and young children under the age of 2 years and children between 5 and 10 years of age.

In healthy, mobile children, usually young athletic adolescents, stress fractures of the proximal femur due to repetitive activity such as running, jumping, and during sports have been described. These children typically present with ongoing pain, increasing with physical activity. Because they are so rare, a broad differential diagnosis has to be considered, and it can be hard to diagnose these stress fractures [277–290].

Rinat et al. described the occurrence of fractures of the proximal femur (Delbet type II and III) in two girls aged 10 and 12 years, due to a trauma caused by (suspected) hyperabduction while sliding on a water slide [291].

The occurrence of Delbet-type I fractures has been described to occur during attempted closed reduction of a traumatic hip dislocation with a nondisplaced physeal fracture in adolescents [292–294].

After Birth: Diseases with an Increased Risk of Fractures of the Proximal Femur

Pathological fractures of the proximal femur are very rare in paediatric patients but may occur in malignant and in benign medical conditions [263]. Physicians should consider a pathological fracture in a child in case of a (confirmed) history of a minor or insignificant trauma (a trauma with a low transfer of energy), or in case of the suspected presence of abnormal findings on radiological imaging.

Pathological fractures of the proximal femur in children, caused by a minor trauma can be found due to generalized changes in mineral density of the bone (e.g. osteogenesis imperfecta) or due to localized changes in density (e.g. in infections, bone cysts, bone tumours, and tumour-like lesions) [263, 295–300]. The proximal femur is one of the most common locations for benign bone tumours in children.

Shrader et al. identified pathologic femoral neck fractures, including two basicervical fractures, in 15 children (9 boys, 6 girls) ranging in age from 18 months to 15 years (mean age, 9 years) between 1960 and 2000: fibrous dysplasia (n = 5 children), unicameral bone cyst (n=2), Ewing's sarcoma (n = 2), osteomyelitis (n = 2), leukaemia (n = 1), rhabdomyosarcoma (n = 1), osteogenesis imperfecta (n = 1), and osteopetrosis (n = 1) [263]. According to authors paediatric patients with pathologic fractures of the proximal femur are at significant risk for complications.

Femoral neck fractures may also occur in children with osteopenia secondary to other conditions, e.g. cerebral palsy or muscular dystrophy.

12.7.3 Fractures of the Femoral Shaft

12.7.3.1 General Aspects of Fractures of the Femoral Shaft

Shaft fractures are the most common femur fractures in children, especially midshaft fractures, accounting for over 60% of all paediatric femur fractures [301]. They account for almost 2% of the fractures in children [242, 245, 302–306]. This includes subtrochanteric and supracondylar fractures (fractures of the upper and lower third of the shaft) [302].

Femoral shaft fractures are more common in boys compared to girls with a ratio of approximately 2.6:1 [307]. Two peak ages can be distinguished, especially in boys: toddlers aged 2–4 years and adolescents above the age of 12 years [242, 245, 304, 308, 309].

The incidence of femoral shaft fractures in children is estimated to be between 11 and 20 per 100.000 children worldwide. In 1999 Hinton et al. reported an annual incidence of 19.15 per 100,000 children [304]. The incidence of femoral shaft fractures seems to have decreased in the last decades. Based on data from the Swedish National Hospital Discharge Registry (SNHDR) von Heideken et al. reported that from 1987 to 2005 a total of 4984 children, aged 0–14 years, had a diagnosis of a femur shaft fracture [303]. They found an overall annual incidence of 16.4 cases (95% CI, 15.9–16.8) per 100,000 children, where during the observed period, the annual incidence of femur shaft fractures declined on average with 3% per year with a total decrease of 42%. The authors do not give an explanation for this decrease but similar findings have been reported in the United Kingdom by Bridgman and in the United States by Wilson and Mooney and Forbes [306, 310]. The authors do suggest that it might be related to the fact that children tend to be less physically active, the increased role of injury prevention research, and safety education campaigns.

In the United Kingdom, Talbot et al. identified in 2018 a total of 1852 isolated, closed fractures of the femoral shaft in children from birth to 15 years of age, indicating a mean annual incidence was 5.82 per 100 000 children (95% confidence interval (CI) 5.20–6.44). The age of peak incidence was two years for both boys and girls; this decreased with increasing age [311].

12.7.3.2 Cause of Fractures of the Femoral Shaft

As stated before, the femur is the largest and strongest bone in the body. Above that, the femoral shaft is protected against blunt force trauma by the surrounding muscles. If a fracture of the shaft is found in a paediatric patient with normal bone, the fracture must have been caused by a trauma with a high energy transfer, either a direct blow to the shaft or an indirect trauma, transmitted at the knee. The protective muscles, however, also are responsible for the displacement, which is often seen in fractures of the femur shaft [312, 313].

The high-energy transfer, which is needed to fracture the femur, can be illustrated by the fact that especially in accidental circumstances (see Sect. 12.7.3.3.3) the fracture often is associated with other injuries, due to a high-energy transfer, e.g. intracranial or intra-abdominal injuries. These injuries regularly result in life-threatening circumstances [313].

Fractures of the femur may also occur in children with bone diseases with an increased risk of fractures (pathological fractures) due to a lesser amount of force (see Sect. 12.7.3.3.5).

12.7.3.3 Manner of Fractures of the Femoral Shaft

Fractures of the femoral shaft can occur before, during, or after birth. If a fracture is sustained after birth, this may have occurred in accidental or in non-accidental circumstances.

Before Birth

In the literature, several case reports are found concerning intrauterine fractures of the shaft. Multiple intrauterine fractures of long bones, including fractures of the shaft, can be found due to medical conditions with increased fragility of bone-like skeletal dysplasias or due to severe maternal (abdominal) trauma [314, 315]. Maternal trauma may occur in accidental and in non-accidental circumstances. Domestic violence (intimate partner violence) may occur in 3–9% of all pregnancies. Studies in selected populations (low-income, predominantly single women) sometimes even show percentages of up to 50% [316].

According to Christensen and Dietz trauma is the most common cause of non-obstetrical maternal deaths [317]. The fact that pregnant patients frequently are injured in accidents means that unborn children also are risk of being injured in utero. Foetal fractures of almost every bone in the body have been described [317].

Isolated intrauterine fractures of the shaft seem to be extremely rare and are hardly ever diagnosed before birth [314, 318].

Wilkinson (1898) was one of the first to describe the occurrence of an isolated intrauterine fracture of the femur [319]. Concerning the circumstances he stated:

- *'Professor Gurlt, who has discussed the subject of intrauterine fractures in an exhaustive paper fortified by cases, published in Berlin in 1857, and later in his classic "Treatise on Fractures," believes that many intra-uterine fractures result from external violence received by the mother during the advanced period of pregnancy'.*

Concerning the occurrence of a femur fracture in his case he stated:

- *'I am unable to assign a positive cause for this fracture. There is a history of an epileptic seizure of the mother in the fourth of fifth month of pregnancy, in which seizure she fell violently over a stove, but aside from the fall no pain followed the accident. Another theory is that the husband, who afterward deserted his wife, owing to a continuance of domestic infidelity, may have abused her, either by striking her or by some other violent means causing the fracture.*
- *My other theory, and the one I wish to call your attention to especially is, that during an epileptic seizure, a compression of great severity produced by an abnormal increase of muscular power of the abdominal muscles would be sufficient to squeeze, as in a vise, the pent-up fetus, which would at that time most likely, be raised up against the abdominal walls, consequently being more exposed to the spasmodic contractions of the muscles or walls of the abdomen'.*

Bucholz and Moulden reported the occurrence of a fracture of the left midshaft in a male foetus due to, what eventually turned out to be after 5 days a fatal car accident of the mother [318]. The boy was born after an emergency caesarean section, but developed multiple medical problems, including seizures, probably caused by neonatal asphyxia. Christensen and Dietz reported the same case and stated that this case report probably was the first that documented radiographically a fracture prior to delivery [317].

Sometimes an intrauterine fracture of the shaft is reported in single case reports without any indication of a skeletal dysplasia or an evident maternal trauma. These fractures often are labelled as spontaneous fractures [315, 320–322]. Forensically seen, the use of the term 'spontaneous' is not correct, better terms would be 'unknown' or 'unexplained'.

Despite the fact that usually it is assumed that a severe maternal trauma is needed, a shaft fracture may probably occur in what is considered to be a less severe or even mild maternal trauma. Alonso et al. described the occurrence in a low-speed frontal collision, while the mother was driving (less than 30 mph) [323]. She was wearing a seat belt, but despite that there was a direct impact onto her abdomen with the steering wheel. She attended the Emergency Department.

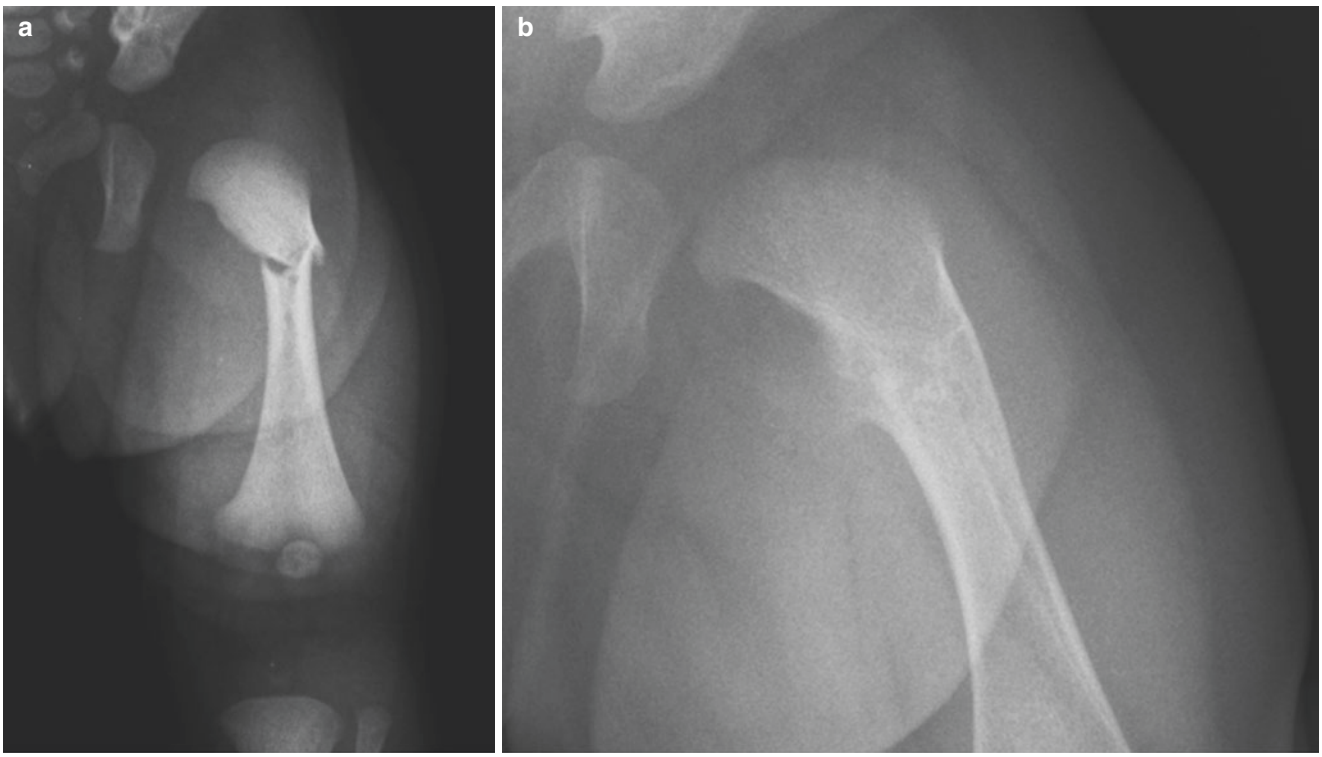


Fig. 12.59 (**a**) One-day-old neonate (birth weight 2215 g) with a femur fracture after a complicated delivery with transverse presentation. (**b**) After 4 months the fracture has healed practically seamless

She had no external injuries. The baby was born 4 weeks later after a normal vaginal delivery. The baby had a hard mass on the right femur. The right femur showed on X-ray an almost united fracture with abundant callus formation with no indication of a skeletal dysplasia.

During Birth

Birth trauma-related fractures of the femur are very rare and may occur both in vaginal deliveries as in caesarean sections [324–326]. (Figs. 12.59a, b and 12.60). Most of these are fractures of the shaft, mainly midshaft. Kancherla et al. evaluated 10 neonates with birth-related shaft fractures, of which 8 were midshaft fractures and 2 subtrochanteric fractures [326]. Frik described four neonates with birth trauma-related femoral subtrochanteric fractures, of which three occurred during a caesarean section [327]. Birth trauma-related femoral shaft fractures may occur bilaterally [328].

Four large epidemiological studies, concerning birth-related fractures in over almost 160,000 neonates showed only 11 fractures of the femur, of which 8 were fractures of the shaft [94, 118, 121, 329]. This would mean an incidence of 0.05 femoral shaft fractures in 1,000 live births. Smaller series show comparable low figures (Table 12.8).

Birth trauma-related fractures of the femoral shaft are associated with shoulder dystocia, caesarean section, twin pregnancies, multiple births, breech presentation, preterm and small for age neonates, osteopenia of prematurity, and osteoporosis (e.g. secondary to copper deficiency) [117, 326, 327, 330]. However, these fractures may also occur in neonates with normal weights, uncomplicated pregnancies, and healthy mothers [330].

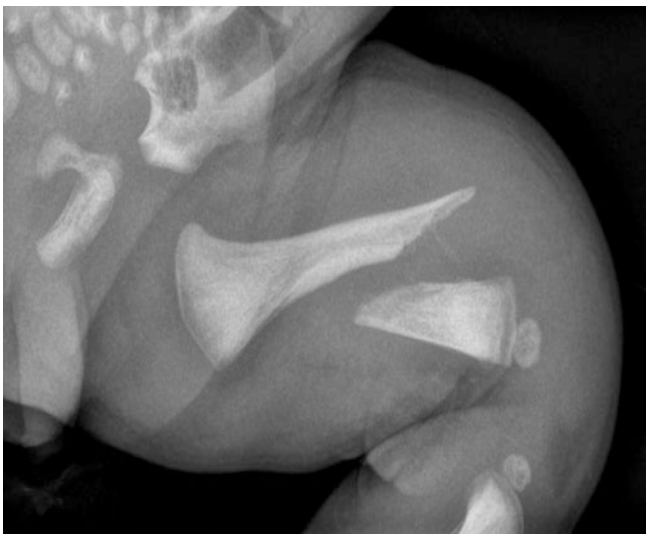


Fig. 12.60 Oblique left femur after a caesarean section

Not all birth trauma-related fractures are immediately identified after birth. A delay in diagnosis up to a few days

Table 12.8 Incidence of birth-related femur fractures

Author	*N*	Incidence per 1000
Bhat [121]	34,946	0.14
Morris [324]	55,296	0.14
Toker—vaginal delivery [325]	184.949	0.03
Toker—caesarean section [325]	38,990	0.31
Basha [120]	34,519	0.17
Kanat Pektaş [468]	31,058	0.32[a]
Rehm [118]	87,461	0.01[b]
Von Heideken [117]	1,855,267	0.024

[a] All caesarean sections
[b] All emergency caesarean section

after birth has been described, even when children are hospitalized [324–326]. Kancherla et al. found a mean delay time of 4 days, before the fracture was diagnosed. Kanai et al. reported a delay in the diagnosis of 9 days in a female neonate with a left spiral femur fracture with associated oedema and hypoaesthesia [331]. The delay was caused by a lack of symptoms and she had a normal physical exam on day 1. On day 1 a whole body X-ray was made which showed on revision a non-displaced fracture of the left femoral shaft. Such a delay may, in theory, lead to a suspicion of non-accidental trauma.

Up to a certain extent, it is possible to differentiate between birth trauma-related femoral shaft fractures and fractures due to trauma after birth (accidental or non-accidental) by evaluating the formation of callus.

Subperiosteal new bone formation and calcification can be found as early as 7 days after birth [332–334]. The absence of subperiosteal new bone formation or callus after 11 days should prompt a physician to consider non-accidental injury [332].

Hosokawa et al. evaluated the findings concerning femoral shaft fractures in 7 neonates without underlying disease. Subperiosteal new bone formation (SPNBF) and callus formation were not detected by day 6 on radiographs. SPNBF was first observed on day 14 (14.29 ± 5.35 days; range 9–23 days), soft callus on day 15 (15.85 ± 4.49 days; rage 10–23 days), and hard callus on day 21 (21.43 ± 5.41 days; range 16–32 days). The 7 neonates without an underlying disease showed SPNBF and soft callus formation by day 23. According to Hosokawa et al., an underlying disease (e.g. osteogenesis imperfecta) may be considered, if SPNBF or callus formation is detected within 6 days after birth. The authors also stated that trauma after birth or an underlying diseases may be considered if SPNBF or callus formation is not detected by day 23.

Based on the data presented above reserve is recommended in excluding birth-related trauma if a femoral shaft fracture is found in a neonate, because of the range in days in first radiologically visible SPNBF, soft and hard callus. Above that, Crompton et al. showed that the subperiosteal new bone formation and callus stages of femoral fracture healing in children under the age of 3 years are slower compared to birth-related clavicular fractures [334]. More research (with larger sample sizes) on this topic is needed.

After Birth: Accidental and Non-accidental Circumstances

The femoral shaft is the most common location in case of femoral fractures. Fractures of the shaft may occur in accidental and in non-accidental circumstances.

In normal bone, a fracture of the shaft usually is caused by a trauma with a high-energy transfer (Sect. 12.7.3.2). After birth this may occur due to a direct blow to the shaft, e.g. in motor vehicle accidents of high-impact sport injuries, or due to an indirect trauma, transmitted at the knee, like in falls from heights and landing on feet [245, 303, 304, 335].

Fractures have also been described to occur due to traditional massage, physiotherapy, or medical procedures, although the occurrence of this type of circumstance is probably extremely rare in paediatric patients (Sect. 12.7.3.3.5). They may also occur due to low-energy trauma, e.g. in diseases with an increased risk of fractures (see Sect. 12.7.3.3.6).

Hinton et al. evaluated the findings of 1,485 paediatric patients under the age of 18 years with acute fractures of the femoral shaft and found that the most common accidental circumstances are age dependent [304]. In children under the age of 6 years falls were the most common accident, in children between 6 to 9 years this was motor vehicle-pedestrian accidents, and in teenagers motor-vehicle accidents. They also found that Firearm-related injuries accounted for 15% of the fractures among black adolescents.

According to Edgington et al. non-accidental trauma should always be considered in young children under the age of 3 years and especially in pre-mobile children [307]. Several large studies describe high rates of non-accidental injury in children under the age of 1 year with a femoral shaft fracture [40, 124, 130, 244, 303, 336–338].

In children of 3 years and older shaft fractures are rarely sustained in non-accidental circumstances since bone at this age is significantly stronger in resisting both torque forces and direct blows [339]. In children of 5 years and older and in adolescents, a shaft fracture is hardly ever the result of non-accidental trauma. The most probable circumstances are a high-energy trauma-related, such as sports trauma or motor vehicle accidents [340–342]. In the United States, shaft fractures increasingly are sustained due to shot wounds [302].

Most studies concerning shaft fractures in paediatric patients compare the findings in children, who sustained the fracture in accidental circumstances, to children, who sustained the fracture in non-accidental circumstances:

- Worlock et al. described data from a retrospective study (inclusion period 1976–1982) on 151 children under the age of 5 years [40]. In their study 116 (76.8%) children

had an accident as the cause of the fracture and 35 (23.2%) a non-accidental cause. The authors do not provide data on the difference between age groups of mobile versus non-mobile infants and children.

- Leventhal et al. described a retrospective study (inclusion period 1979–1983) in children under the age of 3 years who were treated for fractures at the Yale-New Haven Hospital [124]. They found that out of 228 children 26 (11.4%) had a femur fracture. Of these 13 (50%) were sustained in accidental circumstances, in 9 (35%) in non-accidental circumstances, and in 4 (15%) the circumstances were unknown. In children under the age of 12 months, 6 of 10 fractures occurred due to non-accidental trauma and children above the age of 23 months none of 10 fractures were the result of non-accidental trauma.
- Blakemore et al. evaluated data on 42 children aged 1–5 years (mean age 3.1 years) who presented with a femoral fracture between 1979 and 1993 [336]. In this group 16 children were reported to child welfare of which 4 cases went to court. In only 1 case intentional injury was determined to be proven.
- Schwend et al. performed a retrospective analysis in 139 children, under the age of 4 years, with a femoral shaft fracture [337]. In 126 (91%) children the fracture was sustained in accidental circumstances, and in 13 (9%) the circumstances were determined to be non-accidental. The children in the accidental group were slightly older compared to the non-accidental group: 2.4 ± 1.0 versus 1.1 ± 1.0 years. The strongest predictor for non-accidental trauma was the ability to walk, with 10 (42%) out of 24 non-walking children in the non-accidental group versus 3 (2.6%) out of 116 children in the accidental group.
- In a large nationwide study based on the 2000 Healthcare Cost and Utilization Project of Kids' Inpatient Database Loder et al. collected data on 9963 femur fractures [242]. In the vast majority the shaft fracture was sustained either in a fall (35%) (3481 cases, of which 1691 related to sport injuries) or in a motor vehicle accident (33%) (3245 cases). Although the authors not specifically mention the number of fractures, that occurred in non-accidental circumstances, they stated that 15% of all femur fractures under the age of 2 years were inflicted.
- Hui et al. performed a retrospective study in which they included 127 children under the age of 3 years with a femur fracture [338]. Of the 127 femur fractures 14 (11%) were determined to have occurred in non-accidental circumstances. In children under the age of 1 year this was the case in 10 (17%) out of 60 children. According to Hui et al. non-accidental trauma should be excluded in children with a femur fracture under the age of 12 months, non-ambulatory status, delayed presentation, mechanism of injury unwitnessed or inconsistent, and other associated injuries.
- Pandya et al. performed a large retrospective study, in children under the age of 4 years, in an urban level I paediatric trauma centre [130]. In the period 1998–2007, a total of 1485 children, 500 non-accidental (377 <18 months), and 985 accidental (425 <18 months) cases, were included. In the non-accidental group there were 73 (14.6%) femur fractures and in the accidental group 140 (14.2%, $p = 0.85$). In the under 18 months group this was, respectively, 66/377 (17.5%) versus 45/425 (10.6%, $p = 0.057$). Based on their findings the authors concluded that in the under 18 months group '*the odds of femur fracture (1.8 times) were found to be significantly higher in the child abuse group than in the control group*'. For the whole study population the odds ratio for abuse was 1.0 (95% CI: 08–1.4).
- In a retrospective study in children under the age of 4 years with femur fracture Baldwin et al. compared 139 control patients (mean age 26.2 months. IQR 34.8, with 44 infants under the age of 18 months) with 70 cases of non-accidental injury (mean age 4.0 months. IQR 8.3, with 63 infants under the age of 18 months, both age and proportion <18 months $p < 0.001$) [244]. In this study the authors looked at seven risk factors of which three patient characteristics were significant: current polytrauma, physical and/or radiologic evidence of prior trauma, and the history suspicious for abuse. For all three categories there was a significant difference between both groups. Using a multiple logistic regression model the authors calculated odds ratios for the presence of 1, 2, and 3 risk factors these were, respectively, 7.2 (95% CI: 2.2–23.5), 155.5 (95% CI: 41.6–581.0), and 273.0 (95% CI: 28.1–2649.0). Based on a logistic regression equation for each number of risk factors a prediction tool was developed (Fig. 12.57).
- Shrader et al. evaluated the findings in 137 children under the age of 5 years with a femoral shaft fracture (5-year period) (mean age at the time of injury 2.2 years; range 1 month to 4 years [343]. Forty-three children (mean age 1.8 years) (31%) were determined to have injuries suspicious of non-accidental trauma and were referred to Child Protective Services. Shaft fractures in children under the age of 1 year were a highly significant risk factor for suspected non-accidental trauma. Of the 20 children under the age of 1 year, 18 (90%) were referred to Child Protective Services, comprising 42% of those children suspicious of non-accidental trauma.
- Mughal et al. evaluated the findings in 759 paediatric patients, aged from 1 day to 12 years (mean age 4.9 years; median age 3.6 years) with a total of 770 femoral shaft fractures [344]. Eleven patients had bilateral fractures. The most common circumstances were falls (39%) (peak age 2–3 years), followed by motor vehicle accidents (33.7%), of which in 88% pedestrians (peak age 4–5 years)

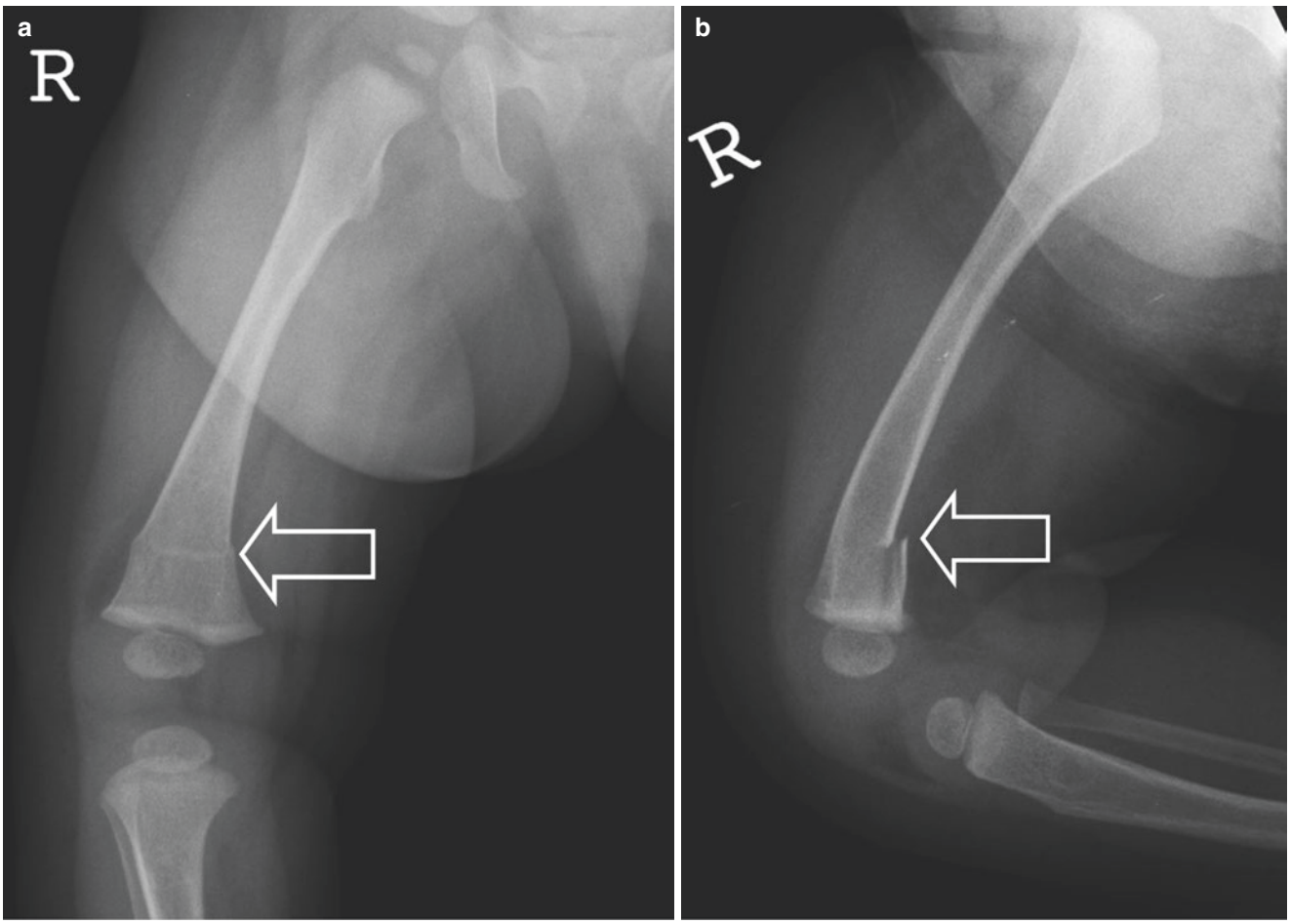


Fig. 12.61 (**a**) Five-month-old girl who had sustained a greenstick fracture of the distal femur (open arrow, A-P view). (**b**) Lateral view of the femur shows a cortical defect (open arrow)

were involved. In children under the age of 1 year, 59.3% were due to non-accidental circumstances. Pathological fractures occurred in 39 patients (5.1%) (Sect. 12.7.3.3.6).

- In a nationwide study of 1,855,267 infants under the age of 12 months, born between 1 January 1997 and 1 December 2004 in Sweden, von Heikeden et al. studied the incidence of femur fractures [117]. From this population 287 infants with a non-birth-related femur fracture were included in the study. Of these fractures 27 (9%) were related to non-accidental trauma, in children under the age of 6 months there were 21 (20%) cases.

Based on the data presented above it is clear that it is imperative that a skeletal survey is performed in non-mobile infants with a shaft fracture without a clear clinical history. This is supported by a study by Cornell et al. which showed that in a series of 19 infants under the age of 12 months who were presented with a femoral fracture 8 (42%) showed occult fractures on the skeletal survey [345].

In young pre-mobile children, an unusual accidental fracture of the distal part of the shaft may occur when a parent falls on the child while the child is carried on the hip of the parent. This can cause a greenstick fracture of the medial distal metaphysis of the femur (Fig. 12.61a, b) due to bowing of the thigh bone, which leads to compression damage to the medial cortex [302].

Ali et al. reported the occurrence of femoral shaft fractures in two boys, aged 4 and 6 years, who were playing in a graveyard, due to a tumbling tombstone [346].

Stress fractures of the femoral shaft (and neck) are uncommon, but are increasingly diagnosed in adolescent athletes participating in sporting activities like soccer, basketball, or athletics. These fractures account for 4% of all stress fractures in paediatric patients [339].

In a few studies femoral fractures in sexually abused children are reported. Hobbs and Wynne found fractures in 5% of a group of 130 sexually abused children, as a sign of physical child abuse [347]. According to this study, however, these fractures are seldom or never the result of sexual acts. In three children they did find fractures resulting from sexual acts. A 5-month-old girl sustained a femoral fracture without dislocation as the result of abuse [348]. Johnson et al report

on a case of a 4-month-old girl who was thought to have been sexually abused and the follow-up skeletal survey revealed a shaft fracture due to sexual abuse [348].

After Birth: Differentiating Accidental from Non-accidental Circumstances

Currently, no association has been found between the morphology of the fracture and distinguishing between accidental or non-accidental injury. The systematic review of Kemp et al. showed that the distribution of transverse, spiral, or oblique fractures do not differ significantly between accidental and non-accidental cases [148]. However, high-quality studies with large sample sizes are lacking.

It is often maintained that a spiral fracture of the shaft of one of the long bones, and in particular the femur, is evidence of child abuse. This is incorrect, it is only possible to evaluate such a fracture when the context of the origin of the fracture is also considered [124, 335, 349, 350]. The only conclusion that can be made with certainty when a spiral fracture of the femur is found in a child is that the fracture is the result or applied torque (rotation along the longitudinal axis of the bone). Torque may occur in non-accidental circumstances (Figs. 12.62 and 12.63). In mobile children torque can occur in accidental circumstances, in which the foot takes a more or less stationary position [124, 125, 147, 337, 351]. The fracture may also occur in a fall in which the knee and hip are more or less stationary, and the child turns the lower leg in relation to the stationary joints. This happens regularly, not just to the femur but also to, e.g. the tibia, as in the 'toddlers' fracture' (Sect. 12.8.3.3).

In a transverse fracture, the fracture line is more or less perpendicular to the long axis of the bone. In an oblique fracture, the fracture line is at an angle of 30–40 degrees to the long axis of the bone. Transverse and oblique fractures may occur due to compression, tension, shearing and bowing, or a combination of these mechanisms (Table 12.1).

Transverse and oblique fractures are frequently seen in accidental and non-accidental circumstances [335]. These fractures may occur as a result of direct blunt force trauma (impact) on the bone (mostly resulting in bowing or shearing, Fig. 12.64), or by indirect trauma, e.g. when a child falls from a significant height and lands on a knee (usually resulting in compression and/or bowing). It may also happen when a parent falls down the stairs while holding the child on an arm and the child lands on the femur (Fig. 12.65) [36]. This is often a trauma with a high-energy transfer.

Oblique fractures are usually the result of a combination of various forms of loading, such as compression with some torque, or compression with bowing [36].

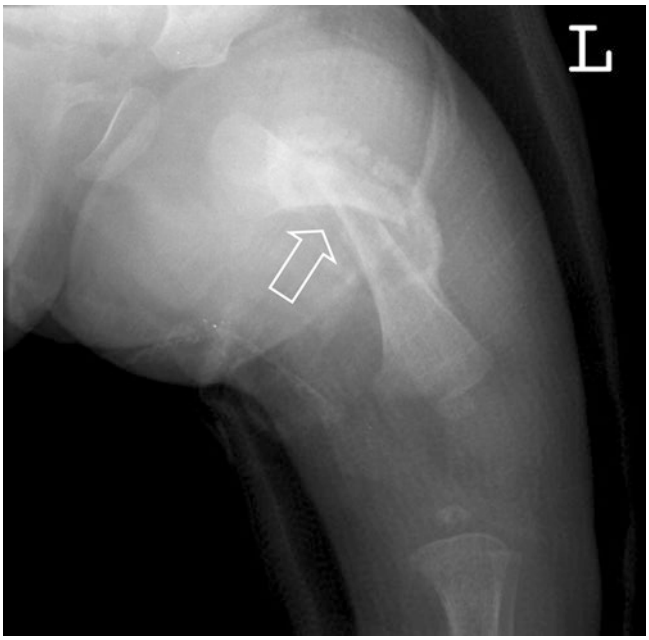


Fig. 12.62 Healing spiral fracture of the left femur (open arrow) in a 3-week-old infant who, according to the parents, had fallen from the couch. The fracture does not correspond with the described biomechanics

Fig. 12.63 Graphic representation of the possible origin of a non-accidental femur fracture

Fig. 12.64 Oblique femur fracture (open arrow) in a 3-year-old boy who had toppled a television (witnessed trauma)

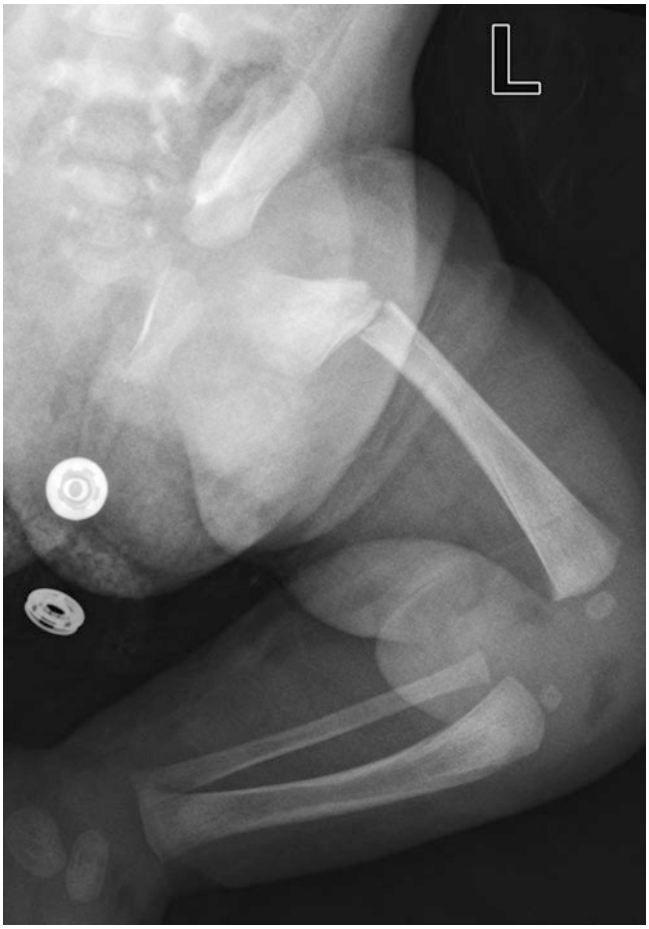


Fig. 12.65 Four-week-old girl who had sustained a proximal femur fracture after a fall from the arm of her mother who tripped over the family dog

While evaluating a shaft fracture, one should realize that it can sometimes be difficult to distinguish radiologically different morphologies, for example a spiral and an oblique fracture, which can look quite similar on different views.

In non-accidental trauma, particularly bowing and shearing are involved. A child may have received a blow or a kick to the upper leg, and the bone bows past the point at which recovery is still possible without a fracture. In younger children, indirectly applied forces may also be involved; for example when a person violently grabs and manipulates the leg, swings the child to and fro, or hits or throws the child against some object [349].

Loos et al. showed that, regardless of working experience, health care professionals in the Netherlands were biased by contextual information towards accidental or non-accidental trauma when assessing images of femur fractures in young children [352]. Context such as low income, single-parent family households, and migrant status may inappropriately influence professionals.

After Birth: Traditional Massage, Physiotherapy, and Medical Procedures

Mboutol-Mandavo et al. reported two neonates with massage-related fractures: a 17-day-old neonate with a midshaft fracture of the right femur and a 1-month-old infant with a fracture of the right clavicle [353]. In the 17-day-old neonate a crack was heard during the massage, after which the child started crying. The massaging was done at home by a grandmother.

Siddiqui et al. described femoral midshaft fractures in 3 neonates, aged 2–3 weeks, following oil massage, which is common practice in India [354]. The massages took place at home 2–3 times a day and were done by a grandmother. All three grandmothers noted that they felt a crack at the time of the oil massage, after which the babies refused to move the affected lower extremity.

Della Grotto et al. reported an 11-day-old neonate, who was admitted to the hospital at day 1 of life [53]. On day 11,

while still in hospital, a swelling of the right leg was noted and on ultrasonography a fracture of the right femoral shaft was diagnosed. Additional radiographs showed an oblique fracture of the right femoral shaft and a metaphyseal corner fracture of the proximal tibia of the same leg. Chart review showed that physiotherapy was provided in the NICU and this was ruled the cause of the fractures.

The medical literature does not report any cases in which a fracture was sustained in a physical examination. However, the authors of this book have been confronted with a 3-day-old neonate with a midshaft femur fracture (Fig. 13.33). According to the mother the child showed pain when she changed the diaper. Patient history and follow-up examination did not show any signs of non-accidental trauma. Postpartum there were no indications for a fracture. On day 3, the paediatric resident performed an examination according to Ortolani. The resident wrote in the dossier that a little snap was heard and that the Ortolani was positive. After this examination the infant showed pain when the diaper was changed. A radiograph of the leg showed a midshaft oblique femur fracture. The successively made skeletal survey did not show any other fractures. The combined facts led to the conclusion that the femur fracture had to be the result of the examination according to Ortolani.

After Birth: Diseases with an Increased Risk of Femoral Shaft Fractures

Pathological fractures of the femoral shaft are relatively rare in children and account for around 4–5% of all paediatric shaft fractures [239, 344]. A pathological fracture is defined as a fracture that occurs without a significant trauma or with a trauma with a seemingly low-energy transfer, usually in pre-existent pathological bone. Fractures of the shaft in a child should alert physicians to a possible underlying disorder, if there is no history of a significant trauma or if there is a history and/or other findings suggestive of non-accidental trauma.

Bone may be 'pathological' due to generalized bone disorders or in disorders with focal manifestations, resulting in an increased fracture risk.

Pathological femoral shaft fractures, due to generalized bone disorders can be seen in children with generalized osteopenia, such as osteogenesis imperfecta (OI) [124, 355]. Other causes of generalized osteopenia in which a fracture of the femoral shaft may occur due to a minor trauma are neurological/neuromuscular disorders, such as cerebral palsy or meningomyocele [239, 356–358]. Ju et al. reported the occurrence of shaft fractures in a 14-year-old boy with undiagnosed cystic fibrosis [359]. While playing baseball he sustained a left midshaft femoral fracture while running. Eight months later, he sustained a right midshaft femoral fracture under similar conditions. After the second fracture, further evaluation revealed low bone mineral density and confirmed the diagnosis of cystic fibrosis.

Osteogenesis imperfecta and cerebral palsy are probably the most common underlying disorders. Children with these disorders sustain femoral shaft fractures most commonly between the age of 6 and 12 years, because, according to Murugappan, they start walking late.

Pathological fractures, due to focal lesions, can be seen in paediatric patients with neoplasms. Usually, these are benign lesions such as non-ossifying fibroma, eosinophilic granuloma (unifocal Langerhans cell histiocytosis), fibrous dysplasia, and bone cysts [360–362]. Pathological femur fractures are seldom seen in paediatric patients with malignant neoplasms, e.g. osteosarcoma or Ewing sarcoma (see Chap. 14) [302, 363].

Pathological fractures, due to focal lesions, can also be seen in paediatric patients with chronic osteomyelitis of the femoral shaft [364, 365].

12.7.4 Fractures of the Distal Femur

12.7.4.1 General Aspects of Fractures of the Distal Femur

Fractures of the distal femur are rare, accounting for only 7% of all fractures of the lower extremity in children. There is a peak incidence between 10 and 12 years. Boy to girl ratio is estimated around 6:1 [366–368]. Distal femur fractures account for approximately 12–19% of all femur fractures in children [242, 301, 369].

Distal femur fractures can be classified as metaphyseal corner fractures, metaphyseal fractures, or physeal fractures [370]:

- Metaphyseal corner fractures (classical metaphyseal lesions, buckle handle fractures) are almost exclusively seen in children under the age of 2 years of age (Sect. 12.3.2).
- Metaphyseal fractures of the distal femur (transverse distal metaphyseal fractures, supracondylar femoral fractures) are the most common type of distal femur fracture (excluding MCF) in infants and young children (Fig. 12.66a, b) [370, 371]. These can be complete or incomplete (greenstick or torus) fractures.
- Physeal fractures of the distal femur are more common in older children and adolescents (Fig. 12.67a, b). These fractures account for around 7% of fractures of the lower extremity and under 1% of all paediatric fractures [372]. Physeal fractures are classified according to the Salter–Harris classification (Sect. 12.3.3) [370]. SH-type II fracture is most common [373, 374]. The epiphysis of the distal femur is particularly prone to growth disturbance

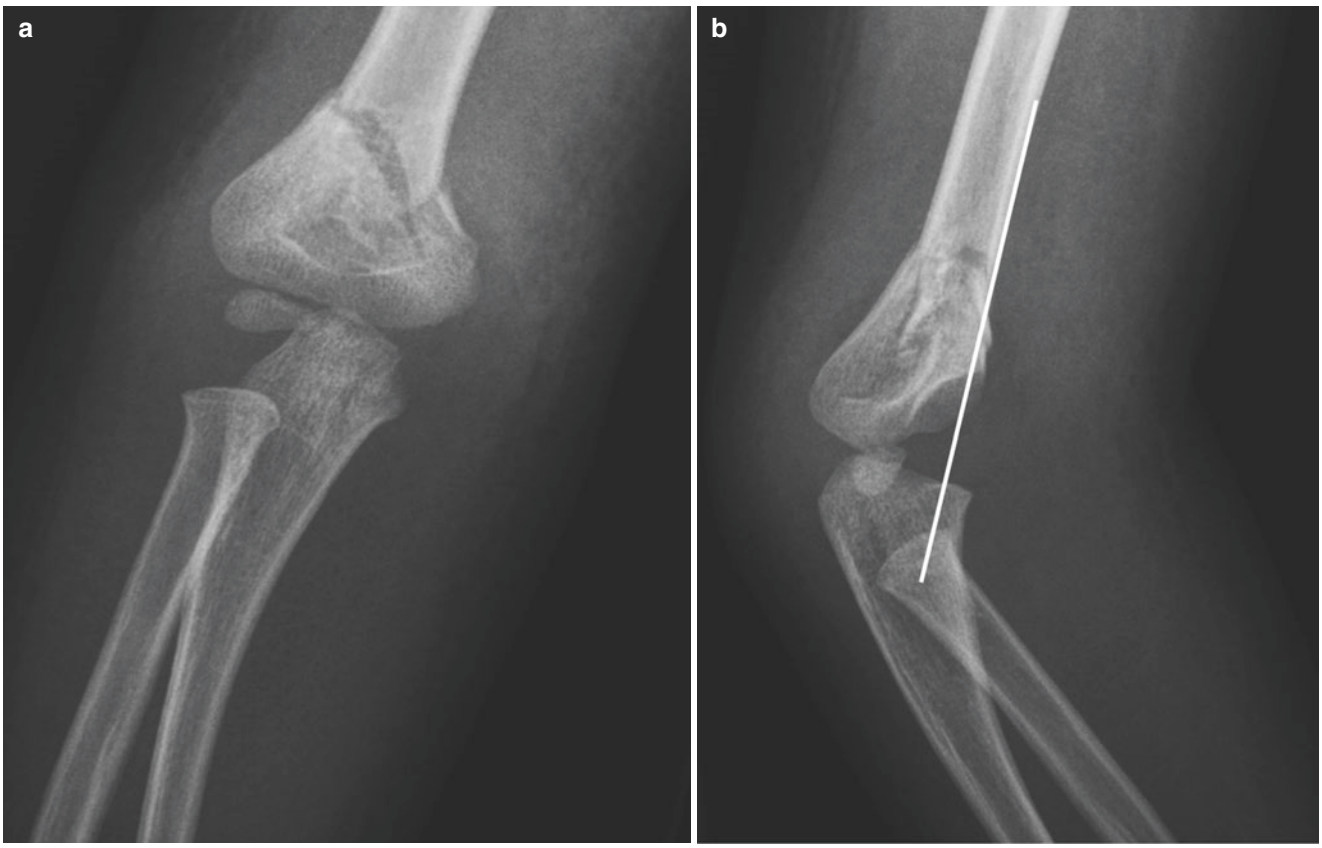


Fig. 12.66 Three-year-old child who fell from a climbing frame. (**a**) AP radiograph shows a supracondylar fracture. (**b**) Lateral radiograph shows posterior displacement of the distal fracture segment. The anterior humeral line (in white) drawn on a lateral view along the anterior surface of the humerus should pass through the middle third of the capitellum. This is clearly not the case in this child

due to a fracture, as this is responsible for approximately 70% of the total growth of the femur [375].

12.7.4.2 Cause of Fractures of the Distal Femur

'Older practioners will remember that this injury commonly happened to some young person who was attempting to 'hook a ride' by scrambling over the rear dashboard of a high-wheeled horse-drawn vehicle. In swinging his leg over the dashboard, the victim caught his foot in the slowly turning spokes of the wheel. His pelvis being fixed on the dashboard and the foot being fixed in the turning wheel, there resulted a twist with hyperextension of the knee; these factors caused the injury under discussion' [376].

As long as the growth plates of the distal femur in children and adolescents are open, the distal femoral epiphysis will be less resistant to trauma and therefore more prone to sustain injuries than the knee ligaments. This vulnerability is further increased because of the undulating shape of the distal femur [373].

Fractures of the distal femur are the result of a significant trauma with a high transfer of energy, as may occur in motor vehicle accidents, falls from height, or contact sports. Two mechanisms are most common [368, 374, 377, 378]:

- A direct blow to the knee joint either from the lateral or the medial side (side impact), causes a valgus or varus bending effect across the joint and on the collateral ligaments. Due to this effect disruption of the ligaments may occur on one side, while compression of bone may occur on the other side. In children with open growth plates, the tensioning of the ligaments at the attachment to the distal femoral epiphysis may result in failure of the bone, eventually leading to fractures of the distal femur. In adolescents, this trauma mechanism can result in the so-called 'unhappy triad' consisting of a rupture of the anterior cruciate ligament (ACL), a medial meniscus tear, and a rupture of the tibial (medial) collateral ligament.
- An indirect trauma due to lateral or medial distortion of the joint, causing a valgus or varus bending effect across

Fig. 12.67 Nine-year-old paraplegic child with a swollen leg. (**a**) Radiography shows an osteopenic femur with florid subperiosteal new bone formation along the diaphysis and distal metaphysis. (**b**) Lateral radiograph shows a SH-I fracture of the distal femur (inset). Most likely the fracture resulted from physical therapy

the joint and on the collateral ligaments, with comparable consequences as in a direct blow. Indirect trauma may occur in landing on the feet after a fall from height.

Often the bending effect is increased by some degree of rotation/torsion due to twisting the knee on the stable foot.

12.7.4.3 Manner of Fractures of the Distal Femur

Before Birth

Gowda et al. described the occurrence of a MCF of the distal femur. According to the authors this MCF was sustained in utero due to external cephalic version for a breech presentation with the hips flexed and knees extended [46]. The foetus was successfully manoeuvred into a cephalic presentation, but because of a blood-stained discharge from the cervix, an emergency caesarean section was done.

During Birth

Injuries related to the femur as a consequence of birth-related trauma are usually found in the shaft and the proximal physis. Few studies have been published on birth-related injuries of the distal femur [71, 72, 109, 379–383]. As for other birth-related long bone fractures, often a complicated vaginal delivery (often in high-birth weight neonates or in breech presentation) or a caesarean section (often secondary to breech presentation) is reported [384].

Eliahou et al. reported a premature neonate with a SH-type 1 fracture of the left distal femur following caesarean section [72]. According to Eliahou et al., the presumed mechanism in caesarean sections is forced traction of the leg with acute angulation or twisting during birth.

Alexander et al. reported the occurrence of metaphyseal fractures in 2 female neonates [385]. The first was born at term after caesarean section because of an extended breech delivery without any progress. She weighed 3.3 kg at birth and was diagnosed on day 6 with metaphyseal fracture of the left distal femur and of the left upper tibia. Skeletal survey otherwise was normal. The second girl was born after caesarean section because of a flexed breech presentation in a primigravida. She weighed 4.14 kg. On day 2 a distal metaphyseal fracture of the right femur was diagnosed.

Bilateral birth-related distal femoral epiphyseal fractures have been reported [386].

Birth-related metaphyseal corner fractures of the distal femur are only very rarely reported. Sieswerda et al. reported the occurrence of an MCF in the distal right femur in a male neonate, born after attempted external version (ECV) and vaginal breech birth [45].

After Birth: Accidental or Non-accidental Trauma

Fractures of the distal femur can occur in accidental and in non-accidental circumstances [40, 244, 387–389].

Metaphyseal and physeal fractures of the distal femur have been described to occur in accidental and in non-accidental circumstances. Metaphyseal fractures (excluding MCF) are the most common type of distal femur fracture in infants and young children, whereas physeal fractures are more common in older children and adolescents.

Fractures of the distal femur usually are sustained due to high-energy trauma, e.g. falls, motor vehicle accidents, or sports-related activities [368]. This especially accounts for children aged 2–11 years [372].

Rex and Kay evaluated the findings, concerning age, site, and fracture patterns, in 14 children with non-accidental femur fractures and compared these with the findings in 33 children with accidental femur fractures. Thirteen of the children with non-accidental fractures of the femur were under the age of 12 months [388]. The authors could not find any specific site or fracture pattern that could allow differentiation between accidental and non-accidental fractures of the femur.

Rewers et al. evaluated epidemiological data concerning femur fractures in 1139 paediatric patients, aged 0–17 years (795 boys and 344 girls) [301]. The most frequent location of femoral fractures was the shaft in 62.5%, followed by the proximal (12.5%) and the distal (11.7%) femur. Almost 1 in 8 fractures involved the shaft in combination with a proximal or distal fracture. Fractures of the shaft, due to non-accidental trauma, were relatively less common, whereas distal fractures and combinations of shaft and distal fractures were more common, compared to fractures due to accidental trauma. Associated injuries were found in 28.6% of the children, more often in older children. Children who sustained femur fractures due to non-accidental trauma, motor vehicle accidents, or car versus pedestrian accidents were 16–20 times more likely to have associated injuries than those with femur fractures as a result of a fall.

Baldwin et al. evaluated the findings in 70 paediatric patients with non-accidental femur fractures and compared these with the findings in 139 paediatric patients with accidental femur fractures [244]. Patients from the accidental group more often had shaft fractures and patients from the non-accidental group more often had fractures of the distal femur. No difference was found between both groups concerning fractures of the proximal femur. The risk of femur fractures due to non-accidental trauma was highest in children under the age of 18 months, in girls (girls to boys ratio 2:1), in polytrauma patients, physical, and/or radiographic evidence of prior trauma and in case of a suspicious history.

In children under the age of 1 year non-accidental trauma should always be considered in case of a fracture of the distal femur. Arkader et al. evaluated the findings in 29 children with complete metaphyseal fractures of the distal femur (two level 1 paediatric trauma centres; 10-year period) [389]. 20 fractures occurred in non-ambulatory infants under the age of 1 year (14 boys, 6 girls; average age 6 months 10 days, with a range of 5 days to 1 year). Non-accidental circumstances were considered confirmed in 10 children and highly suspicious in 5 children. The authors' advice that in all non-ambulatory infants non-accidental circumstances should always be considered.

Nevertheless, a careful analysis of the medical history, concerning accidental circumstances, is always indicated, in children with distal femur fractures. This also accounts for non-ambulatory infants:

- Grant et al. described the finding of identical oblique distal femoral metaphyseal fractures extending through the growth plate in two non-ambulatory infants [390]. The fractures supposedly occurred while playing in an infant stationary activity centre (Exersaucer, Sect. 13.3.5.7). According to the authors, the twisting motion provided by the Exersaucer might have generated enough force to cause the fractures.
- Haney et al. evaluated the findings in 18 children with transverse fractures of the distal femoral metadiaphysis [387]. In 13 children (11 under the age of 1 year; mean age 12 months; median age 8 months) the circumstances were determined to be accidental and in five children (all 5 under the age of 1 year; mean age 8 months; median age 8 months) non-accidental. The authors concluded that impacted transverse fractures of the distal femoral metadiaphysis may occur as a result of accidental short falls of young children. They also were of the opinion that a 'traditional abuse evaluation' should be done in all cases, but that in the absence of additional skeletal findings, and a history of a fall, accidental circumstance likely accounts for the occurrence of the fracture.

Fractures of the distal femur may occur due to physiotherapy. Pickett et al. described an ex-premature infant (pregnancy 33 weeks; birth weight 2077 g) in whom multiple defects to both legs were found at age 4 weeks: extensive periosteal reactions around the knees combined with 'bucket-handle' fractures of both proximal tibiae [391]. Diaphyseal periosteal new bone formation and metaphyseal fragmentation of both tibiae were present. Diametaphyseal periosteal new bone of the distal end of the left femur was present. The proximal medial femoral metaphyses had corner fractures. The osseous lesions appeared to be limited to joints receiving physical therapy for contractures.

After Birth: Diseases with an Increased Risk of Fractures of the Distal Femur

Underlying disorders may predispose children to fractures of the distal femur [378]. This has been described in children with spastic cerebral palsy, neonatal osteomyelitis and septic arthritis, and spina bifida (Fig. 12.68) [392–394]. In these children fractures may occur due to low-energy trauma, e.g. in 'twisting' the leg during physical therapy or while changing diapers or changing the child's position in bed. Because of disuse osteopenia non-ambulatory children, e.g. with cerebral palsy or spina bifida, are susceptible to fractures due to low-energy trauma. Ambulatory children with spina bifida may develop epiphysiolysis, or a chronic separation of the distal femoral physis, and be unaware of it because of altered sensation.

Vander Have et al. described three patients who developed knee stiffness after operative treatment for displaced tibial eminence fractures. The stiffness was treated with manipulation of the knee under anaesthesia [395]. Due to the manipulation the patients sustained distal femoral fractures with subsequent growth arrest.

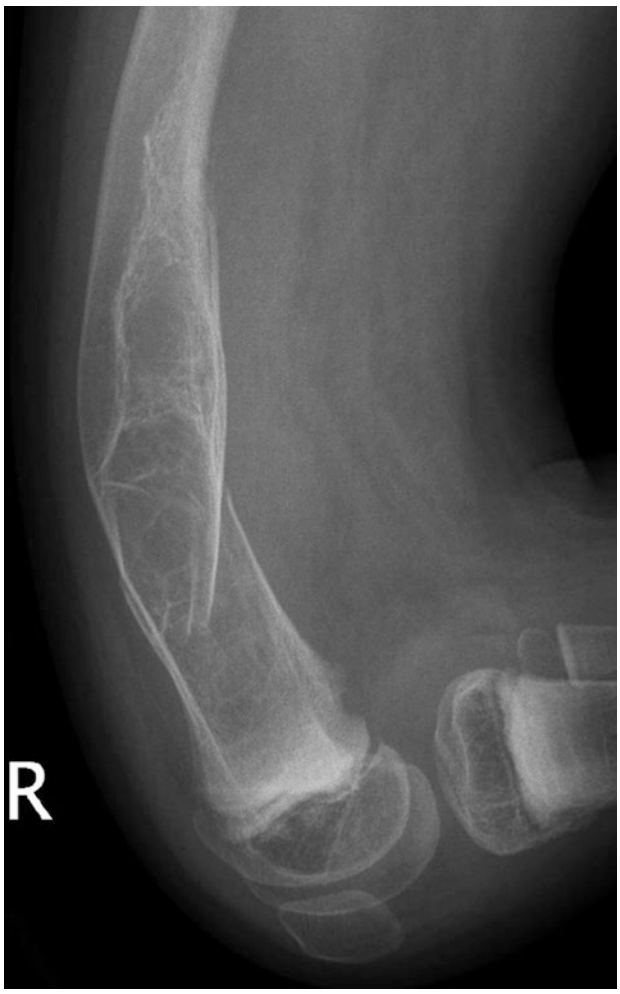


Fig. 12.68 Ten-year-old paraplegic child with spina bifida. After a puppy jumped on her knee she had a slightly swollen left upper leg. Radiography showed a fracture through a severely osteopenic distal femur

12.8 Tibia and Fibula

12.8.1 General Aspects of Fractures of the Tibia and Fibula

Tibial fractures are the third most common fractures in childhood, after fractures of the forearm (distal radius and shaft) and the humerus [396]. These fractures occur most frequently in early mobile and older children and in adolescents. In infants fractures of the lower leg are rare. Fractures of the tibia consist of approximately 15% of all paediatric fractures [308], with a yearly incidence of 11 per 1000 children [397]. The average age of occurrence is 8 years. They are more common in boys than in girls [398, 399].

Almost 40% of all tibia fractures are midshaft fractures. Most of these shaft fractures are oblique or transverse and located in the middle or lower third of the shaft, although spiral fractures are regularly found in toddlers (Childhood Accidental Spiral Tibia fractures, Sect. 12.7.3.2). Paediatric patients under the age of 4 years with lower leg fractures most often showed simple oblique fractures of the tibia [400].

About 30% of the tibia shaft fractures are associated with fibular fractures [398]. Isolated tibial fractures with an intact fibula have a lower risk for shortening, but may pose a risk for varus deformity [401, 402]. Isolated fibula fractures are rare [403].

12.8.2 Cause of Fractures of the Tibia and Fibula

The cause of fractures of the lower leg can be divided into low- and high-energy trauma. According to Chapman and Cohen the cause of lower leg fractures varies depending on the age of the patient. In younger children low energy trauma, e.g. a rotational force due to twisting of the lower leg or falls

from standing height, will be the more common cause. In older children and in adolescents high-energy trauma will be more common, e.g. due to a direct blow to the lower leg that is perpendicular to the bone shaft (pedestrian versus car accidents) or a long-distance fall (Table 12.9) [397].

Table 12.9 Cause of fractures of the lower leg [78, 469–474]

	Cause
Tibia fractures	
• Tibial plateau fracture	Axial loading with valgus or varus forces (e.g. fall from a height or collision with the bumper of a car)
• Tibial spine (intercondylar eminence) fracture	Most often in children aged 8–14 years but may occur in a skeletally mature patients. • Rapid deceleration or hyperextension and/or rotation of the flexed knee, as in sports, often combined with trauma to the distal femur (e.g. falling off a bicycle or during sports)
• Tibial tubercle fracture	Usually following an active quadriceps extension with knee flexed during jumping or sprinting activities such as basketball, diving, football, and gymnastics More common in adolescents than in adults
• Proximal tibial metaphyseal fracture (Cozen's fracture)	Low energy trauma in children, aged 3–6 years: • Valgus force across the knee creating incomplete fracture of proximal tibia and/or torsional force (e.g. a child going down a slide in the lap of an adult with leg extended and the leg caught on the way down) • Often resulting in greenstick fractures with an intact lateral cortex, or in complete fractures
• Tibial shaft fracture	Low energy trauma in younger often pre-school children ('Toddler's fracture'): • Indirect trauma (e.g. falls from standing height) and/or a torsional trauma (twisting) (e.g. when the child's body rotates around a fixed foot, often resulting in a spiral or oblique fracture) High-energy trauma in older children and adolescents (may involve tibia and fibula): • Direct trauma: direct blow to the lower leg that is perpendicular to the bone shaft, usually resulting in a transverse fracture (e.g. pedestrian vs car) • Indirect trauma: long-distance falls
• Tibial plafond fracture	Most common: • High-energy trauma with axial loading (e.g. in falls from height or motor vehicle accidents) Less common: • Low-energy trauma due to rotational forces (e.g. twisting the ankle during skiing)
• Tibial stress fracture	Repetitive submaximal stress, e.g. during athletics
• Open tibia fracture	Usually a high-energy trauma
Epiphyseal transitional fractures around the ankle (Sect. 12.3.3)	
• Tillaux fracture	In adolescents within 1 year prior to physeal closure: • Supination, combined with external rotation force around the ankle
• Triplanar fracture	In slightly younger adolescents than in the Tillaux fracture: • Lateral triplanar fracture: supination, combined with external rotation around the ankle (twisting) • Medial triplanar fracture: adduction, combined with external rotation (twisting)
Fibula fractures	
• Fibula shaft fractures	Usually high-energy trauma, usually midshaft fractures: • Direct trauma: direct blow to the outer aspect of the lower leg • Indirect trauma: landing on heels after a high-distance fall/jump Sometimes low-energy trauma: • Rolling or spraining of the ankle, which stresses the fibula.
• Fibula stress fractures	Repetitive submaximal stress, e.g. during athletics
• Fracture of the lateral malleolus	Twisting or bending of the ankle. The inner side of the ankle is unaffected
• Bimalleolar ankle fracture	The ligaments connecting the ankle and fibula are injured and the resulting stress on the fibula causes a fracture
Combined tibia-fibula fractures	
	Usually high-energy trauma: • Direct trauma: direct blow to the lower leg that is perpendicular to the bone shaft • Indirect trauma: long-distance falls

12.8.3 Manner of Fractures of the Tibia and Fibula

Fractures of the lower leg (tibia and fibula), including MCF, have been described to occur before, during, or after birth. If sustained after birth, lower leg fractures can occur due to accidental and non-accidental circumstances.

12.8.3.1 Before Birth

Lysack et al. described the occurrence of a MCF in an otherwise healthy newborn in the proximal tibia. The MCF was thought to have occurred due to an external cephalic version for a frank breech presentation, followed by an emergency caesarean section [47].

12.8.3.2 During Birth

Tibial fractures have only sporadically been found as a consequence of birth-related trauma in epidemiological studies [47, 118, 404]:

- Basha et al. evaluated the findings in a total of 34 519 live births [120]. Long-bone fractures were found in 8 neonates. In one neonate, born after an emergency caesarean delivery due to breech presentation, a transverse non-displaced right tibial shaft fracture was found. The child also had a midshaft spiral fracture of the right femur and

an old fracture of the left femur. A diagnosis of osteogenesis imperfecta was made.
- Dolivet et al. reviewed the findings in 6840 neonates, born after caesarean section (after exclusion of findings in multiple pregnancies and in caesarean sections before 32 weeks) [405]. They found 10 neonates with at least one fracture. One newborn had a fracture of tibia and fibula. The infant was born after a scheduled section because of breech presentation and macrosomia (birth weight of 3510 g).
- Rehm et al. reviewed the findings in 87,461 consecutive live births. In 66 newborns a fracture was found, of which only one newborn had a tibia fracture [118].

Some descriptions of tibial fractures, sustained during birth, are case based:

- Kaplan et al. described a term female neonate, delivered by caesarean section [404]. At the age of 1 week, while the girl was still hospitalized, a swelling was noted over the lower third of the infant's left tibia with local tenderness and erythema. Radiographs showed an oblique fracture of the midshaft of the left tibia, and a greenstick fracture at the distal end of the right radius, adjacent to the epiphyseal plate. The authors stated that fractures most likely occurred during the caesarean section.
- Mileto et al. described the occurrence of a proximal epiphyseal fracture of the right tibia in a newborn following caesarean section [406]. The birth was complicated by a failure to progress after the membranes had been ruptured for 24 h. Four attempts at vacuum assistance were made prior to performing an emergency caesarean section. The newborn showed swelling and bruising of the right lower leg

The occurrence of MCF due to birth trauma probably is extremely rare, only one case report was found. [49]. Lee et al. describe a MCF of the distal tibia that occurred after an urgent and difficult footling breech delivery [49]. According to the authors, their case shows that the traction and torque placed on the distal extremities during this difficult delivery could be a potential mechanism for the occurrence of a MCF.

12.8.3.3 After Birth: Accidental Circumstances

Accidental fractures of the tibia and fibula are very rare in pre-mobile children and have only been reported as case reports:

- Moineau and Plint described a case of a 9-month-old boy who presented with bilateral buckle fractures of the proximal tibia [407]. Although the authors concluded that in their case the circumstances, under which the fractures were sustained, remained unknown they stated in the discussion of the case *'the parents could not think of, and the babysitter did not admit to, any potential traumatic event while in their care. When reviewing any possible repetitive stresses occurring on his lower limbs, the parents admitted that he was often in his baby stationary activity center, and the sitter had mentioned that he had been in it for a few hours the day he seemed more irritable'*. It thus seems plausible that there is a relation between the use of the exersaucer and the occurrence of fractures.
- Paddock et al. reported the finding of accidental bilateral fibular fractures in a pre-mobile boy, aged 6 months [408]. The parents reported that the infant repeatedly banged his legs against the metal frame of his playpen. The parents videotaped the 'banging', which showed that (according to the instructed radiology expert) the point of impact of the infant's legs against the metal frame was at a similar level to the radiographic abnormalities. The videotaped mechanism was therefore believed to be consistent with the injuries, resulting in a diagnosis of 'self-inflicted' bilateral fibular fractures and not of inflicted injury.

Accidental tibial fractures are very commonly reported in mobile children. Probably the most common accidental tibial fractures in mobile children, usually under the age of 8 years, are isolated spiral fractures of the tibia. These fractures are usually the result of a (minor) accident such as a fall while walking/running or a fall in which the child's body rotates around a fixed foot, often resulting in a spiral or oblique fracture [409–412]. Often these minor accidents are unwitnessed, which can cause concern for non-accidental injury. In most cases the fracture is a non- or minimally dislocated fracture of the lower two-third of the tibia. These fractures were previously referred to as a toddler's fracture, however, the term Childhood Accidental Spiral Tibia fractures (CAST) is now preferred because this type of fracture not only occurs in toddlers (Fig. 12.69) [409].

In mobile children and in adolescents simultaneous fractures of the tibia and fibula are usually seen in accidents (Fig. 12.70). Fractures of tibia and fibula may also occur when the child is seated on the backseat of a bike (usually a bike of one of the parents) and the foot gets caught between the frame and the spokes of the wheel (Figs. 12.71 and 12.72) [413–415]. These easily avoidable injuries are known as 'spokes' injuries and unfortunately, at least in the Netherlands with many cycling parents, these are seen on a regular basis.

Other 'accidental' circumstances, resulting in tibial fractures, which are not often reported in the literature, are (see Chap. 13 for additional information):

- Tibia fracture due to a fall out of bed, crib/cot, or chair, while in hospital (falling distance 30–100 cm) (Sect. 13.3.2).

Fig. 12.69 Childhood accidental spiral tibia fracture in a 22-month-old boy

Fig. 12.70 Distal fracture of tibia and fibula in a 4-year-old boy after high-energy trauma, car vs pedestrian (radiograph was taken in a vacuum splint)

Fig. 12.71 Graphic representation of a spoke injury

Fig. 12.72 Spoke injury in a 4-month-old girl who was seated at the back of her mother's bike. The trauma resulted in an oblique fracture of the tibia (open arrow) and a Salter–Harris type II fracture of the fibula (arrow)

Fig. 12.73 Two-year old who fell on a trampoline. Radiography shows transverse fractures of the distal tibia and fibula

- Trampoline related tibial fractures (Fig. 12.73) (Sect. 13.3.5.11).
- Iatrogenic tibial fractures after the use of an intra-osseous vascular access needle (Sect. 13.4.2.2).
- MCF of the tibia, during IV line placement [52].
- Iatrogenic distal tibia/fibula fractures, including metaphyseal corner fractures, due to orthopaedic surgery in clubfoot (Sect. 13.4.2.3).
- Physiotherapy-related tibial fractures (Sect. 13.4.2.4) [391].
- Stress injuries (Sect. 13.5.2.2).

12.8.3.4 After Birth: Non-accidental Circumstances

Tibia fractures occur frequently in non-accidental trauma. In several studies describing a series of children with non-accidental fractures, the tibia is one of the most commonly affected bones:

- Worlock et al. compared the findings in 35 children (28 children under the age of 18 months; 7 children between 19 and 60 months; 0 children above the age of 60 months) with non-accidental fractures to the findings in 826 children (19 children under the age of 18 months; 97 children between 19 and 60 months; 710 children above the age of 60 months) with accidental fractures [40]. Worlock et al. found non-accidental lower leg fractures only in children under the age of 18 months, including 7 tibial metaphyseal corner fractures (5 proximal, 2 distal) and 5 tibial shaft fractures (1 spiral fracture of the tibia, 2 tibial periosteal reactions, and 2 tibial greenstick fractures).
- King et al. evaluated the findings in 750 children of whom 189 children (age range 1 month to 13 years; median age 7 months) with a total of 429 fractures were considered to have sustained these fractures in non-accidental trauma [96]. They found that fractures of humerus, femur, and tibia were the most common non-accidental fractures and that 26% of the children with non-accidental fractures had non-accidental tibial fractures. Avulsion or metaphyseal corner fractures involving the proximal third of the tibia were most common tibial fractures. Twenty-eight percent of the children had a history of previous fractures.
- Mellick et al. reviewed 31 tibial fractures in 30 children under the age of 5 years (23 boys, 7 girls; age range from 2 months to 4 years and 10 months; 50% were younger than 36 months) [416]. Non-accidental trauma was suspected in 13 children. The suspicion was confirmed in 7 children. 3 out of 7 children had no other fractures. In only 1 out of 13 children with isolated spiral fractures the fracture occurred in non-accidental circumstances.
- In 1990, Mellick and Reesor published findings in probably the same 13 children with isolated spiral tibial fractures, as published by Mellick et al. in 1988 [410]. Of these 13 children, 9 were classified as accidental fractures and 4 were classified as non-accidental fractures. The circumstances in the 4 children with, according to Mellick and Reesor, non-accidental fractures were described as:
  - Leg twisted by caretaker in a 9-month-old boy: The parents initially had no explanation for the injury, which they first noticed after the infant was picked up from the babysitter. Shortly afterwards, the babysitter admitted to grabbing and twisting the extremity after becoming angry with the child.
  - 'Slipped of lap' in a 2-month-old girl: The slipping allegedly occurred while a parent was placing the child into a sitting position on the floor. A subsequent hospital visit, examination, and admission demonstrated rib fractures and bruises on the infant's back.
  - Fell from bed while playing with a 4-year-old brother in a 19-month-old boy: The boy fell a distance of 3 feet from the bed to the floor. When interviewed alone the older brother gave the same explanation for the injury as was presented by the parents. Although no additional evidence for non-accidental trauma was obtained, it was concluded by a child abuse evaluation

that '*the suspicion of child abuse cannot be ruled out due to the nature of the fracture*'.
  - 'Tripped over dog chain' in a 17-month-old boy: The actual fall was reportedly not observed by either parent. The boy was presented for medical care, 3 days after the reported fall. Because of the delay in seeking medical care and inconsistencies in the history, the fracture was designated to be consistent with non-accidental trauma.

  In 3 of these 4 children non-accidental trauma either was confirmed or could not be excluded on plausible grounds. In the 19-month-old boy it is dubious whether non-accidental trauma was maintained as most plausible manner.
- In a third study, Mellick et al. reviewed the data concerning isolated spiral tibial fractures in 55 children under the age of 8 years (age range 12–94 months; mean age 50.7 months; 69% male, 31% female) [409]. In 10 children non-accidental trauma was suspected. In none of these children the suspicion was confirmed after an evaluation by the child protection service.
- Kowal-Vern et al. evaluated the findings in 124 children with fractures under the age of 3 years to determine the frequency of accidental (motor vehicle accidents, pedestrian accidents, other accidents) versus non-accidental trauma [126]. Fractures due to non-accidental trauma were found in 24 children. The authors found that in only 1 of 8 children with lower leg fractures the fractures were sustained due to non-accidental trauma.
- Leventhal et al. evaluated the findings in 215 children under the age of 3 years with a total of 253 fractures [124]. The fractures were sustained in 24.2% in non-accidental circumstances and in 67.4% in accidental circumstances. In 8.4% the circumstances were not known. Concerning lower leg fractures ($n = 35$) they found that 14 (40%) were due to non-accidental trauma and 21 (60%) to accidental trauma. According to the author's non-accidental trauma should be suspected in a child under the age of 1 year with a fracture of the lower leg.
- Banaszkiewicz et al. reviewed the medical records of all children, under the age of 1 year of age presenting to an Emergency Department over a 5-year period (1995–1999) with a fracture [417]. Seventy-four children presented with fractures (age range 2 weeks to 1 year; mean age 5 months). 5 children had a tibial fracture. The authors stated that in 1 child the fracture was definitely sustained in non-accidental circumstances and in 1 child likely. In 1 child it was suspected but not confirmed.
- Coffey et al. found 55 fractures of the lower extremities in 555 children under the age of 18 months [418]. Of these 55 cases 41 were linked to non-accidental trauma. Femur fractures were most common (22 unilateral and 6 bilateral, followed by tibia fractures (14 unilateral and 9 bilateral). Fourteen cases were not linked to non-accidental trauma, 13 femur fractures (12 unilateral and 1 bilateral), and 1 tibia fracture. In other words, Coffey et al. found that 96% (23/24) of all tibial fractures in children under the age of 18 months were due to non-accidental trauma.
- Loder et al. reviewed the findings in 1794 patients under the age of 20 years with injuries due to non-accidental trauma [204]. They found a total of 1053 fractures, of which 119 were fractures of tibia and/or fibula, and/or ankle. Of these 98 were found in children under the age of 1 year, 15 between 1 and 2 years, 3 between 3 and 12 years, and 3 between 13 and 20 years.
- Van As et al. evaluated the physical findings in 1037 children between 1 month and 13 years (median age 16.5 months, average age 44.8 months; male to female ratio 2:1) with injuries due to non-accidental trauma [205]. Of these children 121 had a total of 149 fractures (21 had multiple fractures). Eleven children had fractures of the tibia/fibula.
- Pandya et al. did a large retrospective study, in children under the age of 4 years, in an urban level I paediatric trauma centre [130]. In the period 1998–2007 a total of 1485 children, 500 non-accidental (377 <18 months), and 985 accidental (425 <18 months) cases, were included. In the non-accidental group there were 55 (11.0%) tibia/fibula fractures and in the accidental group 16 (1.6%, $p < 0.001$). In the under 18 months group this was, respectively, 50 (13.3%) versus 5 (1.2%, $p < 0.001$). Based on their findings the authors concluded that in the under 18 months group '*the odds of a humerus fracture (7.5 times) were found to be significantly higher in the child abuse group than in the control group*'. For the whole study population the odds ratio for abuse was 7.5 (95% CI: 4.2–13.2).
- Eren et al. described 16 non-accidental fractures in a premobile 7-month-old girl [419]. Of these 16 fractures 3 were tibial fractures (distal and proximal fractures of right tibia and shaft fracture of the left tibia) and one was a fibular fracture (distal fracture of the right fibula).

Based on the findings in the foregoing literature one can conclude the following concerning fractures of the lower leg:

- Non-accidental tibial shaft fractures probably are less common than tibial metaphyseal corner fractures or fractures of the apophysis of the proximal tibia (apophyseal ring fractures) [40, 96]. Concerning the meaning of tibial metaphyseal corner fractures the reader is referred to Sect. 12.3.2.
- Despite the rarity of non-accidental tibial shaft fractures one should always consider non-accidental trauma in non-mobile children/children who do not (yet) walk [409, 418].

- One should also consider non-accidental circumstances if the explanation of how the fracture occurred does not match the known trauma mechanism(s) (inconsistent history) or when other physical findings are found, which are suggestive of non-accidental circumstances (concomitant injuries) [398, 420].
- Literature on the association between the type of fracture in the shaft of the tibia (spiral, oblique, or transverse) in association with non-accidental trauma is currently lacking.
- Literature on fibula fractures in association with non-accidental trauma is very scarce [124, 421, 422]. Compared to tibia fractures, fractures of the fibula are only rarely reported [124, 130]. Usually, a simultaneous fracture of the tibia is seen.

12.9 Fractures of the Foot

12.9.1 General Aspects of Fractures of the Foot

Fractures of the foot account for 5–13% of all paediatric fractures [423]. These fractures are rare in infants and toddlers, but the incidence increases with age [424]. In children fractures of the foot are more common in boys than in girls.

Between 70% and 90% of all foot fractures in children involve the metatarsals and phalanges [425]:

- Metatarsal fractures are common in older children and adolescents and may account for around 50–70% of all paediatric foot fractures (Fig. 12.74) [424, 426, 427]. The most frequently fractured metatarsal in children under the age of 5 years is the 1st metatarsal and in children above the age of 5 years the 5th metatarsal [426]. Fractures of the 1st and 5th metatarsal can occur isolated, while fractures of the 2nd, 3rd, and 4th metatarsal often occur combined with another metatarsal fracture [426].
- Phalangeal fractures may account for around 20–30% of all paediatric foot fractures [424, 426, 428–431]. These fractures usually are Salter–Harris type I or type II fractures [424].
- Tarsal fractures (fractures of the talus, calcaneus, and of the cuboid, navicular and cuneiform bones) are rare and together account for less than 5–15% of all paediatric foot fractures [427]. Fractures of the calcaneus account for a third of all tarsal fractures. In children the most common talus fracture is a fracture of the neck of the talus [427].

Fig. 12.74 Radiograph of the foot of a 4-year-old child, he was playing in the house when a door (which was removed from the hinges) fell over and landed on his foot. There are transverse fractures of the 2nd and 3rd metatarsal (inset)

12.9.2 Cause and Manner of Fractures of the Foot

In Table 12.10, an overview is given of the cause and accidental circumstances of fractures of the different bones of the foot.

Fractures of the foot can be sustained in accidental and in non-accidental circumstances. In mobile and increasingly more active children fractures of the foot mostly occur due to accidental circumstances such as direct impact, crush injury, or falls from height. Singer et al. evaluated the findings concerning metatarsal fractures in 125 children (75 boys, 50 girls; average age 8.6 years; range 1–17 years) [426]. Most fractures were sustained outdoors, including backyard and playground (30%). Other sites were indoors (25%), sports facilities (25%), and school and child-care facilities (12%).

Table 12.10 Overview of cause and manner of fractures of the different bones of the foot [423–427, 475, 476]

	Causing mechanism
Metatarsal fractures	Direct force: • Direct blow: shaft fracture Indirect force: • Torsional forces applied to the forefoot: metatarsal neck fracture Repetitive stress: • Overuse fractures, e.g. during sporting activities
• Avulsion fracture of the base of the 5th metatarsal	Inversion or adduction force
Phalangeal fractures	Direct blow: • Objects falling on toe • Stubbing toe
• Hallux	Direct blow: • Commonly during sporting activities, especially soccer
Tarsal fractures	
• Talus	Forced dorsiflexion of the foot, when the neck impinges against the anterior lip of the tibia, e.g. in: • Falls from height • Motor vehicle accidents
• Calcaneus	Combination of axial loading (compression) with the talus being driven into the calcaneus: • Usually, fall from height • Traffic accidents Stress fracture at the beginning of walking (sometimes described as 'toddler's fracture')
• Cuboid bone	Direct and indirect force: • Shear force across the midfoot and/or twisting injury a.k.a. as nutcracker mechanism in which the cuboid is compressed between the bases of the 4th and 5th metatarsal in the anterior process of the calcaneus by force abduction on a fixed plantar flexed foot • Load to the heel: fall from height when the foot hits the ground in plantar-flexed position, transmitting axial and rotatory forces up along the lateral column
• Navicular bone	Direct and indirect force, e.g. in a motor vehicle accident
• Cuneiform bones	No mechanism is known in children
Tarsometatarsal injuries (Lisfranc injuries)	Direct forces: • Crushing: object falling on the foot, resulting in rupture of the plantar ligaments Indirect forces (more common than direct forces): • Violent plantar flexion or abduction force alone or in combination. May result from vertical loading in plantar flexion as in falling from a height or trying to break speed with the foot while riding a bicycle • With forced abduction the metatarsals are impacted laterally, fracturing the base of the 2nd metatarsal and crushing the cuboid

In 8% the fractures were sustained in traffic accidents. They found that in children under the age of 5 years (n=40) most fractures (>50%) occurred due to a fall from height. Most fractures in these children were sustained either inside the house (43%) or outside the house in the backyard (40%) during leisure activities. In children above that age ($n = 85$) most fractures (35%) were sustained during sports activities. In these children most fractures occurred due to a fall from standing height on a level surface (including twisting).

Fractures of the feet in children under the age of one year seem to be associated with non-accidental injury (Fig. 12.75). However, they are only rarely reported. Studies on feet fractures in association with non-accidental trauma in children are very limited:

- In 1977, Jaffe and Lasser reported an infant with multiple metatarsal fractures due to non-accidental trauma [432]. They were the first to make a plea to routinely include imaging of the hands and feet in evaluating suspicions of non-accidental circumstances.
- Nimkin et al. evaluated 11 infants under the age of 10 months with fractures of hands and feet due to non-accidental trauma [235]. A total of 22 fractures were noted. Five infants had a total of 7 fractures of the feet (6× metatarsal fractures and 1× proximal phalangeal fracture). The authors found predominantly torus fractures. According to the authors torus fractures are consistent with forced hyperflexion.
- Pandya et al. compared 500 child abuse trauma patients with 985 control (accidental) trauma patients [130]. They found 6-foot fractures in each group, with an OR, adjusted for age and sex, of 3.6 (1.1–12.2) for abuse.

The presence of feet fractures in infants and children who received a skeletal survey for evaluation of suspected non-accidental trauma is low and has been reported in only a few studies (Fig. 12.76) [10, 236–238]:

- Barber et al. reported on a study in 567 children, of whom 313 suffered a total of 1,029 fractures [10]. Eleven children (3.5%) had fractures of the foot. In the study by Kleinman et al. 225 out of 365 children had one or more fractures on the skeletal survey. Six children (2.7%) had a total of 9 fractures of the foot [236].
- Karmazyn et al. studied 930 children of whom 317 had a total of 899 fractures. Two infants had a total of 4 (0.4%) fractures of the hand [237].
- In the, by far largest, study of Lindberg et al. out of 2890 children 1208 had one or more fractures. Of these children 21 (1.7%) had a total of 20 fractures of the hand [238]. In this study, there were 7 children with either a fracture to the hand or foot, but it was not possible to discriminate as the report spoke of, e.g. a digit fracture.

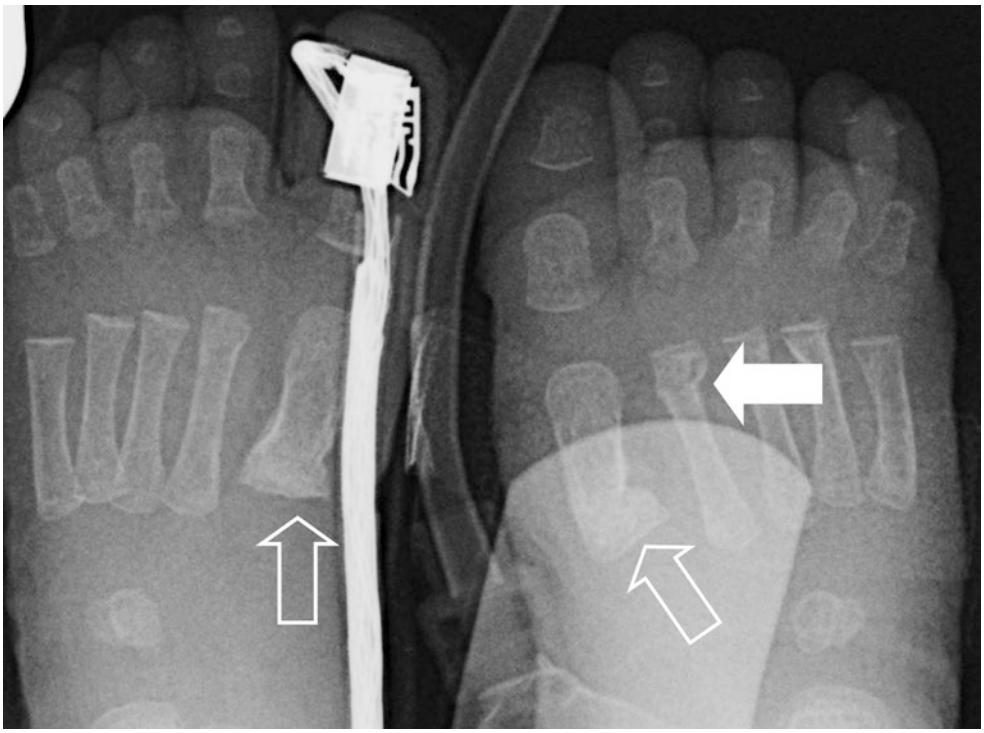


Fig. 12.75 Three-month-old infant who was suspected to be a victim of child abuse. As part of the skeletal survey radiographs of the feet were made, these showed bilateral torus fractures of the base of MT-I (open arrow) and a sub-capital torus fracture of MT-II of the left foot (arrow)

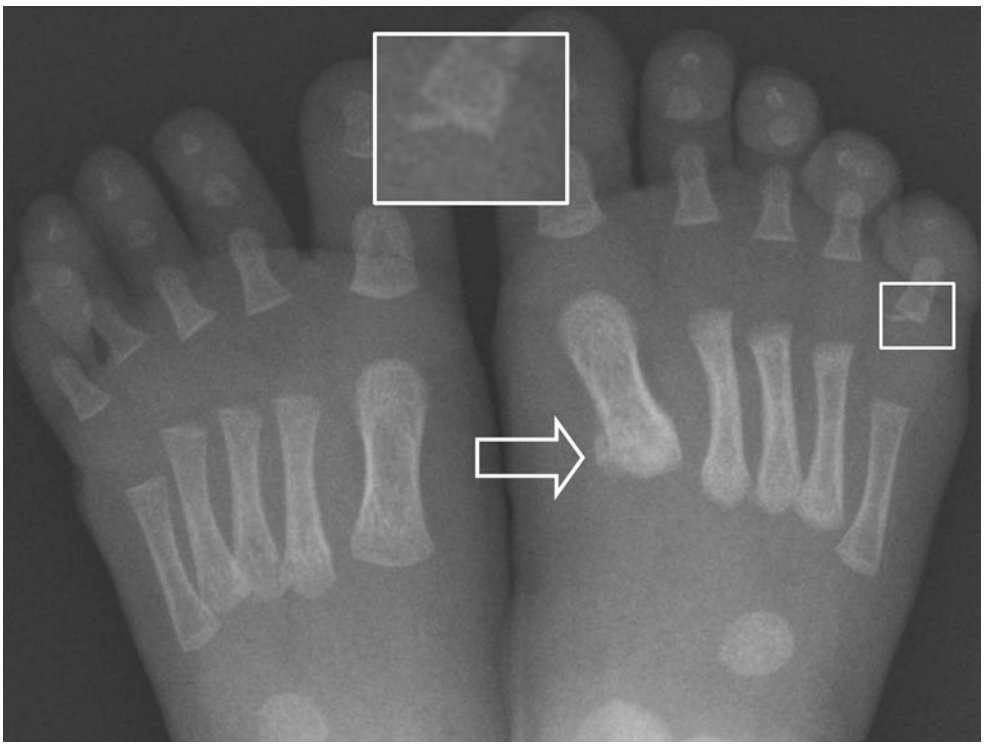


Fig. 12.76 Two-month-old infant who was suspected to be a victim of child abuse. As part of the skeletal survey radiographs of the feet were made, these showed a torus fracture of the base of MT-I of the right foot (arrow) and a SH-III fracture of the base of the proximal phalanx of the 5th toe (inset)

12.10 Subperiosteal Haemorrhage and Periosteal Reaction

12.10.1 Traumatic Sub-periosteal Haemorrhage

The periosteum of the young growing bone differs from adult bones on two important points which increase the risk of subperiosteal haemorrhage. First of all, the periosteum of the young growing bone is loosely attached to the underlying cortical bone with much less anchoring collagen fibres than in older children and adults. Secondly, during growth the number of periosteal blood vessels is tremendous and with a strong circulation through them [38].

Traumatic subperiosteal haemorrhage can occur as a result of direct or indirect physical forces acting on the bone.

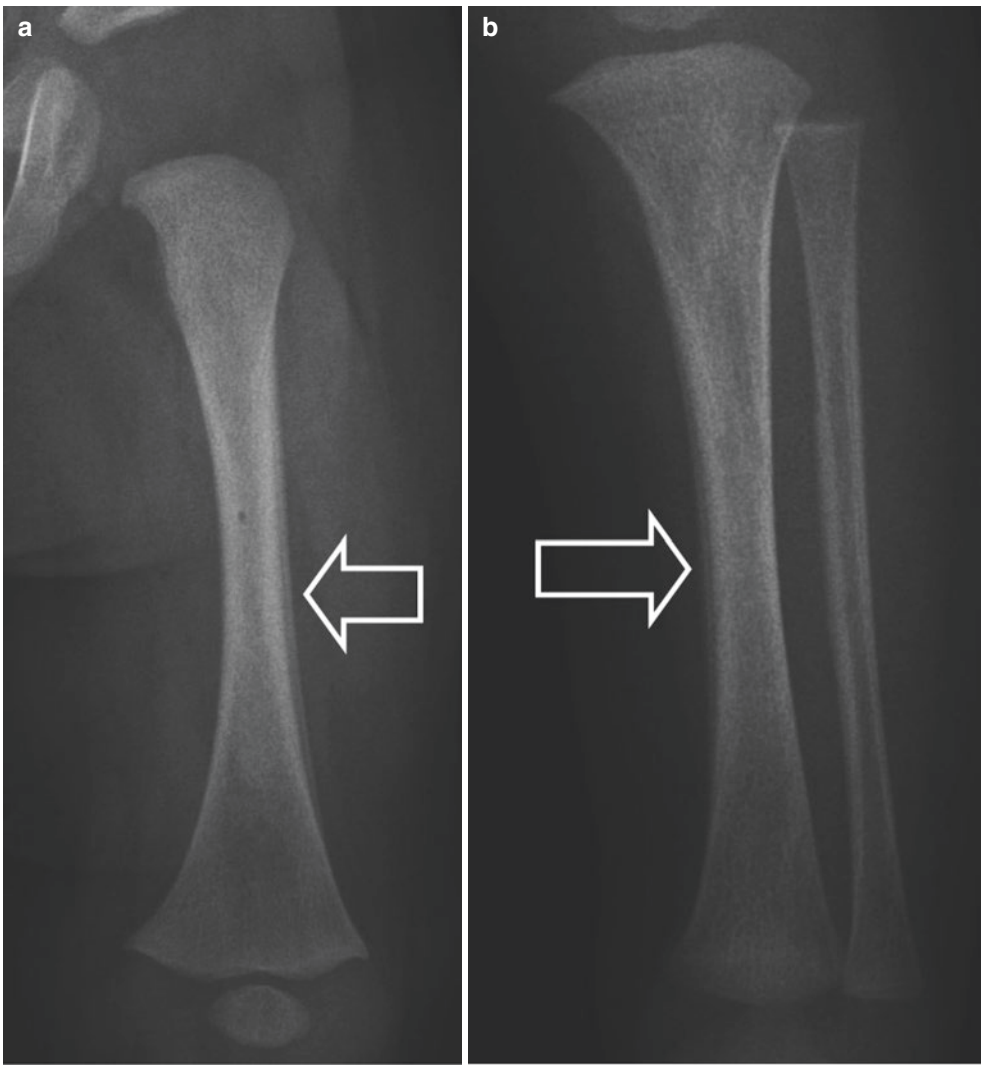


Fig. 12.77 Physiological sub-periosteal new bone formation along the diaphysis of the (**a**) femur and (**b**) the tibia (arrow)

Torsional, tractional, frictional, and blunt impact forces have been described to cause subperiosteal bleeding. Traumatic subperiosteal haemorrhage may be present with or without a visible underlying bone fracture. Extensive subperiosteal hematomas have been described in neurofibromatosis case reports as a rare cause of limb hypertrophy after minor (or no) trauma [433–436].

Whatever cause it has, a subperiosteal haemorrhage will lift the periosteum from the cortex. The presence of the subperiosteal hematoma causes a periosteal reaction consisting of stimulation of the cells in the cambium layer of the periosteum to form subperiosteal new bone. In children, the cambium is thicker than in adults and it has considerable osteoblastic potential. The process from subperiosteal hematoma to periosteal reaction to subperiosteal new bone formation cannot be detected radiographically until calcification has occurred [437]. From fracture dating studies, it is known that SPNBF in long bone fractures is seen as early as day 5–7 after trauma [438–441].

Periosteal reaction with subperiosteal new bone formation can be provoked by any condition that irritates or elevates the periosteum.

Subperiosteal haemorrhage and periosteal reaction due to trauma must be distinguished from periosteal reaction seen in medical conditions such as vitamin C deficiency, vitamin A intoxication, infantile cortical hyperostosis (Caffey's disease), osteomyelitis, malignancies (such as leukaemia), and congenital syphilis [442].

In infants between 1 and 6 months subperiosteal new bone formation of the long bones (tibia, femur, humerus, radius) may represent a normal physiological phenomenon (Fig. 12.77) [443, 444]. Physiological subperiosteal new bone formation tends to be bilateral and with a thickness seldom exceeding 2 millimetres.

12.10.2 Periosteum, Periosteal Reaction, and the Healing of Fractures

After a fracture, the periosteum stays intact in children more often than in adults, because in children the periosteum is relatively thicker, stronger, and more biologically active. When the periosteum stays intact, the presence of the subperiosteal hematoma causes a periosteal reaction resulting in

subperiosteal new bone (Sect. 12.10.1), leading to a continuity of bony tissue will grow over the location of the fracture. This results in a more stable fracture and reduces the chance of dislocation. Essentially, here the periosteum functions as a natural splint.

Moreover, a child's periosteum has greater potential to form bone than that of an adult. This adds extra stimulus to the healing process, resulting in faster remodelling of fractures in children than in adults. Low-grade deviations in alignment will be corrected faster, and even in gross deviations in alignment excellent remodelling can occur.

12.11 Growth Arrest Lines

12.11.1 General Aspects of Growth Arrest Lines

Growth arrest lines (a.k.a. Harris lines, Park lines, growth retardation lines, growth recovery lines, and Zebra lines) are symmetrical transverse sclerotic lines, perpendicular to the long axis of long bones. These lines are evidence of a disturbance in longitudinal growth, which takes place in the metaphyses. The lines are formed in periods when longitudinal growth has temporarily been delayed or even ceased. When growing of the bone is resumed, the arrest lines will 'follow' the longitudinal growth and 'migrate' from the metaphysis towards the diaphysis. They may remain visible for months and may eventually disappear [158, 445].

Radiologically these lines can be recognized by the presence of symmetrical thin white lines in long bones. According to Herring, the lines do not become visible until after normal growth has resumed. The lines are most prominent in rapidly growing ends of bones, e.g. the distal femur and the proximal tibia [445]. Often multiple symmetrical thin white lines are visible, indicating alternating cycles of osseous growth arrest and growth resumption, caused by the occurrence of repetitive pathologic levels of stress during bone development [445, 446].

Growth arrest lines were first described by Harris in 1926/1927 [447, 448]. Park described the influence of nutritional disturbances on the growing bone and on the development of these lines [449].

12.11.2 Growth Arrest Lines Due to Childhood Medical Conditions

Growth arrest lines have been reported in a multitude of childhood medical conditions in which a disturbance (a delay or even a temporary cessation) in growth is seen (causes of 'organic failure to thrive').

Growth arrest lines are reported to occur due to malnutrition in children, due to poor diet or starvation [449]. They may also occur in every disease with a severe and/or chronic course of systemic illnesses, e.g. infections, including septicaemia [449, 450], hypothyroidism [451], parahypothyroidism [452], Cushing's syndrome [453], chronic juvenile arthritis [454], and chemotherapy in children with malignancies and the use of other medication, e.g. bisphosphonates (Figs. 12.78a–c, 12.79, and 12.80) [455–458].

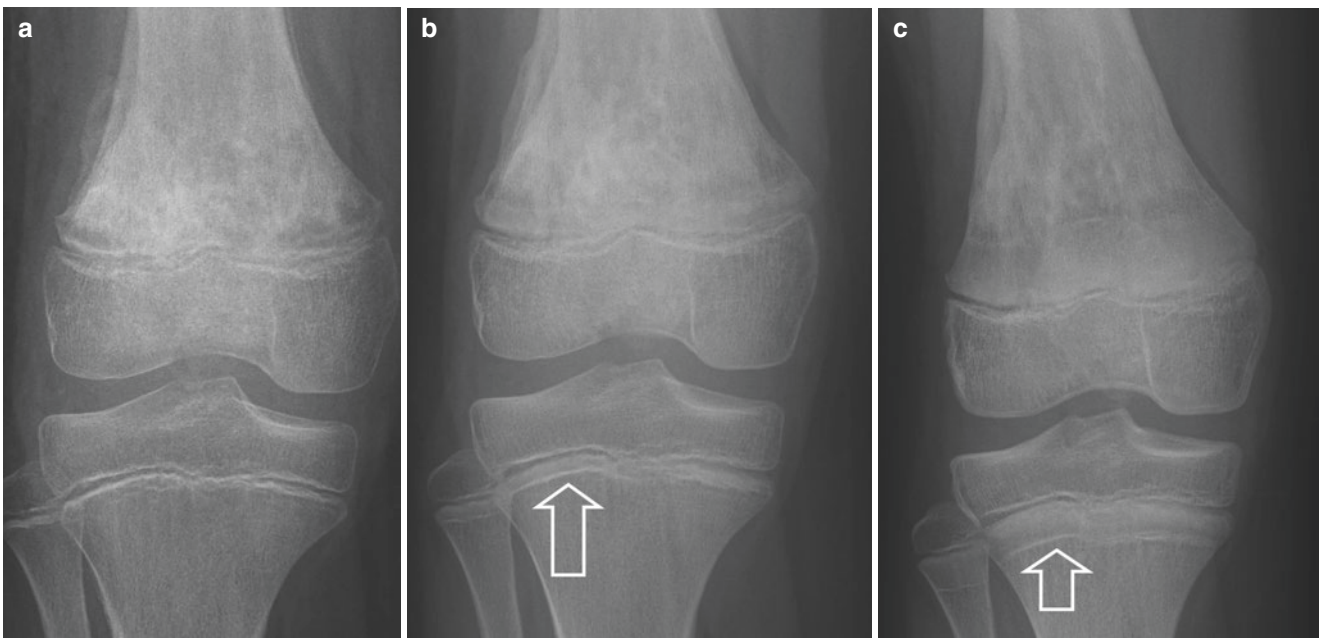


Fig. 12.78 Ten-year-old infant treated for osteomyelitis of the distal femur. (**a**) Radiograph shows a mixed permeative sclerotic aspect of the distal femur. (**b**) Radiograph after 2 months shows a growth retardation line in the proximal tibia (arrow). (**c**) Radiograph after 8 months shows growth of the tibia resulting in the growth retardation line (arrow) moving away from the growth plate

Fig. 12.79 Child with fibrous dysplasia treated with intravenous bisphosphonates. Each growth retardation line corresponds with a course of treatment

Fig. 12.80 Growth retardation lines in a 5-year-old girl after treatment with intravenous bisphosphates, due to fibrous dysplasia in the left maxillary sinus

The lines are also found in children that had been immobilized after orthopaedic surgery [459]. Kennedy et al. reported the occurrence in three girls after a localized trauma. In 2 girls a surgical intervention was needed, in 1 girl no surgical intervention was done [450]:

- Eight-year girl, following a right-sided tibial spine fracture, which required a surgical intervention (open reduction and internal fixation), resulting in growth arrest lines in the right proximal tibia and fibula and in an intra-epiphyseal distal femoral arrest silhouette.
- Nine-year-old girl, following a hyperflexion injury of the left knee, resulting in avulsions of her anterior and posterior cruciate ligaments, which required a surgical intervention, resulting in left-sided femoral, fibular and tibial growth arrest lines and in an intra-epiphyseal distal femoral epiphyseal 'arrest silhouette'.
- Ten-year-old girl, following a comminuted distal tibial fracture after falling from a height, which required an anatomical reduction.

12.11.3 Growth Arrest Lines Due to Non-organic Failure to Thrive

Growth disturbances are not just caused by medical conditions, resulting in a temporary disturbance of longitudinal growth (Sect. 12.11.2). In the Western world, the most common cause of growth and development retardation—in other words, the most common cause of 'failure to thrive'—is not 'organic failure to thrive', but 'mixed organic and non-organic failure to thrive', due to neglect and under stimulation. In neglect, the child is offered insufficient calories (malnourishment—organic failure to thrive) and/or insufficient affective stimulation (non-organic failure to thrive).

As early as 1967, Patton and Gardner mentioned growth arrest lines (metaphyseal growth-retardation lines) in their book on maternal deprivation [460]. Maternal deprivation stands for a serious disturbance in the relation between parent (mother) and child, and a lack of bonding between parent (mother) and child. The deprivation consists of neglect, rejection and isolation of the child. Maternal deprivation syndrome leads to serious growth retardation, delayed skeletal maturation, and retarded motor and intellectual develop-

ment [461]. This multitude of physical symptoms is nowadays summarized in the term 'non-organic failure to thrive'. Khadilkar et al. confirmed the observation of Patton and Gardner that the origin of these lines may involve psychological factors [462].

Based on a study concerning 241 tibiae from a medieval Swiss skeletal material Papageorgopoulou et al. concluded that the development of these lines is a result of normal growth and growth spurts, rather than a pure outcome of nutritional or pathologic stress. Animal tests, however, suggest that the lines are formed after an initial retardation or cessation in growth, followed by resumed growth [449, 459]. According to Khadilkar et al., in children they seem to occur in similar circumstances [462]. In case the process is cyclic (repeated periods of delayed growth interspersed with periods of resumed growth) a large number of lines may be found. These lines will always remain visible, up to and including puberty.

When multiple growth arrest lines are found in a child, mixed organic and non-organic failure to thrive will be, after exclusion of other merely organic causes, the most probable cause [462].

Thus far, only two studies have evaluated whether growth arrest lines can be an indicator of non-accidental trauma:

- Zapala et al. reported that growth arrest lines occur more frequently in infants with a high risk of non-accidental trauma ($n = 21$) compared to infants with a low risk of non-accidental trauma ($n = 52$) [463]. Infants at high risk had a significant intracranial injury, retinal haemorrhages, other skeletal injuries, and clinical determination of high risk (child protection team/social work assessment). Infants at low risk had a skull fracture without significant intracranial injury, history of a fall and clinical determination of low risk. The authors concluded that growth arrest lines are significantly more present in children with high risk of abuse (71%) compared to the low-risk group (38%) ($p < 0.001$; odds ratio 4.0, 95% CI: 1.7–9.5). However, this is equal to a likelihood ratio of 1.9, in other words: growth arrest lines are 1.9 times more likely in infants in the high-risk group than in infants in the low-risk group.
- Spiller et al. describe 135 children, 58 in the low-risk abuse group, 26 in the neglect group, and 51 in the physical abuse group [464]. Children in the neglect group and physical abuse group had 1.73 ($p = 0.007$) and 1.84 ($p < 0.001$) times more growth arrest lines respectively, compared to the low-risk group. The most common locations for growth arrest lines in their population were distal radius, proximal tibia, and distal tibia. In the study of Spiller et al., the specificity for maltreatment (child abuse and neglect) in children with at least 10 growth arrest lines in the long bones was greater than 84%, while sensitivity was less than 35%. This means a LR+ of 2.2, in other words the finding of at least 10 growth arrest lines in the long bones is 2.2 times more likely in children in the high-risk group than in the low-risk group.

References

1. Carter D (1984) The biomechanics of bone. In: Nahum AM, Melvin J (eds) The biomechanics of trauma. Appleton and Lange, pp 15–90
2. Kempe CH, Silverman FN, Steele BF, Droegenmueller W, Silver HK (1962) The battered-child syndrome. JAMA 181:17–24
3. Salter RB, Harris WR (1963) Injuries Involving the Epiphyseal Plate. J Bone Joint Surg 45(3):587–622
4. Tsai A, McDonald AG, Rosenberg AE, Gupta R, Kleinman PK (2014) High-resolution CT with histopathological correlates of the classic metaphyseal lesion of infant abuse. Pediatr Radiol 44(2):124–140
5. Kleinman PK, Marks SC Jr (1995) Relationship of the subperiosteal bone collar to metaphyseal lesions in abused infants. J Bone Joint Surg Am 77A(10):1471–1476
6. Kepron C, Pollanen MS (2015) Rickets or abuse? A histologic comparison of rickets and child abuse-related fractures. For Sci Med Pathol 11(1):78–87
7. Kleinman PK, Marks SC, Blackbourne B (1986) The metaphyseal lesion in abused infants: a radiologic-histopathologic study. AJR Am J Roentgenol 146(5):895–905
8. Kleinman PK, Rosenberg AE, Tsai A (2015) Skeletal trauma: general considerations. In: Kleinman PK (ed) Diagnostic Imaging of child abuse, 3rd edn. Cambridge Publishers, Cambridge, pp 23–52
9. Thompson A, Bertocci G, Kaczor K, Smalley C, Pierce MC (2015) Biomechanical investigation of the classic metaphyseal lesion using an immature porcine model. AJR Am J Roentgenol 204(5):W503-9
10. Barber I, Perez-Rossello JM, Wilson CR, Kleinman PK (2015) The yield of high-detail radiographic skeletal surveys in suspected infant abuse. Pediatr Radiol 45(1):69–80
11. Adamsbaum C, De Boissieu P, Teglas JP, Rey-Salmon C (2019) Classic metaphyseal lesions among victims of abuse. J Pediatr 209:154–9.e2
12. Tsai A, Johnston PR, Perez-Rossello JM, Breen MA, Kleinman PK (2018) The distal tibial classic metaphyseal lesion: medial versus lateral cortical injury. Pediatr Radiol 48(7):973–978
13. Kleinman PK, Marks SC Jr (1996) A regional approach to classic metaphyseal lesions in abused infants: the distal tibia. AJR Am J Roentgenol 166(5):1207–1212
14. Hymel KP, Spivack BS. The biomechanics of physical injury. In: Reece RM, Ludwig S, eds. Child abuse—medical diagnosis and management: Lippincott Williams en Wilkins, 2001; p. 1-22.
15. Kleinman PK, Marks SC Jr (1996) A regional approach to the classic metaphyseal lesion in abused infants: the proximal humerus. AJR Am J Roentgenol 167(6):1399–1403
16. Kleinman PK, Marks SC Jr (1996) A regional approach to the classic metaphyseal lesion in abused infants: the proximal tibia. AJR Am J Roentgenol 166(2):421–426
17. Kleinman PK, Marks SC Jr (1998) A regional approach to the classic metaphyseal lesion in abused infants: the distal femur. AJR Am J Roentgenol 170(1):43–47
18. Osier LK, Marks SC Jr, Kleinman PK (1993) Metaphyseal extensions of hypertrophied chondrocytes in abused infants indicate healing fractures. J Pediatr Orthop 13(2):249–254
19. DeLee JC, Wilkins KE, Rogers LF, Rockwood CA (1980) Fracture-separation of the distal humeral epiphysis. J Bone Joint Surg Am 62(1):46–51

20. Karmazyn B, Marine MB, Wanner MR, Sağlam D, Jennings SG, Hibbard RA (2020) Establishing signs for acute and healing phases of distal tibial classic metaphyseal lesions. Pediatr Radiol. 50(5):715–725
21. Kleinman PK, Marks SC Jr, Spevak MR, Belanger PL, Richmond JM (1991) Extension of growth-plate cartilage into the metaphysis: a sign of healing fracture in abused infants. AJR Am J Roentgenol 156(4):775–779
22. Tsai A, Connolly SA, Ecklund K, Johnston PR, Kleinman PK (2019) Subperiosteal new bone formation with the distal tibial classic metaphyseal lesion: prevalence on radiographic skeletal surveys. Pediatr Radiol
23. Offiah A, van Rijn RR, Perez-Rossello JM, Kleinman PK (2009) Skeletal imaging of child abuse (non-accidental injury). Pediatr Radiol 39(5):461–470
24. Kleinman PK (2008) Problems in the diagnosis of metaphyseal fractures. Pediatr Radiol 38(Suppl 3):S388–SS94
25. Kleinman PK, Nimkin K, Spevak MR et al (1996) Follow-up skeletal surveys in suspected child abuse. AJR Am J Roentgenol 167(4):893–896
26. Harper NS, Eddleman S, Lindberg DM (2013) The utility of follow-up skeletal surveys in child abuse. Pediatrics 131(3):e672-8
27. Harlan SR, Nixon GW, Campbell KA, Hansen K, Prince JS (2009) Follow-up skeletal surveys for nonaccidental trauma: can a more limited survey be performed? Pediatr Radiol 39(9):962–968
28. Zimmerman S, Makoroff K, Care M, Thomas A, Shapiro R (2005) Utility of follow-up skeletal surveys in suspected child physical abuse evaluations. Child Abuse Negl 29(10):1075–1083
29. The Royal College of Radiologists (RCR) and the Society and College of Radiographers (SCoR). The radiological investigation of suspected physical abuse in children. 2017. [05-07-2021]; Available from: https://www.rcr.ac.uk/publication/radiological-investigation-suspected-physical-abuse-children.
30. Drubach LA, Sapp MV, Laffin S, Kleinman PK (2008) Fluorine-18 NaF PET imaging of child abuse. Pediatr Radiol 38(7):776–779
31. Perez-Rossello JM, Connolly SA, Newton AW, Zou KH, Kleinman PK (2010) Whole-body MRI in suspected infant abuse. AJR Am J Roentgenol 195(3):744–750
32. Proisy M, Vivier PH, Morel B et al (2021) Whole-body MR imaging in suspected physical child abuse: comparison with skeletal survey and bone scintigraphy findings from the PEDIMA prospective multicentre study. Eur Radiol
33. Marine MB, Hibbard RA, Jennings SG, Karmazyn B (2019) Ultrasound findings in classic metaphyseal lesions: emphasis on the metaphyseal bone collar and zone of provisional calcification. Pediatr Radiol 49(7):913–921
34. Karmazyn B, Marine MB, Wanner MR et al (2020) Accuracy of ultrasound in the diagnosis of classic metaphyseal lesions using radiographs as the gold standard. Pediatr Radiol
35. Tsai A, McDonald AG, Rosenberg AE, Stamoulis C, Kleinman PK (2013) Discordant radiologic and histological dimensions of the zone of provisional calcification in fetal piglets. Pediatr Radiol 43(12):1606–1614
36. Pierce MC, Bertocci GE, Vogeley E, Moreland MS (2004) Evaluating long bone fractures in children: a biomechanical approach with illustrative cases. Child Abuse Negl 28(5):505–524
37. Kleinman PL, Zurakowski D, Strauss KJ et al (2008) Detection of simulated inflicted metaphyseal fractures in a fetal pig model: image optimization and dose reduction with computed radiography. Radiology 247(2):381–390
38. Caffey J (1957) Some traumatic lesions in growing bones other than fractures and dislocations: clinical and radiological features: The Mackenzie Davidson Memorial Lecture. Br J Radiol 30(353):225–238
39. Loder RT, Bookout C (1991) Fracture patterns in battered children. J Orthopaed Trauma 5(4):428–433
40. Worlock P, Stower M, Barbor P (1986) Patterns of fractures in accidental and non-accidental injury in children: a comparative study. BMJ 293:100–102
41. Lindberg DM, Berger RP, Reynolds MS, Alwan RM, Harper NS (2014) Yield of skeletal survey by age in children referred to abuse specialists. J Pediatr 164(6):1268–73.e1
42. Kleinman PK, Perez-Rossello JM, Newton AW, Feldman HA, Kleinman PL (2011) Prevalence of the classic metaphyseal lesion in infants at low versus high risk for abuse. AJR Am J Roentgenol 197(4):1005–1008
43. Kleinman PK, Marks SC Jr, Richmond JM, Blackbourne BD (1995) Inflicted skeletal injury: a postmortem radiologic-histopathologic study in 31 infants. AJR AmJ Roentgenol 165(3):647–650
44. Thackeray JD, Wannemacher J, Adler BH, Lindberg DM (2016) The classic metaphyseal lesion and traumatic injury. Pediatr Radiol 46(8):1128–1133
45. Sieswerda-Hoogendoorn T, van Rijn RR, Robben SGF (2014) Classic metaphyseal lesion following vaginal breech birth, a rare birth trauma. JoFRI 2(1):2–4
46. Gowda SR, Vecsei FA, Fairhurst J, Aarvold A (2018) Metaphyseal corner fracture caused in utero by external cephalic version—a rare presentation. Case Rep Perinatal Med 7:1
47. Lysack JT, Soboleski D (2003) Classic metaphyseal lesion following external cephalic version and cesarean section. Pediatr Radiol 33(6):422–424
48. O'Connell A, Donoghue VB (2007) Can classic metaphyseal lesions follow uncomplicated caesarean section? Pediatr Radiol 37(5):488–491
49. Lee GS, Methratta ST, Frasier LD (2019) Classic metaphyseal lesion of distal tibia following footling breech delivery. Pediatr Radiol 49(13):1840–1842
50. Buonuomo PS, Ruggiero A, Zampino G, Maurizi P, Attinà G, Riccardi R (2006) A newborn with multiple fractures as first presentation of infantile myofibromatosis. J Perinatol 26(10):653–655
51. Grayev AM, Boal DK, Wallach DM, Segal LS (2001) Metaphyseal fractures mimicking abuse during treatment for clubfoot. Pediatr Radiol 31(8):559–563
52. Burrell T, Opfer E, Berglund L, Lowe LH, Anderst J (2015) A witnessed case of a classic metaphyseal fracture caused during IV line placement in a child: Insight into mechanism of injury. J Forensic Leg Med 35:51–53
53. Della Grotta LM, Marine MB, Harris TL, Karmazyn B (2019) Classic metaphyseal lesion acquired during physical therapy. Clin Imaging 54:100–102
54. Culotta PA, Burge LR, Bachim AN, Donaruma-Kwoh M (2020) Are classic metaphyseal lesions pathognomonic for child abuse? Two cases of motor vehicle collision-related extremity CML and a review of the literature. J Forensic Leg Med 74:102006
55. Kleinman PK, Belanger PL, Karellas A, Spevak MR (1991) Normal metaphyseal radiologic variants not to be confused with findings of infant abuse. AJR Am J Roentgenol 156(4):781–783
56. Quigley AJ, Stafrace S (2014) Skeletal survey normal variants, artefacts and commonly misinterpreted findings not to be confused with non-accidental injury. Pediatr Radiol. 44(1):82–93. quiz 79-81
57. Norrell K, Hennrikus W (2017) The risk of assuming abuse in an infant with an isolated metaphyseal lesion: a case report. JBJS Case Connect 7(3):e69
58. Kleinman PK, Sarwar ZU, Newton AW, Perez-Rossello JM, Rebello G, Herliczek TW (2009) Metaphyseal fragmentation with physiologic bowing: a finding not to be confused with the classic metaphyseal lesion. AJR Am J Roentgenol 192(5):1266–1268
59. Mizuta T, Benson WM, Foster BK, Paterson DC, Morris LL (1987) Statistical analysis of the incidence of physeal injuries. J Pediatr Orthop 7(5):518–523

60. Cepela DJ, Tartaglione JP, Dooley TP, Patel PN (2016) Classifications in brief: Salter-Harris classification of pediatric physeal fractures. Clin Orthop Relat Res 474(11):2531–2537
61. Little JT, Klionsky NB, Chaturvedi A, Soral A, Chaturvedi A (2014) Pediatric distal forearm and wrist injury: an imaging review. Radiographics 34(2):472–490
62. Rogers LF, Poznanski AK (1994) Imaging of epiphyseal injuries. Radiology 191(2):297–308
63. Kothadia S, Birole U, Ranade A (2017) Paediatric Salter-Harris type IV injury of distal tibia with talus fracture. BMJ Case Rep 2017
64. Supakul N, Hicks RA, Caltoum CB, Karmazyn B (2015) Distal humeral epiphyseal separation in young children: an often-missed fracture-radiographic signs and ultrasound confirmatory diagnosis. AJR Am J Roentgenol 204(2):W192–W198
65. Nimkin K, Kleinman PK, Teeger S, Spevak MR (1995) Distal humeral physeal injuries in child abuse: MR imaging and ultrasonography findings. Pediatr Radiol 25(7):562–565
66. Marine MB, Forbes-Amrhein MM (2021) Fractures of child abuse. Pediatr Radiol 51(6):1003–1013
67. Peterson HA, Madhok R, Benson JT, Ilstrup DM, Melton LJ 3rd. (1994) Physeal fractures: Part 1. Epidemiology in Olmsted County, Minnesota, 1979-1988. J Pediatr Orthop 14(4):423–430
68. Tharakan SJ, Lee RJ, White AM, Lawrence JT (2016) Distal humeral epiphyseal separation in a newborn. Orthopedics 39(4):e764-7
69. Gigante C, Kini SG, Origo C, Volpin A (2017) Transphyseal separation of the distal humerus in newborns. Chin J Traumatol = Zhonghua chuang shang za zhi 20(3):183–186
70. Ogden JA, Lee KE, Rudicel SA, Pelker RR (1984) Proximal femoral epiphysiolysis in the neonate. J Pediatr Orthop 4(3):285–292
71. Banagale RC, Kuhns LR (1983) Traumatic separation of the distal femoral epiphysis in the newborn. J Pediatr Orthop. 3(3):396–398
72. Eliahou R, Simanovsky N, Hiller N, Simanovsky N (2006) Fracture-separation of the distal femoral epiphysis in a premature neonate. J Ultrasound Med 25(12):1603–1605
73. Baker AM, Methratta ST, Choudhari AK (2012) Transphyseal fracture of the distal humerus in a neonate. West J Emerg Med 12(2):173
74. Triplet JJ, Samora WP, Balch SJ (2018) Distal humeral physeal separation in a newborn: a case report and review of the literature. Curr Orthop Pract 29(6):611–615
75. Shalaby-Rana E, Hinds TS, Deye K, Jackson AM (2020) Proximal femoral physeal fractures in children: a rare abusive injury. Pediatr Radiol 50(8):1115–1122
76. Jones JCW, Feldman KW, Bruckner JD (2004) Child abuse in infants with proximal physeal injuries of the femur. Pediatr Emerg Care 20(3):157–161
77. Nenopoulos A, Beslikas T, Gigis I, Sayegh F, Christoforidis I, Hatzokos I (2015) The role of CT in diagnosis and treatment of distal tibial fractures with intra-articular involvement in children. Injury 46(11):2177–2180
78. Ahn L, Williams B (2021) Triplane fractures. Orthobullets. [15-07-2021]; Available from: https://www.orthobullets.com/pediatrics/4029/triplane-fractures
79. Caviglia H, Garrido CP, Palazzi FF, Meana NV (2005) Pediatric fractures of the humerus. Clin Orthop Relat Res 432:49–56
80. Hohl JC (1976) Fractures of the humerus in children. Orthop Clin North Am 7(3):557–571
81. Landin LA (1983) Fracture patterns in children. Analysis of 8, 682 fractures with special reference to incidence, etiology and secular changes in a Swedish urban population 1950-1979. Acta Orthop Scand Suppl 202:1–109
82. Landin LA (1997) Epidemiology of children's fractures. J Pediatr Orthop Part B 6(2):79–83
83. Rose SH, Melton LJ 3rd, Morrey BF, Ilstrup DM, Riggs BL (1982) Epidemiologic features of humeral fractures. Clin Orthop Relat Res 168:24–30
84. Shrader MW (2007) Proximal humerus and humeral shaft fractures in children. Hand Clin 23(4):431–435
85. Hannonen J, Hyvönen H, Korhonen L, Serlo W, Sinikumpu JJ (2019) The incidence and treatment trends of pediatric proximal humerus fractures. BMC Musculoskelet Disord 20(1):571
86. Watts E, Shirley E, Skaggs DL (2021) Proximal humerus fracture—pediatric. Orthobullets. [25-11-2021]; Available from: https://www.orthobullets.com/pediatrics/4004/proximal-humerus-fracture%2D%2Dpediatric
87. Popkin CA, Levine WN, Ahmad CS (2015) Evaluation and management of pediatric proximal humerus fractures. J Am Acad Orthop Surg 23(2):77–86
88. Chae W, Khan A, Abbott S, Assiotis A (2019) Proximal humerus fractures in children: experience from a Central London Paediatric Orthopaedic Service. Open Orthop J 13:202–207
89. Wimberly RL (n.d.) Proximal humerus fractures. POSNA. [25-11-2021]; Available from: https://posna.org/Physician-Education/Study-Guide/Proximal-Humerus-Fractures
90. Neer CS 2nd, Horwitz BS (1965) Fractures of the proximal humeral epiphysial plate. Clin Orthop Relat Res 41:24–31
91. Lefèvre Y, Journeau P, Angelliaume A, Bouty A, Dobremez E (2014) Proximal humerus fractures in children and adolescents. Orthop Traumatol Surg Res 100(1 Suppl):S149-56
92. Canpolat FE, Köse A, Yurdakök M (2010) Bilateral humerus fracture in a neonate after cesarean delivery. Arch Gynecol Obstet 281(5):967–969
93. Dias E (2012) Bilateral humerus fracture following birth trauma. J Clin Neonatol 1(1):44–45
94. Camus M, Lefebvre G, Veron P, Darbois Y (1985) Traumatismes obstétricaux du nouveau-né. Enquête rétrospective à propos de 20409 naissances [Obstetrical injuries of the newborn infant. Retrospective study apropos of 20,409 births]. J Gynecol Obstet Biol Reprod (Paris) 14(8):1033–1043
95. Gagnaire JC, Thoulon JM, Chappuis JP, Varnier CH, Mered B (1975) Les traumatismes du membre supérieur du noveau-né constatés à la naissance [Injuries to the upper extremities in the newborn diagnosed at birth]. J Gynecol Obstet Biol Reprod 4(2):245–254
96. King J, Diefendorf D, Apthorp J, Negrete VF, Carlson M (1988) Analysis of 429 fractures in 189 battered children. J Pediatr Orthop 8(5):585–589
97. Jones GP, Seguin J, Shiels WE 2nd (2003) Salter-Harris II fracture of the proximal humerus in a preterm infant. Am J Perinatol 20(5):249–253
98. Dalldorf PG, Bryan WJ (1994) Displaced Salter-Harris type I injury in a gymnast. A slipped capital humeral epiphysis? Orthop Rev 23(6):538–541
99. Ahn JI, Park JS (1994) Pathological fractures secondary to unicameral bone cysts. Int Orthop 18(1):20–22
100. Barber DB, Janus RB, Wade WH (1996) Neuroarthropathy: an overuse injury of the shoulder in quadriplegia. J Spinal Cord Med 19(1):9–11
101. Lock TR, Aronson DD (1989) Fractures in patients who have myelomeningocele. J Bone Joint Surg Am. 71(8):1153–1157
102. Marengo L, Rousset M, Paonessa M et al (2016) Displaced humeral shaft fractures in children and adolescents: results and adverse effects in patients treated by elastic stable intramedullary nailing. Eur J Orthop Surg Traumatol Orthop Traumatol 26(5):453–459
103. Rush J. Scapula fractures. POSNA (Pediatric Orthopaedic Society of North America); [29-07-20]; Available from: https://posna.org/Physician-Education/Study-Guide/Scapula-Fractures.

104. Whooley PJ. Humeral shaft injuries. 2021. [25-11-2021]; Available from: https://dontforgetthebubbles.com/humeral-shaft-injuries/.
105. Bayram S, Mert L, Anarat FB, Chodza M, Ergin ÖN (2018) A Newborn with Multiple Fractures in Osteogenesis Imperfecta: A Case Report. J Orthop Case Rep 8(3):71–73
106. Barker TH (1857) On intrauterine fractures: with an illustrative case. Br Med J 2(39):806–809
107. Madsen ET (1955) Fractures of the extremities in the newborn. Acta Obstet Gynecol Scand 34(1):41–74
108. Sherr-Lurie N, Bialik GM, Ganel A, Schindler A, Givon U (2011) Fractures of the humerus in the neonatal period. Isr Med Assoc J 13(6):363–365
109. Shulman BH, Terhune CB (1951) Epiphyseal injuries in breech delivery. Pediatrics 8(5):693–700
110. Dameron TB Jr, Reibel DB (1969) Fractures involving the proximal humeral epiphyseal plate. J Bone Joint Surg Am 51(2):289–297
111. Lemperg R, Liliequist B (1970) Dislocation of the proximal epiphysis of the humerus in newborns. Acta Paediatr Scand 59(4):377–380
112. Reed MH, Letts RM, Pollock AN (1994) Birth fractures. In: Letts RM (ed) Management of pediatric fractures. Churchill Livingstone, pp 1049–1061
113. Kaya B, Daglar K, Kirbas A, Tüten A (2015) Humerus diaphysis fracture in a newborn during vaginal breech delivery. Case Rep Obstet Gynecol 2015:489108
114. Hamilçıkan Ş, Yılmaz K, Can E (2018) Humeral diaphysis fracture in a neonate after vaginal delivery. Sisli Etfal Hastan Tip Bul 52(1):51–53
115. Tan TS, Mohamed A, Dharmaraj S (2016) Bilateral fractures in a shoulder dystocia delivery. BMJ Case Rep 2016
116. Ryan LM (2011) Midshaft humeral fractures in children. UpToDate. [25-11-2021]; Available from: https://somepomed.org/articulos/contents/mobipreview.htm?40/18/41262
117. von Heideken J, Thiblin I, Högberg U (2020) The epidemiology of infant shaft fractures of femur or humerus by incidence, birth, accidents, and other causes. BMC Musculoskelet Disord 21(1):840
118. Rehm A, Promod P, Ogilvy-Stuart A (2020) Neonatal birth fractures: a retrospective tertiary maternity hospital review. J Obstet Gynaecol 40(4):485–490
119. Suleiman FA, Almaaitah AA, Aqrabawi HE (2016) Upper limb birth trauma in a Jordanian population: a prospective study at King Hussein Medical Centre, Amman, Jordan. JPMA J Pak Med Assoc 66(11):1422–1426
120. Basha A, Amarin Z, Abu-Hassan F (2013) Birth-associated long-bone fractures. Int J Gynaecol Obstet 123(2):127–130
121. Bhat BV, Kumar A, Oumachigui A (1994) Bone injuries during delivery. Indian J Pediatr 61(4):401–405
122. Carter S (2021) Humeral shaft fracture. Radiopaedia. Available from: https://radiopaedia.org/articles/humeral-shaft-fracture-1
123. Strait RT, Siegel RM, Shapiro RA (1995) Humeral fractures without obvious etiologies in children less than 3 years of age: when is it abuse? Pediatrics 96(4 Pt 1):667–671
124. Leventhal JM, Thomas SA, Rosenfield NS, Markowitz RI (1993) Fractures in young children. Distinguishing child abuse from unintentional injuries. Am J Dis Child 147:87–92
125. Thomas SA, Rosenfield NS, Leventhal JM, Markowitz RI (1991) Long-bone fractures in young children: distinguishing accidental injuries from child abuse. Pediatrics 88(3):471–476
126. Kowal-Vern A, Paxton TP, Ros SP, Lietz H, Fitzgerald M, Gamelli RL (1992) Fractures in the under-3-year-old age cohort. Clin Pediatr (Phila). 31(11):653–659
127. Rosenberg N, Bottenfield G (1982) Fractures in infants: a sign of child abuse. Ann Emerg Med 11(4):178–180
128. Williams R, Hardcastle N (2005) Best evidence topic report. Humeral fractures and non-accidental injury in children. Emerg Med J 22(2):124–125
129. Shaw BA, Murphy KM, Shaw A, Oppenheim WL, Myracle MR (1997) Humerus shaft fractures in young children: accident or abuse? J Pediatr Orthop. 17(3):293–297
130. Pandya NK, Baldwin K, Wolfgruber H, Christian CW, Drummond DS, Hosalkar HS (2009) Child abuse and orthopaedic injury patterns: analysis at a level I pediatric trauma center. J Pediatr Orthop 29(6):618–625
131. Pandya NK, Baldwin KD, Wolfgruber H, Drummond DS, Hosalkar HS (2010) Humerus fractures in the pediatric population: an algorithm to identify abuse. J Pediatr Orthop Part B. 19(6):535–541
132. Samardzić M, Grujicić D, Milinković ZB (1990) Radial nerve lesions associated with fractures of the humeral shaft. Injury 21(4):220–222
133. Hannonen J, Sassi E, Hyvönen H, Sinikumpu JJ (2020) A shift from non-operative care to surgical fixation of pediatric humeral shaft fractures even though their severity has not changed. Front Pediatr 8:580272
134. Raza M, Anestis I (2021) Paediatric humeral shaft fractures: an overview and modern management approach. Int J Orthop 8(2):1443–1146
135. Hymel KP, Jenny C (1996) Abusive spiral fractures of the humerus: a videotaped exception. Arch Pediatr Adolesc Med 150(2):226–227
136. Somers JM, Halliday KE, Chapman S (2014) Humeral fracture in non-ambulant infants-a possible accidental mechanism. Pediatr Radiol 44(10):1219–1223
137. Altai Z, Viceconti M, Li X, Offiah AC (2020) Investigating rolling as mechanism for humeral fractures in non-ambulant infants: a preliminary finite element study. Clin Radiol 75(1):78.e9-.e16
138. Silverio L (2018) Distal hymerus fracture in children – emergency management. DynaMed. [25-11-2021]; Available from: https://www.dynamed.com/management/distal-humerus-fracture-in-children-emergency-management
139. Smithuis R (2008) Elbow fractures in children. Radiology Assistant. [25-11-2021]; Available from: https://radiologyassistant.nl/pediatrics/hip/fractures-in-children-1
140. Patel B, Reed M, Patel S (2009) Gender-specific pattern differences of the ossification centers in the pediatric elbow. Pediatr Radiol 39(3):226–231
141. Goodwin SJ, Irwin LJ, Irwin GJ (2019) Gender differences in the order of appearance of elbow ossification centres. Scott Med J 64(1):2–9
142. Brubacher JW, Dodds SD (2008) Pediatric supracondylar fractures of the distal humerus. Curr Rev Musculoskelet Med 1(3-4):190–196
143. Holt JB, Glass NA, Shah AS (2018) Understanding the epidemiology of pediatric supracondylar humeral fractures in the United States: identifying opportunities for intervention. J Pediatr Orthop 38(5):e245–ee51
144. Woon C, Souder C, Skaggs D (2021) Supracondylar fracture—pediatric. Orthobullets. [cited 25-11-2021]; Available from: https://www.orthobullets.com/pediatrics/4007/supracondylar-fracture%2D%2Dpediatric
145. Knipe H (2021) Supracondylar humeral fracture. Radiopaedia. [25-11-2021]; Available from: https://radiopaedia.org/articles/supracondylar-humeral-fracture-2
146. Carter S (2021) Flexion supracondylar fracture. Radiopaedia. [25-11-2021]; Available from: https://radiopaedia.org/articles/flexion-supracondylar-fracture?lang=us
147. Reisdorff EJ, Roberts MR, Wiegenstein JG (1993) Pediatric emergency medicine. WB Saunders Co

148. Kemp AM, Dunstan F, Harrison S et al (2008) Patterns of skeletal fractures in child abuse: systematic review. BMJ 337:a1518
149. Rosado N, Ryznar E, Flaherty EG (2017) Understanding humerus fractures in young children: abuse or not abuse? Child Abuse Negl 73:1–7
150. Lawton L (1994) Fractures of the distal radius and ulna. In: Letts RM (ed) Management of pediatric fractures. Churchill-Livingstone, pp 345–368
151. Rinaldi JM, Hennrikus WL (2018) Are supracondylar fractures of the elbow in children caused by child abuse? Pediatrics 141(1 Meeting Abstract):300
152. Shaath K, Souder C, Skaggs DL (2021) Lateral condyle fracture. Orthobullets. [25-11-2021]; Available from: https://www.orthobullets.com/pediatrics/4009/lateral-condyle-fracture%2D%2Dpediatric
153. Tejwani N, Phillips D, Goldstein RY (2011) Management of lateral humeral condylar fracture in children. J Am Acad Orthop Surg 19(6):350–358
154. Carter S (2021) Lateral humeral condyle fracture. Radiopaedia. [25-11-2021]; Available from: https://radiopaedia.org/articles/lateral-humeral-condyle-fracture-1
155. Offiah AC, Hall CM (2009) Diaphyseal fractures. In: Offiah AC, Hall CM (eds) Radiological atlas of child abuse. Radcliff Publishing Ltd, London, pp 81–117
156. Walsh JJ (2020) Medial humeral condyle fracture. Medscape. [25-11-2021]; Available from: https://emedicine.medscape.com/article/1231290-overview#showall
157. Tepeneu NF (2018) Fractures of the humeral condyles in children—a review. J Med Psych Trauma 1(1):3–13
158. Kleinman PK (2015) Upper extremity trauma. In: Kleinman PK (ed) Diagnostic imaging of child abuse, 3rd edn. Cambridge University Press, Cambridge, pp 121–163
159. Gottschalk HP, Eisner E, Hosalkar HS (2012) Medial epicondyle fractures in the pediatric population. J Am Acad Orthop Surg 20(4):223–232
160. Bauer S, Dunne B, Whitewood C (2012) Simultaneous bilateral elbow dislocation with bilateral medial epicondyle fractures in a 13-year-old female gymnast with hyperlaxity. BMJ Case Rep 2012
161. DeFroda S, McGlone P, Levins J, O'Donnell R, Cruz AI, Kriz PK (2020) Shoulder and elbow injuries in the adolescent throwing athlete. R I Med J (2013) 103(7):21–29
162. Sperry K, Pfalzgraf R (1990) Inadvertent clavicular fractures caused by "chiropractic" manipulations in an infant: an unusual form of pseudoabuse. J Forensic Sci 35(5):1211–1216
163. Shore RM (2018) What are pediatric transphyseal fractures. Medscape. [25-11-2021]; Available from: https://www.medscape.com/answers/415822-185743/what-are-pediatric-transphyseal-fractures
164. Abzug JM, Ho CA, Ritzman TF, Brighton BK (2016) Transphyseal fracture of the distal humerus. J Am Acad Orthop Surg 24(2):e39–e44
165. Makanji H, Glotzbecker M (2021) Distal humerus physeal separation—pediatric. Orthobullets. [25-11-2021]; Available from: https://www.orthobullets.com/pediatrics/4006/distal-humerus-physeal-separation%2D%2Dpediatric
166. McLoughlin E, Stanhope B, Johnson K (2017) The answer is not always in black and white: a frequently missed diagnosis in a child presenting with elbow pain. Emerg Med J 34(8):524–525
167. Patil MN, Palled E (2015) Epihyseal separation of lower end humerus in a neonate-diagnostic and management difficulty. J Orthop Case Rep 5(4):7–9
168. Navallas M, Díaz-Ledo F, Ares J et al (2013) Distal humeral epiphysiolysis in the newborn: utility of sonography and differential diagnosis. Clin Imaging 37(1):180–184
169. Mane PP, Challawar NS, Shah H (2016) Late presented case of distal humerus epiphyseal separation in a newborn. BMJ Case Rep 2016
170. Murthy PG, Vuillermin C, Naqvi MN, Waters PM, Bae DS (2017) Capitellar fractures in children and adolescents: classification and early results of treatment. J Bone Joint Surg Am 99(15):1282–1290
171. Ertl JP (2020) Capitellar fractures. Medscape. [25-11-2021]; Available from: https://emedicine.medscape.com/article/1238203-overview#showall
172. Yoon R, Sanchez-Sotelo J, on behalf of 'American Shoulder and Elbow Surgeons' (2021) Capitellum fractures. Orthobullets. [25-11-2021]; Available from: https://www.orthobullets.com/trauma/1023/capitellum-fractures
173. Thomas EM, Tuson KW, Browne PS (1975) Fractures of the radius and ulna in children. Injury 7(2):120–124
174. Schweich P (2021) Distal forearm fractures in children: diagnosis and assessment. UpToDate. [25-11-2021]; Available from: https://www.uptodate.com/contents/distal-forearm-fractures-in-children-diagnosis-and-assessment
175. Wolfe JA, Wolfe H, Banaag A, Tintle S, Perez Koehlmoos T (2019) Early pediatric fractures in a universally insured population within the United States. BMC Pediatr 19(1):343
176. Edgington J, Glotzbecker M (2021) Both bone forearm fracture—pediatric. Orthobullets. [25-11-2021]; Available from: https://www.orthobullets.com/pediatrics/4126/both-bone-forearm-fracture%2D%2Dpediatric
177. Cheng JC, Shen WY (1993) Limb fracture pattern in different pediatric age groups: a study of 3,350 children. J Orthop Trauma 7(1):15–22
178. Johnson PG, Szabo RM (1993) Angle measurements of the distal radius: a cadaver study. Skeletal Radiol 22(4):243–246
179. Reed MH (1977) Fractures and dislocations of the extremities in children. J Trauma 17(5):351–354
180. Bailey DA, Wedge JH, McCulloch RG, Martin AD, Bernhardson SC (1989) Epidemiology of fractures of the distal end of the radius in children as associated with growth. J Bone Joint Surg Am 71(8):1225–1231
181. Qudsi R, Souder C (2021) Distal radius fractures—pediatric. Orthobullets. [25-11-2021]; Available from: https://www.orthobullets.com/pediatrics/4014/distal-radius-fractures%2D%2Dpediatric
182. Tredwell SJ, Van Peteghem K, Clough M (1984) Pattern of forearm fractures in children. J Pediatr Orthop 4(5):604–608
183. Hermans K, Fransz D, Walbeehm-Hol L, Hustinx P, Staal H (2021) Is a Parry Fracture-an isolated fracture of the ulnar shaft-associated with the probability of abuse in children between 2 and 16 years old? Children (Basel). 8:8
184. Perkins CA, Busch MT, Christino MA et al (2018) Olecranon fractures in children and adolescents: outcomes based on fracture fixation. J Children's Orthop 12(5):497–501
185. Holme TJ, Karbowiak M, Arnander M, Gelfer Y (2020) Paediatric olecranon fractures: a systematic review. EFORT Open Rev 5(5):280–288
186. Edgington J, Andras L (2021) Radial head and neck fractures—pediatric. Orthobullets. [25-11-2021]; Available from: https://www.orthobullets.com/pediatrics/4011/radial-head-and-neck-fractures%2D%2Dpediatric
187. Perron AD, Hersh RE, Brady WJ, Keats TE (2001) Orthopedic pitfalls in the ED: Galeazzi and Monteggia fracture-dislocation. Am J Emerg Med 19(3):225–228
188. Berger R, Souder C (2021) Galeazzi fracture—pediatric. Orthobullets. [25-11-2021]; Available from: https://www.orthobullets.com/pediatrics/4016/galeazzi-fracture%2D%2Dpediatric
189. Allen D (2021) Monteggia fracture—pediatric. Orthobullets. [25-11-2021]; Available from: https://www.orthobullets.com/pediatrics/4015/monteggia-fracture%2D%2Dpediatric
190. Hupin Debeurme M, Powers J (2021) Monteggia fracture. POSNA. [25-11-2021]; Available from: https://posna.org/Physician-Education/Study-Guide/Monteggia-Fracture

191. Korner J, Hansen M, Weinberg A, Hessmann M, Rommens P (2004) Monteggia fractures in childhood diagnosis and management in acute and chronic cases. Eur J Trauma 30:361–370
192. Shonnard PY, DeCoster TA (1994) Combined Monteggia and Galeazzi fractures in a child's forearm. A case report. Orthop Rev 23(9):755–759
193. Maeda H, Yoshida K, Doi R, Omori O (2003) Combined Monteggia and Galeazzi fractures in a child: a case report and review of the literature. J Orthop Trauma 17(2):128–131
194. Knipe H, Jones J (2021) Bowing fracture. Radiopedia. [25-11-2021]; Available from: https://radiopaedia.org/articles/bowing-fracture?lang=us
195. Price CT, Mencio GA (2001) Injuries to the shafts of the radius and ulna. In: Beatty JH, Kasser JR (eds) Rockwood and Wilkins' Fractures in Children, 5th edn. Lippincott Williams en Wilkins, pp 433–482
196. Onimus M, Lebrat J, Scherrer M, Weber R, Cousin J, Samaki M (1991) Pregnancy and traffic accident. A case report [Grossesse et accident de la voie publique. A propos d'un cas]. Rev Fr Gynecol Obstet 86(4):315–317
197. Ho CA, Richards BS, Ezaki M (2014) Congenital band syndrome with pseudarthrosis of the radius and ulna and impending vascular compromise: a case report. J Pediatr Orthop. 34(6):e14–e18
198. Angelis S, Vynichakis G, Trellopoulos A, Mirtsios H, Michelarakis J (2019) Strangling congenital constriction ring band of the forearm with fracture: a rare case report. Cureus 11(3):e4189
199. Thompson KA, Satin AJ, Gherman RB (2003) Spiral fracture of the radius: an unusual case of shoulder dystocia-associated morbidity. Obstet Gynecol 102(1):36–38
200. Ryan LM, Teach SJ, Searcy K et al (2010) Epidemiology of pediatric forearm fractures in Washington, DC. J Trauma 69(4 Suppl):S200-5
201. Watson WL, Ozanne-Smith J (1993) The use of child safety restraints with nursery furniture. J Paediatr Child Health 29(3):228–232
202. Smith GA, Dietrich AM, Garcia CT, Shields BJ (1996) Injuries to children related to shopping carts. Pediatrics 97(2):161–165
203. Smith GA, Bowman MJ, Luria JW, Shields BJ (1997) Babywalker-related injuries continue despite warning labels and public education. Pediatrics 100(2):E1
204. Loder RT, Feinberg JR (2007) Orthopaedic injuries in children with nonaccidental trauma: demographics and incidence from the 2000 kids' inpatient database. J Pediatr Orthop 27(4):421–426
205. Van As AB, Naidoo S, Craig R, Franklin J (2007) Fracture patterns in non-accidentally injured children at Red Cross Children's Hospital. SAJCH. 1(3):102–105
206. Browner EA (2013) Nursemaid's elbow (annular ligament displacement). Pediatr Rev 34(8):366–367. discussion 7
207. Campo TM (2011) A case of subluxation of the radial head: nursemaids' elbow. Adv Emerg Nurs J 33(1):8–14
208. Schunk JE (1990) Radial head subluxation: epidemiology and treatment of 87 episodes. Ann Emerg Med 19(9):1019–1023
209. Michaels MG (1989) A case of bilateral nursemaid's elbow. Pediatr Emerg Care 5(4):226–227
210. Meiner EM, Sama AE, Lee DC, Nelson M, Katz DS, Trope A (2004) Bilateral nursemaid's elbow. Am J Emerg Med 22(6):502–503
211. Vitello S, Dvorkin R, Sattler S, Levy D, Ung L (2014) Epidemiology of nursemaid's elbow. West J Emerg Med 15(4):554–557
212. Thompson GH (2001) Dislocations of the elbow. In: Beaty JH, Kasser JR (eds) Rockwood and Wilkins' fractures in children, 5th edn. Lippincott Williams en Wilkins, pp 705–739
213. Newman J (1985) "Nursemaid's elbow" in infants six months and under. J Emerg Med 2(6):403–404
214. Shaath K, Shirley E (2021) Nursemaid's elbow. Orthobullets. [25-11-2021]; Available from: https://www.orthobullets.com/pediatrics/4012/nursemaids-elbow
215. Rudloe TF, Schutzman S, Lee LK, Kimia AA (2012) No longer a "nursemaid's" elbow: mechanisms, caregivers, and prevention. Pediatr Emerg Care 28(8):771–774
216. Kunkler CE (2000) Did you check your nursemaid's elbow? Orthop Nurs 19(4):49–52. quiz 3-5
217. Yamanaka S, Goldman RD (2018) Pulled elbow in children. Can Fam Physician 64(6):439–441
218. Li N, Khoo B, Brown L, Young T (2021) Nonaxial traction mechanisms of nursemaid's elbow. Pediatr Emerg Care 37(6):e292–e2e4
219. Shah SS, Rochette LM, Smith GA (2012) Epidemiology of pediatric hand injuries presenting to United States emergency departments, 1990 to 2009. J Trauma Acute Care Surg 72(6):1688–1694
220. Nellans KW, Chung KC (2013) Pediatric hand fractures. Hand Clin 29(4):569–578
221. Wahba G, Cheung K (2018) Pediatric hand injuries: practical approach for primary care physicians. Can Fam Physician 64(11):803–810
222. Vadivelu R, Dias JJ, Burke FD, Stanton J (2006) Hand injuries in children: a prospective study. J Pediatr Orthop 26(1):29–35
223. Kreutz-Rodrigues L, Gibreel W, Moran SL, Carlsen BT, Bakri K (2020) Frequency, pattern, and treatment of hand fractures in children and adolescents: a 27-year review of 4356 pediatric hand fractures. Hand (N Y):1558944719900565
224. Chung KC, Spilson SV (2001) The frequency and epidemiology of hand and forearm fractures in the United States. J Hand Surg Am 26(5):908–915
225. Brodeur AE, Monteleone JA (1994) Child maltreatment. A clinical guide and reference. GW Med:32
226. Bauer A (n.d.) Study guide: hand trauma. POSNA (Pediatr Orthop Soc North Am). [25-11-2021]; Available from: https://posna.org/Physician-Education/Study-Guide/Hand-Trauma
227. Worlock PH, Stower MJ (1986) The incidence and pattern of hand fractures in children. J Hand Surg Br. 11(2):198–200
228. Barton NJ (1979) Fractures of the phalanges of the hand in children. Hand 11(2):134–143
229. Hastings H 2nd, Simmons BP (1984) Hand fractures in children. A statistical analysis. Clin Orthop Relat Res 188:120–130
230. Liu EH, Alqahtani S, Alsaaran RN, Ho ES, Zuker RM, Borschel GH (2014) A prospective study of pediatric hand fractures and review of the literature. Pediatr Emerg Care 30(5):299–304
231. Rajesh A, Basu AK, Vaidhyanath R, Finlay D (2001) Hand fractures: a study of their site and type in childhood. Clin Radiol 56(8):667–669
232. Mahabir RC, Kazemi AR, Cannon WG, Courtemanche DJ (2001) Pediatric hand fractures: a review. Pediatr Emerg Care 17(3):153–156
233. Johnson CF, Kaufman KL, Callendar C (1990) The hand as a target organ in child abuse. Clin Pediatr (Phila). 29(2):66–72
234. McClelland CQ, Heiple KG (1982) Fractures in the first year of life. A diagnostic dilemma. Am J Dis Child 136(1):26–29
235. Nimkin K, Spevak MR, Kleinman PK (1997) Fractures of the hands and feet in child abuse: imaging and pathologic features. Radiology 203(1):233–236
236. Kleinman PK, Morris NB, Makris J, Moles RL, Kleinman PL (2013) Yield of radiographic skeletal surveys for detection of hand, foot, and spine fractures in suspected child abuse. AJR Am J Roentgenol 200(3):641–644
237. 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? AJR Am J Roentgenol 197(1):W159–W163
238. Lindberg DM, Harper NS, Laskey AL, Berger RP (2013) Prevalence of abusive fractures of the hands, feet, spine, or pelvis on skeletal survey: perhaps "uncommon" is more common than suggested. Pediatr Emerg Care 29(1):26–29

239. Murugappan KS (2019) Pediatric femur fractures. Medscape. [25-11-2021]; Available from: https://emedicine.medscape.com/article/1246915-overview#a4
240. Buess E, Kaelin A (1998) One hundred pediatric femoral fractures: epidemiology, treatment attitudes, and early complications. J Pediatr Orthop Part B. 7(3):186–192
241. Brown D, Fisher E (2004) Femur fractures in infants and young children. Am J Public Health 94(4):558–560
242. Loder RT, O'Donnell PW, Feinberg JR (2006) Epidemiology and mechanisms of femur fractures in children. J Pediatr Orthop 26:561–566
243. Petković L, Djan I, Gajdobranski D, Marić D, Petković M (2011) Pediatric femur fractures, epidemiology and treatment. Vojnosanit Pregl 68(1):9–14
244. Baldwin K, Pandya NK, Wolfgruber H, Drummond DS, Hosalkar HS (2011) Femur fractures in the pediatric population: abuse or accidental trauma? Clini Orthop Relat Res 469(3):798–804
245. Engström Z, Wolf O, Hailer YD (2020) Epidemiology of pediatric femur fractures in children: the Swedish Fracture Register. BMC Musculoskelet Disord 21(1):796
246. Rokaya PK, Karki DB, Rawal M, Limbu D, Acharya BD, Bhandari PB (2020) Epidemiology of Femur fractures in children: a descriptive cross sectional study based on a rural population of Nepal. JNMA J Nepal Med Assoc 58(228):574–579
247. Valaikaite R, Tabard-Fougère A, Steiger C, Samara E, Dayer R, Ceroni D (2020) A retrospective epidemiological study of paediatric femoral fractures. Swiss Med Wkly 150:w20360
248. Beaty JH (2006) Fractures of the hip in children. Orthop Clin North Am 37(2):223–232. vii
249. Boardman MJ, Herman MJ, Buck B, Pizzutillo PD (2009) Hip fractures in children. J Am Acad Orthop Surg 17(3):162–173
250. Dial BL, Lark RK (2018) Pediatric proximal femur fractures. J Orthop 15(2):529–535
251. Ahn L, Andras L, Shirley E (2021) Proximal femur fractures—pediatric. Orthobullets. [25-11-2021]; Available from: https://www.orthobullets.com/pediatrics/4018/proximal-femur-fractures%2D%2Dpediatric
252. Stone KP, White K (2021) Hip fractures in children. UpToDate. [25-11-2021]; Available from: https://www.uptodate.com/contents/hip-fractures-in-children
253. Bimmel R, Bakker A, Bosma B, Michielsen J (2010) Paediatric hip fractures: a systematic review of incidence, treatment options and complications. Acta Orthop Belg 76(1):7–13
254. Miller WE (1973) Fractures of the hip in children from birth to adolescence. Clin Orthop Relat Res 92:155–188
255. Pape HC, Krettek C, Friedrich A, Pohlemann T, Simon R, Tscherne H (1999) Long-term outcome in children with fractures of the proximal femur after high-energy trauma. J Trauma 46(1):58–64
256. Davison BL, Weinstein SL (1992) Hip fractures in children: a long-term follow-up study. J Pediatr Orthop. 12(3):355–358
257. Azouz EM, Karamitsos C, Reed MH, Baker L, Kozlowski K, Hoeffel JC (1993) Types and complications of femoral neck fractures in children. Pediatr Radiol 23(6):415–420
258. Togrul E, Bayram H, Gulsen M, Kalaci A, Ozbarlas S (2005) Fractures of the femoral neck in children: long-term follow-up in 62 hip fractures. Injury 36(1):123–130
259. Shrader MW, Jacofsky DJ, Stans AA, Shaughnessy WJ, Haidukewych GJ (2007) Femoral neck fractures in pediatric patients: 30 years experience at a level 1 trauma center. Clin Orthop Relat Res 454:169–173
260. Currey JD, Butler G (1975) The mechanical properties of bone tissue in children. J Bone Joint Surg Am 57(6):810–814
261. Ratliff AH (1962) Fractures of the neck of the femur in children. J Bone Joint Surg Br 44-b:528–542
262. Bali K, Sudesh P, Patel S, Kumar V, Saini U, Dhillon MS (2011) Pediatric femoral neck fractures: our 10 years of experience. Clin Orthop Surg 3(4):302–308
263. Shrader MW, Schwab JH, Shaughnessy WJ, Jacofsky DJ (2009) Pathologic femoral neck fractures in children. Am J Orthop (Belle Mead, NJ) 38(2):83–86. discussion 6
264. Wojtowycz M, Starshak RJ, Sty JR (1980) Neonatal proximal femoral epiphysiolysis. Radiology 136(3):647–648
265. Prevot J, Lascombes P, Blanquart D, Gagneux E (1989) Labor trauma-induced epiphysiolysis of the proximal femur. 4 cases. ZKinderchir. 44(5):289–292
266. Towbin R, Crawford AH (1979) Neonatal traumatic proximal femoral epiphysiolysis. Pediatrics 63(3):456–459
267. Milgram JW, Lyne ED (1975) Epiphysiolysis of the proximal femur in very young children. Clin Orthop Relat Res 110:146–153
268. Michail JP, Theodorou S, Houliaras K, Siatis N (1958) Two cases of obstetrical separation (epiphysiolysis) of the upper femoral epiphysis: appearance of ossification centre of the femoral head fifteen-day-old child. J Bone Joint Surg Br Volume 40-b(3):477–482
269. Kennedy PC (1944) Traumatic separation of the upper femoral epiphysis. Am I Roentgenol 51:707
270. Lindseth RE, Rosene HA Jr (1971) Traumatic separation of the upper femoral epiphysis in a new born infant. J Bone Joint Surg Am 53(8):1641–1644
271. Mortens J, Christensen P (1964) Traumatic separation of the upper femoral epiphysis as an obstetric lesion. Acta Orthop Scand 34:238–250
272. Theodorou SD, Ierodiaconou MN, Mitsou A (1982) Obstetrical fracture-separation of the upper femoral epiphysis. Acta Orthop Scand 53(2):239–243
273. Kembhavi RS, James B (2015) A rare case report on bilateral intertrochanteric fractures in a child following child abuse. J Clin Diagnostic Res 9(10):Rd03-5
274. Pastor AJ, Gupta A, Press CM, Gourineni P (2012) Femoral neck fracture as the sentinel sign of child abuse in an infant: a case report. J Pediatr Orthop Part B. 21(6):587–591
275. Gholve P, Arkader A, Gaugler R, Wells L (2008) Femoral neck fracture as an atypical presentation of child abuse. Orthopedics 31(3):271
276. Paris N, Journeau P, Moh Ello N et al (2008) Bilateral upper femoral physis injury in a case of epilepsy in a young child [Décollement épiphysaire fémoral proximal bilatéral chez un nourrisson atteint d'épilepsie généralisée. A propos d'une observation]. Revue de chirurgie orthopedique et reparatrice de l'appareil moteur 94(4):403–406
277. Hayyun MF, Jamil K, Abd-Rashid AH, Ibrahim S (2021) Subcapital femoral neck tension stress fracture—a rare injury in a child: a case report. Malays Orthop J 15(1):132–134
278. Papadimitriou NG, Christophorides J, Papadimitriou A, Beslikas TA, Ventouris TN, Goulios BA (2007) Stress fractures in children: a review of 37 cases. Eur J Orthop Surg Traumatol Orthop Traumatol 17:131–137
279. Er MS, Eroglu M, Altinel L (2014) Femoral neck stress fracture in children: a case report, up-to-date review, and diagnostic algorithm. J Pediatr Orthop Part B 23(2):117–121
280. Lehman RA Jr, Shah SA (2004) Tension-sided femoral neck stress fracture in a skeletally immature patient. A case report. J Bone Joint Surg Am 86(6):1292–1295
281. Boyle MJ, Hogue GD, Heyworth BE, Ackerman K, Quinn B, Yen YM (2017) Femoral neck stress fractures in children younger than 10 years of age. J Pediatr Orthop 37(2):e96–ee9
282. Meaney JE, Carty H (1992) Femoral stress fractures in children. Skeletal Radiol 21(3):173–176
283. St Pierre P, Staheli LT, Smith JB, Green NE (1995) Femoral neck stress fractures in children and adolescents. J Pediatr Orthop 15(4):470–473
284. Walker RN, Green NE, Spindler KP (1996) Stress fractures in skeletally immature patients. J Pediatr Orthop 16(5):578–584

285. Scheerlinck T, De Boeck H (1998) Bilateral stress fractures of the femoral neck complicated by unilateral displacement in a child. J Pediatr Orthop Part B 7(3):246–248
286. Román M, Recio R, Moreno JC, Fuentes S, Collantes F (2001) Stress fracture of the femoral neck in a child. Case report and review of the literature. Acta Orthop Belg 67(3):286–289
287. Fiévez EF, Hanssen NM, Schotanus MG, van Haaren EH, Kort NP (2013) Stress fracture of the femoral neck in a child: a case report. J Pediatr Orthop Part B 22(1):45–48
288. Maezawa K, Nozawa M, Sugimoto M, Sano M, Shitoto K, Kurosawa H (2004) Stress fractures of the femoral neck in child with open capital femoral epiphysis. J Pediatr Orthop Part B 13(6):407–411
289. Lee GW, Park KS, Yoon TR, Eshnazarovich EK (2016) Bilateral femoral neck stress fracture in child: a case report. Hip Pelvis 28(3):169–172
290. Miller F, Wenger DR (1979) Femoral neck stress fracture in a hyperactive child. A case report. J Bone Joint Surg Am 61(3):435–437
291. Rinat B, Bor N, Dujovny E, Rozen N, Rubin G (2021) Pediatric femoral neck fractures after sliding in a water slide: a case report of two patients. J Orthop Case Rep 11(5):68–71
292. Blaiser RD, Hughes LO (2001) Fractures and traumatic dislocations of the hip in children. In: Beaty JH, Kasser JR (eds) Rockwood and Wilkins' fractures in children, 5th edn. Lippincott, Philadelphia, pp 913–914
293. Gaudinez RF, Heinrich SD (1989) Transphyseal fracture of the capital femoral epiphysis. Orthopedics 12(12):1599–1602
294. Swischuk LE (1997) Irritable infant and left lower extremity pain. Pediatr Emerg Care 13(2):147–148
295. De Mattos CB, Binitie O, Dormans JP (2012) Pathological fractures in children. Bone Joint Res 1(10):272–280
296. Miu A (2015) Pathological fractures of the proximal femur due to solitary bone cyst: classification, methods of treatment. J Med Life 8(4):536–543
297. Vynichakis G, Angelis S, Chandrinos M et al (2019) Pathological hip fractures in children and adolescents due to benign tumor or tumor-like lesions. J Long Term Eff Med Implants 29(2):91–99
298. Swiontkowski MF, Winquist RA (1986) Displaced hip fractures in children and adolescents. J Trauma. 26(4):384–388
299. Ortiz EJ, Isler MH, Navia JE, Canosa R (2005) Pathologic fractures in children. Clin Orthop Relat Res 432:116–126
300. Morcos MW, Hamdy RC, Fassier F, Saran N (2019) Treatment of Femur neck fracture in children with osteogenesis imperfecta: two case reports. JBJS Case Connect 9(4):e0449
301. Rewers A, Hedegaard H, Lezotte D et al (2005) Childhood femur fractures, associated injuries, and sociodemographic risk factors: a population-based study. Pediatrics 115(5):e543-52
302. Kasser JR, Beaty JH (2001) Femoral shaft fractures. In: Beaty JH, Kasser JR (eds) Rockwood and Wilkins' Fractures in children, 5th edn. Lippincott Williams and Wilkins, pp 941–980
303. von Heideken J, Svensson T, Blomqvist P, Haglund-Åkerlind Y, Janarv PM (2011) Incidence and trends in femur shaft fractures in Swedish children between 1987 and 2005. J Pediatr Orthop 31(5):512–519
304. Hinton RY, Lincoln A, Crockett MM, Sponseller P, Smith G (1999) Fractures of the femoral shaft in children. Incidence, mechanisms, and sociodemographic risk factors. J Bone Joint Surg Am 81(4):500–509
305. Rennie L, Court-Brown CM, Mok JY, Beattie TF (2007) The epidemiology of fractures in children. Injury 38(8):913–922
306. Bridgman S, Wilson R (2004) Epidemiology of femoral fractures in children in the West Midlands region of England 1991 to 2001. J Bone Joint Surg Br Volume 86(8):1152–1157
307. Edgington J, Shirley E, Sink E (2021) Femoral shaft fractures—pediatric. Orthobullets. [26-11-2021]; Available from: https://www.orthobullets.com/pediatrics/4019/femoral-shaft-fractures%2D%2Dpediatric
308. Galano GJ, Vitale MA, Kessler MW, Hyman JE, Vitale MG (2005) The most frequent traumatic orthopaedic injuries from a national pediatric inpatient population. J Pediatr Orthop 25(1):39–44
309. Stone KP, White K. Femoral shaft fractures in children. UpToDate; 2021. [26-11-2021]; Available from: https://www.uptodate.com/contents/femoral-shaft-fractures-in-children.
310. Mooney DP, Forbes P (2004) Trends in inpatient pediatric trauma care in new England. J Trauma 57(6):1241–1245
311. Talbot C, Davis N, Majid I et al (2018) Fractures of the femoral shaft in children: national epidemiology and treatment trends in England following activation of major trauma networks. Bone Joint J 100-b(1):109–118
312. Keany JE, McKeever D (2019) Femoral shaft fractures in emergency medicine. Medscape. [26-11-2021]; Available from: https://emedicine.medscape.com/article/824856-overview
313. Medda S, Unger T, Halvorson J (2021) Diaphyseal Femur fracture. StatPearls. StatPearls Publishing, Treasure Island (FL)
314. Arioz DT, Koken GN, Koken R, Kose KC, Cevrioglu AS (2008) Isolated intrauterine femoral fracture in an otherwise normal fetus. J Obstet Gynaecol Res 34(1):92–94
315. Senanayake H, Anandakumar C, de Silva MV (2003) Mid-trimester fracture of femur in a normal fetus. J Obstet Gynaecol Res 29(3):186–188
316. Alhusen JL, Ray E, Sharps P, Bullock L (2015) Intimate partner violence during pregnancy: maternal and neonatal outcomes. J Womens Health (Larchmt) 24(1):100–106
317. Christensen EE, Dietz GW (1978) A radiographically documented intra-uterine femoral fracture. Br J Radiol 51(610):830–831
318. Bucholz R, Mauldin D (1978) Prenatal diagnosis of intrauterine fetal fracture. A case report. J Bone Joint Surg Am 60(5):712–713
319. Wilkinson AD (1898) Compound intra-uterine fracture of the femur, with report of a case. JAMA XXXI(10):536
320. Scheier M, Peter M, Hager C, Lang T, Barvinek A, Marth C (2010) Spontaneous isolated midtrimester fracture of tibia and fibula in a normal fetus with in utero healing and good long-term outcome. Fetal Diagn Ther 28(1):58–60
321. Yu M, Xu D, Zhang A, Shen J (2018) Spontaneous fetal femoral fracture: a case report and literature review. J Int Med Res 46(3):1282–1287
322. Raabe E, Dammer U, Kehl S, Beckmann MW, Faschingbauer F. Spontaneous fetal femur fracture. Ultraschall in der Medizin (Stuttgart, Germany : 1980). 2013;34 - KS_CS6_04.
323. Alonso JA, Wright DM, Sochart DH (2005) Intrauterine femoral fracture diagnosed at birth—maternal abdominal trauma versus non-accidental injury. Injury Extra 36:432–433
324. Morris S, Cassidy N, Stephens M, McCormack D, McManus F (2002) Birth-associated femoral fractures: incidence and outcome. J Pediatr Orthop 22(1):27–30
325. Toker A, Perry ZH, Cohen E, Krymko H (2009) Cesarean section and the risk of fractured femur. Isr Med Assoc J 11(7):416–418
326. Kancherla R, Sankineani SR, Naranje S et al (2012) Birth-related femoral fracture in newborns: risk factors and management. J Child Orthop 6(3):177–180
327. Frik S (2016) Management of birth-associated subtrochanteric femur fractures. Acta Orthop Belg 82(4):850–853
328. Cebesoy FB, Cebesoy O, Incebiyik A (2009) Bilateral femur fracture in a newborn: an extreme complication of cesarean delivery. Arch Gynecol Obstet 279(1):73–74
329. Rubin A (1964) Birth injuries: Incidence, mechanisms and end results. Obstet Gynecol 23:218–221
330. Barnes AD, Van Geem TA (1985) Fractured femur of the newborn at cesarean section. A case report. J Reprod Med 30(3):203–205

331. Kanai Y, Honda Y, Honda T, Sanpei M (2018) Delayed birth-related Femur fracture after cesarean section: a case report. AJP Rep 8(3):e158–ee60
332. Cumming WA (1979) Neonatal skeletal fractures. Birth trauma or child abuse? J Can Assoc Radiol 30(1):30–33
333. Hosokawa T, Yamada Y, Sato Y, Tanami Y, Oguma E (2017) Subperiosteal new bone and callus formations in neonates with femoral shaft fracture at birth. Emerg Radiol 24(2):143–148
334. Crompton S, Messina F, Klafkowski G, Hall C, Offiah AC (2021) Validating scoring systems for fracture healing in infants and young children: pilot study. Pediatr Radiol
335. Scherl SA, Miller L, Lively N, Russinoff S, Sullivan CM, Tornetta P 3rd. (2000) Accidental and nonaccidental femur fractures in children. Clin Orthop Relat Res 376:96–105
336. Blakemore LC, Loder RT, Hensinger RN (1996) Role of intentional abuse in children 1 to 5 years old with isolated femoral shaft fractures. J Pediatr Orthop. 16(5):585–588
337. 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
338. Hui C, Joughin E, Goldstein S et al (2008) Femoral fractures in children younger than three years: the role of nonaccidental injury. J Pediatr Orthop 28(3):297–302
339. John R, Sharma S, Raj GN et al (2017) Current concepts in paediatric femoral shaft fractures. Open Orthop J 11:353–368
340. Hedlund R, Lindgren U (1986) The incidence of femoral shaft fractures in children and adolescents. J Pediatr Orthop 6(1):47–50
341. Daly KE, Calvert PT (1991) Accidental femoral fracture in infants. Injury 22(4):337–338
342. Loder RT (1987) Pediatric polytrauma: orthopaedic care and hospital course. J Orthop Trauma 1(1):48–54
343. Shrader MW, Bernat NM, Segal LS (2011) Suspected nonaccidental trauma and femoral shaft fractures in children. Orthopedics 34(5):360
344. Mughal MA, Dix-Peek SI, Hoffman EB (2013) The epidemiology of femur shaft fractures in children. SA Orthop J 12(4):23–27
345. Cornell EM, Powell EC (2018) Skeletal survey yield in young children with Femur fractures. J Emerg Med 55(6):758–763
346. Ali M, Padhye K, Gauthier L (2021) Pediatric femoral shaft fractures secondary to tombstone uprooting—Two case reports. Trauma Case Rep 32:100416
347. Hobbs CJ, Wynne JM (1990) The sexually abused battered child. Arch Dis Child 65(4):423–427
348. Johnson K, Chapman S, Hall CM (2004) Skeletal injuries associated with sexual abuse. Pediatr Radiol 34(8):620–623
349. Kleinman PK (1998) The lower extremity. In: Kleinman PK (ed) Diagnostic imaging of child abuse. Mosby, pp 26–71
350. Frasier LD (2003) Child abuse or mimic. Consult Pediatr 2:212–215
351. Beals RK, Tufts E (1983) Fractured femur in infancy: the role of child abuse. J Pediatr Orthop 3(5):583–586
352. Loos MHJ, Allema WM, Bakx R, Stoel RD, van Rijn RR, Karst WA (2021) Paediatric femur fractures-the value of contextual information on judgement in possible child abuse cases: are we bias? Eur J Pediatr 180(1):81–90
353. Mboutol-Mandavo C, N'Dour O, Ouedraogo SF, Missengue-Bosseba R, Ndiaye D, Ngom G (2016) Fractures du nouveau-né et du nourrisson secondaires au massage traditionnel [Newborn and infant fractures secondary to traditional massage]. Arch Pediatr: organe officiel de la Societe francaise de pediatrie 23(9):963–965
354. Siddiqui SA, Singh MV, Shrivastava A, Maurya M, Gaur VK, Kumar D (2020) Femoral shaft fracture following oil massage in neonates: a single-centre experience. Trop Doct 50(4):387–389
355. Greeley CS, Donaruma-Kwoh M, Vettimattam M, Lobo C, Williard C, Mazur L (2013) Fractures at diagnosis in infants and children with osteogenesis imperfecta. J Pediatr Orthop 33(1):32–36
356. Fry K, Hoffer MM, Brink J (1976) Femoral shaft fractures in brain-injured children. J Trauma 16(5):371–373
357. Torwalt CR, Balachandra AT, Youngson C, de Nanassy J (2002) Spontaneous fractures in the differential diagnosis of fractures in children. J Forensic Sci 47(6):1340–1344
358. Katz JF (1953) Spontaneous fractures in paraplegic children. J Bone Joint Surg Am 35-a(1):220–226
359. Ju DG, Mogayzel PJ Jr, Sponseller PD, Familiari F, McFarland EG (2016) Bilateral midshaft femoral fractures in an adolescent baseball player. J Cyst Fibros 15(4):e41–e43
360. Prince HG, Clay NR (1988) A pathological fracture of the femur through a histiocytic granuloma. Injury 19(2):124–125
361. Chaib B, Malhotra K, Khoo M, Saifuddin A (2021) Pathological fracture in paediatric bone tumours and tumour-like lesions: A predictor of benign lesions? Br J Radiol 94(1125):20201341
362. Cherix S, Bildé Y, Becce F, Letovanec I, Rüdiger HA (2014) Multiple non-ossifying fibromas as a cause of pathological femoral fracture in Jaffe-Campanacci syndrome. BMC Musculoskelet Disord 15:218
363. Srivastava A, Aggarwal AN, Mishra P, Bhateja D (2016) Femoral fracture acting as an "ominous masquerade" in a 7-year-old child. J Clin Orthop Trauma 7(Suppl 1):27–29
364. Pandey PK, Pawar I, Gupta J (2016) Physeal separation and complete resorption of femoral head associated with chronic osteomyelitis of femoral diaphysis in a young child: rare case report. J Pediatr Orthop Part B 25(6):529–532
365. Akinyoola AL, Orimolade EA, Yusuf MB (2008) Pathologic fractures of long bones in Nigerian children. J Child Orthop 2(6):475–479
366. Eid AM, Hafez MA (2002) Traumatic injuries of the distal femoral physis. Retrospective study on 151 cases. Injury 33(3):251–255
367. Arkader A, Warner WC Jr, Horn BD, Shaw RN, Wells L (2007) Predicting the outcome of physeal fractures of the distal femur. J Pediatr Orthop 27(6):703–708
368. Duffy S, Gelfer Y, Trompeter A, Clarke A, Monsell F (2021) The clinical features, management options and complications of paediatric femoral fractures. Eur J Orthop Surg Traumatol Orthop Traumatol 31(5):883–892
369. Bellamy JT, Ward LA, Fletcher ND (2021) Evaluation of pediatric distal femoral physeal fractures and the factors impacting poor outcome requiring further corrective surgery. J Pediatr Orthop Part B 30(1):6–12
370. Stone KP, White K. Distal femoral fractures in children. UpToDate; 2021 [26-11-2021]; Available from: https://www.uptodate.com/contents/distal-femoral-fractures-in-children.
371. Smith NC, Parker D, McNicol D (2001) Supracondylar fractures of the femur in children. J Pediatr Orthop 21(5):600–603
372. Fox S. Distal femoral physeal fractures. Don't Forget the Bubbles; 2021 [26-11-2021]; Available from: https://dontforgetthebubbles.com/distal-femoral-physeal-fractures/.
373. Neal K (2021) Distal Femur Fractures. POSNA. [26-11-2021]; Available from: https://posna.org/Physician-Education/Study-Guide/Distal-Femur-Fractures
374. Shaath K, Shirley E, Skaggs DL (2021) Distal femoral physeal fractures—pediatric. Orthobullets. [26-11-2021]; Available from: https://www.orthobullets.com/pediatrics/4020/distal-femoral-physeal-fractures%2D%2Dpediatric
375. Pritchett JW (1992) Longitudinal growth and growth-plate activity in the lower extremity. Clinical Orthop Relat Res 275:274–279
376. Leavitt PH (1951) Traumatic separation of the lower-femoral epiphysis. N Engl J Med 245:565–566
377. El-Zawawy HB, Silva MJ, Sandell LJ, Wright RW (2005) Ligamentous versus physeal failure in murine medial collateral ligament biomechanical testing. J Biomech 38(4):703–706

378. Price CT (2021) Extra-articular injuries of the knee. TeachMeOrthopedics. [26-11-2021]; Available from: https://teachmeorthopedics.info/extra-articular-injuries-of-the-knee/
379. Ozturk M, Yurtbay A, Keskin D, Polat AV, Selcuk MB (2019) Subperiosteal hemorrhage due to a distal femoral physeal fracture in a neonate. North Clin Istanb 6(3):312–314
380. Jain R, Bielski RJ (2001) Fracture of lower femoral epiphysis in an infant at birth: a rare obstetrical injury. J Perinatol 21(8):550–552
381. Rutherford Y, Fomufod AK, Gopalakrishnan LJ, Beeks EC (1983) Traumatic distal femoral periostitis of the newborn: a breech delivery birth injury. J Natl Med Assoc 75(10):933–935
382. Clarke TA, Edwards DK, Merritt A, Young LW (1982) Radiological case of the month. Neonatal fracture of the femur: iatrogenic? Am J Dis Child 136(1):69–70
383. Mangurten HH, Puppala B, Knuth A (2005) Neonatal distal femoral physeal fracture requiring closed reduction and pinning. J Perinatol 25(3):216–219
384. Trier H (1992) Epiphysiolysis in the distal femur as a birth injury in Cesarean section [Epifysiolyse i distale femur som fødselslaesion ved sectio]. UgeskrLaeger 154(22):1574–1575
385. Alexander J, Gregg JE, Quinn MW (1987) Femoral fractures at caesarean section. Case reports. Br J Obstet Gynaecol 94(3):273
386. McCollough FL, McCarthy RE (1988) Bilateral distal femoral epiphyseal fractures following home delivery: a case report. J Ark Med Soc 84(9):364–366
387. Haney SB, Boos SC, Kutz TJ, Starling SP (2009) Transverse fracture of the distal femoral metadiaphysis: a plausible accidental mechanism. Pediatr Emerg Care 25(12):841–844
388. Rex C, Kay PR (2000) Features of femoral fractures in nonaccidental injury. J Pediatr Orthop 20(3):411–413
389. Arkader A, Friedman JE, Warner WC Jr, Wells L (2007) Complete distal femoral metaphyseal fractures: a harbinger of child abuse before walking age. J Pediatr Orthop 27(7):751–753
390. Grant P, Mata MB, Tidwell M (2001) Femur fracture in infants: a possible accidental etiology. Pediatrics 108(4):1009–1011
391. Pickett WJ 3rd, Johnson JF, Enzenauer RW (1982) Case report 192. Neonatal fractures mimicking abuse secondary to physical therapy. Skeletal Radiol 8(1):85–86
392. Aroojis AJ, Gajjar SM, Johari AN (2007) Epiphyseal separations in spastic cerebral palsy. J Pediatr Orthop Part B 16(3):170–174
393. Aroojis AJ, Johari AN (2000) Epiphyseal separations after neonatal osteomyelitis and septic arthritis. J Pediatr Orthop 20(4):544–549
394. Parsch K (1991) Origin and treatment of fractures in spina bifida. Eur J Pediatr Surg 1(5):298–305
395. Vander Have KL, Ganley TJ, Kocher MS, Price CT, Herrera-Soto JA (2010) Arthrofibrosis after surgical fixation of tibial eminence fractures in children and adolescents. Am J Sports Med 38(2):298–301
396. Cheng JC, Ng BK, Ying SY, Lam PK (1999) A 10-year study of the changes in the pattern and treatment of 6,493 fractures. J Pediatr Orthop 19(3):344–350
397. Chapman J, Cohen J. Tibial and fibular fractures in children. UpToDate; 2020 [26-11-2021]; Available from: https://www.uptodate.com/contents/tibial-and-fibular-shaft-fractures-in-children.
398. Mashru RP, Herman MJ, Pizzutillo PD (2005) Tibial shaft fractures in children and adolescents. J Am Acad Orthop Surg 13(5):345–352
399. Shannak AO (1988) Tibial fractures in children: follow-up study. J Pediatr Orthop 8(3):306–310
400. Weber B, Kalbitz M, Baur M, Braun CK, Zwingmann J, Pressmar J (2021) Lower leg fractures in children and adolescents-comparison of conservative vs ECMES treatment. Front Pediatr 9:597870
401. Cruz AI Jr, Raducha JE, Swarup I, Schachne JM, Fabricant PD (2019) Evidence-based update on the surgical treatment of pediatric tibial shaft fractures. Curr Opin Pediatr 31(1):92–102
402. Sarmiento A, Gersten LM, Sobol PA, Shankwiler JA, Vangsness CT (1989) Tibial shaft fractures treated with functional braces. Experience with 780 fractures. J Bone Joint Surg Br Volume 71(4):602–609
403. Audigé L, Slongo T, Lutz N, Blumenthal A, Joeris A (2017) The AO pediatric comprehensive classification of long bone fractures (PCCF). Acta Orthop 88(2):133–139
404. Kaplan M, Dollberg M, Wajntraub G, Itzchaki M (1987) Fractured long bones in a term infant delivered by cesarian section. Pediatr Radiol 17(3):256–257
405. Dolivet E, Delesalle C, Morello R et al (2018) A case-control study about foetal trauma during caesarean delivery. J Gynecol Obstet Hum Reprod 47(7):325–329
406. Mileto C, Hashem J, Culbertson MD, Sadeghpour R, Tucci J (2014) Proximal tibial epiphyseal fracture in a newborn following cesarean section: a case report. JBJS Case Connect 4(3):e84
407. Moineau G, Plint A (2005) Tibial fractures possibly linked to use of a baby stationary activity center. Pediatr Emerg Care 21(3):181–183
408. Paddock M, Horton D, Offiah AC (2020) Bilateral fibular fractures in a pre-ambulant infant. Pediatr Radiol
409. Mellick LB, Milker L, Egsieker E (1999) Childhood accidental spiral tibial (CAST) fractures. Pediatr Emerg Care 15(5):307–309
410. Mellick LB, Reesor K (1990) Spiral tibial fractures of children: a commonly accidental spiral long bone fracture. Am J Emerg Med 8(3):234–237
411. Tenenbein M, Reed MH, Black GB (1990) The toddler's fracture revisited. Am J Emerg Med 8(3):208–211
412. Sarmah A (1995) 'Toddler's fracture'? A recognised entity. Arch Dis Child 72(4):376
413. D'Souza LG, Hynes DE, McManus F, O'Brien TM, Stephens MM, Waide V (1996) The bicycle spoke injury: an avoidable accident? Foot Ankle Int 17(3):170–173
414. Roffman M, Moshel M, Mendes DG (1980) Bicycle spoke injuries. J Trauma 20(4):325–326
415. Slaar A, Karsten IH, Beenen LF et al (2015) Plain radiography in children with spoke wheel injury: A retrospective cohort study. Eur J Radiol 84(11):2296–2300
416. Mellick LB, Reesor K, Demers D, Reinker KA (1988) Tibial fractures of young children. Pediatr Emerg Care 4(2):97–101
417. Banaszkiewicz PA, Scotland TR, Myerscough EJ (2002) Fractures in children younger than age 1 year: importance of collaboration with child protection services. J Pediatr Orthop 22(6):740–744
418. Coffey C, Haley K, Hayes J, Groner JI (2005) The risk of child abuse in infants and toddlers with lower extremity injuries. J Pediatr Surg 40(1):120–123
419. Eren MB, Bilgiç E, Çetin S, Deresoy FA, Öztürk T, Balta O (2021) Sixteen fractures in a seven-month-old child caused by nonaccidental trauma. Case Rep Orthop Res:185–191
420. Patel NK, Horstman J, Kuester V, Sambandam S, Mounasamy V (2018) Pediatric tibial shaft fractures. Indian J Orthop 52(5):522–528
421. Taitz J, Moran K, O'Meara M (2004) Long bone fractures in children under 3 years of age: is abuse being missed in Emergency Department presentations? J Paediatr Child Health 40(4):170–174
422. Clarke NM, Shelton FR, Taylor CC, Khan T, Needhirajan S (2012) The incidence of fractures in children under the age of 24 months—in relation to non-accidental injury. Injury 43(6):762–765
423. Petnehazy T, Schalamon J, Hartwig C et al (2015) Fractures of the hallux in children. Foot Ankle Int 36(1):60–63
424. Kay RM, Tang CW (2001) Pediatric foot fractures: evaluation and treatment. J Am Acad Orthop Surg 9(5):308–319

425. Rammelt S, Godoy-Santos AL, Schneiders W, Fitze G, Zwipp H (2016) Foot and ankle fractures during childhood: review of the literature and scientific evidence for appropriate treatment. Revista brasileira de ortopedia. 51(6):630–639
426. Singer G, Cichocki M, Schalamon J, Eberl R, Höllwarth ME (2008) A study of metatarsal fractures in children. J Bone Joint Surg Am 90(4):772–776
427. Ribbans WJ, Natarajan R, Alavala S (2005) Pediatric foot fractures. Clin Orthop Relat Res 432:107–115
428. Mäyränpää MK, Mäkitie O, Kallio PE (2010) Decreasing incidence and changing pattern of childhood fractures: A population-based study. J Bone Miner Res 25(12):2752–2759
429. Owen RJ, Hickey FG, Finlay DB (1995) A study of metatarsal fractures in children. Injury 26(8):537–538
430. Cooper C, Dennison EM, Leufkens HG, Bishop N, van Staa TP (2004) Epidemiology of childhood fractures in Britain: a study using the general practice research database. J Bone Miner Res 19(12):1976–1981
431. Rosendahl K, Myklebust R, Ulriksen KF et al (2021) Incidence, pattern and mechanisms of injuries and fractures in children under two years of age. BMC Musculoskelet Disord 22(1):555
432. Jaffe AC, Lasser DH (1977) Multiple metatarsal fractures in child abuse. Pediatrics 60(4 Pt 2):642–643
433. Herrera-Soto JA, Crawford AH, Loveless EA (2005) Ossifying subperiosteal hematoma associated with neurofibromatosis type 1. Diagnostic hesitation: a case report and literature review. J Pediatr Orthop Part B 14(1):51–54
434. Steenbrugge F, Verstraete K, Poffyn B, Uyttendaele D, Verdonk R (2001) Recurrent massive subperiosteal hematoma in a patient with neurofibromatosis. Eur Radiol 11(3):480–483
435. Lavell A, Jones CW, Wong D, Counsel P, Carey-Smith R (2017) Plexiform neurofibroma causing an ossifying subperiosteal haematoma: a rare case in the tibia of an 11-year-old girl. Skeletal Radiol 46(10):1405–1413
436. Blitman NM, Levsky JM, Villanueva-Siles E, Thornhill BA (2007) Spontaneous hemorrhage simulating rapid growth of a benign subperiosteal plexiform neurofibroma. Pediatr Radiol 37(9):925–928
437. Kenan S, Abdelwahab IF, Klein MJ, Hermann G, Lewis MM (1993) Lesions of juxtacortical origin (surface lesions of bone). Skeletal Radiol 22(5):337–357
438. Islam O, Soboleski D, Symons S, Davidson LK, Ashworth MA, Babyn P (2000) Development and duration of radiographic signs of bone healing in children. AJR Am J Roentgenol 175(1):75–78
439. Prosser I, Lawson Z, Evans A et al (2012) A timetable for the radiologic features of fracture healing in young children. AJR Am J Roentgenol 198(5):1014–1020
440. Prosser I, Maguire S, Harrison SK, Mann M, Sibert JR, Kemp AM (2005) How old is this fracture? Radiologic dating of fractures in children: a systematic review. AJR Am J Roentgenol 184(4):1282–1286
441. Warner C, Maguire S, Trefan L, Miller A, Weinman J, Fadell M (2017) A study of radiological features of healing in long bone fractures among infants less than a year. Skeletal Radiol 46(3):333–341
442. Rana RS, Wu JS, Eisenberg RL (2009) Periosteal reaction. AJR Am J Roentgenol 193(4):W259–W272
443. Ved N, Haller JO (2002) Periosteal reaction with normal-appearing underlying bone: a child abuse mimicker. Emerg Radiol 9(5):278–282
444. Kwon DS, Spevak MR, Fletcher K, Kleinman PK (2002) Physiologic subperiosteal new bone formation: prevalence, distribution, and thickness in neonates and infants. AJR Am J Roentgenol 179(4):985–988
445. Herring W. Growth arrest lines. Learning Radiology. Recognizing the basics; 2020 [27-11-2021]; Available from: http://learningradiology.com/archives2008/COW%20334-Growth%20arrest%20lines/growtharrestcorrect.htm.
446. Bell DJ, Morgan MA (2021) Growth arrest lines. Radiopaedia. [27-11-2021]; Available from: https://radiopaedia.org/articles/growth-arrest-lines
447. Harris HA (1926) The growth of the long bones in childhood with special reference to certain bony striations of the metaphysis and the role of vitamins. Arch Intern Med 38:785–793
448. Harris HA (1927) Cessation of growth in long bones in health and disease. Mayo Clin Proc 2:228–229
449. Park EA (1964) The imprinting of nutrional disturbances on the growing bone. Pediatrics 33(Suppl):815–862
450. Kennedy JW, Irwin GJ, Huntley JS (2014) Growth arrest lines and intra-epiphyseal silhouettes: a case series. BMC Res Notes 7:27
451. Boyages SC, Halpern JP, Maberly GF et al (1988) A comparative study of neurological and myxedematous endemic cretinism in western China. J Clin Endocrinol Metab 67(6):1262–1271
452. Rosen RA, Deshmukh SM (1985) Growth arrest recovery lines in hypoparathyroidism. Radiology 155(1):61–62
453. Bessler W (1982) Vertebral growth arrest lines after Cushing's syndrome. A case report. Diagn Imaging 51(6):311–315
454. Fiszman P, Ansell BM, Renton P (1981) Radiological assessment of knees in juvenile chronic arthritis (juvenile rheumatoid arthritis). Scand J Rheumatol 10(2):145–152
455. Schwartz AM, Leonidas JC (1984) Methotrexate osteopathy. Skeletal Radiol 11(1):13–16
456. Meister B, Gassner I, Streif W, Dengg K, Fink FM (1994) Methotrexate osteopathy in infants with tumors of the central nervous system. Med Pediatr Oncol 23(6):493–496
457. Bar-On E, Beckwith JB, Odom LF, Eilert RE (1993) Effect of chemotherapy on human growth plate. J Pediatr Orthop 13(2):220–224
458. Etxebarria-Foronda I, Gorostiola-Vidaurrazaga L (2013) Zebra lines: Radiological repercussions of the action of bisphosphonates on the immature skeleton. Rev Osteoporos Metab Miner 5(1):39–41
459. Silverman FN (1994) Variants due to diseases of bone. In: Silverman FN, Kuhn JP (eds) Caffey's pediatric x-ray diagnosis: an integrated imaging approach, 9th edn. Mosby, pp 1521–1527
460. Patton RG, Gardner LI (1967) Growth failure in maternal deprivation. Charles C Thomas, Springfield, USA
461. Patton RG, Gardner LI (1962) Influence of family environment on growth: the syndrome of 'maternal deprivation'. Pediatrics 30(6):957–962
462. Khadilkar VV, Frazer FL, Skuse DH, Stanhope R (1998) Metaphyseal growth arrest lines in psychosocial short stature. Arch Dis Child 79(3):260–262
463. Zapala MA, Tsai A, Kleinman PK (2016) Growth recovery lines are more common in infants at high vs. low risk for abuse. Pediatr Radiol 46(9):1275–1281
464. Spiller LR, Kellogg ND, Mercado-Deane MG, Zarka AI, Gelfond JAL (2020) Growth recovery lines: a specific indicator of child abuse and neglect? Pediatr Radiol 50(2):207–215
465. Obdeijn MC, van der Vlies CH, van Rijn RR (2010) Capitate and hamate fracture in a child: the value of MRI imaging. Emerg Radiol 17(2):157–159
466. UpToDate. Ossification centers of the elbow. UpToDate; 2021 [16-12-2021]; Available from: https://www.uptodate.com/contents/image?imageKey=EM%2F73226&topicKey=EM%2F6537&source=see_link.
467. Shahareh B (2021) Radius and ulnar shaft fractures. Orthobullets. [25-11-2021]; Available from: https://www.orthobullets.com/trauma/1025/radius-and-ulnar-shaft-fractures
468. Kanat Pektaş M, Koyuncu H, Kundak AA (2019) Long bone fractures in neonatal intensive care units of Afyonkarahisar: Five-year's experience. Turk J Obstet Gynecol. 16(4):219–223

469. Gogi N, Deriu L (2017) Common paediatric lower limb injuries. Surgery (Oxford) 35(1):27–32
470. Norvell JG (2017) Tibia and Fibula fracture in the ED. Medscape. [26-11-2021]; Available from: https://emedicine.medscape.com/article/826304
471. Macknet D, Yen Y, Joughin E (2021) Tibial eminence fracture. Orthobullets. [26-11-2021]; Available from: https://www.orthobullets.com/pediatrics/4022/tibial-eminence-fracture
472. Fares A (2021) Tibial plafond fractures. Orthobullets. [26-11-2021]; Available from: https://www.orthobullets.com/trauma/1046/tibial-plafond-fractures
473. Macknet D, Swarup I, Sink E (2021) Proximal Tibia metaphyseal fractures—pediatric. Orthobullets. [26-11-2021]; Available from: https://www.orthobullets.com/pediatrics/4025/proximal-tibia-metaphyseal-fractures%2D%2Dpediatric
474. Edgington J, Swarup I, Vitale M (2021) Tibial shaft fractures—pediatric. Orthobullets. [26-11-2021]; Available from: https://www.orthobullets.com/pediatrics/4026/tibial-shaft-fractures%2D%2Dpediatric
475. Polyzois VD, Vasiliadis E, Zgonis T, Ayazi A, Gkiokas A, Beris AE (2006) Pediatric fractures of the foot and ankle. Clin Podiatr Med Surg 23(2):241–255. v
476. Chaturvedi A, Mann L, Cain U, Chaturvedi A, Klionsky NB (2020) Acute fractures and dislocations of the ankle and foot in children. Radiographics 40(3):754–774

Accidental Trauma

13

Rick R. van Rijn, Roel Bakx, Heike C. Terlingen, and Rob A. C. Bilo

Contents

13.1 Introduction

As discussed in previous chapters fractures in children occur regularly and in most cases the circumstances will be accidental. If a physician finds a fracture in a child, an extensive differential diagnosis is possible. In this chapter, the different accidental trauma scenarios are discussed and illustrated with examples.

13.2 Birth Trauma

13.2.1 Introduction

In older children, pain is often an indication for the presence of a fracture. However, in neonates it is difficult to establish pain. Often the presence of a fracture can only be established by behaviour, muscle tone, heartbeat, and symptoms such as nausea and vomiting or limited use of a body part [1].

R. R. van Rijn
Department of Radiology and Nuclear Medicine, Amsterdam UMC, University of Amsterdam, Amsterdam, The Netherlands

Department of Forensic Medicine, Netherlands Forensic Institute, The Hague, The Netherlands

R. Bakx
Paediatric Surgical Centre Amsterdam, Amsterdam UMC, Free University, Amsterdam, The Netherlands

H. C. Terlingen
Department of Forensic Medicine, Netherlands Forensic Institute, The Hague, The Netherlands

R. A. C. Bilo (✉)
Veilig Thuis Rotterdam Rijnmond (Center for the Reporting of Child Abuse, Domestic Violence and Elder Abuse), Rotterdam, The Netherlands

R. A. C. Bilo et al. (eds.), *Forensic Aspects of Paediatric Fractures*, https://doi.org/10.1007/978-3-031-12041-1_13

Fractures resulting from birth are not always diagnosed immediately post-partum, unless there are obvious symptoms, such as a clearly visible swelling and/or abnormal position. It is quite likely that physicians will completely overlook some fractures due to the lack of obvious symptoms. Research by Morris et al. showed that there was a delay in diagnosis in the majority of children that had sustained a birth-related femur fracture [2]. When there are no clinical symptoms, skull fractures may also be overlooked, even after a vacuum-assisted vaginal delivery. Simonson et al. showed in a study in 913 successful vacuum-assisted, full-term deliveries that of the 235 neonates admitted to the neonatal intensive care skull fracture were present in 5.0% of cases [3]. Clavicle fractures too are often diagnosed as late as several weeks after birth, due to the then-present callus formation [4]. Birth trauma as a cause of fractures is also discussed in several other chapters in this book.

13.2.2 Incidence and Prevalence of Fractures Resulting from Birth

Birth-related fractures occur infrequently. In the medical literature, a great number of studies on the incidence of birth-related fractures can be found (Table 13.1) [5–14]. Based on these publications it can be established that the clavicle fracture is most prevalent (Fig. 13.1), followed by fractures of the humerus (Fig. 13.2), femur (Fig. 13.3), and skull. Rib fractures are only reported in exceptional situations [15].

Many of the fractures that have been described to occur in non-accidental trauma, have also been reported, usually in case reports, as a birth trauma (Fig. 13.4a, b). Hence, it is essential that in the post-partum period, a thorough obstetric history is taken. In their research population, Bhat et al. found a higher incidence of fractures reported in cases with-

Table 13.1 Incidence of birth-related fractures

Authors	Year	Babies (*n*)	Delivery	Fractures (*n* (%))	Location (*n, % of total fractures)*)
Rubin [5]	1964	15,435		51 (0.33)	• Clavicle (43, 0.28) • Humerus (7, 0.05) • Skull (1, 0.01)
Camus et al. [6]	1985	20,409		123 (0.6)	• Clavicle (105, 0.51) • Humerus (7, 0.03) • Skull (7, 0.03) • Femoral shaft (2, 0.01) • Epiphysis (2, 0.01)
Bhat et al. [7]	1994	34,946		35 (0.1)	• Clavicle (16, 0.05) • Humerus (7, 0.02) • Femur (5, 0.01) • Skull (4, 0.01) • Orbit (1, 0.003) • Epiphysis distal femur (1, 0.003) • Dislocation elbow (1, 0.003)
Groenendaal & Hukkelhoven [9]	2007	158,035	Vaginal and caesarean	1174 (0.74)	• Clavicle[a] • Humerus • Femur • No other fractures were mentioned
Basha t al [209].	2013	34,519		10 (0.03)	• Femur (7, 0.02) • Tibia (1, 0.003) • Humerus (2, 0.006)
Ahn et al. [12] [b]	2015	77,543	Vaginal and caesarean	319 (0.41)	• Clavicle (319, 0.41)
Suleiman et al. [210]	2016	5030	Vaginal, instrumented, and caesarean	27 (0.54)	• Clavicle (20, 0.40) • Humerus (3, 0.06) • Acromion (1, 0.02) • Not reported (3, 0.06)
		36,286	Caesarean	19 (0.05)	• Clavicle (19, 0.05)
Vitner et al. [10]	2019	4534	Vacuum-assisted	163 (3.6)	• Clavicle (86, 1.9) • Humerus (50, 1.1) • Skull (27, 0.6)
von Heideker et al. [211][c]	2020	1,855,267		233 (0.012)	• Humerus (188, 0.01) • Femur (45, 0.002)

[a] Number of fractures not available
[b] Focus on clavicle fractures only
[c] Focus on humerus and femur fracture only

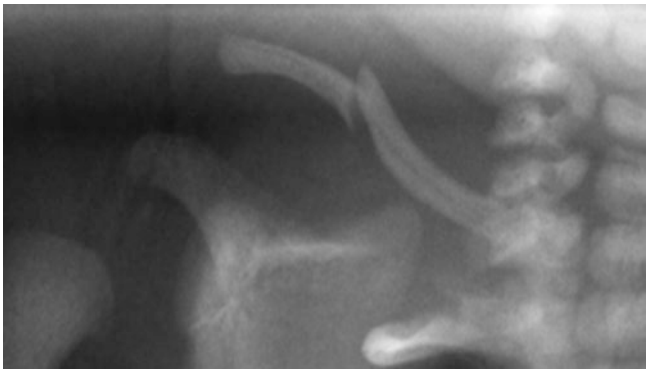


Fig. 13.1 One-day-old infant (birth weight 3400 g) after uncomplicated delivery. On physical examination, a swelling was seen at the site of the right clavicle. Radiograph showed a mid-clavicular fracture

Fig. 13.2 One-day-old neonate (birth weight 3350 g) after vacuum extraction with shoulder dystocia. Radiograph showed a mid-shaft humerus fracture

out prenatal care, after a complicated delivery, after a Caesarean section, or in cases of maternal diabetes [7]. Vitner et al. assessed the difference in outcome in vacuum-assisted

Fig. 13.3 One-day-old neonate (birth weight 2125 g) after primary Caesarean section for transverse presentation. At physical examination a swelling on the left femur was seen. Radiograph showed an oblique fracture of the proximal femur

vaginal delivery in women with (N = 251) and without (N = 4534) gestational diabetes mellitus and found that in the first group significantly more neonates had a broken humerus (p = 0.003) but no difference in clavicle and skull fractures [10]. Krispin et al. evaluated the difference in vacuum-assisted vaginal delivery in which cup detachment occurred (N = 146) or not (N = 1633), there was no difference in the incidence of fractures between the two groups [11]. Wen et al. studied 732,818 vaginal deliveries and showed a significant temporal decline in the rates of fractures from 2.35 to per 1000 live births in 2004 to 1.97 per 1000 live births in 2013 ($P < 0.001$) [16]. This might reflect the overall improvement of obstetric care.

One specific fracture that can easily be overlooked is an epiphyseal separation fracture [17–26]. These are most commonly encountered in the distal humerus, but have been reported in other anatomic locations as well, and may mimic a metaphyseal corner fracture. For these separation fractures ultrasonography is a method of choice in the differential diagnosis whereas in more complex cases MRI can be advised (Fig. 13.5a–c) [26–28].

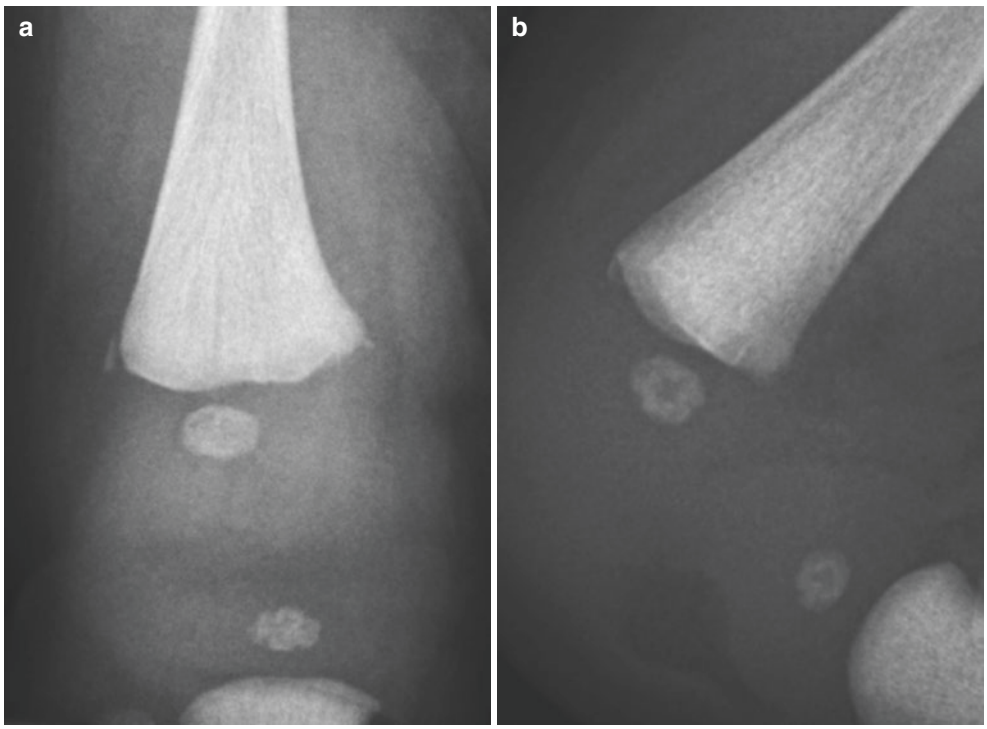


Fig. 13.4 A painful knee was observed in a 2-day-old newborn after a complicated breech delivery. (**a**) AP and (**b**) lateral radiographs show metaphyseal corner fractures

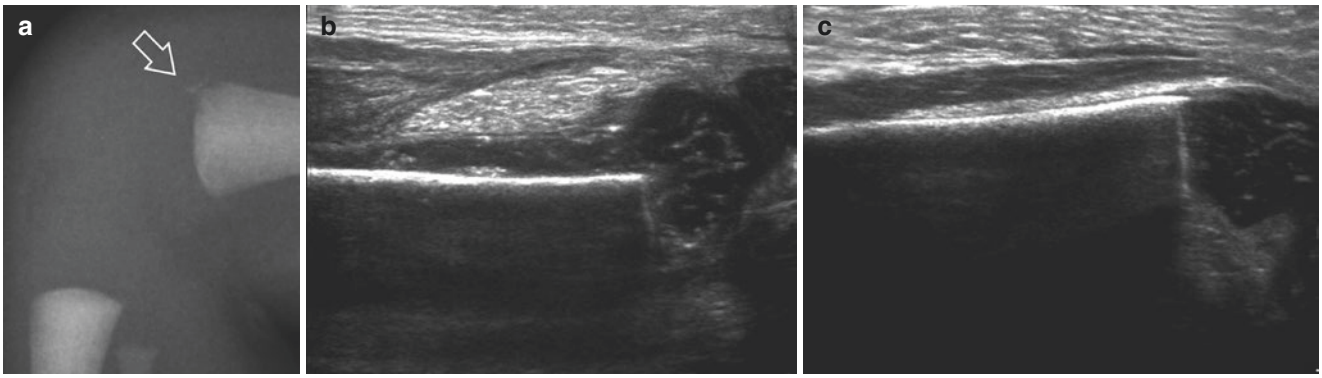


Fig. 13.5 A 2-day-old newborn presents with a swollen and painful knee after a caesarean section. (**a**) Lateral radiograph of the knee shows a subtle metaphyseal avulsion (arrow). (**b**) Ultrasonography revealed an accompanying slipped epiphysis. (**c**) Normal contralateral knee for comparison

13.2.3 Conclusion

Clinicians should be aware that when they are confronted with a neonate with a fracture in the first weeks after birth that birth trauma, even if this was not reported or clinical findings were not present after birth, it might be very difficult to differentiate between birth trauma and non-accidental injury [29]. Fracture healing shows margins making dating at this young age unreliable (see Chap. 4) [30].

13.3 Accidental Fractures

13.3.1 General Considerations

Accidental injuries in children are common with approximately one-third of children sustaining a fracture before the age of 17 years [31]. In a Canadian longitudinal study of 96,359 children up to the age of 10 years there were 12,811 fractures of an upper limb, 4237 fractures of a lower limb, 1620 fractures of the skull, and 217 fractures of the neck or

trunk [32]. In light of these figures, accidental trauma should be in any differential diagnosis when a child presents with a fracture. Physicians are expected to be able to distinguish between accidental and non-accidental injuries. However, this is not always easy. After regular diagnostic methods have established the nature and extent of the injury, physicians have four important aids at their disposal:

- Clinical history, i.e. the statement given by the parents/carers regarding the origin of the injury.
- Developmental level of the child.
- Theoretical data concerning the cause of fractures and the circumstances under which fractures can be sustained.

In order to explain the presence of a specific injury, parents may mention an accident, whereas the physician is able to establish based on the level of development of the child and the scientific data that the injury is consistent with the circumstances mentioned, or it is highly unlikely or even impossible for the sustained injury to result from the reported accident.

In an accidental injury, the clinical history often provides a more or less conclusive explanation for the cause of the injury and the circumstances under which the injury occurred. The patient history is regularly supported by statements from witnesses.

Based on a number of key ages, Table 13.2 provides a global overview of motor development in children up to 5 years of age. Key ages are a selection from many age levels and the most suitable frame of reference for diagnostic purposes. Every age mentioned in these fields of development is 'p50 aged'. This means that 50% of all children have reached that specific level of functioning at that age, and master these functions at a more or less adult level. The table does not claim to be complete and only provides an indication of the general level of motor development of a child at a given age [33]. In non-mobile or partly mobile children it regularly occurs that the clinical history states that the child was responsible for the sustained fracture; for example, when the child fell from the dressing table or out of bed. The question the physician should answer in these cases is whether at the moment of the reported incident, the infant had the motor skills to fall in the manner described. This calls for careful assessment of the level of development of the child. This can take place based on the one hand on data in the clinical history, and on the other hand on data from the child welfare centre. Only when these data are not available, then a theoretical assessment of the motor skills can be made, based on scientific data. An important element is the moment at which a child can turn and crawl, since this creates the movement potential that makes falling feasible. Tables 13.3, 13.4, 13.5 and 13.6 provide an overview of reference values regarding the age at which such skills are present [34].

Table 13.2 Overview of key ages and their general motor development [33]

Age	Skill	General motor development
4 weeks	• Control muscles of the eye	• Positive head lag
16 weeks	• Balance head	• Stabile head balance • Symmetric posture
28 weeks	• Grip and manual manipulation	• Sits and leans forward supported on the hands • Stable stance when supported • Asymmetric neck reflex disappears (22–26 weeks)
40 weeks	• Control trunk and fingers: Sitting, crawling, and picking	• Sits without support • Crawls • Pulls up to a stance • Grip reflex at the feet disappears (40 weeks–18 months)
52 weeks	• Control of legs and feet: The child stands erect and starts exploring	• Walks holding on to one hand • Walks along an object (such as coffee table or settee)
18 months	• Control of larynx function: Words and word combinations	• Walks independently • Able to sit up independently
24 months	• Control of bladder and bowel functions	• Is capable of running • Can play football
36 months	• Speaks in sentences	• Can stand on one leg • Jumps from the bottom step of the stairs
48 months	• Understands numbers and shapes	• Hops well on one leg • Jumps forward on both legs
60 months	• Child ready for school and prepared to play with other children	• Hops equally well on either leg

Table 13.3 Overview of the reference values for supine-prone rotation [34]

Author	*P* value	Age (weeks)
Bayley	50	28
	95	43
BOS 2–30	5	19
	50	28
	95	41
D.O.S.	50	19
	90	38
Gesell	50	24
Helbrügge	–	30
Illingworth	–	28
Schlesinger	10	17
	50	24
	90	32
	99	39
	100	41

Table 13.4 Overview of the reference values of turning prone-supine [34]

Author	*P* value	Age (weeks)
Illingworth	–	24
Schlesinger	10	14
	50	23
	90	32

Table 13.5 Overview reference values for supine-prone and prone-supine rotation [34]

Author	*P* value	Age (weeks)
Schlesinger	10	22
	50	28
	90	36
Sheridan	–	26
Touwen	80	29–32

Table 13.6 Overview of reference values for crawling along (abdomen touching the surface) [34]

Author	Similar characteristic	*P* value	Age (weeks)
Bayley	Pre-walking locomotion	50 95	31 47
Gesell	Pulling self along on abdomen	50	<40
Helbrügge	Prone crawl position	–	39
Illingworth	Prone crawl position Crawls by pulling self forward with hands	–	40
Schlesinger	Crawls forwards, stomach touching the floor	10 50 90 95 98 100	28 36 46 51 53 55
Sheridan	Attempts to crawl; sometimes succeeds	–	39
Touwen	Moves forward on stomach by using arms and legs	80	45–48

The reader is referred to Chaps. 2 and 5–12, in which extensive data are given concerning the cause of specific fractures and the circumstances under which these fractures can occur.

In evaluating the presence of a fracture in a child, based on scientific data, the following questions can be answered:

- Which skeletal injuries can be sustained in an accident/fall?
- Which injuries are less or not plausible in an accident/fall?

In retrospective studies, the following injuries were found after an accident/fall:

- Haematomas, contusions, excoriations, and lacerations on the head and the rest of the body.
- Fractures of the skull, clavicles, and long bones.
- Brain damage, such as concussion, contusion, and intracranial haemorrhages.

Studies showed that severe brain damage and death after a short-distance fall are improbable. When brain damage was found after such a fall it generally was focal instead of diffuse. Furthermore, none of the children died as a consequence of a fall from cot or crib. Due to complicating factors in falls from a bunk bed or shopping trolley, the risk for more serious injuries and death increased slightly.

Based on earlier-mentioned studies, the following variables that affect the occurrence of serious injuries, including fractures and/or death could be indicated:

- The distance of the fall.
- The velocity of the fall or (rather) the initial velocity at the start of the fall.
- Free or complicated fall (for instance on an object).
- Properties of the landing surface (for instance a concrete surface as opposed to a surface covered in foam-supported carpet).
- The manner in which the energy is spread when landing (percentage of body surface, possible fractures).

13.3.2 Short-Distance Fall, Serious Injuries, Hospital Admissions, and Death

13.3.2.1 Introduction

One of the most prevalent explanations for the occurrence in young children of serious injuries (including fractures) and eventually visits to emergency department, hospital admissions, or even death is that a child allegedly fell over a relatively short distance (under 1.5 m) (Figs. 13.6a, b and 13.7).

In the literature one may come across case reports on relatively serious accidental injuries in such a fall. Often there are complicating factors involved in the fall. Wheeler and Shope described the occurrence of a depression fracture of the calvarium in a 7-month-old child after falling on a toy car [35], although fractures of the calvarium may occur due to an uncomplicated short-distance fall [36]. Most epidemiological studies, however, show that serious injuries are rare in accidental short-distance falls, compared to the number of falling children.

In the first years of life, approximately 50% of children will experience a short-distance fall, defined as a fall over a distance under 1.5 to 2 metres (Fig. 13.8a, b) [36–38]. This

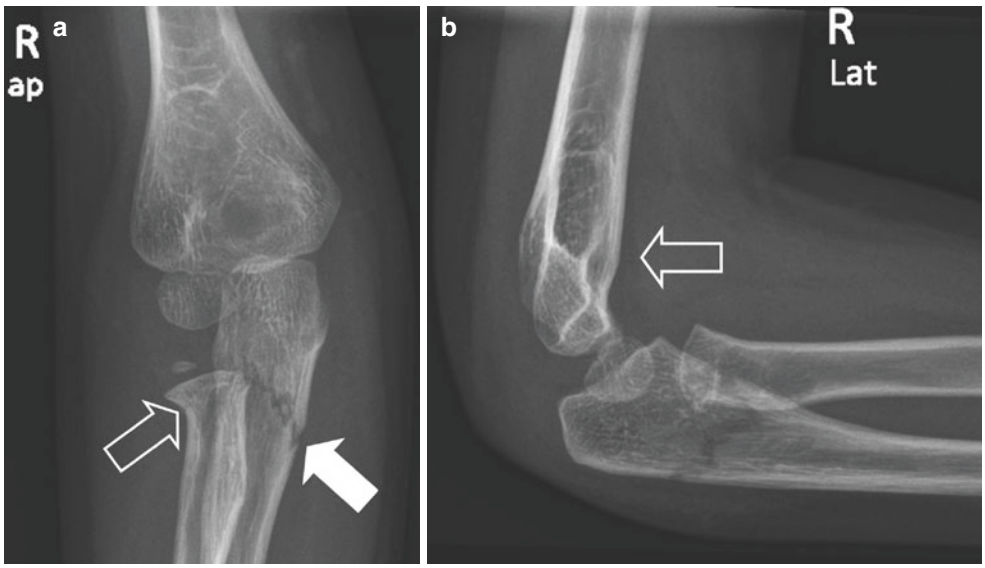


Fig. 13.6 Five-year-old child who fell out of a play rack from a height of approximately 1.5 metre. (**a**) AP radiograph shows a fracture of the neck of the radius (open arrow) and an oblique proximal ulnar fracture (arrow). (**b**) Lateral radiograph shows an anterior fat pad sign (arrow)

Fig. 13.7 Spiral tibia fracture in a 5-year-old child who fell off a play rack from a height of approximately 2 metre

type of fall often necessitates a visit to the emergency department (approximately 1 in 100 children under the age of 1 year) [39]. Approximately 1 in 1000 children will be hospitalized for such a fall [40, 41].

In 1993, Rivara concluded that approximately 1 in 250,000 children under the age of 1 year will die from such a fall [40]. In 2008, Chadwick et al. found a much lower estimated mortality rate from short-distance falls (falls under 1.5 m in vertical height) in infants and young children between birth and their fifth birthday [41]. They reviewed published materials, including 5 book chapters, 2 medical society statements, 7 major literature reviews, 3 public injury databases, and 177 peer-reviewed, published articles indexed in the National Library of Medicine. Based on this review, the best estimate (at the moment of the review) of the mortality rate for short-distance falls is under 0.48 deaths per one million young children per year.

13.3.2.2 Data from Literature

From 1977 onwards, various articles have been published in which the authors investigated whether children will sustain injuries in a fall and, if so, what type of injury.

Helfer et al. reported injuries in 246 children <5 years old [42]. The study population consisted of two groups, the first one hundred and sixty-one children whose parents had filled out a questionnaire when they visited their physician because their child had fallen over a distance of <100 cm (bed or couch) outside the hospital. Due to the fall, 3 children sustained a clavicle fracture (age ranged between 6 months and 5 years), two children a skull fracture (age < 6 months), and one child a humerus fracture (age < 6 months). According to the parents, 80% of children did not sustain any injuries, irrespective of the distance of the fall. The second group consisted of 85 children who had fallen from cots or examination tables while hospitalized (the so-called hospital incident reports). In 57 children no injuries were found, in 17 children there was superficial damage to the skin such as abrasions,

Fig. 13.8 Five-year-old girl who fell out of a play rack from a height of approximately 1.5 metre. (**a**) There is an obvious fracture of the left lower arm (Photo printed with permission). (**b**) Lateral radiograph shows transverse fractures of the radius and ulna

20 children suffered bruises and one child sustained a skull fracture. The authors concluded that in this group there were no serious head injuries after a short-distance fall, but that it was possible to sustain fractures.

Chadwick et al. looked at 283 children of all ages who were presented at the emergency department of a paediatric hospital in San Diego between August 1984 and March 1988 [43]. In their study, there were 7 deaths after a fall from a distance of less than 4 feet (approximately 1.2 metre) and in all cases there were factors in the clinical history that made it unrealistic that indeed there had been a short-distance fall.

Kravitz et al. reported on a questionnaire study on 536 infants under the age of 1 year [38]. In this study, the parents/caregivers reported that a total of 255 infants (47.6%) at least sustained a single fall in the first year of life. In 34 cases 6.3% of more than one fall was reported. In the whole study population three (1.2% of all falls) skull fractures and no other fractures were reported.

Nimityongskul and Anderson looked into the occurrence of injuries in 76 children (age range: from neonate to 16 years old) who had fallen out of their bed, crib/cot, or chair while hospitalized [44]. Fifty-seven children were < 5 years old, 23 children <1-year-old and falling distance was 30–100 cm. Most children sustained superficial injuries (haematomas of the scalp and lacerations of the face). One child (12 months old) sustained an occipital skull fracture, another child (with osteogenesis imperfecta) a fracture of the tibia. No rib fractures were found. Fourteen children had sustained facial bruising or bruising on the scalp. Nimityongskul and Anderson did not find any serious injuries to the head, neck, spine, and extremities. Based on their findings they consider the statement of the parents that their child had fallen over a short distance only suspect of non-accidental circumstances when serious injuries were found.

Lyons and Oates report on 207 children <6 years of age who had fallen from crib/cot (n = 124) or bed (n = 83) while hospitalized [45]. The distance of the fall ranged from 65 cm (bed rail down) to 110 cm (bed rail up) in a fall from crib or cot, and from 50 to 85 cm (including bed rail) in a fall from a bed. In 31 children injuries were found.

- Twenty-nine children suffered contusions of little consequence and small lacerations.
- One child suffered a linear skull fracture (age 10 months, fall from cot).
- One child sustained a clavicle fracture (age 21 months, fall from cot, bed rail up).

In 26 of 31 children the injury was located on the head. None of the children had serious, multiple, or life-threatening injuries. Lyons and Oates concluded that serious head injuries are unlikely in a short-distance fall.

Tarantino et al. report on 167 children less than 10 months old (average age 5.2 months, 56% boys) that had experienced a fall of less than 1.25 m and consequently presented at the emergency department [46]. Tarantino et al. excluded children that had experienced a complicated fall, such as a fall from a baby walker, a fall down the stairs, or a fall on an object. They also excluded situations in which the carer fell on the child during the fall. Of the included children, 55% fell out of bed, 20% from the arms of a carer, 16% fell from a bed/couch and 10% fell from other objects. Of the 167 included children 10% was hospitalized. However, the majority of children had few or no injuries. Twenty-five children sustained serious head injuries: 16 closed skull trauma (of whom 12 had a skull fracture), two an intracranial haem-

orrhage, and seven fracture of one of the long bones (Figs. 13.9 and 13.10). Additional examination showed that the two children with an intracranial haemorrhage sustained these due to non-accidental trauma. After these two children were excluded, it appeared that the risk for serious injury was only present in a fall from the arms of the carer. Tarantino et al. advise, based on their findings, always to consider non-accidental trauma in children with intracranial injury and/or multiple injuries, when the parents mention a short-distance fall. Bechtel et al. draw attention to the risks of a fall from the arms of a carer [47, 48]. They also point out that such a fall may lead to intracranial haemorrhages.

Haney et al. evaluated the findings in 307 children under the age of 5 years, regarding any short-distance fall before the age of 2 years [37]. A total of 209 falls were reported in 122 children. Only 24% of the children sustained any injury due to falling. Most children (85%) had bruises or bumps. Forty children were brought for medical care, while only 13 of these 40 children were in need of medical care. Two children sustained concussions and four children had a permanent injury (cutaneous scars) due to the fall. No fractures due to short-distance falls were reported in this study. Children who fell on a hard surface were 6 times more likely to have an injury compared with children who fell on a soft surface ($P = 0.001$). They also found that fall height was a significant predictor of injury risk (proportional relation: the higher the fall, the higher the risk). Based on their findings they concluded that:

Fig. 13.9 Two-year-old infant who had fallen out of bed. Radiograph showed a supracondylar humerus fracture (arrow)

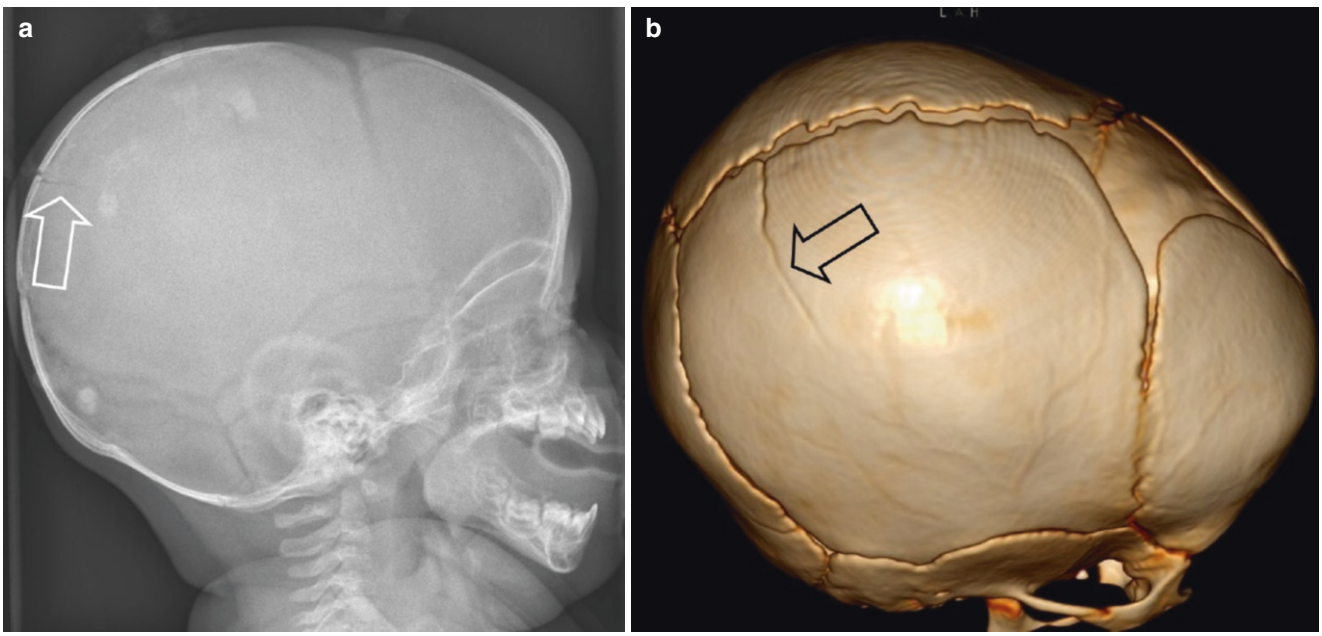


Fig. 13.10 Six-month-old infant who fell from a baby changing table. (**a**) Conventional radiograph shows a linear skull fracture (arrow). (**b**) 3D-CT provides a better overview of the extent of the fracture

- Short-distance falls rarely caused injuries.
- A history of a short fall in a seriously injured child should raise the suspicion of non-accidental circumstances.

As part of the Avon Longitudinal Study of Parents and Children (ALSPAC), Warrington and Wright researched the prevalence of accidents in non-mobile children around the home [49]. ALSPAC included 13,822 mothers/sets of parents. Via written questionnaires, Warrington and Wright asked the parents of 6-month-old children to describe every accident since birth. They asked them to describe: type of fall, distance, injuries sustained, and the medical help provided (if sought). 11,466 forms were returned. The parents reported 3357 fall accidents in 2554 children. Fifty-three percent of children fell out of a bed or from the couch. Twelve percent either fell from an arm while being carried or the person that carried the child fell holding the child. In less than 1% of cases the cause of the accident was omitted. In the other 34% there was great diversity in types of fall, e.g. a fall from a table, chair, or changing table or from a baby bouncer. Of all children, 76% of children experienced one fall, 5% had three falls or more. The number of falls increased with the age of the child. Less than 25% took place before the age of 4 months. In only 14% of children there were visible injuries, of which 56% were haematomas. In 97% there was a visible injury to the head. In less than 1% (21 children) concussion or a fracture was found. One hundred and sixty-two children were taken to hospital after the fall, 18 were hospitalized. In three children the hospital physicians found a skull fracture; however, this was no reason for hospitalization. No skull fractures were seen after a fall from a bed or couch. One child sustained a clavicle fracture after a fall from a bed. Warrington et al. concluded that children <6 months old regularly take a fall, but that this seldom leads to injuries. Serious injuries (here defined as concussion or a fracture) are very rare: it occurs in less than 1% of children. None of the children had intracranial injuries such as subdural or epidural haematomas.

Hennrikus et al. evaluated 115 young patients with orthopaedic injuries, according to the parents sustained at home after a fall from a piece of furniture [50]. One hundred and thirteen children sustained fractures or dislocations. Two children suffered penetrating injuries (pen, needle). In six children the physicians reported non-accidental circumstances: two (out of four) children were < 1 year old, four (of 83 children) were 1–5 years old. Hennrikus et al. concluded that orthopaedic injuries sustained around the home are usually the result of a fall (from bed or couch), unless the child is less than 1 year old.

Mulligan et al. reported on 916 infants under the age of 1 year who were presented following a fall at a paediatric trauma centre in Sydney Australia [51]. Of these infants 110 (11.6%) were admitted, of whom 20 were reported to child protection services due to concerns of non-accidental trauma. The most common reason for admission was head injuries (90 infants, 85%), of these skull fractures (80 infants, 75.5%) were the most common head injury. There was one fatal case, a 5-month-old infant who fell from a cot on a hard floor and who sustained a skull fracture and extradural haemorrhage. This study shows a higher incidence of severe trauma, but as the study was done in a tertiary referral centre this may well be the result of a referral bias.

Johnson et al. evaluated the type and nature of the head injuries in 72 children <5 years old (4 months to 4 years and 9 months) of whom it had been irrevocably established that they had fallen as the result of an accident [52]. The distance of the fall ranged from <50 cm to >3 m; most children fell over a distance of less than 1 m. Forty-nine children fell on a hard surface and 23 on a soft surface. Fifty-two children sustained visible injuries: 35 of them fell on a hard surface and 17 on a soft surface. The surface area of the fall did not seem to lead to significant differences in the injuries that were found; however, the distance of the fall did make a difference. All children who fell over a distance of >1.5 m had sustained visible injuries to the head. This was also true for 95% of children that fell over a distance of >1 m. In 32 children a radiograph of the skull was made. Four children had sustained a skull fracture: three linear fractures (twice from a fall of >1 m and once by a fall of 80–90 cm against the stone surround of a fireplace). The fourth child sustained a basilar fracture after a fall over 3 m from a window on the first floor. Johnson et al. concluded that in the majority of children the most common accidents in and around the home do not cause noticeable injuries in the majority of children. Skull fractures occur probably in less than 5% of children. It requires a fall of at least 1 m, or a fall on an object that results in a 'small-area impact'.

Ibrahim et al. reported on 285 children, aged 0-48 months, hospitalized with accidental head injuries after falls from different heights [53]. 98 children (67 infants and 31 toddlers) did fall from a height under 3 feet (approximately 1 m). Skull fractures and scalp/facial soft tissue injuries were seen more often in infants (0-12 months) than in toddlers (12-48 months): soft tissue injuries in 84% of infants versus 42% of toddlers and skull fractures in 73% versus 23%. Skull fractures without soft tissue injuries and multiple skull fractures were only found in infants in resp. 5% and 8%. No evidence of impact (soft tissue injury and/or skull fracture) was found in 12% of infants and 58% of toddlers. Low height falls resulted in primary intracranial injury (subdural haematoma, epidural haematoma, subarachnoid haematoma, parenchymal contusion/laceration, intraparenchymal haemorrhage and/or diffuse axonal injury) in 55% of infants and in 42% of toddlers. Primary intracranial injury without soft tissue or skull fracture was found in infants (6%) and toddlers (16%). Retinal haemorrhages (scattered white centered intraretinal haemorrhages in the left eye) were found in one infant after a fall from a caregiver's lap. The infant had a large ipsilateral epidural haemorrhage. In toddlers retinal haemorrhages were only found after falls from over 3 feet. There were no long bone fractures after heights under 3 feet, neither in infants, nor in toddlers.

Monson et al. describe 14 neonates (in a total number of 88.774 births over a period of 3 years) who had fallen shortly after birth while hospitalized [54]. Seven children fell to the floor when the parent was lying on the bed or seated in a chair and fell asleep holding the infant. In four children the fall took place in the delivery room, two children fell out of their bassinette when wheeled down the hall and one child fell from an infant swing. Thirteen infants had a normal external examination when discharged from hospital. One child had a large haematoma on the forehead, which was still visible at discharge. Another child had to be transferred to the regional hospital due to a depressed skull fracture. The fracture, sustained after a fall of 50–70 cm that occurred immediately after the child was delivered, did not cause any notable clinical symptoms. There was no indication of neurosurgical intervention. After 2 days the child could be safely discharged from hospital. Only in one child a CT was performed, which did not show any intracranial lesions. If one of the other children incurred intracranial lesions as a result from the fall, they did not result in any noticeable clinical symptoms.

In a small study of 11 newborns with in-hospital falls by Ruddick et al. only three newborns showed clinical findings after a fall [55]. In a subgroup of six newborns where, at the discretion of the clinician, a skull radiograph was performed there were three new-borns with skull fractures. In two of these three cases there was no soft-tissue swelling.

13.3.2.3 Conclusions

Based on the literature it is evident that short-distance falls are common events in the first years of life and that they will rarely cause injury and rarely, if ever, result in serious or life-threatening injuries. Fractures of the skull, clavicle, or long bones are reported, although less often than one would expect (Figs. 13.11a, b and 13.12a, b). Serious multiple non-life-threatening injuries of the head, neck, spine, and extremities are not reported. More serious injuries can be sustained, such as a depression fracture of the skull, if there are complicating factors (e.g. falling on an object). A history of a short fall in a seriously injured child should raise the suspicion of non-accidental circumstances. It is important to note that in daily routine in many, if not most, cases of short-distance falls no radiological examinations will be done. As, e.g. skull fractures can be clinically silent we do not know the exact incidence of these kinds of fractures. From a forensic medical point of view, more research into the exact incidence of fractures after a short-distance fall would be welcome.

13.3.3 Fall from a Considerable Height

13.3.3.1 Introduction

Most children that fall from a considerable height are under the age of 6 years. They fall over a distance of 3–7 m (one to two floors), in or in the vicinity of their home, more often during the warm seasons [56–60].

13.3.3.2 Data from Literature

As described earlier in this book, a child who falls from a considerable height will in particular sustain injuries to the head and neck area [57, 59, 61]. The most prevalent injuries are (in order of occurrence):

- External and visible injuries [56–59, 62–64].
- Skull fractures, of the cranium as well as the base of the skull, possibly with intracranial abnormalities [56, 59, 62, 63, 65].
- Fractures of the extremities [56, 57, 59].
- Fractures of the spine [56, 66].

Only occasionally more than one body part is injured [56, 59].

A remarkable discovery in the study of Wang et al. is that orthopaedic and thoracic injuries (fractures of the extremities, lung contusion, and pneumothorax) are more frequently seen

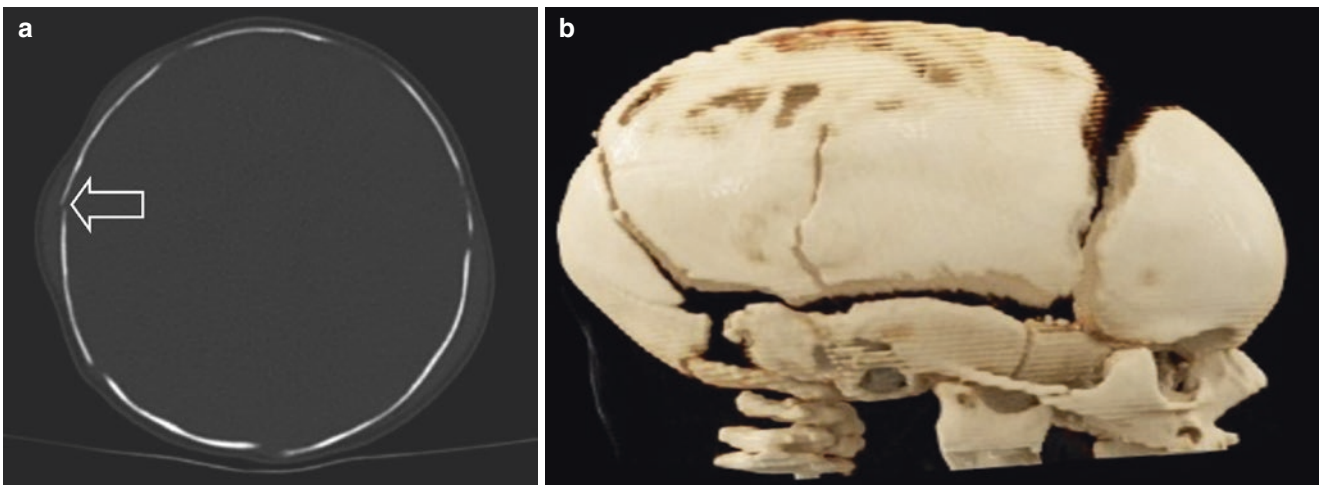


Fig. 13.11 Three-week-old infant who fell out of the arms of her mother. (**a**) Axial CT shows a linear fracture of the right parietal bone (arrow) and bilateral soft tissue swelling. (**b**) Cinematic rendering of the skull shows a linear fracture

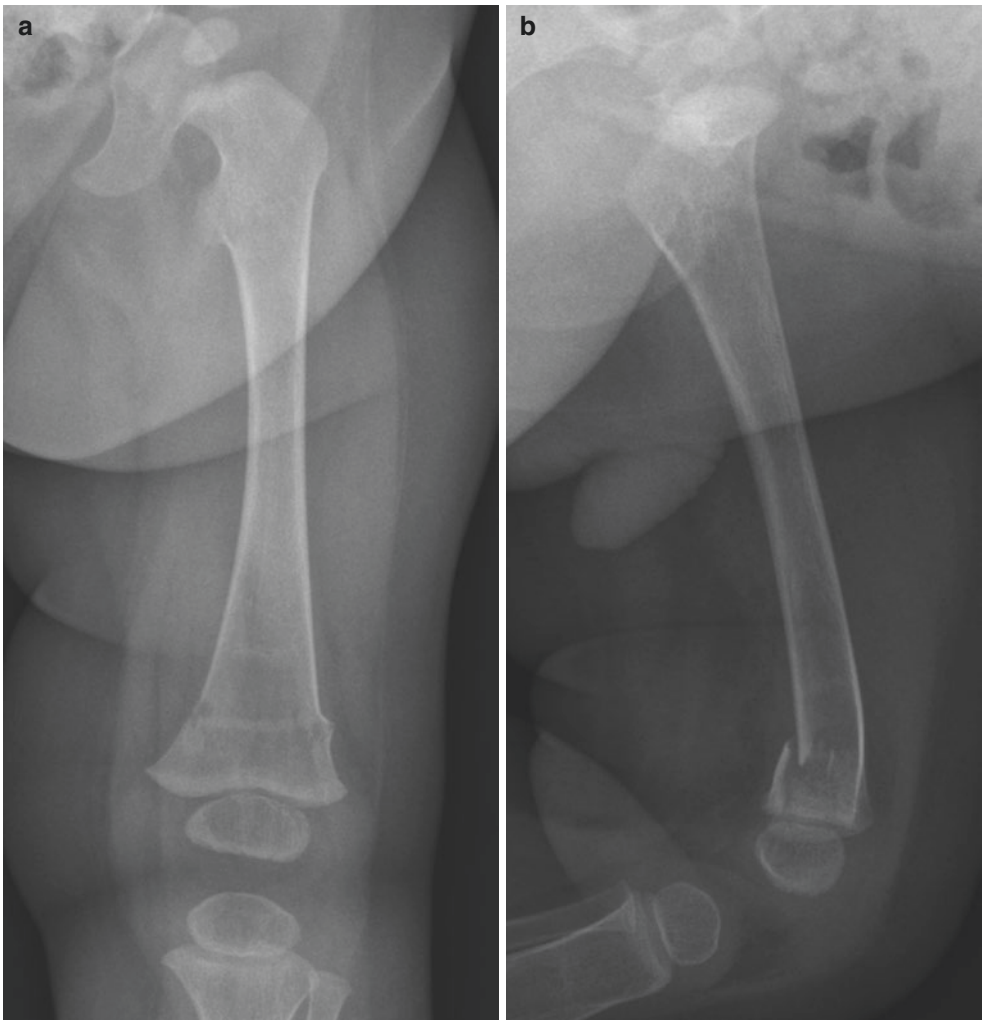


Fig. 13.12 Eight-month-old infant who fell out of the arm of a parent. (**a**) AP radiograph of the left femur shows a fracture of the distal metaphysis. (**b**) Lateral radiograph shows buckling of the posterior cortex

in falls over a distance of more than 4–5 m, and that abdominal injuries (liver lacerations, visceral, and spleen injuries) are more frequently seen in a fall of less than 4–5 m [64].

13.3.3.3 Conclusions

Although this type of fall carries a high morbidity, it seldom leads to lasting defects or death [56, 59]. Morbidity increases with fall distance, although a fall distance of less than 5 m can also be lethal [56, 62]. In these cases intracranial injuries are the main cause of death (Fig. 13.13a, b) [62]. As in most cases the clinical history and circumstances are clear falls from a considerable height most often will not present as a diagnostic problem.

13.3.4 Fall with Caretaker

13.3.4.1 Introduction

Every parent's fear is tripping while carrying a young child and unfortunately this is something that does happen. Interestingly the incidence of these falls is unknown. However, when Googling 'tripping with baby in arm' numerous statements from parents are found in which they reported to have tripped while holding a baby. The commonly reported trauma mechanism is losing footing on the stairs (either up or down) and tripping over pets or objects. In many cases, the parent/caregiver holds on to the child but some report to have lost their grip and dropped the child while falling. Although Google certainly is not a scientific database, going over these statements two points arise, first in nearly all cases the child did well and in many cases no medical attention was sought and second nearly all parents report to be ashamed about the incident.

In our experience both locally and within the Dutch Expertise Centre for Child Abuse [67], we have encountered a group of cases in which a femoral fracture reportedly occurred due to a tripping accident of the parent. The clinical information in these cases was that the parent/caregiver was carrying the child on his/her lower arm placed in the knee cavity facing towards the person carrying the child. When this person fell/tripped he/she pressed her lower arm against the body and used the other arm to break the fall. As a result, the child fell backward but did not fall to the ground. In several cases with this reported trauma mechanism we found distal femoral fractures at the level of the distal metaphysis/diaphysis (Fig. 13.14a, b). Further work-up in these cases was negative and we believe that this is a true accidental trauma mechanism.

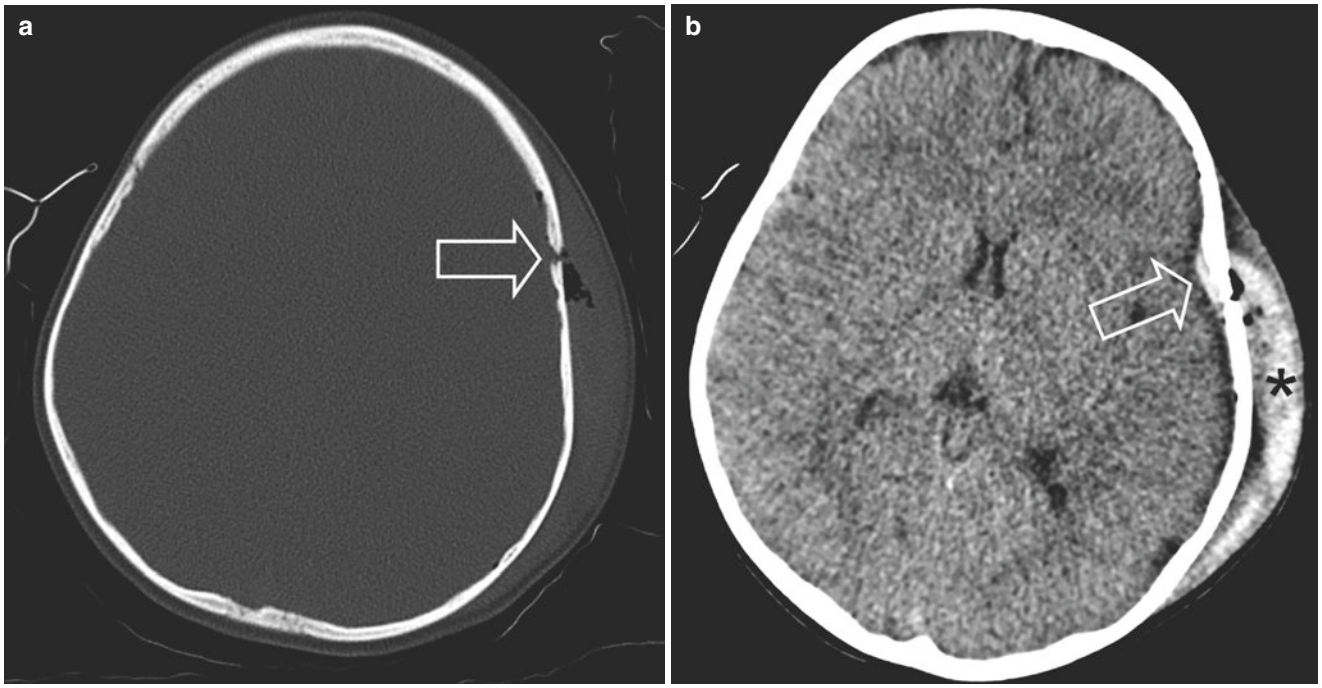


Fig. 13.13 (**a**) Twenty-two-month-old infant who had fallen from a 4-m high window. The skull CT shows a fracture of the parietal bone (arrow). (**b**) CT at soft-tissue setting shows a small, probably epidural, haematoma (*open arrow*) and a soft-tissue haematoma (asterisk)

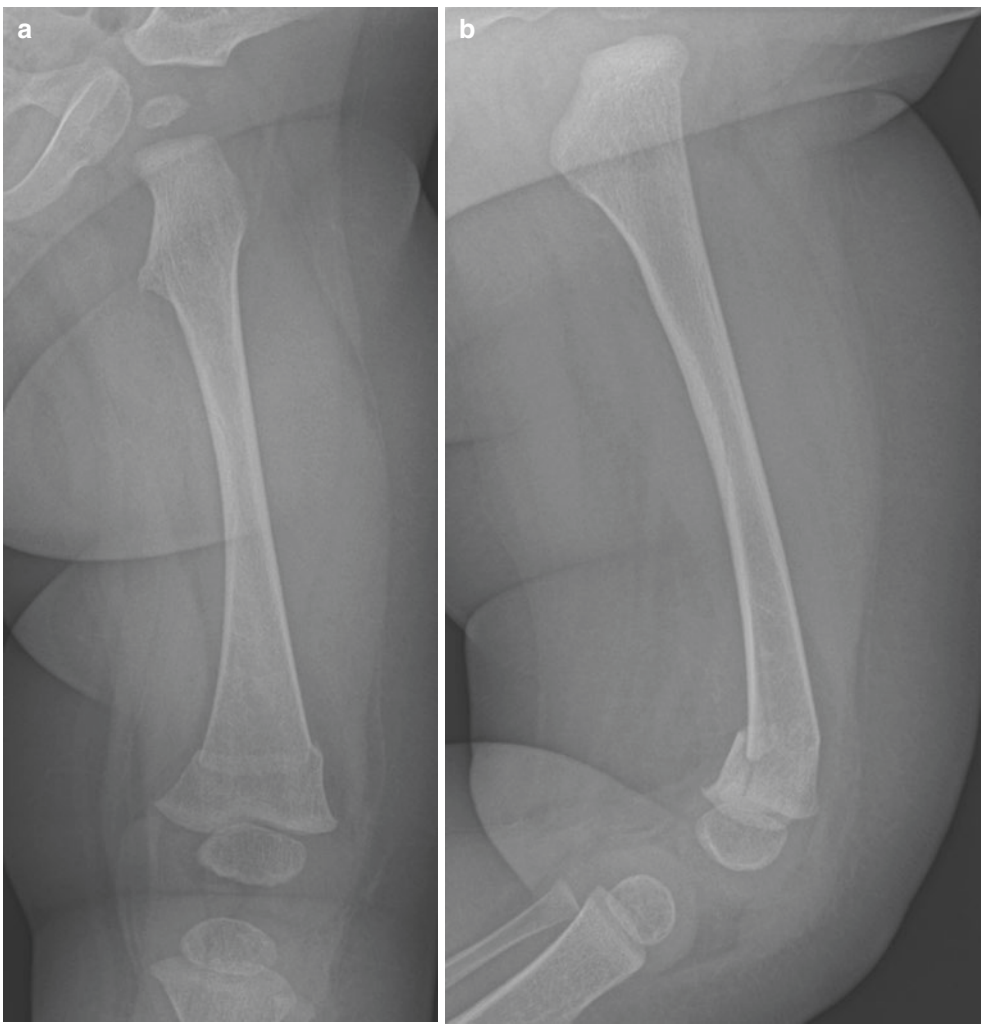


Fig. 13.14 Eight-month-old infant who was, facing towards the mother, carried by mother on her arm. When she fell, she tried to break her fall with her arm, at the same time the infant fell backwards over her lower arm (with the arm acting as a lever). (**a**) AP radiograph shows a distal femur fracture. (**b**) Lateral radiograph shows buckling of the posterior cortex

13.3.4.2 Conclusion

Although it seems that falls of parents/caregivers while carrying a child occur regularly, there is no dedicated literature on this specific trauma mechanism.

13.3.5 Falls Involving Objects

Injuries in young children are often explained as resulting from a fall from or on an object. In this section specific objects which most often are mentioned will be discussed, our aim is to give an overview of the evidence behind injuries resulting from a fall from or with an object. By no means this is intended to provide a definitive all-inclusive overview of the literature available and inevitably there also will be some overlap with other parts of this book.

13.3.5.1 Bed or Couch

Introduction

As presented in paragraph 13.3.2 falls from low heights are a relatively common experience in childhood. Subsequently, it is often stated in the medical history of young children with a fracture (Fig. 13.15) or a head trauma, that the child sustained the injuries due to a fall off a bed or couch.

Data from Literature

Already in 1977 Helfer et al. studied the incidence of injuries as a result from a fall in young children [42]. For this they interviewed parents and performed a retrospective review of their hospital emergency data. A total of 256 children (161 questionnaire and 85 hospital admission), aged <6 years, with a total of 254 (169 questionnaire and 85 hospital admission) falls from a bed or sofa were included in the study resulting in a total of 43 injuries. Of these injuries 75 were minor and in 7 cases fractures were found (3 clavicle fractures, 3 skull fractures, and 1 humerus fracture). In the questionnaire group, the skull fractures and humerus fractures occurred in children <6 months of age and the clavicle fractures in older children. In the hospital admission group 1 skull fracture was found, however, the age was not reported. It is of interest to note that the authors state that in the 85 hospital cases a total of 40 radiographs were taken, i.e. this rules out that skeletal surveys were done and thus inflicted injury could certainly have been misdiagnosed.

Fig. 13.15 Three-year-old child who fell off the backrest of the bench. Radiograph shows a fracture of the radius and a fracture line in the ulna

Nimityongskul and Anderson described 76 children under the age of 16 years who fell out of bed in the period 1980–1985 [44]. Of these 23 were 1 year and 16 were 2 years of age and despite the title of the paper 'The likelihood of injuries when children fall out of bed' only 36 children fell out of bed, stretcher or crib. In the whole study, only one occipital skull fracture was reported, although it is unknown what the trauma mechanism was in this case.

Lyons et al. looked at children <6 years of age, who fell out of a bed while admitted to their hospital [45]. In a 9.5-year period this happened to 235 children, in all cases the children were evaluated after the fall and an incident report was made. This data was used for study purposes. There were 124 falls from cribs, which are used for children up to 36 months of age, leading to 29 trivial injuries and 2 fractures, i.e. 1 skull fracture and 1 clavicle fracture. In the older age group 13 minor injuries were found.

Tarantino et al. looked at 167 children, aged ≤10 months of age, who sustained a short vertical fall [46]. Of these children 91 rolled or fell off a bed, 26 rolled or fell off a couch, and 16 fell off another object (e.g. chair or changing table). In this group, 119 (71.3%) suffered a minor injury and 14 (8.4%) significant injury (i.e. intracranial injury, fractures). In the total study group in 10% of children with a minor injury and 48% of children with a significant injury a skeletal survey was made. Unfortunately, the authors do not specify the injuries by trauma modality so more detail cannot be given here.

Macgregor reported on 85 children, 66 < 6 years of age, who were presented to an accident and emergency department with injuries related to a fall off a bed [68]. In this study, the majority of children 72 (85%) fell while sleeping and only a minority while playing or getting in or out of a bunk bed. Although a total of 25 fractures were reported, 18 in children <6 years of which 8 were from a top bunk, it is not specified how the other 10 fractures in this younger age group occurred.

Hennrikus et al. reported on 115 children who were referred to a department of orthopaedic surgery after they sustained injuries after a fall; 36 from a bed, 36 from a couch, and 25 from a different piece of furniture lower than 1.2 metre [50]. In their study population the average age was 4 years (range, 8 months–12 years), with only 1 < 1 year of age and 83 between 1 and 5 years. Not surprisingly a high number of children, 113 (98.3%), suffered a fracture or dislocation. In 6 (5%) cases, including the patient <1 year of age, suspected child abuse was reported.

Conclusions

The literature shows that there is a relatively low number of injuries after a fall from a bed or couch in children. However, one should keep in mind that the true prevalence of these injuries after a fall from the bed or couch is not known as there will be many more children who fall while sleeping or playing on a bed or couch, and may or may have not a minor injury, than there are presented to a hospital. In keeping with most publications it is recommended to rule out non-accidental trauma in children under the age of 1 year (or pre-mobile children) when they are presented with a fracture that reportedly occurred after a fall from a bed or couch.

13.3.5.2 Bunk Bed

Introduction

At face value one may be inclined to think that a fall from a bunk bed, barring the greater distance of the fall, can be compared with a fall from a low bed. However, data from the literature show that the risk for serious injury is considerably higher in a fall from a bunk bed (Fig. 13.16a, b).

Data from Literature

Mack et al. studied the US National Electronic Injury Surveillance System (NEISS) in the period 2001–2004 and found that a total of 23,000 children aged 0–9 years (14,600 children <6 years) were treated annually in emergency departments for bunk bed fall-related injuries [69]. D'Souza et al. evaluated data of children and adolescents treated in emergency departments for a 16-year period (1990–2005) in the United States, a total of 572,580 children and adolescents aged ≤21 years, on average 35,790 cases annually, i.e. average of 42 per 100,000 population (ages ≤21 years), were treated during that time frame [70]. The incidence of injuries was the highest in the 3- to 5-year-old age group (190,200 cases [95% CI: 161390–219,010]). As D'Souza and Mack used the same database there is an overlap in data, but in this study also children over the age of 6 years were included. In this large study population, a total of 3431 fractures were reported. Based on the Canadian Hospitals Injury Reporting and Prevention Program McFaull et al. reported 6002 presentations in Canadian emergency departments in a 20-year period for injuries related to bunk beds [71]. Over the study period the incidence remained stable. Based on data from the EU Injury Database it is estimated that in the European Union annually 19,000 injuries occur in children 0–14 years of age that are severe enough to require presentation at an emergency department [72].

Selbst et al. compared a group of children with injuries resulting from a fall from a bunk bed ($n = 68$) with a control

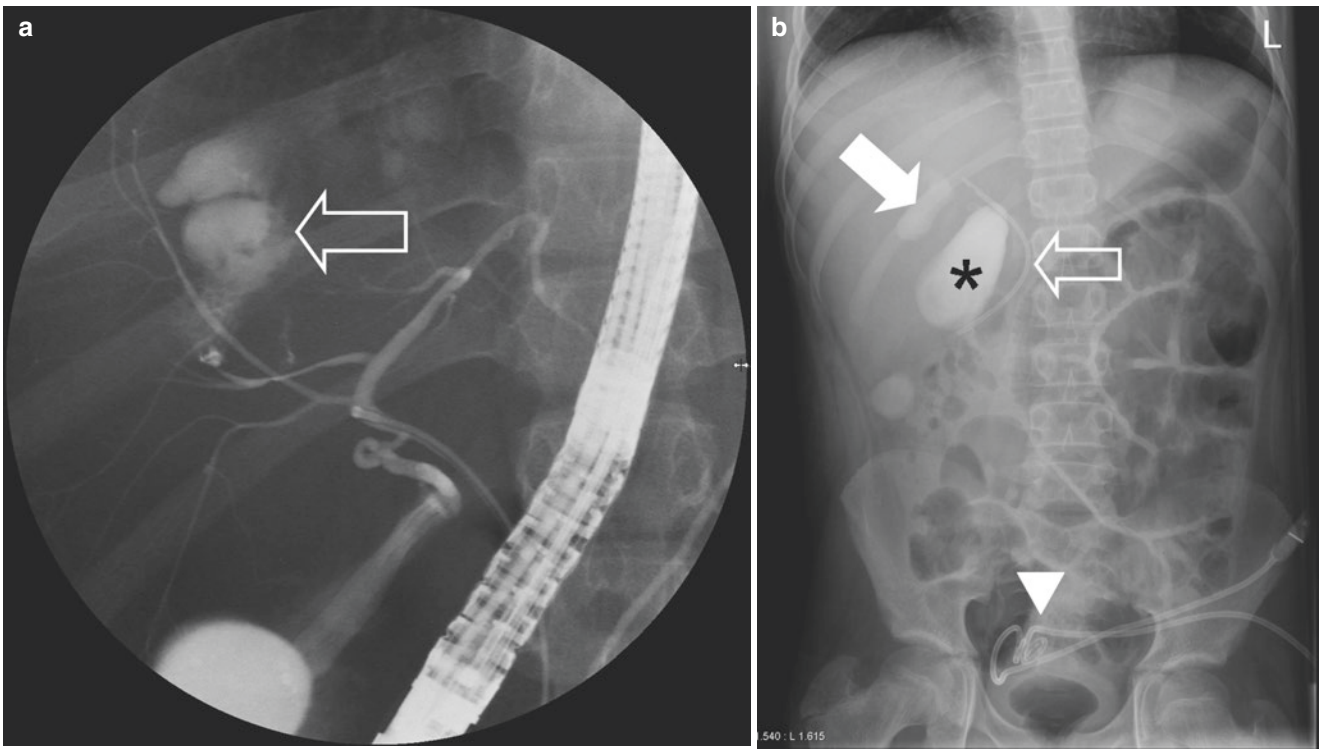


Fig. 13.16 (**a**) Thirteen-year-old child who had fallen out of a bunk bed on top of a drum set. Endoscopic retrograde pancreaticography (ERCP) shows extravasation of contrast (arrow) from an intrahepatic bile duct from a liver laceration. (**b**) Abdominal radiograph after ERCP shows a stent in the common bile duct (open arrow), contrast in the liver laceration (arrow) and gallbladder (asterisk). Because of ascites, pigtail catheters were positioned (arrow point)

group of children that had presented at the emergency department for other reasons, but did sleep in a bunk bed (n = 54) [73]. The average age in the injury group was 5.1 years against 6.2 years in the control group. Seventy percent of the injury group and 48% of the control group was under the age of 6 years. The injuries were sustained by a fall from the upper bed (58%), a fall from the ladder (11%), or a fall from the lower bed (12%). The moment that the injury was sustained varied. In 29% it happened while sleeping (n = 19; 12 children <6 years), in 20% while climbing in or out of the upper bed, and in 43% during play in and around the bed. Fifty-two percent of the children showed head injuries, 12% facial injuries, 13% injuries of the lower extremities, and 10% of the upper extremities (Figs. 13.17a, b and 13.18). Lacerations (40%) and contusions (19%) were seen most frequently. Twelve percent of the children had a concussion and 10% suffered fractures. Six children (9%) had to be hospitalized: four with a concussion, one with a skull fracture with subdural haemorrhage, and one with a laceration close to the eye. The authors concluded that injuries are frequently sustained in a fall from a bunk bed and may be serious. In a fall from the upper bed, injuries to the head and face were most likely. Also, the more severe injuries were sustained by a fall from the upper bed.

MacGregor carried out a prospective study into the severity of injuries resulting from falling from bunk beds (upper and lower bed) and cribs [68]. The study comprised 85 children in the age of 5 months or more (43 boys and 42 girls). Fifty-seven children fell from a crib or the lower bed, 28 children fell from the upper bed. Seventy-eight percent of children (n = 72) was <6 years old (lower bed and crib, n = 52; upper bed, n = 20). The article is not very specific on which injury occurred in which fall (upper or lower bed, crib). However, MacGregor described head trauma in 27 children, of which seven showed noticeable neurological symptoms such as unconsciousness, lethargy, or vomiting. It was remarkable to see that there were no skull fractures or intracranial haemorrhages found in any of the children. This was not even the case in complicated falls when the child while falling hit, for example, other pieces of furniture before hitting the floor. The remaining 20 children who had sus-

Fig. 13.18 Five-year-old infant who fell off the top bunk bed. Radiograph shows a mid-clavicular fracture

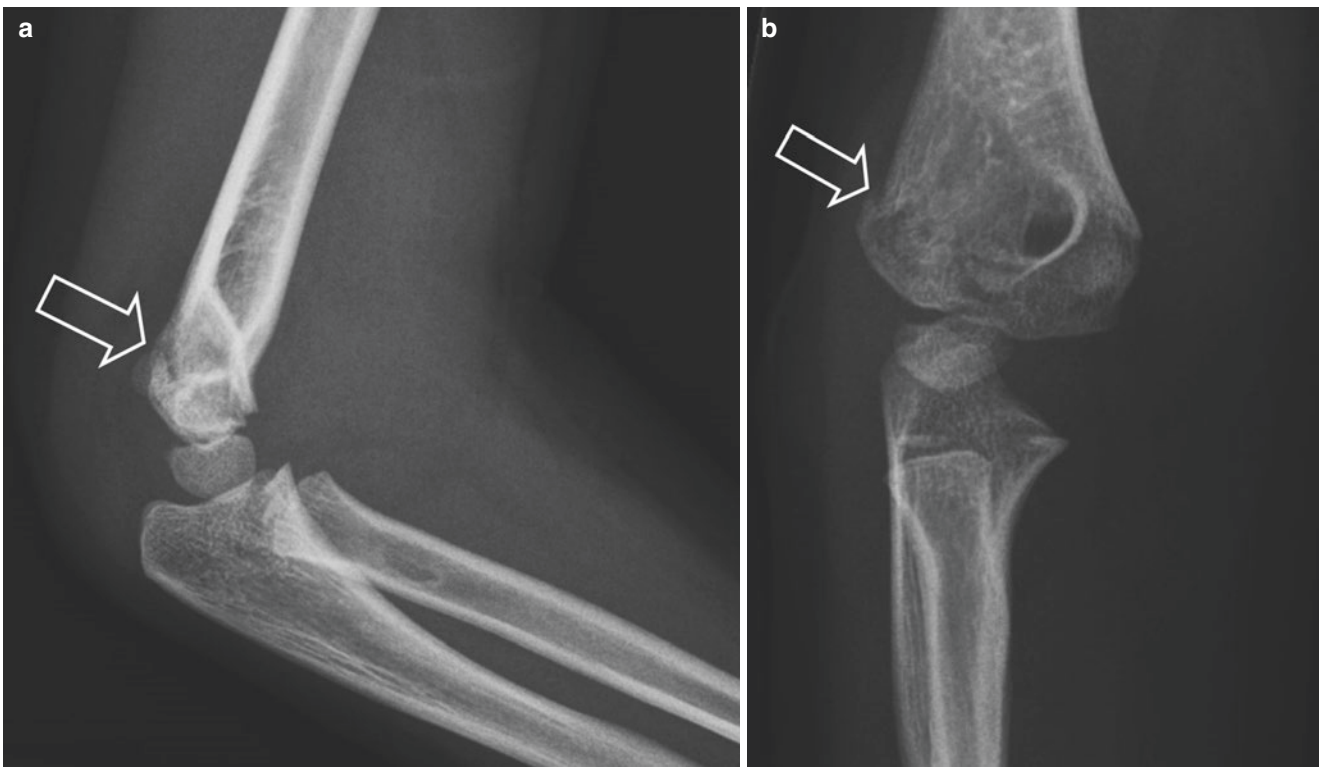


Fig. 13.17 (**a**) AP and (**b**) lateral radiographs of the elbow of a 6-year-old child who fell out of a bunk bed

tained head injuries did not show any neurological problems. Twelve children had a laceration of the scalp or face. Since MacGregor did not clearly distinguish between a fall from the upper and lower bed and a crib, only limited interpretation of the data is possible. Only intracranial injuries and skull fractures can be compared to other studies. Also, the author did not indicate how he eliminated non-accidental trauma to explain the injuries in a child.

A retrospective study by Mayr et al. confirmed the data from earlier studies on this subject [68]. They analyzed 218 bunk bed accidents in children. Of the children in the study, 23.8% was <3 years old. The main causes of injury were a fall from the upper bed while asleep (31.5%) or during play (34.4%) or a fall down the ladder (23.2%). Serious injury was found in 91 children (41.7%): multiple injuries (n = 3), skull fractures (n = 7), concussion (n = 44), fractures of the long bones (n = 33), Lisfranc's tarsometatarsal dislocations (n = 2), and lacerations of the spleen (n = 2) (Figs. 13.19 and 13.20). Sixty percent of children had less severe injuries, such as fractures in other locations than the skull or long bones (n = 18), contusions and sprains (n = 89), lacerations of the skin (n = 18), and dental fractures (n = 2). The authors concluded: '*there is only one recommendation: no bunk beds*'. Remarkable in this study is that again there are no serious intracranial injuries. In none of the children in this study intracranial haemorrhages were found, in spite of the large number and diversity of injuries after a fall from a bunk bed. Anyway, Johnson is of the opinion that Lisfranc's metatarsal dislocations should be considered typical 'bunk-bed' fractures [74].

Belechri et al. compared the injuries sustained by children up to 14 years of age from a fall from a bunk bed (n = 197) and a normal bed (n = 1684) [75]. In 8% of the 197 falls from a bunk bed, the child fell off the ladder. A fall from a bunk bed occurs mainly during sleep. In these cases, there were usually more serious injuries than in a fall from a regular bed: concussions, fractures, multiple injuries, and other injuries that resulted in hospitalization.

Conclusions

In spite of the large number, severity, and diversity of the injuries that are sustained by children that fall from a bunk bed, intracranial injuries are almost totally absent. Only the article by Selbst et al. mentioned a child that sustained a skull fracture and subdural haemorrhage. It is also remark-

Fig. 13.19 Two-year-old infant who fell from a bunk bed refusing to bear weight on her leg. Radiograph shows a distal tibia and fibula fracture (arrow)

Fig. 13.20 A 4-year-old child who fell from a bunk bed indicated pain in her hand. Radiograph shows an intra-articular fracture of the middle phalanx of the fifth finger

able that the studies did not report even one child that died after a fall from a bunk bed [73].

The US Consumer Product Safety Commission and the American Academy of Pediatrics have advised that children under the age of 6 years should not sleep in the upper bunk and that children should be discouraged from playing on bunk beds [76].

13.3.5.3 Pram

Introduction

From an early age onwards, children have been carried in prams/strollers. Especially the more mobile children regularly fall from prams, and in such cases there is very likely that the child will fall on its head.

Data from Literature

Watson and Ozanne evaluated the relation between accidents related to children's furniture and prams and the prevention of injuries [77]. Just over 6% of the injuries in children up to 3 years old and 19% of children up to 1-year old resulted from such an accident. The majority of injuries related to prams and high chairs appeared to be due to a fall (respectively, 75% and 83%). Watson and Ozanne consider the risk of sustaining serious injuries as high, since 96% of children who had fallen from a pram and 75% of children that had fallen from a high chair landed on their head. From 1985 to 1988, in Victoria (Australia), 1 child died from a fall from a pram and 1 from a fall from a high chair.

Couper et al. performed a retrospective study into accidents with prams (n = 149) [78]. Seventy percent of children were between 9 and 15 months old. Sixty percent suffered injuries to the head, face and/or teeth and/or had a concussion. Eleven children (7.4%) had to be hospitalized. The article did not mention any children that had died from the fall.

Lee and Fong describe a 10-month-old girl that suffered an epidural haemorrhage after a fall from a pram [79]. She did not sustain a skull fracture and post-operative made a full recovery. Moreover, Lee and Fong report that a review of the literature on similar situations showed that a fall from a pram seldom results in serious and/or life-threatening injuries, but does cause head injuries. Three children died after a fall from a pram that had been reported by the parents. In the end, it was found that in two children the injuries were due to non-accidental circumstances (child abuse).

Powell et al. executed the most comprehensive (retrospective) study into pram-related injuries in children up to 3 years old (n = 64.373, over a period of 5 years) [80]. The average age of the children was 11 months. The purpose of the study was a report on the incidence of this type of accident, the circumstances and the types of injury. Seventy-six percent of the injuries resulted from a fall from the pram, particularly on the head (44%) or to the face (43%). The injuries were: contusions and abrasions (38%), lacerations (24%), intracranial injuries (22%), and fractures of the extremities (3%). Two percent of children were hospitalized; 70% of the hospitalizations were for head injuries. The article did not mention any children that died from the fall. Several years later, also based on data from the National Electronic Injury Surveillance System (1990–2010) Fowler et al. evaluated stroller or carrier-related injuries in children ≤5 years of age [81]. In the study period, there were an estimated 261,879 (95% CI, 204,095–319,664) stroller-related injuries, of these children 42.0% were < 1 year of age. Like in other studies the most common location for injuries was the head and face (86.5%).

Arnholz et al. describe bilateral skull fractures in a 6-week-old infant who fell from a distance of about 90 cm from a pram and landed with the top of the head on concrete stairs (Fig. 13.21) [82]. Two symmetrically located haemorrhages on the scalp were found as associated injuries.

Tripathi et al. reported on 248 pram- (N = 136) and stroller-related (N = 111) injuries collected in young children (median age 12.5 months) over a 3-year period in 1 an emergency department of a paediatric hospital in Singapore [83]. In all children most injuries (90.7%) were to the head and/or face and only a few of these required medical care. There were 17 (6.9%) fractures/dislocations, however, the authors, except for one depressed skull fracture in an 8-month-old child, did not describe the actual fracture locations. In general, the injuries were the result of blunt force trauma (97.6%), were under supervision of an adult (79.4%), and happened in and around the house (46.8%).

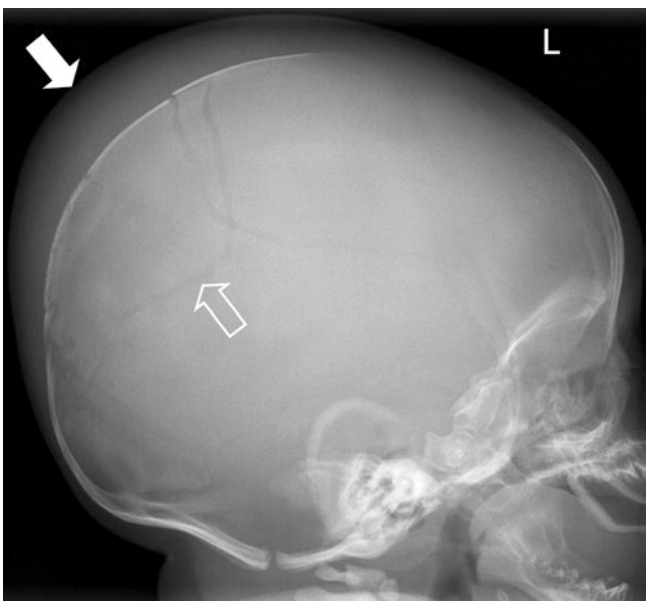


Fig. 13.21 Ten-year-old child who fell out of his travel bassinet down the stairs (14 steps). After the fall he was drowsy, a radiograph of the skull showed soft-tissue swelling (arrow) and a linear biparietal fracture (open arrow)

Conclusions

A fall from a pram, in particular in children under the age of 1 year, is not an unusual occurrence. Often children will fall on their head. Serious intracranial injuries (a typical impact injury: epidural haemorrhages) are only reported in case reports. Lasting damage is rare, as is death.

13.3.5.4 Infant Seating Device

Introduction

Infant seating devices are widely used and may well be one of the most sold infant consumer products [84]. These products are sold under a wide variety of names which sometimes overlap, making evaluation of the literature at times difficult. For this section, the seating devices have been into two groups: bounce chairs (Fig. 13.22) and variations on that theme and car seats used outside a vehicle as a seating device.

Bounce Chair Data from Literature

In their article, Farmakakis et al. report on 181 children that sustained injuries by a fall with a bounce chair [85]. Beaudin et al. reported on the risk of injuries with the inappropriate use of seating devices based on data from the Cincinnati Children's Hospital [86]. In their study there were, among others, 22 children with injuries related to a bouncy seat and 2 related to a Bumbo seat (these were actually recalled in 2007 due to safety issues and should only be used if a restraint belt is fitted). All children were admitted to the hospital, one of them to the paediatric intensive care unit. The authors did not specify the injuries sustained as a result of these incidents. Wickham and Abrahamson reported children <1 year with head injury, who, in an one-year period presented at their accident and emergency department [87]. In this study period 131 children were seen with head injuries of these 17 (13%) were related to bounce chair or car seat, and in all cases of bounce chair injuries the cause was a fall of the bounce chair from an elevated surface.

Fig. 13.22 Bouncy chair

Car Seat Data from Literature

Infant car seats are essential in preventing injuries in babies and infants when they are involved in a car crash and as such they are an indispensable tool in preventing serious injuries or even death. It has been reported that due to the use of car seats in the United States there has been a decrease in fatal injuries by 71% [88]. However, these seats are also used outside the vehicle as an infant seating device and if used incorrectly then they can pose a threat to the welfare of the child. Incorrect use may result in injuries.

Parikh and Wilson looked, based on NEISS data, at car seat accidents in the United States when used outside a car in the period 2003–2007 [89]. They estimated that in this period a total of 43,562 children were seen in emergency departments due to such accidents. In the majority of cases (at least 49%) the accident occurred at home and in all cases the majority of accidents (64.8%) were caused by infants falling out of their car seats. In these cases the infants either fell when carried or when the car seat was placed on an elevation (e.g. a table or car). In all of these cases the authors assumed that the infants were unrestrained in their seat, although data to support this statement was available for only 16.8% of cases. In 14.6% the accident was caused by the car seat itself falling from a height. The head and neck were most often (84.3%) injured and internal organ injuries, soft-tissue injuries, and fractures occurred in, respectively, 45.3%, 33.4%, and 6.6%. In most of the infants, the injuries were minor and they could be treated as out-patients (89.9%) with only a minority admitted (8.4%) and a very small percentage of fatal injuries (0.1%). In a Canadian study, based on BC Children's Hospital data from the Canadian Hospital Injury Reporting and Prevention Program, by Desapriya et al. similar results were found [90]. Although the study only included 87 children falls from elevated surfaces (43%) were the most common reported trauma mechanism. In a study by Pollack-Nelson attention was brought to the risk of car seats toppling when placed on a soft surface, e.g. bed or couch [91]. Although rare in the study period from 1990 to 1997, a total of 16 cases of suffocation after the seat overturned were reported.

Another trauma mechanism reported by Greenberg et al. is a fall out of a car seat when it is used as a carrier [92]. They reported on 62 children <18 months who fell out or with a car seat of them a subset of 9 children fell while being carried, four of these children sustained intracranial injury. In the whole study group a staggering 87% of children were not buckled into the car seat when the incident occurred.

Conclusions

Although infant seating devices are generally safe, improper use can lead to serious injuries. Under all circumstances it is advised to follow the instructions and have the child restrained/buckled up when seated. Based on the reported injuries and trauma mechanisms in the literature placement of infants in seating devices on an elevated surface should under all circumstances be discouraged. In 2017, the US Consumer Product Safety Commission introduced a federal mandatory standard to improve the safety of infant bouncer seats, in this standard manufacturers must make fall hazard warnings more visible to parents/caregivers [93]. Among others they also advise parents/caregivers to always use the bouncer on the floor, and never on an elevated surface, to never place the bouncer on a soft surface, and to always use restraints and adjust restraints to fit snugly.

Although slightly off-topic the following is also of interest, Liaw et al. reported, based on 2004–2014 data from the US National Center for Fatality Review and Prevention that in that period out of a total of 11,779 infant sleep-related deaths, 348 (3.0%) occurred in infant seating devices [94]. Of these cases in 62.9% a car seat was involved and in the majority of cases the guidelines for use were not followed. In the discussion, the authors state that infants should never sleep unsupervised in car seats and that, although their data did not provide detail regarding buckling, they agree that infants should also always be buckled with the 5-strap harness, even when asleep. Batra et al. reported, based on between 2004 and 2008 US Consumer Product Safety Commission data, on 47 fatalities involving among other seating devices (31 car seats and 4 bouncers) [95]. In all cases the cause of death was either positional asphyxia or strangulation (resulting from improper use of the straps). Barber et al. reported on a series of coronial autopsies, between January 1996 and December 2011, performed in Great Ormond Street Children's Hospital, London [96]. Out of a total of 1465 infant deaths 14 were related to a car seat (0.96%) incident. In 9 cases (64%) the car seat was used as either a cot or a seat. None of the deaths were the result of a fall.

13.3.5.5 High Chair

Introduction

From the moment that children are well able to sit up and eat solid food, parents/carers will usually place them in a high chair at the dining-room table. In spite of the straps in these chairs, children regularly fall out of them.

Data from the Literature

In Europe, it is estimated that within the European Union approximately 7700 injuries to children 0–4 years of age occur which are severe enough that medical attention is sought (Fig. 13.23a, b) [72].

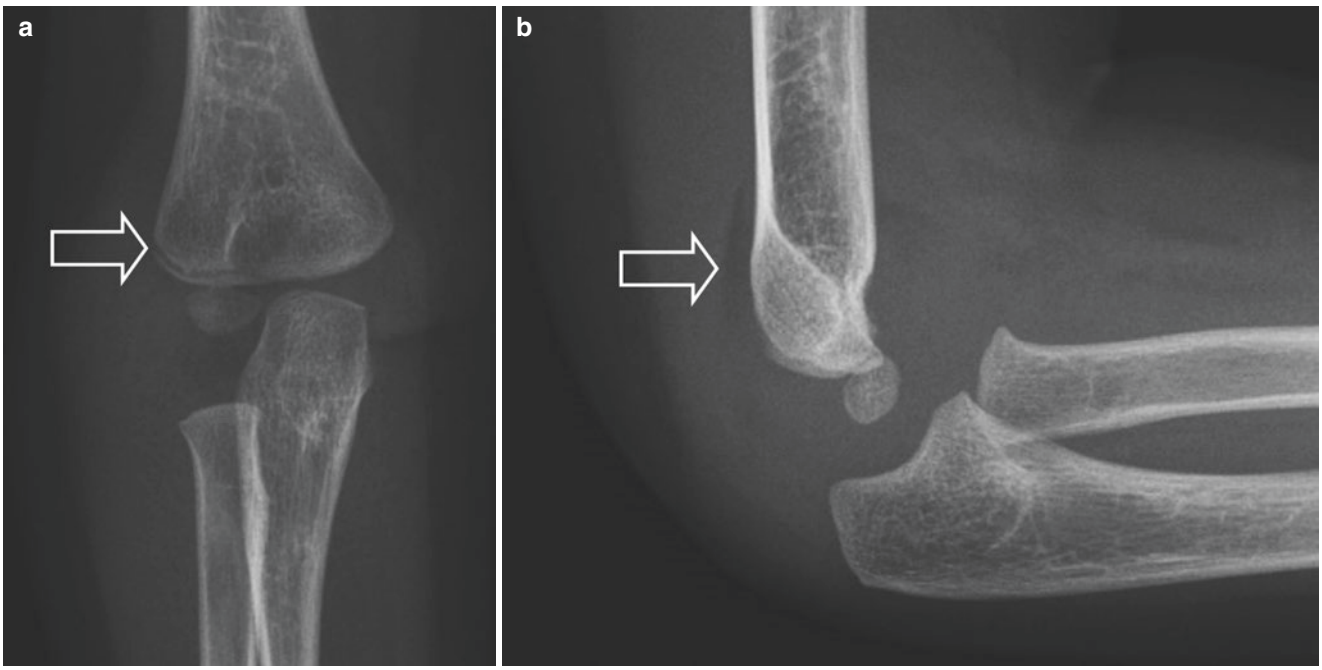


Fig. 13.23 Two-year-old child who fell out of a high chair. (**a**) AP radiograph of the elbow shows a lateral condyle fracture (arrow). (**b**) On the lateral radiograph the fracture cannot be seen, but there clearly is a posterior fat-pad sign (arrow)

Watson and Ozanne evaluated the relation between accidents resulting from children's furniture and prams and the occurrence of injuries [77]. In this study 20 cases of falls involving a high chair could be included, the most common injury was to the head (75%) other injuries were a fractured clavicle and 2 cases of a fractured ulna and radius.

By means of a questionnaire directed to the parents, Mayr et al. evaluated 103 children that had fallen from a high chair and presented at the emergency department [97]. Fifty percent of children wanted to stand up in the chair before they fell down, and 14% of accidents occurred because the high chair toppled over. Most children had suffered head injuries: contusions or haematomas on the head or lacerations of the scalp or face (68.9%), skull fractures (15.5%), and concussions (13.6%). The article does not mention any children that died as a result of the fall or ended up in a life-threatening situation.

Powell et al. carried out the most comprehensive (retrospective) study into injuries in children up to the age of 3 years old, related to a fall from a high chair (n = 40,650, average age 10 months, over a period of 5 years) [98]. The purpose of the study was to compile a report on the incidence of this type of accident, the circumstances, and the types of injury. Over 5000 children (13%) fell from a booster seat and over 4000 children fell from a youth chair. Predominantly head (44%) and facial (39%) injuries were found: contusions and abrasions (36%), lacerations (25%), intracranial injuries (21%), and fractures (8%). Two percent of the children were hospitalized. The article does not mention any children that died as a result of the fall.

Kurinski et al. described data from the NEISS from 2003 to 2010 [99]. They found that annually there were on average 9421 high chair-related injuries in children aged up to 3 years of age in the United States and that in the study period there was a significant increase of 8926 injuries in 2003 to 10,930 injuries in 2010. A fall was the most reported trauma mechanism (92.8%). During the study period closed head injury occurred in 26,649 children (95% CI: 18,753–34,545) and fractures in 6169 (95% CI: 4578–7760).

Although most injuries are a result of a fall out of the chair, backward falls have also been described. Mayr et al. reported that in 9 (mean age of 16 months, range: 9–30 months) of their 103 cases a backward fall occurred when their highchair tipped over [97]. Atkinson et al. described 8 cases of backward falls with occipital impact, in one of these cases a fall occurred when a high chair tipped over [100]. In this case of a 10-month-old boy a subdural haematoma and unilateral retinal haemorrhages were found. A full workup disclosed no other injuries or cause to doubt this consistent clinical history.

A rare fatal injury mechanism was described by Souheil et al. who described a case of a 2-year-old girl who died as a result of accidental asphyxiation [101]. In this case the child herself had climbed in the chair and fastened the waist strap and not the crotch strap, this allowed her to slip out of the chair and accidentally hang herself on the waiststrap.

Conclusions

Children, in particular those <1 year old, regularly fall from or with high chairs. The literature mentions serious or even life-threatening injuries only in a very small proportion of children. A fatality from a fall is only mentioned in the article of Watson, this child was not included in their study sample [77].

A common finding in all studies was the lack of use of restraints, even if they were present, this would therefore be an easy method to prevent falls from highchairs.

13.3.5.6 Baby Jumpers

Introduction

The baby jumper, also known as a suspended baby exercisers or bouncers, is a recreational device for children who are well able to keep their head upright, but cannot walk yet. The baby is seated in a chair made of soft cloth that is hung from the ceiling, the door opening, or a frame by a spring. By pressing his/her toes against the floor, the infant can push up him-/herself up, and in this manner can move up and down. A baby bouncer is not made for swinging. According to the Consumers and Safety Council its use is not without risks. When incorrectly hung or attached, the jumper may come loose. Also, the baby may jump too hard or fall out of the jumper.

Data from Literature

Although warnings are mentioned by several organizations and newspaper articles mention injuries, actual data on the incidence and case reports are extremely rare [102–105]. In a study on nursery product-related injuries, based on 21 years of NEISS data, the group of baby walkers/jumpers/exercisers related injuries accounted for 16.2% of all causes. Claydon presents a case of a fatal fall from a baby jumper by a child <1-year old (Fig. 13.24) [106]. The author mentions with good reason that to sustain life-threatening head trauma, it is not necessary for the child to fall from any great height. The head of the child in question was no more than 60 cm from the floor. The child landed on its head on thick carpet.

Conclusions

Although injuries can certainly occur in the use of baby jumpers there is insufficient literature to specify which injuries can specifically be found.

Fig. 13.24 Baby jumper

13.3.5.7 Exersaucer

Introduction

An exersaucer is a stationary device in which a non-mobile infant can sit in an upright position. By pushing his feet against the floor or base the infant can rotate and play with the toys on the exersaucer. It is a predecessor to the infant walker (see Sect. 13.3.5.8). The exersaucer is reported to have a beneficial influence on the infant's motor development and is therefore used by parents [107].

Data from Literature

In 2001, Grant et al. presented two cases in which young children (respectively 7 and 4.5 months of age) presented with a distal femoral fracture [108]. In both cases, the parents reported the use of an exersaucer for their child, although in neither case the fracture was reported directly after use. The parents of the 7-month-old child brought the exersaucer to the hospital and stated that they did not adjust the height of the seat and as a result the child could plant her feet on the base and twist. In both cases imaging revealed no other fractures, and after a full workup both cases were ruled as accidental related to the exersaucer.

Moineau and Plint described a case of a 9-month-old boy who presented with bilateral buckle fractures of the proximal tibia [109]. Although the authors conclude that in their case the circumstances, under which the fractures were sustained, remain unknown they state in the discussion of the case '*the parents could not think of, and the babysitter did not admit to, any potential traumatic event while in their care. When reviewing any possible repetitive stresses occurring on his lower limbs, the parents admitted that he was often in his baby stationary activity center, and the sitter had mentioned that he had been in it for a few hours the day he seemed more irritable*'. It thus seems plausible that there is a relation between the use of the exersaucer and the occurrence of fractures.

Conclusions

Although rare, there have been reports of lower extremity fractures related to the use of an exersaucer. In all reported cases the fracture was not identified directly after the use of the exersaucer but the full clinical workup led to the conclusion that this could have been the only explanation.

The same exersaucer as reported in the publication by Grant et al. can, once a child can walk, also be used as an activity table. However, it has been shown that the cap on one end of the product can loosen and fall off, posing a fall hazard to a young child [110]. After receiving reports of dislodged end caps which resulted in minor injuries, including bumps and bruises and in one case a broken collarbone, this toy has been withdrawn from the market.

13.3.5.8 Baby Walker

Introduction

Many parents use baby walkers for their infants either because they believe its use will accelerate gait (for which there is no evidence), it will increase the leg strength of the baby, or just because it is deemed to be entertaining [111–113]. The literature shows that baby walker-related injuries happen regularly. In the United States, the number of reported accidents has increased since the eighties [114, 115]. In a study based on the data from the NEISS data from 1990 to 2014 Sims et al. reported that in that period 230,676 children under the age of 15 months were presented at an emergency department for baby walker-related injuries [116]. Based on an evaluation of nearly 28,000 children up to 4 years of age who presented at the emergency department due to accidents with baby walkers, Trinkoff and Parks concluded that the majority of these accidents could have been avoided (Fig. 13.25) [114].

Data from Literature

In 1982, Fazen and Felizberto performed a study into the use of baby walkers and the frequency and severity of the injuries that resulted from accidents with baby walkers [117]. For this purpose they approached the parents of 49 children

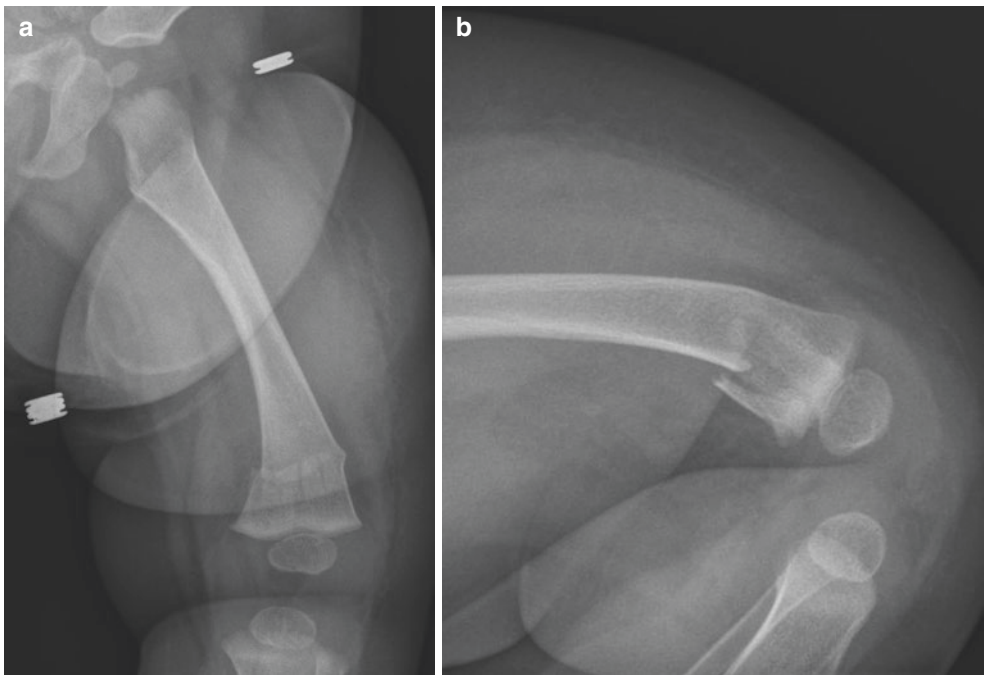


Fig. 13.25 Eight-month-old infant who fell, while being unrestrained, out of an stroller. (**a**) AP radiograph of the left femur shows a distal metaphyseal fracture. (**b**) Lateral radiograph shows buckling of the posterior cortex

8–14 months of age. Most parents (86%; $n = 42$) appeared to have baby walkers. Their children were put in the baby walker for the first time when they were 4 months old. Approximately 50% of the children had experienced an accident with the baby walkers at some time: a tip over, a fall down the stairs or entrapment of fingers. In two children medical assistance was required. Both sustained head and neck injuries after a fall down the stairs in their baby walker. Fazen and Felizberto concluded that injuries from accidents with baby walker are a regular occurrence, but were not as severe as had been reported up to then. Their study revealed that particularly children <7 months old fell down the stairs, whereas tipping over happened more often in children >8 months.

Kavanagh and Banco evaluated the data of 195 children of 5–15 months of age [118]. One hundred and fifty children (77%) had been put in a baby walker by their parents. In 47 children (31%) the accident caused visible injuries: bruises/contusions and abrasions in 38 children, head trauma (including skull fractures) in five children, lip lacerations in two children, perforation of the palate in one child and a dental avulsion, also in one child. Thirty-eight children tipped over, 15 children fell down the stairs, and two children had been pushed over by their sibling. A 7-month-old child had a large haematoma fronto-parietal and multiple skull fractures (two left frontal, of which one impression fracture and one fracture to the left parietal bone) after a fall down the stairs. The child remained conscious and the neurological examination showed no defects.

Wellmann and Paulson studied children that presented at a large emergency department over a period of 23 months for baby walker-related accidents [119]. They reported that 97% of the children had sustained head and facial injuries. Sixty-eight percent of the children had fallen down the stairs. In 22% of injuries, it was necessary to get a surgical or dental evaluation in addition to the paediatric examination.

Stoffman et al. investigated the relation between head injuries and the use of baby walkers in 52 children of <24 months old [120]. In children of <1 year old, it was shown that 42% of head injuries were caused by accidents with baby walkers. In children aged 12–24 months, baby walkers were not at all involved in sustaining head injuries. In all baby walker-related head injuries the child had fallen down the stairs. Three children sustained a skull fracture.

Rieder et al. carried out a prospective study into the mechanisms and patterns of baby walker-related injuries [121]. The study was initiated by the death of a 6-month-old infant who had fallen down a 14-step staircase onto a concrete floor and had received fatal injuries. Over a period of 1 year, Rieder et al. saw 139 children of 4–15 months old that had sustained injuries. Twenty-nine of them had sustained a fracture, and 123 children had fallen down the stairs (89%). Ten children had fallen from a baby walker. Three children had sustained burns and 3 others had pulled an object on top of themselves. Ninety-three children sustained a skull fracture, of whom two had a depression fracture. Furthermore, in three children a fracture of the lower arm was found, and in two children a clavicle fracture. One child suffered a fracture of the nasal septum. There also were lacerations ($n = 6$), abrasions ($n = 3$), and burns (=3). In six children dental injuries were found. The most serious injuries were seen in falls down the stairs; all fractures but one resulted from falls down

the stairs. Approximately 90% of children that had sustained a closed head injury had also fallen down the stairs.

Partington et al. studied the relation between the origin of head injuries (except for facial injuries) and the use of baby walkers in children <24 months [122]. Over a period of 3 years, they saw 129 that had suffered a head injury. In 19 of the children (14.7%) the injuries were caused by an accident with a baby walker. The average age of these children was 8.7 months. Eighteen children fell down the stairs, of which nine had sustained a skull fracture (six linear, two multiple linear, and one complex). None of the children required surgical intervention.

Al-Nouri and Al-Isami interviewed a random selection of 100 mothers, who visited the emergency department with their child <4 years old, and of those 83 stated that they used a baby walker for their babies [123]. Of these children 78 (94%) sustained an accident leading to a total of 148 injuries. The majority of these injuries were minor and involved head trauma (82% of injuries), in one child a skull fracture was reported.

Coats and Allen carried out a retrospective study on children <24 months that presented at the emergency department because of an accident with a baby walker [124]; in a total of 1049 visits, 22 children. The most serious injuries seen in these children were skull fractures (n = 3). The majority of injuries were caused by a fall down the stairs.

Chiavielo et al. carried out a 44-month prospective study into the incidence and severity of baby walker-related injuries in children of 3–17 months old (n = 65) [125]. Of these children 95% was <1 year old. The researchers excluded all children suspected of having sustained in non-accidental circumstances. The injuries were sustained by a fall down the stairs (71%, n = 46), tipping over (21%, n = 14), a fall from the porch (3%, n = 2), and burns (5%, n = 3). Most injuries were found on the head and in the face (97%). Furthermore, injuries were found on the extremities (6%) and the trunk (3%). Most of the injuries were light, although 19 children had suffered severe injuries: skull fracture (15%, n = 10), concussion (12%, n = 8), intracranial haemorrhages (8%, n = 5), third-degree burns (3%, n = 2) and a fracture of the cervical spine (2%, n = 1). One child in the study died, after sustaining a skull fracture, a subdural haemorrhage, and a fracture of a cervical vertebra. When the burn patients were excluded, severe injuries were only seen in children that fell down the stairs.

Mayr et al. studied retrospectively the data of 172 children of 7–14 months of age that had sustained baby walker-related injuries over a period of 3.5 years [126]. They found the following injuries: skull fracture (n = 19), concussion (n = 23), contusions and lacerations to the head (n = 125), including dental luxation), and fractures or distortions of the upper extremities (n = 3).

The study of Petridou et al. on 49 children with baby walker-related injuries confirms the earlier-mentioned data [127]. The majority of injuries were sustained around the age of 9–10 months. The most prevalent cause of injury was a fall down the stairs, in particular in the younger children. The majority of injuries were light. Three children sustained fractures.

Smith et al. investigated baby walker-related injuries in 271 children that presented at the emergency department over a period of 3 years [128]. Their age ranged from 4 to 36 months (average age 9.2 months, 62% boys). Sixty-nine percent of children had sustained the injury from a fall down the stairs. The risk for skull fractures and the necessity of being hospitalized increased with the number of steps the child had fallen down. One hundred and fifty-nine children (58.6%) sustained skin injuries (contusion, haematoma, abrasion), 35 (12.9%) had a concussion and/or head injuries and 33 (12.2%) had lacerations. Twenty-six children (9.6%) sustained a skull fracture (17 parietal, 8 frontal, and 1 occipital) and 4 (1.5%) showed other fractures (three clavicle and one radius and ulna). Nine children (3.3%) had sustained a nose bleed, four (1.5%) a dental avulsion, and one (0.4%) a burn. In three children a depression fracture of the skull was found, two of those also had a second skull fracture without depression. Three children that had suffered a skull fracture also had intracranial haemorrhages, of which twice a subdural haemorrhage. The skull fractures occurred only in the group that had fallen down the stairs. Ten children (3.7%) were hospitalized; they all had a skull fracture. Smith concluded that fractures of the extremities are rare in baby walker-related falls.

In the study by Sims et al. 7–10 months old accounted for 68.3% of all cases [116]. Based on their study data the authors calculated that in the period 1990–2014 in the United States a total of 115,897 (95% CI 94,575–137,220) sustained a closed head injury and 11,798 (95% CI 8435–15,160) a fracture. In the majority of cases (74.1%) a fall down the stairs was the trauma mechanism, the second cause (14.7%) was a fall from the baby walker itself.

In 2001, the American Academy of Pediatrics (AAP) issued an advice on baby walkers [115]. According to the AAP, in 1999 approximately 8800 children <15 months were treated in emergency departments for baby walker-related injuries. The majority of injuries were caused by falls down the stairs and head injuries were seen frequently. Between 1973 and 1998, physicians reported that 34 children had died as a result of a fall with a baby walker. The AAP discourages the use of baby walkers due to the considerable risk for light to very severe injuries and death. Since there are no positive indications for its use, their advice is to prohibit the production and sale of baby walkers.

Conclusions

After a fall with a baby walker, in which falls down the stairs are the major trauma mechanism, the majority of injuries seen are to the head or the face and are relatively harmless.

Skull fractures occur very regularly, whereas fractures of the extremities are rarely seen. In 2–3% of the reported children, intracranial haemorrhages are seen. Unfortunately fatalities are also reported regularly [115].

13.3.5.9 Stair

Introduction

Almost all parents have experienced that a child fell down the stairs signifying that it is a relatively common event. According to the NEISS data in the period 1990 through 2012 a total of 3,667,512 patients under the age of 10 years were treated at emergency departments for injuries related to fall from stairs [129]. Zielenski et al. evaluated data, from the same database, from 1999 to 2008 focussing on children aged <5 years [130]. They found that in this study period a total of 931,886 (95% CI: 799,283-1,064,490) children, i.e. on average 255 children per day or 46.5 injuries per 10,000 children annually in the United States, were treated for injuries.

Falls from a stair are also reported in cases in which, after a thorough work-up, it was concluded that injuries were due to non-accidental trauma (Fig. 13.26a, b). In an article by Pierce et al. the authors refer to a case review of 100 children with non-accidental injuries in the Children's Hospital of Pittsburgh. In 11 cases a fall downstairs was reported in the clinical history [131]. There were severe injuries; 9 children had fractures, 4 had permanent sequelae, and 3 died. According to the authors the perpetrator in most cases confessed to fabricating the stair fall history.

Data from Literature

The study by Zielenski et al. also provides data on the trauma mechanism and injuries sustained by children (Table 13.7) [130]. During the study period there was a significant decrease in stair-related injuries, a large part (but not exclusively) of this decrease could be attributed to a decrease in baby walker-related injuries.

In a prospective study, Joffe and Ludwig described 363 children from 1 month to nearly 19 years old with injuries resulting from a fall down the stairs (average age: 55 months) [132]. Fifty-four children were < 1 year old. Ten children were being carried by their parent/carer. Twenty-four were in a baby walker when they fell down the stairs. Children suspected to have non-accidental injuries were excluded from the study. The majority of children had superficial injuries, 73% sustained injuries to head and neck. Head injuries were more frequently seen in children <4 years of age. In 28%, injuries of the extremities were found, in particular distally. Only in 2% of children injuries to the trunk were seen. Six

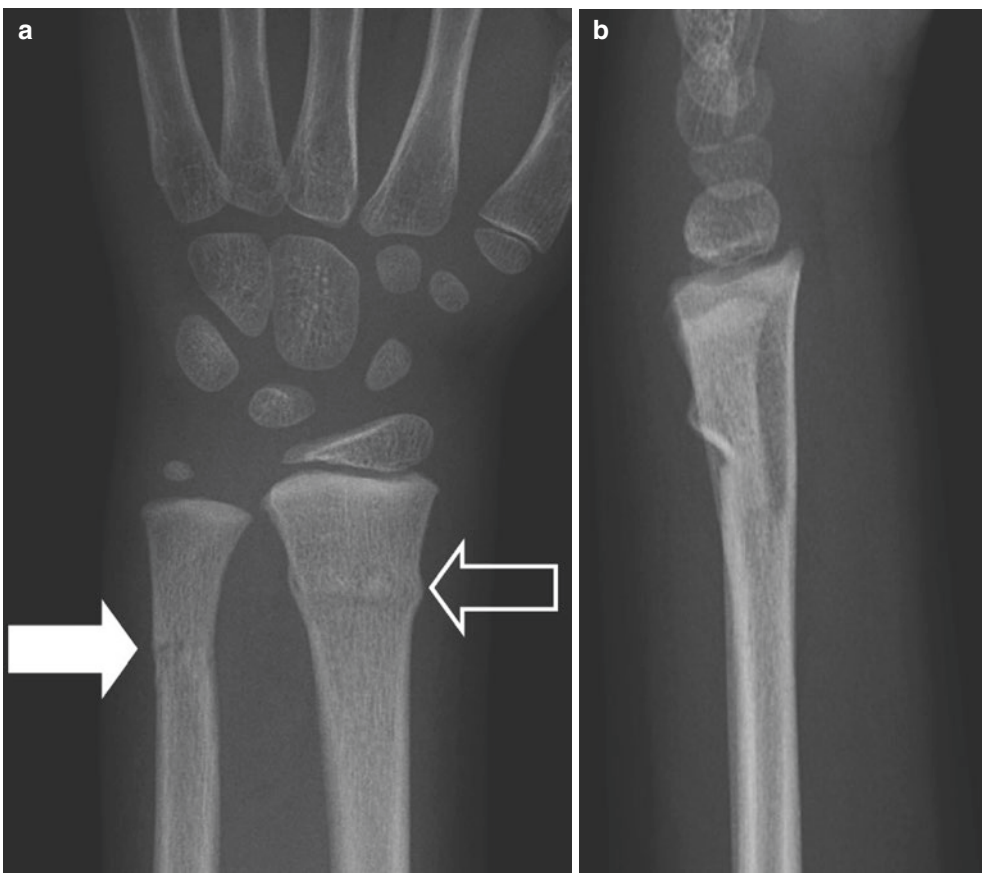


Fig. 13.26 Six-year-old child who was pushed down the stairs. (**a**) AP radiograph of the wrist shows a greenstick fracture of the distal ulna (arrow) and a cortical irregularity of the distal radius (open arrow). (**b**) lateral radiograph shows a torus fracture of the distal radius

Table 13.7 Stair-related trauma mechanism and injuries (children <5 years) based on National Electronic Injury Surveillance System data 1999–2008 [130]

	<1		1		2		3		4	
	National incidence	%	National incidence	%	National incidence	%	National incidence	%	National incidence	%
Injury mechanism										
Carried	33,531	24.5	12,171	4.0	4999	2.2	1392	0.9	1054	0.9
Fell in baby walker	22,226	16.2	3145	1.0	#		#		#	
Jumped	#		1506	0.5	4519	2.0	3687	2.5	4355	3.7
Fell from stroller	6860	5.0	2704	0.9	381	0.2	#		#	
Rode toy	#		2370	0.8	3791	1.7	2223	1.5	1561	1.3
Tripped on object	#		547	0.2	346	0.2	#		#	
Fell without object/ action	74,037	54.0	279,519	92.6	212,778	93.7	141,220	94.5	109,129	93.6
Injury type										
Soft tissue	54,487	43.3	115,215	38.4	72,239	31.9	41,362	27.8	32,838	28.2
Laceration	8797	6.5	61,836	20.6	68,890	30.4	56,269	37.8	47,191	40.5
Closed head	43,644	32.3	71,166	23.7	39,299	17.4	21,916	14.7	13,277	11.4
Fracture	10,047	7.4	24,948	8.3	26,410	11.7	19,483	13.1	16,421	14.1
Dislocation	#		6557	2.2	5003	2.2	1527	1.0	571	0.5
Miscellaneous	13,707	10.2	20,115	6.7	14.411	6.4	8417	5.6	6107	5.2

$N < 20$

percent of the children ($n = 22$) had sustained fractures due to falling downstairs. Sixteen children had suffered a fracture of one of the extremities (15 of them were > 4 years old). Six children had suffered a skull fracture (all <3 years old). Four of the six skull fractures were sustained in ten children who fell on the stairways from the hands of their parents/carers. None of the children showed rib, spinal, pelvic, or hip fractures. The group of children of 6–12 months old counted 40 injuries in total. Twenty-four of those injuries were seen in the 24 children that had fallen in their baby walker (see paragraph 13.3.5.6). Only 2.7% of children had sustained injuries to more than one body part. Children who had fallen more than four steps down the stairs did not sustain more injuries than children who had fallen less than four steps down the stairs, irrespective of their age. Also, the injuries were of similar severity. Three percent was admitted to the hospital. None of the children experienced a life-threatening situation. Also, none of the children was admitted to the intensive care unit, no intracranial haemorrhages or brain contusions were found, and none of the children died. Joffe and Ludwig concluded that there was no relation between the number of steps a child falls down and the severity of the injury, and that a fall from a staircase is less serious than a free fall from the same height. When a child presents with multiple and severe injuries to the trunk or extremities, and according to the clinical history he/she has fallen down the stairs, then non-accidental circumstances should be considered.

Chiaviello et al. also studied the effects of a fall down the stairs in 69 children of <5 years of age (average age 2 years), including three children who had fallen together with their parent/carer [133]. Unlike Joffe, they excluded accidents with baby walkers. They also excluded children suspected to have non-accidental injuries. The majority of the injuries were not serious. Fifteen children had sustained serious injuries, such as concussion (11 children, 16%), skull fracture (five children, 7%), brain contusion (two children, 3%), subdural haemorrhages (one child, 1%), and a fracture of the second cervical vertebra (one child, 1%). The three children that had been carried by their parent/carer who fell on the child against the staircase sustained the most serious injuries: two children suffered a skull fracture, and one of the children showed a small subdural haemorrhage and contusion. This was also the child who had sustained fracture of the second cervical vertebra. Chaviello et al. concluded that in the majority of falls there were no serious injuries. Injuries of head and neck prevailed and serious head injury can occur after an accidental fall. However, if a child has injuries to multiple body regions or serious truncal or extremity injuries then alternative mechanisms, including non-accidental injury, should be considered.

Docherty et al. studied stair falls in Sheffield, United Kingdom, in the fiscal years 2005/6 and 2006/7 and found 239 paediatric cases median age of 1 year (range 0–15 years) [134]. Of these children 82/239 (34%) fell a full flight of stairs, i.e. more than 11 steps, of these children 68 (83%) sustained injuries. Overall 216/239 (90%) children suffered some form of injuries after a fall from a stair, the majority had a minor head and facial injury (165/239, 69%). There were a total of 28 fractures, it is of interest to note that all 5 skull fractures in this study population were found in children who were dropped by carers on the stairs.

Pierce et al. developed an injury plausibility model for children with femur fractures after a fall from the stair (Fig. 13.27) [131]. This model is based on four categories

Fig. 13.27 Thirteen-month-old child who suffered a witnessed fall from the fourth step of the stairs. Radiograph shows a transverse mid-diaphyseal femur fracture

(1) history details; (2) biomechanical compatibility of the fracture morphologic features; (3) time to seeking care; and (4) other injuries. Applying the model to their study population showed a difference between falls in accidental and non-accidental circumstances. Although this is a very interesting approach the authors state that *'Additional research is needed to develop and test this model and similar models that provide more objective injury assessment and differentiation between abusive and noninflicted trauma'*.

One of the most widely advised interventions to prevent falls from stairs is the use of 'baby gates'. These, however, can also give rise to accidents [135]. These injuries can be the result of, e.g. contact injuries, including cuts/lacerations from sharp parts, open gates, gates collapsing, or children climbing over the gate (Fig. 13.28). In the latter group the injuries were more severe leading to more hospitalizations.

Conclusions

Based on the literature, and experience of most parents, children regularly fall down the stairs. Usually there are few or no injuries. However, when injuries are sustained, they are usually seen in the head, neck, and distal extremities (Figs. 13.29 and 13.30a, b). When a child presents with multiple and severe injuries to the trunk or extremities, and it is stated that the injuries were sustained due to falling down the stairs, then non-accidental circumstances should be considered/excluded.

Fig. 13.28 Five-and-a-half-year-old child who sustained a spiral fracture after a fall down the stairs. The clinical history showed that he had climbed over the stair barrier holding a toy in his hand, caught his leg in the spindles and lost his balance. A police investigation at the site of the accident confirmed the clinical history

13.3.5.10 Shopping Cart

Introduction

Children are often placed in shopping carts; in the designated seats as well as in the cart itself. A fall from a shopping cart is a regular occurrence, possibly due to diminished attention of the parents while shopping.

Data from Literature

Shopping cart-related injuries are a major source of injuries in children [136]. In the United States, from 1990 to 2011, 530,494 children younger than 15 years were treated in emergency departments [137]. This means that on average

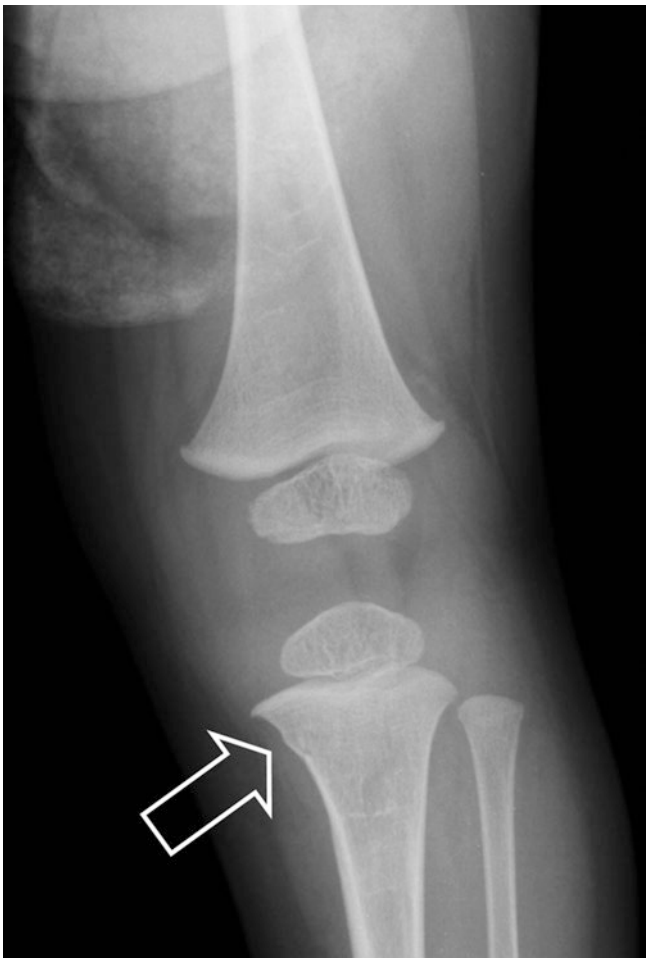


Fig. 13.29 Fourteen-month-old infant after witnessed fall down the stairs. The proximal tibia shows a torus fracture (arrow)

24,113 children annually, or 66 children daily are seen with shopping cart-related injuries. The majority of cases (84.5%) were children aged 0–4 years. Wright et al. performed a similar study in a shorter time frame and also found that children <5 years were most commonly injured [138].

Smith et al. evaluated retrospectively the emergency department data of over 75,000 shopping cart-related injuries in children of 15 years old and younger (84% <5 years old) [139]. The most common trauma mechanism was a fall out of the shopping cart. Head and neck injuries were the most prevalent (74%). Of the children that had sustained injuries, 2.7% had to be hospitalized (93% <4 years old), mostly due to fractures (45%), followed by internal injuries (22%), and concussion (17%). The body location that was most often injured was the head (78.1%) followed by the upper extremities (13.8%). Again the majority of head injuries (90.7%) were sustained by children under the age of 5 years. In the whole study population a total of 1326 fractures were reported. Unfortunately, the fractures were not specified.

In another prospective study, Smith et al. evaluated 62 children from 4 months to 10 years old (average age 2.8 years), who had presented at the emergency department due to shopping cart-related injuries over a period of 15 months [140]. Twelve children were presented by ambulance. Forty-nine children (79%) were found to have sustained a head injury. Fractures were found in 11 children (18%: 5 skull fractures, 2 femur fractures, 1 metatarsal fracture, 1 clavicle fracture, and 1 radius and ulna fracture). Nine children (14%) had sustained lacerations and 30 children (48%) had suffered superficial injuries. Most children sus-

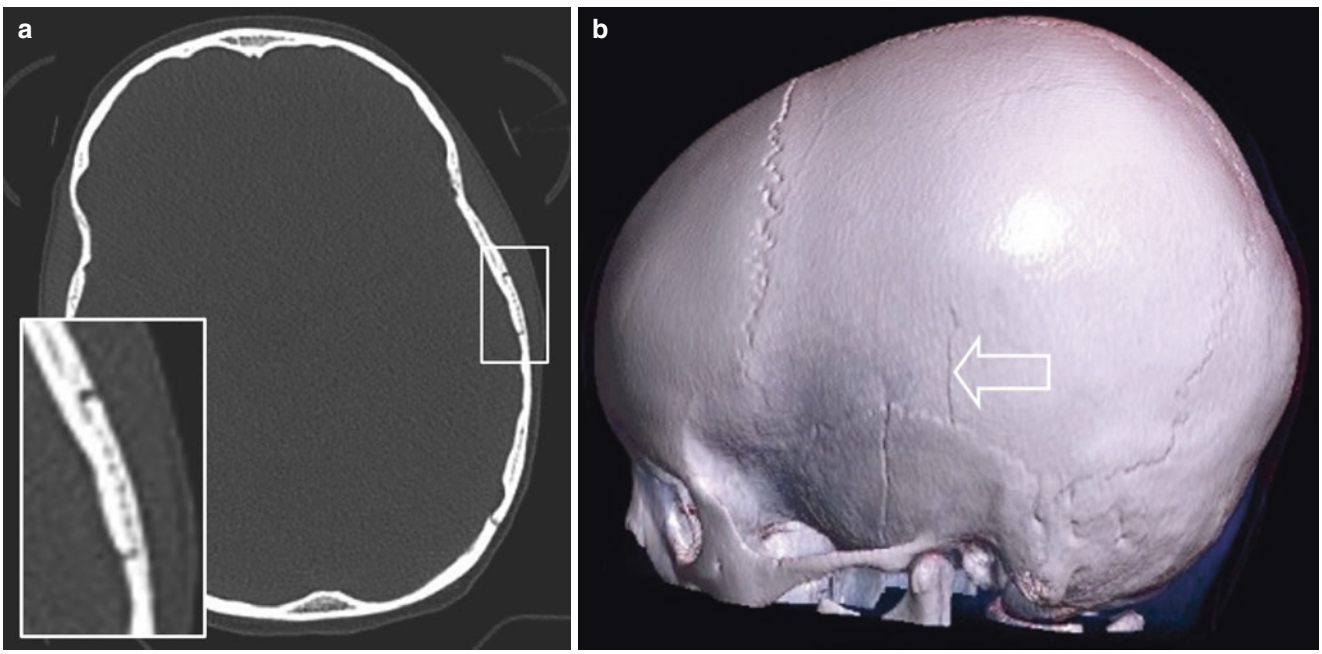


Fig. 13.30 Five-year-old child who was brought to the emergency department after a fall down a flight of stairs, on physical examination anisocoria was seen. (**a**) Axial CT shows a fracture of the inner and external table of the temporal bone (inset). (**b**) 3D-CT shows a linear fracture of the temporal bone (arrow)

tained the injuries by falling from the cart (58%), followed by toppling over the cart (26%). Injuries resulting from a fall out of the cart occurred in all age groups, toppling over of the cart was mostly seen in children <1-year old. Smith et al. concluded that accidents with shopping carts may lead to serious and potentially life-threatening injuries, although there were no cases of serious (intra)cranial injury—even in spite of a fall on a solid (often concrete) surface. No intracranial haemorrhages were found.

Parry et al. evaluated retrospectively 282 hospitalizations of children up to 15 years of age over a period of 10 years as the result of shopping cart-related injuries [141]. Of the hospitalized children, 92% was less than 5 years old, 65% was less than 2 years old. Ninety percent of the injuries resulted from a fall out of the cart. Eighty-four percent of the injuries were seen to the head or face. The physicians considered 22% of the injuries to be serious. None of the children died from the fall.

Jensen et al. described an extremely rare fatal case of asphyxiation of a 19-month-old girl who, while playing in the backyard, got caught with her head between the bottom of the basket and the horizontal base frame [142].

Conclusions

Although the literature shows that a fall from a shopping cart may lead to life-threatening injuries, there are up to the present day no reports on children that died as a consequence of such a fall. In the (albeit very limited) literature, there are also no reports on intracranial haemorrhages. The American Academy of Pediatrics advises parents among others to place the child in a safety belt or harness at all times when in a shopping cart and to never put a child in the basket. Also, have another adult present to supervise the child during shopping.

13.3.5.11 Trampoline

Introduction

Trampolines are, besides as source of fun and pleasure, increasingly a source of trauma. In the United States, an annual increase of 3.85% (95% confidence interval [CI]: 0.51–7.30) between 2008 and 2017 in trampoline-related fractures was seen [143]. The incidence increased from 35.3 per 100,000 person-years in 2008 to 53.0 per 100,000 person-years in 2017.

Most children who sustain injuries during trampoline jumping are older than the children that belong to the most vulnerable group for non-accidental injuries. However, there is an overlap between the youngest group of children with trampoline-related injuries (<5 years old) and the oldest victims with severe non-accidental injuries (2–5 years).

Although many injuries occur in a household situation there is also an increase in trampoline park injuries. Using data of the NEISS from 2010 to 2014 Kasmire showed that trampoline park injuries more often led to lower extremity injuries, serious open injuries, and spinal cord injuries leading to more hospital admissions [144]. Although technically not a trampoline, inflatable bouncers lead to similar trauma mechanisms and injuries [145–147].

Data from Literature

Woodward et al. report on 114 children with injuries due to trampoline accidents [148]. The average age was 8 years. The youngest children ran the highest risk for injuries. In 55% of children injuries of the extremities were found (Fig. 13.31). Head and neck injuries were seen in 37% of children. Seventy-five percent underwent radiography, 23% was hospitalized and 17% had to have an operation. Meyerber et al. compared children with trampoline-related injuries (N = 107) to other children with injuries, due to other (non-trampoline related) circumstances (N = 999) [149]. There was an equal distribution in fracture rate between the two groups: 34 in the trampoline group (31.8%) versus 309 in the control group (31.0%). However, surgery was required more often in the trampoline group (3.7% versus 1.8%—not significant). Also, treatment in the operating room under gen-

Fig. 13.31 Nearly 7-year-old child who has fallen from the trampoline at the day-care centre. A radiograph of the right wrist shows a torus fracture (see inset)

eral anaesthesia was more often required in the trampoline group (6.5% versus 4.7%).

Chalmers et al. evaluated 2098 hospitalizations and two deaths related to the use of trampolines over a period of 10 years [150]. Eighty percent of children fell from the trampoline on the ground. Fractures were the most prevalent injuries (68%). The arms were the most commonly involved body part. The study did not show that serious head and neck injuries were seen on a regular basis.

Larson and Davis reported on 217 children and adults that had sustained trampoline injuries [151]. Their ages ranged from 18 months to 45 years of age (average age 10 years). Forty-three percent was between 5 and 9 years old. The following injuries were found: fractures (39%), sprains and strains (25%), lacerations (21%), and contusions (16%). The patients had sustained injuries to: elbow or lower arm (26%), head and neck (21%), ankle or foot (18%), knee or leg (15%), trunk or back (9%), shoulder or arm (6%) and wrist or hand (4%). None of the patients sustained lasting neurological damage.

McDermott et al. described retrospectively 88 children (33 boys and 55 girls, average age 8.6 years) who had presented over a period of 6 months at their local hospital with a fracture due to a trampoline accident [152]. Most fractures were located in the upper extremities (69%). Thirty-six children (41%) had to have a surgical intervention; the others could be treated conservatively. In 40% of cases, playing on the trampoline was supervised by a parent/carer.

The earlier-mentioned data were more or less confirmed by Hume et al. [153]. In the 114 cases they examined (95%, age < 20 years) sprains and strains (40%) were the most frequently seen injuries. The legs were the most common place of injury.

Smith carried out a retrospective analysis of approximately 249,000 children ≤18 years old who had been treated between 1990 and 1995 for a trampoline accident [154]. Well over 70% of injuries were found in the extremities. Smith found several age-specific injury patterns:

- There is an inversely proportional relation between the age of the child and the relative frequency of injuries to the upper extremities, fractures, and dislocations.
- There is a proportional relation between the age of the child and the occurrence of skin lesions (haematomas, contusions) and injuries to the lower extremities.
- There is an inversely proportional relation between the age of the child and face, head and neck injuries, and lacerations.

Hospitalizations were indicated in 3.3% of children. Main reasons for hospitalization were fractures and dislocations (83%). Children who could go home after their visit to the emergency department had sustained predominantly skin lesions (53%), fractures and dislocations (30%), and lacerations (14%). Smith concludes that trampoline injuries are an important cause of (lethal) injuries.

Smith and Shield carried out a prospective study into trampoline accidents in 214 children from 1 to16 years of age (average age: 9.4 years) [155]. Most injuries were found in the lower extremities (36%), followed by the upper extremities (31.8%), the head (14.5%), the trunk (9.8%), and the neck (7.9%). Most frequently seen were skin lesions (haematomas and contusions) (51.9%), followed by fractures (34.6%) and lacerations (11.7%). Fractures of the extremities were most often seen in the upper extremities. Skin lesions were seen predominantly in the lower extremities. Lacerations were most prevalent to the head and the face, especially in children <6 years of age. This was also the group with the largest number of skin lesions. Other studies show a wide diversity of injuries related to trampoline accidents ranging from head/neck trauma to nerve injuries [156–162]. Lee et al., using the South Korea's Emergency Department-based Injury In-depth Surveillance registry, performed a retrospective study into children with trampoline-related injuries between January 2011 and December 2016 [163]. During the study period, there was a significant increase in the incidence of trampoline-related injuries. The most common injury site was the lower extremity (45%) with fractures being most frequently diagnosed overall (34.3%).

Boyer et al. were the first to focus on fractures of the proximal tibia in trampoline-related injuries [164]. They described cases in which children, 2–5 years (mean age 3.9), jumped on the trampoline with another person and sustained a fracture of the proximal tibia metaphysis. This was explained by the fact that a trampoline will recoil if the larger child jumps up, if the smaller child lands both the downward force of the child as well as the upward force of the trampoline will impact the child leading to a fracture. Later on, other authors have also reported this specific finding which has come to be known as a 'trampoline fracture' (Fig. 13.32a–c) [165–168]. Jääskelä et al. performed a retrospective study of children with proximal tibia fractures as a result of trampoline trauma in the period 2006 to 2017 [169]. They found, based on population data from Statistics Finland an incidence of 9.5 per 100,000 children (2006 to 2009) increasing to 22.0 per 100,000 (2014 to 2017) in the region of Oulu Arc and Oulu. Based on their findings the authors conclude that '*Probably many of these specific injuries could have been prevented by avoiding situations where more than one child jumps on the trampoline at the same time*'.

Conclusions

Most trampoline accidents do not result in life-threatening injuries, however, there is an increasing number of injuries

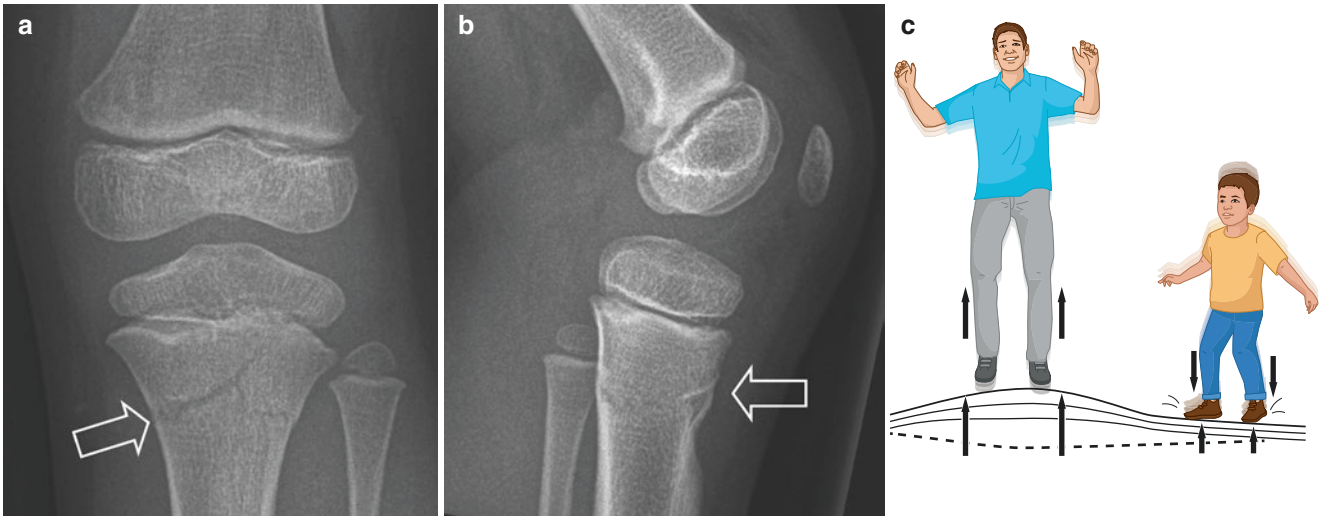


Fig. 13.32 3.5-year-old child who was on a trampoline with an adult. (**a**) AP radiograph of the knee shows a linear fracture of the proximal tibia (arrow). (**b**) Lateral radiograph shows buckling of the anterior cortex of the tibial tuberosity (arrow). (**c**) Graphic representation of a trampoline-induced tibia fracture. When the heavier person jumps, the trampoline mat recoils striking the descending smaller person (child). This causes sufficient forces to cause fracture of the proximal tibia

which can be severe. On average the age of children with trampoline injuries is higher than the age of children who have serious non-accidental injuries.

13.4 Medical Procedures

13.4.1 Introduction

In normal circumstances a child will not sustain injuries during daily care or due to medical treatments. However, as there always are exceptions to the rule we present a number of cases in which this took place.

13.4.2 Medical Procedures

Medical procedures have long been known to have negative side effects for children and can result in iatrogenic trauma [170]. In this section, we will discuss several iatrogenic causes of fractures the clinicians should be aware of.

13.4.2.1 Physical Examination

The medical literature does not report any cases in which a fracture was sustained in a physical examination. However, the authors of this book have been confronted with a 3-day-old neonate that had sustained a mid-shaft femur fracture (Fig. 13.33). According to the mother the child showed pain when she changed the diaper. Patient history and follow-up examination did not show any signs of non-accidental trauma. Post-partum there were no indications for a fracture.

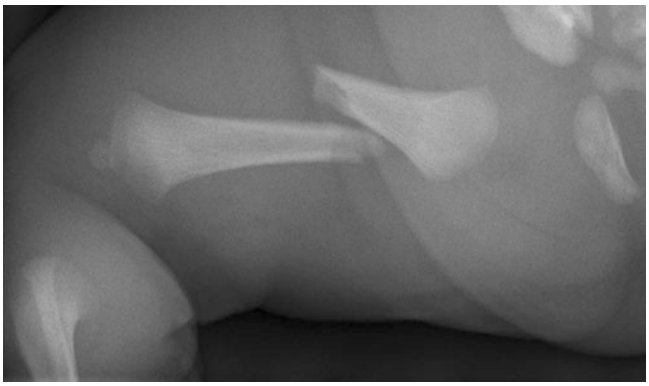


Fig. 13.33 Femur fracture in a neonate after incorrectly preformed Ortolani

On day 3, the paediatric resident performed an examination according to Ortolani. The resident wrote in the dossier that a little snap was heard and that the Ortolani was positive. After this examination the infant showed pain when the diaper was changed. A radiograph of the leg showed a mid-shaft oblique femur fracture. The successively made skeletal survey did not show any other fractures. The combined facts led to the conclusion that the femur fracture had to be the result of the examination according to Ortolani.

13.4.2.2 Lines and Punctures

In daily clinical work physicians and nurses hold children and manipulate them in order to insert lines and perform punctures. In rare cases this can lead to iatrogenic fractures in, especially the young child.

Burell et al. described the case of a 20-day-old neonate where the nursing staff manipulated the left foot, involving, involved simultaneous torque and tension/distraction of the left ankle, in order to insert an IV line [171]. During this manipulation, a 'pop' was heard and the child started to cry, a radiograph on day 30 revealed the presence of a metaphyseal corner fracture. Given the clear clinical history this was attributed to the manipulation on day 20 of life.

Harty and Kao describe two children that presented at the emergency department for bone abnormalities [172]. According to the physicians, the abnormalities had possibly been caused by 'child abuse'. In both children cortical bone defects were found in the proximal tibiae, which were thought to be healing fractures. In the end it appeared that in both children intra-osseous vascular access needles had been used. Bowley describes a case of a 2-year-old child that had sustained an iatrogenic tibia fracture after the use of an intra-osseous vascular access needle [173]. In the discussion of their case they mention two more children that had sustained an iatrogenic fracture after a bone needle had been placed. La Fleche et al. reported on a 3-month-old girl who was presented at the emergency department where i.v. access was impossible and a intra-osseous needles in both tibias were placed [174]. On outpatient check-up, three days after discharge and 14 days after presentation, bilateral healing tibial shaft fractures were diagnosed.

Another intervention in which children have to be held in a position is a lumbar puncture, in these positions the spine is flexed to obtain access to the spinal canal. Habert and Haller describe a case of a 7-week-old girl who underwent a lumbar puncture [175]. Because of suspected necrotising enterocolitis the child had AP and lateral abdominal radiographs before and after the procedure and this showed a post-intervention compression fracture of L3. In the discussion the authors mention the existence of a second similar case.

13.4.2.3 Orthopaedic Surgery in Clubfeet

There are several papers that describe iatrogenic distal tibia/fibula fractures, including metaphyseal corner fractures [176–179].

Wesely et al. were among the first to point out the complications of clubfoot treatment [178]. In a series of more than 300 cases of congenital club feet they reported four anterior compression fractures of the distal tibia and fibula metaphyses in non-surgical cases. They also mention torus fractures of the tibia and fractures of the distal fibula, for these they, however, do not provide the number of cases they encountered. Ranjan et al. reported on 196 patients (302 feet) and found 10 cases (14 feet) of nonoperative treatment, according to the Ponseti method, related distal tibial/fibula fractures [177]. Grayev et al. reported on 8 children, age range 1–4 months, who underwent casting for clubfeet [176]. In these children, who underwent surgery by five different orthopaedic surgeons in three centres, fractures of the distal tibia and/or fibula were seen on radiography. In one child the skeletal survey showed multiple rib fractures, diagnosed as resulting from non-accidental trauma. In four children metaphyseal lesions mimicking metaphyseal corner fractures were found. Volz et al. reported on four children who following nonoperative treatment with a cast developed distal tibial fractures [179]. Although this is not directly related to the intervention, but a result of disuse osteopenia, it is worthwhile considering this aetiology when confronted with such a case.

13.4.2.4 Physiotherapy

A number of articles refer to fractures sustained during physiotherapy. There can only be confusion with non-accidental fractures when the physiotherapy takes place outside the hospital and/or by the parents [180, 181].

Pickett et al. describe an ex-premature infant (pregnancy 33 weeks; birth weight 2077 gram) in whom multiple defects to both legs were found at age 4 weeks: extensive periosteal reactions around the knees combined with 'bucket-handle' fractures of both proximal tibiae [182]. Diaphyseal periosteal new bone formation and metaphyseal fragmentation of both tibiae were present. Diametaphyseal periosteal new bone of the distal end of the left femur was present. The proximal medial femoral metaphyses had corner fractures. The osseous lesions appeared to be limited to joints receiving physical therapy for contractures.

Helfer et al. describe four children of <1-year old (three ex-premature babies and one term infant) who, in their opinion, sustained serious bone damage due to passive exercises [180]. In the three ex-premature infants, the parents/carers executed the exercises advised by the hospital. In the fourth child the exercises were started on the initiative of the babysitter. Due to the results of the radiological examination, the physicians suspected the injuries in these children to be inflicted. Helfer et al. draw attention to the risk of this type of exercise for infants. It appeared that in all cases the parents executed the exercises far more strenuously than was intended. The context, as described by Helfer et al., does not always completely exclude non-accidental trauma. In one case, the father abused alcohol while in charge of the child. Only in one case the authors state specifically that after extensive investigation non-accidental circumstances were excluded. In a letter, as a reaction to this article London et al. emphasize the danger of including potentially maltreating families in (what is more or less) medical treatment such as passive exercises or physiotherapy in already vulnerable children [181].

Simonian and Staheli draw attention to fractures inflicted around the knee joint in passive exercising for contractures around the knee joint [183]. They reported two children, who sustained two separate peri-articular fractures from overzealous manipulation for knee contracture. The male neonate had no underlying disorder and sustained asynchronous ipsi-

lateral distal femoral and proximal tibial fractures and the 11-year-old boy who was diagnosed with amyoplasia sustained bilateral proximal tibial fractures.

Della Grotto et al. report on an 11-day-old neonate, who was admitted to the hospital on day 1 of life [184]. On day 11, while still in hospital, swelling of the right leg was noted and on ultrasonography a fracture of the right femoral shaft was diagnosed. Additional radiographs showed an oblique fracture of the right femoral shaft and a metaphyseal corner fracture of the proximal tibia of the same leg. Chart review showed that physiotherapy was provided in the NICU and this was ruled the cause of the fractures.

The occurrence of rib fractures resulting from physiotherapy has also been reported. Chalumeau et al. describe five boy infants (average age 3 months) within a period of 4 years who were shown to have sustained a rib fracture after physiotherapy [185]. In a prospective study by Gorincour et al., there were six children of less than 2 years old that had sustained either lateral rib fractures or possibly had remains of rib fractures as a result of physiotherapy [186]. The authors stated that in all children non-accidental trauma could be ruled out based on plausible grounds. Chanelière et al. described two children with lateral rib fractures after they had received physiotherapy for bronchiolitis [187].

Osteopathic manipulative treatment is increasingly used in the Western world in this treatment direct force, using an activating force to move tissue through range-of-motion barriers, and/or indirect force, disengaging dysfunctional body parts away from restrictive barriers is applied to the patient. Hayes and Bezilla performed a retrospective analysis in 346 children, under the age of 19 years, who underwent osteopathic manipulative treatment in Pennsylvania and Virginia, USA [188]. In their study the authors found no iatrogenic injuries. However, there is a Dutch case report of a rare fatality after 'cranio-sacral manipulation' in a 3-month-old girl [189]. The therapist positioned the child on her side and bent the spine, with the chin touching the chest, this was repeated and the child developed a loud breathing which was explained as deep sleep. After approximately 10 minutes the child was returned on her back and at that time she was pale and unresponsive. CPR was started and the child was transported to a paediatric hospital, although she was still alive at arrival she suffered dramatic hypoxic brain damage and 12 h after admission she died.

Although strictly speaking not physiotherapy, several case reports can be found describing fractures after traditional baby massage. The first report concerned two young infants, aged respectively 17 days and 1 month old who suffered respectively a femur and clavicle fracture [190]. The authors describe that the parents heard the fracture of the femur during the massage session. Siddiqui et al. described 3 children, 2–3 weeks of age, who suffered a mid-shaft femur fracture after forceful oil massage by caring grandmothers [191].

13.5 Sports

13.5.1 Introduction

Over the last few decennia, the number of children who are engaged in sports has increased. In the last years the physical, sexual and verbal maltreatment of young sporters, especially in elite sports, has gained more attention [192]. In most cases, this does not lead to physical injuries and is in older children and therefore outside the scope of this book. The incidence of sports-related injuries in children of school age has been estimated to be 3–11%, in which the majority of sports injuries (such as distortions and contusions) is not serious and will heal quickly. Serious sports injuries most commonly are overuse injuries, fractures, and ruptures and occur in all sports [193–206]. In most cases the reported trauma is in keeping with the injuries found, however, in rare cases questions can arise.

13.5.2 Data from Literature

13.5.2.1 Acute Fractures

Fractures sustained while playing sports are of the same nature and severity as 'normal' accidental fractures, such as Salter–Harris fractures and shaft fractures, since the biomechanics are often the same. Possibly Salter–Harris fractures of the distal femur and fractures of the patella are more frequently seen in sports-related trauma.

Fractures sustained while playing sports are seldom suspect for non-accidental injuries, due to an adequate clinical history and age (Fig. 13.34a, b). Moreover, often the trauma has been witnessed by a number of people. Questions can however arise if a reported trauma is considered to be insufficient to have caused the diagnosed fracture, e.g. a wrist fracture in young soccer keeper who pertinently denied having fallen on his hand. We have experienced such a case where the child said the fracture must have occurred while stopping a ball (Fig. 13.35). Initially, this raised questions but a literature study revealed that this indeed is a known trauma mechanism and that it has been reported before [207, 208].

13.5.2.2 Chronic Osseous Injuries (Stress Injuries)

The radiological image of stress injuries may suggest non-accidental injury, especially when the clinical history does not immediately point in the direction of overuse. One will find stress fractures and chronic avulsion fractures that are accompanied by ample callus formation, sclerosis, and sometimes bone resorption.

Stress fractures are often seen in the feet (metatarsals 2 and 3), tibia, and fibula (Figs. 13.36 and 13.37); chronic avulsion fractures are seen in epiphysis, e.g. the wrist (Fig. 13.38a, b), and apophyses, e.g. in the pelvis at the level where the muscles are attached to the anterior inferior and superior iliac spinous process and the ischial tuberosity.

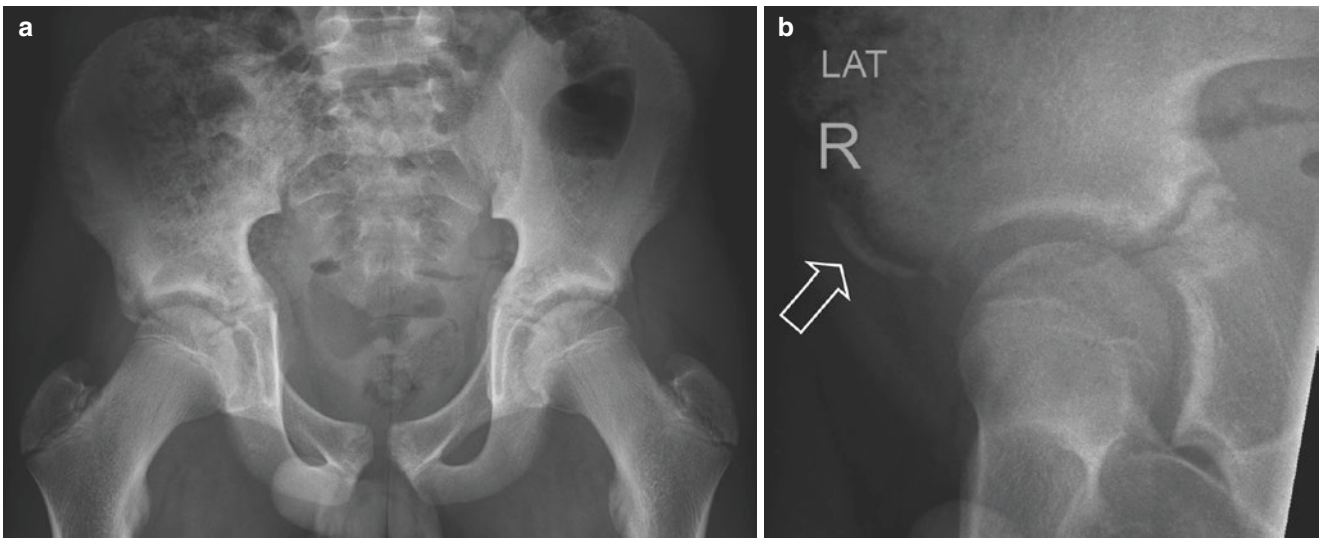


Fig. 13.34 Fifteen-year-old soccer player with acute pain in the hip. (**a**) Radiograph of the pelvis shows a cortical irregularity at the level of the anterior inferior iliac spine (AIIS). (**b**) Axial hip shows an avulsion fracture of the AIIS (arrow)

Fig. 13.35 AP radiograph of the right wrist of a 6-year-old child who stopped a soccer ball with his hand. There is a transverse fracture of the distal radius and a torus fracture of the distal ulna (arrow)

Fig. 13.36 Multiple fracture lines, cortical widening, and sclerosis in the tibia shaft of a young kick-boxer, corresponding to stress fractures (see insert). After plaster immobilization the abnormalities disappeared

Fig. 13.37 Stress fracture of metatarsal 4 (March fracture). Extensive callus formation (see inset). Situation after stapled talo-calcaneal arthrodesis (arrow)

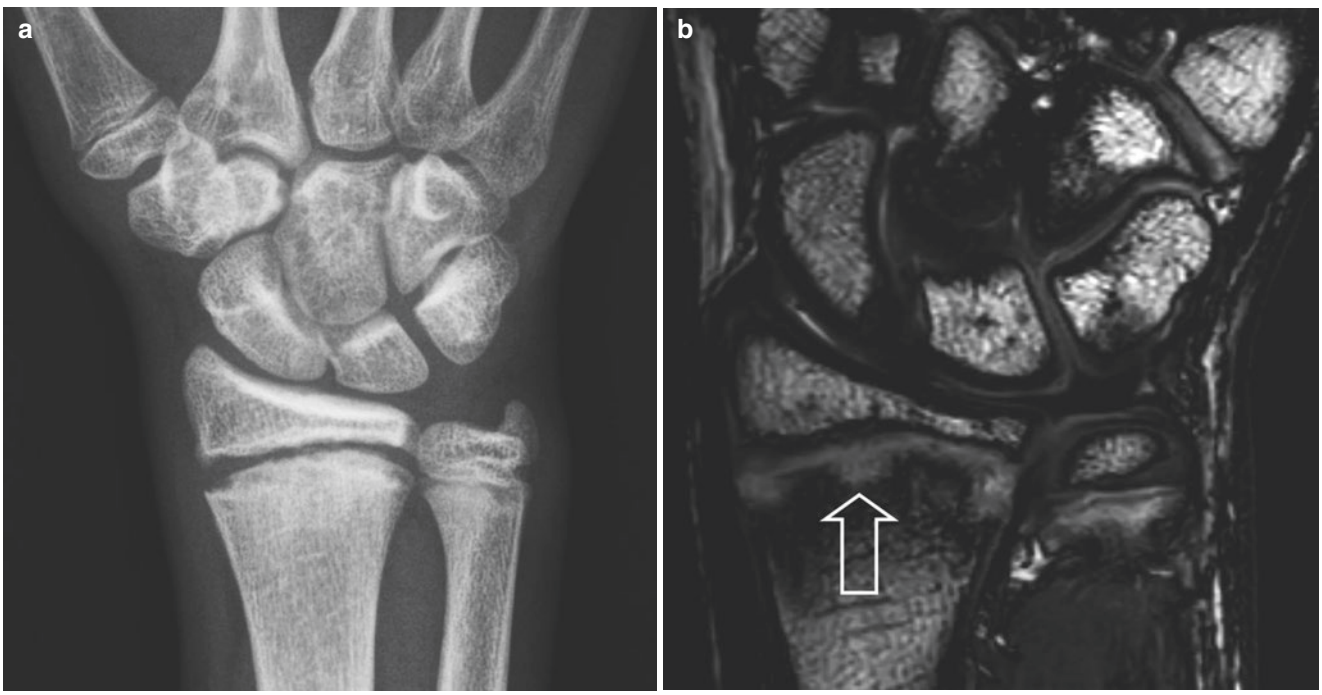


Fig. 13.38 Fifteen-year-old high-level competitive gymnast with wrist pain. (**a**) Radiograph shows widening of the physis of the distal radius and slight irregularity of the metaphysis. (**b**) T2-weighted MRI shows periphyseal bone marrow oedema (arrow)

References

1. Jaarsma AS (2007) Botbreuken bij pasgeborenen [Fractures in neonates]. Patient Care 34:9–12
2. Morris S, Cassidy N, Stephens M, McCormack D, McManus F (2002) Birth-associated femoral fractures: incidence and outcome. J Pediatr Orthop 22:27–30
3. Simonson C, Barlow P, Dehennin N, Sphel M, Toppet V, Murillo D, Rozenberg S (2007) Neonatal complications of vacuum-assisted delivery. Obstet Gynecol 109:626–633
4. Joseph PR, Rosenfeld W (1990) Clavicular fractures in neonates. Am J Dis Child 144:165–167
5. Rubin A (1964) Birth injuries: incidence, mechanisms and end results. Obstet Gynecol 23:218–221
6. Camus M, Lefebvre G, Veron P, Darbois Y (1985) Traumatismes obstétricaux du nouveau-né. Enquête rétrospective à propos de 20409 naissances [Obstetrical injuries of the newborn infant. Retrospective study apropos of 20,409 births]. J Gynecol Obstet Biol Reprod (Paris) 14:1033–1043
7. Bhat BV, Kumar A, Oumachigui A (1994) Bone injuries during delivery. Indian J Pediatr 61:401–405
8. Choi HA, Lee YK, Ko SY, Shin SM (2017) Neonatal clavicle fracture in cesarean delivery: incidence and risk factors. J Matern Fetal Neonatal Med 30:1689–1692
9. Groenendaal F, Hukkelhoven C (2007) Botbreuken bij voldragen pasgeborenen [Fractures in full-term neonates]. Ned Tijdschr Geneeskd 151:424
10. Vitner D, Hiersch L, Ashwal E, Nassie D, Yogev Y, Aviram A (2019) Outcomes of vacuum-assisted vaginal deliveries of mothers with gestational diabetes mellitus. J Matern Fetal Neonatal Med 32:3595–3599
11. Krispin E, Aviram A, Salman L, Chen R, Wiznitzer A, Gabbay-Benziv R (2017) Cup detachment during vacuum-assisted vaginal delivery and birth outcome. Arch Gynecol Obstet 296:877–883
12. Ahn ES, Jung MS, Lee YK, Ko SY, Shin SM, Hahn MH (2015) Neonatal clavicular fracture: recent 10 year study. Pediatr Int 57:60–63
13. Loeser JD, Kilburn HL, Jolley T (1976) Management of depressed skull fracture in the newborn. J Neurosurg 44:62–64
14. Biber MP (1976) Iatrogenic skull fracture depression by use of a head clamp. JAMA 235:414–415
15. van Rijn RR, Bilo RA, Robben SG (2009) Birth-related mid-posterior rib fractures in neonates: a report of three cases (and a possible fourth case) and a review of the literature. Pediatr Radiol 39:30–34
16. Wen Q, Muraca GM, Ting J, Coad S, Lim KI, Lisonkova S (2018) Temporal trends in severe maternal and neonatal trauma during childbirth: a population-based observational study. BMJ Open 8:e020578
17. Mane PP, Challawar NS, Shah H (2016) Late presented case of distal humerus epiphyseal separation in a newborn. BMJ Case Rep 2016
18. El-Adl WA, Elgohary HS, Elshennawy MM (2014) Epiphyseal separation of the proximal humerus after birth trauma. Eur J Orthop Surg Traumatol Orthop Traumatol 24:863–867
19. Franco A, Chaturvedi A (2018) Neonatal distal femoral physeal injury secondary to mechanical trauma of birth: a case report. Clin Imaging 51:65–67
20. Gigante C, Kini SG, Origo C, Volpin A (2017) Transphyseal separation of the distal humerus in newborns. Chin J Traumatol = Zhonghua chuang shang za zhi 20:183–186
21. Jacobsen S, Hansson G, Nathorst-Westfelt J (2009) Traumatic separation of the distal epiphysis of the humerus sustained at birth. J Bone Joint Surg Br 91:797–802
22. Malik S, Khopkar SR, Korday CS, Jadhav SS, Bhaskar AR (2015) Transphyseal injury of distal humerus: a commonly missed diagnosis in neonates. J Clin Diagn Res 9:Sd01-02
23. Patil MN, Palled E (2015) Epihyseal separation of lower end humerus in a neonate-diagnostic and management difficulty. J Orthop Case Rep 5:7–9
24. Tharakan SJ, Lee RJ, White AM, Lawrence JT (2016) Distal humeral epiphyseal separation in a newborn. Orthopedics 39:e764-767
25. Varghese J, Teng M, Huang M, Balsam D (2017) Birth injuries to growth plates: a sheep in wolves' clothing. J Clin Ultrasound 45:511–514
26. Kay M, Simpkins C, Shipman P, Whitewood C (2017) Diagnosing neonatal transphyseal fractures of the distal humerus. J Med Imaging Radiat Oncol 61:494–499
27. Fette A, Mayr J (2012) Slipped distal humerus epiphysis in tiny infants easily detected and followed-up by ultrasound. Ultraschall Med. (Stuttgart, Germany : 1980) 33:e361–e363
28. Supakul N, Hicks RA, Caltoum CB, Karmazyn B (2015) Distal humeral epiphyseal separation in young children: an often-missed fracture-radiographic signs and ultrasound confirmatory diagnosis. AJR Am J Roentgenol 204:W192-198
29. Pfeifer CM, Henry MK, Caré MM, Christian CW, Servaes S, Milla SS, Strouse PJ (2021) Debunking fringe beliefs in child abuse imaging: AJR expert panel narrative review. AJR Am J Roentgenol
30. Warner C, Maguire S, Trefan L, Miller A, Weinman J, Fadell M (2017) A study of radiological features of healing in long bone fractures among infants less than a year. Skelet Radiol 46:333–341
31. Cooper C, Dennison EM, Leufkens HG, Bishop N, van Staa TP (2004) Epidemiology of childhood fractures in Britain: a study using the general practice research database. J Bone Miner Res 19:1976–1981
32. Spady DW, Saunders DL, Schopflocher DP, Svenson LW (2004) Patterns of injury in children: a population-based approach. Pediatrics 113:522–529
33. Bilo RAC, Voorhoeve HWA, Koot JM (2008) Kind in ontwikkeling—een handreiking bij de observatie van jonge kinderen. Elsevier, Tijdstroom
34. Brouwers-de Jong EA, Burgmeijer RJF, Laurent de Angulo MS (1996) Ontwikkelingsonderzoek op het consultatiebureau—handboek bij het vernieuwde Van Wiechenonderzoek
35. Wheeler DS, Shope TR (1997) Depressed skull fracture in a 7-month-old who fell from bed. Pediatrics 100:1033–1034
36. Hajiaghamemar M, Lan IS, Christian CW, Coats B, Margulies SS (2019) Infant skull fracture risk for low height falls. Int J Legal Med 133:847–862
37. Haney SB, Starling SP, Heisler KW, Okwara L (2010) Characteristics of falls and risk of injury in children younger than 2 years. Pediatr Emerg Care 26:914–918
38. Kravitz H, Driessen G, Gomberg R, Korach A (1969) Accidental falls from elevated surfaces in infants from birth to one year of age. Pediatrics 44(Suppl):869–876
39. Gallagher SS, Finison K, Guyer B, Goodenough S (1984) The incidence of injuries among 87,000 Massachusetts children and adolescents: results of the 1980-81 statewide childhood injury prevention program surveillance system. Am J Public Health 74:1340–1347
40. Rivara FP, Alexander B, Johnston B, Soderberg R (1993) Population-based study of fall injuries in children and adolescents resulting in hospitalization or death. Pediatrics 92:61–63
41. 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

42. Helfer RE, Slovis TL, Black M (1977) Injuries resulting when small children fall out of bed. Pediatrics 60:533–535
43. Chadwick DL, Chin S, Salerno C, Landsverk J, Kitchen L (1991) Deaths from falls in children: how far is fatal? J Trauma 31:1353–1355
44. Nimityongskul P, Anderson LD (1987) The likelihood of injuries when children fall out of bed. J Pediatr Orthop 7:184–186
45. Lyons TJ, Oates RK (1993) Falling out of bed: a relatively benign occurrence. Pediatrics 92:125–127
46. Tarantino CA, Dowd MD, Murdock TC (1999) Short vertical falls in infants. Pediatr Emerg Care 15:5–8
47. Bechtel K, Stoessel K, Leventhal JM, Ogle E, Teague B, Lavietes S, Banyas B, Allen K, Dziura J, Duncan C (2004) Characteristics that distinguish accidental from abusive injury in hospitalized young children with head trauma. Pediatrics 114:165–168
48. Lueder GT (2005) Retinal hemorrhages in accidental and nonaccidental injury. Pediatrics 115:192; author reply 192
49. Warrington SA, Wright CM (2001) Accidents and resulting injuries in premobile infants: data from the ALSPAC study. Arch Dis Child 85:104–107
50. Hennrikus WL, Shaw BA, Gerardi JA (2003) Injuries when children reportedly fall from a bed or couch. Clin Orthop Relat Res:148–151
51. Mulligan CS, Adams S, Tzioumi D, Brown J (2017) Injury from falls in infants under one year. J Paediatr Child Health 53:754–760
52. Johnson K, Fischer T, Chapman S, Wilson B (2005) Accidental head injuries in children under 5 years of age. Clin Radiol 60:464–468
53. Ibrahim NG, Wood J, Margulies SS, Christian CW (2012) Influence of age and fall type on head injuries in infants and toddlers. Int J Dev Neurosci 30:201–206
54. Monson SA, Henry E, Lambert DK, Schmutz N, Christensen RD (2008) In-hospital falls of newborn infants: data from a multihospital health care system. Pediatrics 122:e277-280
55. Ruddick C, Platt MW, Lazaro C (2010) Head trauma outcomes of verifiable falls in newborn babies. Arch Dis Child Fetal Neonatal Ed 95:f144–f145
56. Lallier M, Bouchard S, St-Vil D, Dupont J, Tucci M (1999) Falls from heights among children: a retrospective review. J Pediatr Surg 34:1060–1063
57. Vish NL, Powell EC, Wiltsek D, Sheehan KM (2005) Pediatric window falls: not just a problem for children in high rises. Inj Prev 11:300–303
58. Mayer L, Meuli M, Lips U, Frey B (2006) The silent epidemic of falls from buildings: analysis of risk factors. Pediatr Surg Int 22:743–748
59. Musemeche CA, Barthel M, Cosentino C, Reynolds M (1991) Pediatric falls from heights. J Trauma 31:1347–1349
60. Randazzo C, Stolz U, Hodges NL, McKenzie LB (2009) Pediatric tree house-related injuries treated in emergency departments in the United States: 1990-2006. Acad Emerg Med Off J Soc Acad Emerg Med 16:235–242
61. 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 14:201–207
62. Kim KA, Wang MY, Griffith PM, Summers S, Levy ML (2000) Analysis of pediatric head injury from falls. Neurosurg Focus 8:e3
63. Murray JA, Chen D, Velmahos GC, Alo K, Belzberg H, Asensio JA, Demetriades D, Berne TV (2000) Pediatric falls: is height a predictor of injury and outcome? Am Surg 66:863–865
64. Wang MY, Kim KA, Griffith PM, Summers S, McComb JG, Levy ML, Mahour GH (2001) Injuries from falls in the pediatric population: an analysis of 729 cases. J Pediatr Surg 36:1528–1534
65. Shields BJ, Burkett E, Smith GA (2011) Epidemiology of balcony fall-related injuries, United States, 1990-2006. Am J Emerg Med 29:174–180
66. Babu RA, Arimappamagan A, Pruthi N, Bhat DI, Arvinda HR, Devi BI, Somanna S (2017) Pediatric thoracolumbar spinal injuries: the etiology and clinical spectrum of an uncommon entity in childhood. Neurol India 65:546–550
67. van Rijn RR, Affourtit MJ, Karst WA, Kamphuis M, de Bock LC, van de Putte E (2019) Implementation of the Dutch expertise Centre for child abuse: descriptive data from the first 4 years. BMJ Open 9:e031008
68. Macgregor DM (2000) Injuries associated with falls from beds. Inj Prev 6:291–292
69. Mack KA, Gilchrist J, Ballesteros MF (2007) Bunk bed-related injuries sustained by young children treated in emergency departments in the United States, 2001-2004, National Electronic Injury Surveillance System—all Injury Program. Inj Prev 13:137–140
70. D'Souza AL, Smith GA, McKenzie LB (2008) Bunk bed-related injuries among children and adolescents treated in emergency departments in the United States, 1990-2005. Pediatrics 121:e1696-1702
71. McFaull SR, Fréchette M, Skinner R (2012) Emergency department surveillance of injuries associated with bunk beds: the Canadian Hospitals Injury Reporting and Prevention Program (CHIRPP), 1990-2009. Chronic Dis Inj Can 33:38–46
72. Sengölge M, Vincenten J (2013) Child product safety guide; potentially dangerous products. European Child Safety Alliance, EuroSafe, Birmingham, UK
73. Selbst SM, Baker MD, Shames M (1990) Bunk bed injuries. Am J Dis Child 144:721–723
74. Johnson GF (1981) Pediatric Lisfranc injury: "bunk bed" fracture. AJR Am J Roentgenol 137:1041–1044
75. Belechri M, Petridou E, Trichopoulos D (2002) Bunk versus conventional beds: a comparative assessment of fall injury risk. J Epidemiol Community Health 56:413–417
76. Rasch H (2012) Rest easy with these bunk bed safety tips. https://www.aappublications.org/content/33/7/22.6 Date accessed: 14-08-2021
77. Watson WL, Ozanne-Smith J (1993) The use of child safety restraints with nursery furniture. J Paediatr Child Health 29:228–232
78. Couper RT, Monkhouse W, Busutil M, Thompson P (1994) Stroller safety. Med J Aust 160:335–338
79. Lee AC, Fong D (1997) Epidural haematoma and stroller-associated injury. J Paediatr Child Health 33:446–447
80. Powell EC, Jovtis E, Tanz RR (2002) Incidence and description of stroller-related injuries to children. Pediatrics 110:e62
81. Fowler E, Kobe C, Roberts KJ, Collins CL, McKenzie LB (2016) Injuries associated with strollers and carriers among children in the United States, 1990 to 2010. Acad Pediatr 16:726–733
82. Arnholz D, Hymel KP, Hay TC, Jenny C (1998) Bilateral pediatric skull fractures: accident or abuse? J Trauma 45:172–174
83. Tripathi M, Tyebally A, Feng JX, Chong SL (2017) A review of stroller-related and pram-related injuries to children in Singapore. Inj Prev 23:60–63
84. Callahan CW, Sisler C (1997) Use of seating devices in infants too young to sit. Arch Pediatr Adolesc Med 151:233–235
85. Farmakakis T, Alexe DM, Nicolaidou P, Dessypris N, Petridou E (2004) Baby-bouncer-related injuries: an under-appreciated risk. Eur J Pediatr 163:42–43
86. Beaudin M, Maugans T, St-Vil D, Falcone RA Jr (2013) Inappropriate use of infant seating devices increases risks of injury. J Pediatr Surg 48:1071–1076
87. Wickham T, Abrahamson E (2002) Head injuries in infants: the risks of bouncy chairs and car seats. Arch Dis Child 86:168–169
88. The National Highway Traffic Safety Administration — U.S. Department of Transportation (2004) The status of occupant protection in America. https://one.nhtsa.gov/people/injury/

research/6thAnnualBUAReport/pages/Status.htm Date accessed: 14-08-2021
89. Parikh SN, Wilson L (2010) Hazardous use of car seats outside the car in the United States, 2003-2007. Pediatrics 126:352–357
90. Desapriya EB, Joshi P, Subzwari S, Nolan M (2008) Infant injuries from child restraint safety seat misuse at British Columbia Children's hospital. Pediatr Int 50:674–678
91. Pollack-Nelson C (2000) Fall and suffocation injuries associated with in-home use of car seats and baby carriers. Pediatr Emerg Care 16:77–79
92. Greenberg RA, Bolte RG, Schunk JE (2009) Infant carrier-related falls: an unrecognized danger. Pediatr Emerg Care 25:66–68
93. U.S. Consumer Product Safety Commission (CPSC) (2017) CPSC approves new federal standard for infant bouncers. https://www.cpsc.gov/content/cpsc-approves-new-federal-standard-for-infant-bouncers Date accessed: 14-08-2021
94. Liaw P, Moon RY, Han A, Colvin JD (2019) Infant deaths in sitting devices. Pediatrics 144
95. Batra EK, Midgett JD, Moon RY (2015) Hazards associated with sitting and carrying devices for children two years and younger. J Pediatr 167:183–187
96. Bamber AR, Pryce J, Ashworth MT, Sebire NJ (2014) Sudden unexpected infant deaths associated with car seats. Forensic Sci Med Pathol 10:187–192
97. Mayr JM, Seebacher U, Schimpl G, Fiala F (1999) Highchair accidents. Acta Paediatrica (Oslo, Norway : 1992) 88:319–322
98. Powell EC, Jovtis E, Tanz RR (2002) Incidence and description of high chair-related injuries to children. Ambul Pediatr 2:276–278
99. Kurinsky RM, Rochette LM, Smith GA (2014) Pediatric injuries associated with high chairs and chairs in the United States, 2003-2010. Clin Pediatr (Phila) 53:372–379
100. Atkinson N, van Rijn RR, Starling SP (2018) Childhood falls with occipital impacts. Pediatr Emerg Care 34:837–841
101. Souheil M, Audrey F, Anny G, Sebastien RJ, Bertrand L (2011) Fatal accidental hanging by a high-chair waist strap in a 2-year-old girl. J Forensic Sci 56:534–536
102. Government of Canada (2018) Suspended baby jumpers. https://www.canada.ca/en/health-canada/services/infant-care/suspended-baby-jumpers.html Date accessed: 14-08-2021
103. U.S. Consumer Product Safety Commission (CPSC) (2005) CPSC, Kids II Inc. Announce Recall of Doorway Baby Jumpers. https://www.cpsc.gov/Recalls/2005/cpsc-kids-ii-inc-announce-recall-of-doorway-baby-jumpers Date accessed: 14-08-2021
104. Tanner J (2013) Baby Einstein recall after children suffer skull fractures, bruises. Pix11com
105. Glanfield E (2015) Three-month-old girl died after being left in a baby bouncer for several hours while her mother slept on the sofa. Mailonline
106. Claydon SM (1996) Fatal extradural hemorrhage following a fall from a baby bouncer. Pediatr Emerg Care 12:432–434
107. Abbott AL, Bartlett DJ (2001) Infant motor development and equipment use in the home. Child Care Health Dev 27:295–306
108. Grant P, Mata MB, Tidwell M (2001) Femur fracture in infants: a possible accidental etiology. Pediatrics 108:1009–1011
109. Moineau G, Plint A (2005) Tibial fractures possibly linked to use of a baby stationary activity center. Pediatr Emerg Care 21:181–183
110. U.S. consumer product safety commission (CPSC) (2009) Evenflo® Recalls Children's Activity Centers Due to Fall Hazard. https://www.cpsc.gov/Recalls/2009/evenflo-recalls-childrens-activity-centers-due-to-fall-hazard Date accessed: 14-08-2021
111. Chagas PSC, Fonseca ST, Santos TRT, Souza TR, Megale L, Silva PL, Mancini MC (2020) Effects of baby walker use on the development of gait by typically developing toddlers. Gait Posture 76:231–237
112. Yaghini O, Goodarzi M, Khoei S, Shirani M (2020) Effect of baby Walker use on developmental status based on ages and stages questionnaire score (ASQ). Iran J Child Neurol 14:105–111
113. Burrows P, Griffiths P (2002) Do baby walkers delay onset of walking in young children? Br J Community Nurs 7:581–586
114. Trinkoff A, Parks PL (1993) Prevention strategies for infant walker-related injuries. Public Health Rep. (Washington, DC : 1974) 108:784–788
115. American Academy of Pediatrics (AAP) (2001) Injuries associated with infant walkers. Pediatrics 108:790–792
116. Sims A, Chounthirath T, Yang J, Hodges NL, Smith GA (2018) Infant Walker-related injuries in the United States. Pediatrics 142
117. Fazen LE 3rd, Felizberto PI (1982) Baby walker injuries. Pediatrics 70:106–109
118. Kavanagh CA, Banco L (1982) The infant walker. A previously unrecognized health hazard. Am J Dis Child 136:205–206
119. Wellman S, Paulson JA (1984) Baby walker-related injuries. Clin Pediatr (Phila) 23:98–99
120. Stoffman JM, Bass MJ, Fox AM (1984) Head injuries related to the use of baby walkers. Can Med Assoc J 131:573–575
121. Rieder MJ, Schwartz C, Newman J (1986) Patterns of walker use and walker injury. Pediatrics 78:488–493
122. Partington MD, Swanson JA, Meyer FB (1991) Head injury and the use of baby walkers: a continuing problem. Ann Emerg Med 20:652–654
123. Al-Nouri L, Al-Isami S (2006) Baby walker injuries. Ann Trop Paediatr 26:67–71
124. Coats TJ, Allen M (1991) Baby walker related injuries—a continuing problem. Arch Emerg Med 8:52–55
125. Chiaviello CT, Christoph RA, Bond GR (1994) Infant walker-related injuries: a prospective study of severity and incidence. Pediatrics 93:974–976
126. Mayr JM, Gaisl M, Purtscher K, Noeres H, Schimpl G, Fasching G (1994) Baby walkers—an underestimated hazard for our children? Eur J Pediatr 153:531–534
127. Petridou E, Simou E, Skondras C, Pistevos G, Lagos P, Papoutsakis G (1996) Hazards of baby walkers in a European context. Inj Prev 2:118–120
128. Smith GA, Bowman MJ, Luria JW, Shields BJ (1997) Babywalker-related injuries continue despite warning labels and public education. Pediatrics 100:E1
129. Blazewick DH, Chounthirath T, Hodges NL, Collins CL, Smith GA (2018) Stair-related injuries treated in United States emergency departments. Am J Emerg Med 36:608–614
130. Zielinski AE, Rochette LM, Smith GA (2012) Stair-related injuries to young children treated in US emergency departments, 1999-2008. Pediatrics 129:721–727
131. Pierce MC, Bertocci GE, Janosky JE, Aguel F, Deemer E, Moreland M, Boal DK, Garcia S, Herr S, Zuckerbraun N, Vogeley E (2005) Femur fractures resulting from stair falls among children: an injury plausibility model. Pediatrics 115:1712–1722
132. Joffe M, Ludwig S (1988) Stairway injuries in children. Pediatrics 82:457–461
133. Chiaviello CT, Christoph RA, Bond GR (1994) Stairway-related injuries in children. Pediatrics 94:679–681
134. Docherty E, Hassan A, Burke D (2010) Things that go bump ... bump ... bump: an analysis of injuries from falling down stairs in children based at Sheffield Children's Hospital. Emerg Med J 27:207–208
135. Cheng YW, Fletcher EN, Roberts KJ, McKenzie LB (2014) Baby gate-related injuries among children in the United States, 1990-2010. Acad Pediatr 14:256–261
136. Committee on Injury Violence and Poison Prevention; American Academy of Pediatrics (2006) Shopping-cart-related injuries to children. Pediatrics 118:825–827

137. Martin KJ, Chounthirath T, Xiang H, Smith GA (2014) Pediatric shopping-cart-related injuries treated in US emergency departments, 1990-2011. Clin Pediatr (Phila) 53:277–285
138. Wright JW, Griffin R, MacLennan PA, Rue LW 3rd, McGwin G Jr (2008) The incidence of shopping cart-related injuries in the United States, 2002–2006. Accid Anal Prev 40:1253–1256
139. Smith GA, Dietrich AM, Garcia CT, Shields BJ (1995) Epidemiology of shopping cart-related injuries to children. An analysis of national data for 1990 to 1992. Arch Pediatr Adolesc Med 149:1207–1210
140. Smith GA, Dietrich AM, Garcia CT, Shields BJ (1996) Injuries to children related to shopping carts. Pediatrics 97:161–165
141. Parry ML, Morrison LG, Chalmers DJ, Wright CS (2002) Shopping trolley-related injuries to children in New Zealand, 1988-97. J Paediatr Child Health 38:51–54
142. Jensen L, Charlwood C, Byard RW (2008) Shopping cart injuries, entrapment, and childhood fatality. J Forensic Sci 53:1178–1180
143. Hadley-Miller N, Carry PM, Brazell CJ, Holmes KS, Georgopoulos G (2020) Trends in trampoline fractures: 2008-2017. Pediatrics 145:e20190889
144. Kasmire KE, Rogers SC, Sturm JJ (2016) Trampoline Park and home trampoline injuries. Pediatrics 138
145. Avoian T, Choi PD, Manjra N, Weiss J (2008) Inflatable bouncer-related fractures in children. J Pediatr Orthop 28:656–659
146. Ferro V, D'Alfonso Y, Vanacore N, Rossi R, Deidda A, Giglioni E, Reale A, Raucci U (2016) Inflatable bouncer-related injuries to children: increasing phenomenon in pediatric emergency department, 2002-2013. Eur J Pediatr 175:499–507
147. Kirketerp-Møller K, Balslev N, Lohmann M (1996) Ulykker med hoppepuder hos 0-19-årige i Danmark i 1993 [accidents caused by inflatable bouncers in 0-19 year-olds in Denmark in 1993]. Ugeskr Laeger 158:2251–2253
148. Woodward GA, Furnival R, Schunk JE (1992) Trampolines revisited: a review of 114 pediatric recreational trampoline injuries. Pediatrics 89:849–854
149. Meyerber M, Fraisse B, Dhalluin T, Ryckewaert A, Violas P (2019) Trampoline injuries compared with other child activities. Archives de pediatrie : organe officiel de la Societe francaise de pediatrie 26:282–284
150. Chalmers DJ, Hume PA, Wilson BD (1994) Trampolines in New Zealand: a decade of injuries. Br J Sports Med 28:234–238
151. Larson BJ, Davis JW (1995) Trampoline-related injuries. J Bone Joint Surg Am 77:1174–1178
152. McDermott C, Quinlan JF, Kelly IP (2006) Trampoline injuries in children. J Bone Joint Surg Br 88:796–798
153. Hume PA, Chalmers DJ, Wilson BD (1996) Trampoline injury in New Zealand: emergency care. Br J Sports Med 30:327–330
154. Smith GA (1998) Injuries to children in the United States related to trampolines, 1990-1995: a national epidemic. Pediatrics 101:406–412
155. Smith GA, Shields BJ (1998) Trampoline-related injuries to children. Arch Pediatr Adolesc Med 152:694–699
156. Brown PG, Lee M (2000) Trampoline injuries of the cervical spine. Pediatr Neurosurg 32:170–175
157. Cools MJ, Carneiro KA (2018) Facial nerve palsy following mild mastoid trauma on trampoline. Am J Emerg Med 36:1522.e1521–1522.e1523
158. Maclin MM 2nd, Novak CB, Mackinnon SE (2004) Ulnar nerve injury associated with trampoline injuries. South Med J 97:720–723
159. Menting T, Staal E (2012) Een meisje met een pijnlijke heup na spagaat [a girl with a painful hip after a splits]. Ned Tijdschr Geneeskd 156:A3320
160. Ono H, Sase T, Takasuna H, Tanaka Y (2019) Playground equipment-related head injuries requiring hospitalization in children. Pediatr Int 61:293–297
161. Ranneries TN, Balle J, Homøe P (2018) Larynxtraumer hos børn efter fald på trampolin [Laryngeal traumas in children caused by trampoline accidents]. Ugeskrift for laeger 180
162. Korhonen L, Salokorpi N, Suo-Palosaari M, Pesälä J, Serlo W, Sinikumpu JJ (2018) Severe trampoline injuries: incidence and risk factors in children and adolescents. Eur J Pediatr Surg 28:529–533
163. Lee G, Kim DK, Park JW, Kwak YH, Jung JY (2020) Trampoline-related injuries in children: a nationwide cross-sectional study in South Korea. Clin Exp Emerg Med 7:190–196
164. Boyer RS, Jaffe RB, Nixon GW, Condon VR (1986) Trampoline fracture of the proximal tibia in children. AJR Am J Roentgenol 146:83–85
165. Bruyeer E, Geusens E, Catry F, Vanstraelen L, Vanhoenacker F (2012) 'Trampoline fracture' of the proximal tibia in children: report of 3 cases and review of literature. JBR-BTR 95:10–12
166. Kakel R (2012) Trampoline fracture of the proximal tibial metaphysis in children may not progress into valgus: a report of seven cases and a brief review. Orthop Traumatol Surg Res 98:446–449
167. Stranzinger E, Leidolt L, Eich G, Klimek PM (2014) The anterior tilt angle of the proximal tibia epiphyseal plate: a significant radiological finding in young children with trampoline fractures. Eur J Radiol 83:1433–1436
168. Choi ES, Hong JH, Sim JA (2018) Distinct features of trampoline-related orthopedic injuries in children aged under 6 years. Injury 49:443–446
169. Jääskelä M, Kuivalainen L, Victorzon S, Serlo W, Lempainen L, Sinikumpu JJ (2020) Trampoline-related proximal tibia impaction fractures in children: a population-based approach to epidemiology and radiographic findings between 2006 and 2017. J Child Orthop 14:125–131
170. Spackman TJ (1973) Pediatric trauma: medical abuse of infants. Radiol Clin N Am 11:633–656
171. Burrell T, Opfer E, Berglund L, Lowe LH, Anderst J (2015) A witnessed case of a classic metaphyseal fracture caused during IV line placement in a child: insight into mechanism of injury. J Forensic Legal Med 35:51–53
172. Harty MP, Kao SC (2002) Intraosseous vascular access defect: fracture mimic in the skeletal survey for child abuse. Pediatr Radiol 32:188–190
173. Bowley DM, Loveland J, Pitcher GJ (2003) Tibial fracture as a complication of intraosseous infusion during pediatric resuscitation. J Trauma 55:786–787
174. La Fleche FR, Slepin MJ, Vargas J, Milzman DP (1989) Iatrogenic bilateral tibial fractures after intraosseous infusion attempts in a 3-month-old infant. Ann Emerg Med 18:1099–1101
175. Habert J, Haller JO (2000) Iatrogenic vertebral body compression fracture in a premature infant caused by extreme flexion during positioning for a lumbar puncture. Pediatr Radiol 30:410–411
176. Grayev AM, Boal DK, Wallach DM, Segal LS (2001) Metaphyseal fractures mimicking abuse during treatment for clubfoot. Pediatr Radiol 31:559–563
177. Ranjan R, Sud A, Adhikary D, Sinha A, Chand S (2019) Incidence and risk factors for iatrogenic distal tibia/fibula fracture during Ponseti technique of clubfoot treatment. J Pediatr Orthop B 28:572–578
178. Weseley MS, Barenfeld PA, Barrett N (1972) Complications of the treatment of clubfoot. Clin Orthop Relat Res 84:93–96
179. Volz R, Paulsen M, Morcuende J (2009) Distal tibia/fibula fractures following clubfoot casting—report of four cases. Iowa Orthop J 29:117–120
180. Helfer RE, Scheurer SL, Alexander R, Reed J, Slovis TL (1984) Trauma to the bones of small infants from passive exercise: a factor in the etiology of child abuse. J Pediatr 104:47–50
181. Landon R, Noronha PA, Levy HB (1984) Bone trauma caused by passive exercise. J Pediatr 105:172–173

182. Pickett WJ 3rd, Johnson JF, Enzenauer RW (1982) Case report 192. Neonatal fractures mimicking abuse secondary to physical therapy. Skelet Radiol 8:85–86
183. Simonian PT, Staheli LT (1995) Periarticular fractures after manipulation for knee contractures in children. J Pediatr Orthop 15:288–291
184. Della Grotta LM, Marine MB, Harris TL, Karmazyn B (2019) Classic metaphyseal lesion acquired during physical therapy. Clin Imaging 54:100–102
185. Chalumeau M, Foix-L'Helias L, Scheinmann P, Zuani P, Gendrel D, Ducou-le-Pointe H (2002) Rib fractures after chest physiotherapy for bronchiolitis or pneumonia in infants. Pediatr Radiol 32:644–647
186. Gorincour G, Dubus JC, Petit P, Bourliere-Najean B, Devred P (2004) Rib periosteal reaction: did you think about chest physical therapy? Arch Dis Child 89:1078–1079
187. Chanelière C, Moreux N, Pracros JP, Bellon G, Reix P (2006) Fractures costales au cours des bronchiolites aiguës virales: à propos de 2 cas [rib fractures after chest physiotherapy: a report of 2 cases]. Arch Pediatr 13:1410–1412
188. Hayes NM, Bezilla TA (2006) Incidence of iatrogenesis associated with osteopathic manipulative treatment of pediatric patients. J Am Osteopath Assoc 106:605–608
189. Holla M, Ijland MM, van der Vliet AM, Edwards M, Verlaat CW (2009) Overleden zuigeling na 'craniosacrale' manipulatie van hals en wervelkolom [death of an infant following 'craniosacral' manipulation of the neck and spine]. Ned Tijdschr Geneeskd 153:828–831
190. Mboutol-Mandavo C, N'Dour O, Ouedraogo SF, Missengue-Bosseba R, Ndiaye D, Ngom G (2016) Fractures du nouveau-né et du nourrisson secondaires au massage traditionnel [Newborn and infant fractures secondary to traditional massage]. Arch Pediatr 23:963–965
191. Siddiqui SA, Singh MV, Shrivastava A, Maurya M, Gaur VK, Kumar D (2020) Femoral shaft fracture following oil massage in neonates: a single-centre experience. Tropical doctor:49475520940480
192. Ohlert J, Vertommen T, Rulofs B, Rau T, Allroggen M (2020) Elite athletes' experiences of interpersonal violence in organized sport in Germany, The Netherlands, and Belgium. Eur J Sport Sci:1–10
193. Adams AL, Schiff MA (2006) Childhood soccer injuries treated in U.S. emergency departments. Acad Emerg Med Off J Soc Acad Emerg Med 13:571–574
194. Hostetler SG, Xiang H, Smith GA (2004) Characteristics of ice hockey-related injuries treated in US emergency departments, 2001-2002. Pediatrics 114:e661-666
195. Kerr ZY, Collins CL, Comstock RD (2011) Epidemiology of bowling-related injuries presenting to US emergency departments, 1990-2008. Clin Pediatr (Phila) 50:738–746
196. Nation AD, Nelson NG, Yard EE, Comstock RD, McKenzie LB (2011) Football-related injuries among 6- to 17-year-olds treated in US emergency departments, 1990-2007. Clin Pediatr (Phila) 50:200–207
197. Pollard KA, Gottesman BL, Rochette LM, Smith GA (2013) Swimming injuries treated in US EDs: 1990 to 2008. Am J Emerg Med 31:803–809
198. Pollard KA, Shields BJ, Smith GA (2011) Pediatric volleyball-related injuries treated in US emergency departments, 1990-2009. Clin Pediatr (Phila) 50:844–852
199. Randazzo C, Nelson NG, McKenzie LB (2010) Basketball-related injuries in school-aged children and adolescents in 1997-2007. Pediatrics 126:727–733
200. Reid JP, Nelson NG, Roberts KJ, McKenzie LB (2012) Track-related injuries in children and adolescents treated in US emergency departments from 1991 through 2008. Phys Sportsmed 40:56–63
201. Shields BJ, Smith GA (2006) Cheerleading-related injuries to children 5 to 18 years of age: United States, 1990-2002. Pediatrics 117:122–129
202. Singh S, Smith GA, Fields SK, McKenzie LB (2008) Gymnastics-related injuries to children treated in emergency departments in the United States, 1990-2005. Pediatrics 121:e954-960
203. Gram MCD, Clarsen B, Bø K (2021) Injuries and illnesses among competitive Norwegian rhythmic gymnasts during preseason: a prospective cohort study of prevalence, incidence and risk factors. Br J Sports Med 55:231–236
204. Kox LS, Kraan RBJ, Mazzoli V, Mens MA, Kerkhoffs G, Nederveen AJ, Maas M (2020) It's a thin line: development and validation of Dixon MRI-based semi-quantitative assessment of stress-related bone marrow edema in the wrists of young gymnasts and non-gymnasts. Eur Radiol 30:1534–1543
205. Kraan RBJ, Kox LS, Oostra RJ, Kuijer P, Maas M (2020) The distal radial physis: exploring normal anatomy on MRI enables interpretation of stress related changes in young gymnasts. Eur J Sport Sci 20:1197–1205
206. Rui P, Ashman JJ, Akinseye A (2019) Emergency department visits for injuries sustained during sports and recreational activities by patients aged 5-24 years, 2010-2016. Natl Health Stat Rep:1–15
207. Boyd KT, Brownson P, Hunter JB (2001) Distal radial fractures in young goalkeepers: a case for an appropriately sized soccer ball. Br J Sports Med 35:409–411
208. Macgregor DM (2003) Don't save the ball! Br J Sports Med 37:351–353
209. Basha A, Amarin Z, Abu-Hassan F (2013) Birth-associated long-bone fractures. Int J Gynaecol Obstet 123:127–130
210. Suleiman FA, Almaaitah AA, Aqrabawi HE (2016) Upper limb birth trauma in a Jordanian population: a prospective study at King Hussein Medical Centre, Amman, Jordan. JPMA J Pak Med Assoc 66:1422–1426
211. von Heideken J, Thiblin I, Högberg U (2020) The epidemiology of infant shaft fractures of femur or humerus by incidence, birth, accidents, and other causes. BMC Musculoskelet Disord 21:840

Normal Variants, Congenital, and Acquired Disorders

14

Rick R. van Rijn, Jopje M. Ruskamp, Nicole L van Woerden,
Rutger A. J. Nievelstein, Simon G. F. Robben,
and Rob A. C. Bilo

Contents

R. R. van Rijn
Department of Radiology and Nuclear Medicine, Amsterdam UMC, University of Amsterdam, Amsterdam, The Netherlands

Department of Forensic Medicine, Netherlands Forensic Institute, The Hague, The Netherlands

J. M. Ruskamp
Department of Paediatrics, University Medical Center Utrecht, Utrecht, The Netherlands

N. L. van Woerden
Department of Forensic Medicine, Netherlands Forensic Institute, The Hague, The Netherlands

R. A. J. Nievelstein
Department of Radiology, University Medical Center Utrecht, Utrecht, The Netherlands

S. G. F. Robben (✉)
Department of Radiology, Maastricht University Medical Center, Maastricht, The Netherlands
e-mail: s.robben@mumc.nl

R. A. C. Bilo
Veilig Thuis Rotterdam Rijnmond (Center for the Reporting of Child Abuse, Domestic Violence and Elder Abuse), Rotterdam, The Netherlands

R. A. C. Bilo et al. (eds.), *Forensic Aspects of Paediatric Fractures*, https://doi.org/10.1007/978-3-031-12041-1_14

14.1 Introduction

Although in the differential diagnosis of fractures in childhood one should particularly be aware of accidental trauma (Chap. 13), it is not uncommon that normal variants, congenital and/or acquired defects lead to the incorrect conclusion that these 'fractures' were sustained in non-accidental circumstances (Table 14.1) [1–5]. In this chapter, we discuss the most important differential diagnoses of which the radiological findings might be misinterpreted as fractures due to non-accidental trauma.

A suspicion may occur due to a true mimic, which is a normal variant or a disorder, simulating on imaging a fresh or a healing/healed fracture, e.g. Caffey's disease or Alagille syndrome. Suspicion can also occur if a child sustains a fracture in a (previously unknown) medical condition with an increased risk of fractures due to weakening of the bone, e.g. osteogenesis imperfecta. A fracture in such a disorder should not be considered to be a true mimic. After all, it is a real fracture and a fracture is always caused by trauma, in which the loading of the bone exceeded the maximum load-bearing capacity. Finding a fracture in a child with such a condition does not automatically indicate under which circumstances the fracture was sustained. Fractures due to weakening of bone can be considered to be a mimic of inflicted fractures in non-accidental trauma, because an adequate clinical history may be lacking.

Some disorders can be considered to be true mimics, but also show an increased risk of fractures. Menkes syndrome (OMIM #309400) and copper deficiency, for example are true mimics, because of the presence of metaphyseal spurs, suggesting metaphyseal corner fractures, and periosteal reactions, suggesting healing fractures. In both disorders, however, there is also an increased risk of fracturing, because of weakening of the bone (osteoporosis) due to disturbances in bone metabolism and for that reason may mimic inflicted fractures.

In a multidisciplinary team effort based on a combination of the patient's clinical history, laboratory tests, and radiological examination, it is usually possible to reach the correct diagnosis [6]. In this chapter, we discuss the most important differential diagnoses of which the radiological findings might be misinterpreted as fractures due to non-accidental trauma.

Table 14.1 Differential diagnosis in disease-related fractures in infancy and childhood [1–4]

Fractures	
Disorders related to collagen production	Osteogenesis imperfecta Copper deficiency Menkes syndrome Bruck syndrome
Congenital mineralization disorders	Prematurity: Metabolic bone disease of prematurity Neuromuscular disorders Vitamin D-resistant rickets (or hypophosphatemic rickets) X-linked hypophosphatemia Liver abnormalities, such as Alagille syndrome Malabsorption Familiar osteoporosis Osteopetrosis Cole Carpenter syndrome Congenital CMV infection
Acquired mineralization disorders	Vitamin D deficiency based on malnutrition: Rickets Use of diuretics, glucocorticoids, and methotrexate Intoxications, such as lead Cerebral paresis and spasticity
Other increased risk disorders	Congenital pain insensitivity disorders: Spina bifida Congenital pain insensitivity Muscular dystrophy
Periosteal reactions	
Radiological differential diagnosis in the absence of fractures	Normal variants: Such as: Physiological thickening of the long bones (femur, tibia, humerus) in neonates and infants Congenital syphilis Osteomyelitis Septic arthritis Osteoid osteoma and other tumours Leukaemia Vitamin C deficiency: Scurvy Caffey's disease: Infantile cortical hyperostosis Hurler disease: Mucopolysaccharidosis type I Sickle cell anaemia Vitamin use-related disorders Hypervitaminosis A Vitamin E therapy Prostaglandin E treatment Metastases of a neuroblastoma The use of intra-osseous vascular access needles
Normal variants (examples)	Subperiosteal new bone formation (shaft of femur, tibia, and humerus, usually bilateral) in normal, healthy neonates, and infants Normal metaphyseal variants Accessory growth centres Unfused growth plate of the shoulder Unfused apophysis of the fifth metatarsal Accessory skull sutures Accessory ossicles Vascular/nutrient lines

14.2 Normal Variants

When evaluating radiographs of children, there are a number of normal variants that may cause confusion, and even lead to a false accusation of parents or caregivers. When in doubt it is advised to contact an experienced paediatric radiologist and/or to use Keat's reference standard work 'Atlas Of Normal Roentgen Variants That May Simulate Disease' [7].

14.2.1 Subperiosteal New-Bone Formation

At a very young age, subperiosteal new bone formation around the shaft of the femur, tibia, and humerus may be seen in normal, healthy infants (Fig. 14.1). This newly formed bone, which may radiologically be mistaken for a healing fracture, is most prominently present in children from 1 to 4 months old. Kwon et al. studied 101 neonates and

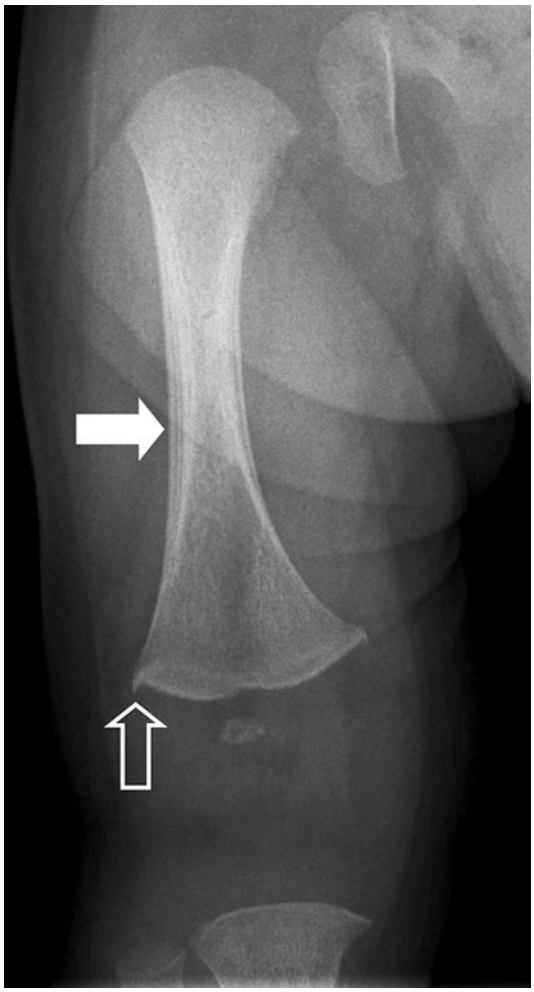


Fig. 14.1 Femur of a young infant, showing physiological subperiosteal new bone formation (arrow) and metaphyseal spur (open arrow)

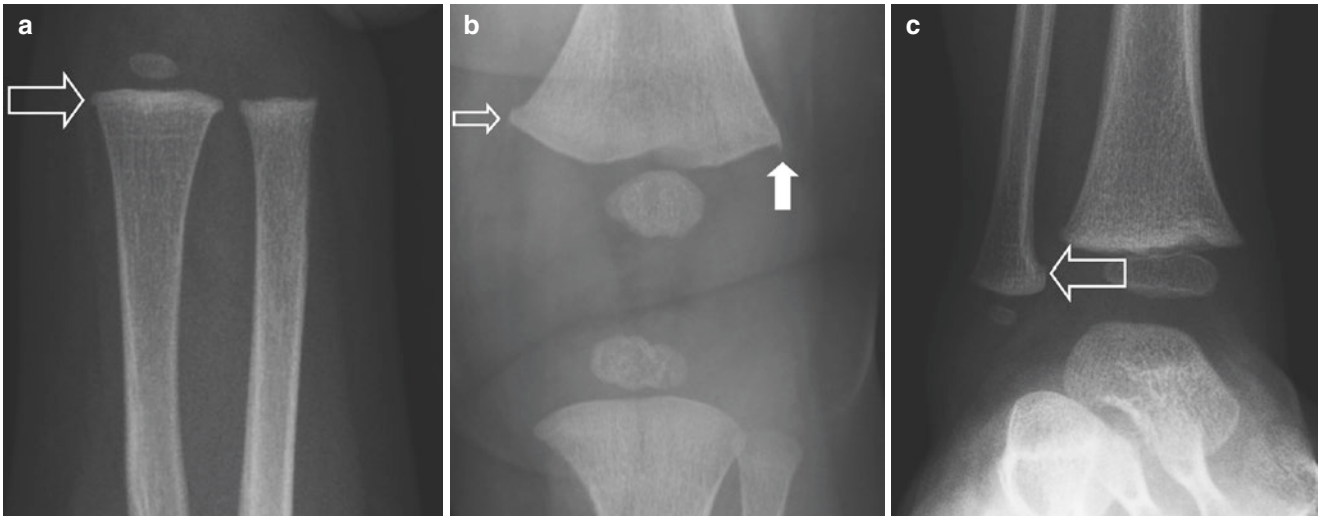


Fig. 14.2 (**a**) Physiological metaphyseal collar in the distal radius (open arrow). (**b**) Physiological metaphyseal collar (open arrow) and metaphyseal spur (arrow) in the distal femur metaphysis. (**c**) Physiological metaphyseal collar (open arrow) at the medial side of the distal fibula

infants who had died from sudden infant death syndrome (age range, 2 weeks–8 months). They found subperiosteal new bone formation in 35 infants (35%), all of whom were between 1 and 4 months of age [8]. Subperiosteal newly formed bone is usually seen bilaterally; however, it may be more prominently present unilaterally [3, 9]. In physiological, subperiosteal newly formed bone, there is no obvious uptake of isotopes on a bone scan [10].

14.2.2 Metaphyseal Variants

Normal metaphyseal variants should not be mistaken for metaphyseal corner fractures due to non-accidental trauma. This category comprises thickened edges of the metaphyses (collar, step off) exactly where the epiphyseal plate is attached (Fig. 14.2a–c) [11, 12]. This collar is usually present in the proximal tibia, proximal fibula, distal femur, distal radius and distal ulna, and is regularly seen bilaterally [13]. In young children pointed metaphyseal 'spurs' can also be found, which to the untrained eye of a radiologist may look very similar to metaphyseal corner fractures. This spur is made of cortical bone that grows under the perichondrial ring of the epiphyseal plate. Spurs may be seen in the distal femur (Figs. 14.1 and 14.2b), the lateral aspect of the distal radius, the medial aspect of the distal ulna and the metacarpals (Fig. 14.3), and metatarsals. In 25% of cases this image is seen bilaterally. Finally, the metaphysis may show medial widening, especially in the proximal tibia and humerus (Fig. 14.4).

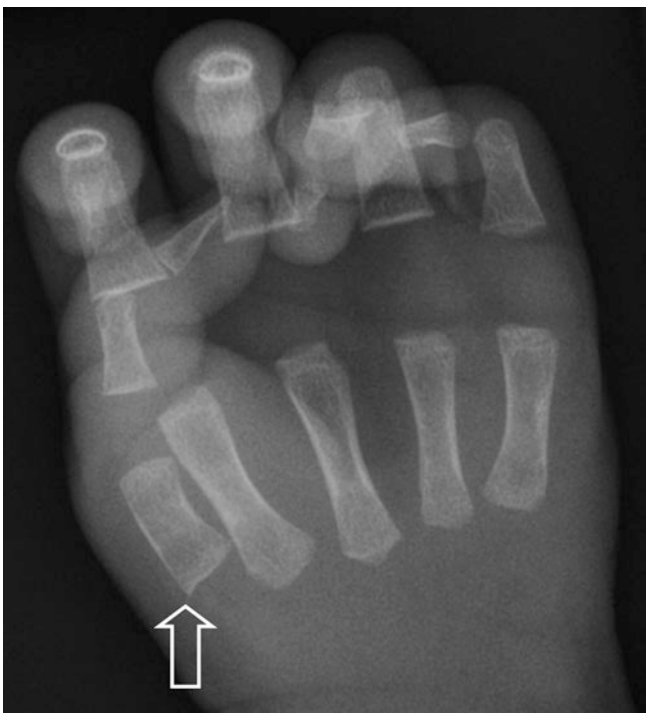


Fig. 14.3 Metaphyseal spur on the base of metacarpal 1 of the right hand (open arrow)

14.2.3 Variants of the Cortex

In 4% of children, a cortical irregularity is seen on the medial side of the proximal tibia. In 25% of these children this is present in both legs (Figs. 14.5 and 14.6a, b) [13]. This

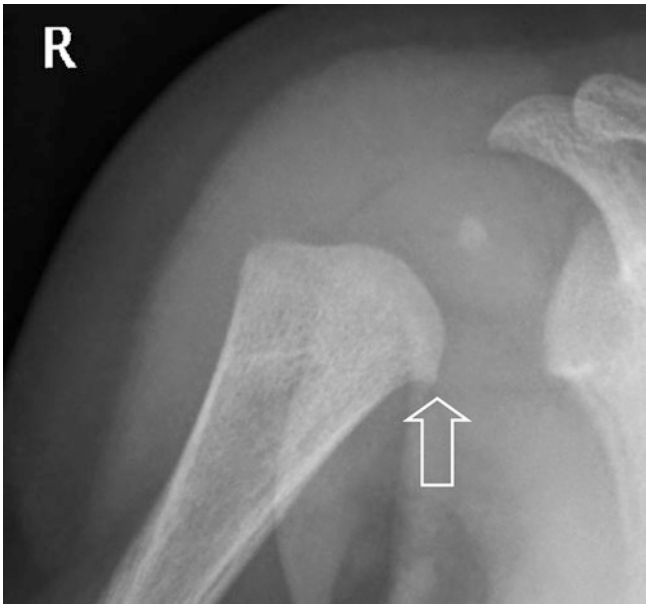
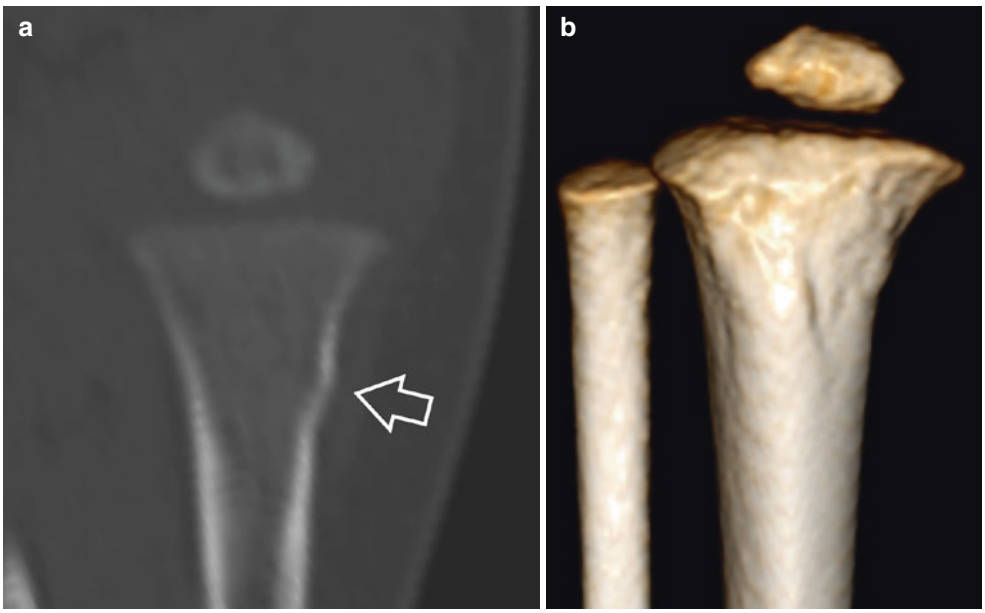


Fig. 14.4 Medial extension of the proximal metaphysis of the humerus (open arrow)

Fig. 14.5 Cortical irregularity is seen on the medial side of the proximal tibia on a conventional radiograph (arrow)

Fig. 14.6 (**a**) Cortical irregularity in another patient seen on the medial side of the proximal tibia on CT (arrow). (**b**) 3D reconstruction of the proximal tibia



irregularity may look like a healing fracture and consequently lead to an incorrect diagnosis.

14.2.4 Accessory Growth Centres and Sutures

One of the most important properties of the childhood skeleton is growth. Besides the normal growth centres, accessory growth centres may be seen (Fig. 14.7a, b) [7], which may be interpreted erroneously as fractures, and as such lead to confusion.

The presence of normal variants of sutures of the skull, may also lead to an erroneous diagnosis of a skull fracture (Figs. 14.8 and 14.9), (Sects. 5.3.4.7 and 5.5.4) [14–24]. With respect to accessory sutures the occipital bone requires specific attention. Embryonically this bone is derived from four segments: the basilar, squamous, and two condylar segments [25, 26]. The basilar segment, also known as the basiocciput, is formed from four segments. The squamous segment, also known as the supraoccipital, is formed from three segments. During embryogenesis this can lead to a wide variety of accessory sutures that both radiologists and clinicians should be aware of. Variance in the development of the occipital bone can also lead to the development of a so-called Inca bone, a larger solitary bone island at the level of the posterior fontanelle [27, 28].

Besides variants in sutures also accessory bones within the sutures, the so-called 'Wormian bones' can be found [29–31]. In a normal population, one or more Wormian bones are reported to be present in up to 50% of children [31]. With the increased use of CT, they are more likely to be reported by radiologists. Although the presence of a higher than a usual number of Wormian bones is a well-known radiographic sign of osteogenesis imperfecta, there are certainly more diseases that can also have this imaging finding (Table 14.2). This finding is therefore by no means diagnostic of osteogenesis imperfecta.

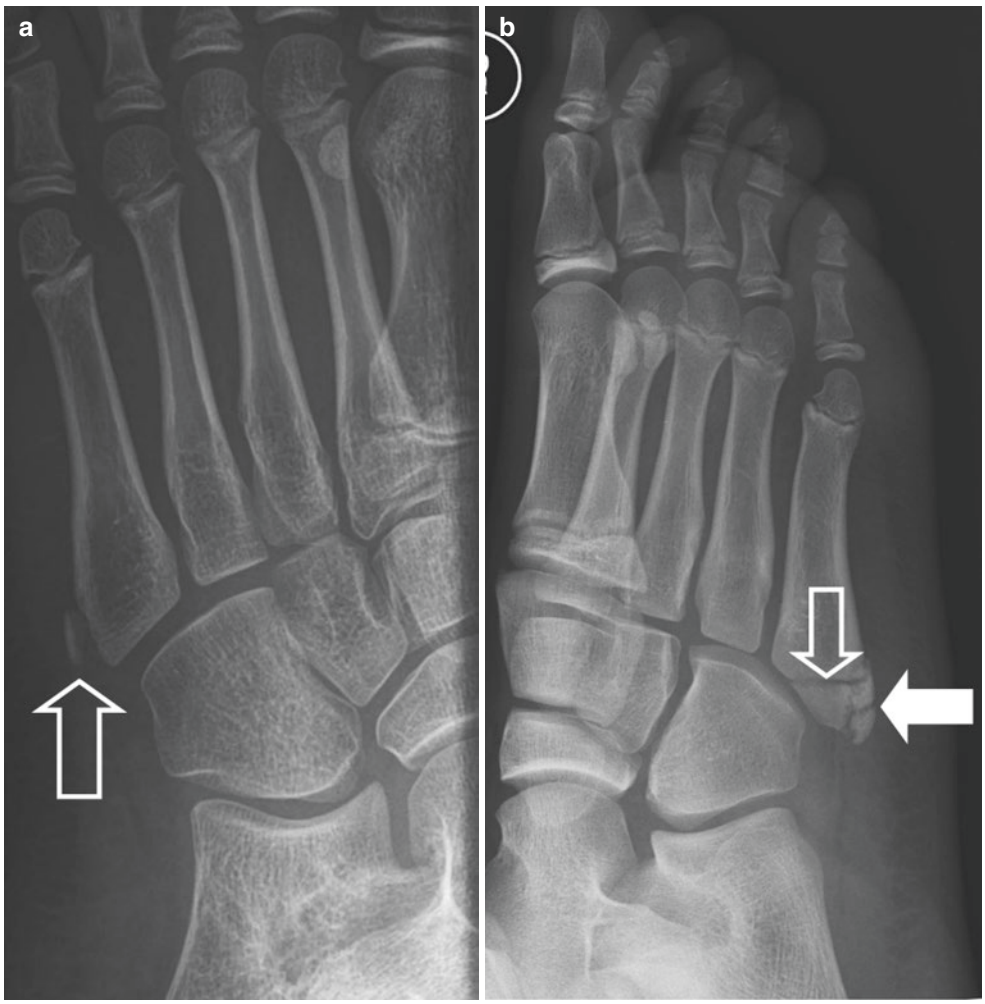


Fig. 14.7 (**a**) Accessory ossicle at the base of metatarsal 5 (open arrow). When one is not familiar with this phenomenon, it may be mistaken for an avulsion fracture. (**b**) Accessory ossicle at the base of metatarsal 5 (arrow). There is also a 'Jones fracture' visible at the base of metatarsal 5 (open arrow)

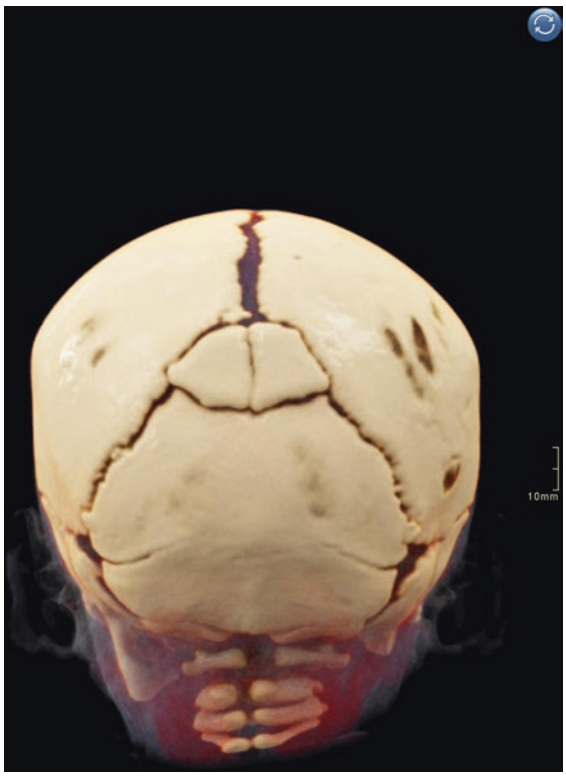


Fig. 14.8 Three-dimensional cinematic rendering showing bilateral mendosal sutures and a bifid Inca bone

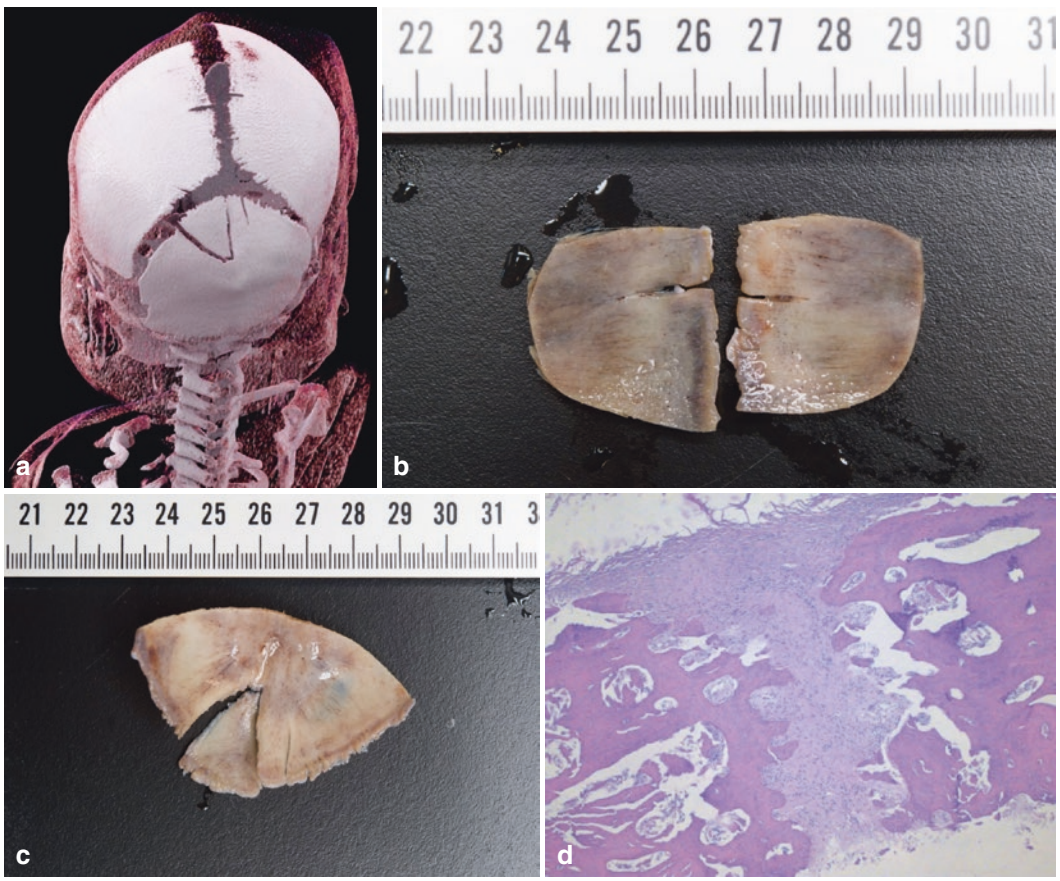


Fig. 14.9 (**a**) Post-mortem CT of an 18-day-old neonate found in a bag, 3D cinematic rendering shows an Obelian suture, i.e. a suture connecting the two parietal foramina, and two 'fracture lines' or accessory sutures in the occipital bone. The 'fracture lines' however, are in keeping with the borders between the ossification centres that form the cranial segment of the occipital bone. (**b**) Specimen photo showing the Obelian suture. (**c**) Specimen photo showing the 'fracture lines' in the occipital bone. (**d**) Histological slide clearly shows connective tissue between the bone segments proving the presence of accessory sutures

Table 14.2 Diseases that may present with 'wormian bones'

Consistently present	Inconsistently present
Cretinism	Pyknodysostosis
Metaphyseal dysplasia, type Jansen	Sclerosteosis
Menkes syndrome[a]	Hydrocephalus
Acro-osteolysis	Osteopetrosis
Prader-Willi syndrome	Down syndrome
Cleido-cranial dysostosis	Rickets[a]
	Hypophosphatasia
	Progeria

[a]These diseases should also be included in the radiological differential diagnosis of non-accidental trauma

14.3 Osteogenesis Imperfecta

14.3.1 Introduction

Osteogenesis imperfecta (OI) is a heterogeneous group of diseases, which classically demonstrates fragility of the skeletal system and susceptibility to fractures of the long bones or vertebral compressions from mild or inconsequential trauma. The spectrum of OI is extremely broad, ranging from a form that is lethal in the perinatal period to a mild form in which the diagnosis may be equivocal in an adult [32]. Structural or quantitative defects in type 1 collagen cause the full spectrum of OI (Fig. 14.10). Together with

Fig. 14.10 (**a**) Specimen, dating from approximately 1850, from the Vrolik collection of the Academic Medical Centre Amsterdam. Gerardus Vrolik (1775–1859) described this specimen, which is considered to be one of the first reported cases of OI. A very wide sagittal suture can be seen, the ribs are fragile but intact. Both tibiae show signs of healing fractures (Courtesy of R.J. Oostra, conservator of the Vrolik museum). (**b**) Lateral view of the specimen shows multiple wormian bones in the skull

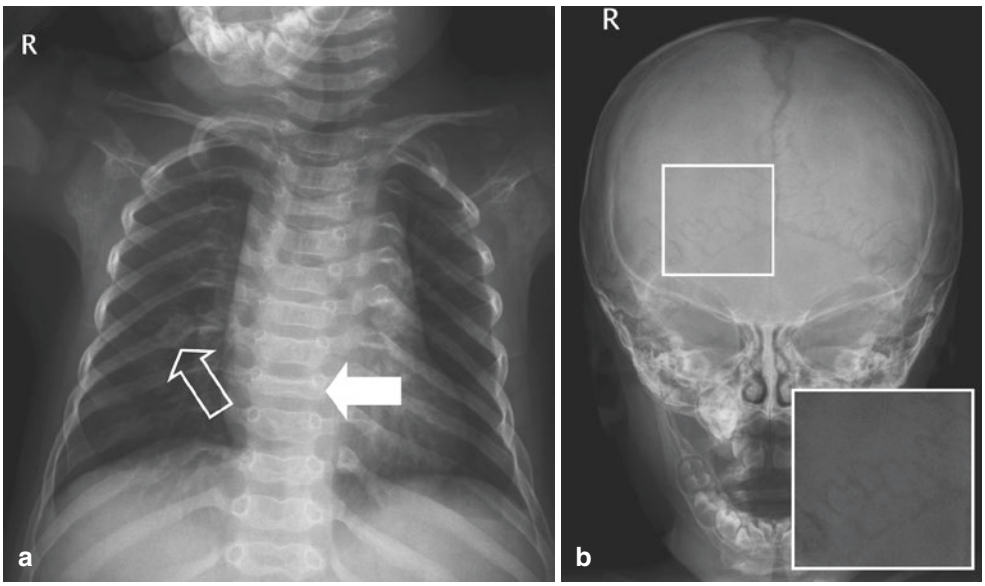


Fig. 14.11 (**a**) One-year-old boy who presented with a femur fracture after falling off the counter, in the presence of multiple witnesses. The chest radiograph shows old rib fractures (open arrows) and multiple collapsed vertebrae (arrow). (**b**) Skull view of the same patient shows multiple wormian bones (see inset). Osteogenesis imperfecta was genetically confirmed

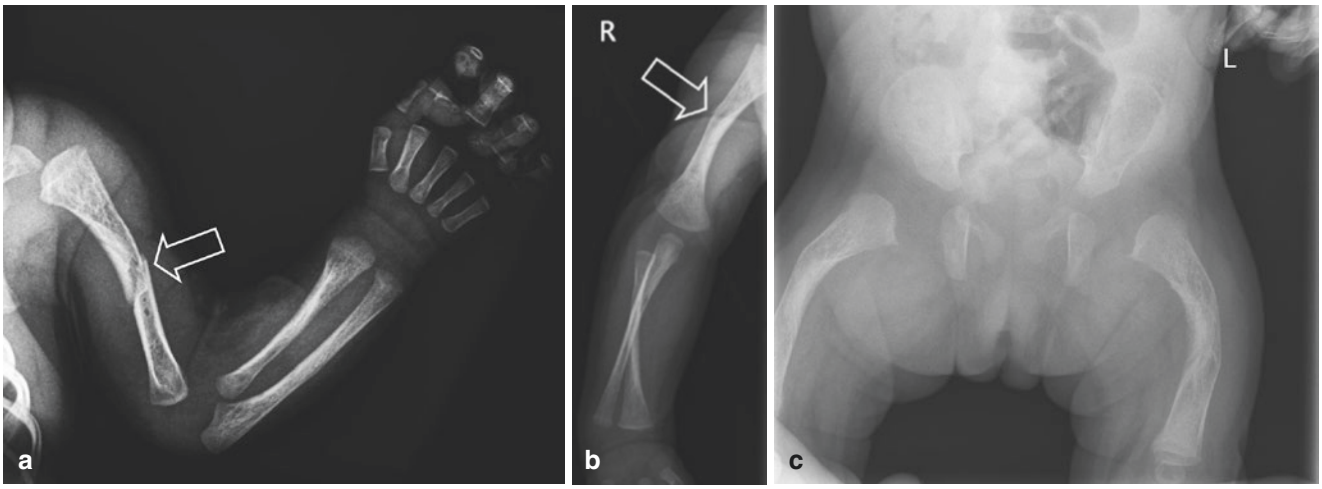


Fig. 14.12 (**a**) One-week-old girl with swollen painful left arm. Radiograph shows a fresh mid-shaft oblique humerus fracture (open arrow). An additional skeletal survey was made. (**b**) The right arm also shows a mid-shaft fracture of the humerus (open arrow), which is difficult to date. (**c**) Pelvic view shows bilateral bowing of the femurs and sclerosis, an image corresponding with healing fractures. Osteogenesis imperfecta was genetically confirmed

non-accidental trauma OI is the most common cause for the presence of multiple fractures, often at various stages of healing, without a plausible explanation (Fig. 14.11a, b). This makes it an important differential diagnosis but it must be borne in mind that OI is considerably less prevalent compared to non-accidental trauma in children [33, 34].

14.3.2 Clinical Presentation

In OI there is a defect in the synthesis of type I collagen production, predominantly (in up to 85% of cases) caused by mutations in the collagen I genes (*COL1A1* and *COL1A2*) [35–37]. Type I collagen is an important protein in the extracellular matrix of many tissues. The disease is equally distributed between boys and girls, and is often seen in other family members. It is an autosomal dominant disorder, although spontaneous mutations do occur. In the skeleton, a defect in the synthesis of type I collagen will lead to osteoporosis, which makes it possible for minimal trauma to cause multiple fractures (Fig. 14.12a–c). The protein is also present in ligaments, skin, teeth, sclera, and blood vessels. Consequently, symptoms can occur to a higher or lesser degree in all these systems. Besides the defect in the synthesis of collagen type I, two more mutations have been reported; a mutation of the *CRTAP* gene, which causes a mild to severe recessive rhizomal form of OI [38]. Furthermore, mutations have been reported in *CRTAP* together with *LEPRE1*, which leads to an autosomal recessive form of

Table 14.3 Classification of osteogenesis imperfect as proposed by van Dijk et al. [35]

Type	Gene
I	*Col1A1/2*
II-A[a]	*Col1A1/2*
II-B[c]	*CRTAP*
II-C[d]	*LEPRE-1*[b]
III	*PPIB*
IV	
V	Unknown
VI[e]	

[a] No individuals with OI due to *LEPRE1, CRTAP* or *PPIB* mutations were diagnosed with OI type IIA
[b] No *LEPRE1* mutations causing OI type IV have been reported
[c] OI type II-B with longer survival and OI type III with early death show considerable clinical and radiological overlap
[d] OI Type II-C is extremely rare and its existence is even doubted
[e] OI type VI main distinguishing feature from type V is histological

OI [39–41]. This genetic basis makes the input and expertise of clinical geneticists invaluable [42–44].

Sillence et al. provide a classification in four subtypes [45], based on the mode of inheritance, radiological presentation (including age of presentation) and scleral blueness. The incidence figures provided are based on research on Australian children. In 2004, Rauch and Glorieux published an overview in the Lancet in which they widened the Sillence classification to seven subtypes [46]. In later years more types have been described leading to a total of 20 types of OI, many of which are extremely rare and only described in a few patients. In 2010, van Dijk et al. proposed a revised classification of the Sillence criteria I, II-A, II-B, II-C, III, and IV, in which they mentioned the causative gene and the clinical picture (Table 14.3) [35].

In 85% of children with OI, fractures will heal at the same speed and in the same manner as in children without OI [47]. Children with OI types I and II (80% of all patients) usually present no diagnostic problem (Fig. 14.13).

Young children without OI may also have blue sclerae. Consequently, in children with inflicted fractures there may be the erroneous impression that they have pathological bone fragility that fits OI. The presence of aberrant teeth (dentogenesis imperfecta) may either support or confirm the suspected OI. Rib fractures are frequently seen in all types, as is bowing of the lower extremities. Metaphyseal corner fractures may also be seen in children with OI [47, 48]. Astley described metaphyseal corner fractures in seven children of a group of 41 children with OI [48]. He deems it impossible that one could erroneously suspect non-accidental trauma in these children, because of the other noticeable signs fitting OI. On the other hand, Albin et al. are convinced that the presence of metaphyseal defects is pathognomic for non-accidental trauma, and that for this reason it is possible to differentiate between osteogenesis imperfecta and non-accidental trauma [49].

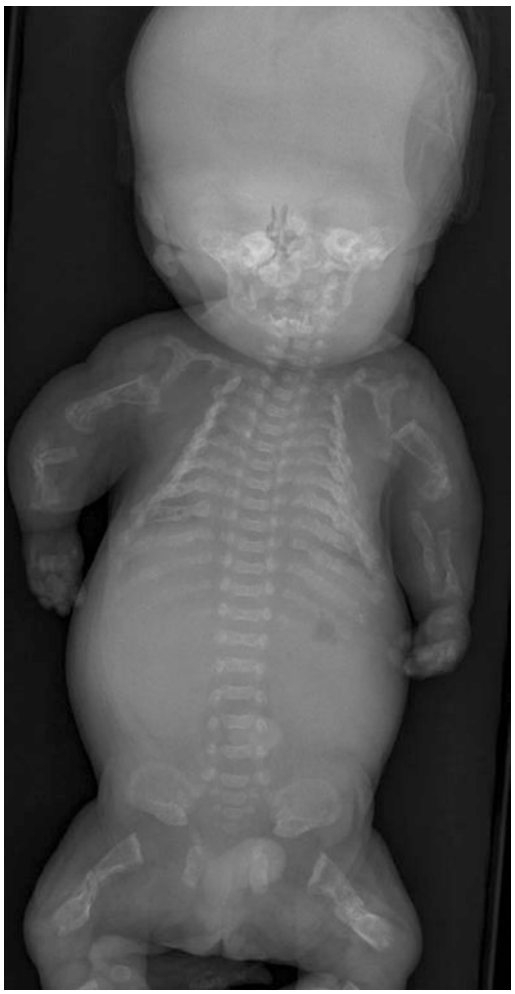


Fig. 14.13 Stillborn neonate whose skeleton shows innumerable healing fractures of nearly every bone. Osteogenesis imperfecta type IIA was genetically confirmed

14.3.3 Additional Examinations

To experienced radiologists, the diagnosis OI will generally, in view of its characteristic lesions, not present many problems. When OI is suspected, radiological examination is essential. In prenatal ultrasound it is also possible to find characteristic defects, in those cases it often concerns type II.

In atypical cases, the biochemical analysis of the synthesis and structure of collagen may be used [50]. In order to differentiate with non-accidental trauma, a skin biopsy for the purpose of a fibroblast culture is not indicated. Steiner et al. concluded that based on clinical and radiological data, OI can be diagnosed in nearly all children. According to Steiner et al., biochemical collagen analysis should be restricted to the very rare situation in which there is continued diagnostic doubt regarding non-accidental trauma [51]. Lachman evaluated a large series of OI cases from the International Skeletal Dysplasia Registry and based on his

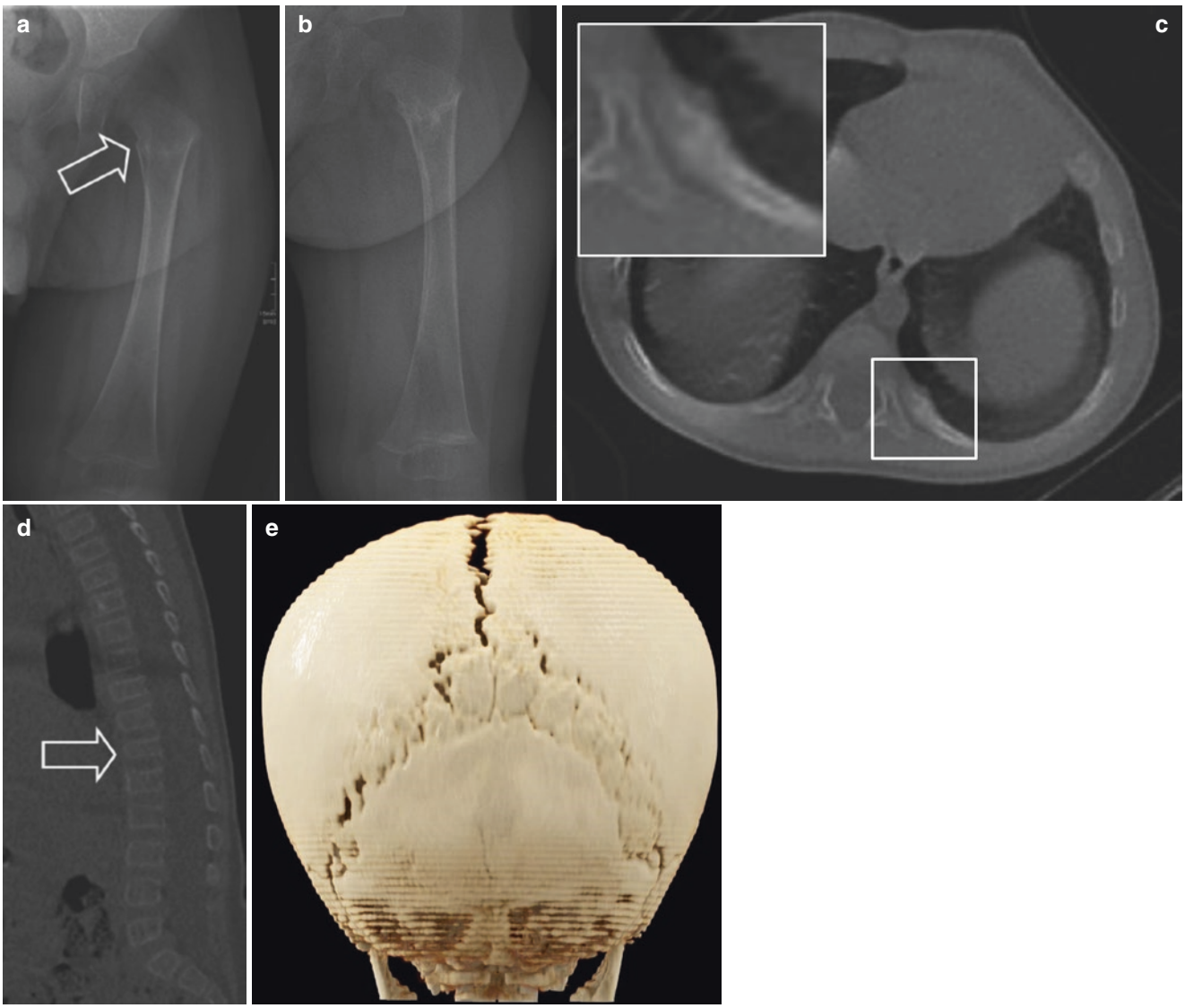


Fig. 14.14 Wormian bones and vertebral fractures in a 1-year-old infant who presented with pain in the left hip. (**a**) radiograph shows a fracture of the proximal femur. (**b**) follow-up radiography, as part of the skeletal survey, 15 days later shows sclerosis around the fracture line as a sign of healing. (**c**) Chest CT shows a healing posterior rib fracture (inset), (**d**) CT of the spine shows a fracture of the 12th thoracic vertebra, and (**e**) 3D cinematic rendering of the skull shows multiple Wormian bones. After thorough assessment the fractures were all ascribed to OI

findings suggested a diagnostic algorithm [52]. If the clinical evaluation and radiographs show clear diagnostic signs of OI, the diagnosis should be made. If there is a normal bone density but there remains doubt about the diagnosis of OI genetic testing should be performed. If genetic testing is negative then the diagnosis of OI can be rejected.

14.3.4 Osteogenesis Imperfecta and Non-accidental Trauma

In most cases, the differential diagnosis between OI and inflicted fractures due to non-accidental trauma is based on the clinical history (including a family history), physical examination, and radiological imaging. In most cases, it concerns children with type I. The blue sclerae, the skull defects ('wormian' bones), and the family history will soon clarify matters (Fig. 14.14a, b). Wormian bones and occasionally blue sclerae are also present in children with type III. In type IV no blue sclerae or skull lesions are seen. Theoretically, it complicates the differentiation; however, this type occurs so rarely that only in exceptional cases it may lead to mistakes. The chance that a child less than 1 year old will be diagnosed with type IV (without a positive clinical history, a normal skull radiograph, and normal teeth) is estimated to be 3:1,000,000 [53].

One should be aware that non-accidental trauma most certainly also happens in children with OI. Even if this disor-

der provides a plausible explanation for the fractures and bruises that correspond with the minimal trauma recorded in the clinical history, one should still exclude non-accidental trauma. Knight and Bennett describe a child with OI in whom non-accidental trauma could only be confirmed after the attending physicians had found facial abnormalities that proved that the child had been beaten [54].

14.4 Rickets

14.4.1 Introduction

> *'We affirm therefore, that this disease doth very rarely invade children presently after their birth, or before they are six moneths old; (yea, perhaps before the ninth moneth) but after that time it beginneth by little and little daily to rage more and more to the period of eighteen moneths, then is attaineth its pitch and exaltation, and as it were resteth in it, till the child be two years and six months old: so that the time of the thickest invasion is that whole year, which bears date from the eighteenth month, two years and a half being expired, the disease falleth into its declination, and seldom invadeth the child, for the reasons already alledged.'*
> Francis Glisson, 1597–1677 [55]

Rickets, or in the history known as the 'English disease' was thought to be a disease of the past but in recent years it is on the rise again making it a relevant differential diagnosis, if it is suspected that fractures were sustained in non-accidental circumstances [56–64]. Rickets can be classified as calciopenic or phosphopenic. Calciopenic rickets most commonly is caused by a nutritional deficiency of vitamin D and/or calcium. This type of rickets is the most common type of rickets in children and is also known as nutritional rickets. Phosphopenic rickets usually is caused by renal phosphate wasting. Another rare type is vitamin D-resistant rickets. As a result of the disturbance, the ossification of the cartilaginous tissue is too slow which leads to irregularities in the metaphyses (Fig. 14.15a, b).

The onset and presentation of rickets depend on the cause and the severity of the deficiency, e.g. nutritional rickets may usually become evident only after several months of a vitamin D-deficient diet [65–67]. Vitamin D deficiency is mostly found in infants and toddlers/preschool children, with a peak age of 3–18 months [68, 69]. However, rickets can also be found in neonates, if the mother had a vitamin D deficiency during pregnancy, and in older children, as long as the growth plates have not closed [68, 70]. In post-pubertal adolescents and adults with closed growth plates and completed skeletal maturation, vitamin D deficiency will lead to osteomalacia [66, 70]. There are numerous causes of rickets, amongst others malabsorption of fat, diseases of the liver or kidney, the use of medication (e.g. phenobarbital or phenytoin), deficient diets (e.g. lactovegetarians), and insufficient exposure to sunlight, e.g. dark-skinned children (Table 14.4) [71–73].

From a medical point of view, there is an increased interest in vitamin D-deficient rickets. The British Paediatric and Adolescent Bone Group states that deficiency should be defined as a plasma concentration of 25 hydroxyvitamin D of less than 25 nmol/L (10 ng/mL), insufficiency being

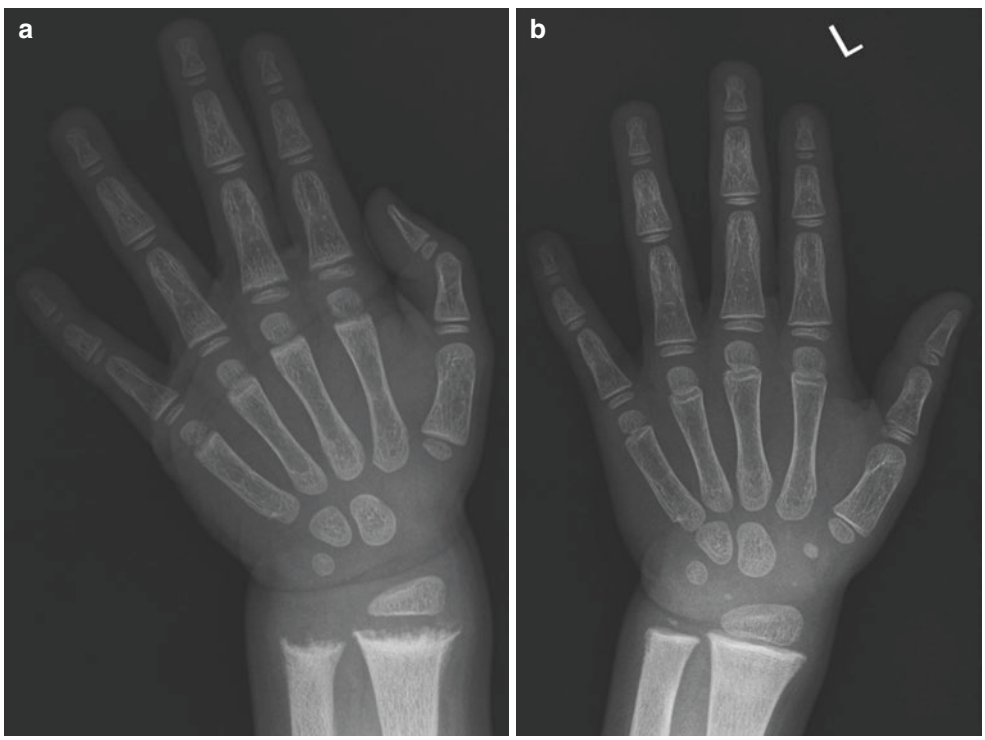


Fig. 14.15 (**a**) Two-year-old girl with rickets. The distal radius shows metaphyseal widening (splaying), concavity (cupping), and irregularities (fraying). (**b**) Two months after the therapy was initiated, the image has nearly been normalized and only a small amount of sclerosis of the distal metaphysis of the radius remains

Table 14.4 Causes of rickets

Vitamin D deficiency
Deficient diet
Deficient endogenous synthesis
Metabolic vitamin D disorders
Pseudovitamin D deficiency
Use of anticonvulsives
Chronic kidney failure
Disorders of the gastrointestinal tract
Gastrointestinal malabsorption disorders
Partial or total gastrectomy
Hepatobiliary diseases
Chronic pancreatic insufficiency
Acidosis
Distal tubular acidosis (classic or type I)
Secondary forms of renal acidosis
Ureterosigmoidostomy
Medication-induced
Chronic acetazolamide use
Chronic salmiac use
Chronic kidney failure
Phosphate deficiency
Inherited:
X-linked hypophosphatemic rickets
Acquired:
Low dietary phosphate contents
Sporadic hypophosphatemic osteomalacia (phosphate diabetes)
Tumour-associated rickets
Osteomalacia
Neurofibromatosis
Fibrous dysplasia
Renal tubular disorders
Primary renal tubular disorders
Renal tubular disorders associated with systemic metabolic abnormalities cystinosis
Glycogenosis
Lowe syndrome
Systemic disorders with associated renal abnormalities
Congenital
Wilson's disease
Tyrosinemia
Neurofibromatosis
Acquired
Multiple myeloma
Nephrotic syndrome
Kidney transplantation
Primary mineralization defects
Inherited
Acquired
Fluor treatment
Bisphosphonate treatment
Rapid bone formation, with or without relative defects in bone resorption
Postoperative hyperparathyroidism with osteitis fibrosa cystica
Osteopetrosis
Defective matrix synthesis
Fibrogenesis imperfecta ossium
Others
Magnesium-dependent conditions
Axial osteomalacia
Parenteral nutrition
Aluminium intoxication
Isophosphamide treatment

25–50 nmol/L and sufficiency a concentration greater than 50 nmol/L [74]. In 2002, Chesney spoke of a 'third wave' of rickets because the disease seemed to have reappeared in the last decade of the twentieth century for the third time in the past 100 years [75]. The first wave of rickets occurred during the industrial revolution in the Western world, when the smog in large cities blocked the sun's ultraviolet rays, resulting in a blockage of the formation of vitamin D3 (cholecalciferol) in the skin. In fact, rickets was the first paediatric disorder due to environmental pollution [56, 76]. The discovery of cod-liver oil was an effective remedy for this problem. The second wave of rickets was the result of breast-feeding by women who did not get enough exposure to sunlight because their religious beliefs prescribed nearly full body coverage [75]. Chesney saw the third wave in the United States mainly in dark-skinned infants, particularly among African American children, whose mothers were breast-feeding. These mothers seldom took their children outside and they were exclusively breastfed. The reasons for avoiding the outside varied from concerns over sunshine, fear of violence, and degeneration of the neighbourhood. Some mothers worked at home on the telephone or with a personal computer. This third wave was also seen in the Middle East and Europe, particularly in infants born of mothers with full body coverage and wearing veils [77–80].

Ladhani et al. stated that vitamin D deficiency had clearly reappeared as a problem in the United Kingdom, especially in 'at risk' ethnic minority groups [81]. Ladhani et al. evaluated the data of 65 children with rickets, of whom 39 were of Asian origin, 24 Afro-Caribbean, and 2 Eastern European. The occurrence of the third wave in England probably is best illustrated by the number of hospitalizations due to rickets, being the highest in England in five decades in the period 2007–2011 [82]. According to Goldacre et al. hospitalization rates for rickets in paediatric patients under the age of 15 years were low in the 1960s and 1970s and decreased further in the 1980s and 1990s, but increased in the 2000s. Most hospitalized children were under the age of 5 years.

Concerning this third wave Moon et al. stated *'... despite the suggestion of a secular increase in the incidence of rickets, this observation may be driven more by changes in population demographics than a true alteration to age, sex and ethnicity-specific incidence rates; indeed rickets remains uncommon overall and is rarely documented in fair-skinned children'* [83].

According to Roberts and Gaillard rickets is more prevalent in a number of distinct populations:

- Premature infants (especially if on parenteral nutrition).
- Infants with unbalanced nutrition (protracted exclusive breast-feeding; non-vitamin D supplemented formula-fed infants; vegetarian diets).

Table 14.5 Risk factors for nutritional rickets [90]

Maternal factors
Vitamin D deficiency
Dark skin pigmentation
Full body clothing cover
High latitude during winter/spring season
Other causes of restricted sun (UVB) exposure, e.g.: Predominant indoor living, disability, pollution, cloud cover
Low vitamin D diet
Low calcium diet
Poverty, malnutrition, special diets
Infant/childhood factors
Neonatal vitamin D deficiency secondary to maternal deficiency/ vitamin D deficiency
Lack of infant supplementation with vitamin D
Prolonged breast-feeding without appropriate complementary feeding from 6 months
High latitude during winter/spring season
Dark skin pigmentation and/or restricted sun (UVB) exposure, e.g. Predominant indoor living, disability, pollution, and cloud cover
Low vitamin D diet
Low calcium diet
Poverty, malnutrition, special diets
Risk factors are prevented by:
Sun exposure (UVB content of sunlight depends on latitude and season)
Vitamin D supplementation
Strategic fortification of the habitual food supply
Normal calcium intake

- Maternal vitamin D deficiency (lack of sun exposure/dark skin in sun-poor countries; lack of outdoor time; clothing that eliminates sun exposure).

Munns et al. formulated, based on a systematic review, an overview of risk factors for nutritional rickets and osteomalacia in their 'Global Consensus Recommendations on Prevention and Management of Nutritional Rickets' (Table 14.5). According to the authors the risk of vitamin D deficiency could be reduced by sun exposure (UVB content of sunlight depends on latitude and season), vitamin D supplementation, strategic fortification of the habitual food supply, and normal calcium intake.

14.4.2 Clinical History—Clinical Manifestations

In case of a suspicion of vitamin D deficiency in a child, the medical history should contain extensive information about the risk factors as described in Table 14.5. In case of a newborn with suspected vitamin D deficiency or a breast-fed child one should also take an extensive medical history of the mother.

Complaints suggesting a vitamin D deficiency are, amongst others, restlessness, sleeping problems, muscular pain, impaired growth, possibly resulting in stunted height, and developmental delay (Table 14.6) [68, 84]. Sleeping problems can be due to tenderness and pain, especially in the long bones of the lower extremity, which may wake the child at night [84]. The developmental delay may concern a delay in crawling, sitting, or walking. Peridontitis, abnormal development of teeth, and dental decay have also been described as vitamin D deficiency.

Table 14.6 Clinical manifestations in rickets

Pain or sensitive bones
Skeletal deformation
Bowing of the long bones of the legs
Pectus carinatum
Ricketsian rosary
Asymmetrical or deformed skull
Pelvic and spinal deformities, including scoliosis and kyphosis
Increased risk for sustaining fractures
Dental abnormalities
Muscular spasms
Growth disturbances, possibly resulting in stunted height

In severe cases of hypocalciaemia, due to vitamin D deficiency, tetany (muscle twitching and sharp bending of the wrist and ankle joints), and hypocalcaemic seizures can occur [85]. Also, intellectual disability has been described to occur due to hypocalciaemia in children with vitamin D deficiency [68, 84]. Some children may develop cardiomyopathy with cardiac failure, which may result in death [68].

14.4.3 Physical Examination

If rickets is suspected, a complete physical examination, including a dental examination, should take place. Because deformation of parts of the skeleton and tenderness and pain of the 'bones' are characteristic findings in children with rickets, the whole skeleton should be carefully inspected and palpated. While examining the head, one may notice craniotabes (soft skull bones), frontal bossing (prominent forehead), and a delay in closing of skull sutures. Also, an asymmetric or deformed skull can be seen.

Inspection of the thorax may show the presence of a rachitic rosary (bead-like nodules) at the costochondral junction. Pectus excavatum and carinatum can occur in children with rickets (Fig. 14.16).

Swelling of the joints, e.g. of the wrists and ankles can also be seen [68]. Bowing of the lower leg and knock knees can be present [70, 86].

14.4.4 Biochemical Testing

In case of rickets biochemical testing will show low serum levels of 25(OH)D (25 hydroxyvitamin D), calcium, phosphorus, and of urinary calcium), combined with high levels of serum parathyroid hormone (PTH), alkaline phosphatase

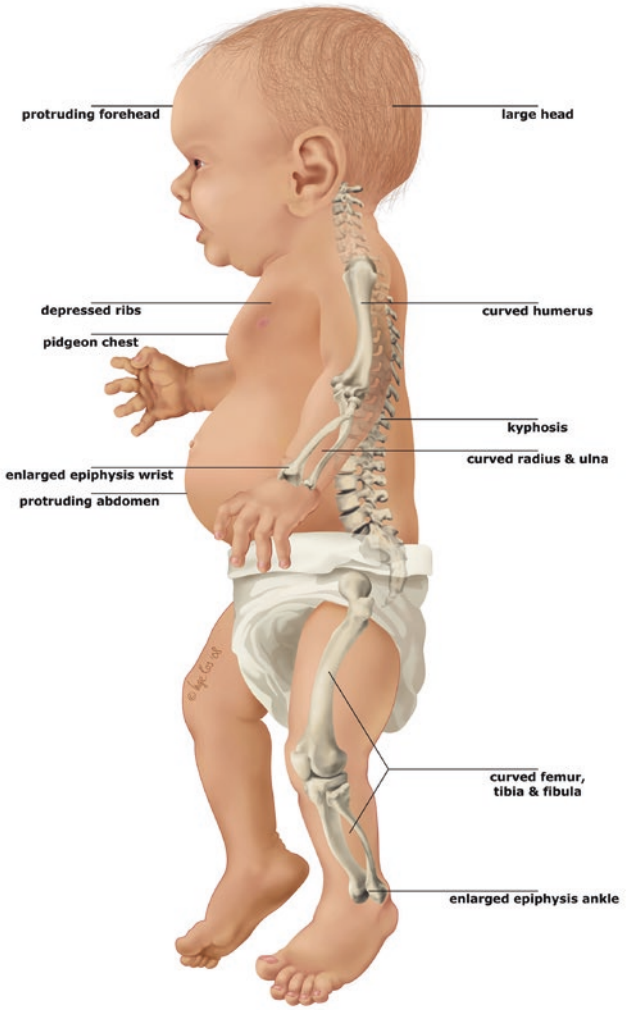


Fig. 14.16 Graphic representation of the anomalies that can be found in rickets

(ALP), and of urinary phosphorus [68, 87]. If both serum inorganic phosphorus and PTH levels are normal, the diagnosis of rickets is unlikely [87]. One should realize that serum alkaline phosphatase is not only elevated in rickets but also in healing fractures [88]. Based on biochemical testing it is possible to differentiate vitamin D-deficient rickets from X-linked hypophosphatemic rickets (see Sect. 14.6.4) and hypophosphatasia (see Sect. 14.7.5) (Table 14.7).

There is no worldwide consensus concerning normal (sufficient), insufficient, and deficient serum levels of 25 (OH) D. Normal 25 (OH)D values vary from over 10 to 12 ng/ml (25–30 nmol/l, e.g. the Netherlands) to over 30 ng/ml (75 nmol/l, e.g. Canada, Japan), while deficiency is defined by some organizations as vitamin D levels under 20 ng/ml (50 nmol/l, e.g. United States, Japan) [89]. Based on a systematic review, Munns et al. recommended that deficiency should be defined as a plasma concentration of 25 (OH)D (25 hydroxyvitamin D) of less than 12 ng/mL (30 nmol/L), insufficiency being 12–15 ng/ml (30–50 nmol/l) and sufficiency a concentration above 15 ng/ml (50 nmol/l) [90]. According to Munns et al., the incidence of nutritional rickets increases with 25(OH)D levels under 12 ng/ml (30 nmol/l). Munns et al. added: *'It should be noted that nutritional rickets has been reported in children with 25(OH) D concentrations above 30 nmol/l and that it may not occur with very low 25(OH)D concentrations but is more likely to occur with deficiency sustained over time,* i.e. *chronic deficiency'*. They further remarked that *'Most children with vitamin D deficiency are asymptomatic'*. The British Paediatric and Adolescent Bone Group states that deficiency should be defined as a plasma concentration of 25 (OH)D of less than 10 ng/ml (25 nmol/l), insufficiency being 10–15 ng/ml (25–50 nmol/l) and sufficiency a concentration above 15 ng/ml (50 nmol/l) [74].

Table 14.7 Laboratory findings differentiating nutritional rickets, X-linked hypophosphatemic rickets, and hypophosphatasia

Parameter	Nutritional rickets	X-linked hypophosphatemic rickets	Hypophosphatasia
Alkaline phosphatase	↑	↑	↓
Pyridoxal 5′-phosphate	–	↓	↑
Calcium	↓	Normal	↑ or normal
Phosphate	↓	↓	↓ or normal
Parathyroid hormone	↑	↑ or normal	↓ or normal
Vitamin D	↓	↓ or normal	Normal

14.4.5 Radiological Examination

As stated before, the diagnosis of nutritional rickets is made on the basis of history, physical examination, and biochemical testing, but should always be confirmed by appropriate radiographs [90].

Plain radiography of the metaphyseal sites (wrists and ankles) is usually performed to confirm the diagnosis [89]. In classic cases this shows widening and fraying of the metaphyses and a ricketsian rosary (Figs. 14.15 and 14.17). Since the radiographical lesions are symmetrically present throughout the whole body, even in the presence of fractures, it will be no problem to differentiate with fractures sustained in non-accidental circumstances (Fig. 14.18). In milder cases, the metaphyseal abnormality may strongly resemble metaphyseal corner fractures (Fig. 14.19). In these cases, comprehensive laboratory tests and repeating the tests at a 2-week follow-up will provide valuable information. When the disease is more protracted bowing of the long bones, in particular of the legs, may occur (Fig. 14.20).

According to Roberts and Gaillard, the deficient/insufficient mineralization due to vitamin D deficiency is most evident in the growing skeleton at the metaphyseal zones of provisional calcification, where there is an excess of non-mineralized osteoid. This will result in widening of the growth plate and abnormal configuration of the metaphysis [65, 91]:

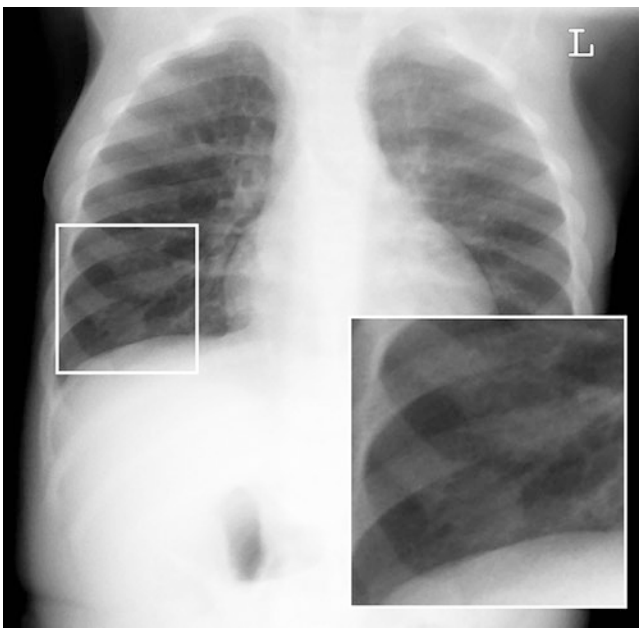


Fig. 14.17 Seventeen-month-old boy with rickets. The chest radiograph shows irregularities of the costochondral junctions (inset), resulting in the image known as ricketsian rosary

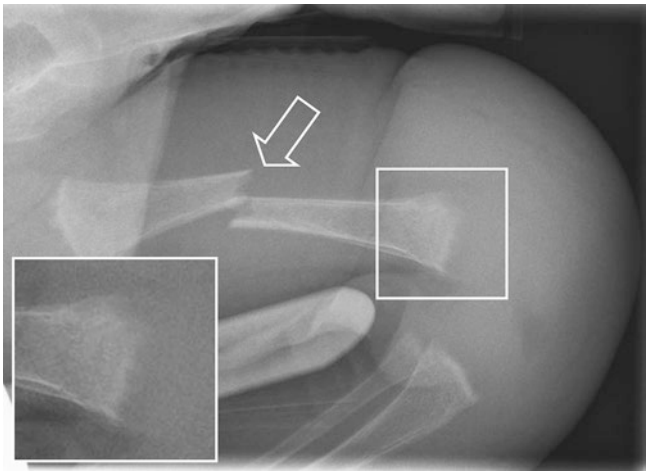


Fig. 14.18 Eight-month-old boy with a transverse mid-shaft femur fracture (open arrow) without evident trauma in the patient history. The distal femur metaphysis shows severe splaying, cupping and fraying, corresponding with rickets. Laboratory tests showed a vitamin D deficiency

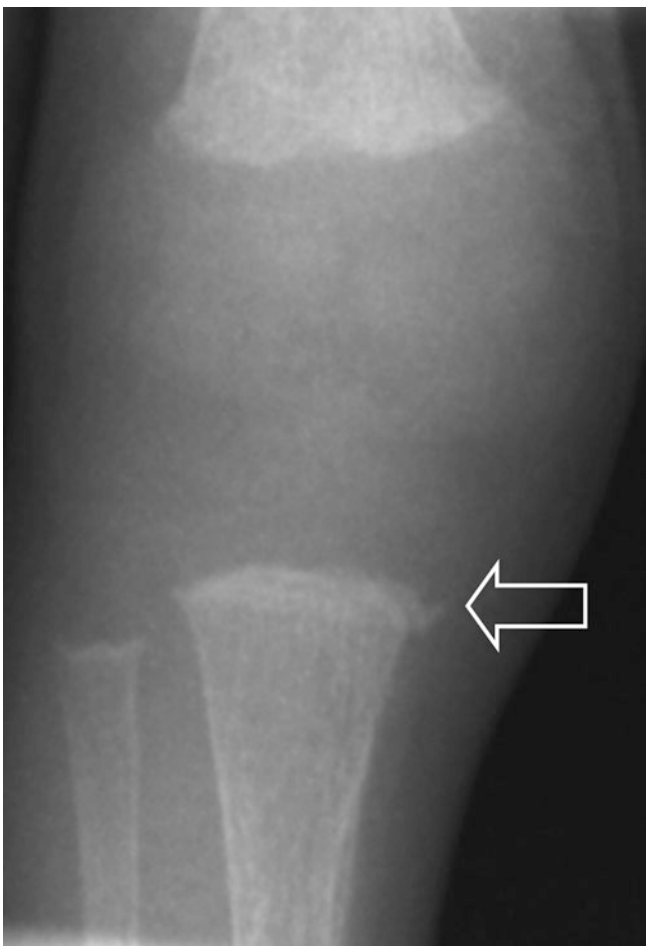


Fig. 14.19 Premature infant, born at 27 weeks pregnancy. A radiograph on day 56 shows an irregular aspect of the proximal metaphysis of the tibia (open arrow). Laboratory tests confirmed the diagnosis of rickets. This anomaly could be interpreted as a metaphyseal corner fracture

- Fraying: Indistinct margins of the metaphysis.
- Splaying: Widening of metaphyseal ends.
- Cupping: Concavity of metaphysis.

These features are most prominent in bones where growth is greatest:

- Knee: Distal femur, proximal tibia.
- Wrist: Especially the ulna.
- Anterior rib ends: Rachitic rosary.

In rickets even bones that appear sufficiently mineralized are weak. This can result in the bowing of long bones, most commonly seen in the lower extremities once the child is walking. The legs bow outward with variable deformity of the hips (coxa vara and coxa valga) (Fig. 14.20).

Other bone deformities are also noted such as genu valga (knock-knees) and vara as well as protrusio acetabuli/impression of the sacrum and femora into the pelvis, leading to a triradiate configuration of the pelvis. The lower ribs may also be drawn inwards inferiorly, due to inwards pulling by the diaphragm (Harrison's sulcus) [65, 70]. Some children may develop scoliosis/kyphosis due to the deficiency. Sometimes pseudo-fractures are present. As a rule, periosteal reactions and new bone formation are abundantly present. Fractures may occur in children with rickets (see Sect. 15.3.2).

In the early stages of rickets, radiographs may depict no pathology. Chemical changes in blood serum, however, can already be found at this time. The distal radius and ulna typically demonstrate rachitic lesions early on radiographs. In preterm neonates and young infants, radiographs of the knee may be more reliable than those of the wrist [92]. In more advanced stages of rickets, radiographic changes are pathognomonic; however, the underlying cause needs to be established using clinical and biochemical assessments. False-negative findings can occur in the early phase of disease. In healing rickets, the zones of provisional calcification become denser than the diaphysis. In addition, cupping of the metaphysis may become more apparent [65].

Fig. 14.20 Bow legs in a child treated for rickets

14.4.6 Rickets and Non-accidental Trauma

Although fractures resulting from rickets can give rise to the incorrect conclusion that these fractures were sustained in non-accidental circumstances, it does not mean that the presence of a vitamin D-related disorder combined with fractures excludes non-accidental trauma [93]. Duncan and Chandry describe a little girl who presented at the age of 3 months with multiple fractures [94]. The infant was also diagnosed with rickets. When she suddenly died at the age of 5 months, unnatural death was suspected. However, this could not be confirmed. Three years onwards, non-accidental trauma was confirmed in another child of that family. However, this does not prove in any way that the first child also died in non-accidental circumstances.

Vitamin D deficiency can also be the result of neglecting a child; for example when parents/carers fail or refuse to give vitamin D supplements. Children with a nutrition-based vitamin D deficiency are also at risk for osteopenia (reduced bone density). Severe osteopenia (osteoporosis, see Sect. 14.7.3) may lead to an increased risk for fractures. Often it is difficult to differentiate between fractures sustained by physical violence and fractures sustained by minimal force on a weakened bone structure. Comprehensive damage to non-weight bearing parts of the skeleton, such as clavicles, ribs, lower arms, and hands, are also suspect in children with rickets. This is certainly true when radiological examination reveals signs of healing fractures. In recent years this differential diagnosis has become an issue of debate in courtrooms (see Sect. 15.3.2).

Kepron and Polanen looked at the histology of rickets versus fractures sustained in non-accidental circumstances

Table 14.8 Histologic and radiologic features of fractures, metaphyseal fractures, and rickets (from Kepron and Polanen, reprinted with permission) [95]

	Radiology	Histopathology
General fractures	Fracture line	Fracture line with physical disruption of cortex and trabeculae
	Periosteal new bone formation	Haemorrhage, fibrin clot, inflammatory infiltrate, marrow necrosis
	Loss of fracture line demarcation (with healing)	Chronic inflammation, granulation tissue
	Soft callus formation	Osteoid and woven bone formation
	Hard callus formation	Increased activity of osteoclasts and osteoblasts
	Remodeling of callus	
Metaphyseal fractures	Lucency within the subphyseal region of the metaphysis	Physical disruption of the trabeculae of the primary spongiosa
	With complete fracture across the metaphysis, the fracture fragment may appear as a linear density ('bucket handle lesion') or triangular fragments at the edges of the metaphyses ('corner fractures'), depending on the angle of projection	Undermining of the subperiosteal bone collar ± haemorrhage and fibrin (acutely)
	Sclerosis along the fracture margins (with healing) ± periosteal new bone formation (many heal without)	Decrease in cellularity and trabecular density within the metaphysis
		Osteoclast activation, granulation tissue, and fibrosis (with healing)
Rickets	Osteopenia	Widening of the growth plate; architectural disarray
	Decreased mineralization of the zone of provisional calcification	Extension of cartilage into the primary spongiosa
	Irregularity of the metaphyseal margin	Irregular border between growth plate and primary spongiosa with the maintenance of physical continuity between zones
	Transverse widening of the metaphysis and metaphyseal cupping	Blood vessels penetrate the growth plate
	Periosteal new bone formation	Abnormal trabeculae within the diaphysis
	Bowing of the diaphyses of long bones	Necrosis and inflammatory infiltrates are not features

[95]. In their publication, they present a good overview of both the histologic as well as the radiologic differences between fractures in general, metaphyseal corner fractures and rickets (Table 14.8).

14.5 Syndromes and Congenital Disorders

14.5.1 Introduction

In the medical literature one can find case reports on suspected inflicted fractures (e.g. unexplained fresh or healing/healed fractures) in children with skeletal abnormalities belonging to certain syndromes and congenital disorders. In this paragraph a short overview is given; be aware the overview does not claim to be complete.

14.5.2 Sickle Cell Anaemia

Sickle cell anaemia (OMIM # 603903) is an autosomal recessive inherited disease in which a mutation in the β-globin gene leads to binding between β1 and β2 chains of two haemoglobin molecules HbS [96]. As a result, a polymer nucleus grows and subsequently fills the erythrocyte which leads to disruption of its architecture and shape, causing sickle-shaped erythrocytes [97]. It is one of the most common monogenic diseases in the world with millions of people suffering from this disease. It is seen in particular in people (themselves or their ancestors) that hail from Africa, the Mediterranean countries and the Arabic peninsula, India, and parts of South and Central America. Generally, the diagnosis can easily be made with a microscopic test.

The symptoms of sickle cell anaemia are due to abnormal erythrocytes that take on a sickle shape resulting in early breakdown, which leads to anaemia. When the sickle-shaped cells occlude small vessels, it may cause pain and infection. These symptoms come in episodes of acute illness, e.g. vaso-occlusive pain, acute chest, and cholelithiasis and progressive organ damage, e.g. stroke, autosplenectomy, pulmonary hypertension, and haemolytic anaemia. In young infants septicaemia is an important cause of death.

On a conventional radiograph, periostitis (Fig. 14.21) and radiolucencies with blurred margins are visible. These are present in bone infarcts as well as in osteomyelitis (which is more prevalent in patients with sickle cell anaemia). After a

Fig. 14.21 Child with confirmed patient history of sickle cell anaemia presented at the emergency department with pain in the right upper arm. The radiograph shows extensive periosteal reaction (open arrow). Furthermore, there is an extensive anomaly in the medullary cavity, corresponding with a bone infarction (arrow)

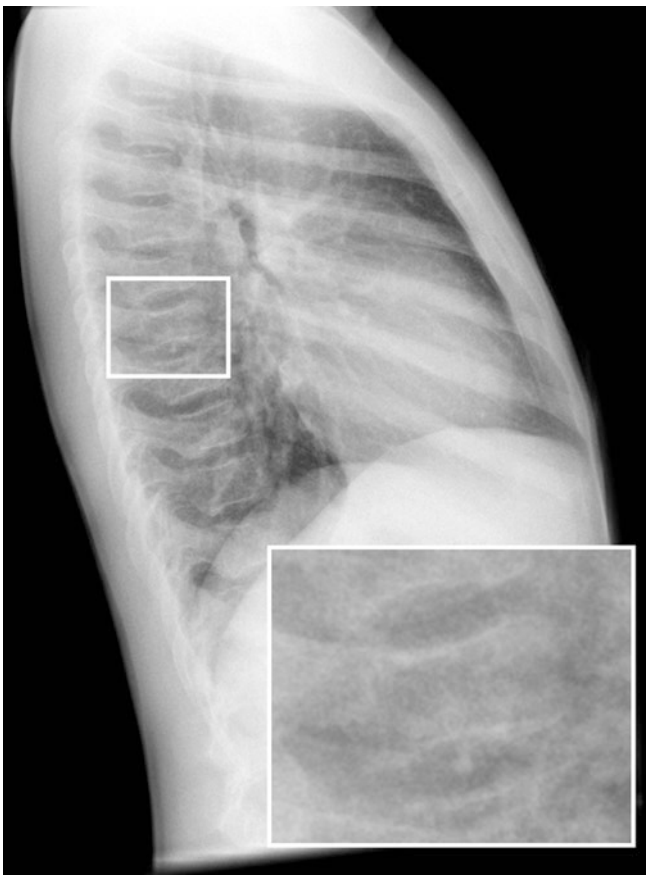


Fig. 14.22 Eleven-year-old boy with a confirmed history of sickle cell anaemia. Routine radiograph shows the characteristic H-shaped collapsed vertebra (see inset)

period of time, the bone will start to show sclerosis. Since this image may resemble a healing fracture, it may cause confusion in the differential diagnosis [98]. Quite distinguishing for sickle cell anaemia are the centrally located depression fractures of the vertebral corpora, which result in the signature H-shaped vertebrae (Fig. 14.22).

14.5.3 Alagille Syndrome

Alagille syndrome (arteriohepatic dysplasia, OMIM #118450) is an autosomal dominant disease with variable expression [99]. In this syndrome, various organs (liver, heart, kidneys, eyes, and skeleton) may be affected. Furthermore, often typical facial features are seen (prominent forehead, hypertelorism, small chin, and saddle nose). Mental retardation may also be present (mostly mild to moderate). In the United States, the incidence is approximately 1:100,000 live-born children.

Most children with Alagilles syndrome are seen for the first time before the age of 6 months for neonatal jaundice based on cholestasis (70%) or cardiac symptoms (17%). Sometimes there is a deficiency of fat-soluble vitamins (A, D, E, and K).

The reported skeletal defects refer to the vertebrae, the so-called 'butterfly vertebrae' (Fig. 14.23), and to the ribs and arms/hands (shortened radius, ulna, and digital phalanges) [100]. It is also possible that post-fracture bone deformation will not spontaneously correct itself [101].

The diagnosis is made based on the earlier-mentioned complaints, complemented with genetic and (if so required) pathological examinations (liver biopsy).

14.5.4 Duchenne Muscular Dystrophy

Duchenne muscular dystrophy (OMIM #310200) is a recessive X-linked inherited progressive proximal muscular dystrophy with pseudohypertrophy of the calf muscles. It is the most prevalent form of muscular dystrophy seen in childhood and has an incidence of 1:3500 boys. Usually, onset is before the age of 3 years, and after a period of being wheel-

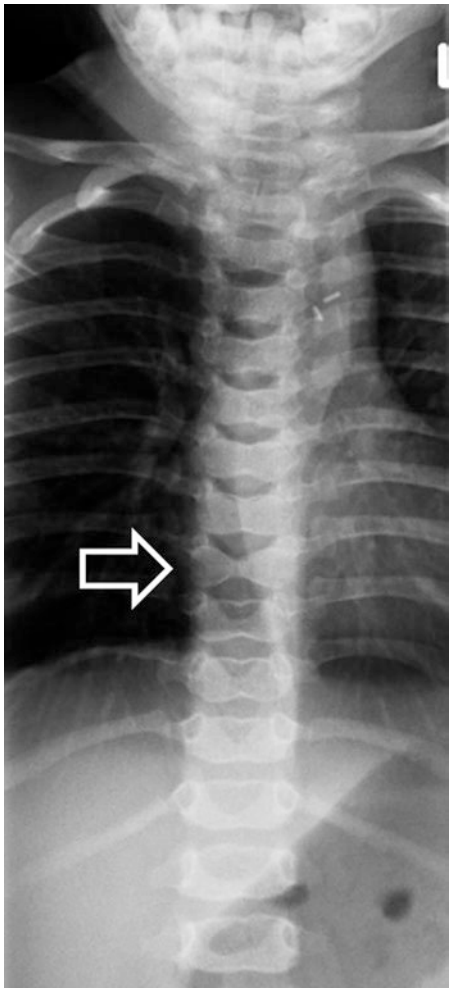


Fig. 14.23 One-year-old infant with pulmonary arterial hypertension and renal cysts. The chest radiograph shows a mid-thoracic butterfly vertebra (open arrow)

chair dependent, the patient generally dies before the age of 21 years from respiratory failure [102].

In children weight-bearing physical activities are critical for bone health. In children with Duchenne muscular dystrophy mobility and weight-bearing physical activities are reduced, which may result in fractures, due to decreasing bone health and increasing fragility [103]. This most commonly concerns fractures of vertebral bodies and/or long bones (Fig. 14.24a–c) [104].

McDonald et al. reported on a population of 378 patients (average age 12 years; range 1–25 years). Of this group, 79 (20.9%) had experienced a fracture [105]. In this population, no rib fractures were reported. Since it is generally possible to make a firm diagnosis, the differential diagnosis should present no problem in these children.

Children with Duchenne muscular dystrophy often show the development of a progressive (kypho)scoliosis of the spine, due to fractures of the vertebral bodies [104, 106]. This is most commonly seen in young adolescents (mean age of onset 13.29 years; progression rate 11.48° per year) [107].

14.5.5 Congenital Pseudarthrosis

Congenital pseudarthrosis of the tibia is a relatively rare defect, associated with neurofibromatosis type 1 (NF1). Fifty-five percent of patients with congenital pseudarthrosis also have NF1 [108]. Congenital pseudarthrosis is the result of segmental mesodermal dysplastic bone development. Although the defect is linked to neurofibromatosis, no neurofibromas are visible near the pseudarthrosis. In 99% of cases the defect is unilateral [108]. Because of segmental bone weakness, there is progressive anterolateral bowing of the tibia (often also fibula), which may finally break. Congenital pseudarthrosis of other bones is found to a lesser degree. In case there is a fracture, it will happen in the first 2 years of life. After the fracture has been sustained, no spontaneous healing takes place, which results in a real pseudarthrosis. Treatment of the fracture is protracted, difficult, and sometimes even without success, which will lead to amputation.

Crawford distinguishes four radiological types [109]. Typical anterolateral bowing is always present:

- Type I: Normal medullary cavity.
- Type II: Narrowed medullary cavity with concomitant cortical thickening.
- Type III: Presence of cysts, sometimes with a fracture.
- Type IV: Actual pseudarthrosis. After the fracture, a pseudarthrotic image develops in which the fracture ends may assume an osteolytic-like configuration.

Type II can simulate non-accidental trauma when the patients have not sought medical help, because the image can be interpreted as a healing fracture that has been badly reduced (Fig. 14.25a, b). Type IV may be interpreted as pseudarthrosis due to non-immobilization of the fracture in a neglected child (Fig. 14.25c).

14.5.6 Caffey's Disease

Caffey's disease (OMIM #114000), also known as infantile cortical hyperostosis, is a little understood autosomal dominant disorder that manifests itself by a gross periosteal reaction during infancy [110]. It mainly involves the long bones (often asymmetrically). However, the disease may also manifest itself in different locations, such as the mandible, ribs, scapula, and clavicle [111, 112]. Spine, phalanges, and pel-

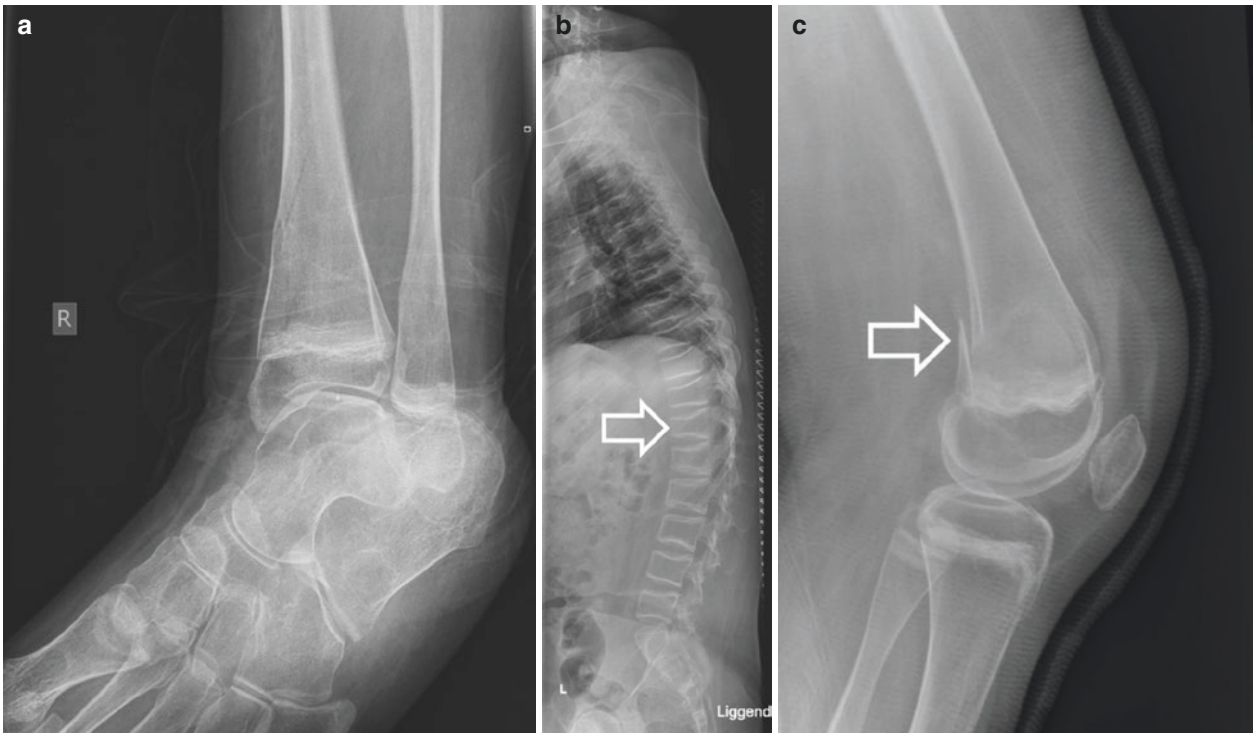


Fig. 14.24 Sixteen-year-old adolescent boy with Duchenne muscular dystrophy. Radiography shows insufficiency fractures of the (**a**) ankle, (**b**) multiple vertebral bodies, most pronounced at level thoracic 12, and (**c**) the distal femur

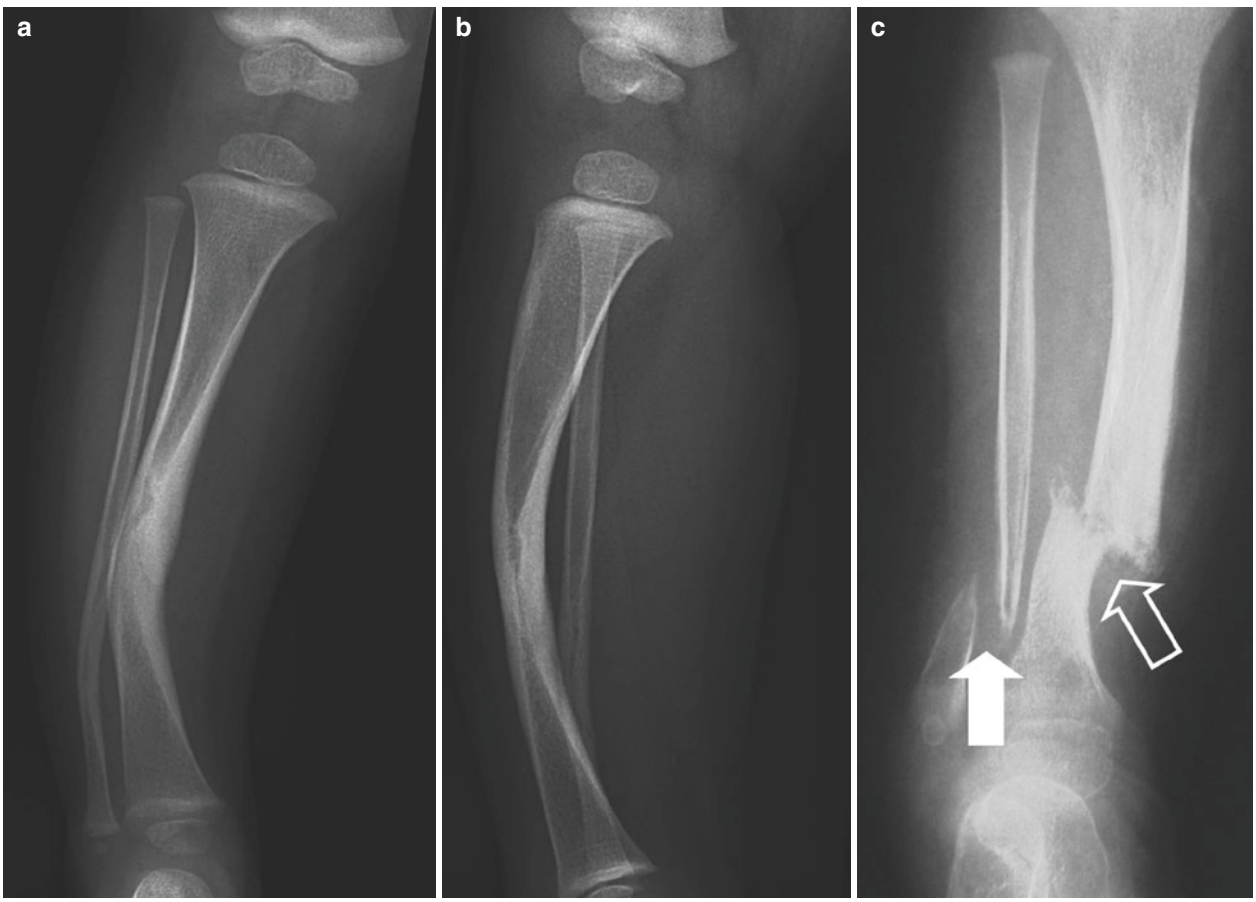


Fig. 14.25 (**a**) Congenital pseudarthrosis of the tibia, Crawford type II. Antero-posterior view of the lower leg. Anterolateral bowing with thickening of the cortical bone and narrowing of the medullary cavity. (**b**) Lateral view of the lower leg. (**c**) Crawford type IV with typical pseudarthrosis of the tibia (open arrow) and osteolytic-like pseudarthrosis of the fibula (arrow)

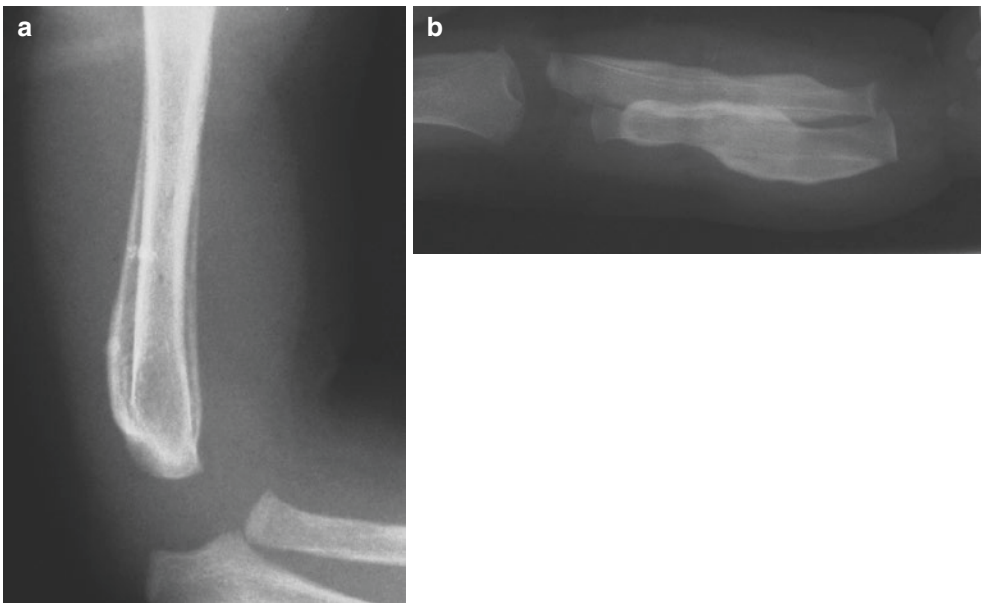


Fig. 14.26 (**a**) Two-month-old girl with Caffey's disease. Clinical presentation showed painful, slightly swollen limbs. Radiographs showed extreme periosteal reaction of the distal humerus without fractures. (**b**) Extreme periosteal reaction along the complete length of the radius and ulna without fractures. After a year the girl was symptom-free and the bone anomalies had all disappeared

vis are hardly ever affected. Its autosomal dominant inheritance is reported to have variable expression [113]. Caffey's disease is self-limiting, appears in the first 5 months after birth and by the age of 3 years the clinical and radiological abnormalities have disappeared. It is sometimes present at birth and has in rare cases been identified in utero [114–118].

The patients have swollen and painful extremities, are irritable and show a (sub)febrile temperature. ESR and alkaline phosphatase are often elevated. Conventional radiographs show extensive subperiosteal new bone formation in the affected bones. In the extremities, the epiphyses and metaphyses are usually spared (Fig. 14.26a, b). As a result of subperiosteal haemorrhages, extensive subperiosteal new bone formation can also be found in non-accidental injuries. Consequently, Caffey's disease can simulate non-accidental trauma and vice versa [119, 120]. However, in contrast to Caffey's disease, in non-accidental trauma fractures are a regular feature and the periosteal reaction is predominantly metaphyseal.

Other disorders associated with pronounced periosteal reactions, and as such may cause differential diagnostic problems, are hypervitaminosis A, prostaglandin-E1 medication in children with duct-dependent cardiac defects, leukaemia, syphilis, some storage diseases (I-cell disease, mucolipidosis type II, GM gangliosidosis type I), vitamin C deficiency and hypertrophic osteoarthropathy. These diseases can be differentiated from Caffey's disease on the basis of clinical, clinical-chemical, and radiological results.

14.5.7 Menkes Disease

Menkes disease (OMIM #309400) is a progressive neurodegenerative disease based on a congenital, X-linked recessive defect in copper metabolism [121]. Copper is required for enzymes essential to the formation of bone, nerve tissue, and other structures.

The disease is seen nearly exclusively in boys. Yet, there are a few case reports on girls with Menkes [122, 123]. The incidence is not well known. In Australia, Danks estimates it at 1:40,000 live births [124]. Over the period 1976–1987, Tonnesen et al. estimated the incidence in Denmark, France, The Netherlands, the United Kingdom, and Germany to be 1:298,000 live births [125]. On the other hand, Gu et al. found a much lower incidence in Japan, 1:4.9 million boys [125].

Birth-related skull fractures have been reported as the first diagnostic clue to the diagnosis [126, 127]. But in most cases, the onset of the disease occurs in the first weeks to months after birth. Initially, development progresses normally, after which there is a delay with loss of the earlier acquired skills. Hypotonia and convulsions may also be present, as is 'failure to thrive'. The prognosis is poor: generally, the children will die before the age of 4 years, although sporadically there has been the odd child that survived longer, even past the age of 21 years [128].

A striking feature is the hair anomaly, and not just on the scalp, but also of the lashes and eyebrows. In light-skinned people, the hair is often without colour and sometimes silver or steel-grey in colour. In black-haired ethnical groups, the hair may be blonde or brown in colour. It is sparsely present and fuzzy or stubbly to the touch. It is crinkly and breaks easily. It resembles glass wool. Consequently, Menkes disease is also known as 'kinky hair disease' or 'steely hair disease'.

Besides the hair anomaly, the children often have growth problems, anterior rib defects (flaring), and 'wormian bones' on radiographs (Fig. 14.27a, b). Due to the disturbances in bone metabolism, which causes osteoporosis, there is a risk

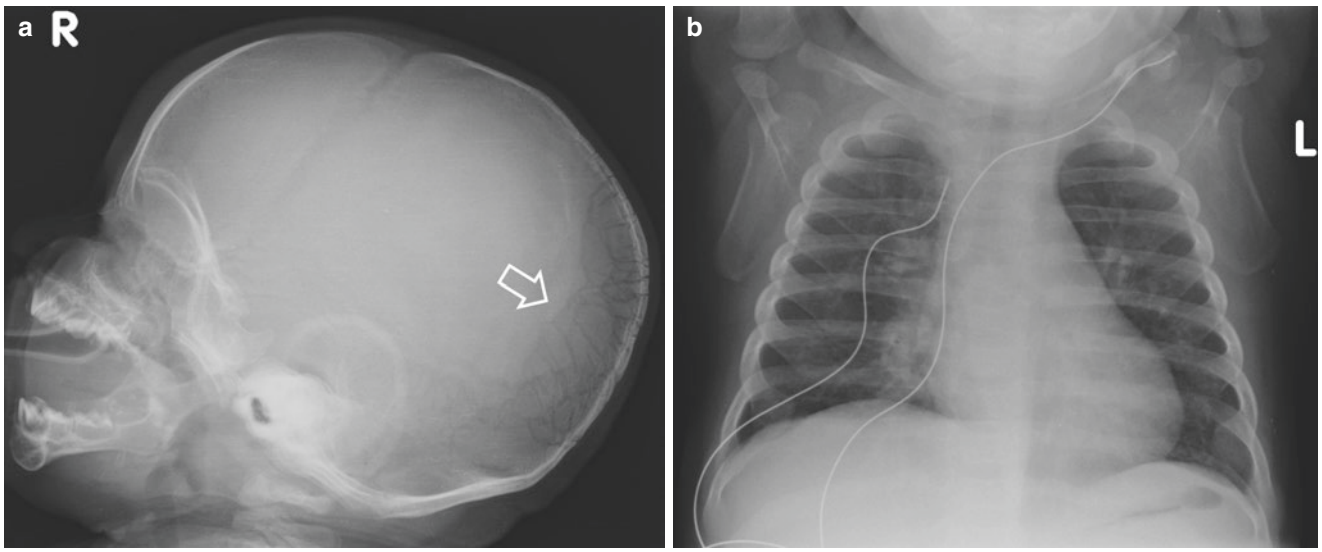


Fig. 14.27 Six-month-old infant diagnosed with Menkes disease. (**a**) Lateral radiograph of the skull shows multiple wormian bones (arrow). (**b**) AP chest radiograph shows cupping/flaring of the ribs

of fractures. Moreover, metaphyseal defects and periosteal reactions may be found. On radiographs, this set of anomalies is indistinguishable from fractures resulting from non-accidental trauma. However, the clinical history, combined with the above-mentioned symptoms should make it possible to differentiate between disease and non-accidental trauma.

In the medical literature, there are multiple case reports that describe cases of Menkes disease that initially were mistaken for non-accidental trauma. Most of them focus on neuroradiological findings however, some focus on the skeletal findings:

- In 1974 Adam et al. were the first to include Menkes syndrome in the differential diagnosis of inflicted injuries, due to non-accidental trauma (child abuse) [129]. They described the findings in two boys: a 10-week-old boy and a 6-month-old boy. The findings were studied by serial radiologic examination of the extremities and by selected studies of the central nervous system. This was before the standardized use of CT scans for the evaluation of the brain. A suspicion of non-accidental trauma arose in the 10-week-old boy because of the finding of *'multiple bilateral metaphyseal fractures with striking periosteal new bone formation involving both humeri, femurs, tibiae and fibulae'* and a negative history. The authors concluded that the finding of flared and fragmented metaphyses and the evidence of brain damage in children with Menkes syndrome could be mistaken for signs of trauma, because of the similarity of these findings to the findings in, what they called, the 'infant abuse syndrome' (non-accidental trauma).
- Seay et al. described the clinical courses and the findings in serial computerized tomography (CT) scans of four patients with Menkes disease [130]. The authors concluded that Menkes disease should be suspected in male infants with psychomotor deterioration and seizures, or when trauma is suspected from subdural hematoma and multiple fractures.
- Grünebaum et al. described four children with copper deficiency who did not have Menkes disease [131]. All four showed 'sickle-shaped metaphyseal spurs', two children showed fractures of these spurs. This case report seems to indicate that the metaphyseal defects in Menkes' syndrome may be the result of copper deficiency.
- Wendler and Mutz described an 10-week-old infant with multiple fractures [132]. The child developed *'marked cortical thickening of many bones, which raised the suspicion of a battered child syndrome. Unusual progression of bone thickening and hitherto undescribed excessive bone remodeling led to the diagnosis of Menkes 'kinky hair disease, …'*.
- Jankov et al. described a neonate with a rapidly progressing fatal syndrome. The boy died on day 27. He had been seen because of an acute presentation with severe intra-abdominal bleeding, haemorrhagic shock, and multiple fractures. The physicians made the diagnosis at autopsy, which was confirmed by copper accumulation in the fibroblast culture [133].
- Cronin et al. described the findings in a 6-month-old boy with Menkes syndrome whose symptoms, including bone and connective tissue disturbances, originally were thought to be from non-accidental trauma [134].
- Hill et al. evaluated the findings in 35 children with Menkes disease and found 4 children with apparent C2 posterior arch defects consistent with spondylolysis or incomplete/delayed ossification [135]. According to the authors these findings may simulate cervical spine fractures, due to non-accidental trauma.
- Wacks et al. described a 5-month-old boy in whom, on a chest radiograph obtained for a cough and shortness of

breath, a healing right posterior seventh rib fracture was seen [136]. A skeletal survey showed irregularity of the bilateral distal radial, ulnar, and femur metaphyses, indicative of healing fractures, and wormian bones along the lambdoid sutures. The metaphyseal irregularities, however, are those normally seen in Menkes disease and not metaphyseal corner fractures. Droms et al. reported the same case a year later [137].

- Akinsey et al. described a case of a 12-week-old boy with a 2-day history of fussiness and vomiting [138]. Because of dehydration an intra-osseous needle was placed and on follow-up radiography, to confirm proper placement, healing metaphyseal fractures were reported. On further work-up CT of the head showed Wormian bones and CTA showed tortuous vessels, and based on these findings in combination with the physical exam and laboratory findings the correct diagnosis of Menkes disease could be made.

14.5.8 Pain Insensitivity in Spina Bifida

In spina bifida there may be insensitivity to pain in the lower extremities. When there is incomplete paralysis, an effort will be made to have children with this disorder walk with devices such as splints. As a result, abnormal stress on the joints may lead to damage to the epiphyseal plate and the metaphysis, possibly resulting in a fracture. Moreover, patients with a severe form of spina bifida will develop immobilization-related osteoporosis. The combination of osteoporosis and pain insensitivity may lead to fractures that are only noticed at a later stage (Fig. 14.28).

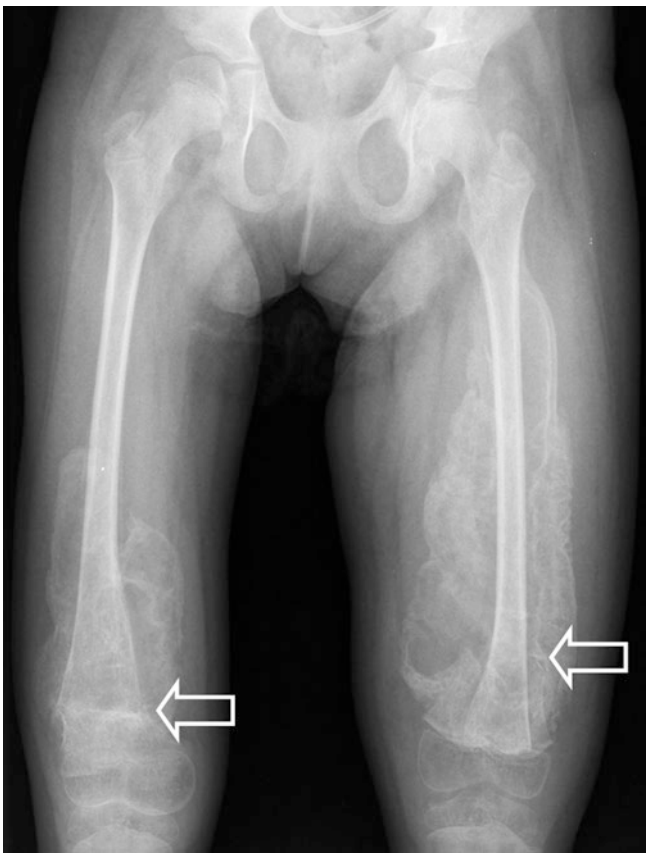


Fig. 14.28 Six-and-a-half-year-old girl with spina bifida showed bilateral swollen knees at physical examination. Radiographs revealed bilateral distal metaphyseal femur fractures (open arrows) with extensive new bone formation. Based on the clinical history, non-accidental trauma was excluded

14.5.9 Congenital Pain Insensitivity

Congenital pain insensitivity (OMIM # 243000) is an autosomal recessive disease. Children with this disease have normal intelligence. The only aberrant neurological finding is their insensitivity to pain, which may lead to a plethora of unaccounted-for injuries. A similar congenital disorder is congenital insensitivity to pain with anhidrosis (OMIM # 256800), also known as hereditary sensory and autonomic neuropathy (Fig. 14.29). Especially in young children, repeated damage to the growing skeleton will not be noticed. This may cause defects to metaphyses and epiphyses. A meticulous neurological examination and careful clinical history will make it possible to differentiate from non-accidental trauma [139, 140].

Fig. 14.29 Six-year-old girl with hereditary sensory and autonomic neuropathy type IID (HSAN2D) (a serious defect in pain sensitivity) with a swollen left foot. A radiograph of the foot showed a torus fracture of metatarsal I (open arrow)

14.6 Skeletal Dysplasias

14.6.1 Introduction

Skeletal dysplasias are a heterogeneous group of disorders characterized by anomalies in bone and cartilage development and growth. Although the prevalence of skeletal dysplasias (350:1,000,000) is many times higher than that of bone tumours (20:1,000,000), trainee radiologists generally pay little attention to these disorders [141]. The resulting lack of knowledge may result in the unjust allocation of a radiological finding such as a metaphyseal spur in Jeune's 'asphyxiating thoracic dysplasia' (Fig. 14.30a, b, OMIM %208,500) to non-accidental trauma. Collaboration with clinical geneticists is essential in these cases [43].

14.6.2 Metaphyseal Chondroplasia Type Schmid

Metaphyseal chondroplasia type Schmid (OMIM #156500) is a rare autosomal dominant inherited skeletal dysplasia, characterized by irregular margins of the metaphyses (Fig. 14.31) [142–145]. The metaphyseal defects cause bowing and shortening of the extremities during growth. The metaphyseal defects are very similar to rickets (see Sect. 14.4) and may be confused with metaphyseal corner fractures.

14.6.3 Spondylometaphyseal Dysplasia 'Corner Fracture Type'

Spondylometaphyseal dysplasia 'corner fracture type' (Sutcliffe type) (OMIM %184,255) is a rare skeletal dysplasia characterized by short stature and an aberrant, waddling gait [142, 145, 146]. Often the diagnosis is not made until the age of 2–3 years when an increasingly abnormal gait pattern is noticed.

From a radiological point of view, the most important anomalies are, as already indicated by its name, vertebral and metaphyseal anomalies, the latter having irregular margins. The metaphyses show triangular fragments, which may lead to the incorrect diagnosis of 'metaphyseal corner fractures' when one is not familiar with this dysplasia (Fig. 14.32a–c).

14.6.4 X-Linked Hypophosphatemia

X-linked hypophosphatemia (OMIM #307800) is an X-linked dominant disease that is the most common genetic form of rickets and osteomalacia. Due to the rarity of the disease the diagnosis is often delayed [147]. Younger children can present with varus deformity of the legs, which in itself can be physiological and thus lead to a delay in diagnosis (Fig. 14.33) [148]. Other presenting symptoms consist of delayed growth speed, joint enlargement, and dental infection.

The metaphyseal radiological findings of rickets can, as discussed before, look like metaphyseal corner fractures to the untrained eye and subsequently lead to the erroneous diagnosis of non-accidental trauma.

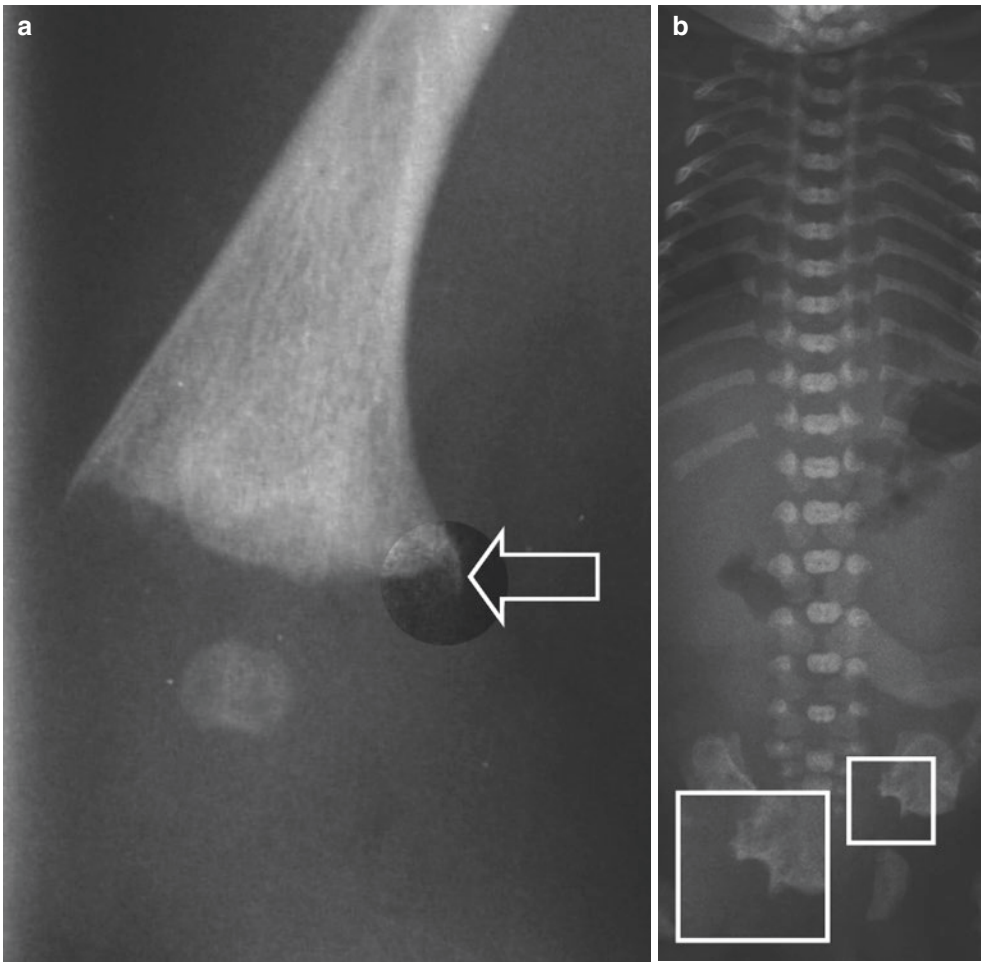


Fig. 14.30 (**a**) Neonate with a narrow chest. Radiographs of the knee showed a metaphyseal spur which may be confused with a metaphyseal corner fracture (open arrow). (**b**) Image of another patient with the same clinical presentation. Radiographs of spine and pelvis show a narrow chest and relatively short ribs. The pelvis shows spurs of the ilium (see inset). Based on, among other things, the radiological examination, the diagnosis Jeune's asphyxiating thoracic dysplasia could be made

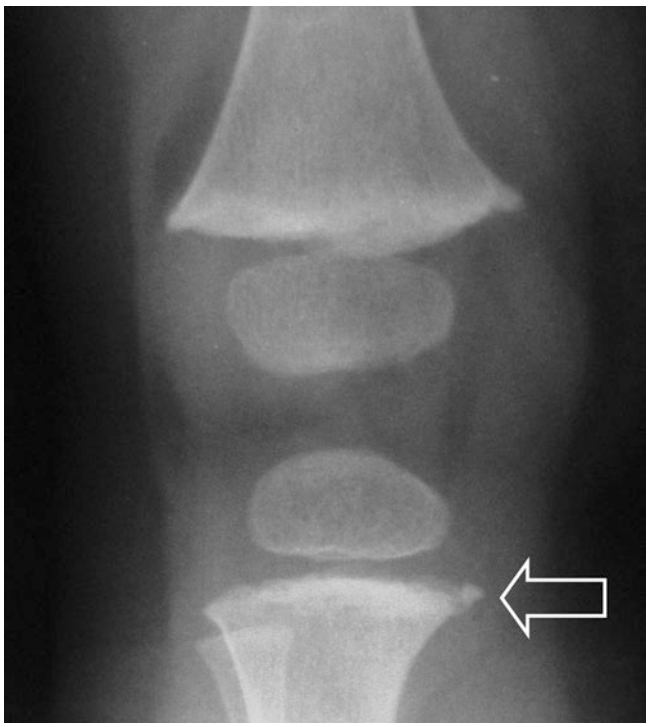


Fig. 14.31 Two-year-old child with metaphyseal chondrodysplasia type Schmid. The irregularities of the proximal metaphysis of the tibia have a strong resemblance to metaphyseal corner fractures (open arrow)

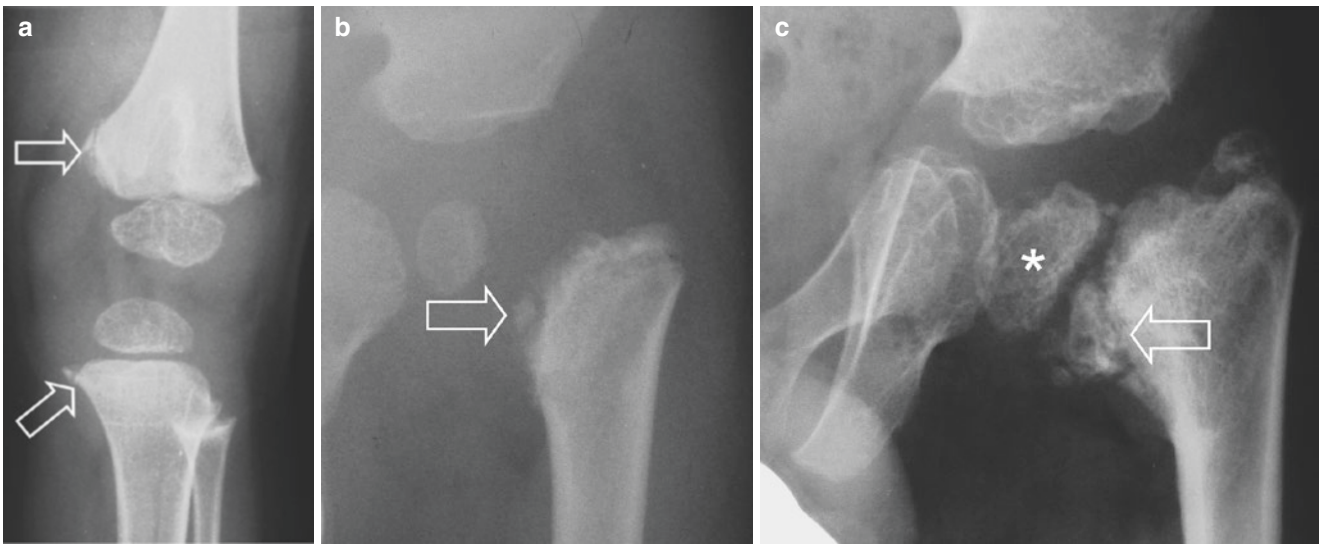


Fig. 14.32 (**a**) Two-year-old child with spondylometaphyseal dysplasia, corner fracture type. The distal femur metaphysis as well as the proximal tibia metaphysis show anomalies that strongly resemble metaphyseal corner fractures (open arrows). (**b**) Hip radiograph of the same patient shows an anomalous aspect of the proximal metaphysis of the femur (open arrow). (**c**) Radiological image of the left hip at 13 years of age shows an irregular metaphysis (open arrow) with strong developmental retardation and also deformation of the femoral head (asterisk)

Fig. 14.33 Three-year-old boy with X-linked hypophosphatemia. Lower limb radiographs show ricketsian bowing of the legs

14.7 Metabolic Disorders

14.7.1 Introduction

Metabolic bone disorders are a heterogeneous group of diseases with a wide variety of abnormal skeletal findings. Children with a metabolic bone disorder can form a diagnostic dilemma [149]. In the medical literature case reports can be found regarding fractures suspected to be due to non-accidental trauma in skeletal abnormalities compatible with metabolic disorders. In this paragraph an overview is presented. The overview does not claim to be complete.

14.7.2 Osteopetrosis

The term osteopetrosis (OMIM #166600) relates to a group of anomalies in which osteoclastic activity is suppressed, resulting in increased bone density (sclerosis) and ultimately in abnormal bone modelling [142].

In the context of a differential diagnosis in suspected non-accidental fractures, it is important that infantile osteopetrosis is mentioned. In this disorder, the metaphyses may show a translucent area and have an irregular aspect (Figs. 14.34 and 14.35a, b). The presence of generalized skeletal sclerosis and metaphyseal undertubulation makes it possible to come to the correct diagnosis. There are multiple case reports of fractures in infants with osteopetrosis, however, the dense skeleton on conventional radiography should lead to a straightforward diagnosis [150–154].

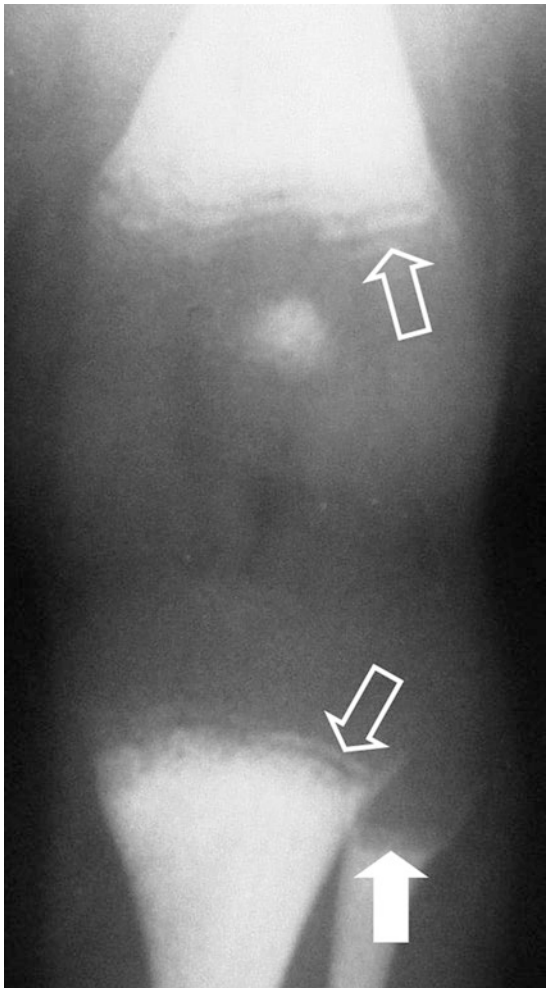


Fig. 14.34 Neonate with osteopetrosis. The distal femur and proximal tibia show irregular metaphyses (open arrow). In particular, in the proximal metaphysis, the image could be confused with a bucket-handle fracture (metaphyseal corner fracture). The proximal fibula also shows an anomalous aspect

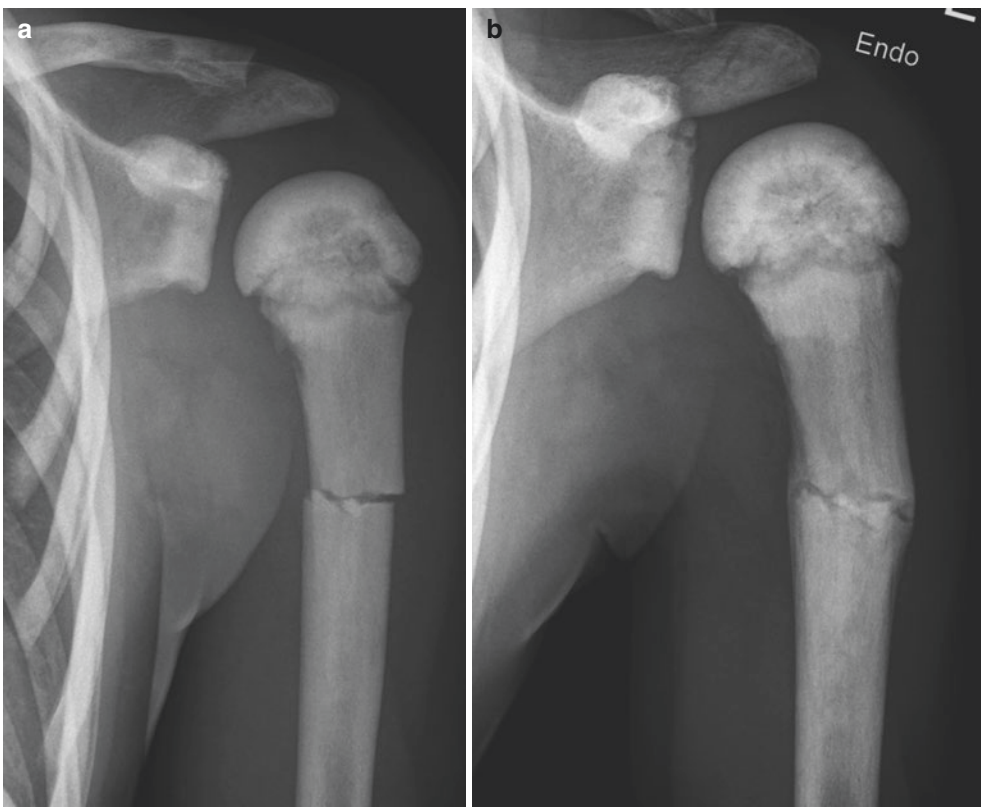


Fig. 14.35 (**a**) Ten-year-old child who presented with pain after a fall. Radiograph shows a transverse fracture in dense sclerotic bone. (**b**) Radiograph after 10 weeks shows a healing fracture. There was a strong familial history of osteopetrosis but parents did not pursue genetic testing

Paradoxically rickets is a well-known complication in children with osteopetrosis, this combination is also known as 'Osteopetrorickets' [152, 155–160]. In children with osteopetrorickets it is hypothesized that, despite a positive body calcium balance, the osteoclasts are unable to maintain the calcium-phosphorus product in the extracellular fluid [152]. On radiology, the long bones show a diffuse increase in bone density in combination with growth plate widening, metaphyseal splaying, and cupping.

14.7.3 Osteoporosis

The World Health Organisation defines osteoporosis as a systemic disease characterized by low bone mass and micro-architectural regression of bone tissue, resulting in increased fragility of the skeleton and risk for fractures. Paediatric osteoporosis may result from, e.g. chronic disease, malnutrition, immobilization, and genetic defects (Table 14.9) [161, 162]. The diagnosis of paediatric osteoporosis is, according to the International Society for Clinical Densitometry, based on 2 diagnostic criteria [163, 164]. First the presence of one or more vertebral compression fractures without a history of trauma. Second, the presence of a low bone mineral content or bone mineral density, which is corrected for age, sex, and body size and expressed as a Z score, should be less than −2.0 and the presence of a clinically significant fracture history as being abnormal. The latter is defined as one or more of the following: A) two or more long bone fractures by age 10 years and B) three or more long bone fractures at any age up to age 19 years.

Table 14.9 Causes of osteoporosis in childhood

Chronic diseases	Immobilization Anorexia nervosa Asthma Coeliac disease Neuromuscular diseases Chronic kidney failure Cystic fibrosis Diabetes mellitus Epilepsy Human immunodeficiency virus infection Inflammatory bowel disease Malignancies Organ transplantation Rheumatic diseases Sickle cell disease Thalassemia Turner syndrome
Endocrinopathies	Cushing's syndrome (hypercortisolemia) Growth hormone deficiency Hyperthyroidism Hyperparathyroidism Hyperprolactinemia Hypopituitarism Hypothyroidism Gonadal steroids deficiency/hypogonadism
Medication use	Anticonvulsive drugs Corticosteroids Cyclosporine A Heparin Lithium Methotrexate Various chemotherapeutics

A specific form of childhood osteoporosis is idiopathic juvenile osteoporosis, a self-limiting primary osteoporosis of unknown origin, seen mainly in children in their second decade of life (Fig. 14.36a, b) [165, 166]. The diagnosis of childhood osteoporosis is not always straightforward, since the commonly used techniques are validated for adults [167]. In osteoporosis, the most frequently seen fractures are vertebral and metaphyseal. In children with multiple fractures osteoporosis should be excluded.

14.7.4 Dysostosis Multiplex Congenita

Dysostosis multiplex congenita is a group of storage diseases of complex proteins that have a large number of aspects in common. These include mucopolysaccharidosis (such as Hurler disease (OMIM #607014) and Hunter disease (OMIM #309900)), gangliosidosis, and mucolipidosis.

Clinical manifestation depends on the degree of storage and the organs in which the metabolite is stored. When storage occurs in the brain, progressive mental retardation will be the primary symptom. Other clinical symptoms are: typically coarse facial features, opaque corneas, and organomegaly. Radiological lesions are: incomplete modelling of the long bones, epiphyseal dysplasia, broad ribs, abnormal configuration of the corpora vertebrae, in particular, at the thoracolumbar transition (so-called 'vertebral beaking' or 'hook-shaped vertebra'; Fig. 14.37). Periosteal reaction may be very pronounced in GM1 gangliosidosis and mucolipidosis II (I-cell disease) [142].

Suspected dysostosis multiplex congenita is usually based on clinical and radiological anomalies and is confirmed by biochemical analysis of urine and blood for abnormal metabolites. However, the younger the child, the more difficult it is to make the diagnosis, since at a young age the clinical presentation has not yet fully developed, and consequently the radiographs may appear to be normal.

In patients with dysostosis multiplex, an injury may unjustly be suspected based on the periosteal reaction in GM1 gangliosidosis and mucolipidosis II (I-cell disease) [119]. Also, when observed cursory, the spinal anomalies may be considered spinal fractures after non-incidental injuries. The clinical presentation and the radiological anomalies in the remaining skeleton are usually sufficient to reach the correct diagnosis.

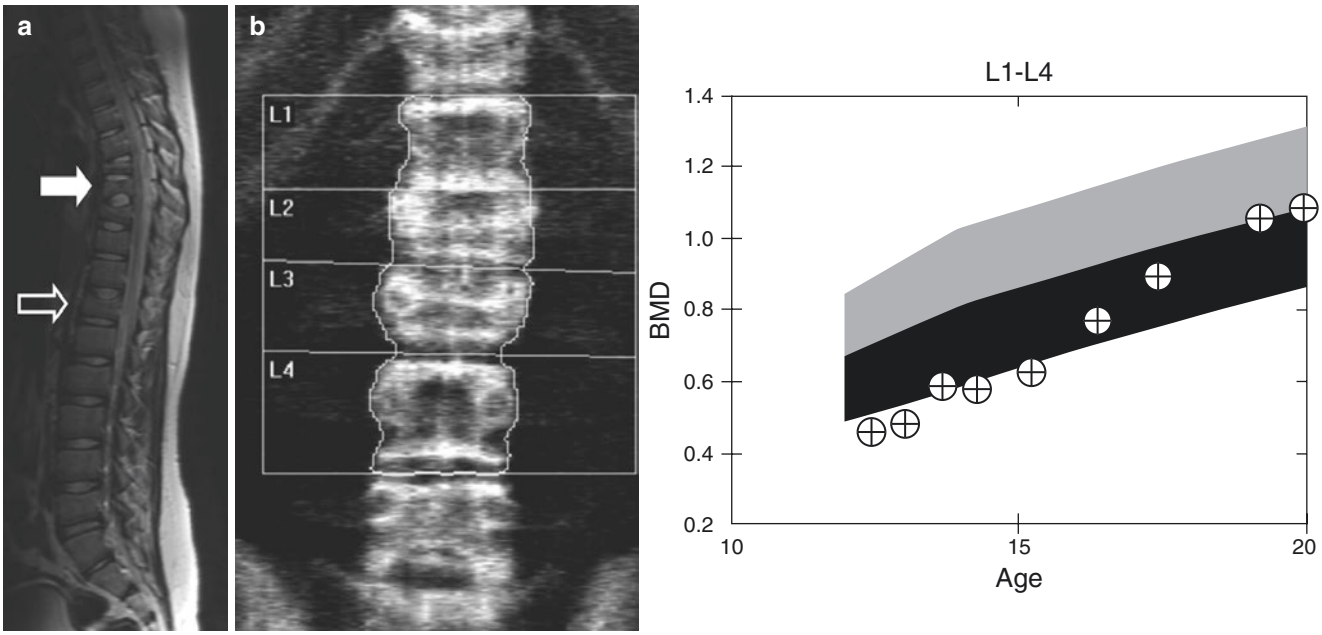


Fig. 14.36 (**a**) Twelve-year-old boy with idiopathic osteoporosis. The MRI of the spine shows collapsed vertebrae at levels Th7–9 (arrow) and Th12 (open arrow). (**b**) DXA scan of this patient, presenting the values of 12–20 years of age, shows normalization of bone mineral density

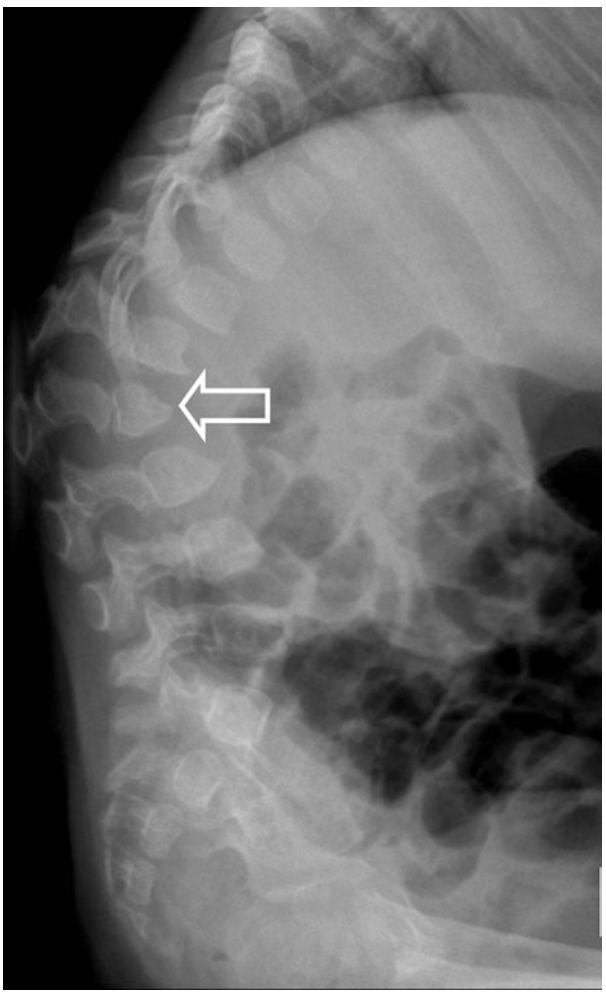


Fig. 14.37 Seven-month-old boy with mucopolysaccharidosis Hurler type. At several levels the lumbar spine shows considerably increased kyphosis and anterior beaking of the vertebral corpora

14.7.5 Hypophosphatasia

Hypophosphatasia (OMIM #241500) is a rare disorder caused by a mutation of the gene coding for the enzyme alkaline phosphatase [168, 169]. The prevalence is estimated to be 1:100,000. There are six categories, depending on age: the perinatal (fatal), benign perinatal, infantile, child and adult forms, and odontohypophosphatasia [168]. In the latter category only dental anomalies are present.

In young children decreased mineralization of the cranium is seen with wide sutures, which later progresses to craniosynostosis, a noticeable bowing of the long bones, sometimes even angular (kyphomelia), fractures and pseudofractures and irregular metaphyseal ossification defects (Fig. 14.38a, b) [142, 170]. Due to its heterogenic presentation, it may initially be difficult to diagnose, and the fractures, bowing and metaphyseal irregularities may even be reminiscent of non-accidental injury.

Moulin et al. describe a 9-year-old girl and her sister who frequently sustained fractures after trivial injuries. They had normal growth, normal sclerae, no rickets and only minor dental abnormalities. In the end, hypophosphatasia appeared to be the cause [171]. A clinical presentation of this kind may also look like non-accidental injuries within the home.

Ultimately, the diagnosis is made by DNA sequencing, measuring serum alkaline phosphatase activity, and proving an increased concentration of phosphoethanolamine and calcium in urine and pyridoxal 5′-phosphate and calcium in blood. By DNA sequencing, approximately 95% of mutations in severe hypophosphatasia (perinatal and infantile forms) can be found [168]. It is important to make the correct

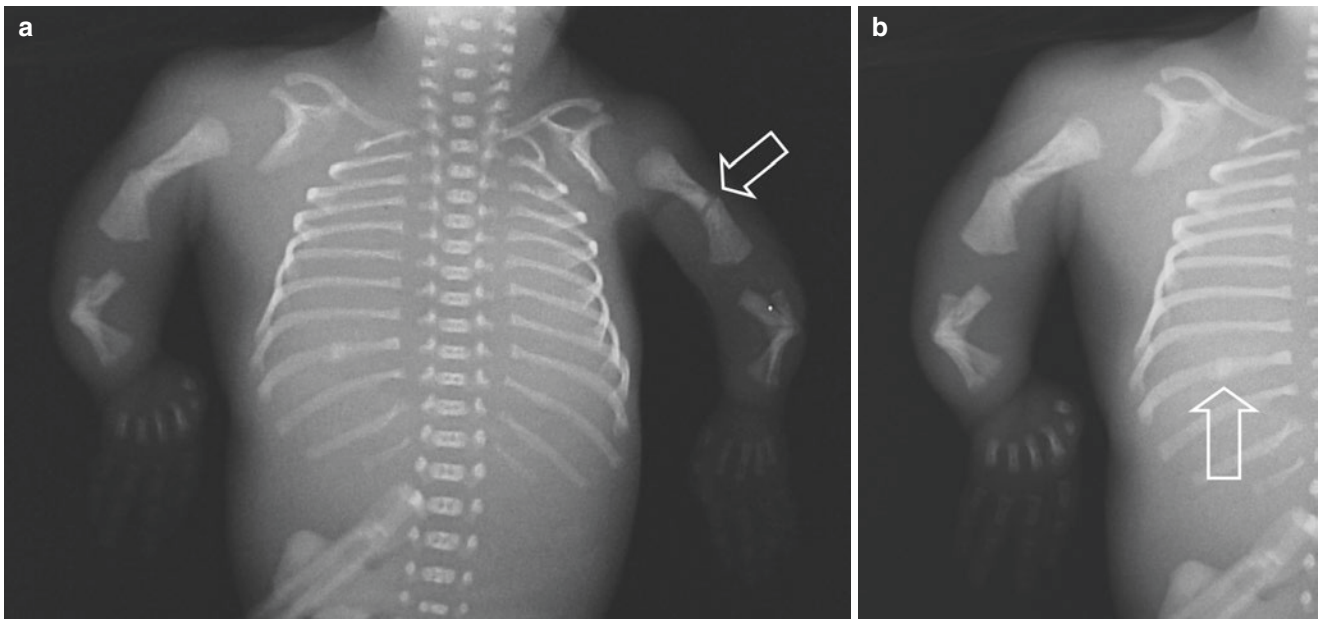


Fig. 14.38 (**a**) Neonate with hypophosphatasia, lethal perinatal variant. Bilateral angular bowing 'kyphomelia' of radius and ulna, and to a lesser extent of both humeri with (pseudo) fractures (open arrow). (**b**) Detail view of the chest shows a healing mid-posterior rib fracture (open arrow)

diagnosis as treatment with high-dose vitamin D, calcium supplements, or bisphosphonates can lead to an exacerbation of the symptoms of hypophosphatasia.

14.8 Infectious Diseases

14.8.1 Introduction

In the medical literature case reports can be found regarding suspected non-accidental trauma in skeletal anomalies compatible with infectious diseases. In this paragraph an overview is presented of the disorders. The overview does not claim to be complete.

14.8.2 Osteomyelitis

Osteomyelitis in childhood is a relatively rare diagnosis, with an estimated prevalence of 1:10,000 children under 12 years of age [172]. Since the course of the illness is often slow, it is often not diagnosed until it reaches a well-advanced stage.

In osteomyelitis, metaphyseal abnormalities and periosteal reactions may be found, which can resemble metaphyseal and other fractures, and as such result in an incorrect diagnosis of non-accidental trauma (Figs. 14.39, 14.40a–d, and 14.41a, b). Taylor et al. described a 7-month-old infant that had sustained a fracture of the left proximal humerus, without a clear explanation. Initially, non-accidental trauma was suspected [173]. However, follow-up examination showed that the radiological anomalies looked more like a

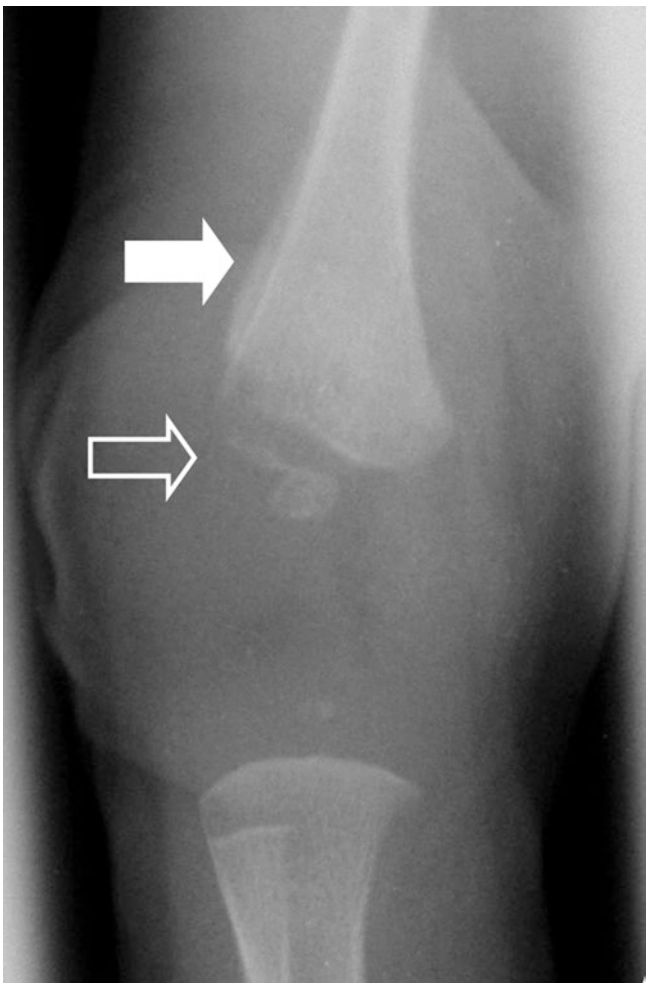


Fig. 14.39 Neonatal osteomyelitis of the distal femur. There is a periosteal metaphyseal reaction visible (arrow). Furthermore, there may be a metaphyseal corner fracture (open arrow)

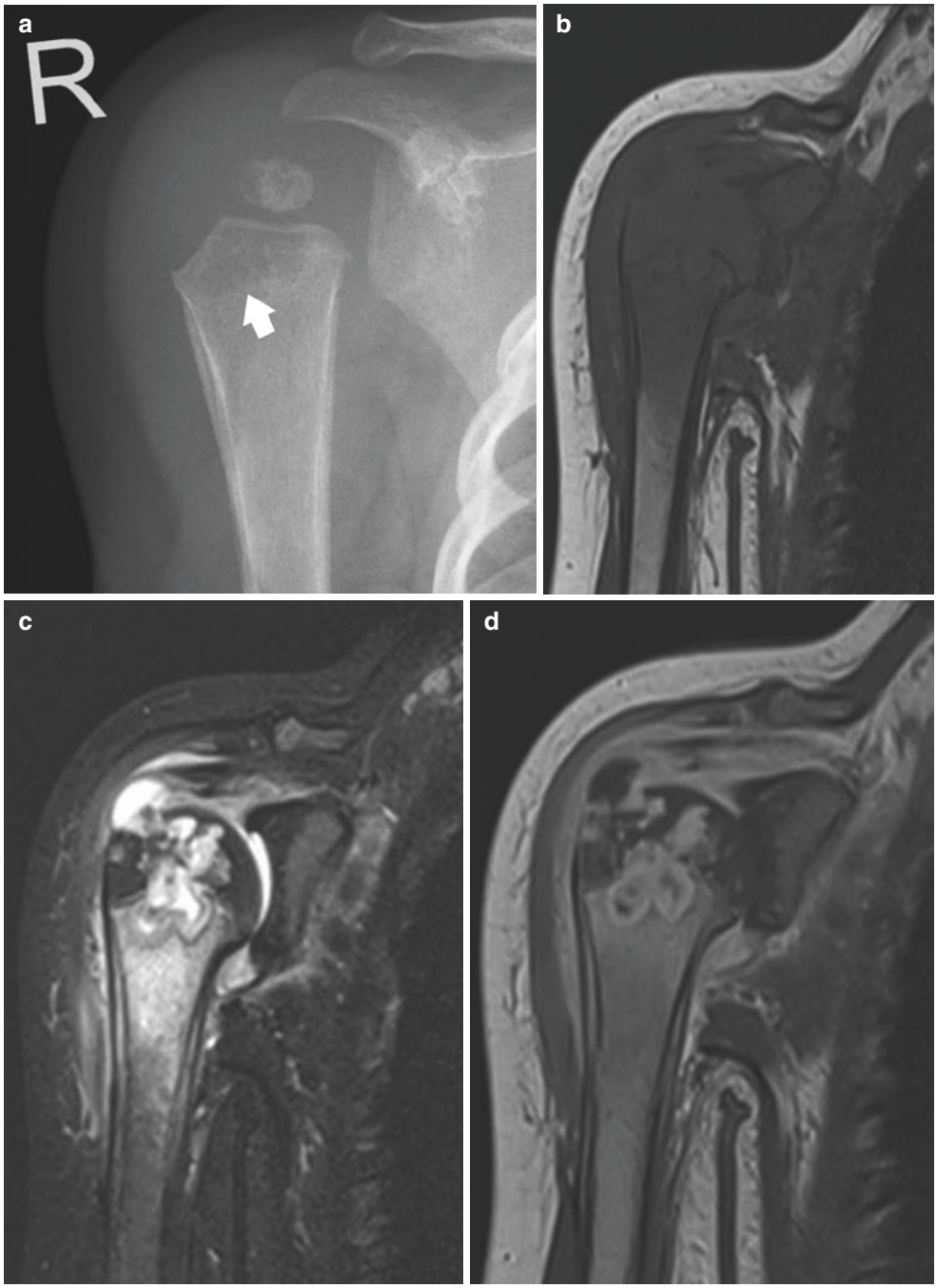


Fig. 14.40 Thirteen-month-old infant with since 4 weeks reduced use and motion of the right arm. (**a**) Radiograph of the shoulder shows a lytic ill-defined area in the proximal humerus metaphysis (arrow). (**b**) Coronal T1 MRI shows a lesion extending into the epiphysis. Coronal T2 (**c**) and coronal T1 after Gadolineum (**d**) show epi-metaphyseal osteomyelitis with concomitant synovial enhancement of the shoulder joint

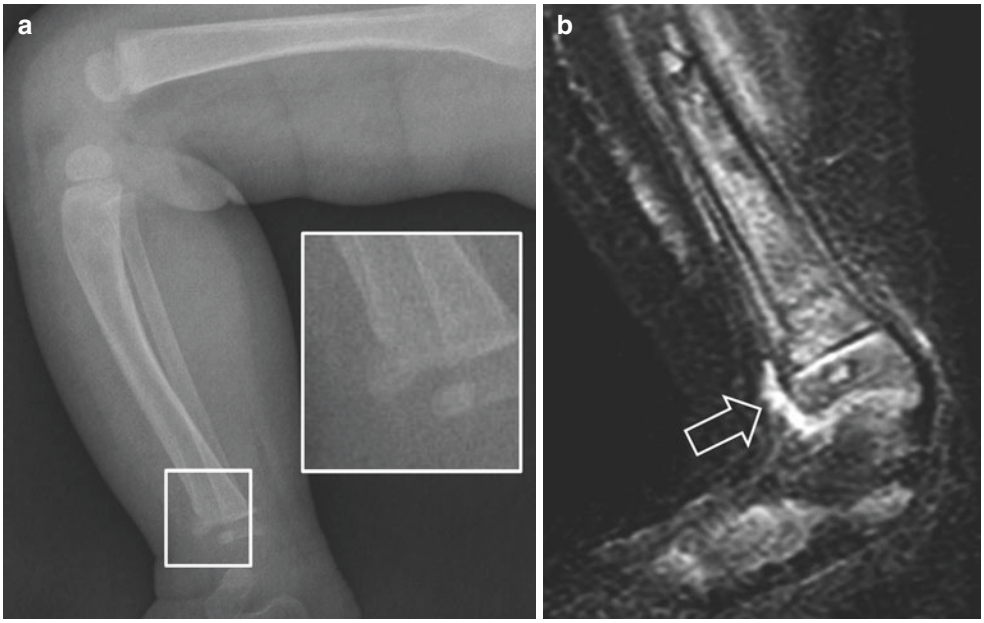


Fig. 14.41 Three-month-old infant with a painful lower leg, warm to the touch, clinically in keeping with osteomyelitis. (**a**) Lateral radiograph of the ankle shows a cortical irregularity described by a non-paediatric radiologist as a metaphyseal corner fracture (inset). (**b**) Sagittal T1 weighted MRI post-Gadolineum shows diffuse enhancement of the soft tissue and distal tibia (arrow). Follow-up imaging was consistent with evolving osteomyelitis

pathological fracture. Biopsy showed an *S. aureus* infection.

In meningococcal septicaemia, the epiphyseal plate may be affected. Initially, this will not show up in the radiological examination. The possible results will not be visible until a few years later: the epiphyseal plate will show central premature closure, which leads to a characteristic deformation (Fig. 14.42). However, this deformation may also be the result of an experienced trauma. In these cases, the clinical history is conclusive. Especially the slow progression of the clinical presentation may present the clinician with a diagnostic dilemma.

Fig. 14.42 Eight-year-old boy, who suffered meningococcal septicaemia at the age of 18 months. This resulted in premature partial closing of the epiphyseal plate (open arrow), resulting in joint deformation

14.8.3 Chronic Relapsing Multifocal Osteomyelitis

Chronic relapsing multifocal osteomyelitis is a disease that affects the metaphyses of the long bones, in particular in older children [174, 175]. Spine, pelvis, and shoulder girdle are also involved, but to a lesser degree (Fig. 14.43a, b).

The presentation of the patient will depend on the location of the inflammation, where despite the name also single bones (often the pelvis or long bones, especially of the lower extremity) can be affected. Systemic symptoms such as

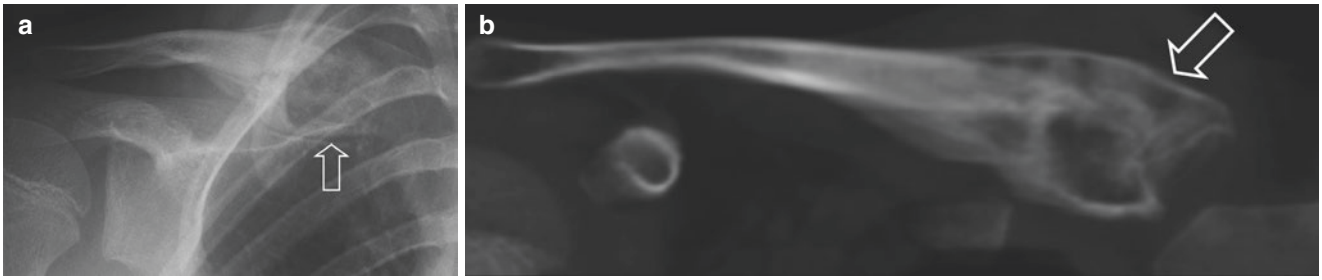


Fig. 14.43 (**a**) Eight-year-old girl with swelling of the proximal clavicle (open arrow). Later, a sacral focus was found in keeping with the diagnosis of chronic recurrent multifocal osteomyelitis. (**b**) CT of the clavicle (coronal reconstruction) clearly shows the sclerotic abnormality in the proximal clavicle

weight loss and fever are seldom seen [176]. Because of the lack of systemic symptoms, the periosteal reaction seen during the healing process may present a source of diagnostic dilemmas. The clinical history has a pivotal role in the diagnosis.

14.8.4 Congenital Syphilis

Over the past few years, and particularly in the United States, physicians have seen an increase in the incidence of syphilis in women of reproductive age. This may lead to an increase in congenital syphilis [139, 177]. In the differential diagnosis serological tests are often conclusive. Primary skeletal involvement is rare, but when present the findings tend to be bilateral polyostotic affecting primarily the long bones [178].

Solomon and Rosen described a series of 112 children with serologically confirmed congenital syphilis [179]. In these children, the bones most frequently affected were: tibia, femur, and ulna (Fig. 14.44). The most prevalent abnormalities on the radiographs are metaphyseal osteomyelitis and periosteal reactions. Pathological fractures of the metaphysis and periosteal new bone formation may mimic skeletal lesions seen in non-accidental trauma, and the image may even resemble lesions at various stages of healing. In the patient group of Solomon and Rosen, 31% of children had bone lesions corresponding with trauma, as described by Caffey [180]. Rasool and Govender described a series of 302 clinically suspected cases of congenital syphilis in whom bone changes were found in 192 children [181]. Of these 192 children periostitis was seen in 102 cases, osteitis in 20 and metaphyseal changes in 71 children. Sixty-one children showed a combination of two or more of these radiological abnormalities. Two children were lost to follow-up in the remaining 59 cases all radiological abnormalities normalized over time.

There have been several reports in literature of infants who presented with fractures initially thought to be the result

Fig. 14.44 Neonate with congenital syphilis. A radiograph of the knee shows a metaphyseal periosteal reaction in the distal femoral metaphysis (arrow). In the proximal tibia metaphysis a cortical radiolucency is visible, known as Wimberger sign (open arrow)

of non-accidental trauma. Jacobs et al. presented a case of an infant admitted twice at 3 and 4 months of age with long bone fractures and suspected non-accidental trauma [182]. Idrissi et al. presented a similar case of a 2-month-old adopted girl, of whom maternal information was unknown, who presented with a humerus fracture and diffuse osteolysis of the humeri and femora [183]. Lim et al. described a case

of an 8-week-old infant with congenital syphilis, who presented with numerous fractures which were initially thought to be inflicted [177]. The common finding in these cases was that the skeleton showed moderate to severe osteopenia.

14.9 Oncological Diseases

14.9.1 Introduction

Oncological diseases in childhood are relatively rare (168 in 1,000,000 children aged 0 to 14 years in the United States over the period 2013–2017) [184]. It is often forgotten that they occur significantly less frequently than skeletal dysplasias. Because they are so rare, they may present a diagnostic problem. Due to centralized treatment, which has an advantageous effect on the therapeutic result [185], radiologists not specialized in paediatric oncology will generally have limited knowledge of this topic, which may lead to problems when interpreting examinations.

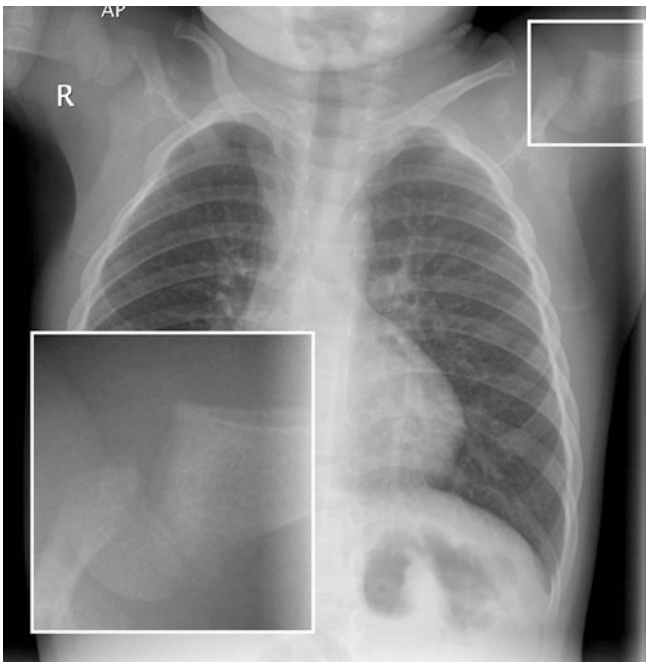


Fig. 14.45 Nearly 2-year-old boy with B-cell leukaemia. At the edge of the chest radiograph a periosteal reaction of the proximal humerus is visible (see inset)

14.9.2 Malignancies

14.9.2.1 Leukaemia

Leukaemia is the most prevalent oncological disease in childhood, with an estimated incidence of 50:1,000,000 in the Western world. In over 75% of cases it is acute lymphatic leukaemia (ALL). The clinical symptoms are generally due to decreased blood production: anorexia, pallor, fever, joint pain, haematomas and lymphadenopathy [186]. Generally, complaints will have been present for some weeks before the diagnosis is made.

Due to joint complaints, patients are regularly first referred to an orthopaedic surgeon, which is often followed by a radiological examination. The latter may show osteopenia, metaphyseal radiolucencies, periosteal reactions, osteosclerosis and pathological fractures or a combination of the above (Figs. 14.45 and 14.46) [119, 187]. When adequate clinical information is absent, the radiological manifestations may be hard to interpret. On the whole, when ALL is suspected, the diagnosis will be simple and fast.

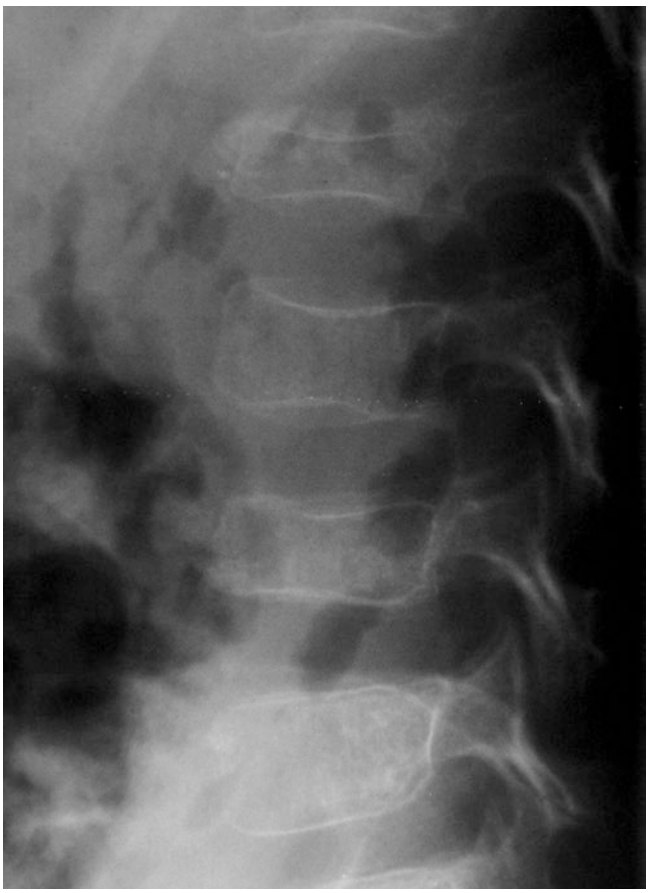


Fig. 14.46 Five-year-old boy with collapse of the spinal corpora in leukaemia

14.9.2.2 Ewing Sarcoma

Ewing sarcomas are predominantly seen in the second decade of life, and at that age will not present any diagnostic dilemmas. However, they may also present at a younger age and then, due to their radiological manifestation, they may cause confusion. Radiologically, Ewing sarcoma is an aggressive tumour that may show an erosive aspect as well as an abundant periosteal reaction (onion skin aspect) (Fig. 14.47) [188, 189]. The periosteal reaction in particular may cause confusion when incorrectly interpreted as an old fracture.

Fig. 14.47 'Onion peeling' in an Ewing sarcoma (open arrow). Especially when this is the only visible aspect of the tumour, there may be confusion with a healing fracture

14.9.3 Benign Diseases

14.9.3.1 Osteoid Osteoma

An osteoid osteoma is a small benign neoplasm, which is predominantly seen in the cortical bone of the long bones [190, 191]. The disorder is usually seen in boys and 50% of patients with this disorder are between 10 and 20 years old.

Over 50% of osteoid osteomas are located in the femur or tibia, mainly diaphyseal or diametaphyseal. The classical presentation is pain, mainly at night, which reacts well to acetylsalicylic acid. Pain complaints are related to hypervascularization of the osteoid osteoma, and prostaglandins probably have an important role, which would explain the adequate reaction to acetylsalicylic acid (prostaglandin synthetase inhibitor).

On radiographs, a cortical osteoid osteoma is visible as a small radiolucent focus (nidus) in a considerably widened sclerotic cortex, which is the result of a protracted periosteal reaction, often with a multi-layered aspect. This may mimic a healing fracture (Fig. 14.48a, b). However, it is seldom confused with non-accidental injuries, since the pattern of complaints is fairly typical and the age of the average patient usually leads to a reliable clinical history.

A small percentage of osteoid osteomas is not found in cortical bone, but in trabecular bone or subperiosteum, predominantly periarticularly. In this manifestation of osteoid osteoma, the sclerotic reaction is far less pronounced and confusion with non-accidental injury is possible.

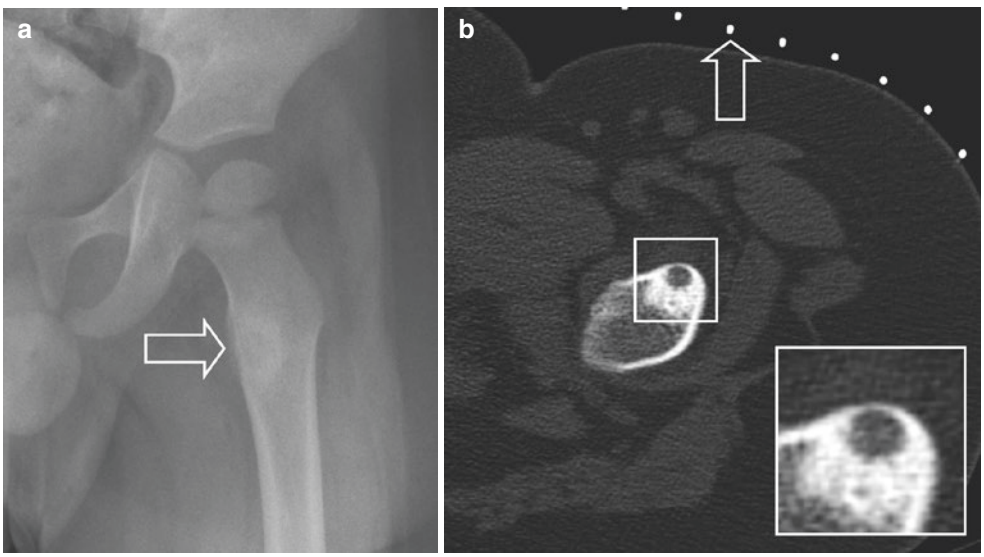


Fig. 14.48 (**a**) Ten-month-old boy with pain in the left leg. A radiograph of the femur shows cortical sclerosis (open arrow). Centrally a radiolucency can be seen. (**b**) A CT of the femur during radiofrequency ablation (see markers on the skin—open arrow) shows a cortical radiolucency with a central nidus (see inset)

14.9.3.2 Bone Cysts

Although rare in infancy, cystic bone lesions, either simple bone cysts (SBC) or aneurysmal bone cysts (ABC), do occur. Both entities have a predilection for the proximal metaphysis of the humerus and femur [192, 193]. Due to their location and expansive growth these tumours can lead to pathologic fractures.

Simple bone cysts are cystic, fluid-filled lesions, which in general are unicameral but may show septation [192]. The most common presentation is a pathologic fracture. The imaging findings, a lytic expansile lesion on conventional imaging with, if a fracture is present, a 'fallen fragment' and a cystic lesion on MRI are pathognomic for this entity (Fig. 14.49).

Aneurysmal bone cysts are benign blood-filled bone tumours which show growth, and consequently expansion of bone, over time [192, 194]. In a large US study of 238 patients the youngest patient was 18 months (mean, 16.1 years) [195]. Although pain and swelling are the most common complaints, pathologic fractures are not uncommonly a presenting symptom. The imaging findings, a lytic expansile lesion on conventional imaging and on MRI a multicystic tumour with multiple fluid-fluid levels, are almost pathognomonic for this diagnosis (Fig. 14.50a–d). Both in SBC as well as in ABC the diagnosis of a pathologic fracture should be relatively straightforward.

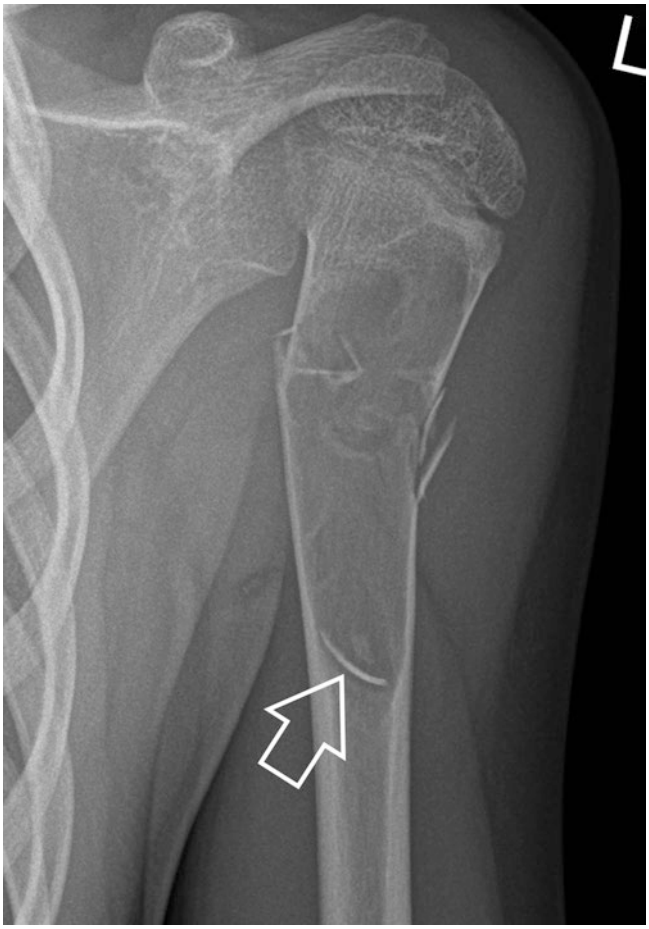


Fig. 14.49 Seven-year-old child with a pathologic fracture of the left proximal humerus. The humerus shows a fracture through a lytic lesion in the proximal metaphysis and diaphysis, there is a fallen fragment within the cyst (arrow). This finding is pathognomonic for a solitary bone cyst (SBC)

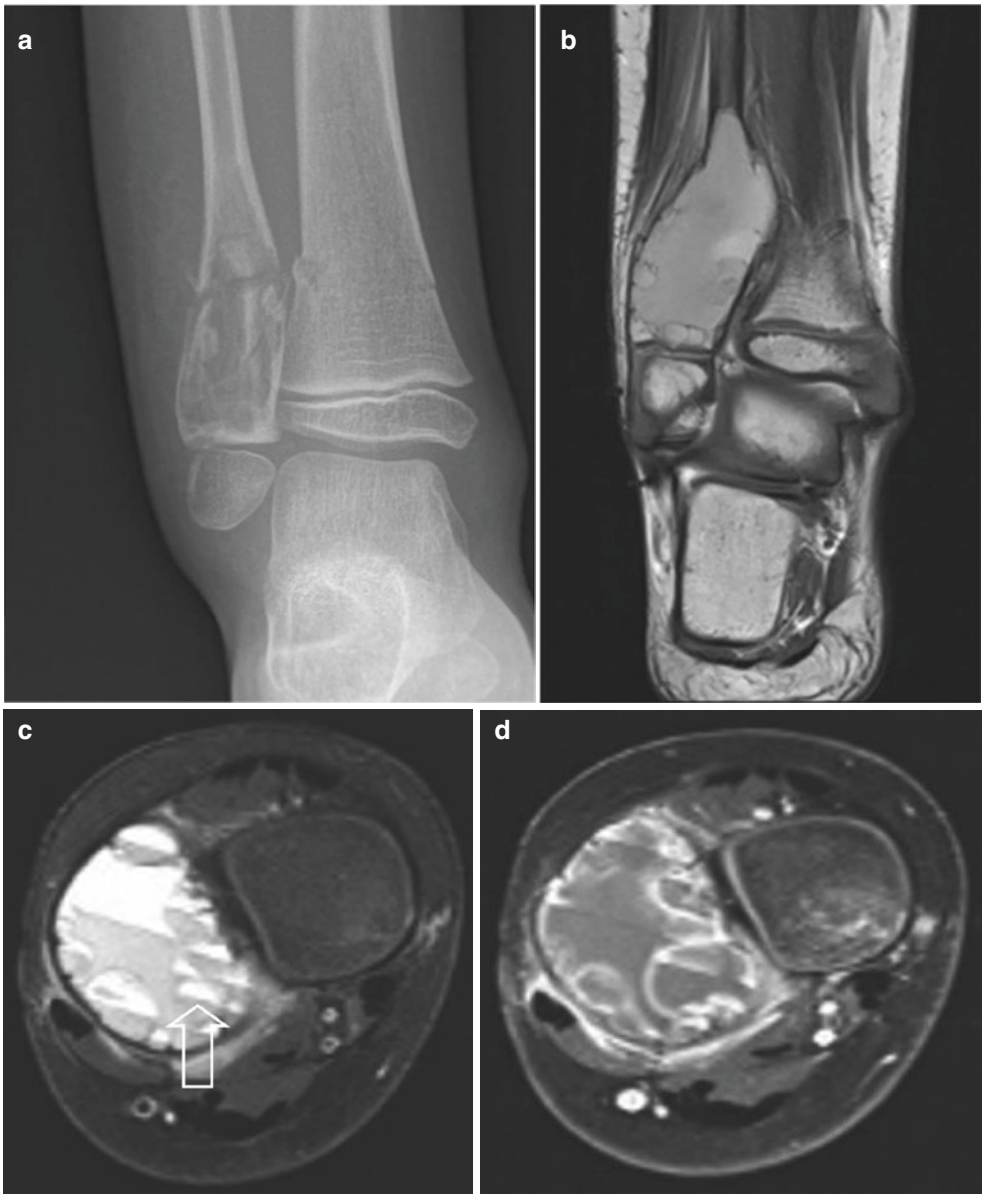


Fig. 14.50 Six-year-old child who sustained a right tibia and fibula fracture while playing in a playground. (**a**) conventional radiography shows a linear fracture through the distal tibia and a pathologic fracture through an expansive lytic lesion in the distal fibula. (**b**) Coronal T1-weighted MRI shows an expansile multicystic lesion in the distal fibula. (**c**) Axial T2-weighted MRI shows multiple fluid-fluid levels (arrow) after administration of Gadolineum (**d**) septal enhancement is seen. The imaging findings, in the absence of a solid component are pathognomonic for an aneurysmal bone cyst (ABC)

14.10 Medication-Related Abnormalities

14.10.1 Introduction

When evaluating the radiological examination of children, the clinical history is generally known. However, the radiologist should also take the use of medication into consideration. Several medications may influence bone development and growth. These medications have not always been prescribed by physicians, and accordingly some cannot be recorded in the medical dossier.

14.10.2 Corticosteroids

In childhood there are a number of indications for the use of corticosteroids, such as asthma, juvenile rheumatoid arthritis, inflammatory bowel disease (IBD) and organ transplantations. Corticosteroid use may lead to disturbances in bone mineralization. Protracted use may cause osteoporosis [196, 197]. The primary mechanism of corticosteroid-induced osteoporosis is decreased bone formation. Even in childhood this may result in insufficiency fractures (Fig. 14.51).

Fig. 14.51 Ten-year-old girl with IBD, for which she was treated with prednisone. As a consequence of the therapy corticosteroid-induced osteoporosis developed, which resulted in multiple vertebral fractures (open arrows)

14.10.3 Methotrexate

Methotrexate-induced lesions are characterized by osteopenia (in particular in the lower extremities), dense metaphyseal banding, growth retardation, and metaphyseal fractures that may strongly resemble metaphyseal corner fractures [198, 199]. However, these lesions only occur after protracted use in relatively high doses [200].

14.10.4 Hypervitaminosis A

Children with hypervitaminosis A may present with a great variety of clinical complaints such as anorexia, pruritis, lip fissures, stiff joints and bone pain, multiple nodular soft tissue swelling, alopecia, and hepatosplenomegaly [201]. From a radiological point of view it manifests as the result of hypercalcaemia, in particular by periostitis, in which the ulna is most frequently affected [201]. This periostitis, which in severe cases is palpable, may suggest an experienced trauma.

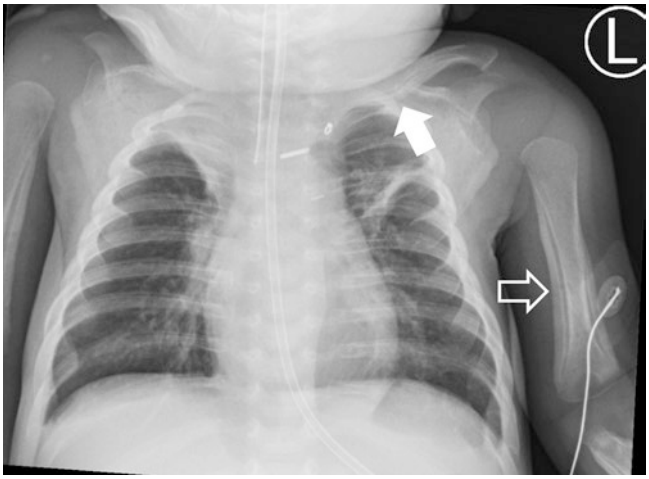


Fig. 14.52 Two-month-old infant was treated with an interrupted aortic arch, for which prostaglandin treatment was initiated. The chest radiograph shows periosteal reaction of the clavicle (arrow) and the humerus (open arrow)

In these cases the clinical history should provide the answer. Hereby one should be aware that vitamin A, and products in which it is present in a relatively high dose, is generally freely obtainable. Consequently, parents/carers may inadvertently give their child an overdose [202–204]. But also administered in a medical setting, for example as adjuvant therapy in children with neuroblastoma or vitamin A deficiency, hypervitaminosis A has been reported in exceptional cases [205, 206]. In the literature, there is one case that reports on an iatrogenic overdose in an autistic child. His parents put him on a special 'autism diet' that contained extremely high doses of vitamin A: 100,000 IU/day for 3 months, followed by 3 months of 150,000 IU/day (the recommended dose of vitamin A is 1200–1600 IU/day) [207, 208]. When these cases present, determining blood values may clarify matters, although this may also provide a false-negative result [209].

14.10.5 Prostaglandins

Prostaglandins are used in neonates with duct-dependent congenital heart disease to bridge the time to operation [210]. Treatment with prostaglandins may cause periosteal reactions in the long bones (Fig. 14.52) [211]. Generally, it takes 30–40 days for these changes to present, although it has been reported as early as 9 days after treatment was initiated [212]. The periosteal changes may be sufficiently pronounced to resemble Caffey's disease (see Sect. 14.5.6); however, in prostaglandin treatment the mandible is spared.

14.10.6 Bisphosphonates

Bisphosphonates are derivatives of pyrophosphate and bind to the bone surface. In bone resorption the osteoclasts absorb the bisphosphonates. Depending on the presence of nitrogen atoms, the osteoclasts are inhibited. When no nitrogen atoms are present (clodronate and etidronate), there will be interference with the energy supply of the osteoclast, leading to apoptosis [213]. In the more potent nitrogen-containing bisphosphonates (alendronate, ibandronate, and residronate) inhibition of the essential proteins in the osteoclasts is seen, which will also result in apoptosis [214].

Bisphosphonates have been developed for therapeutic use in postmenopausal women. Due to their optimal effect in ideal circumstances (efficacy) and their minimal side effects, they are at the moment the medication of choice [215]. However, paediatric medicine can also accommodate treatment with bisphosphonates, such as in osteogenesis imperfecta, fibrous dysplasia, idiopathic osteoporosis, juvenile idiopathic arthritis and in some children with tumours [216–219]. In children these medications are often administered intravenously, resulting in the characteristic image of successive growth lines corresponding with the number of treatments (Figs. 14.53, 14.54a, b, and 14.55a, b) [220]. When a radiologist is not familiar with this image, he/she may consider failure to thrive as a diagnosis. However, in day-to-day practice this should not be a problem (see Sect. 12.11).

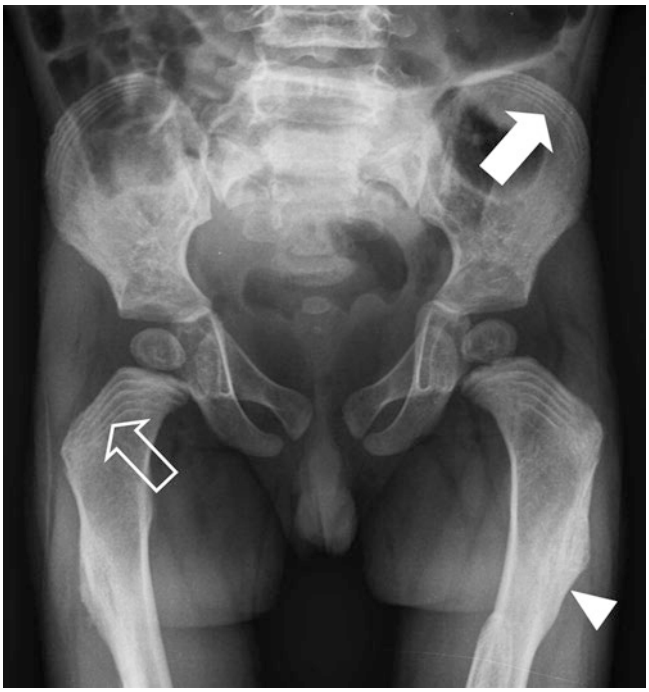


Fig. 14.53 Patient from Fig. 14.11 a year after treatment with intravenous pamidronate (a bisphosphonate). The pelvic film shows sclerotic bands in both proximal femurs (open arrow) and near the iliac crest (arrow). Every sclerotic band equals an intravenous treatment. Healing retardation of the left femoral fracture is also visible (arrow point)

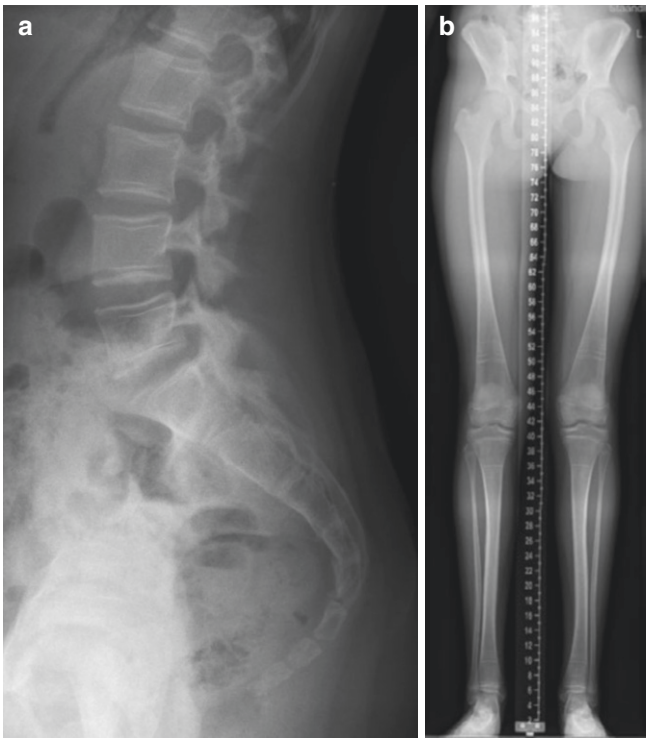


Fig. 14.54 Thirteen-year-old child on bisphosphonate treatment because of OI type I. (**a**) Radiograph of the lumbar spine shows a bone in bone configuration. (**b**) Leg length radiographs show multiple growth lines

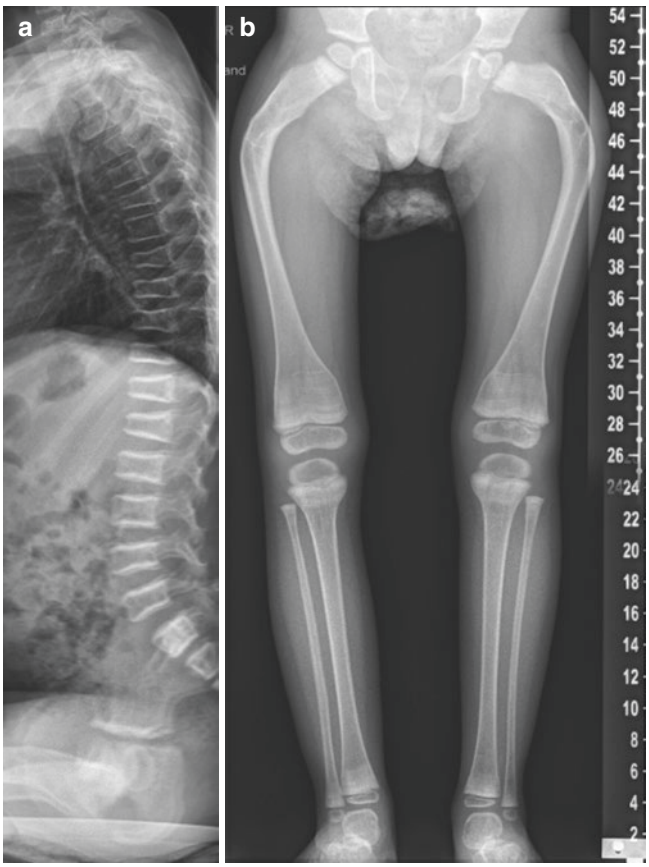


Fig. 14.55 Five-year-old child on bisphosphonate treatment because of OI. (**a**) Lateral radiograph of the spine shows multiple vertebral fractures and sclerotic vertebral end plates. (**b**) Leg length radiographs show bowing of the proximal femora and dense sclerotic metaphyses of the distal femora and proximal tibiae

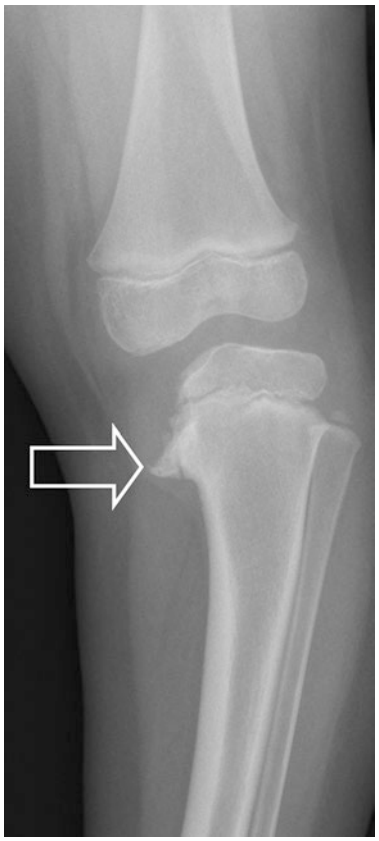


Fig. 14.56 Four-year-old girl with genu vara. Radiological examination shows a strong deviant aspect of the proximal metaphysis of the tibia (open arrow). Since this girl is well over 2 years old, this should not cause a diagnostic dilemma

14.11 Other Disorders

14.11.1 Blount's Disease

Blount's disease (OMIM 188700) is a deformity on the medial side of the proximal metaphysis of the tibia leading to genu vara [221, 222]. In the more serious cases the metaphysis of the distal femur is also affected, but to a lesser degree. Generally, it is assumed that this is a subclinical compression injury. It is more frequently seen in young children with preexisting tibia vara, in which case the medial side is already overloaded [223]. The compression leads to growth defects at the medial side of the tibia, increasing the tibia vara. A variant at adolescent age has also been reported, and was associated with obesity [224, 225]. The disease may present at both ages bilateral as well as unilateral.

The radiological image shows a deformity of the medial metaphysis of the tibia with irregular margins; the metaphysis has been displaced downwards; the pointed end of the metaphysis is directed downwards (Fig. 14.56). This may cause fragmentation of the metaphysis. This image could be interpreted incorrectly as a metaphyseal avulsion fracture in a non-accidental injury; however, the fact that these children are much older and have been known to have bow legs for some time should be sufficient to reject this erroneous diagnosis.

14.11.2 Epilepsy

Patients that are affected by epilepsy and/or spasticity are at increased risk for fractures. The literature provides several reasons: accidental trauma during epileptic seizure (e.g. as result of a fall), non-epilepsy-related accidental trauma, the seizure itself, decreased bone density due to inactivity and anticonvulsant drugs, increased muscular tone with contractures, and decreased muscle mass. Prasad et al. calculated that the relative risk of sustaining any fracture in children and young adults (1–21 years) with epilepsy (person years at risk = 143,336) compared to those without (person years at risk = 56,310) was 14.4 (95% CI: 13.8–15.0) [226].

In children, epilepsy is one of the most prevalent neurological anomalies [227]. It may occur isolated but is often seen combined with spasticity. The fracture rate in epileptic patients is three times that of the general population [228, 229]. The fractures are mostly fall-related and associated with an epileptic seizure. This risk increases in the presence of more risk-increasing factors such as spasticity and decreased bone density.

Sheth et al. report four mechanisms that apply to epilepsy-related fractures: the seizure itself or a fall related to the seizure, accidental trauma not related to the seizure, and a pathological fracture resulting from decreased bone density [228]. In newly diagnosed patients, the incidence of seizure-related fractures was very low, 5% [230].

Fractures caused by the seizure itself are rare, but can occur and have mainly been described in adult patients [231–237]. However, there are also paediatric cases to be found in literature. Paris et al. describe a case of a boy who developed epilepsy at the age of 2 months [238]. At the age of 9 months he was admitted for adjustment of his medication and during admission he suffered four major generalized seizures. After these seizures his left hip was less mobile and on radiography a slipped capital epiphysis was seen. One month later a similar episode led to a right-sided slipped capital epiphysis. Aoudi et al. described a case of a 5-month-old boy with a history of epilepsy, who after a tonic/clonic convulsive insult developed a painful left hip [239]. Radiography showed a slipped capital epiphysis, the diagnosis was confirmed using MRI. Atmaca et al. described the case of an 11-month-old boy who has a history of convulsive episodes as a result of hypoxic postnatal encephalopathy [240]. After a seizure, he developed pain in his right hip and after having been treated by the general practitioner for septic arthritis, he was referred after 1.5 months. On physical examination, he held his right leg in abduction and external rotation and on radiography a diagnosis of missed slipped capital epiphysis was made. Based on the clinical history it was decided that this was caused by the epileptic seizure. Ballal et al. describe two cases of epileptic seizure-induced fractures of the proximal femora [241]. The first was a girl of 22 months who after a tonic-clonic seizure remained restless and had an abnormal position of her right hip, radiography showed a slipped capital epiphysis. The second case was a 9-year-old boy, with a history of grand mal seizures and a previous right-sided proximal femoral fracture, who after a tonic-clonic insult developed a deformation of the right hip. On radiography, a transepiphyseal fracture of the left proximal femur was seen as well as a subtrochanteric fracture of the right femur distal to a plate, which had been inserted 3 years previously. Jacoby et al. describe a case of a preterm male neonate who a few hours after delivery developed a tonic-clonic insult [242]. Chest radiographs on days 1 and 3 of life showed no signs of birth-related trauma, but a chest radiograph on day 18 of life revealed bilateral acromial fractures with callus formation. The authors postulate that these fractures were the result of the tonic-clonic insult.

Schnadower et al. describe bilateral femur fractures in an adolescent with primary vitamin-D deficiency due to a hypocalcaemic seizure [243]. Presedo et al. pose that 2% of all fractures in spastic children may be the result of a seizure, but refrain from reporting whether it concerns a fracture by the seizure itself or a fracture due to seizure-related trauma (fall) [244].

In children with normal bone density (no anti-epileptica or inactivity osteopenia), no fractures of the extremities due to notably increased muscle tone during a seizure are known.

The prevalence of fractures in spastic children with an average age of 10 years is 6%, of which 45% has no identifiable cause, 32% is due to trauma, and 11% is caused by medical proceedings or physiotherapy [244]. It usually concerns the lower extremities (82%). The main risk factors are immobility, osteoporosis, and the use of anti-epileptica. Lingam et al. described 5 spastic patients (10–19 years old) with five femur fractures and one cruris fracture without identifiable causal incident [245]. In this case, the authors maintain it was a combination of inactivity osteoporosis, increased muscle tone with contractures and decreased muscle mass. Anti-epileptica, such as phenobarbital, phenytoin, primidone, valproate, carbamazepine, and oxacarbazepine cause decreased bone density [227, 246, 247]. Babayigit demonstrated this in 68 children who had been on anti-epileptica for over a year [247]. Sheth et al. found a pathological fracture due to reduced bone density in 15% of fractures in epileptic children [228].

There are no comprehensive studies known on fractures in spasticity and/or epilepsy in the age group up to 2 years old.

In conclusion, one may pose that patients with epilepsy and/or spasticity are at higher risk of fractures, especially of the lower extremities, in relatively minor trauma and sometimes even without identifiable cause. Of course, this does not imply that there are no non-accidental injuries in this patient group. In each patient that presents with a fracture, this subject must be open for discussion. After all, intentional or unintentional negligence in the medical treatment or care in these often institutionalized, fragile patients can also be considered non-accidental injuries. Therefore, when fractures are present in epileptic children of less than 2 years old without comorbidity, also non-accidental trauma should be considered.

14.11.3 Vitamin C Deficiency

Infants are protected from congenital vitamin C deficiency by vitamin C storage in utero. As vitamin C is not stored in

the body dietary intake is essential. Neonates deplete this storage when after birth they receive vitamin C-deficient artificial nutrition [248, 249]. When post-partum there is total vitamin C deprivation, it will still take at least 5 months before the supply has been depleted. The first signs of scurvy will occur after 1–3 months of insufficient intake of vitamin C [250]. Since a severe vitamin C deficiency in pregnant women results in early abortion, congenital vitamin C deficiency is unknown [251].

Scurvy nowadays is hardly ever seen in children, especially in the Western industrialized world [251, 252]. In the last decades only a few cases have been reported, with cases related to dietary regimens or underlying diseases [253–268]. In non-Western and non- or less industrialized countries, case reports and announcements on epidemics still surface regularly [269–277].

Vitamin C has a catalyzing role in the formation of collagen and many symptoms of vitamin C deficiency result from a disturbance in collagen formation: impaired wound healing, increased fragility of capillary walls, and osteoporosis. If a child already has incisors (at 7 months usually the incisors of the mandible), haemorrhagic areas will be found at their base. The gums are swollen. In adults, teeth may fall out when the deficiency has been present for a protracted period, which will reduce the state of the gums even further.

Even in the early stages, the radiological images are rather characteristic: a dense widened zone of provisional calcification (Frankel line), an adjacent hyperlucent line (Trümmerfeld zone), the Frankel line can show a spur (Pelkin spur) which can also resemble a metaphyseal corner fracture, and 'ringed' epiphyses (Wimberger rings) and slight osteoporosis (Fig. 14.57). At a later stage, examination may show swelling of the ends of the long bones, in particular the distal ends of the femur. These swellings are due to subperiosteal haemorrhages that will only in time be visible on radiographs. Externally, a shiny, livid (blue-black) skin will be visible at the location of the swelling. Gulko et al. described the findings of scurvy on MRI in four children [261]. On T1-weighted imaging the metaphyseal bone marrow showed a low signal intensity and on T2-weighted imaging a high signal intensity. After administration of Gadolineum bone marrow enhancement can be seen. Periosteal elevation along the metaphyseal cortex is also seen. Other authors also have described MRI findings of scurvy with cases where imaging findings even resemble osteomyelitis [257, 267, 268]. The elevation of the periosteum can not only be seen on MRI but also on ultrasonography [264]. Bone involvement in scurvy typically is symmetrical, just like in rickets [278]. For an overview of the clinical manifestations, the reader is referred to Table 14.10. The development of the clinical spectrum over time is shown in Fig. 14.58.

In 2014, the American Academy of Pediatrics issued a clinical report concerning the evaluation of children with

Fig. 14.57 Vitamin C deficiency (scurvy). The radiograph shows an evident osteopenia with the characteristic hyperdense zone of provisional calcification (Frankel line) and the relative dense outer edge of the epiphysis (Wimberger ring—open arrow)

fractures [279]. According to this report, vitamin C deficiency can result in metaphyseal lesions, resembling metaphyseal corner fractures. However, according to this report, *'other characteristic bone changes, including osteopenia, increased sclerosis of the zones of provisional calcification, dense epiphyseal rings, and extensive calcification of subperiosteal and soft tissue hemorrhages, will point to the diagnosis of scurvy'*.

The clinical symptoms of vitamin C deficiency can be severe and the clinical course can be dramatic. Sometimes the clinical symptoms are limited without even showing classical scurvy symptoms.

Vitoria et al. reported a child with scurvy, who was fed exclusively with almond beverages and almond flour between the age 2.5 and 11 months [280]. The child was admitted to the hospital with a fracture of the distal left femur and periosteal reaction on the right femur, irritability, and failure to thrive. He had typical radiologic signs of scurvy (osteopenia,

Table 14.10 Summary of clinical manifestations of scurvy [281]

Organ system	Signs and symptoms	Differential diagnoses
Skin	Hyperkeratosis Perifollicular haemorrhages Ecchymoses Purpura/pseudovasculitis Splinter haemorrhages Poor wound healing Nail changes Corkscrew hair Alopecia	Cutaneous vasculitis Bleeding disorders Zinc deficiency Systemic connective tissue diseases
Ocular	Conjunctival haemorrhage Retinal haemorrhages	Bleeding disorders Accidental trauma/injuries Non-accidental trauma Retinal vein occlusion
Oral	Bleeding gums Gingivitis Gingival hypertrophy Necrotic gums Loosening of teeth	Bleeding disorders Haematologic malignancy Granulomatous diseases Periodontal disease
Musculoskeletal	Myalgia and arthralgia Limb pain Weakness Extremity swelling Subperiosteal haemorrhage Hemarthrosis Intramuscular hematoma Osteopenia/osteoporosis	Juvenile rheumatoid arthritis Connective tissue diseases Multifocal osteomyelitis Hematologic malignancy Neuroblastoma Bone tumours Bleeding disorders Non-accidental trauma Rickets Neuromuscular diseases
Haematology	Anaemia Bleeding tendency	Bleeding disorders Haematologic malignancy
Neuropsychiatric	Apathy and irritability Intracerebral bleeding Subdural haematoma	Bleeding disorders Accidental trauma/injuries Non-accidental trauma
Constitutional	Fatigue Failure to gain weight Loss of appetite	Endocrine/metabolic diseases Haematologic/oncologic diseases Immune deficiency Chronic infections (HIV, tuberculosis etc.)

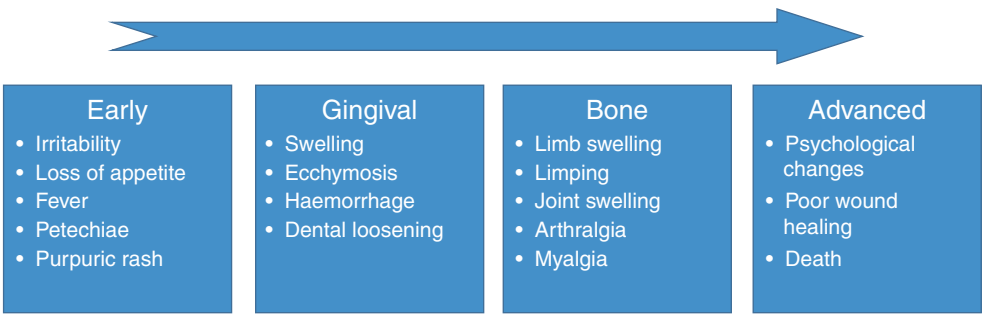


Fig. 14.58 The development of the clinical spectrum of scurvy in time (adapted from [278])

cortical thinning, Wimberger ring, Frankel line, fracture, and periosteal reaction). His plasma vitamin C level was very low. Vitamin C suppletion was started and over the following 3 months, his general condition, the pain in the legs, and the radiologic features improved, the plasmatic vitamin C level was normalized and the child started walking.

Kitcharoensakkul et al. reported 3 children, aged 5 years (one girl and 2 boys) with lower extremity pain and refusal to walk [281]. Only one child had gingival lesions on the initial presentation. They were finally diagnosed with scurvy/vitamin C deficiency, due to extremely limited intake of fruits and vegetables. Vitamin C suppletion was started and all children recovered completely and started walking again.

Ceglie et al. described the findings in three previously healthy children, who were referred for leg pain and refusal to walk, due to scurvy [282]. The complaints had started months

before and subtly advanced. Two children had gingival hyperplasia and petechiae, one child reported night sweats and gingival bleeding in the past few weeks. A nutritional screening revealed low or undetectable levels of ascorbic acid.

Lund et al. reported a previously healthy 3-year-old girl, who presented at a rheumatology department for abnormal gait [283]. She had also developed lower extremity weakness and was admitted to the hospital. The laboratory evaluation showed that her vitamin C level was undetectable. Analysis of her diet showed that she refused to eat fruits and vegetables. She had no other characteristic physical findings of scurvy. Vitamin C suppletion was started and the girl had an immediate and complete recovery.

Liebling et al. reported a 12-year-old boy with anorexia nervosa, who presented with asymmetric painful swelling of multiple fingers of both hands [284]. Imaging demonstrated soft tissue and bone marrow oedema of several phalanges, without arthritis, concerning an inflammatory process. Laboratory evaluation showed an extremely low vitamin C level and a moderately low vitamin D level. Vitamin C suppletion was started and within 3 weeks digital abnormalities resolved on physical exam.

In 1932 Gilman and Tanzer stated *'The hemorrhagic diathesis of scurvy has been recognized since the time of Hippocrates. Hemorrhage occurs most commonly beneath the periosteum of the long bones and into joint spaces but frequently involves the skin, mucous membranes, orbits and serous cavities. Reports of hemorrhage associated with the meninges during the active scorbutic state are sufficiently rare to deserve note'* [285]. They found 13 cases of intracranial haemorrhages, due to scurvy, since the first description in 1668: *'Seven being subdural, three extradural and three unclassifiable. Five of the seven cases of subdural bleeding were found in infants or young children'*.

Miura et al. reported a 5-month-old girl, who was admitted to the hospital with *'bleeding tendencies such as purpura, vomiting and bulging of the anterior fontanelle. On admission coagulation studies including bleeding time, PT, APTT, platelet aggregation (ADP, collagen, epinephrine and ristocetin) and so on, revealed no abnormal findings except positive Rumpel-Leede test. Roentgenograms of the lower extremities showed subperiosteal hemorrhage, thinning of cortex and a scurvy line. Subdural hematoma was found in CT-scanning and she was diagnosed as scurvy with subdural hematoma. We performed an operation of a subdural-peritoneal shunt and prescribed vitamin C. The prognosis was good. Since it was found that the formula milk for this baby had been prepared with boiling water, the level of vitamin C was assayed. The result revealed that the level of vitamin C in the formula with boiling water was decreased to 42.6% of the original source'* [286].

Verma et al. reported a 3-year-old boy, who presented with proptosis of the left eye with ulceration for 15 days [287]. He was not able to walk anymore for the last 2 months, due to painful swelling of the right thigh. He was fed with diluted cow's milk and rice since late infancy and lacked fruit and vegetables. He was extremely irritable and undernourished with weight, height, and head circumference well below the third percentile. Costochondral junctions were sharply angulationed. Radiological examination of the right femur showed a thin cortical outline of the epiphysis, splayed metaphysis with evidence of a Pelkan spur, submetaphyseal lucencies in the distal femur and subperiosteal haemorrhage. MRI of the brain showed bilateral extradural haematomas compressing the frontal lobes. There was a subperiosteal haematoma in the left orbit along with subretinal haemorrhage and retinal detachment. He was treated with vitamin C and on follow-up 8 weeks later had recovered with no evidence of the orbital mass on clinical or radiological study.

Aziz et al. described the findings in an 11-month-old child who presented with a 2-week history of spontaneous superior left lid oedema associated with alteration of the general condition [288]. Ophthalmologic examination found limitations to movement in all fields of gaze in the left eye. The left eye fundus showed signs of extrinsic compression with vascular tortuosity, moderate papillary oedema, retinal folds between papilla and macula, and loss of foveal reflex. A skeletal survey showed metaphyseal fractures of both thigh bones associated with a bilateral calcified subperiosteal hematoma, and Pond's fracture. Stomatological examination revealed parodontopathy and dermatological assessment found numerous bruises of different ages. Initially, non-accidental trauma was suspected, but the child's diet revealed a very low vitamin C intake. The laboratory evaluation revealed a severe vitamin C deficit. Vitamin C was supplemented and the child's condition improved within a few days.

Retinal haemorrhages, due to vitamin C deficiency, have been described in adults, but as far as we know, have never been reported in children [289–291].

14.11.3.1 Vitamin C Deficiency and Suspected Non-accidental Trauma

In children with vitamin C deficiency the symptoms and physical findings, especially bruising and skeletal findings, initially may be mistaken for inflicted injuries, due to non-accidental trauma.

In 1966 Berant and Jacobs described the findings in a boy, aged 2 years and 8 months [292]. He was admitted to the hospital because of tender swelling of both legs, left shoulder, and left arm, which had started a few days before the admission. The boy had been ill for about 3 months before the admission with unexplained high fevers (initially intermittent, later continuous) and increasing lethargy and pallor. At the same time, he began to limit his activities and became more irritable. He started crying, when being handled. On

admission he looked neglected and was pale. During physical examination it was noticed that the costochondral junctions were prominent as in a rachitic rosary. The left shoulder and left arm were swollen and very tender. While manipulating the arms crepitations were felt, which gave the clinical impression that a fracture was present. The thighs were swollen and very tender along their entire length. Pitting oedema was present on both legs and feet. The neurologic examination was normal except for the lack of spontaneous movement, the peculiar resting position, and his limited vocabulary. The differential diagnosis consisted of sepsis, battered child, leukaemia, syphilitic pseudoparalysis, Still's disease (systemic onset juvenile rheumatoid arthritis), and nutritional deficiency disease. According to the authors his posture, the extreme tenderness, the oedema, and the crepitus were possibly due to an epiphyseal fracture but could also be due to scurvy. The radiological examination showed an increased density of the epiphyseal lines and cortical thinning in the vicinity of the epiphysis, and calcifying periosteum over a subperiosteal haematoma around the left humerus. It was concluded that the child had multiple nutritional deficiencies, of which vitamin C deficiency was the most dominant. The boy initially was breast-fed, but after 6 months the breast-feeding was stopped. After that the boy would only eat milk and porridge. Vitamin C suppletion was started together with oral iron, multivitamin preparations, and a blood transfusion. The boy's health gradually improved. If a vitamin C deficiency is diagnosed in a child, one should always analyze the circumstances under which the deficiency occurred. In some children the deficiency is the result of severe neglect.

Mimasaka (2000) reported death due to vitamin C deficiency in a 6-year-old girl [293]. Because the girl had extensive bruises, initially death was suspected to be due to non-accidental trauma. During the forensic autopsy, many bruises were seen on the face, on the torso, and lower extremities. The gums were swollen and some teeth were missing. Subperiosteal haemorrhaging was found on the humerus, tibia, and femur. Ultimately, the findings on autopsy could be explained as the result of a serious and long-term vitamin C deficiency. Mimasaka concluded that the death was not due to non-accidental trauma but to serious neglect. The parents locked up the child, whenever they left the home, the parents did not seek medical attention, when the child got ill, and the child was not fed properly. No intracranial haemorrhages were found in this child.

According to Greeley (2011) subdural and retinal haemorrhages have been described to occur due to vitamin C deficiency, but whenever these haemorrhages were vitamin C related, other clinical features characteristic of scurvy were always apparent [294]. Greeley did not find any reports in the medical literature of infants whose only manifestations of scurvy were subdural and retinal haemorrhages, without cutaneous bruising, gingival changes, or bone and joint findings on examination or radiograph. He is of the opinion that a diagnosis of scurvy in children with subdural and retinal haemorrhages is purely speculative if other findings, belonging to the clinical spectrum of symptoms are absent.

Although the clinical symptoms of scurvy can be severe and scurvy is a potentially fatal disease, it is easily curable with ascorbic acid (vitamin C) and the response to suppletion of vitamin C usually is rapid and dramatic [265, 295, 296].

14.11.4 Copper Deficiency

Due to the skeletal findings, copper deficiency can be considered as a true mimic of inflicted injuries (a disorder, which simulates on imaging a fresh or a healing/healed fracture). Metaphyseal spurs, suggesting metaphyseal corner fractures, and periosteal reactions, suggesting healing fractures have been described in children with copper deficiency. However, in children with copper deficiency there is also a real increased risk of fractures, because of weakening of the bone (osteoporosis) due to disturbances in the bone metabolism and fractures may occur due to trauma. For that reason copper deficiency may mimic inflicted fractures (Sect. 2.3). Marquardt et al. reported two extremely low birth weight preterm infants with complicated medical courses requiring prolonged parenteral nutrition for short-gut syndrome. This led to the development of cholestasis [297]. Both children never left the hospital after birth. Between 5 and 6 months of age, they developed signs of copper deficiency, which initially led to a suspicion of non-accidental trauma. Radiographic findings (osteoporosis, metaphyseal changes, and physeal disruptions), however, indicated metabolic bone disease. Copper levels were low.

Copper deficiency in children was first described in the fifties of the twentieth century [298, 299]. It is a rare disorder and it was initially only reported in infants with malnutrition and after prolonged parental nutrition [300–302]. In the 1970s, pure copper deficiency (copper deficiency without malnutrition or prolonged parental nutrition) was described for the first time [302, 303]. According to Carty up to 1988 copper deficiency had never been described in infants who were fed exclusively breast milk or in full-term infants who were fed a formula known to contain adequate amounts of copper [304]. Carty also mentioned that since 1983 most, though not all, formula milk freely available in the United Kingdom contained enough copper to prevent dietary copper deficiency.

Cordano et al. were probably the first to describe skeletal findings (*'scurvy-like bone changes'*) in four severely malnourished infants [305]. Ashkenazi et al. described the skeletal findings in a 6-month-old prematurely born infant with primary copper deficiency: *'long-bone changes on radiolog-*

ical examination, particularly osteoporosis with blurring and cupping of the metaphyses' [302]. Grünebaum et al. reported four children with copper deficiency [131]. All four showed symmetrical *'sickle-shaped metaphyseal spurs'*. Two children showed fractures of these spurs. Allen et al. described the presence of '*osteoporosis, metaphyseal cupping, widening of the zone of provisional calcification, multiple undisplaced fractures, and periosteal elevation with subperiosteal calcification*' [306]. Schmidt et al. described the findings in five preterm infants (25th to 30th week of gestation) suffering from alimentary copper deficiency [307]. They found (starting in the third to 12th week of life) general skeletal osteoporosis and retardation of the skeletal age, metaphyseal radiodense lines, irregular metaphyses, cupping, and spurring of the metaphyses, followed by multiple fractures and subperiosteal new-bone formation and enlarged costochondral junctions.

References

1. Mendelson KL (2005) Critical review of 'temporary brittle bone disease'. Pediatr Radiol 35:1036–1040
2. Altman DH, Smith RL (1960) Unrecognized trauma in infants and children. J Bone Joint Surg Am 42-a:407–413
3. O'Neill JA Jr, Meacham WF, Griffin JP, Sawyers JL (1973) Patterns of injury in the battered child syndrome. J Trauma 13:332–339
4. Bronicki LM, Stevenson RE, Spranger JW (2015) Beyond osteogenesis imperfecta: causes of fractures during infancy and childhood. Am J Med Genet C Semin Med Genet 169:314–327
5. Bishop N, Sprigg A, Dalton A (2007) Unexplained fractures in infancy: looking for fragile bones. Arch Dis Child 92:251–256
6. Shur N, Summerlin ML, Robin NH, Moreno-Mendelson A, Shalaby-Rana E, Hinds T (2020) Genetic consultations in cases of unexplained fractures and haemorrhage: an evidence-based approach. Curr Opin Pediatr. Publish Ahead of Print
7. Keats TE, Anderson MW (2012) Atlas of normal roentgen variants which may simulate disease. Saunders
8. Kwon DS, Spevak MR, Fletcher K, Kleinman PK (2002) Physiologic subperiosteal new bone formation: prevalence, distribution, and thickness in neonates and infants. AJR Am J Roentgenol 179:985–988
9. Shopfner CE (1966) Periosteal bone growth in normal infants. A preliminary report. Am J Roentgenol Radium Therapy, Nucl Med 97:154–163
10. Conway JJ, Collins M, Tanz RR (1993) The role of bone scintigraphy in detecting child abuse. Semin Nucl Med 23:321–333
11. Norrell K, Hennrikus W (2017) The risk of assuming abuse in an infant with an isolated metaphyseal lesion: a case report. JBJS Case Connect 7:e69
12. Karmazyn B, Wanner MR, Marine MB, Tilmans L, Jennings SG, Hibbard RA (2019) The added value of a second read by pediatric radiologists for outside skeletal surveys. Pediatr Radiol 49:203–209
13. Kleinman PK, Belanger PL, Karellas A, Spevak MR (1991) Normal metaphyseal radiologic variants not to be confused with findings of infant abuse. AJR Am J Roentgenol 156:781–783
14. Weir P, Suttner NJ, Flynn P, McAuley D (2006) Normal skull suture variant mimicking intentional injury. BMJ 332:1020–1021
15. Miller AJ, Kim U, Carrasco E (2010) Differentiating a mendosal suture from a skull fracture. J Pediatr 157:691
16. Naldemir IF, Guclu D, Baki Altınsoy H, Berk Canga H, Onbas O (2018) Accessory occipital suture mimicking fracture in head trauma. Am J Emerg Med 36:530.e537–530.e538
17. Tharp AM, Jason DR (2009) Anomalous parietal suture mimicking skull fracture. Am J Forensic Med Pathol 30:49–51
18. Wiedijk JEF, Soerdjbalie-Maikoe V, Maat GJR, Maes A, van Rijn RR, de Boer HH (2016) An accessory skull suture mimicking a skull fracture. Forensic Sci Int 260:e11–e13
19. Idriz S, Patel JH, Ameli Renani S, Allan R, Vlahos I (2015) CT of normal developmental and variant anatomy of the pediatric skull: distinguishing trauma from normality. Radiographics 35:1585–1601
20. Burkhard K, Lange LM, Plenzig S, Verhoff MA, Kölzer SC (2016) Schädelfraktur oder akzessorische Sutur bei einem kind? [skull fracture or accessory suture in a child?]. Archiv fur Kriminologie 237:172–181
21. Cirpan S, Aksu F, Mas N (2015) The incidence and topographic distribution of sutures including Wormian bones in human skulls. J Craniofac Surg 26:1687–1690
22. Eklund MJ, Carver KC, Stalcup ST, Riemer EC, Taylor MA, Hill JG (2016) Atypical accessory intraparietal sutures mimicking complex fractures in a neonate. Clin Imaging 40:806–809
23. Nakahara K, Miyasaka Y, Takagi H, Kan S, Fujii K (2003) Unusual accessory cranial sutures in pediatric head trauma—case report. Neurol Med Chir 43:80–81
24. Currarino G (1976) Normal variants and congenital anomalies in the region of the obelion. AJR Am J Roentgenol 127:487–494
25. Bernard S, Loukas M, Rizk E, Oskouian RJ, Delashaw J, Tubbs RS (2015) The human occipital bone: review and update on its embryology and molecular development. Childs Nerv System 31:2217–2223
26. Choudhary AK, Jha B, Boal DK, Dias M (2010) Occipital sutures and its variations: the value of 3D-CT and how to differentiate it from fractures using 3D-CT? Surg Radiol Anat 32:807–816
27. Hanihara T, Ishida H (2001) Os incae: variation in frequency in major human population groups. J Anat 198:137–152
28. Shapiro R, Robinson F (1976) The os incae. AJR Am J Roentgenol 127:469–471
29. Bellary SS, Steinberg A, Mirzayan N, Shirak M, Tubbs RS, Cohen-Gadol AA, Loukas M (2013) Wormian bones: a review. Clin Anat 26:922–927
30. Cremin B, Goodman H, Spranger J, Beighton P (1982) Wormian bones in osteogenesis imperfecta and other disorders. Skelet Radiol 8:35–38
31. Marti B, Sirinelli D, Maurin L, Carpentier E (2013) Wormian bones in a general paediatric population. Diagn Interv Imaging 94:428–432
32. Van Dijk FS, Sillence DO (2014) Osteogenesis imperfecta: clinical diagnosis, nomenclature and severity assessment. Am J Med Genet A 164a:1470–1481
33. Zarate YA, Clingenpeel R, Sellars EA, Tang X, Kaylor JA, Bosanko K, Linam LE, Byers PH (2016) COL1A1 and COL1A2 sequencing results in cohort of patients undergoing evaluation for potential child abuse. Am J Med Genet A 170:1858–1862
34. Leventhal JM, Martin KD, Asnes AG (2010) Fractures and traumatic brain injuries: abuse versus accidents in a US database of hospitalized children. Pediatrics 126:e104-115
35. Van Dijk FS, Pals G, Van Rijn RR, Nikkels PG, Cobben JM (2010) Classification of osteogenesis imperfecta revisited. Eur J Med Genet 53:1–5
36. van Dijk FS, Huizer M, Kariminejad A, Marcelis CL, Plomp AS, Terhal PA, Meijers-Heijboer H, Weiss MM, van Rijn RR, Cobben JM, Pals G (2010) Complete COL1A1 allele deletions in osteogenesis imperfecta. Genet Med 12:736–741
37. Andersson K, Dahllöf G, Lindahl K, Kindmark A, Grigelioniene G, Åström E, Malmgren B (2017) Mutations in COL1A1 and

COL1A2 and dental aberrations in children and adolescents with osteogenesis imperfecta—a retrospective cohort study. PLoS One 12:e0176466
38. Ward LM, Rauch F, Travers R, Chabot G, Azouz EM, Lalic L, Roughley PJ, Glorieux FH (2002) Osteogenesis imperfecta type VII: an autosomal recessive form of brittle bone disease. Bone 31:12–18
39. Barnes AM, Chang W, Morello R, Cabral WA, Weis M, Eyre DR, Leikin S, Makareeva E, Kuznetsova N, Uveges TE, Ashok A, Flor AW, Mulvihill JJ, Wilson PL, Sundaram UT, Lee B, Marini JC (2006) Deficiency of cartilage-associated protein in recessive lethal osteogenesis imperfecta. N Engl J Med 355:2757–2764
40. Cabral WA, Chang W, Barnes AM, Weis M, Scott MA, Leikin S, Makareeva E, Kuznetsova NV, Rosenbaum KN, Tifft CJ, Bulas DI, Kozma C, Smith PA, Eyre DR, Marini JC (2007) Prolyl 3-hydroxylase 1 deficiency causes a recessive metabolic bone disorder resembling lethal/severe osteogenesis imperfecta. Nat Genet 39:359–365
41. Van Dijk FS, Nesbitt IM, Nikkels PG, Dalton A, Bongers EM, van de Kamp JM, Hilhorst-Hofstee Y, Den Hollander NS, Lachmeijer AM, Marcelis CL, Tan-Sindhunata GM, van Rijn RR, Meijers-Heijboer H, Cobben JM, Pals G (2009) CRTAP mutations in lethal and severe osteogenesis imperfecta: the importance of combining biochemical and molecular genetic analysis. Eur J Hum Genet 17:1560–1569
42. Pepin MG, Byers PH (2015) What every clinical geneticist should know about testing for osteogenesis imperfecta in suspected child abuse cases. Am J Med Genet C Semin Med Genet 169:307–313
43. Shur N, Carey JC (2015) Genetic differentials of child abuse: is your case rare or real? Am J Med Genet C Semin Med Genet 169:281–288
44. Pereira EM (2015) Clinical perspectives on osteogenesis imperfecta versus non-accidental injury. Am J Med Genet C Semin Med Genet 169:302–306
45. Sillence DO, Senn A, Danks DM (1979) Genetic heterogeneity in osteogenesis imperfecta. J Med Genet 16:101–116
46. Rauch F, Glorieux FH (2004) Osteogenesis imperfecta. Lancet 363:1377–1385
47. Plotkin H (2019) Osteogenesis imperfecta. http://www.emedicine.com/ped/topic1674.htm Date accessed: 17-07-2019
48. Astley R (1979) Metaphyseal fractures in osteogenesis imperfecta. Br J Radiol 52:441–443
49. Ablin DS, Greenspan A, Reinhart M, Grix A (1990) Differentiation of child abuse from osteogenesis imperfecta. AJR Am J Roentgenol 154:1035–1046
50. Gahagan S, Rimsza ME (1991) Child abuse or osteogenesis imperfecta: how can we tell? Pediatrics 88:987–992
51. Steiner RD, Pepin M, Byers PH (1996) Studies of collagen synthesis and structure in the differentiation of child abuse from osteogenesis imperfecta. J Pediatr 128:542–547
52. Krakow D, Lachman RS, Kleinman PK (2015) Differential diagnosis III: osteogenesis imperfecta. In: Kleinman PK (ed) Diagnostic imaging of child abuse. Cambridge University Press, pp 254–267
53. Reece RM (2001) Child abuse—medical diagnosis and management. Lippincott Williams & Wilkins, pp 149–151
54. Knight DJ, Bennet GC (1990) Nonaccidental injury in osteogenesis imperfecta: a case report. J Pediatr Orthop 10:542–544
55. Dunn PM (1998) Francis Glisson (1597-1677) and the "discovery" of rickets. Arch Dis Child Fetal Neonatal Ed 78:F154–F155
56. Zhang M, Shen F, Petryk A, Tang J, Chen X, Sergi C (2016) "English disease": historical notes on rickets, the bone-lung Link and child neglect issues. Nutrients 8
57. Hernigou P, Auregan JC, Dubory A (2019) Vitamin D: part II; cod liver oil, ultraviolet radiation, and eradication of rickets. Int Orthop 43:735–749
58. Akkermans MD, van der Horst-Graat JM, Eussen SR, van Goudoever JB, Brus F (2016) Iron and vitamin D deficiency in healthy young children in Western Europe despite current nutritional recommendations. J Pediatr Gastroenterol Nutr 62:635–642
59. Kehler L, Verma S, Krone R, Roper E (2013) Vitamin D deficiency in children presenting to the emergency department: a growing concern. Vitamin D deficiency in Birmingham's children: presentation to the emergency department. Emerg Med J 30:717–719
60. Kubota T, Nakayama H, Kitaoka T, Nakamura Y, Fukumoto S, Fujiwara I, Hasegawa Y, Ihara K, Kitanaka S, Koyama S, Kusuda S, Mizuno H, Nagasaki K, Oba K, Sakamoto Y, Takubo N, Shimizu T, Tanahashi Y, Hasegawa K, Tsukahara H, Yorifuji T, Michigami T, Ozono K (2018) Incidence rate and characteristics of symptomatic vitamin D deficiency in children: a nationwide survey in Japan. Endocr J 65:593–599
61. Thacher TD, Fischer PR, Tebben PJ, Singh RJ, Cha SS, Maxson JA, Yawn BP (2013) Increasing incidence of nutritional rickets: a population-based study in Olmsted County, Minnesota. Mayo Clin Proc 88:176–183
62. Ward LM, Gaboury I, Ladhani M, Zlotkin S (2007) Vitamin D-deficiency rickets among children in Canada. CMAJ 177:161–166
63. Wheeler BJ, Dickson NP, Houghton LA, Ward LM, Taylor BJ (2015) Incidence and characteristics of vitamin D deficiency rickets in New Zealand children: a New Zealand Paediatric Surveillance Unit study. Aust N Z J Public Health 39:380–383
64. Aldana Sierra MC, Christian CW (2021) Vitamin D, rickets and child abuse: controversies and evidence. Pediatr Radiol 51:1014–1022
65. Roberts D, Gaillard F (2020) Rickets. https://radiopaedia.org/articles/rickets Date accessed: 28-11-2021
66. Krebs NF, Primak LE (2011) Vitamin and mineral deficiences; Pediatric nutrition and nutritional disorders. In: Marcdante KJ, Kliegman RM, Jenson HB, Behrman RE (eds) Nelson essentials of Pediatrics. Saunders Elsevier, pp 114–122
67. Wagner CL, Greer FR (2008) Prevention of rickets and vitamin D deficiency in infants, children, and adolescents. Pediatrics 122:1142–1152
68. National Organization for Rare Disorders (NORD) (2020) Rare Disease Database: vitamin D deficiency rickets. https://rarediseases.org/rare-diseases/rickets-vitamin-d-deficiency/ Date accessed: 28-10-2020
69. Creo AL, Thacher TD, Pettifor JM, Strand MA, Fischer PR (2017) Nutritional rickets around the world: an update. Paediatr Int Child Health 37:84–98
70. van Rijn RR (2018) Rickets imaging. https://emedicine.medscape.com/article/412862-overview Date accessed: 14-08-2021
71. Lemoine A, Giabicani E, Lockhart V, Grimprel E, Tounian P (2020) Case report of nutritional rickets in an infant following a vegan diet. Arch Pediatr 27:219–222
72. Pettifor JM (2004) Nutritional rickets: deficiency of vitamin D, calcium, or both? Am J Clin Nutr 80:1725s–1729s
73. Stoutjesdijk E, Schaafsma A, Nhien NV, Khor GL, Kema IP, Hollis BW, Dijck-Brouwer DAJ, Muskiet FAJ (2017) Milk vitamin D in relation to the 'adequate intake' for 0-6-month-old infants: a study in lactating women with different cultural backgrounds, living at different latitudes. Br J Nutr 118:804–812
74. Arundel P, Ahmed SF, Allgrove J, Bishop NJ, Burren CP, Jacobs B, Mughal MZ, Offiah AC, Shaw NJ (2012) British paediatric and adolescent bone Group's position statement on vitamin D deficiency. BMJ 345:e8182
75. Chesney RW (2002) Rickets: the third wave. Clin Pediatr (Phila) 41:137–139
76. Schwarz M (2017) Rickets. https://emedicine.medscape.com/article/985510-overview Date accessed: 29-11-2021

77. Glew RH, Crossey MJ, Polanams J, Okolie HI, VanderJagt DJ (2010) Vitamin D status of seminomadic Fulani men and women. J Natl Med Assoc 102:485–490
78. Guzel R, Kozanoglu E, Guler-Uysal F, Soyupak S, Sarpel T (2001) Vitamin D status and bone mineral density of veiled and unveiled Turkish women. J Womens Health Gend Based Med 10:765–770
79. Buyukuslu N, Esin K, Hizli H, Sunal N, Yigit P, Garipagaoglu M (2014) Clothing preference affects vitamin D status of young women. Nutr Res 34:688–693
80. Glerup H, Mikkelsen K, Poulsen L, Hass E, Overbeck S, Thomsen J, Charles P, Eriksen EF (2000) Commonly recommended daily intake of vitamin D is not sufficient if sunlight exposure is limited. J Intern Med 247:260–268
81. Ladhani S, Srinivasan L, Buchanan C, Allgrove J (2004) Presentation of vitamin D deficiency. Arch Dis Child 89:781–784
82. Goldacre M, Hall N, Yeates DG (2014) Hospitalisation for children with rickets in England: a historical perspective. Lancet 383:597–598
83. Moon RJ, Harvey NC, Davies JH, Cooper C (2014) Vitamin D and skeletal health in infancy and childhood. Osteoporos Int 25:2673–2684
84. Marson BA, Price KR, Hunter JB (2017) Vitamin D deficiency in children. https://www.sfh-tr.nhs.uk/media/3770/vitamin-d-deficiency-in-children.pdf Date accessed: 28-10-2020
85. Bloom E, Klein EJ, Shushan D, Feldman KW (2004) Variable presentations of rickets in children in the emergency department. Pediatr Emerg Care 20:126–130
86. Dijksman T, van Doorn K (2011) Een meisje met kromme beentjes [A girl with bowed legs]. Ned Tijdschr Geneeskd 155:A2345
87. Meštrović T (2018) Rickets diagnosis. https://www.news-medical.net/health/Rickets-Diagnosis.aspx Date accessed: 14-08-2021
88. Chesney RW (2008) Rickets or abuse, or both? Pediatr Radiol 38:1217–1218
89. Saggese G, Vierucci F, Prodam F, Cardinale F, Cetin I, Chiappini E, De' Angelis GL, Massari M, Miraglia Del Giudice E, Miraglia Del Giudice M, Peroni D, Terracciano L, Agostiniani R, Careddu D, Ghiglioni DG, Bona G, Di Mauro G, Corsello G (2018) Vitamin D in pediatric age: consensus of the Italian Pediatric society and the Italian Society of Preventive and Social Pediatrics, jointly with the Italian Federation of Pediatricians. Ital J Pediatr 44:51
90. Munns CF, Shaw N, Kiely M, Specker BL, Thacher TD, Ozono K, Michigami T, Tiosano D, Mughal MZ, Mäkitie O, Ramos-Abad L, Ward L, DiMeglio LA, Atapattu N, Cassinelli H, Braegger C, Pettifor JM, Seth A, Idris HW, Bhatia V, Fu J, Goldberg G, Sävendahl L, Khadgawat R, Pludowski P, Maddock J, Hyppönen E, Oduwole A, Frew E, Aguiar M, Tulchinsky T, Butler G, Högler W (2016) Global consensus recommendations on prevention and management of nutritional rickets. J Clin Endocrinol Metab 101:394–415
91. Thacher TD, Fischer PR, Pettifor JM, Lawson JO, Manaster BJ, Reading JC (2000) Radiographic scoring method for the assessment of the severity of nutritional rickets. J Trop Pediatr 46:132–139
92. Shore RM, Chesney RW (2013) Rickets: part II. Pediatr Radiol 43:152–172
93. Zeiss J, Wycliffe ND, Cullen BJ, Conover S, Wood BP (1988) Radiological case of the month. Simulated child abuse in drug-induced rickets. Am J Dis Child 142:1367–1368
94. Duncan AA, Chandy J (1993) Case report: multiple neonatal fractures—dietary or deliberate? Clin Radiol 48:137–139
95. Kepron C, Pollanen MS (2015) Rickets or abuse? A histologic comparison of rickets and child abuse-related fractures. Forensic Sci Med Pathol 11:78–87
96. Rees DC, Williams TN, Gladwin MT (2010) Sickle-cell disease. Lancet 376:2018–2031
97. Frenette PS, Atweh GF (2007) Sickle cell disease: old discoveries, new concepts, and future promise. J Clin Invest 117:850–858
98. Madani G, Papadopoulou AM, Holloway B, Robins A, Davis J, Murray D (2007) The radiological manifestations of sickle cell disease. Clin Radiol 62:528–538
99. Brooks AS, Dooijes D (2003) Van gen naar ziekte; arteriohepatische dysplasie—het syndroom van Alagille [From gene to disease: arteriohepatic dysplasia or Alagille syndrome]. Ned Tijdschr Geneeskd 147:1213–1215
100. Scheimann A (2006) Alagille syndrome. https://reference.medscape.com/article/926678-differential Date accessed: 14-08-2021
101. de Halleux J, Rombouts JJ, Otte JB (1998) Evolution des déviations osseuses post fracturaires chez une enfant atteinte d'ostéodystrophie hépatique sur syndrome d'[AlagilleEvolution of post-fracture bone deformities in an infant with hepatic osteodystrophy on Alagille syndrome]. Revue de chirurgie orthopedique et reparatrice de l'appareil moteur 84:381–386
102. Metules T (2002) Duchenne muscular dystrophy. RN 65:39–44. 47; quiz 48
103. Boyce AM, Gafni RI (2011) Approach to the child with fractures. J Clin Endocrinol Metab 96:1943–1952
104. Ma J, McMillan HJ, Karagüzel G, Goodin C, Wasson J, Matzinger MA, DesClouds P, Cram D, Page M, Konji VN, Lentle B, Ward LM (2017) The time to and determinants of first fractures in boys with Duchenne muscular dystrophy. Osteoporos Int 28:597–608
105. McDonald DG, Kinali M, Gallagher AC, Mercuri E, Muntoni F, Roper H, Jardine P, Jones DH, Pike MG (2002) Fracture prevalence in Duchenne muscular dystrophy. Dev Med Child Neurol 44:695–698
106. Karol LA (2007) Scoliosis in patients with Duchenne muscular dystrophy. J Bone Joint Surg Am 89(Suppl 1):155–162
107. Liang WC, Wang CH, Chou PC, Chen WZ, Jong YJ (2018) The natural history of the patients with Duchenne muscular dystrophy in Taiwan: a medical center experience. Pediatr Neonatol 59:176–183
108. Hefti F, Bollini G, Dungl P, Fixsen J, Grill F, Ippolito E, Romanus B, Tudisco C, Wientroub S (2000) Congenital pseudarthrosis of the tibia: history, etiology, classification, and epidemiologic data. J Pediatr Orthop B 9:11–15
109. Crawford AH (1986) Neurofibromatosis in children. Acta Orthop Scand Suppl 218:1–60
110. Caffey J (1957) Infantile cortical hyperostosis; a review of the clinical and radiographic features. Proc R Soc Med 50:347–354
111. France AK, Kain JJ, Gourishankar A (2016) Facial swelling in an infant. JAMA Otolaryngol Head Neck Surg 142:293–294
112. Ramesh V, Sankar J (2017) Infantile cortical hyperostosis of scapula presenting as pseudoparalysis in an infant. Indian Pediatr 54:157–158
113. Saul RA, Lee WH, Stevenson RE (1982) Caffey's disease revisited. Further evidence for autosomal dominant inheritance with incomplete penetrance. Am J Dis Child 136:55–60
114. Barba WP, Freriks DJ (1953) The familial occurrence of infantile cortical hyperostosis in utero. J Pediatr 42:141–150
115. Bercau G, Gonzalez M, Afriat R, Lecolier B, de Kermadec S (1991) La difficulté du diagnostic de la maladie de Caffey in utero. A propos d'un cas simulant l'ostéogenèse imparfaite létale [the difficulty of diagnosing Caffey's disease in utero. Apropos of a case simulating lethal osteogenesis imperfecta]. Annales de pediatrie 38:15–18
116. Labrune M, Guedj G, Vial M, Bessis R, Roset M, Kerbrat V (1983) Maladie de Caffey à début anténatal [Caffey's disease with antenatal onset]. Archives francaises de pediatrie 40:39–43
117. Langer R, Kaufmann HJ (1985) Pränatale Diagnosestellung bei Caffey'scher Erkrankung (infantile kortikale Hyperostose) [prenatal diagnosis of Caffey's disease (infantile cortical hyperostosis)]. Klinische Padiatrie 197:473–476

118. Schweiger S, Chaoui R, Tennstedt C, Lehmann K, Mundlos S, Tinschert S (2003) Antenatal onset of cortical hyperostosis (Caffey disease): case report and review. Am J Med Genet A 120a:547–552
119. Ved N, Haller JO (2002) Periosteal reaction with normal-appearing underlying bone: a child abuse mimicker. Emerg Radiol 9:278–282
120. Lo HP, Lau HY, Li CH, So KT (2010) Infantile cortical hyperostosis (Caffey disease): a possible misdiagnosis as physical abuse. Hong Kong Med J = Xianggang yi xue za zhi 16:397–399
121. Menkes JH, Alter M, Steigleder GK, Weakley DR, Sung JH (1962) A sex-linked recessive disorder with retardation of growth, peculiar hair, and focal cerebral and cerebellar degeneration. Pediatrics 29:764–779
122. Kapur S, Higgins JV, Delp K, Rogers B (1987) Menkes syndrome in a girl with X-autosome translocation. Am J Med Genet 26:503–510
123. Abusaad I, Mohammed SN, Ogilvie CM, Ritchie J, Pohl KR, Docherty Z (1999) Clinical expression of Menkes disease in a girl with X;13 translocation. Am J Med Genet 87:354–359
124. Danks DM, Cartwright E, Campbell PE, Mayne V (1971) Is Menkes' syndrome a heritable disorder of connective tissue? Lancet 2:1089
125. Tønnesen T, Kleijer WJ, Horn N (1991) Incidence of Menkes disease. Hum Genet 86:408–410
126. Freidl P, Rauter L, Moser R, Kerbl R (2015) Intrapartum acquired skull fracture as first sign of Menkes disease. Klin Padiatr 227:33–34
127. Ubhi T, Reece A, Craig A (2000) Congenital skull fracture as a presentation of Menkes disease. Dev Med Child Neurol 42:347–348
128. Chang CH (2019) Menkes disease. https://emedicine.medscape.com/article/1180460-overview Date accessed: 14-08-2021
129. Adams PC, Strand RD, Bresnan MJ, Lucky AW (1974) Kinky hair syndrome: serial study of radiological findings with emphasis on the similarity to the battered child syndrome. Radiology 112:401–407
130. Seay AR, Bray PF, Wing SD, Thompson JA, Bale JF, Williams DM (1979) CT scans in Menkes disease. Neurology 29:304–312
131. Grünebaum M, Horodniceanu C, Steinherz R (1980) The radiographic manifestations of bone changes in copper deficiency. Pediatr Radiol 9:101–104
132. Wendler H, Mutz I (1985) Menkes-Syndrom mit exzessiven Skelettveränderungen [Menkes syndrome with excessive skeletal changes]. RoFo : Fortschritte auf dem Gebiete der Rontgenstrahlen und der Nuklearmedizin 143:351–355
133. Jankov RP, Boerkoel CF, Hellmann J, Sirkin WL, Tümer Z, Horn N, Feigenbaum A (1998) Lethal neonatal Menkes' disease with severe vasculopathy and fractures. Acta Paediatr 87:1297–1300
134. Cronin H, Fussell JN, Pride H, Bellino P (2012) Menkes syndrome presenting as possible child abuse. Cutis 90:170–172
135. Hill SC, Dwyer AJ, Kaler SG (2012) Cervical spine anomalies in Menkes disease: a radiologic finding potentially confused with child abuse. Pediatr Radiol 42:1301–1304
136. Wacks NP, Schoppel K, Sell PJ, Guggina T (2016) Opening Pandora's box: a chest radiograph in a 5-month-old with bronchiolitis. Hosp Pediatr 6:642–645
137. Droms RJ, Rork JF, McLean R, Martin M, Belazarian L, Wiss K (2017) Menkes disease mimicking child abuse. Pediatr Dermatol 34:e132–e134
138. Akinseye ON, Yazdani R, Tornow KA, Reeder KN, Clarke RL, Pfeifer CM (2019) Imaging findings of Menkes disease, a radiographic mimic of abusive trauma. Radiol Case Rep 14:993–996
139. Akbarnia BA, Campbell RM (1990) The role of the orthopedic surgeon in child abuse. In: Morrissy RT, Winter RB (eds) Lovell and Winter's pediatric orthopaedics. Lippincott, Williams and Wilkins
140. Spencer JA, Grieve DK (1990) Congenital indifference to pain mistaken for non-accidental injury. Br J Radiol 63:308–310
141. Offiah AC, Hall CM (2003) Radiological diagnosis of the constitutional disorders of bone. As easy as A, B, C? Pediatr Radiol 33:153–161
142. Spranger JW, Brill PW, Poznanski A (2002) Bone dysplasias: an atlas of genetic disorders of skeletal development. Oxford University Press
143. Elliott AM, Field FM, Rimoin DL, Lachman RS (2005) Hand involvement in Schmid metaphyseal chondrodysplasia. Am J Med Genet A 132a:191–193
144. Richmond CM, Savarirayan R (1993) Schmid metaphyseal chondrodysplasia. In: Adam MP, Ardinger HH, Pagon RA et al (eds) GeneReviews(®). University of Washington, Seattle, Seattle (WA)
145. England J, McFarquhar A, Campeau PM (1993) Spondylometaphyseal dysplasia, corner fracture type. In: Adam MP, Ardinger HH, Pagon RA et al (eds) GeneReviews(®). University of Washington, Seattle, Seattle (WA)
146. Currarino G, Birch JG, Herring JA (2000) Developmental coxa vara associated with spondylometaphyseal dysplasia (DCV/SMD): "SMD-corner fracture type" (DCV/SMD-CF) demonstrated in most reported cases. Pediatr Radiol 30:14–24
147. Emma F, Cappa M, Antoniazzi F, Bianchi ML, Chiodini I, Eller Vainicher C, Di Iorgi N, Maghnie M, Cassio A, Balsamo A, Baronio F, de Sanctis L, Tessaris D, Baroncelli GI, Mora S, Brandi ML, Weber G, D'Ausilio A, Lanati EP (2019) X-linked hypophosphatemic rickets: an Italian experts' opinion survey. Ital J Pediatr 45:67
148. Lambert AS, Zhukouskaya V, Rothenbuhler A, Linglart A (2019) X-linked hypophosphatemia: management and treatment prospects. Joint Bone Spine 86:731–738
149. El Demellawy D, Davila J, Shaw A, Nasr Y (2018) Brief review on metabolic bone disease. Acad Forensic Pathol 8:611–640
150. Bodamer OA, Bravermann RM, Craigen WJ (2001) Multiple fractures in a 3-month-old infant with severe infantile osteopetrosis. J Paediatr Child Health 37:520–522
151. Georgiev H, Alexiev VA (2013) Case report of LCP pediatric hip osteosynthesis of a proximal femoral fracture in a child with marble bone disease. Pan Afr Med J 15:66
152. Gonen KA, Yazici Z, Gokalp G, Ucar AK (2013) Infantile osteopetrosis with superimposed rickets. Pediatr Radiol 43:189–195
153. van Rijn RR, Smets AM (2003) Diagnose in bleed (132). Een 3-jarige jongen met groeiachterstand. Osteopetrose [diagnostic image (132). A 3-year old boy with growth retardation]. Ned Tijdschr Geneeskd 147:606–607
154. Wijeratne N, Choy KW, Lu ZX, Brown J, Doery JC (2016) A girl with bone sclerosis and fracture. Clin Chem 62:684–687
155. Biyyam DR, Done S (2010) Osteopetrorickets: infantile malignant osteopetrosis paradoxically complicated by rickets. Pediatr Radiol 40:782
156. Demirel F, Esen I, Tunc B, Tavil B (2010) Scarcity despite wealth: osteopetrorickets. J Pediatr Endocrinol Metab 23:931–934
157. Kaplan FS, August CS, Fallon MD, Gannon F, Haddad JG (1993) Osteopetrorickets. The paradox of plenty. Pathophysiology and treatment. Clin Orthop Relat Res:64–78
158. Patra S, Pemde HK, Singh V, Chandra J (2009) Infantile osteopetrorickets. Indian J Pediatr 76:1182–1183
159. Ozdirim E, Altay C, Pirnar T (1981) Osteopetrosis with rickets in infancy. Turk J Pediatr 23:211–218
160. Kirubakaran C, Ranjini K, Scott JX, Basker M, Sridhar G (2004) Osteopetrorickets. J Trop Pediatr 50:185–186
161. Loud KJ, Gordon CM (2006) Adolescent bone health. Arch Pediatr Adolesc Med 160:1026–1032

162. van der Sluis IM, de Muinck Keizer-Schrama SM (2001) Osteoporosis in childhood: bone density of children in health and disease. J Pediatr Endocrinol Metab 14:817–832
163. International Society for Clinical Densitometry (ISCD) (2019) Skeletal health assessment in children from infancy to adolescence. https://iscd.org/learn/official-positions/pediatric-positions/ Date accessed: 14-08-2021
164. Steffey CL (2019) Pediatric osteoporosis. Pediatr Rev 40:259–261
165. Lorenc RS (2002) Idiopathic juvenile osteoporosis. Calcif Tissue Int 70:395–397
166. Tan LO, Lim SY, Vasanwala RF (2017) Primary osteoporosis in children. BMJ Case Rep 2017
167. van Rijn RR, van der Sluis IM, Link TM, Grampp S, Guglielmi G, Imhof H, Glüer C, Adams JE, van Kuijk C (2003) Bone densitometry in children: a critical appraisal. Eur Radiol 13:700–710
168. Mornet E (2007) Hypophosphatasia. Orphanet J Rare Dis 2:40
169. Szabo SM, Tomazos IC, Petryk A, Powell LC, Donato BMK, Zarate YA, Tiulpakov A, Martos-Moreno G (2019) Frequency and age at occurrence of clinical manifestations of disease in patients with hypophosphatasia: a systematic literature review. Orphanet J Rare Dis 14:85
170. Thacher TD, Pettifor JM, Tebben PJ, Creo AL, Skrinar A, Mao M, Chen CY, Chang T, San Martin J, Carpenter TO (2019) Rickets severity predicts clinical outcomes in children with X-linked hypophosphatemia: utility of the radiographic rickets severity score. Bone 122:76–81
171. Moulin P, Vaysse F, Bieth E, Mornet E, Gennero I, Dalicieux-Laurencin S, Baunin C, Tauber MT, De Gauzy JS, Salles JP (2009) Hypophosphatasia may lead to bone fragility: don't miss it. Eur J Pediatr 168:783–788
172. Blyth MJ, Kincaid R, Craigen MA, Bennet GC (2001) The changing epidemiology of acute and subacute haematogenous osteomyelitis in children. J Bone Joint Surg Br 83:99–102
173. Taylor MN, Chaudhuri R, Davis J, Novelli V, Jaswon MS (2008) Childhood osteomyelitis presenting as a pathological fracture. Clin Radiol 63:348–351
174. Girschick H (2002) Chronic recurrent multifocal osteomyelitis in children. http://www.orpha.net/data/patho/GB/uk-CRMO.pdf Date accessed: 14-08-2021
175. Menashe SJ, Aboughalia H, Zhao Y, Ngo AV, Otjen JP, Thapa MM, Iyer RS (2020) The many faces of Pediatric chronic recurrent multifocal osteomyelitis (CRMO): a practical location- and case-based approach to differentiate CRMO from its mimics. J Magn Reson Imaging:e27299
176. Brown T, Wilkinson RH (1988) Chronic recurrent multifocal osteomyelitis. Radiology 166:493–496
177. Lim HK, Smith WL, Sato Y, Choi J (1995) Congenital syphilis mimicking child abuse. Pediatr Radiol 25:560–561
178. Toohey JS (1985) Skeletal presentation of congenital syphilis: case report and review of the literature. J Pediatr Orthop 5:104–106
179. Solomon A, Rosen E (1975) The aspect of trauma in the bone changes of congenital lues. Pediatr Radiol 3:176–178
180. Caffey J (1957) Some traumatic lesions in growing bones other than fractures and dislocations: clinical and radiological features: the Mackenzie Davidson Memorial Lecture. Br J Radiol 30:225–238
181. Rasool MN, Govender S (1989) The skeletal manifestations of congenital syphilis. A review of 197 cases. J Bone Joint Surg Br 71:752–755
182. Jacobs K, Vu DM, Mony V, Sofos E, Buzi N (2019) Congenital syphilis misdiagnosed as suspected nonaccidental trauma. Pediatrics:144
183. Idrissi ML, Ismaili L, Bouharrou A, Hida M (2011) La syphilis congénitale révélée par une fracture spontanée [Congenital syphilis revealed by a spontaneous fracture]. Pan Afr Med J 10:42
184. SEER Cancer Statistics Review 1975–2017 Childhood Cancer. https://seer.cancer.gov/csr/1975_2017/ Date accessed: 14-08-2021
185. McGregor LM, Metzger ML, Sanders R, Santana VM (2007) Pediatric cancers in the new millennium: dramatic progress, new challenges. Oncology (Williston Park) 21:809–820. discussion 820, 823-804
186. Crist WM, Pui CH (1996) The leukemias. In: Behrman RE, Kliegman RM, Arvin AM (eds) Nelson textbook of pediatrics
187. Sinigaglia R, Gigante C, Bisinella G, Varotto S, Zanesco L, Turra S (2008) Musculoskeletal manifestations in pediatric acute leukemia. J Pediatr Orthop 28:20–28
188. Miller SL, Hoffer FA (2001) Malignant and benign bone tumors. Radiol Clin N Am 39:673–699
189. Kaste SC (2011) Imaging pediatric bone sarcomas. Radiol Clin N Am 49(749–765):vi–vii
190. Kransdorf MJ, Stull MA, Gilkey FW, Moser RP Jr (1991) Osteoid osteoma. Radiographics 11:671–696
191. Iyer RS, Chapman T, Chew FS (2012) Pediatric bone imaging: diagnostic imaging of osteoid osteoma. AJR Am J Roentgenol 198:1039–1052
192. Mascard E, Gomez-Brouchet A, Lambot K (2015) Bone cysts: unicameral and aneurysmal bone cyst. Orthop Traumatol Surg Res 101:S119–S127
193. van der Woude HJ, Smithuis R Bone tumours: Osteolytic—well defined. https://radiologyassistant.nl/musculoskeletal/bone-tumors/osteolytic-well-defined Date accessed: 15-07-2021
194. Park HY, Yang SK, Sheppard WL, Hegde V, Zoller SD, Nelson SD, Federman N, Bernthal NM (2016) Current management of aneurysmal bone cysts. Curr Rev Musculoskelet Med 9:435–444
195. Vergel De Dios AM, Bond JR, Shives TC, McLeod RA, Unni KK (1992) Aneurysmal bone cyst. A clinicopathologic study of 238 cases. Cancer 69:2921–2931
196. Leonard MB (2007) Glucocorticoid-induced osteoporosis in children: impact of the underlying disease. Pediatrics 119(Suppl 2):S166–S174
197. Sarinho ESC, Melo V (2017) Glucocorticoid-induces bone disease: mechanisms and importance in pediatric practice. Revista paulista de pediatria : orgao oficial da Sociedade de Pediatria de Sao Paulo 35:207–215
198. Meister B, Gassner I, Streif W, Dengg K, Fink FM (1994) Methotrexate osteopathy in infants with tumors of the central nervous system. Med Pediatr Oncol 23:493–496
199. Roebuck DJ (1999) Skeletal complications in pediatric oncology patients. Radiographics 19:873–885
200. Schwartz AM, Leonidas JC (1984) Methotrexate osteopathy. Skelet Radiol 11:13–16
201. Siegel NJ, Spackman TJ (1972) Chronic hypervitaminosis a with intracranial hypertension and low cerebrospinal fluid concentration of protein. Two illustrative cases. Clin Pediatr (Phila) 11:580–584
202. Mahoney CP, Margolis MT, Knauss TA, Labbe RF (1980) Chronic vitamin a intoxication in infants fed chicken liver. Pediatrics 65:893–897
203. Gamble JG, Ip SC (1985) Hypervitaminosis a in a child from megadosing. J Pediatr Orthop 5:219–221
204. Frame B, Jackson CE, Reynolds WA, Umphrey JE (1974) Hypercalcemia and skeletal effects in chronic hypervitaminosis A. Ann Intern Med 80:44–48
205. Grissom LE, Griffin GC, Mandell GA (1996) Hypervitaminosis A as a complication of treatment for neuroblastoma. Pediatr Radiol 26:200–202
206. Eid NS, Shoemaker LR, Samiec TD (1990) Vitamin A in cystic fibrosis: case report and review of the literature. J Pediatr Gastroenterol Nutr 10:265–269
207. Kimmoun A, Leheup B, Feillet F, Dubois F, Morali A (2008) Hypercalcémie révélant une hypervitaminose A iatrogène chez

un enfant atteint de troubles autistiques [hypercalcemia revealing iatrogenic hypervitaminosis A in a child with autistic troubles]. Arch Pediatr: organe officiel de la Societe francaise de pediatrie 15:29–32
208. Olson JA (1987) Recommended dietary intakes (RDI) of vitamin A in humans. Am J Clin Nutr 45:704–716
209. Mendoza FS, Johnson F, Kerner JA, Tune BM, Shochat SJ (1988) Vitamin A intoxication presenting with ascites and a normal vitamin A level. West J Med 148:88–90
210. Kerins DM, Murray R, FitzGerald GA (1991) Prostacyclin and prostaglandin E1: molecular mechanisms and therapeutic utility. Prog Hemost Thromb 10:307–337
211. Matzinger MA, Briggs VA, Dunlap HJ, Udjus K, Martin DJ, McDonald P (1992) Plain film and CT observations in prostaglandin-induced bone changes. Pediatr Radiol 22:264–266
212. Poznanski AK, Fernbach SK, Berry TE (1985) Bone changes from prostaglandin therapy. Skelet Radiol 14:20–25
213. Russell RG, Croucher PI, Rogers MJ (1999) Bisphosphonates: pharmacology, mechanisms of action and clinical uses. Osteoporos Int 9(Suppl 2):S66–S80
214. Reszka AA, Rodan GA (2004) Nitrogen-containing bisphosphonate mechanism of action. Mini Rev Med Chem 4:711–719
215. Boonen S, Vanderschueren D, Venken K, Milisen K, Delforge M, Haentjens P (2008) Recent developments in the management of postmenopausal osteoporosis with bisphosphonates: enhanced efficacy by enhanced compliance. J Intern Med 264:315–332
216. Cimaz R (2002) Osteoporosis in childhood rheumatic diseases: prevention and therapy. Best Pract Res Clin Rheumatol 16:397–409
217. Glorieux FH (2007) Treatment of osteogenesis imperfecta: who, why, what? Horm Res 68(Suppl 5):8–11
218. Glorieux FH, Rauch F (2006) Medical therapy of children with fibrous dysplasia. J Bone Miner Research 21(Suppl 2):P110–P113
219. Batch JA, Couper JJ, Rodda C, Cowell CT, Zacharin M (2003) Use of bisphosphonate therapy for osteoporosis in childhood and adolescence. J Paediatr Child Health 39:88–92
220. Price AP, Abramson SJ, Hwang S, Chou A, Bartolotta R, Meyers P, Katz DS (2011) Skeletal imaging effects of pamidronate therapy in osteosarcoma patients. Pediatr Radiol 41:451–458
221. Blount WP (1937) Tibia vara: osteochondrosis deformans tibiae. J Bone Joint Surg 19:1–29
222. Janoyer M (2019) Blount disease. Orthop Traumatol Surg Res 105:S111–s121
223. Bateson EM (1968) The relationship between Blount's disease and bow legs. Br J Radiol 41:107–114
224. Thompson GH, Carter JR (1990) Late-onset tibia vara (Blount's disease). Current concepts. Clin Orthop Relat Res:24–35
225. Henderson RC (1992) Tibia vara: a complication of adolescent obesity. J Pediatr 121:482–486
226. Prasad V, Kendrick D, Sayal K, Thomas SL, West J (2014) Injury among children and young adults with epilepsy. Pediatrics 133:827–835
227. Samaniego EA, Sheth RD (2007) Bone consequences of epilepsy and antiepileptic medications. Semin Pediatr Neurol 14:196–200
228. Sheth RD, Gidal BE, Hermann BP (2006) Pathological fractures in epilepsy. Epilepsy Behav E&B 9:601–605
229. Wirrell EC (2006) Epilepsy-related injuries. Epilepsia 47(Suppl 1):79–86
230. Appleton RE (2002) Seizure-related injuries in children with newly diagnosed and untreated epilepsy. Epilepsia 43:764–767
231. Ach K, Slim I, Ajmi ST, Chaieb MC, Beizig AM, Chaieb L (2010) Non-traumatic fractures following seizures: two case reports. Cases J 3:30
232. Granhed HP, Karladani A (1997) Bilateral acetabular fracture as a result of epileptic seizure: a report of two cases. Injury 28:65–68
233. Zijlmans GJ, Huijbregts JE, van Nielen KM (2006) Collumfractuur in bed door een epileptische aanval [fracture of the femoral neck in bed caused by an epileptic seizure]. Ned Tijdschr Geneeskd 150:747–749
234. Haronian E, Silver JW, Mesa J (2002) Simultaneous bilateral femoral neck fracture and greater tuberosity shoulder fracture resulting from seizure. Orthopedics 25:757–758
235. Sikkink CJ, van der Tol A (2000) Unilateral transverse acetabular fracture with medial displacement of the femoral head after an epileptic seizure. J Trauma 48:777–778
236. Hughes CA, O'Briain DS (2000) Sudden death from pelvic hemorrhage after bilateral central fracture dislocations of the hip due to an epileptic seizure. Am J Forensic Med Pathol 21:380–384
237. Murzic WJ, Taylor JK, Bargar WL (1993) Seizure-induced femur fracture after total hip replacement. Orthopedics 16:906–908. discussion 908-909
238. Paris N, Journeau P, Moh Ello N, Haumont T, Randriabololona RA, Métaizeau JD, Lascombes P (2008) Décollement épiphysaire fémoral proximal bilatéral chez un nourrisson atteint d'épilepsie généralisée. A propos d'une observation [Bilateral upper femoral physis injury in a case of epilepsy in a young child]. Rev Chir Orthop Reparatrice Appar Mot 94:403–406
239. Aoudi K, Vialle R, Thevenin-Lemoine C, Abelin K, Mary P, Damsin JP (2009) Traumatic transepiphyseal separation of the upper femoral epiphysis following seizures in a 5-month-old child: a case report. Childs Nerv Syst 25:1039–1041
240. Atmaca H, Memişoğlu K, Baran T (2012) Neglected femoral neck fracture in patient with seizure: a case of a delayed fixation of type 1A fracture in 11-month-old infant. Eur J Orthop Surg Traumatol Orthop Traumatol 22(Suppl 1):173–176
241. Ballal MS, Dawoodi A, Sampath J, Bass A (2008) Traumatic transepiphyseal separation of the upper femoral epiphysis following seizures in two children with cerebral palsy. J Bone Joint Surg Br 90:382–384
242. Jacoby J, Nicholls AJ, Clarke NM, Fairhurst J (2011) Bilateral acromial fractures in a neonate with epileptic encephalopathy. Pediatr Radiol 41:788–789
243. Schnadower D, Agarwal C, Oberfield SE, Fennoy I, Pusic M (2006) Hypocalcemic seizures and secondary bilateral femoral fractures in an adolescent with primary vitamin D deficiency. Pediatrics 118:2226–2230
244. Presedo A, Dabney KW, Miller F (2007) Fractures in patients with cerebral palsy. J Pediatr Orthop 27:147–153
245. Lingam S, Joester J (1994) Spontaneous fractures in children and adolescents with cerebral palsy. BMJ 309:265
246. Sheth RD (2004) Bone health in pediatric epilepsy. Epilepsy Behav 5(Suppl 2):S30–S35
247. Babayigit A, Dirik E, Bober E, Cakmakci H (2006) Adverse effects of antiepileptic drugs on bone mineral density. Pediatr Neurol 35:177–181
248. von Harnack GA (1977) Säuglings-Skorbut (Möller-Barlowsche Krankheit). In: von Harnack GA (ed) Kinderheilkunde. Springer Verlag
249. Fain O (2005) Musculoskeletal manifestations of scurvy. Joint Bone Spine 72:124–128
250. Hodges RE, Baker EM, Hood J, Sauberlich HE, March SC (1969) Experimental scurvy in man. Am J Clin Nutr 22:535–548
251. Finberg L (1998) Concepts and diet analysis. Saunders manual of pediatric practice. WB Saunders Company, p 23
252. Rajakumar K (2001) Infantile scurvy: a historical perspective. Pediatrics 108:E76
253. Burches Greus E, Lecuona López C, Ardit Lucas J, Aguilar Bacallado F, García Vicent C, Tomás Ratés C, Alvarez Angel V, Donderis Folgado P (1991) Diagnóstico radiológico en la enfermedad de Möller-Barlow (Escorbuto). A propósito de un Caso

[radiologic diagnosis of Möller-Barlow disease (scurvy). Apropos of a case]. Anales espanoles de pediatria 34:243–246
254. Maroscia D, Negrini AP, Salsano G, Merola S, Travaglio MD, Contaldi G (1992) Scorbuto: una malattia non ancora scomparsa. A proposito di un caso [Scurvy: a disease that has not yet disappeared. Apropos a case]. Radiol Med 83:462–464
255. Hoeffel JC, Lascombes P, Mainard L, Durup de Baleine D (1993) Cone epiphysis of the knee and scurvy. Eur J Pediatr Surg 3:186–189
256. Riepe FG, Eichmann D, Oppermann HC, Schmitt HJ, Tunnessen WW Jr (2001) Special feature: picture of the month. Infantile scurvy. Arch Pediatr Adolesc Med 155:607–608
257. Harknett KM, Hussain SK, Rogers MK, Patel NC (2014) Scurvy mimicking osteomyelitis: case report and review of the literature. Clin Pediatr (Phila) 53:995–999
258. Ma NS, Thompson C, Weston S (2016) Brief report: scurvy as a manifestation of food selectivity in children with autism. J Autism Dev Disord 46:1464–1470
259. Golriz F, Donnelly LF, Devaraj S, Krishnamurthy R (2017) Modern American scurvy - experience with vitamin C deficiency at a large children's hospital. Pediatr Radiol 47:214–220
260. Hahn T, Adams W, Williams K (2019) Is vitamin C enough? A case report of scurvy in a five-year-old girl and review of the literature. BMC Pediatr 19:74
261. Gulko E, Collins LK, Murphy RC, Thornhill BA, Taragin BH (2015) MRI findings in pediatric patients with scurvy. Skelet Radiol 44:291–297
262. Nastro A, Rosenwasser N, Daniels SP, Magnani J, Endo Y, Hampton E, Pan N, Kovanlikaya A (2019) Scurvy due to selective diet in a seemingly healthy 4-year-old boy. Pediatrics 144
263. Duggan CP, Westra SJ, Rosenberg AE (2007) Case records of the Massachusetts General Hospital. Case 23-2007. A 9-year-old boy with bone pain, rash, and gingival hypertrophy. N Engl J Med 357:392–400
264. Polat AV, Bekci T, Say F, Bolukbas E, Selcuk MB (2015) Osteoskeletal manifestations of scurvy: MRI and ultrasound findings. Skelet Radiol 44:1161–1164
265. Weinstein M, Babyn P, Zlotkin S (2001) An orange a day keeps the doctor away: scurvy in the year 2000. Pediatrics 108:E55
266. Choi SW, Park SW, Kwon YS, Oh IS, Lim MK, Kim WH, Suh CH (2007) MR imaging in a child with scurvy: a case report. Korean J Radiol 8:443–447
267. Niwa T, Aida N, Tanaka Y, Tanaka M, Shiomi M, Machida J (2012) Scurvy in a child with autism: magnetic resonance imaging and pathological findings. J Pediatr Hematol Oncol 34:484–487
268. Brennan CM, Atkins KA, Druzgal CH, Gaskin CM (2012) Magnetic resonance imaging appearance of scurvy with gelatinous bone marrow transformation. Skelet Radiol 41:357–360
269. Ratanachu-Ek S, Sukswai P, Jeerathanyasakun Y, Wongtapradit L (2003) Scurvy in pediatric patients: a review of 28 cases. J Med Assoc Thailand = Chotmaihet thangphaet 86 Suppl 3:S734–S740
270. Paul DK, Lahiri M, Garai TB, Chatterjee MK (1999) Scurvy persists in the current era. Indian Pediatr 36:1067
271. Ahuja SR, Karande S (2002) An unusual presentation of scurvy following head injury. Indian J Med Sci 56:440–442
272. Yilmaz S, Karademir S, Ertan U, Kuyucu S, Hallioğlu O, Ocal B, Maviş N (1998) Scurvy. A case report. Turk J Pediatr 40:249–253
273. Caksen H, Odabaş D (2002) Keratomalacia and scurvy in a severely malnourished infant. Pediatr Dermatol 19:93–95
274. Narchi H, Thomas M (2000) A painful limp. J Paediatr Child Health 36:277–278
275. Najera-Martínez P, Rodríguez-Collado A, Gorian-Maldonado E (1992) Escorbuto. Estudio de 13 casos [Scurvy. A study of 13 cases]. Boletin medico del Hospital Infantil de Mexico 49:280–285
276. Cheung E, Mutahar R, Assefa F, Ververs MT, Nasiri SM, Borrel A, Salama P (2003) An epidemic of scurvy in Afghanistan: assessment and response. Food Nutr Bull 24:247–255
277. Miraj F, Abdullah A (2020) Scurvy: forgotten diagnosis, but still exist. Int J Surg Case Rep 68:263–266
278. Agarwal A, Shaharyar A, Kumar A, Bhat MS, Mishra M (2015) Scurvy in pediatric age group—a disease often forgotten? J Clin Orthop Trauma 6:101–107
279. Flaherty EG, Perez-Rossello JM, Levine MA, Hennrikus WL (2014) Evaluating children with fractures for child physical abuse. Pediatrics 133:e477–e489
280. Vitoria I, López B, Gómez J, Torres C, Guasp M, Calvo I, Dalmau J (2016) Improper use of a plant-based vitamin C-deficient beverage causes scurvy in an infant. Pediatrics 137:e20152781
281. Kitcharoensakkul M, Schulz CG, Kassel R, Khanna G, Liang S, Ngwube A, Baszis KW, Hunstad DA, White AJ (2014) Scurvy revealed by difficulty walking: three cases in young children. J Clin Rheumatol 20:224–228
282. Ceglie G, Macchiarulo G, Marchili MR, Marchesi A, Rotondi Aufiero L, Di Camillo C, Villani A (2019) Scurvy: still a threat in the well-fed first world? Arch Dis Child 104:381–383
283. Lund RM, Becker ML, Shapiro S, Allison T, Harris JG (2019) Scurvy presenting with limp and weakness: a case report. BMC Pediatr 19:228
284. Liebling EJ, Sze RW, Behrens EM (2020) Vitamin C deficiency mimicking inflammatory bone disease of the hand. Pediatr Rheumatol Online J 18:45
285. Gilman BB, Tanzer RC (1932) Subdural hematoma in infantile scurvy: report of case with review of literature. JAMA 99:989–991
286. Miura T, Tanaka H, Yoshinari M, Tokunaga A, Koto S, Saito K, Izumi J, Inagaki M (1982) A case of scurvy with subdural hematoma. Rinsho Ketsueki 23:1235–1240
287. Verma S, Sivanandan S, Aneesh MK, Gupta V, Seth R, Kabra S (2007) Unilateral proptosis and extradural hematoma in a child with scurvy. Pediatr Radiol 37:937–939
288. Aziz A, Matonti F, Bautrant V, Foletti JM, Denis D (2017) Infantile scurvy revealed by spontaneous orbital hematoma. Orbit 36:170–172
289. Bloxham CA, Clough C, Beevers DG (1990) Retinal infarcts and haemorrhages due to scurvy. Postgrad Med J 66:687
290. Adetona N, Kramarenko W, McGavin CR (1994) Retinal changes in scurvy. Eye (Lond) 8(Pt 6):709–710
291. Errera MH, Dupas B, Man H, Gualino V, Gaudric A, Massin P (2011) Une cause inhabituelle d'anomalies rétiniennes, hémorragies rétiniennes révélatrices de scorbut [Unusual retinal abnormality: retinal hemorrhages related to scurvy]. Journal francais d'ophtalmologie 34:186
292. Berant M, Jacobs J (1966) A "pseudo" battered child. Clin Pediatr (Phila) 5:230–237
293. Mimasaka S, Funayama M, Adachi N, Nata M, Morita M (2000) A fatal case of infantile scurvy. Int J Legal Med 114:122–124
294. Greeley CS (2011) Conditions confused with head trauma. In: Jenny C (ed) Child abuse and neglect diagnosis, treatment and evidence. Saunders Elsevier, pp 441–450
295. Pailhous S, Lamoureux S, Caietta E, Bosdure E, Chambost H, Chabrol B, Bresson V (2015) Le scorbut, Une vieille maladie toujours d'actualité: à propos de deux cas [scurvy, an old disease still in the news: two case reports]. Arch Pediatr: organe officiel de la Societe francaise de pediatrie 22:63–65
296. Solanki M, Baweja DK, Patil SS, Shivaprakash PK (2011) Ascorbic acid deficiency: a case report. J Dent Child (Chic) 78:115–119
297. Marquardt ML, Done SL, Sandrock M, Berdon WE, Feldman KW (2012) Copper deficiency presenting as metabolic bone disease in extremely low birth weight, short-gut infants. Pediatrics 130:e695–698

298. Brubaker C, Sturgeon P (1956) Copper deficiency in infants; a syndrome characterized by hypocupremia, iron deficiency anemia, and hypoproteinemia. AMA J Dis Child 92:254–265
299. Schubert WK, Lahey ME (1959) Copper and protein depletion complicating hypoferric anemia of infancy. Pediatrics 24:710–733
300. Graham GG, Cordano A (1969) Copper depletion and deficiency in the malnourished infant. Johns Hopkins Med J 124:139–150
301. Karpel JT, Peden VH (1972) Copper deficiency in long-term parenteral nutrition. J Pediatr 80:32–36
302. Ashkenazi A, Levin S, Djaldetti M, Fishel E, Benvenisti D (1973) The syndrome of neonatal copper deficiency. Pediatrics 52:525–533
303. al-Rashid RA, Spangler J (1971) Neonatal copper deficiency. N Engl J Med 285:841–843
304. Carty H (1988) Brittle or battered. Arch Dis Child 63:350–352
305. Cordano A, Baertl JM, Graham GG (1964) Copper deficiency in infancy. Pediatrics 34:324–336
306. Allen TM, Manoli A 2nd, LaMont RL (1982) Skeletal changes associated with copper deficiency. Clin Orthop Relat Res:206–210
307. Schmidt H, Herwig J, Greinacher I (1991) Skelettveränderungen bei Frühgeborenen mit Kupfermangel [The skeletal changes in premature infants with a copper deficiency]. RoFo : Fortschritte auf dem Gebiete der Rontgenstrahlen und der Nuklearmedizin 155:38–42

The Role of the Expert Witness and the Abuse of Differential Diagnoses in Court

15

Rick R. van Rijn and Rob A. C. Bilo

Contents

15.1 Introduction

The suspicion that a child sustained injuries in non-accidental circumstances (inflicted injuries, child abuse) does not only have a medical and social but also a potentially legal impact. This sets it apart from all other medical pediatric signs, symptoms, and physical findings in need of a proper differential diagnosis. Probably the most important difference is that when these cases go to court the suspicion can, and in most cases will, be debated by forensic medical experts/expert witnesses who act for the court, the public prosecutor, or for the defense.

It is the duty of an expert witness to explain the reasons for the suspicion and to explain the differential diagnoses of the physical findings to laypersons (e.g., child protection and the police) or to the participants in court procedures (judge, public prosecutor, and defense lawyer). The explanation has to be done in understandable lay language and in a neutral and preferably evidence-based way.

If the debate between medical experts is conducted in accordance with the current state of medical science, it creates an equal playing field between all participants, which are involved in the criminal court procedures.

The fact that a defense lawyer is allowed to question the conclusions of a medical expert on which the prosecution by the public prosecutor and the evidence of the prosecutor at least partially is based, is not only right but essential, as this forms the basis of proper and well-balanced legal proceedings. It is in the interest of the defendant and it offers him/her maximum protection. The defense lawyer should ensure that the rights of the defendant are respected and that he/she gets

R. R. van Rijn
Department of Radiology and Nuclear Medicine, Amsterdam UMC, University of Amsterdam, Amsterdam, The Netherlands

Department of Forensic Medicine, Netherlands Forensic Institute, The Hague, The Netherlands
e-mail: r.r.vanrijn@amsterdamumc.nl

R. A. C. Bilo (✉)
Veilig Thuis Rotterdam Rijnmond (Center for the Reporting of Child Abuse, Domestic Violence and Elder Abuse), Rotterdam, The Netherlands

R. A. C. Bilo et al. (eds.), *Forensic Aspects of Paediatric Fractures*, https://doi.org/10.1007/978-3-031-12041-1_15

a fair trial. In the case of Powell versus Alabama, the court stated "*Even the intelligent and educated layman has small and sometimes no skill in the science of law. If charged with crime, he is incapable, generally, of determining for himself whether the indictment is good or bad. He is unfamiliar with the rules of evidence. Left without the aid of counsel he may be put on trial without a proper charge, and convicted upon incompetent evidence, or evidence irrelevant to the issue or otherwise inadmissible. He lacks both the skill and knowledge adequately to prepare his defence, even though he have a perfect one. He requires the guiding hand of counsel at every step in the proceedings against him. Without it, though he be not guilty, he faces the danger of conviction because he does not know how to establish his innocence. If that be true of men of intelligence, how much more true is it of the ignorant and illiterate, or those of feeble intellect*" [1].

The debate, if done according to the rules of expert testimony, also gives the judges the opportunity to arrive at a well-balanced judgement.

15.2 Expert Witness

15.2.1 Duties and Responsibilities of the Expert Witness

The primary function of an expert witness is to express his independent expert opinion based on the information that is provided. An expert can be employed in different capacities, for example, at arbitrations, tribunals, and litigation. A witness is a person giving sworn evidence to a tribunal or court of law. There are basically two types of witnesses:

- *Witnesses of Fact who may give evidence of fact but may not normally give opinions.*

- *Expert Witnesses who may give opinion evidence within their expertise and in addition evidence of facts.*

An Expert Witness can be anyone with knowledge or experience in a particular field or discipline beyond that to be expected of a layman. The Expert Witness's duty is to give to the Court or tribunal an impartial opinion on particular aspects of matters within his expertise which are in dispute.

In England & Wales and many other jurisdictions the Court must give permission for an Expert Witness to give evidence.

An Expert Witness is not an expert adviser who is normally appointed by a party to assist in the formulation and preparation of a party's claim or defense. An expert adviser does not have an overriding duty to the court but to the party instructing him.

The Academy of Experts [2]

Already in 1897 Brouardel[1] made a statement about the duties and responsibilities of medical expert witnesses in court, which still is valid today: "*If the law has made you a witness, remain a man of science. You have no victim to avenge, no guilty or innocent person to convict or save – you must bear testimony within the limits of science*."

In a court case in the UK concerning a suspicion of child abuse, the judge quoted an overview of duties and responsibilities of experts, which came from a verdict by judge Cresswell, concerning a claim on a policy of marine insurance [3, 4]. This shows that duties and responsibilities of experts are always the same, irrespective of their field of expertise, whether this is marine engineering or forensic medicine:

[1]Forensic pathologist, chair of the Department of Forensic Medicine, Faculty of Medicine, Sorbonne University, Paris, France (1837–1906).

- *Expert evidence presented to the court should be and should be seen to be the independent product of the expert uninfluenced as to form or content by the exigencies of litigation.*
- *An expert witness should provide independent assistance to the court by way of objective unbiased opinion in relation to matters within his expertise. An expert witness in the High Court should never assume the role of advocate.*
- *An expert witness should state the facts or assumptions on which his opinion is based. He should not omit to consider material facts that detract from his concluded opinion.*
- *An expert witness should make it clear when a particular question or issue falls outside his expertise.*
- *If an expert's opinion is not properly researched because he considers that insufficient data is available then this must be stated with an indication that the opinion is no more than a provisional one.*
- *If after exchange of reports, an expert witness changes his view on a material matter, such change of view should be communicated … to the other side without delay and when appropriate to the court.*
- *Where expert evidence refers to photographs, plans, calculations … survey reports or other similar documents, these must be provided to the opposite party at the same time as the exchange of reports.*

In 2002 Butler-Sloss and Hall wrote an article on the interaction between medical expert witnesses, the courts, and the law [5]. They made the following statements about the serving and mandatory neutral role of medical experts in court and about the complicated interaction between law and medicine, in which in fact the message of Brouardel is repeated:

- *"Most judges have no more medical expertise than the average intelligent lay person. … are best assisted by experts who provide opinions based firmly on clinical findings and recognized medical knowledge. ....*
- *Medical opinions given should always be well researched and thorough. Expert witnesses must resist any urge to present without appropriate warning an opinion that is controversial in the profession. ….*
- *Of course, genuine disagreements will arise; but where an expert advances a hypothesis to explain an injury, he or she owes a very heavy duty to explain to the court that what is being advanced is a hypothesis, to say whether the hypothesis is widely accepted, and to place before the court all the material contradicting that hypothesis. ….*
- *Without them* (the medical experts)*, we could not do our job."*

In 2010 the judges in a UK appellate court (R v Butler, 2010) formulated the most important criterion about the medical differential diagnosis of physical findings in inflicted head injury in children, namely: "*…. a realistic possibility of an unknown cause must not be overlooked…*" [6]. Their opinion about the medical differential diagnosis of inflicted head injury is valid for all cases in which physical findings and medical experts play a role. In determining what constitutes a plausible medical differential diagnosis, one should always keep in mind that a differential diagnosis does not merely exist of a list of possibilities, but also is closely connected with the probability of certain possibilities. This was formulated by the judges in the UK appellate court in 2010 as "*a realistic possibility*." A good example of the difference between certain possibilities and the probability of these possibilities, although not primarily concerning fractures, was given in the 2008 article on the evidence in non-accidental head injury, which was written by David for Pediatric Radiology [7]. In this review he gave two tables, one of causes of subdural bleeding and one of causes of retinal hemorrhages. Although David stated that he did also include causes in adults and that these were not relevant in children (possibilities without probability), the tables are sometimes used as starting point in the differential diagnosis ("*diagnosis by exclusion*") in suspected inflicted head injury in children. This leads to a confusion of tongues, in which possibilities and probabilities are used as synonyms. One might wonder how realistic (how probable) breakdancing, head banging, weight lifting, or boxing are as a cause of subdural bleeding in a young child under the age of 1 year, although it has been described in the medical literature as causes of subdural bleeding in adolescents and adults. The same accounts for retinal hemorrhages. How realistic (how probable) are bungee jumping, altitude, crushing injury to chest, or chest compression from a safety belt as causes of retinal hemorrhage in an infant or a young child, despite their description in the adolescent and adult medical literature? Even if one looks at causes that were quoted by David and that could be relevant in infants, one should always ask how realistic (how probable), e.g., extra corporeal membrane oxygenation, diabetes, and sickle cell anemia are as possibilities in infants, given the findings and circumstances in a specific child.

As an expert witness, whether this is for the prosecution or for the defense, one should always keep in mind what the duties of an expert witness are in legal procedures or in court and what the court demands from the expert witness [8, 9]:

- Experts should provide a straightforward, not a misleading opinion. This implies that they must present arguments in a fashion that they truly believe to be as near as possible to the objective truth, not as a partisan biased in favor of one side or the other.

- Experts should be objective and not omit factors/facts which do not support their opinion. This selectivity of material in an expert's report may lead to what would appear to be an unbiased report becoming a misleading one.
- The expert's opinions and conclusions should be properly researched. This implies that the expert should be up to date with the relevant current medical literature.

In the USA the Daubert standard applies, this rule of evidence is used to assess the admissibility of expert witness testimony [10]. This standard states that: A witness who is qualified as an expert by knowledge, skill, experience, training, or education may testify in the form of an opinion or otherwise if:

- The expert's scientific, technical, or other specialized knowledge will help the trier of fact to understand the evidence or to determine a fact in issue.
- The testimony is based on sufficient facts or data.
- The testimony is the product of reliable principles and methods.
- The expert has reliably applied the principles and methods to the facts of the case.

15.2.2 The Radiology Report in Court

Much has been written on how to write a radiology report, whether it should be free prose or a structured/template report, and how to get your message across [11–19]. However, most of this is not based on evidence supported guidelines but on personal or institutional preferences. In general, a radiology report has to contain the following information:

- Patient data: In most cases this information will be automatically retrieved from the electronic medical record (EMR). But it is advised to check these details, especially in twins.
- Clinical history and study question: In most cases this information will also be retrieved from the study request as found in the EMR. However, in many cases this, depending on the referring specialty, will only contain a limited amount of information and it is therefore advised to check the EMR for more relevant information.
- Name, date, and time of the study: Especially when multiple similar studies are performed on 1 day the time of the study should be mentioned. Be aware that the reported time may differ from system to system, some will use the timestamp from the EMR, i.e., the time the study was ended by the technician, but others will use the time data as provided by the radiology hardware itself (e.g., the CT scanner or MRI scanner).
- Technique: It is important to state which imaging technique was used, the technical quality of the examination, and if national or international guidelines were followed [20, 21]. If the reported study has been obtained from a different hospital, this should specifically be mentioned.
- Comparison: State with which other examinations the current examination is being compared.
- Findings: A clear and concise description of all relevant imaging findings. Findings that are relevant to the clinical request or suspected pathology should be mentioned first [18]. Besides positive findings, relevant negative and incidental findings should also be described. The use of ambiguous terms and abbreviations should be avoided.
- Conclusion: This is the most important part of the radiology report, here all the items come together in a short conclusion stating the most likely or definite diagnosis. If it is not possible to come to a diagnosis or in case of an unknown imaging finding, the radiologist should honestly state this and not hide this in verbose prose. The conclusion is also the place to recommend follow-up imaging, according to national or international guidelines, or additional imaging necessary to clarify unclear imaging findings.

Although the radiology report is the most important way for radiologists to communicate their findings, it is good to put some emphasis on the use of the radiology report in court. In a normal clinical setting, the report is primarily used to convey the radiology findings to another clinician of whom the radiologist can expect a proper level of clinical knowledge. This is in contrast to radiology reports that might or will be used in a court of law, and that will be read by laymen such as police, prosecution, and lawyers [22, 23]. Although every radiology report could in theory become a document in legal proceedings, the likelihood of that happening in case of suspected child abuse is significantly higher. The reporting radiologist should be aware of that. The findings reported or missed can have far-reaching consequences in legal proceedings. This is especially the case when reporting for court as an expert witness. Besides the issues addressed in the next section, there are some items that the radiologist should keep in mind when writing these expert reports.

- Know your readership: In most cases legal reports will be read by non-medical trained readers. Although most of us will consider it obvious to replace humerus with upper

arm, there are numerous routine words, e.g., posterior and proximal, that can also lead to confusion. It is good to communicate with non-physicians to see if they understand what you intend to express.

- Keep it organized: Although most radiologists will prefer the use of prose in their reports, the use of a structured report has been shown to decrease the risk of missing findings or failing to report these findings [19, 24]. We advise radiologists to use structured or standardized reports whenever possible. A potential structure for a forensic radiology expert report could be:
  1. Qualifications as an expert.
  2. Materials available for review.
  3. Clinical and forensic information available at time of review.
  4. Radiological findings; per study in a structured way.
  5. Interpretation of the radiology findings from a forensic perspective.
  6. Conclusion; preferably using a Bayesian approach (Chap. 17).
  7. Expert statement; stating that the findings are based on the experts' own opinion and that this opinion is unbiased [25].
- Take your time: In contrast to daily routine where timely reporting is of importance to patient care, it is important to plan time to write a forensic radiology expert report. Under normal conditions proofreading is advised but in forensic reporting this is of the utmost importance. Many radiologists will make use of some form of voice recognition and although this has the advantage of significantly improving turnaround times it comes at a price being recognition errors. Overall voice recognition errors in reports have been shown to range from 4.8% to 89%, with reported word errors up to 38.7% [26, 27]. Several studies have shown that this percentage is significantly higher compared to professional transcribed reports. Although voice recognition errors can be a source of amusement, it can lead to serious errors as well, e.g., one versus none. Beside recognition errors care has to be taken in other aspects such as to count correctly (e.g., rib level) and correctly use left and right.
- Use evidence: In contrast to clinical work where experience is, in general, sufficient to come to a diagnosis in court experience only is not sufficient. Preferably every conclusion, e.g., accidental versus non-accidental, timing of injuries/fractures, should be accompanied with relevant literature references.
- Keep an open mind: Consider a relevant differential diagnosis and if the findings are more in keeping with a disease report it like that and do not overstate or understate radiological findings. It is not the radiologist's role to provide evidence in favor of child abuse but to objectively report his/her findings and place them in an evidence-based perspective.

15.2.3 Guidelines

There are several radiological professional societies that have issued guidelines for expert witness in court [25, 28, 29]. These guidelines echo what has been mentioned and what is discussed in Sect. 15.2.1 and also advises on how to approach expert witness casework. Besides these dedicated guidelines, most countries will have general guidelines explaining the duties of expert witnesses in criminal or civil court (e.g., [30]).

A broader guideline was presented by the Ray E. Helfer Society in 2020. After a consensus-building process involving all members of the society presented new guidelines detailing standards for testifying in cases of suspected child maltreatment were published [31]. For this guideline, the authors used the 2017 guidelines on "Expert Witness Participation in Civil and Criminal Proceedings" of the American Academy of Pediatrics as a foundation [32]. Most of the points addressed in this guideline have been discussed above, but certain points of interest will be highlighted here:

- *Unless retired from clinical practice, expert witnesses should be actively engaged in clinical practice in the medical specialty or area of medicine about which they testify. If retired from clinical practice, they should demonstrate active engagement in the field (*e.g.*, CME or conferences on child maltreatment).*
- *Compensation for expert witness work must not be contingent on the outcome of the case.*
- *The testimony presented should reflect generally accepted standards within the expert's specialty or area of practice. Physician experts who know that their opinions are held by a minority of their community should state that transparently.*
- *When raising a differential diagnosis to explain findings that may be the result of abuse, physician experts should include only disease entities that are generally accepted in clinical medicine, and which could reasonably be relevant to the case at hand.*
- *A series of patients assembled from an individual's work as an expert has a high risk of selection bias, which limits generalizability. If a case or series of cases is identified or assembled from a physician's work as an expert, this should be clearly stated in the methods section of the related manuscript.*

- *Individuals who seek to publish data from cases in which they served as expert witnesses should clearly report their role and whether they received remuneration to the journal and within the published manuscript.*

In this guideline the authors, on behalf of the Ray E. Helfer Society, clearly state that the society has a policy in place in order to ensure that members can be held accountable for violations of these standards. They also make a strong statement toward hospital administrations to hold its faculty accountable for unethical testimony in court proceedings. Unfortunately this currently often is not the case and defense witnesses who regularly cross the lines of acceptable behavior are permitted to continue to work and use their affiliations in court testimonies.

Finally, based on our own experience, we advise every radiologist who is asked to appear in court to:

- Ask advice from colleagues who have appeared in court before. This will take away some of the anxiety when appearing in court.
- Familiarize him/herself with the local legal guidelines and practices. And to understand the role that each professional plays in court. Knowing and understanding this will avoid discussions, misunderstandings, and disillusion in the legal system.
- To use a standardized expert report, for this several standard forms can be found online.
- Stay within his/her own field of expertise and not answer any questions outside that field.
- Every expert report should end with a statement similar to the one proposed by the Royal Australian and New Zealand College of Radiologists "*[the Expert Witness] has made all the inquiries which [the Expert Witness] believes are desirable and appropriate and that no matters of significance which [the Expert Witness] regards as relevant have, to [the Expert Witness's] knowledge, been withheld from the Court*" [25].

15.2.4 The Expert Witness in Court: The Differential Diagnosis

> In theory there is no difference between theory and practice, while in practice there is.
> – Benjamin Brewster [33]

All duties and responsibilities, as mentioned in Sect. 15.2.1, will be agreed upon by all medical experts involved in court cases. However, litigation all over the world (especially in the USA) has led to the emergence of differential diagnoses (alternative explanations) by medical "experts" that lack merits. This phenomenon is seen occasionally in medical literature but also in the courtroom where it can lead to erroneous or delayed court rulings [34–37]. It is interesting to note that many alternative explanations are already used for decades in court cases. It may concern existing medical condition, in which physical findings are wrongly interpreted, manipulated, and/or over-interpreted. Examples are osteogenesis imperfecta, rickets, vitamin C deficiency, and Ehlers–Danlos syndrome [38]. However, it may even concern non-existing medical conditions, like temporary brittle bone disease [39, 40]. Despite the fact that these existing and non-existing alternative explanations over and over have been rejected by solid medical evidence, these explanations still pop up in court, either unchanged or slightly adapted.

In 1991 Taitz, a pediatrician from Sheffield, evaluated a series of court cases (1981 to 1990) in which he was asked to provide evidence [41]. The reason for this evaluation was a claim (among others) by Paterson and McAllion in 1989 that "*children with fractures (usually multiple) attributed to child abuse may in fact have metabolic bone diseases, such as osteogenesis imperfecta*" [42]. The evaluation of Taitz concerned 22 infants and children with fractures (mean age 6 months), in whom non-accidental trauma was suspected. The alternative explanations he encountered in these court cases were osteogenesis imperfecta in 9 children, copper deficiency in 2, metabolic disease of prematurity in 2, and in eight osteogenesis imperfecta, copper deficiency, and "temporary brittle bone disease" were all offered as alternative diagnoses. Fourteen children had at least one rib fracture, associated with limb fractures in 10 children and with limb and skull fractures in one child. Sixteen children had rib fractures or metaphyseal fractures, or both. In two patients, who were siblings, type I osteogenesis imperfecta had been diagnosed. Apart from the fractures, the bones were radiologically normal in all except the two children with type I osteogenesis imperfecta. No evidence of copper deficiency, such as deficient copper intake associated with extreme prematurity or malnutrition, or of osteopenia, delayed bone age, refractory hypochromic anemia, or neutropenia was found in any of the 10 children in whom it had been suggested. Temporary brittle bone disease was proposed as a diagnosis for seven children. The fractures ceased immediately after the children were placed in foster care. All children had additional evidence of abuse (Table 15.1). In 20 cases the courts

Table 15.1 Evidence of child abuse in children and infants with unexplained fractures [41]

Brain damage	1
Multiple evidence of abuse and neglect	2
Subdural hemorrhage	2
Facial bruises and torn frenulum	2
Multiple bruises and oral injury	1
Multiple bruises	5
Previous bruising suggestive of non-accidental trauma	1
Neglect	2
Confession by perpetrator	1
Fractures only	5

decided that the fractures were inflicted. In the remaining two with proven osteogenesis imperfecta type I, there was other evidence of abuse. The children were placed in foster care and only one further fracture occurred, 4 years after placement, when the child fell off a wall. In their 1989 article Paterson and McAllion stated about bruising in children with fractures due to osteogenesis imperfecta: "*Though bruising with little or no obvious trauma is a well-recognised feature of osteogenesis imperfecta, it is a paradoxical but frequent finding that fractures may not be accompanied by the superficial evidence of trauma that might be expected*" [42]. Taitz found that in the evaluated cases it was claimed in 10 children that absence of bruising at the site of the fracture was evidence of metabolic bone disease. However, according to Taitz, 9 of these 10 children had clear additional evidence of non-accidental trauma.

An example of introducing a new alternative explanation is found in a 2010 case report by Barnes et al., concerning a 4-month-old child. The child collapsed unexpectedly and was diagnosed with subdural, subarachnoid, and retinal hemorrhages and eventually fatal cerebral edema [43]. According to the authors, the occurrence of the hemorrhages and fatal edema could be explained by dysphagic choking and the following efforts to resuscitate the child. The report was severely criticized. Greeley (2014) used this case report to illustrate that: *'Theories appear to have been created not with a clinical gap to fill, but explicitly to be used in court—to be "backwards compatible" from the courtroom into clinical relevance. An example of an attempt at a "backwards compatible" theory was published as a case report in 2010 (by Barnes et al.)* [44].' Concerning the case-report Greeley also mentioned also that *'each of the authors served as expert witnesses for the defence in the trial which convicted the infant's father'* and that *'the authors excluded important information, such as a healing rib fracture, from their report.'* According to Greeley, the report was also not peer reviewed. Edwards (2015) also stated that the authors had omitted important information (cited from the article by Edwards [45]):

- *Although the authors did not disclose their source of information or their role as expert witnesses for the defense in the criminal trial that arose from the child's death, there is unequivocal evidence that the report was based on the Zavion Thomas, Jr. case from Austin, Texas. Both the child in the case report and the patient in Austin died at 4 months of age. When admitted to the hospital, both had identical initial laboratory data. Identical images to those included in the case report can be found in the imaging findings of the child from Austin….*
- *Clearly the case report was based on the child who died in Austin, Texas. Nevertheless, the report contained omissions and misrepresentations of the facts of the case.*
- *Four month-old Zavion Thomas, Jr., for whom this author provided consultation, died as a result of abusive head trauma in May 2006 in Austin, Texas. He had rib fractures in different stages of healing, diffuse brain swelling, bilateral subdural hemorrhages, subarachnoid hemorrhages, cerebellar herniation, diffuse swelling of the cervical spinal cord, and extensive bilateral retinal hemorrhages with bilateral retinoschisis and detachments as well as hemorrhage into the posterior third of both orbits. The child's biological father was convicted of serious bodily injury to a child and sentenced to 55 years in prison. His conviction was upheld on appeal....*
- *The case report also omitted the fact that the child had a healing rib fracture. In fact, the child had a healing rib fracture with callus formation of the right lateral seventh rib, in addition to three recent rib fractures. …. Therefore, there is reason to believe that rib fractures after C-sections are very unlikely. This child had been delivered by C-section.*
- *The case report misrepresented the pediatric ophthalmologist's findings. Although there were bilateral retinoschisis and retinal detachments, the authors mentioned only "extensive bilateral RH [retinal hemorrhage] and retinal elevation." They omitted the retinoschisis.*
- *The case report misrepresented the child's bleeding. It stated, "Clinically the patient was having bleeding from IV sites and his ears." In fact, he never had any clinically significant bleeding, although he had IV lines, an arterial line, and numerous venipunctures, including femoral and external jugular punctures.*
- *The case report suggested that a venous thrombosis played a role in the child's problems. However, the pediatric radiologists and neuroradiologists who reviewed the child's imaging studies saw no evidence of venous thrombosis, and the autopsy found no evidence of venous thrombosis. Furthermore, it is doubtful that any causal relationship exists between intracranial venous thrombosis and subdural hemorrhage in young children.*
- *The case report misrepresented the autopsy findings. It described the autopsy stating, "Although NAI [nonaccidental injury] could not be ruled out, the autopsy findings provided further evidence that the child's injury could result from a dysphagic choking type of acute life threatening event (ALTE) as consistently described by the caregiver." In fact, the autopsy showed a well-healed fracture of the right seventh rib; three recent fractures of the right fifth, sixth, and seventh ribs; marked swelling of the brain; bilateral subdural hemorrhages; subarachnoid hemorrhages; and herniation of the cerebellar tonsils. The Medical Examiner stated, "It is my opinion, based on the investigation of the circumstances and the autopsy findings that the decedent … came to his death as a result of severe closed head injury (shaken baby syndrome)." He concluded that the manner of death was homicide.*
- *The case report omitted the legal outcome. The authors stated, "The injuries in this case were attributed to shaken baby syndrome before the brain injuries were completely*

evaluated. The father was charged with fatally shaking the child." In fact, the father was charged with causing serious bodily injury to a child by shaking, impact, and/or both. He was also charged with capital murder. The jury found him guilty of causing serious bodily injury to a child and sentenced him to 55 years in prison. The jury deadlocked on capital murder, and a mistrial was declared on that charge. His subsequent appeal was unsuccessful.

- *In addition to the omissions and misrepresentations, the case report is not consistent with the authors' sworn testimony regarding the cause of the child's death. When the authors testified at the father's trial, they expressed the opinion that the child's death occurred as a result of choking that resulted in hypoxia. They argued that the hypoxia then caused brain damage, disseminated intravascular coagulopathy (DIC), intracranial bleeding, and retinal hemorrhage. They never made reference to increased intrathoracic or vascular pressure from choking. None of the authors used the phrase "dysphagic choking" in their testimony. They explained that hypoxia from choking caused the brain damage and that hypoxia and DIC caused the intracranial and ocular bleeding. In contrast, their assessment in the case report was that dysphagic choking caused increased intrathoracic pressure that resulted in increased vascular pressure in the head. They indicated that the increased vascular pressure would cause rupture of blood vessels with resulting subdural, subarachnoid, and retinal hemorrhaging.*

Edwards concluded: "*The use of scientifically unsupported alternative explanations for abusive head trauma based on inaccurate and biased information constitutes further victimization of the abused child and represents a travesty of justice*" [45].

With the previous data in mind, several alternative explanations/differential diagnoses which have been presented in the past in court and are still regularly presented will be discussed in the following sections. These alternative explanations were either found in verdicts of court cases or in the medical literature. We also included explanations we encountered during our own expertise work in court procedures.

> The court is not an appropriate forum for the presentation of new or unsubstantiated theories of causation of disease. Rather, the court must ensure the accuracy of legal decisions.
> –Servaes et al. [46]

15.3 Abuse of Existing Diseases in the Differential Diagnosis

The differential diagnoses discussed in this part of the chapter are all related to diseases which indeed are real and can have a place in the differential diagnosis of fractures in infants and children. For the clinically relevant information the reader is referred to Chap. 14 of this book. In this part of the chapter, we will only discuss the abuse of these differential diagnoses in court.

15.3.1 Fractures, Bruises, and Non-Accidental Trauma

Taitz already showed in 1991 that one cannot draw any conclusion from the absence of bruises in children with fractures, concerning the amount of energy required to cause the fractures or concerning the presence of a skeletal disease with an increase of fractures (e.g., osteogenesis imperfecta) [41]. He also showed that one cannot draw any conclusions concerning the circumstances under which the fractures were sustained (accidental or non-accidental trauma) from the absence of bruising near the location of fractures. Despite the evidence given by Taitz (Sect. 15.2.4), some medical experts/authors wrongly and without any evidence still maintain that the force necessary to fracture a normal bone should lead to externally visible evidence of trauma (bruising), mainly based on the suggestion of Paterson and others [ref]. Furthermore the absence of bruising would proof that only very little force is needed to break a bone and, as such, that the fracture results from a skeletal disease, e.g., metabolic bone disease of infancy, osteogenesis imperfecta, or copper deficiency [42, 47, 48]. In 1994 Paterson stated in a court case, concerning suspected child abuse in a 10-week-old boy with several fractures, that: "*Had these fractures been sustained as a result of a series of deliberate injuries inflicted on a child with normal bones, it would be almost inconceivable that evidence of such injuries would not be obvious*" [3]. Since then a lot of research has been published confirming the initial 1991 conclusions of Taitz and contradicting the opinion of Paterson:

Mathew et al. performed a prospective study into the presence of bruising around the location of the fracture in 88 children (with a total 93 fractures) that showed no signs of bone pathology with in total 93 fractures (49 boys, 39 girls; age 12 months to 13 years and 11 months) [48]. All children were seen within 24 h after the fracture had been sustained. There were 17 undisplaced, 46 displaced, and 30 angulated fractures. Seventy fractures were sustained in simple falls and 23 fractures in falls from heights. Only in eight (9%) fractures, either displaced or superficially located, bruising was found in the initial phase. No bruising was found in fractures that showed no dislocation or in fractures that were well covered by soft tissue. In 13 other fractures, bruising appeared within 24 h after hospitalization. Ultimately, 25 (28%) fractures were accompanied by bruising 1 week after the fracture was sustained. According to Mathew et al. it is impossible to distinguish between fractures that are the result of bone disease and fractures, sustained in non-accidental circumstances based on the presence or absence of bruising

near the location of the fracture. It appears that in acutely sustained fractures in children, local bruising is less common than one would expect. Based on the absence of bruising, non-accidental trauma should never be excluded.

Starling et al. analyzed the relationship of perpetrators to their victims in case of inflicted skeletal trauma [49]. They evaluated a total of 630 fractures in 194 patients (median number of fractures per patient 2). The maximum number of fractures in a single patient was 31. Of the 630 fractures, the sites most commonly fractured were the ribs (50.2%), legs (21.8%), and arms (12.9%). They did not find any relation between fractures and the presence of bruising. Although bruising was the most commonly associated injury, only 20.8% of the children had a bruise overlying a fracture. After scalp bruises and subgaleal hematomas associated with skull fractures were excluded, less than 10% of children had fracture-related bruising.

Peters et al. evaluated the findings in 192 children with inflicted fractures [50]. In 111 children no bruising was found (57.8%). Forty patients (20.8%) had bruising near the site of at least 1 fracture. Of these, bruising or subgaleal hematoma near the site of a skull fracture was seen most often (43.3% of children with calvarium fractures). Bruising associated with extremity fractures was seen much less commonly, ranging from 3.8% ($n = 2$) of children with tibia fracture to 16.7% ($n = 1$) of children with fibula fracture. Rib fractures also were associated uncommonly with bruising. When skull fractures are excluded, 45 (8.1%) of 555 fractures had bruising near the fracture site, in 13 (6.8%) patients.

Valvano et al. did a retrospective study into the value of presence or absence of bruising in the differentiation between abusive and non-abusive fractures in 150 children with acute fractures [51]. A bruise and fracture were considered by the authors associated if both occurred on the same body site. Ninety-three children had fractures due to non-accidental trauma and 57 to accidental trauma. Bruising associated with a fracture was found for 26% of abused and 25% of non-abused children. Most children (61%) had no bruises anywhere on the body, and this did not differ significantly by cause of injury. The authors concluded that the presence or absence of bruising was not useful to differentiate between abusive and non-abusive fractures.

15.3.2 Vitamin D Deficiency: Rickets

As already discussed in Sect. 14.4 to the untrained eye the radiological findings in a child with rickets can lead to a suspicion or even an incorrect conclusion that the fractures in a child were inflicted. Rickets due to vitamin D deficiency acquired a status as courtroom diagnosis in the first decade of the twenty-first century [52].

However, on the other side, in court the suggestion or even the incorrect ascertainment of rickets as alternative explanation for the occurrence of fractures in a young child can lead to incorrectly motivated judicial rejection of the suspicion of child abuse, and the risk of continued, because not legally proven, child abuse [53, 54].

15.3.2.1 Vitamin D Levels

In Sect. 14.4.4, biochemical testing in rickets is presented. However, there are experts with an outlying view on normal vitamin D levels. In a review article, Holick defined a 25 (OH)D level above 30 ng/mL (75 nmol/L) as normal, 21–29 ng/mL (52 to 72 nmol/L) as indicative of insufficient, and under 20 ng/mL (50 nmol/L) as deficient. By using these values Holick established that, based on data derived from two articles, vitamin D deficiency is present in 52% of Latin-American and African-American adolescents in Boston and in 48% of pre-adolescent white girls from Maine [55–58]. However, Holick's definition of normal 25 (OH)D values, insufficiency, and deficiency was refuted in letters submitted to the New England Journal of Medicine and the authors of the submitted letters state that these values were not medically supported [59, 60]. There is also a discrepancy between the recommendations of Holick and the guidelines of the American Academy of Pediatrics, who in 2017 adopted the recommendations, as described by Munns et al. [61, 62]. It is of importance to note that Holick is one of the strongest propagators for the use of vitamin D supplements and that as such he reportedly has received significant support from the vitamin D industry [63, 64].

15.3.2.2 The Keller and Barnes Commentary on Rickets Versus Child Abuse

In 2008, a commentary by Keller and Barnes on the topic of rickets versus (child) abuse was published in Pediatric Radiology [53]. This commentary can be considered to be a hallmark publication concerning the use of rickets as a courtroom diagnosis. A large part of the text is dedicated to the epidemiology of rickets in children and mothers. Subsequently, Keller and Barnes presented four cases of infants, aged 2–4 months, with multiple fractures. According to the authors, all mothers presented with a decreased vitamin D level. However, none of the children had been checked for their 25(OH)D levels at the time when the fractures were diagnosed. According to the authors, none of the cases was suspect of child abuse. Furthermore, they literally posed the question: "*Would children with so many inflicted fractures not be in serious pain or be restless?*." The remaining part of the publication discussed the similarities between rickets and child abuse.

Because of the, in the meantime acquired, status of the commentary in court and the impact on jurisprudence, due to the use of the commentary by certain expert witnesses, it warrants a careful evaluation.

At the time the article was published, it was accompanied by four comments in the same issue of Pediatric Radiology

[65–68]. The main point of criticism was that it was absolutely not clear how the children that were described had been selected for the commentary of Keller and Barnes.

Jenny, at the time head of the American Academy of Pediatrics Section on Child Abuse, suspected that the cases had been provided by lawyers [68]. Jenny stated that the authors presented several cases of infants with multiple bony lesions and that they in the case presentations implied that these children were suffering from vitamin D deficiency rickets, despite the fact that the diagnosis of rickets apparently was not made in any of the children. She also mentioned that it was not stated in the commentary whether only a suspicion arose because of the finding of multiple fractures in the reported children or that the suspicion in the reported children led to a diagnosis of child abuse. Jenny also stated: "*In my practice, a child protection program in a northern climate that evaluates over 1800 children per year for alleged abuse or neglect, we have been checking every child with multiple fractures for metabolic bone diseases for several years and have not yet identified a single child with vitamin D deficiency. One of my colleagues, however, did find one child, a solely breast-fed 9-month-old with obviously demineralized bones.*"

Slovis and Chapman also criticized the obscure way in which the patients had been selected: "*The authors do not give us their selection criteria for the patients presented, i.e. exclusion criteria and total pool from which they were selected. It appears that the patients were selected from among those involved in litigation concerning whether child abuse was present*" [66]. The authors also remarked that only one of the children had vitamin D levels or calcium phosphate, alkaline phosphatase, or parathyroid hormone values reported at the time they were supposed to have rickets. They continued that all children, that were presented in the commentary, were under the age of 4 months and consequently must have suffered from congenital rickets. In all four cases, laboratory tests that could have confirmed this fact, were absent. Indeed, in the medical literature one can find descriptions of children with congenital rickets, but in a critical review of the literature cited by Keller and Barnes, the children that had been described (six of the seven children were radiologically examined) were shown to have metaphyseal lesion that corresponded with rickets [68–73]. Anyway, these peer-reviewed publications were a selection from the medical literature and cannot be considered to be a systemic review of the available literature at the time of the publication of Keller and Barnes. With regard to the fractures, purely on a radiological basis all one could conclude, according to Slovis and Chapman, was that they were present. It was remarkable that one child showed a spinal fracture which Keller and Barnes contributed to rickets, although in the literature there are no cases found, in which spinal fractures were attributed to rickets. This does not exclude the possibility, but it does not make it very probable either.

In 2009 Feldman wrote a letter to the editor of Pediatric Radiology, in which he explained his involvement in 3 of the 4 cases (cases 1, 2, and 4) that were presented by Keller and Barnes in their commentary [74]. At the end of the letter, Feldman stated: "*Neither Dr. Keller nor Dr. Barnes provided clinical care to these children. All the cases came to them as defense witnesses in the legal system. It is a serious breach of research bias for them to not disclose the source of their case material in their article. Likewise, both physicians are active as defense witnesses in child abuse cases. It is a serious breach of conflict of interest to not disclose in their article that they profit personally from promoting the existence of congenital rickets as legitimate disease and as an explanation for multiple fractures in young infants.*"

Feldman also mentioned the verdicts in the cases: "*In case 2 the dependency judge ruled that the child had been abused and opined that there was sufficient evidence for a criminal conviction. In case 4 the child was ruled in dependency court to have suffered abuse and in criminal court the child's father was convicted of the abuse. Case 3 was placed in a relative's care under court-ordered dependency. In case 1 the dependency judge ruled in favor of the parents, largely on the basis of Dr. Keller's testimony and quirks of how the trial proceeded. A different judge heard the defense's case about a half-year after the state's case was presented. There was no opportunity to present rebuttal of Dr. Keller's testimony.*"

Feldman concluded, concerning the lack of information given by the authors about the court procedures: "*In any case, it is a serious breech of ethical disclosure, even if Keller and Barnes disagreed with those outcomes, to assert in their article that these cases prove the existence of congenital rickets as a cause of infant fractures, without disclosing that bodies as significant as the trial courts deemed their opinions to be incorrect*."

The suspicion of Jenny and of Slovis and Chapman, that the Keller and Barnes cases were provided by lawyers and/or came from civil and criminal court procedures, turned out to be correct, as shown by Feldman and, in response to the letter of Feldman, admitted by Keller and Barnes: "*The four cases in our rickets vs. abuse commentary indeed represent alleged child abuse cases that we reviewed on behalf of the defense*" [74, 75].

In court procedures, concerning suspicion of non-accidental trauma in children, fact finding is of the utmost importance. In case of court procedures judicial evidence to the benefit or detriment of the accused, which is based on the findings in a (forensic) medical evaluation (medicine-based evidence), should be scientifically sound and preferably evidence based. If partially or completely based on case reports the information in the case reports should be undisputed and easy to evaluate by a medically trained outsider on the basis of the information described in the case report. No information, vital to a proposed diagnosis (in this situation rickets), should be missing.

The discussion between Keller and Barnes on the one hand and Feldman on the other, concerning the findings in the cases, the interpretation of the findings and the outcome of the court procedures, does not offer enough information to reliably and objectively interpret the findings in the cases as described by them (initially and in response), without choosing sides in the discussion.

In other words, it is not possible, based on the information in the case reports, to establish all (forensic) medical facts in the cases, described by Keller and Barnes, except that these cases came from court procedures, that all cases concerned children with fractures, and that Keller and Barnes (as expert witnesses for the defense) disagreed with Feldman (as expert witness for the prosecution) about the circumstances under which the fractures were sustained. This way of selecting cases to proof an alternative explanation greatly increases the risk of selection bias and makes correct interpretation of the data impossible.

Because of this, Greeley's statement in response to a 2010 case report by Barnes et al. seems already to be valid in 2008 concerning this commentary by Keller and Barnes: "*Theories appear to have been created not with a clinical gap to fill, but explicitly to be used in court—to be "backwards compatible" from the courtroom into clinical relevance*" [43, 44].

In 2009 Strouse stated in an editorial in Pediatric Radiology, concerning the discussion, that arose in response to the commentary by Keller and Barnes: "*With the letters to the editor from Feldman and Keller and Barnes, we hereby conclude this foray into the abyss of vitamin D deficiency, rickets and child abuse. Further debate is welcomed in your local coffee house, conference room, an online chat room or other chosen arena*" [74–76]. After this statement Strouse returned to the only facts, that can be used in a forensic pediatric evaluation and were, at the time of publication of the editorial, evidence based and not hypothetical:

- *"There is a pandemic of vitamin D deficiency in the world. Evidence of widespread vitamin D deficiency in women of childbearing age is irrefutable. The significance of the maternal deficiencies to their infants is not fully determined.*"
- *"There is no concrete evidence in the literature that vitamin D deficiency in infants younger than 6 months of age renders them susceptible to the same types of fractures as have been accepted to bear high specificity for child abuse.*"

He concluded the editorial with:

- *"Agree or disagree, Keller and Barnes have set forth a hypothesis in their commentary and the following letter. It is an unproven hypothesis.*"

In 2012, Slovis et al. wrote in an editorial about inflicted head injury in children the following [77]:

- *"... There is no scientific evidence to connect vitamin D levels to fractures in the fetus or neonate.*"
- *"The vitamin D level is being used by some to obscure the difference between vitamin D insufficiency/deficiency and rickets—a bone disease defined by biochemical and radiographic features. The National Institute of Medicine has declared that >20 ng/ml of the 25-OH form of vitamin D is adequate for bone health; in other words, children with greater than this level do not suffer fractures due to vitamin D deficiency* [78]. *There is no scientific evidence to establish fractures as being secondary to rickets without the concomitant biochemical profile and radiographic or histological bone changes of rickets.*"

In 2013, 5 years after the original commentary, Strouse stated in Pediatric Radiology [79]:

- *"So what has happened in the 5 years since "Keller & Barnes" was published?*
- *Since 2008, neither Keller nor Barnes has published an academic work on rickets, vitamin D deficiency, or radiography of child abuse. Nothing.*
- *Looking broadly beyond Keller and Barnes, since 2008 no study in the literature has shown that fractures considered to be highly specific for child abuse are caused by vitamin D insufficiency or deficiency.*"
- *"To the contrary, since 2008 there have been studies that serve to refute the hypotheses of "Keller & Barnes"* [80–82].
- "*The hypotheses set forth in "Keller & Barnes" thus remain hypothetical and unsubstantiated.*"
- "*Perhaps better stated, the hypotheses of "Keller & Barnes" have been disproved. Whether in legal proceedings or in medical literature, it is inexcusable and inappropriate to cite "Keller & Barnes," particularly without simultaneously citing the accompanying commentary by Slovis and Chapman* [2] *and the subsequent commentary by Feldman* [5]. *To do so is deceptive. Moreover it is deceptive to cite "Keller & Barnes" without the context provided by the subsequent studies noted in the previous paragraph.*"
- *"Our understanding of metabolic bone disease in infants continues to improve. The commentary by Keller and Barnes made us pause to learn more. Otherwise, it changed nothing.*"

As earlier mentioned in this book, the ascertainment of non-accidental trauma in children is team work, and not a result that by itself is pathognomonic for the ascertainment. In

the cases that were presented by Keller and Barnes, relevant data enabling adequate evaluation are absent. Missing relevant data included the presence of other signs of trauma, indicative dating of fractures or indications for accidental trauma.

Finally, it is important to realize that, even if one assumes that the children, that were reported by Keller and Barnes, were indeed suffering from congenital rickets, this in itself does not exclude child abuse.

15.3.2.3 Other Publications Concerning Rickets Versus Child Abuse

Although the commentary of Keller and Barnes cannot withstand the critical test with regard to the description of the four cases and the link between rickets and non-accidental trauma, it still is cited in court cases. In the meantime several new articles have been published in which the hypothesis of Keller and Barnes is more or less repeated, again without sound scientific evidence and/or acceptance by the medical scientific community. Consequently, any person involved in child protection or in forensic pediatrics should be familiar with these articles and the related comments.

In 2020 Strouse stated in an editorial concerning inflicted head injury in children [83]: "*Others have put forth rickets and vitamin D deficiency as an explanation for the skeletal findings of child abuse even though such findings are clearly not caused by rickets. Such claims are pure fabrication*" [53, 84, 85].

In the article by Ayoub et al., to which Strouse referred, the (what Ayoub et al. called) hypothesis was reviewed that metaphyseal lesions would represent traumatic changes in abused infants by evaluating nine classic metaphyseal lesion (CML) studies, which were performed by the same principal investigator. They also compared these lesions with the findings in healing rickets [84]. They concluded that the hypothesis that classic metaphyseal lesions are secondary to child abuse was/is poorly supported by the findings in the evaluated studies. According to Ayoub et al., the histologic and radiographic features of CML are similar to healing infantile rickets and until classic metaphyseal lesions are experimentally replicated and independently validated, their traumatic origin remains unsubstantiated. Miller, one of the authors of the original 2014 Ayoub et al. article, and Mirkin repeated the message, formulated in the 2014 article in an article in Medical Hypotheses [86]. Miller also was the first author of a 2019 article: "*Findings of metabolic bone disease in infants with unexplained fractures in contested child abuse investigations: a case series of 75 infants*" [87].

The before mentioned publications and other publications concerning "rickets/metabolic bone disease versus child abuse" were and still are heavily criticized in the medical literature, just like the 2008 Keller and Barnes commentary [54, 88]. The reader is referred to the relevant literature, because it is impossible to summarize all criticism, concerning, e.g., the misrepresentation of the professional background of participating scientists in the literature, that is evaluated by Ayoub et al., and the omission of relevant evidence-based medical literature in their evaluation [89–94]. However, the article and conclusions of Ayoub et al. were not only criticized in the medical literature. The views, concerning rickets, as described by Keller and Barnes or later by Ayoub et al. have also been tested for their scientific value and their reliability as "judicial" evidence in a number of court cases. Especially the judiciary way of reasoning is well exemplified in the following two court cases:

Court Case 1

In 2018 Ayoub and Miller testified as expert witnesses for the defense before M.F. Andrews, Circuit Judge, in the Circuit Court of the sixth judicial circuit of the State of Florida in and for Pinellas County criminal division [95]. This case dealt with a motion by the defendant for post-conviction relief.

According to the verdict by judge Andrews, the defendant "*raised a single claim of newly discovered evidence based on a scientific article published in a medical journal in january 2014 by doctors Ayoub et al.* [84] *and an opinion rendered by dr. Miller on February 10, 2015, based on the 2014 article and a review of K.D.'s medical history. Defendant alleged that he is entitled to a new trial because the 2014 article and dr. Miller's report establish that the victim's injuries in this case were the result of an undiagnosed medical condition, such as a metabolic bone disease, scurvy, rickets, copper deficiency, or maternal use of drugs, rather than child abuse.*" … "*Defendant alleges that if the jury had the benefit of this 'unequivocal diagnosis', the trial would have resulted in an acquittal.*"

On page 4 of the verdict Miller is cited: "*Particularly, dr. Miller indicated that the lack of bruising on K.D. is inconsistent with the allegation of violent handling of the baby; stating that violent handling of a baby is often expected to compromise the integrity of the skin, leading to a bruise, when a bone is broken by severe trauma. Dr. Miller opined that the lack of bruising in combination with the large number of fractures is the result of bone fragility rather than trauma. Dr. Miller opined that the lack of internal lung injury in light of the multiple rib fractures suggests that the physical forces that caused the rib fractures were not excessive and that some of the healing rib fractures could have been from the birthing process. Dr. Miller indicated that based on his own study, CML's are not really fractures, but rather findings of metabolic bone disease.*"

On page 6 of the verdict Ayoub is cited: "*He indicated that he is an expert in diagnostic radiology and had testified as an expert in rickets twice. he testified that he has testified for the defense around 80 times. He testified that he sees metabolic bone disease nearly every day in his regular practice. He testified that he has reviewed around 1,500 known cases of rickets in his career. He further testified that he*

reviews between 70 and 250 x-rays per day. In preparation for this case, dr. Ayoub testified that he reviewed digitized versions of K.D.'s x-rays, taken between April 5, 1993 and May 1994."

Page 9: "*Dr. Ayoub testified that he compared K.D.'s x-rays to known metabolic bone disease x-rays, and he saw signs of metabolic bone disease in K.D.'s x-rays. More specifically, he indicated that he saw signs of healing fractures in K.D.'s x-rays. He testified that a feature of bone fragility disorders is that normal handling could cause bone fractures. He testified that K.D. had healing infantile rickets, as well as possibly a copper deficiency.*"

'Dr. Ayoub testified that it is possible for a child with infantile rickets to also be abused. He believes that there are true fractures and signs of metabolic bone disease. .... He testified that the pattern of injuries is consistent with metabolic bone disease.'

On page 10 Miller is cited again: "*He conceded that he has testified around 100 times as an expert and has only testified for the defense. He testified that in each of those cases, there was another explanation for the injuries other than child abuse.*"

On page 19 and 20 of the verdict the judge examined the credibility of expert witnesses. The judge stated: "*.... The court was presented with two defense witnesses, drs. Ayoub and Miller, who this court cannot believe were objective in their testimony. Between the two of them, they have testified in nearly two hundred cases where there was alleged child abuse. Collectively they have reviewed thousands of x-rays. And yet, not once, in nearly two hundred cases, has either defense expert found a case where child abuse was the cause of the child's injuries. In the years each has served as expert witnesses there has not been one case of broken arms, ribs, clavicle, legs or fractures of the skulls, which either doctor has found to be the result of child abuse. The revelations exceed the limits of credulity. Dr. Ayoub admits that of the thousands of cases he has reviewed, the only cases of child abuse he has seen in his practice are accompanied by an admission from the abuser or observation by a witness. In other words, he is predisposed to find that no matter how severe the injury, unless there is a witness to the abuse or an admission by the abuser, there must be some other cause of the child's injuries. The revelations explain why both doctors are frequent defense witnesses. It also explains why neither has ever testified for the prosecution. This court is challenged to believe that any jury, presented with these revelations, would believe that either doctor's opinion are credible. '(S)cientific recognition requires the testimony of impartial experts or scientists.…*"

Page 20: "*.... It is clear from the testimony of various experts participating in the evidentiary hearing, that metabolic bone disorder is not selective or regional. Throughout the hearing testimony of rickets and a possible copper deficiency, the Court was never advised of any studies that isolate such disorders to one side or one region of the body. Yet, K.D.'s injuries were exclusively confined to his left side. It again stretches the limits of credulity that possible bone diseases drs. Ayoub and Miller ascribe to K.D. were unlike bone diseases that afflict any other person especially any other infant in that they were confined to one side of K.D.'s body. .... Even if rickets, copper deficiency, or some other metabolic bone disorder could have afflicted K.D. at the time of various injuries, none of the various explanations account for the injuries being on one side of K.D.'s body.*"

Page 21: "*.... In fact, there was no evidence presented either at trial or at the evidentiary hearing that K.D. suffered any nutritional deficiencies. After K.D. was removed from the care of the defendant, the child suffered no further injuries further undermining the theory of K.D. having brittle bones or vitamin D deficiency.*"

Page 23: "*.... The alleged newly discovered evidence authored by drs. Ayoub and Miller has been published since 2014, and both admit that their theory had not generally accepted in the scientific community.*"

"*The evidence presented at hearing and already discussed herein establishes that both drs. Ayoub and Miller testify exclusively for the defense. The evidence also shows that neither is objective in their analysis of the evidence finding no case of actual child abuse where there is not a confession or witness to the abuse. Because both drs. Ayoub and Miller testify exclusively for the defense it is clear that there are other incentives for the testimony they offer. .... This Court can not say that either dr. Ayoub or dr. Miller is an impartial expert. ….*"

On page 24 and 25 of the verdict Circuit Judge Andrews concluded: "*Both drs. Ayoub and Miller concluded that it is possible for a child with a metabolic bone disease to also be abused. Dr. Ayoub specifically testified that he could not exclude child abuse from this case. .... Both drs. Ayoub and Miller conceded that their opinion as stated in the 2014 article is not generally accepted in the medical community. They both testified that the medical community still relies on dr. Kleinman's textbook and the opinion that CML's are highly specific to child abuse. Thus, the opinion of drs. Ayoub and Miller is clearly a fringe opinion and would be inadmissable or, if admitted, likely unpersuasive when compared to the generally accepted opinions and evidence presented at the original trial. Both doctors would have to concede that their opinion is essentially rejected by the rest of the scientific community. Additionally, a differential diagnosis alone is insufficient to prove causation.…*"

Court Case 2

Ayoub also testified in 2018 in the United Kingdom in the family court before Lord Justice Jackson, sitting as a Judge of the Family Court at the Royal Courts of Justice [96]. In this court applications were made to discharge a care order and to reopen settled findings of non-accidental injuries.

Ayoub, as expert witness for the defendant, stated (section 23 of the judgement): "*That C suffered from metabolic bone disease is beyond certain.*" The judge raised a number of concerns about the evidence given by Ayoub. In his judgement, the judge motivated these concerns in sections 33–45 of the judgement as follows:

'33. Dr Ayoub is a general radiologist practising in Springfield, Illinois. ....

34. Dr Ayoub accepted the proposition that his clinical practice concerns adults and his legal work concerns babies. He has never trained in pediatric radiology and his clinical work does not include infants (children under 1 year old). He has seen just one case of severe rickets in his clinical practice. By contrast, his legal work, stretching back over 10 years, is entirely focussed on infants. This involves him in writing reports and giving evidence, always for the defense, in cases where adults have been accused of causing fractures in infants. 70% of these cases are civil, the remainder criminal. This is unpaid work that he performs out of an avowed conviction that many cases of alleged child abuse are misdiagnosed and are in fact cases of metabolic bone disorder, particularly rickets, which he described as "vastly underdiagnosed". He considers that he has an ethical duty to correct this situation. When I asked him where he considered his views would be considered controversial, he said that they would to those who are unaware or ignorant of the published support for them.

35. Dr Ayoub stated that he is sent 3–5 legal cases every week to consider, has been consulted in about 500 cases, has written approximately 200 court reports and has appeared approximately 80 times as a witness in proceedings in the United States. He has been engaged in two other cases in this country and one in Sweden. In every case in which he has written a report, he has expressed the opinion that the child in question suffered from a metabolic bone disorder. In a television interview given in about 2010, he said this: "I've not seen any high-risk family. I don't believe any case of fractures I've seen has been as a result of real physical child abuse, that it's metabolic."

36. ... (list of letters and articles by Ayoub).

37. Those working in the field will recognise the references to Dr Colin Paterson, proponent of the theory of temporary brittle bone disease, who was criticised by Singer J in Re X (Non-accidental Injury: Expert Evidence) [2001] 2 FLR 90 and who was struck off the medical register in 2004, and to Dr Charles Hyman, the California paediatrician, whose approach as an expert witness was the subject of criticism by Cobb J in Re AD and AM (above). Dr Ayoub said that he knows and sympathises with Dr Paterson and also with Dr Andrew Wakefield, whose views on the effects of childhood vaccination led to him being struck off the medical register in 2010.

38. Dr Ayoub's forensic work has not surprisingly attracted public attention in the United States, and he regularly speaks publicly on how in his view metabolic bone conditions, such as rickets, mimic child abuse.

39.-41. ...

41. Dr Ayoub's involvement arose from an approach by the parents themselves ahead of their criminal trial. He knows that there are rules governing the terms on which experts give evidence in our courts, but he did not profess to know what they contain.

42. It is not seriously disputed between the parties that if the Family Court had been asked to approve the prior instruction of Dr Ayoub as an expert witness, it would have been unable to do so. There are two fundamental reasons. Firstly, he does not have the necessary expertise to offer an opinion to a court on the origin of radiological appearances in infants, particularly pre-term infants, as they are a patient cohort of which he has no clinical experience. Secondly, his approach is shot through with the dogma that child abuse is over-diagnosed. It does not matter for this purpose whether he is right or wrong. The expert with a scientific prejudice may perform a service to science by asking questions that challenge orthodoxy, but be unsuited to be an expert witness, a role that requires objectivity when giving answers.

43. Nothing in Dr Ayoub's evidence in the present case led me to a different view. He made himself available at an early hour at personal inconvenience and gave his evidence in a serious manner. However, his evidence was characteristic of his general approach. Having taken up a position, he advanced it with the tenacity of an advocate and was dismissive of alternative possibilities. He entertained no doubts about the correctness of his opinion, a dangerous mindset for any expert witness.

44. I therefore conclude that the family or criminal courts in England and Wales are unlikely to find that Dr Ayoub meets the requirement that an expert witness must be objective and unbiased. At all events, if it is proposed that he should give evidence in any future case concerning fractures in infants or young children in this jurisdiction, the relevant court should be made aware of the matters contained in this judgment.

45. Here however, his evidence has been heard and I will refer to it as appropriate.'

Concerning the possibility of alternative explanations for the findings, the judge stated in section 123–125 of his judgement:

"Rickets:

- *123. The possibility that C suffered from healing rickets was fully considered in 2014/2015. Now, Dr Ayoub alone argues that the diagnosis should be made, along perhaps with copper and vitamin C deficiency. I do not accept this view. It is contradicted by all the other witnesses and has no sound radiological or biochemical foundation.*

Scurvy:

- *124. I remain satisfied that C did not suffer from scurvy. There is no biochemical or radiological support for this theory, and such assay results as there are speak against it. Given the vitamin C that was being administered to C, it is extremely improbable that he was deficient.*

Copper Deficiency:

- *125. Based on the normal copper level result and the lack of clinical signs of this rare haematological condition, there is no support for Dr Ayoub's opinion that this may be a feature of C's condition.*"

15.3.2.4 The Scientific Evidence Concerning Rickets Versus Child Abuse

Publications like these and defense expert witness presentations in court have led to studies into the relationship between vitamin D levels and fracture risk, the radiological findings in children with low vitamin D levels, and the difference between metaphyseal corner fractures and ricketsian metaphyseal fraying. In Sect. 15.3.2.1, the relationship between vitamin D levels and fracture risk has been dealt with extensively.

The second question to answer could be if a lower vitamin D serum level leads to radiological findings consistent with rickets (Fig. 15.1a–c). Servaes et al. studied 79 infants with fractures with subsequent skeletal survey, median age 4 months, with a vitamin D level ranging 11.6–88.9 ng/mL and which was low (<30 ng/mL) in 27 infants [97]. On radiographic imaging questionable demineralization was seen in seven infants and mild to moderate demineralization in four. In seven infants widening of the sutures, claimed by the protagonists of D deficiency as an alternative to skeletal abuse most easily recognized feature of rickets, was present. However, in four children intracranial hemorrhage was present which can also lead to widening of the sutures. There were no cases with metaphyseal changes. The authors concluded that although in their population of infants suboptimal vitamin D levels were frequently found but that radiographic evidence of rickets is uncommon.

Perez-Rosello et al. studied radiographs of the wrists and knees of 40 infants, aged 8 to 24 months, with a vitamin D level under 20 ng/mL [81]. All radiographs were scored using the Thatcher scale by experienced pediatric radiologists [98]. The radiologists found radiological findings in keeping with rickets in 2 infants (5%, interrater agreement 65%, $k = 0.33$) and signs of demineralization in 2 infants (5%, interrater agreement 70%, $k = 0.37$). The authors conclude that "*When an otherwise healthy infant or toddler presents with unexplained fractures and vitamin D deficiency and radiographic and biochemical markers are absent, rickets is an unlikely explanation for the osseous injuries*."

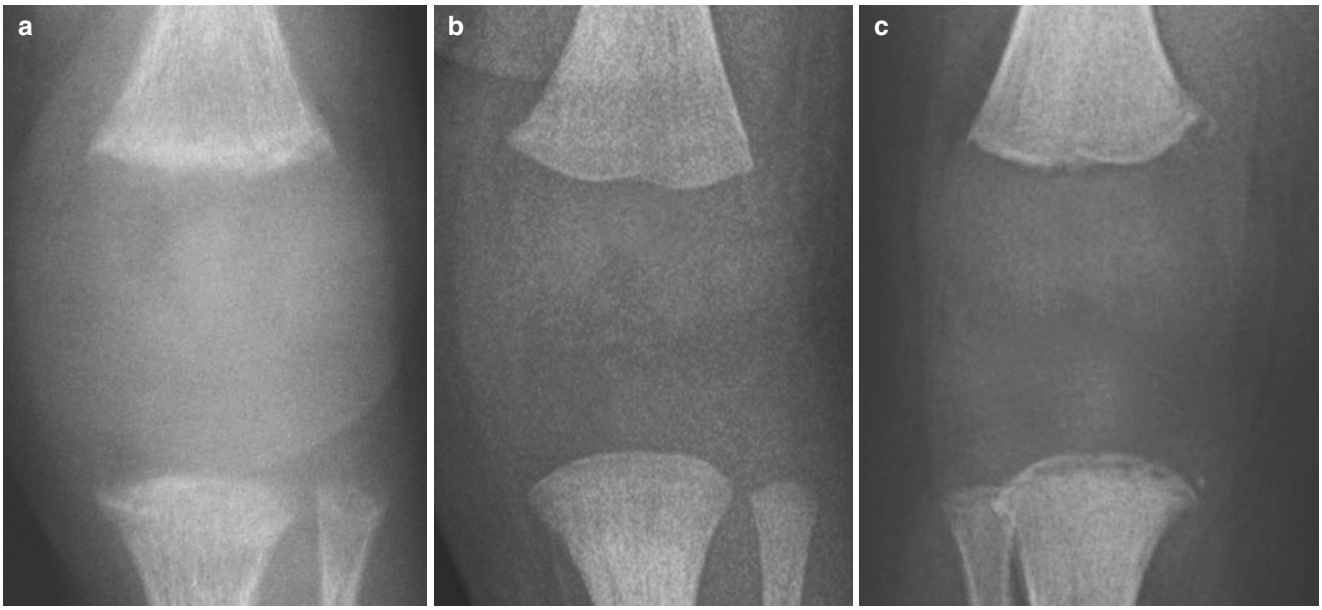


Fig. 15.1 Radiological finding in rickets of the knee compared to normal and CML. (**a**) Rickets in a 6-week-old premature infant. The metaphyseal margins are irregular and unsharp defined, osteopenia with coarsening of the medullary trabeculae and undermining of cortical bone. (**b**) Normal infant for comparison. (**c**) Infant after non-accidental injury with CMLs: corner fractures (femur and tibia) and bucket-handle (tibia)

The final question is whether rickets indeed radiologically mimic fractures as seen in child abuse, e.g., metaphyseal corner fractures, or should radiologists be able to tell the difference.

In a commentary in Pediatric Radiology, Oestreich presented the clear differences between the two radiological findings and summarized his commentary as follows: "*If there is suspicion of rickets at the end of a tubular bone one must carefully evaluate all of the imaged bones. If the metaphyseal collars of Laval-Jeantet are intact and if the zones of provisional calcification are calcified, then the child does not have rickets. Rickets is systemic and symmetrical; classic metaphyseal fractures are not. Patterns of fracture considered to be of high specificity for child abuse are distinctly different from healing rickets and this should not be denied*" [99].

Perez-Rosello et al. studied post-mortem radiologic and histopathologic data from 9 infants, mean age, 3.9 months (age range, 1–9 months), who died of possible non-accidental trauma and who met the following inclusion criteria: (a) The medical examiner determined that the infant had sustained a head injury and that the manner of death was a homicide, (b) at least one CML was evident at skeletal survey, (c) CMLs were confirmed at autopsy, and (d) non-CML fractures were also present [100]. In this population a total of 72 fractures were found and there were no cases with radiological findings consistent with rickets.

Another approach was taken by Chapman et al., who studied a group of 45 infants, age range of 2–24 months, with nutritional ($N = 32$), congenital ($N = 4$), or secondary ($N = 4$) rickets [82]. In 7 children, all with nutritional rickets, fractures were diagnosed on radiography in all cases overt radiographic evidence of rickets was present. The fractures all were structural insufficiency fractures and in none of the infants fractures with a high sensitivity for child abuse, i.e., metaphyseal corner fractures, posterior medial rib fractures, skull fractures, or vertebral fractures, were seen.

Kepron and Polanen looked at the histology of rickets versus child abuse related fractures [101]. In their publication they present a good overview of both the histologic and the radiologic differences between fractures in general, metaphyseal corner fractures and rickets. Based on their study they concluded that "*The maturation disturbance in the growth plate that occurs in rickets is a distinctive entity that cannot be confused histologically with healing fractures, including the classical metaphyseal lesion*."

Finally, with respect to the publication by Keller and Barnes we would like to quote Strouse, editor-in-chief of Pediatric Radiology, who in an editorial, published 5 years after the publication by Keller and Barnes stated "*Perhaps better stated, the hypotheses of "Keller & Barnes" have been disproved. Whether in legal proceedings or in medical literature, it is inexcusable and inappropriate to cite "Keller & Barnes," particularly without simultaneously citing the accompanying commentary by Slovis and Chapman and the subsequent commentary by Feldman* [66, 74]. *To do so is deceptive*" [79].

15.3.3 Vitamin C Deficiency, Vaccinations, and Fractures

Despite the overwhelming scientific evidence that congenital or acquired vitamin C deficiency in the Western industrialized world can be considered to be a virtually non-existing medical condition, some "expert witnesses" still maintain that vitamin C deficiency in infants can result in clinical findings resembling either the findings in inflicted head injury (often referred to by these authors as "shaken baby syndrome") or resembling fractures due to non-accidental trauma without the presence of other signs or symptoms of vitamin C deficiency. According to these "expert witnesses" the deficiency often is congenital or vaccine induced.

As already stated, congenital vitamin C deficiency in neonates, due to vitamin C deficiency in pregnant women, does not exist. Vitamin C deficiency, whether or not vaccine induced, was introduced in the courtroom around the turn of the century as an alternative explanation for the occurrence in children of subdural and retinal hemorrhages, and (often) multiple fractures.

In 2002 Clemetson published an article in "Medical Hypothesis" with the title: "Barlow's disease" [102]. The summary of the article was as follows: "*The classical form of Barlow's disease or infantile scurvy, with bruises, broken bones and sores that will not heal, is rarely seen today, but it seems to be reappearing under a different guise, when infants with borderline vitamin C depletion are assaulted with too many inoculations at one time. Moreover, it is now sometimes mistakenly diagnosed as child abuse.*" According to Clemetson: "S*ubdural hemorrhages, multiple bone fractures and subperiosteal hemorrhages do occur in infantile scurvy, but all too often now the subperiosteal hemorrhages lifting the growing sheath right off the bone, are thought to be the result of fractures, instead of being recognized as revealing their cause. Even this X-ray finding, formerly known as being characteristic of the healing phase of infantile scurvy, is now often said to be indicative of child abuse.*" As a reference for this statement he used an article by Barlow, published in 1883, and a book by Hart & Lessing, published in 1913 [103, 104].

In this article Clemetson related to a court case, in which, according to Clemetson, the findings in a child (bruises in the orofacial region and on the scalp, cerebral edema, subdural hemorrhages, healing fractures at costochondral junctions of the fifth, sixth, and seventh ribs on the left side and a fracture of the tenth rib which may have occurred as a result of han-

dling during the autopsy—cited from Clemetson, 2002) and the resulting death could be explained as "*concatenation of unfortunate circumstances causing vitamin C deficiency and capillary fragility*" in a premature child after inoculations.

Clemetson added a critical addendum to his article: "*While it is readily appreciated that histaminemia due to inoculations, when added to the histaminemia of borderline ascorbic acid depletion, could cause endothelial damage leading to bleeding into the tissues, one would not expect bone fragility until and unless there were an almost complete deficiency of this vitamin. The explanation most likely is that the escape of blood into the tissues soon causes hemolysis and hemolysis rapidly destroys ascorbic acid, leading to frank scurvy.*"

In fact, Clemetson did not give any proof, only hypotheses, for the statements in his 2002 article and admitted in this addendum, that there was no scientific evidence, concerning the statements.

As already mentioned before, in 2014 Greeley stated, concerning courtroom diagnoses like the vitamin C deficiency hypothesis of Clemetson: "*Theories appear to have been created not with a clinical gap to fill, but explicitly to be used in court—to be "backwards compatible" from the courtroom into clinical relevance.*" This is what already happened in the 2002 Clemetson article. Clemetson stated, concerning the conviction of the father of the deceased child, as follows: "*If there is any justice in this world .. (the father) should be released from prison and so should all others falsely accused of child abuse.*"

In 2004 the father was released after an evidentiary hearing, in which the judge overturned the conviction of the father, partially because of the acceptance by the court of the still unproven hypotheses concerning possibly vaccine-induced vitamin C deficiency. Other alternative explanations for the occurrence of the findings in the deceased child, that were not proven in 2004 and still are not proven in 2014, used in the hearing were [105, 106]:

- An adult male cannot generate the needed force by shaking alone, without impact.
- Six vaccines at once, while ill, 13 days prior to the fatal event.
- Vomiting with aspiration.
- Shaking cannot produce unilateral retinal hemorrhages, as observed in the deceased baby, for biomechanical reasons.
- Late hemorrhagic disease of the new-born.
- Four "probable" healing rib fractures of various ages without any bruising or damage to the lung. According to one of the expert witnesses in the hearing each traumatic fracture has a 75% probability of damaging the lung, he stated. Thus, the probability that four independent traumatic fractures will not damage the lung is [0.25]4 or 0.4%).
- The improbability of breaking just one rib in each of four presumed episodes of violent squeezing.
- Temporary brittle bone disease and vitamin C deficiency as explanation for the rib fractures.

The 2002 article of Clemetson was published in Medical Hypothesis. "Medical Hypotheses" is a journal, that was founded in 1975 and is published by Elsevier. In the beginning it was intended as a forum for unconventional ideas in medicine and in related biomedical sciences without the traditional filter of "peer review" being applied to these ideas. This means that a check of the scientific basis and thus the scientific value of the hypotheses in the articles was not tested by other scientists. The only condition for placement was that the ideas had to be coherently and clearly expressed. In this way, the magazine wanted to provide a platform to discuss controversial topics off the beaten path. A striking example of, according to the editor, a coherently and clearly expressed unconventional idea is found in an article by Flensmark, who presented a hypothesis that an association does exist between the use of heeled footwear and the occurrence of schizophrenia [107]. Medical Hypothesis was Elsevier's only magazine that was not subject to peer review prior to publication. The editor was of the opinion that full responsibility for the content and the integrity of the content rested with the authors. This policy was only amended in 2010, due to a scandal concerning AIDS denialism, and the journal now uses, forced by Elsevier, review by the editor and by external reviewers since 2010. This is called by the journal a "Medical Hypotheses custom-made review system" [108]. Despite the fact that Clemetson kept repeating his ideas about vitamin C deficiency, he never gave any scientifically sound proof for his hypothesis [109–114]. In 2006 Clemetson even stated: "*The hypothesis that subdural hemorrhages, retinal petechiae, and spontaneous fractures of the ribs and long bones can occur as an early variant of scurvy at about 8 to 10 weeks of age has not been adequately studied, and, therefore, has not been disproven*" [115]. This statement is a typical example of a reversal of the burden of proof, which, according to accepted scientific standards, rests on the person who formulates a hypothesis and not on the person who contradicts the hypothesis: "I do not have to proof my hypothesis. If you cannot proof that my hypothesis is incorrect, it must be right."

Several other authors used and still use Clemetson's unproven hypotheses to explain the occurrence of certain physical findings, like hemorrhages (intracranial and retinal), fractures and bruises in children, suspected to be sustained due to non-accidental trauma [116–122]. Just like Clemetson, these authors did not provide scientific evidence, but just shared their personal beliefs about vitamin C.

Nevertheless, one of these articles (Innis 2006) was used in the 2016 SBU report about "shaking" as scientific evi-

dence that subdural hematoma or brain edema could occur due to vaccine-induced vitamin C deficiency [116]. The authors of the SBU report did not mention or perhaps even did not notice that vaccine-induced vitamin C deficiency not only was (and still is) an unproven hypothesis, but like temporary brittle bone disease, in fact a non-existing medical condition, despite the fact that subdural hematoma, retinal hemorrhages, and fractures have been described to occur due to vitamin C deficiency (but never without the presence of more classical symptoms of vitamin C deficiency).

15.3.4 Ehlers–Danlos Syndrome

In recent years there has been a debate, not in the medical literature, but almost exclusively in the media (lay press, blogs and non-medical websites) and in court where experts for the defense proposed Ehlers–Danlos syndrome (EDS) as an alternative explanation in cases in whom it was suspected that a child sustained fractures due to non-accidental trauma [35, 123–125]. Ehlers–Danlos syndrome (EDS) is not a single connective tissue disorder, but consists of a group of relatively rare genetic disorders, caused by a defect in collagen synthesis and characterized by, among other, hyperelasticity of the skin, joint hypermobility, and tissue fragility [126, 127]. This group of connective tissue disorders is currently classified in a system of 13 subtypes (Table 15.2) [128]. The exact prevalence of EDS is not known. The overall prevalence of Ehlers–Danlos syndromes is estimated to be 1 in 2500 to 1 in 5000 people, of which hypermobile EDS probably is the most common genetic connective tissue disorder, followed by vascular EDS and classical EDS [128].

Table 15.2 International classification and prevalence of Ehlers–Danlos syndromes [128, 129]

		Old	Prevalence
All subtypes			1 in 2500 to 1 in 5000
EDS subtype			
Classical	cEDS	I, II	1 in 20,000 to 1 in 40,000
Classical-like	clEDS		Unknown
Cardiovascular	cvEDS		1 in 1,000,000
Vascular	vEDS	IV	1 in 10,000
Hypermobile	hEDS	III	1 in 5000 to 1 in 20,000
Arthrochalasia	aEDS	VIIa	Under 1 in 1,000,000 (around 30 patients reported)
Dermatosparaxis	dEDS	VII	Under 1 in 1,000,000 (around 12 patients reported)
Kyphoscoliotic	kEDS	VI	1 in 100.000 (around 60 patients reported)
Brittle cornea syndrome	BCS		Under 1 in 1,000,000
Spondylodysplastic	spEDS		Under 1 in 1,000,000
Musculocontractural	mcEDS		Under 1 in 1,000,000
Myopathic	mEDS		Unknown
Periodontal	pEDS		Unknown

In this group of disorders, not only the integrity of the connective tissue collagen is affected, but also of the vessels [130, 131]. Due to this vascular fragility, minimal trauma can cause faster and more extensive bleeding, due to damage of the vessels (especially the capillaries) in the subcutaneous connective tissue than in children without EDS. Symptoms of EDS can already be present in infancy but will usually be more prominent at a toddler's age. Referral to a specialized (pediatric) dermatologist is advocated in suspected cases.

15.3.4.1 Ehlers–Danlos Syndrome and Suspected Non-Accidental Trauma

Unexplained bruising, soft tissue injuries, and dislocations can be due to non-accidental trauma, but can also be occurred to varying degrees in the different types of EDS, and to differing degrees between individuals with the same form of EDS [132]. The increased risk of damage to the vessel wall in minor trauma and the resulting often extensive bleeding in the subcutaneous connective tissue may lead to suspicions of non-accidental trauma, especially in undiagnosed children. Suspicions of inflicted cutaneous injuries due to the presence of extensive bruises and striking scars ("cigarette paper tissue scars") have been described regularly in children with EDS, most commonly in children with classical EDS, but also in vascular EDS [133–140]. In neonates and infants, intracranial hemorrhages have been described as case reports in certain types of EDS (vascular, kyphoscoliotic, dermatosparaxis) [141–145].

In 2015 Castori pointed out that extensive or severe mucocutaneous injuries after only a minor trauma in children in EDS could be confused with inflicted injuries [125]. He also draw attention to the possible association of Ehlers–Danlos syndrome and bone fragility, as was posed by lay people in the period before 2015. He stated that:

- "*The existence of a true increased risk of fracture in Ehlers-Danlos syndrome is still a matter of debate in children and adults with little and conflicting evidence.*"
- *"The hypothesis of Ehlers-Danlos syndrome as an alternative explanation for infantile fractures remains speculative.*"

Based on his own experience and data derived from the medical literature Castori concluded: "*... EDS should be considered in the differential diagnosis of children with a suspicion of non-accidental injury. There is no evidence in the medical literature to indicate a predisposition to fractures in infants at risk for many EDS subtypes, especially classic, vascular and hypermobility types. The Beighton score is a simple screening tool, but suffers of low specificity especially in children and is uncommonly applied to infants. Hence, before a formal diagnosis of EDS in the case of a previous diagnosis of non-accidental injury, extensive personal and*

family assessment is needed, preferably in the setting of a highly specialized center. Finally, it is also important to emphasize that a diagnosis of EDS does not exclude the possibility of concomitant abuse."

Holick et al. described 72 infants under the age of 1 year with multiple fractures diagnosed to be caused by non-accidental trauma. According to the authors, 67 infants (93%) had clinical evidence of Ehlers–Danlos/hypermobility syndrome and/or a family history with a confirmed clinical diagnosis of at least one parent having this syndrome and 5 infants (7%) had vitamin D deficiency/infantile rickets without evidence of EDS. Three infants with EDS were diagnosed as osteogenesis imperfecta (OI)/EDS overlap syndrome. The most common fractures noted at diagnosis were ribs and extremity fractures (including classic metaphyseal lesions). The authors were of the opinion that EDS, OI/EDS and vitamin D deficiency/infantile rickets are associated in infants with fractures due to fragility and that these fractures can wrongfully be interpreted as sustained in by non-accidental trauma.

The publication of Holick et al. was heavily criticized. On behalf of the Ehlers-Danlos Society an open letter was written by Tinkle as a response to the publication of Holick et al. [146]. Tinkle stated: "*..., it has been argued that (hypermobile) EDS, not abuse, is a reason for unexplained bone fractures in infants. Many such cases have been described, mostly in the lay press, publicized worldwide, and propagated by a few individuals with little scientific merit.*" According to Tinkle, the clinical criteria for diagnosing (hypermobile) EDS, as used by Holick et al., were not clear. Above that, Holick et al. did not examine approximately one-third of the infants. Despite that, they diagnosed EDS in these children. Holick et al. used joint laxity in the parents to diagnose EDS in the parents and, therefore, the infants. According to Tinkle, joint laxity alone does not define EDS. Although most forms of EDS, including the hypermobile type of EDS, are thought to be autosomal dominant conditions, diagnosis in a parent does not equal diagnosis in the child. The diagnosis in a parent simply defines a risk. The children, diagnosed with vitamin D deficiency, had no blood values, suggestive of a vitamin D deficiency. Neither did these children show signs of rickets on X-rays. According to Tinkle, the publication of Holick et al. did not scientifically support the hypothesis that either or both factors are a cause of unexplained fractures in infancy and "*therefore should not be used in defence arguments.*"

Rolfes et al. did a retrospective, population-based, case-controlled study, concerning children, diagnosed with EDS from 1976 to 2015, with complete records for at least their first year of life [147]. The validity of the diagnosis was ascertained using the 2017 International Classification of the Ehlers–Danlos. Rolfes et al. identified 219 potential cases. Twenty-one children had complete records for the first year of life and sufficient evidence in the medical record to support the EDS diagnosis (14 hypermobile, 2 classical, 4 vascular, and 1 arthrochalasia). The EDS children were compared to 63 controls. None of the children had fractures in the first year of life; however, once children became mobile 11 of 21 EDS cases (52.4%) and 15 of 63 controls (23.8%) had one or more fractures during childhood, odds ratio of 3.4 (95% CI: 1.20–9.66). Despite the fact that EDS had been proposed by Holick et al. as a reasonable alternative explanation of multiple and unexplained fractures in young children that may mimic non-accidental injury, Rolfes et al. found little evidence in the medical literature to support this proposition. The authors also found no evidence in the medical literature that infants with common forms of EDS (hypermobile, classic, and vascular type) are predisposed to more frequent fractures and also identified a higher incidence of fractures in older, ambulatory children with these EDS types.

Hakim and Tinkle formulated the following key questions in differentiating between non-accidental trauma and EDS [132]:

- Is there a confirmed diagnosis of EDS and how has this presented over time, as an individual? It is important to have a clear picture of the "natural history"—e.g., how the condition has affected the child to date. Not every child with EDS has the same set of concerns or severity of concerns. Is what has happened normal or abnormal for the child? If it is abnormal, why might this have happened? Are there explanations other than non-accidental trauma for the change in the pattern or type of injury or behavior?
- Has one biological parent or have both biological parents been diagnosed with EDS or a similar condition? If so, this may help to guide opinion but ultimately it is important to know whether the child or adolescent has the condition.
- Have those raising the concern consulted a professional who has experience in diagnosing and treating EDS, if they do not have that expertise themselves?
- (with regard to fractures) Have other medical causes known to be associated with fractures been considered, and is the type of fracture a recognized or reasonably a potential complication of that condition?

They further advise to consult child protection/a legal representative familiar with local policy and law and to work, whenever possible, with the health, welfare, and law enforcement staff. It is inevitable in complex cases that multidisciplinary input is needed, but in the presence or possible presence of EDS this should include expert opinion in EDS and the heritable disorders of connective tissue.

It is interesting to notice that Holick et al. started the introduction of their publication in 2017 with: "*Child Abuse*

and Neglect (CAN) is a serious public health crisis that has major implications for the welfare of the child involved. Inflicted non-accidental skeletal injuries are the second most common manifestation of child abuse after soft tissue injuries." Up to September 2018 Holick never concluded in a single case in which he testified or was consulted that the injuries in a child were inflicted (Armstrong [148]):

- *In the past 7 years, Holick said, he has consulted or testified as an expert witness in more than 300 child-abuse cases throughout the USA as well as the United Kingdom, New Zealand, Australia, Germany, and Canada. In almost every case, he has made the same finding: instead of blaming any injuries on abuse, he has diagnosed the child with a rare genetic disorder, Hypermobile Ehlers-Danlos syndrome, a condition that affects the connective tissues of the skin, bones, and joints. A handful of studies on adults have linked EDS to bone fragility, and Holick argues that children with the disorder have weaker bones, which can fracture from normal handling. So far, his theory is not supported by the scientific literature, but Holick is convinced that "thousands, if not tens of thousands" of parents worldwide have been falsely accused of fracturing their children's bones. "It's just terrible," he told me. "I feel so sorry for these parents."*
- *In all the cases he has worked on, Holick has never concluded that a child was being abused. On the rare occasions when he didn't diagnose EDS, he attributed the bone fractures to rickets or vitamin D deficiency. Many geneticists and bone specialists find it troubling that he diagnoses EDS in nearly 100 percent of the cases he examines. According to the National Institutes of Health, EDS affects, at the most, 0.02 percent of people worldwide. The rate at which Holick diagnoses the disorder "doesn't fall into the mathematical probability of chance," Brad Tinkle, a clinical geneticist at Peyton Manning Children's Hospital, in Indianapolis, said. Holick retorts that his clients don't come to him by chance; parents contact him after doing their own research and realizing that they or their children have symptoms of EDS. He adds that he hasn't seen a single actual abuser pretending to have EDS and contacting him in search of a "get out of jail free" card.*
- *Holick regularly diagnoses children with EDS without seeing them in person. "I already know on the phone they have EDS," he said, adding that he questions the parents about potential symptoms. "I almost don't have to ask. I know the answer."*
- *In 2017, he co-published an article about his EDS work in the little-known journal Dermato-Endocrinology, where he is an associate editor. (He was unable to get more prestigious medical journals to publish the study.) In the study, Holick examined the cases of 72 children who the authorities believed had been abused. He diagnosed 67 of them with EDS. In a third of the cases, he based his diagnosis on physical exams of relatives of the children, not the children themselves.*

In July 2019, Armstrong reported that [149]:

- *Boston Medical Center had notified the Massachusetts medical board that it has restricted the work of a world-renowned endocrinologist (Holick) criticized for espousing controversial theories as an expert witness for people accused of child abuse. ….*
- *Last September, ProPublica and The New Yorker reported that Holick had testified in hundreds of child abuse cases worldwide and almost always blamed broken bones and other injuries on a rare genetic disorder. At the time, Boston Medical Center said that it had barred Holick from treating or evaluating children under age 13 beginning in May 2017. But Holick continued evaluating children in suspected abuse cases as part of an approved research project, and it now turns out that the discipline was not reported to the board until this past February.*

15.3.5 Copper Deficiency

Copper deficiency entered the courtrooms in the United Kingdom as an alternative explanation for the occurrence of fractures, due to non-accidental trauma, somewhere in the eighties, both in criminal and in civil court procedures (Regina versus Lees and Lees, Lord Justice Lane 1987 & Judgement of the Honorable Mr. Justice Hollis in Wardship Proceedings, Middlesbrough 1987 both cited from Taitz and Taylor 1988) [150, 151].

In 1987 Chapman, a pediatric radiologist, stated about copper deficiency and suspicions of child abuse:"*It also causes skeletal abnormalities and fractures, and recently copper deficiency has been offered to the courts (and given wide publicity) as an explanation of the radiological findings in children whose parents have been charged with abuse. The case of one such child with multiple rib, long bone, and metaphysical fractures was recently heard by the Court of Appeal. In dismissing the appeal the Lord Chief Justice rejected the defence's evidence on copper deficiency and suggested that it actually strengthened the prosecution case.*" Chapman further stated that: "*Copper deficiency is rare—only about 100 cases have been reported—and fractures caused by copper deficiency are even rarer: there have been 16 documented cases, and only five in term infants* (up to 1987)" (Regina versus Lees and Lees, Lord Justice Lane 1987 cited from Chapman 1987) [151]. According to Chapman: "*The differentiation of metaphyseal abnormalities caused by copper deficiency from metaphyseal fractures*

caused by child abuse and from normal developmental variants, such as cupping of the anterior ends of ribs and metaphyseal breaking, is not difficult for the experienced."

In 1987 Paterson stated in response to Chapman that over a period of 10 years he had seen, both in England and abroad, 35 infants who seemed to have had a temporary, severe osteogenesis imperfecta-like disorder [151]. According to Paterson "*the combination of reduced bone strength and maintained mineral content makes it likely that the disorder, like osteogenesis imperfecta and experimental lathyrism, represents a collagen defect*." He was of the opinion that "*the most probable cause of such a temporary collagen defect is copper deficiency, which may be transient but leaves behind bone of abnormal strength for some months.*"

Chapman replied: "*I am glad to see that Dr Paterson welcomes the beginnings of a debate in the medical press, as up to this time the possible role of copper deficiency as a cause of fractures in cases of suspected child abuse has been raised by him only in the courts. Dr Paterson now believes that he has personally seen 35 cases with fractures. This is a remarkable number in view of the fact that the total number of reported cases* (of copper deficiency with fractures) *in the world is only about 15*" [152].

According to Carty clinical experience and knowledge of the existing literature should enable the differentiation between the findings in copper deficiency (and osteogenesis imperfecta) and fractures due to non-accidental trauma and that diagnostic confusion should not arise [153]. Carty stated: "*The presence of normal bone and in particular the presence of normal wrists and knees renders the diagnosis of copper deficiency of sufficient severity to cause fractures unlikely and this can be stated even in the absence of a serum copper concentration. The absence of anaemia and neutropenia and the physical stigmata further make the diagnosis virtually untenable.*"

Shaw stated: "*If the patient is a full term infant, under 5 months of age, who has been breast fed, or has received a milk with a copper concentration of 40 microgram/dl or more then the diagnosis is unlikely. Such a case has never been described* (up to 1988)." According to Shaw, skull fractures and cerebral hemorrhage had never been described in infants with copper deficiency, rib fractures had never been described in full-term infants with copper deficiency, and the demonstration of normal bones, and in particular wrists and knees, makes the diagnosis very unlikely [154].

Although copper deficiency since the eighties has not regularly been used as an alternative explanation in court, copper deficiency keeps popping up as alternative explanation (Sect. 15.3.5) or as part of other alternative explanations, e.g., temporary brittle bone disease (Sect. 15.4.1).

15.4 Non-Existing Disease in the Differential Diagnosis

The differential diagnosis discussed in this part of the chapter is a fictitious disease which has been discredited as a true clinical diagnosis. In this part of the chapter we will discuss the abuse of this fictitious differential diagnosis in court.

15.4.1 Temporary Brittle Bone Disease

In 1993, Paterson et al. described 39 children who presented with a set of symptoms that they considered to be a variant of osteogenesis imperfecta [39]. They called it "temporary brittle bone disease" (TBBD). As the name already implies, it supposedly was a temporary disease in which the presence of fractures is limited to the first year of life. The affected children would be susceptible to sustaining fractures after minor trauma for just a short period of time. The disorder heals spontaneously, without any visible pathology. Paterson et al. suspected that these symptoms were due to a temporary, self-limiting period of copper deficiency; although no evident proof was found in the limited study into serum copper contents.

Usually, the disorder starts with a period of vomiting, followed by diarrhea, anemia, hepatomegaly, incidences of respiratory arrest, neutropenia, and edema. The most common radiological findings were metaphyseal corner fractures, rib fractures, diaphyseal fractures and periosteal reactions along the long bones, anomalies at the costochondral junction and retarded bone age. Only 31% of children had a radiologically visible osteopenia.

It did not take long before doubt arose regarding the existence of TBBD, since children with confirmed copper deficiency hardly ever show fractures [155–157]. Not just the medical world criticized Paterson, also representatives from the legal world issued their comments [158, 159]. One of the children in the series that Paterson described had sustained injuries as a result of child abuse. The authors did not report this in their article. This led to concern that a full investigation into injuries in children would (no longer) take place, since the medical world could assume that one single disease could completely explain the anomalies.

In 2001, it was proclaimed in a court case in the United Kingdom that the testimony of an expert witness in the field of TBBD was not only inadmissible, but also that the scientific foundation was found to be inadequate. According to the judge, the study of the expert witness in question, doctor Paterson, was subjective, unreliable, unscientific, and unproven [160]. In 2004, the General Medical Council

(GMC) retracted the qualifications of Paterson as pathologist. According to the GMC, he had failed as expert witness in two court cases in which the parents were accused of child abuse [161]. According to the GMC Paterson suggested that children could suffer from temporary brittle bones, resulting in multiple fractures. He also omitted relevant facts from his evidence, which did not support his conclusion. His actions were seen by the GMC as "an unacceptable risk to the safety of children." The chairwoman of the GMC stated: "*you risked misleading the court and undermining the confidence which the judiciary is entitled to place in expert medical witnesses*" [162].

In 2005, the Society for Pediatric Radiology and the European Society for Paediatric Radiology jointly published an article [40]. Both societies maintain there is no scientific basis at all on which TBBD can be accepted a disease entity. Only a limited number of medical professionals believe, based on speculations that TBBD exists. Moreover, they use conflicting ideas regarding the disorder and its origin. A few of the causes they put forward are:

- Bone or collagen pathology [39].
- Copper deficiency [39].
- Decreased in utero activity in children with reduced bone density [163, 164].

In his article, Mendelson concludes that there is no scientific foundation at all for any of the above-mentioned hypotheses as cause of TBBD. Consequently, this diagnosis should no longer be made [40]. Later on other authors have shared their concerns and pointed to the invalidity of the evidence behind this diagnosis [165–167]. This has not stopped Paterson to publish subsequent papers on "temporary brittle bone disease" [168–173]. Unfortunately, the paper by Paterson et al. is still cited and included in, e.g., systematic reviews [174, 175].

The SBU (Swedish Agency for Health Technology Assessment and Assessment of Social Services) included osteogenesis imperfecta and brittle bone disease as two separate medical conditions in their list of alternative explanations for the physical findings in "Shaken Baby Syndrome" [172, 176, 177]. Both conditions were included as examples of skeletal diseases, in which subdural and retinal hemorrhages could occur. The inclusion of the 2004 Ganesh paper, which describes retinal hemorrhages after minor trauma in three children with type 1 osteogenesis imperfecta, was completely justified. The SBU included the second paper not as an example of osteogenesis imperfecta, but as an example of brittle bone disease. The SBU probably forgot that brittle bone disease is an alternative name of osteogenesis imperfecta. Above that, they used an article by Paterson and Monk as reason for the inclusion of brittle bone disease [172]. The article however did not describe the findings in "brittle bone disease," but the findings in "temporary brittle bone disease," which is, as shown before, a non-existing disease. The SBU did not mention that the General Medical Council (GMC) retracted in 2004 the qualifications of Paterson as pathologist.

References

1. U.S. Supreme Court (1932) Powell v. Alabama. 287 US 45
2. The Academy of Experts What is an expert witness? https://academyofexperts.org/users-of-experts/what-is-an-expert-witness/. Accessed 2 Aug 2021
3. Hargrove P (1994) Re AB (Child Abuse: Expert Witnesses). England and Wales High Court (Family Division). https://www.casemine.com/judgement/uk/5a8ff87c60d03e7f57ec142a. Accessed 01 09 2020
4. Cresswell J (1993) National Justice Compania Naviera SA v Prudential Assurance Co Ltd ('The Ikarian Reefer'). In: Queens bench division (commercial court) (ed) 2 Lloyd's Rep 68
5. Butler-Sloss E, Hall A (2002) Expert witnesses, courts and the law. J R Soc Med 95:431–434
6. Stow TMF (2010) R v Butler. England and Wales Court of Appeal (Criminal Division - [2010] EWCA Crim 1269). https://www.bailii.org/ew/cases/EWCA/Crim/2010/1269.html. Access date 01-09-2020
7. David TJ (2008) Non-accidental head injury – the evidence. Pediatr Radiol 38:370–377
8. Williams C (1993) Expert evidence in cases of child abuse. Arch Dis Child 68:712–714
9. Strouse PJ, Callahan MJ, Annam A, Brown SD, Chung T, Guillerman RP, Jaramillo D (2021) Guidelines for expert testimony in pediatric radiology. Pediatr Radiol 51:1275–1280
10. U.S. Supreme Court (2019) Federal rules of evidence
11. Hartung MP, Bickle IC, Gaillard F, Kanne JP (2020) How to create a great radiology report. Radiographics 40:1658–1670
12. Mityul MI, Gilcrease-Garcia B, Mangano MD, Demertzis JL, Gunn AJ (2018) Radiology reporting: current practices and an introduction to patient-centered opportunities for improvement. AJR Am J Roentgenol 210:376–385
13. Ganeshan D, Duong PT, Probyn L, Lenchik L, McArthur TA, Retrouvey M, Ghobadi EH, Desouches SL, Pastel D, Francis IR (2018) Structured reporting in radiology. Acad Radiol 25:66–73
14. Wallis A, McCoubrie P (2011) The radiology report–are we getting the message across? Clin Radiol 66:1015–1022
15. Bosmans JM, Peremans L, De Schepper AM, Duyck PO, Parizel PM (2011) How do referring clinicians want radiologists to report? Suggestions from the COVER survey. Insights Imaging 2:577–584
16. Larson DB (2018) Strategies for implementing a standardized structured radiology reporting program. Radiographics 38:1705–1716
17. European society of Radiology (ESR) (2018) ESR paper on structured reporting in radiology. Insights Imaging 9:1–7
18. European Society of Radiology (ESR) (2011) Good practice for radiological reporting. Guidelines from the European Society of Radiology (ESR). Insights Imaging 2:93–96
19. Nobel JM, Kok EM, Robben SGF (2020) Redefining the structure of structured reporting in radiology. Insights Imaging 11:10
20. American College of Radiology (ACR) (2011) ACR appropriateness Criteria – Suspected Physical Abuse Child
21. The Royal College of Radiologists (RCR) and the Society and College of Radiographers (SCoR) (2017) The radiological investigation of suspected physical abuse in children. https://www.rcr.

ac.uk/publication/radiological-investigation-suspected-physical-abuse-children. Accessed 2 Aug 2021
22. Strouse PJ, Peréz-Rosselló JM, Moreno JA (2021) The radiology report in child abuse. Pediatr Radiol 51:1065–1069
23. Adamsbaum C, Méjean N, Merzoug V, Rey-Salmon C (2010) How to explore and report children with suspected non-accidental trauma. Pediatr Radiol 40:932–938
24. Rocha DM, Brasil LM, Lamas JM, Luz GVS, Bacelar SS (2020) Evidence of the benefits, advantages and potentialities of the structured radiological report: an integrative review. Artif Intell Med 102:101770
25. The Royal Australian and New Zealand College of Radiologists (2012) Guidelines for RANZCR Fellows who act as expert witnesses
26. Hammana I, Lepanto L, Poder T, Bellemare C, Ly MS (2015) Speech recognition in the radiology department: a systematic review. Health Inf Manag 44:4–10
27. Blackley SV, Huynh J, Wang L, Korach Z, Zhou L (2019) Speech recognition for clinical documentation from 1990 to 2018: a systematic review. J Am Med Inform Assoc 26:324–338
28. American College of Radiology (ACR) (2017) ACR practice guideline on the expert witness in radiology and radiation oncology
29. TRCoR (2005) Board of the faculty of clinical radiology and board of the faculty of clinical oncology. Providing expert advice to the court: guidance for members and fellows
30. College gerechtelijk deskundigen (2015) Gedragscode Nederlands Register Gerechtelijk Deskundigen. https://zoek.officielebekendmakingen.nl/stcrt-2015-47958.html. Accessed 2 Aug 2021
31. Miller AJ, Narang S, Scribano P, Greeley C, Berkowitz C, Leventhal JM, Frasier L, Lindberg DM (2020) Ethical testimony in cases of suspected child maltreatment: the Ray E. Helfer society guidelines. Acad Pediatr 20:742
32. Paul SR, Narang SK (2017) Expert witness participation in civil and criminal proceedings. Pediatrics 139:e20163862
33. Brewster B (1882) Theory and practice. The Yale Literary Magazine, p 47
34. Leventhal JM, Edwards GA (2017) Flawed theories to explain child physical abuse: what are the medical-legal consequences? JAMA 318:1317–1318
35. Shur N (2019) A case of broken bones and systems: the threat of irresponsible testimony. Am J Med Genet A 179:429–434
36. Shur N, Hinds T, Shalaby-Rana E, Tinkle B, Frasier L, Bulas D, Summar M, Jackson A (2019) Invited commentary: his life was lost but his heart still beats: in honor of children harmed by child abuse. Am J Med Genet A 179:2329–2332
37. Pfeifer CM (2021) Evolving forensic controversies in child abuse imaging. Pediatr Radiol 51:1079–1083
38. Pfeifer CM, Henry MK, Caré MM, Christian CW, Servaes S, Milla SS, Strouse PJ (2021) Debunking fringe beliefs in child abuse imaging: AJR expert panel narrative review. AJR Am J Roentgenol 217(3):529
39. Paterson CR, Burns J, McAllion SJ (1993) Osteogenesis imperfecta: the distinction from child abuse and the recognition of a variant form. Am J Med Genet 45:187–192
40. Mendelson KL (2005) Critical review of 'temporary brittle bone disease'. Pediatr Radiol 35:1036–1040
41. Taitz LS (1991) Child abuse and metabolic bone disease: are they often confused? BMJ 302:1244
42. Paterson CR, McAllion SJ (1989) Osteogenesis imperfecta in the differential diagnosis of child abuse. BMJ 299:1451–1454
43. Barnes PD, Galaznik J, Gardner H, Shuman M (2010) Infant acute life-threatening event—dysphagic choking versus nonaccidental injury. Semin Pediatr Neurol 17:7–11
44. Greeley CS (2014) "Shaken baby syndrome" and forensic pathology. Forensic Sci Med Pathol 10:253–255
45. Edwards GA (2015) Mimics of child abuse: can choking explain abusive head trauma? J Forensic Legal Med 35:33–37
46. Servaes S, Brown SD, Choudhary AK, Christian CW, Done SL, Hayes LL, Levine MA, Moreno JA, Palusci VJ, Shore RM, Slovis TL (2016) The etiology and significance of fractures in infants and young children: a critical multidisciplinary review. Pediatr Radiol 46:591–600
47. Paterson CR (1987) Child abuse or copper deficiency? Br Med J (Clin Res Ed) 295:213–214
48. Mathew MO, Ramamohan N, Benet GC (1998) Importance of bruising associated with paediatric fractures: prospective observational study. BMJ 317:1117–1118
49. Starling SP, Sirotnak AP, Heisler KW, Barnes-Eley ML (2007) Inflicted skeletal trauma: the relationship of perpetrators to their victims. Child Abuse Negl 31:993–999
50. Peters ML, Starling SP, Barnes-Eley ML, Heisler KW (2008) The presence of bruising associated with fractures. Arch Pediatr Adolesc Med 162:877–881
51. Valvano TJ, Binns HJ, Flaherty EG, Leonhardt DE (2009) Does bruising help determine which fractures are caused by abuse? Child Maltreat 14:376–381
52. Aldana Sierra MC, Christian CW (2021) Vitamin D, rickets and child abuse: controversies and evidence. Pediatr Radiol 51:1014–1022
53. Keller KA, Barnes PD (2008) Rickets vs. abuse: a national and international epidemic. Pediatr Radiol 38:1210–1216
54. Cannell JJ, Holick MF (2018) Multiple unexplained fractures in infants and child physical abuse. J Steroid Biochem Mol Biol 175:18–22
55. Holick MF (2007) Vitamin D deficiency. N Engl J Med 357:266–281
56. Gordon CM, DePeter KC, Feldman HA, Grace E, Emans SJ (2004) Prevalence of vitamin D deficiency among healthy adolescents. Arch Pediatr Adolesc Med 158:531–537
57. Gordon CM, Feldman HA, Sinclair L, Williams AL, Kleinman PK, Perez-Rossello J, Cox JE (2008) Prevalence of vitamin D deficiency among healthy infants and toddlers. Arch Pediatr Adolesc Med 162:505–512
58. Sullivan SS, Rosen CJ, Halteman WA, Chen TC, Holick MF (2005) Adolescent girls in Maine are at risk for vitamin D insufficiency. J Am Diet Assoc 105:971–974
59. Bouillon R, Norman AW, Lips P (2007) Vitamin D deficiency. N Engl J Med 357:1980–1981. author reply 1981-1982
60. Baroncelli GI (2007) Vitamin D deficiency. N Engl J Med 357:1981. author reply 1981-1982
61. Wagner CL, Greer FR (2008) Prevention of rickets and vitamin D deficiency in infants, children, and adolescents. Pediatrics 122:1142–1152
62. Folsom LJ, DiMeglio LA (2017) Recommendations released on prevention, management of rickets. https://www.aappublications.org/news/aapnewsmag/2017/02/10/Rickets021017.full.pdf. Accessed 2 Aug 2021
63. Szabo L (2018) The man who sold America on vitamin D—and profited in the process. New York Times. https://khn.org/news/how-michael-holick-sold-america-on-vitamin-d-and-profited/. Accessed 01 09 2020
64. OpenPaymentsData.CMS.gov Centers for medicare & medicaid services. https://openpaymentsdata.cms.gov/physician/196430. Accessed 2 Aug 2021
65. Slovis TL, Chapman S (2008) Vitamin D insufficiency/deficiency – a conundrum. Pediatr Radiol 38:1153
66. Slovis TL, Chapman S (2008) Evaluating the data concerning vitamin D insufficiency/deficiency and child abuse. Pediatr Radiol 38:1221–1224
67. Chesney RW (2008) Rickets or abuse, or both? Pediatr Radiol 38:1217–1218

68. Jenny C (2008) Rickets or abuse? Pediatr Radiol 38:1219–1220
69. Moncrieff M, Fadahunsi TO (1974) Congenital rickets due to maternal vitamin D deficiency. Arch Dis Child 49: 810–811
70. Kirk J (1982) Congenital rickets – a case report. Aust Paediatr J 18:291–293
71. al-Senan K, al-Alaiyan S, al-Abbad A, LeQuesne G (2001) Congenital rickets secondary to untreated maternal renal failure. J Perinatol 21:473–475
72. Mohapatra A, Sankaranarayanan K, Kadam SS, Binoy S, Kanbur WA, Mondkar JA (2003) Congenital rickets. J Trop Pediatr 49:126–127
73. Zeidan S, Bamford M (1984) Congenital rickets with maternal pre-eclampsia. J R Soc Med 77:426–427
74. Feldman K (2009) Commentary on "congenital rickets" article. Pediatr Radiol 39:1127–1129. author reply 1130-1122
75. Keller KA, Barnes PD (2009) Reply regarding rickets vs. abuse: the evidence. Pediatr Radiol 39:1130–1132
76. Strouse PJ (2009) Vitamin D deficiency vs. child abuse: what do we know now and where do we go? Pediatr Radiol 39:1033
77. Slovis TL, Strouse PJ, Coley BD, Rigsby CK (2012) The creation of non-disease: an assault on the diagnosis of child abuse. Pediatr Radiol 42:903–905
78. Ross AC, Manson JE, Abrams SA, Aloia JF, Brannon PM, Clinton SK, Durazo-Arvizu RA, Gallagher JC, Gallo RL, Jones G, Kovacs CS, Mayne ST, Rosen CJ, Shapses SA (2011) The 2011 report on dietary reference intakes for calcium and vitamin D from the Institute of Medicine: what clinicians need to know. J Clin Endocrinol Metab 96:53–58
79. Strouse PJ (2013) 'Keller & Barnes' after 5 years—still inadmissible as evidence. Pediatr Radiol 43:1424
80. 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
81. 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
82. Chapman T, Sugar N, Done S, Marasigan J, Wambold N, Feldman K (2010) Fractures in infants and toddlers with rickets. Pediatr Radiol 40:1184–1189
83. Strouse PJ (2018) Shaken baby syndrome is real. Pediatr Radiol 48:1043–1047
84. Ayoub DM, Hyman C, Cohen M, Miller M (2014) A critical review of the classic metaphyseal lesion: traumatic or metabolic? AJR Am J Roentgenol 202:185–196
85. Paterson CR (2015) Fractures in rickets due to vitamin D deficiency. Curr Orthopaed Pract 26:261–264
86. Miller M, Mirkin LD (2018) Classical metaphyseal lesions thought to be pathognomonic of child abuse are often artifacts or indicative of metabolic bone disease. Med Hypotheses 115:65–71
87. Miller M, Stolfi A, Ayoub D (2019) Findings of metabolic bone disease in infants with unexplained fractures in contested child abuse investigations: a case series of 75 infants. J Pediatr Endocrinol Metab 32:1103–1120
88. Paterson CR (2010) Bone disorders that cause fractures and mimic non-accidental injury. Acta Paediatr 99:1281–1282
89. Wood BP (2014) Commentary on "a critical review of the classic metaphyseal lesion: traumatic or metabolic?". AJR Am J Roentgenol 202:197–198
90. Kleinman PK (2014) Classic metaphyseal lesions. AJR Am J Roentgenol 202:W603
91. Brown SD, Servaes S, Hayes LL, Society for Pediatric Radiology Child Abuse Committee (2014) SPR Child Abuse Committee Response regarding classic metaphyseal lesion. AJR Am J Roentgenol 203:W232
92. Paddock M, Adamsbaum C, Barber I, Raissaki M, van Rijn R, Offiah AC (2020) European Society of Paediatric Radiology (ESPR) Child Abuse Taskforce Committee: a response to Miller et al. J Pediatr Endocrinol Metab 33:941
93. Culotta P, Greeley C, Mehollin-Ray A, Donaruma-Kwoh M (2019) Letter to the Editor regarding "Multiple unexplained fractures in infants and child physical abuse". J Steroid Biochem Mol Biol 186:226–227
94. Feldman KW, Done S (2010) Vitamin D deficiency rickets and allegations of non-accidental injury. Acta Paediatr 99:486–487
95. District Court of Appeal of Florida SD (2017) State of Florida v. James S. Duncan (defendant), Case No. 2D16–2625
96. St Helens Borough, Council v M and F (2018) Baby with multiple fractures – rehearing EWFC 1. https://www.familylawweek.co.uk/site.aspx?i=ed187538. Accessed 2020 01-09
97. Servaes S, States L, Wood J, Schilling S, Christian CW (2020) Rachitic change and vitamin D status in young children with fractures. Skelet Radiol 49:85–91
98. Thacher TD, Fischer PR, Pettifor JM, Lawson JO, Manaster BJ, Reading JC (2000) Radiographic scoring method for the assessment of the severity of nutritional rickets. J Trop Pediatr 46:132–139
99. Oestreich AE (2018) Distinguishing child abuse fractures from rickets. Pediatr Radiol 48:305–307
100. Perez-Rossello JM, McDonald AG, Rosenberg AE, Tsai A, Kleinman PK (2015) Absence of rickets in infants with fatal abusive head trauma and classic metaphyseal lesions. Radiology 275:810–821
101. Kepron C, Pollanen MS (2015) Rickets or abuse? A histologic comparison of rickets and child abuse-related fractures. Forensic Sci Med Pathol 11:78–87
102. Clemetson CA (2002) Barlow's disease. Med Hypotheses 59:52–56
103. Barlow T (1883) On cases described as 'acute rickets', which are probably a combination of scurvy and rickets; the scurvy being an essential and the rickets being a variable element. Med Chir Trans 66:159–219
104. Hart C, Lessing O (1913) Der Skorbut der kleinen Kinder (Möller-Barlowsche Krankheit). Verlag von Ferdinand Enke, Stuttgart
105. Struthers M (2004) Rapid response: 'shaken baby' conviction overturned. BMJ 328:719. https://doi.org/10.1136/bmj.328.7442.719. Accessed 2020 08 11
106. Gorski D (2012) The antivaccine lie that just won't die: the claim that shaken baby syndrome is really due to "vaccine injury". https://sciencebasedmedicine.org/the-antivaccine-lie-that-just-wont-die-shaken-baby-syndrome-is-really-due-to-vaccine-injury/. Accessed 2 Aug 2021
107. Flensmark J (2004) Is there an association between the use of heeled footwear and schizophrenia? Med Hypotheses 63:740–747
108. Wikipedia (2020) Medical hypothesis. https://en.wikipedia.org/wiki/Medical_Hypotheses. Accessed 2 Aug 2021
109. Clemetson CA (2003) Child abuse or Barlow's disease? Pediatr Int 45:758
110. Clemetson CA (2004) Capillary fragility as a cause of substantial hemorrhage in infants. Med Hypotheses Res 1:121–129
111. Clemetson CA (2004) Is it 'shaken baby syndrome' or Barlow's disease variant? J Am Phys Surg 9:78–80
112. Clemetson CA (2004) Shaken baby syndrome: a medicolegal problem. N Z Med J 117:U1160
113. Clemetson CA (2004) Elevated blood histamine caused by vaccinations and vitamin C deficiency may mimic the shaken baby syndrome. Med Hypotheses 62:533–536
114. Clemetson CA (2004) Rapid response: "shaken baby", or Barlow's disease variant? BMJ 328:719

115. Clemetson CA (2006) Caffey revisited: a commentary on the origin of 'Shaken Baby Syndrome'. J Am Phys Surg 11:20–21
116. Innis MD (2006) Vaccines, apparent life-threatening events, Barlow's disease, and questions about "shaken baby syndrome". J Am Phys Surg 11:17–19
117. Paterson CR (2010) Multiple fractures in infancy: scurvy or non-accidental injury? Orthop Res Rev 2:45–48
118. Innis MD (2012) Malnutrition, liver dysfunction, subdural and retinal haemorrhages and encephalopathy in children resulting from a deficiency or abnormality of vitamins C, D and K. JOM 27:117–122
119. Innis MD (2013) Autoimmune tissue scurvy misdiagnosed as child abuse. Clin Med Res 2:154–157
120. Innis MD (2014) Tissue scurvy misdiagnosed as shaken baby syndrome homicide. Clin Med Res 2014:6–8
121. Paterson CR, Ayoub D (2015) Congenital rickets due to vitamin D deficiency in the mothers. Clin Nutr 34:793–798
122. Paterson CR, Paterson EC (2019) Maternal vitamin deficiencies causing bone disorders and fractures in infants. Med Res Arch 7:1–13
123. Holick MF, Hossein-Nezhad A, Tabatabaei F (2017) Multiple fractures in infants who have Ehlers-Danlos/hypermobility syndrome and or vitamin D deficiency: a case series of 72 infants whose parents were accused of child abuse and neglect. Dermatoendocrinol 9:e1279768
124. Shur N, Tinkle B, Summar M, Frasier L, Shalaby-Rana E, McIntosh B (2019) Response to letter, broken bones, and irresponsible testimony: enough is enough already: the flawed Ehlers-Danlos syndrome infant fragility theory should not rule. Am J Med Genet A 179:2335–2337
125. Castori M (2015) Ehlers-Danlos syndrome(s) mimicking child abuse: is there an impact on clinical practice? Am J Med Genet C Semin Med Genet 169:289–292
126. Pauker SP, Stoler JM (2020) Clinical manifestations and diagnosis of Ehlers-Danlos syndromes. https://www.uptodate.com/contents/clinical-manifestations-and-diagnosis-of-ehlers-danlos-syndromes. Accessed 2 Aug 2021
127. George MP, Shur NE, Peréz-Rosselló JM (2021) Ehlers-Danlos syndrome: what the radiologist needs to know. Pediatr Radiol 51:1023–1028
128. The Ehlers-Danlos Society (2020) EDS types. https://www.ehlers--danlos.com/eds-types/. Accessed 5 July 2021
129. National Organisation for Rare Disorders (NORD) (2017) Ehlers Danlos Syndromes. https://rarediseases.org/rare-diseases/ehlers-danlos-syndrome/. Accessed 5 July 2021
130. Leung AK, Chan KW (2001) Evaluating the child with purpura. Am Fam Physician 64:419–428
131. De Paepe A, Malfait F (2004) Bleeding and bruising in patients with Ehlers-Danlos syndrome and other collagen vascular disorders. Br J Haematol 127:491–500
132. Hakim A, Tinkle B Child protection and EDS. https://www.ehlers-danlos.com/child-protection-and-eds/. Accessed 5 July 2021
133. Abtahi-Naeini B, Shapouri J, Masjedi M, Saffaei A, Pourazizi M (2014) Unexplained facial scar: child abuse or Ehlers-Danlos syndrome? N Am J Med Sci 6:595–598
134. Morais P, Ferreira O, Magina S, Silva C, Leão M, Maia A, Azevedo F (2013) Classic Ehlers-Danlos syndrome: case report and brief review of literature. Acta Dermatovenerol Croat 21:118–122
135. Lee AC (2008) Bruises, blood coagulation tests and the battered child syndrome. Singap Med J 49:445–449. quiz 450
136. Wardinsky TD, Vizcarrondo FE, Cruz BK (1995) The mistaken diagnosis of child abuse: a three-year USAF Medical Center analysis and literature review. Mil Med 160:15–20
137. Roberts DL, Pope FM, Nicholls AC, Narcisi P (1984) Ehlers-Danlos syndrome type IV mimicking non-accidental injury in a child. Br J Dermatol 111:341–345
138. Owen SM, Durst RD (1984) Ehlers-Danlos syndrome simulating child abuse. Arch Dermatol 120:97–101
139. van Oostrom CG, Werkman HP, Fiselier TJ (1984) Bruising: a coagulation disorder, abuse or abnormality of the vessel wall? Tijdschr Kindergeneeskd 52:152–155
140. Saulsbury FT, Hayden GF (1985) Skin conditions simulating child abuse. Pediatr Emerg Care 1:147–150
141. Tosun A, Kurtgoz S, Dursun S, Bozkurt G (2014) A case of Ehlers-Danlos syndrome type VIA with a novel PLOD1 gene mutation. Pediatr Neurol 51:566–569
142. Solomons J, Coucke P, Symoens S, Cohen MC, Pope FM, Wagner BE, Sobey G, Black R, Cilliers D (2013) Dermatosparaxis (Ehlers-Danlos type VIIC): prenatal diagnosis following a previous pregnancy with unexpected skull fractures at delivery. Am J Med Genet A 161a:1122–1125
143. Bar-Yosef O, Polak-Charcon S, Hoffman C, Feldman ZP, Frydman M, Kuint J (2008) Multiple congenital skull fractures as a presentation of Ehlers-Danlos syndrome type VIIC. Am J Med Genet A 146a:3054–3057
144. Kato T, Hattori H, Yorifuji T, Tashiro Y, Nakahata T (2001) Intracranial aneurysms in Ehlers-Danlos syndrome type IV in early childhood. Pediatr Neurol 25:336–339
145. Byard RW, Keeley FW, Smith CR (1990) Type IV Ehlers-Danlos syndrome presenting as sudden infant death. Am J Clin Pathol 93:579–582
146. Tinkle B (2018) An open letter on Ehlers-Danlos Syndromes, child abuse, and bone fractures. https://www.ehlers-danlos.com/pdf/MEDICAL-PAPERS/EDS-and-Child-Abuse-14Mar2018.pdf. Accessed 5 July 2021
147. Rolfes MC, Deyle DR, King KS, Hand JL, Graff AH, Derauf C (2019) Fracture incidence in Ehlers-Danlos syndrome – a population-based case-control study. Child Abuse Negl 91:95–101
148. Armstrong D (2018) The child abuse contrarian. https://www.propublica.org/article/michael-holick-ehlers-danlos-syndrome-child-abuse-contrarian. Accessed 05 July 2021
149. Armstrong D (2019) Boston hospital reports disciplining of renowned child abuse skeptic. https://www.propublica.org/article/boston-hospital-reports-disciplining-of-renowned-child-abuse-skeptic. Accessed 5 July 2021
150. Taitz LS, Taylor CJ (1988) Copper deficiency and non-accidental injury. Arch Dis Child 63:1111
151. Chapman S (1987) Child abuse or copper deficiency? A radiological view. Br Med J (Clin Res Ed) 294:1370
152. Chapman S (1987) Author's reply. Br Med J (Clin Res Ed) 295:213–214
153. Carty H (1988) Brittle or battered. Arch Dis Child 63:350–352
154. Shaw JC (1988) Copper deficiency and non-accidental injury. Arch Dis Child 63:448–455
155. Ablin DS, Sane SM (1997) Non-accidental injury: confusion with temporary brittle bone disease and mild osteogenesis imperfecta. Pediatr Radiol 27:111–113
156. Ablin DS (1998) Osteogenesis imperfecta: a review. Can Assoc Radiol J 49:110–123
157. Chapman S, Hall CM (1997) Non-accidental injury or brittle bones. Pediatr Radiol 27:106–110
158. Lynch MA (1995) A judicial comment on temporary brittle bone disease. Arch Dis Child 73:379
159. Wissow LS (1995) Child abuse and neglect. N Engl J Med 332:1425–1431
160. Dyer O (2004) Doctor accused of misrepresenting evidence in child abuse cases. BMJ 328:187

161. Dyer O (2004) GMC strikes off proponent of temporary brittle bone disease. Br Med J (Clin Res Ed) 328:604
162. BBC News (2004) Child injury 'expert' struck off. http://news.bbc.co.uk/2/hi/uk_news/scotland/3533487.stm. Accessed 5 July 2021
163. Miller ME (2005) Hypothesis: fetal movement influences fetal and infant bone strength. Med Hypotheses 65:880–886
164. Miller ME, Hangartner TN (1999) Temporary brittle bone disease: association with decreased fetal movement and osteopenia. Calcif Tissue Int 64:137–143
165. Marcovitch H, Mughal MZ (2010) Cases do not support temporary brittle bone disease. Acta Paediatr 99:485–486
166. Nicholl R, Banerjee I, Williams B, Poblete X (2012) Can we have a permanent end to 'temporary brittle bone disease'? Arch Dis Child 97:762–763
167. Spivack BS, Otterman GJ (2010) Does temporary brittle bone disease exist? Not by the evidence offered. Acta Paediatr 99:486
168. Paterson CR (2009) Temporary brittle bone disease: fractures in medical care. Acta Paediatr 98:1935–1938
169. Paterson CR (2011) Temporary brittle bone disease: the current position. Arch Dis Child 96:901–902
170. Paterson CR, Monk EA (2011) Temporary brittle bone disease: relationship between clinical findings and judicial outcome. Pediatr Rep 3:e24
171. Paterson CR, Mole PA (2012) Joint laxity in the parents of children with temporary brittle bone disease. Rheumatol Int 32:2843–2846
172. Paterson CR, Monk EA (2013) Temporary brittle bone disease: association with intracranial bleeding. J Pediatr Endocrinol Metab 26:417–426
173. Paterson CR, Monk EA (2014) Clinical and laboratory features of temporary brittle bone disease. J Pediatr Endocrinol Metab 27:37–45
174. Pandya NK, Baldwin K, Kamath AF, Wenger DR, Hosalkar HS (2011) Unexplained fractures: child abuse or bone disease? A systematic review. Clin Orthop Relat Res 469:805–812
175. Karst WA, van Rijn RR (2011) Letter to the editor: unexplained fractures: child abuse or bone disease: a systematic review. Clin Orthop Relat Res 469:2654–2655; author reply 2656
176. Swedish Agency for Health Technology Assessment and Assessment of Social Services (SBU) (2016) Traumatic shaking – the role of the triad in medical investigations of suspected traumatic shaking: a systematic review. Stockholm
177. Ganesh A, Jenny C, Geyer J, Shouldice M, Levin AV (2004) Retinal hemorrhages in type I osteogenesis imperfecta after minor trauma. Ophthalmology 111:1428–1431

The Use and Misuse of the Medical Literature

16

Christopher S. Greeley

Contents

16.1 Introduction

Academic publishing has, for centuries, been the gatekeeper of new knowledge. This has been typically in the format of monthly, bound, journals with a finite number of articles published, resulting in a filtering and stifling of information exchange. This difficulty has made access to information a precious commodity and had put extraordinary power and influence in the hands of the few who could publish or get published. The rise of the internet over the past 20 years has led to a 'democratization of knowledge' and increased flow of information worldwide [1]. Additionally, as more and more information portals and publications have utilized online formats, the costs for publishing and promoting information have dropped dramatically. This lowered cost for publishing and promoting of generated work has added fuel to the fire of academic productivity, providing a positive feedback loop to increase the demand for more avenues to publish and promote scholarly work. And so the cycle is self-perpetuating. The medical literature is not immune to this increased information flow. This increased information flow has made it more and more difficult for practitioners to stay up on the knowledge explosion [2]. In reality most busy practitioners read only the abstract of an article and do not routinely read much else. If the paper is actually read, most professionals tend to read it as they would a novel, with little

C. S. Greeley (✉)
Division of Public Health Paediatrics, Department of Paediatrics, Baylor College of Medicine and Texas Children's Hospital, Houston, TX, USA
e-mail: greeley@bcm.edu

R. A. C. Bilo et al. (eds.), *Forensic Aspects of Paediatric Fractures*, https://doi.org/10.1007/978-3-031-12041-1_16

appreciation of some of the intentional structures of a well-crafted manuscript. The published literature is a living body with its own strengths and its weaknesses. Knowledge grows and uncertainty narrows. The published medical literature is the recorded history of this fluid process. If we are to be skilled and adept end-users of the medical literature, we have to be active readers. We have to appreciate not only each paper, its nuance and flaws, but the environment and history into which it is placed. The purpose of this article is to provide a framework for critically evaluating articles and note some pitfalls that sophisticated readers should be aware of. I will be focusing on the published literature as it applies to Abusive Head Trauma (AHT) as the content area to provide examples of how to read, in context, the medical literature.

16.2 Evidence-Based Medicine

Critical literature appraisal is one of the central components of the Evidence-Based Medicine (EBM) framework. Evidence-based medicine is widely credited to have originated in McMaster University (Ontario, Canada) in the 1990s. The Evidence-Based Working Group framed a 'new paradigm' of patient care and medical scholarship [3, 4]. This paradigm shift entreated the scholarly practitioner to develop systematic literature appraisal skills, and to appreciate evidence quality rating and the evaluation of the rigour and quality of the research reviewed. Per the Working Group, EBM is 'the conscientious, explicit, and judicious use of current best evidence in making decisions about the care of individual patients'.

As there are excellent resources available for a deeper exploration of EBM [5–7], the following are some of the primary principles. The framework of EBM has five underlying components as outlined by Dr. Sackett [4, 8] and constitutes the *Critical Appraisal Exercise* [3, 8]:

- Convert these information needs into answerable questions.
- Track down, with maximum efficiency, the best evidence with which to answer them (and making increasing use of secondary sources of the best evidence).
- Critically appraise that evidence for its validity (closeness to the truth) and usefulness (clinical applicability).
- Integrate the appraisal with clinical expertise and apply the results in clinical practice.
- Evaluate one's own performance.

It is apparent that a critical component of the EBM framework is an exacting appraisal of the literature to provide the guidance for the clinician. To practice EBM, a reader needs to actively engage the manuscript, and not simply read the abstract conclusion. The following discussion will highlight the importance of active reading, and also provide some tips on how to avoid pitfalls.

16.3 Journals

16.3.1 Introduction

A modern problem confronting contemporary scholars is the sheer volume of what is being published. This was best highlighted by Fraser and Dunstan [2]. They reported that a new entrant into cardiology (in 2010) who wanted to read 'everything' about echocardiography would have to read 156,661 articles. If this new entrant read five papers per hour for 8 h per day, for 5 days a week, for 50 weeks per year, it would take them over 11 years to complete this task. During this 11 years, an additional 82,142 papers would be published, requiring an additional 8 years to read. This dramatic increase in published literature, on top of the literature that was already published, would be insurmountable without a clear strategy on how to efficiently, and effectively, sift through the wheat from the chaff. In the face of this explosion of published material, unfortunately there has a decrease in the quality of publications, as denoted by an increase in the retraction rate for articles [9]. So readers are confronted with an increasing volume of material, of worsening quality.

Contributing to the challenges reading medical literature is the modern day hazard: the rise of 'predatory publishing'. *Predatory Publishing* is dubious journals seek submissions of manuscripts with the goal being to solicit 'journal processing fees' from the author [9–13]. This is the biomedical publishing equivalent of an online scam. Many in academia receive steady stream of emails requesting submissions to unheard of journals with the promise of a rapid review and publication, usually within 1–2 weeks. This phenomenon has taken on the moniker of *Academic Spam* [14]. This first becomes apparent early in an academic publishing career. After the first few publications, a young academic will begin receiving legitimate appearing solicitations professing awe at a recent publication they have and pleading for future submissions be directed to a journal that is unfamiliar to them. The young scholar may feel prideful that they are now being pursued to submit their 'groundbreaking work', for a discounted 'processing fee' [15]. It is these processing fees that is what the predatory journal is truly seeking [1, 16]. The offer of a rapid turnaround and a 'special discount' may be an attractive proposition for a young academic to pay a small sum to 'get that paper off my desk'. This is particularly true when in academics, publication count is often a metric used for promotion and tenure.

An investigation by the science writer John Bohannon for *Science* reported on the scope and global reach of predatory

publishers [17]. Bohannon performed a journalistic sting, creating 304 spoof manuscripts which appeared to come out of an African University, claiming to cure various forms of cancer. He then sent the faux-manuscript to journals for consideration of publication. Despite being fictitious, not scientifically sound and containing fabricated data, 157 journals accepted the manuscript for publication. Only 36 journals generated any peer review comments with 16 of those publishing the manuscript despite the reviewer's recommendations for rejection. The 157 journals offered to publish the spoof manuscript, after the author paid a processing fee to the journal. While the preeminent biomedical journals, with long provenances of high-quality scholarship would not publish the sham submission, predatory journals see any submission as a potential source of money.

A first step is avoiding predatory publications, readers could search in more established sources of medical publications. As noted in the EBM Clinical Appraisal Exercise, a critical component is the searching of high-quality data. While there exist a number of reliable publishing databases and clearinghouses, one of the most well-known is PubMed [18]. This is the online portal for the U.S. National Library of Medicine (NLM) within the U.S. National Institutes of Health. For a journal to be indexed on PubMed it undergoes a quality review by NLM staff. Publication in PubMed does not 'certify' that the journal or article is of high-quality, but the odds are higher than what would be identified without this filter [12]. Another helpful strategy is the 'Think. Check. Submit' checklist that was developed by a coalition of publishing organizations [19]. This process provides a framework for authors (and readers) to question the legitimacy of a journal that they are unfamiliar with.

16.3.2 Editorial Boards and Editors

An offshoot of the rise of academic spam is the rise of solicitations to join editorial boards [20]. The invitation often requires simply the submission of a CV and the promise to submit a minimum number of articles per year to the journal (usually 1). An unscrupulous journal may find benefit in attracting submissions if the 'editorial board' is filled with accomplished and meritorious researchers and scholars. Again, there may be merit to a junior academic to have editorial board membership for promotion or tenure. The journal *Nature* reported a sting operation where a fictitious scientist was created with the expressed goal to get on as many editorial boards as possible [20]. Similar to the sting by Bohannon, Katarzyna Pisanski and colleagues created an online profile with fake degrees and unpublished book chapters with an online CV. This avatar of a scientist, with no published academic articles, was submitted to 360 journals. While most journal either did not respond, or out-rightly rejected the fictitious, unpublished persona, at least a dozen journals appointed (her) to the editorial board; many dependent upon payment of US$50–750.

A critical feature for understanding the medical literature is understanding that journals are not simply platforms to publish scholarly work. Journals and their editors may have a particular perspective that they wish to be promoted. Editors have a tremendous amount of decision power, even to override legitimate peer reviewers, to promote pet theories or thwart competitors. There may be unspoken pressure on potential authors to adjust a manuscript in order to increase the chances of publication. Or, as Alan Sokal, a professor of Mathematics at New York University said after publishing a fanciful, nonsensical paper [21], [he wanted to] 'publish an article liberally salted with nonsense if it (a) sounded good and (b) flattered the editors' ideological preconceptions' [22].

16.3.3 Journal Quality

It is important to be aware that journals themselves can contain both high-quality and low-quality papers. Each paper needs to be considered on its own merits. Often people seek a metric to assess an overall quality of a journal, as a barometer for whether to trust papers that are contained within. While no perfect scoring system exists, a journal's Impact Factor (IF) is often cited as a measure of the quality of the articles it publishes. While this would be attractive, and many journals tout their IF, the IF has some significant flaws which limit its utility [23, 24]. A journal's IF is simply the rate at which the 'average citable' research paper the journal publishes are cited within 2 years after publication [24]. Thus a journal with an IF of 2.0 means that the 'average citable' paper the journal publishes is cited an average of 2 times in the subsequent 2 years after it is published. Clearly, the IF is in no way a reflection of the quality of the journal or the papers it published, and does not help a reader about any specific paper they are reading [25]. High-quality papers may not get cited, or are cited after 2 years from publication, and low-quality papers may get cited regularly as other scholars criticize it. For example, after the publication of the Wakefield et al. paper in The Lancet in 1998 promoting a relationship between the MMR vaccine and autism, it was cited over 140 times. It currently has been cited more than 3750 times [26, 27]. It being widely cited in no way is a reflection of its quality, nor the quality of The Lancet…but it contributed to The Lancet's IF. By comparison, the 1953 paper by Watson and Crick describing the structure of DNA has only been cited 1664 times [28].

16.4 Peer Review

Peer Review is often cited as a strong defence against flawed publications. Peer review is a mechanism by which a submitted manuscript is reviewed by a series of qualified reviewers prior to publication. These reviewers are to have expertise in the subject of the submitted manuscript and are to review the manuscript for scientific and methodologic rigour. The goal is to have experts in the field provide a gatekeeping function, ensuring only high-quality science gets published. By convention, peer review is voluntary and uncompensated. As there is no 'Gold Standard' in peer review process, there is great variability in the journal expectations for the reviewers. In reality, peer review being voluntary and uncompensated places it last on a long list of things to do by busy professionals. As the volume of publications has exploded, rigorous peer review has been an unfortunate victim. Sometimes the peer review process itself can be bypassed or even corrupted [29]. It is important to see peer review as a minimum threshold and not as a stamp of 'Truth'.

An example of the potential flaws of peer review was most clearly described by Godlee et al. in 1998 [30]. The researchers took a paper which had already been accepted to the British Medical Journal (a high-quality journal which has been in publication since 1840) and introduced 8 weaknesses into the design, analysis, or interpretation. The edited manuscript was then sent out to 420 reviewers. Of the 221 reviewers who returned a review, the mean number of weaknesses identified was 2. Ninety percent identified 3 or fewer weaknesses. Thus, it is apparent that even in high-quality journals, peer review may not provide the safety net that many think it does.

While peer review can be a challenge for journals wishing to publish high-quality science, for many journals peer review is simply absent. This is nicely described in a report by Ray [31]. The author submitted four manuscripts in response to email solicitations from 10 suspect predatory publications. In reality, the four manuscripts were written by either the author's 13-year-old daughter or her 15-year-old friend. For example, one manuscript was titled 'Only Girls Should Bake Gender Roles' which was a psychology paper that was submitted in an eighth-grade science class. The unedited papers were submitted to the journals. Nine journals responded to the submission, with eight accepting it for publication. Six of the eight accepted the manuscript unaltered. The reason provided by the one journal which rejected the manuscript was that the word count was too low. The editor indicated that if the paper were lengthened they would reconsider it.

16.5 Literature Appraisal

As noted in the EBM framework, being able to rigorously evaluate medical literature is the link between understanding a clinical (or research) question and an answer to that question. Literature appraisal requires active reading and interpretation of the published literature. Most professionals simply read the abstracts (and sometimes just the conclusion) of each paper, taking the authors interpretation as the correct one. Such a superficial reading runs a great risk of being misled. There are a number of pitfalls that lead to readers being misled. The most common reasons for being misled are: the conclusion in the abstract may not be supported by the data in the paper, it was the wrong study type for the question the authors were trying to answer, or the quality of the actual study was weak. Without a critical reading of the published literature, the reader will be unable to appreciate the subtleties of the quality of the study, accuracy of the analysis, or the truth of the conclusions. I will outline some strategies to minimize the risks of falling for these traps.

Most medical research papers have a similar structure. This is often referred to as the IMRaD format. This stands for Introduction, Methods, Results, and Discussion. The Introduction usually is 2–3 paragraphs and sets the stage and provides background for the study the authors are reporting on. The Method (also referred to as *Materials and Methods*) is the specifics of how the study was performed. It describes the specific steps that were taken in the research study. The Results usually simply report on the findings of the study, often with tables and figures. The Discussion section is where the authors describe what they think the study shows, their interpretation of the data, and the implications for practice or future research. Not included in the IMRaD format is the References. The References are a listing of the citations used to support the research or conclusions of the study. Of these, the two most important sections of a paper are the two sections which are most frequently not read: the Materials and Methods section, and the References.

16.5.1 Materials and Methods

The Materials and Methods section is the most important section of a paper. It should read first. It will help the reader decide if the paper is worth spending time on in the first place. If, from reading the Materials and Methods of the study, it is apparent that the study is the wrong design or is poorly done, the analysis is incorrect, or the data are incomplete, then it would be better not to read the paper and run the risk of being distracted or misled.

16.5.1.1 Study Designs

To gain a nuanced understanding of the relative strength and weaknesses of a paper, the reader should have a working knowledge of the different study designs employed in clinical research. It is important to recognize that each study design has its own strengths and weaknesses and is intended to answer different research questions. Once the reader appreciates the different study designs, they are able to answer two important questions about a paper: (1) did the authors use the correct study design to answer the question that they posed, and (2) were the conclusions that the authors made justified by the methodology and data presented.

Case Reports describe the clinical data on a single subject [32, 33]. This should be familiar to most readers. The single case typically focuses on a particular finding, feature, or outcome that seems noteworthy to the authors. Case reports are common in the medical literature and often present clinical information which do not reflect the true nature of a disease or condition. Often the presentation, findings, or clinical course are atypical. By virtue of their uniqueness case reports should be viewed as solely hypothesis generating and rarely, if ever, influence practice or policy.

Case Series are similar to case reports and present a small number (typically fewer than 10) of collected case reports, usually with a similar feature [33]. Like case reports, case series should be seen as hypothesis generating and need to be interpreted in context. How the patient cohort was collected very much influences the conclusions that could be made. Rarely are the subjects identified sequentially, and they usually represent a convenience sample of subjects that the authors have accumulated. If subjects were not identified sequentially, the series may have been subject to a surveillance bias in how they were identified. An example of a sequentially collected series was reported by Love and colleagues [34].

Cross-sectional Surveys describe a particular feature or finding in large number of subjects at a single point in time [32]. As the name implies, the reader can imagine a population or cohort sliced in a cross section, at one time point, and a particular variable reported. These studies can provide valuable data on disease prevalence or incidence and are often used in public health research. As cross-sectional studies often report on large samples of subjects, no conclusions can be made on an individual subject. For example, knowing the rate of a specific disease in a population gives no information about if a particular subject in that population actually has that disease. This is referred to as the 'Ecological Fallacy' [35]. Cross-sectional surveys are intended to answer questions of disease prevalence. Cross-sectional surveys(studies) that collect data at a single point in time are unable to report on trends or incidence rates, but if sequential, repeated cross-sectional data are collected, then rates or trends can be reported.

Case Control Studies are designed to match subjects with a particular disease or finding (the 'outcome') with control subjects without the disease or finding. This will allow for comparison of a specific potential 'cause' (often referred to as the 'exposure') [32]. The goal is to match 'cases' with 'controls' to all the ability to 'look back' in time to see 'exposure' differences between the two. The cases and controls are matched in an attempt to replicate the effect of randomization by creating a balance between the two groups any important 'known' (but not 'unknown') variables. The Case Control design is valuable for subjects with a rare condition or finding. By comparing otherwise similar subjects with and without a rare condition or finding, potential causes (or 'exposures') may be identified. Case control studies are designed to attempt to answer questions of association and conventionally report results as odds. An example of a case control study was reported by Price et al. [36]. The researchers identified a cohort of children under 10 years who died from blunt abdominal trauma over a 16-year period. These 33 children were then compared with children who died of natural causes who received CPR (comparison group 1) and died of non-vehicular blunt abdominal trauma (comparison group 2) over the same period. As a result of this research, the authors were able to conclude 'The likelihood of CPR-related primary abdominal trauma in child homicides is very low' [36].

Cohort Studies report on two groups of subjects with different risks (or 'exposures') are followed for a period of time to assess outcomes of interest [32]. This study design can be seen as the reverse of the case control design. Whereas case control study design identifies subjects with a disease or condition and explores potential 'exposures' or causes, the cohort study design identified subjects with an 'exposure' of interest and reports on potential outcomes or diseases that are different from comparator subjects. Cohort studies are commonly used when randomization of the 'exposure' would be unethical (smoking, violence) or impossible (poverty) to perform. This research design often requires many years of follow-up, but can provide valuable data on disease causation or outcomes. Similar to case series studies, it is important for cohort studies to clearly identify how the cohort was collected. If not clearly described, there may be hidden variables or biases which account for reported findings. For example, caution needs to be exercised among the 35 infants and children identified by Matshes et al. who were accumulated from 3 different medical examiner offices over an undefined period of time [37].

This cohort was accumulated in a non-sequential, unspecified manner and as inclusion criteria were not described (i.e. how each subject was identified), conclusions need to be guarded. These subjects may have been a biased, non-representative cohort and were included because they fit a preconceived or subconscious purpose. In this way, a poorly performed or incompletely described cohort study may present data that misleads the reader to draw conclusions that are spurious.

Randomized Controlled Trial (RCT) is the study design with which physicians and clinical researchers will be most familiar. In this design two (or more) groups of subjects are randomly allocated to a 'treatment' arm or a 'control' arm [32]. Each group is then objectively assessed for the effects of the treatment. RCTs can be either single-blinded (only the subjects are unaware of which group they are in) or double-blinded (neither the subject nor the investigator is aware of group allocation). The process of randomization allows for important known and unknown variables (age, gender, health) to be evenly balanced between the two groups. This balance allows the investigator to more confidently conclude that any differences between the groups at the end of the study are due to the effects of the 'treatment' or intervention. In the field of child abuse, true RCTs are most commonly seen in prevention studies. It would be unethical to randomize a child to getting abused or shaken. Subjects can be sequentially identified and placed into 'case' and 'control' groups (i.e. traumatic, non-traumatic), but they obviously cannot be randomized into those groups. Important quality criteria of an RCT include true blinding of subjects and the investigator, true randomization, and each group receiving the same measures and outcome evaluations blinded assessors.

Systematic Reviews utilize a comprehensive literature search strategy to answer a well-defined clinical question. The comprehensive search protocol should involve dozens of keywords and combinations that are used to search a number of different databases. The intention of a systematic review is be able to aggregate the total of the medical literature regarding the particular question [38]. If studies' designs and variables within a systematic review are similar, their results can be combined into a meta-analysis. Nested within a systematic review, a *Meta-Analysis* utilizes the findings of the Systematic Review, and combines them in an effort to report an aggregate result. This simulates as if the studies identified by the Systematic Review were actually a large, single study. This attempts to provide a single overall estimate to report what 'The Literature' supports regarding a specific clinical question. Importantly, Systematic Reviews and Meta-analyses within the EBM framework are more appropriately identified as strategies for literature appraisal and interrogation, as opposed to a specific research methodology. Alper and Haynes more precisely frame them as a 'lens' through which a better appreciation of the underlying literature can be achieved [39].

16.5.1.2 Methodological Pitfalls

There are a number of traps that readers of the biomedical literature need to be aware of. Most are the result of poor study design, but some are rhetorical sleights that authors utilize to persuade the reader to conclusions that are not supported by the presented data. The following will highlight a number of the pitfalls and provide some examples in the published literature.

Bias: While bias is a term that usually is associated with an intention to deceive, bias, in clinical research, is simply 'systematic error' which can distort the data [40, 41]. As opposed to intentional deception, bias often is a result of poor study design or implementation. Subjects may be identified, surveilled or analyzed systematically in a way that unobservedly skews results to a particular direction or result. For example, a researcher may recruit subjects from a particular clinic, but that clinic population is not representative of the full community. Results from that study would be skewed (or biased) and not be generalizable to the full population. Common methods to reduce bias in research are subject randomization, consecutive recruitment of subjects, enrolment stratification, prospective study design, and investigator blinding [41].

Circular Reasoning: Within the child abuse research literature, this is one of the most critical aspects of research design that requires careful attention when reading the Materials and Methods section. Circular reasoning within clinical research is when the outcome variable is part of the inclusion criteria [42]. This is often referred to as *Begging the Question*. This is a common concern in many medical and forensic studies when there exists no 'Gold Standard' for the outcome (i.e. SIDS, child abuse, mental illness, migraines). An example of circular reasoning would be, if the child abuse 'case' definition includes a particular finding (i.e. subdural hematoma), the researchers note that subdural hematomas are most often seen in child abuse cases. In this way, it is critical for the researcher to design the study that the 'case' definition and the outcome are completely independent.

16.5.2 References

The second most important section of an academic paper is the Reference section. The references of a paper are the listing of citations that the authors have used to support the points made in the manuscript. It is quite important for the reader to critically scrutinize the references which authors use. There are two common pitfalls that can be identified by a close reading of the references.

The first is chain citation. This is akin to the childhood game of *telephone*. This is when one child whispers a phrase that is then repeated into the ear of a neighbour. This phrase is then repeated continually until the end of the line, when the original phrase has morphed in the retelling. After being repeatedly cited in multiple texts, the original fact is no longer accurately reported. This is clearly described by Casaulta et al. regarding the oft described phenomenon that children with G-6-PD deficiency have an abnormal sweat chloride test [43]. The authors report that this 'fact' often taught in medical settings but the authors were unable to identify the source of data for the original tables. The fact that alleged false-positive sweat testing results are regularly included in textbook tables in apparent cut-and-paste fashion. This began after being hypothesized at a conference in 1975, despite data never having been reported in published literature to support it.

The second pitfall that readers may not appreciate is how often the citation used by the author does not support the point the author is trying to make. While this is often the result of poor attention to detail, it may also be intentional. The authors may believe that a particular paper supports the point that is being made, but they do not actually read the reference to confirm that it actually does, or the author may simply put a reference in assuming the reader will not 'fact check' them. While increasing the effort required to read an article, reading the references used to support specifically critical points within a manuscript will regularly reveal how frequently there are differences between the reference and the way it is portrayed in the paper. Along the same lines, a common sleight of hand technique by authors to bolster an unsupported opinion is to create a 'citation sandwich'. This is when an unsupported (and uncited) phrase or opinion is preceded and followed by well-supported, cited material. The unsupported, usually incorrect, opinion then has the appearance of being supported by being surrounded by references, while it itself is unsupported.

16.6 Rhetorical Pitfalls

Often despite the actual results of their study, within the Discussion section of manuscripts, authors will try to craft their argument for conclusions that they were expecting or wanting. Like all arguments, the reader should be mindful of how the author may try to make their case for conclusions they are promoting, which may not align with the data or results of their study. Authors may utilize several rhetorical tricks that pose pitfalls to the reader. I will highlight some of the most common within the published medical literature.

16.6.1 Association Is Not Causation

Realistically it is difficult to 'prove' that something is a 'cause' of an 'effect'. There are a number of strategies to assess causation. The Bradford Hill criteria provide one of the most accepted frameworks to help determine causal relationships [44]. These criteria serve as a guide for understanding how to demonstrate some variable 'causes' an outcome. Of these nine criteria, only one 'temporality' (the cause must occur before the event) is a *must*. A common pitfall occurs as a result of overgenerous interpretation of a relationship between two events. Often causation is assigned simply because one event occurs after the other (*cum hoc ergo propter hoc*, or 'after this, therefore because of this'). There may be a relationship between two events, but they both may be related through or to a third event (which was unmeasured), often referred as a *Moderator*. In this way they are simply associated with each other through the third factor. For example, people who attend billiard halls frequently have a higher reported rate of lung and oesophageal cancers. The billiard halls are themselves not responsible for the increased cancer rates. It is due to the fact that people who spend time in billiard halls tend to drink alcohol and smoke more than those who don't. While there is an association between billiard hall attendance and lung and oesophageal cancers, the increased cancer rate is due to the drinking and smoking and not the billiard hall.

16.6.2 Hasty Generalizations

Hasty generalizations, as the name suggests, are when the results are over-exaggerated based upon data that the research generated. As noted above, this is commonly apparent with the over-dependence on using case reports to support clinical or forensic decisions. As noted earlier, case reports are outliers. Given that child abuse, as compared to many paediatric conditions, is somewhat rare, case reports play an outsized role in the published literature. To generalize conclusions from a case report and then extrapolate them to a larger population runs the grave risk of being truly misled. An obvious example is the case report of a 15-year-old girl who survived untreated rabies (a uniformly fatal condition) [45]. Using this case report to extrapolate that rabies is a survivable disease would be the incorrect conclusion. The authors of that report rightly note 'Survival of this single patient does not change the overwhelming statistics on rabies, which has the highest case fatality ratio of any infectious disease'.

16.6.3 Argument from Ignorance (Argumentum Ad Ignorantiam)

The logical fallacy pitfall is when there is an argument that because something has not yet been shown to be true, it must then be false. The reverse is also a logical fallacy (i.e. because something has not yet been shown to be false, it must therefore be true) [42]. There are many ideas we believe to be true (or false) that don't require research to demonstrate; that have *en face* validity. These are usually dangerous, or unethical (or simply unfounded) to critically study (i.e. jumping out of an airplane without a parachute is dangerous, or smoking causes lung cancer, or surgical outcomes are improved if scrubs are blue…or that shaking an infant is dangerous).

Straw Man: Most people are familiar with the concept of the Straw Man argument. This is most popular in the political arena and is when an opposing position is misrepresented in such a manner that it appears clearly inferior. This misrepresented view is then readily easily refuted, with one combatant claiming victory [46]. In a manuscript, this would appear within the discussion section when the authors are presenting an opposing view they are attempting to refute. This opposing view would be skewed in such a way to make it easy for the reader to find it un-credible. One key strategy to combat this is by reading the references which are used to represent to opposing view and ensure that it is being presented accurately.

16.7 Examples of Misuse of the Medical Literature

To get a better sense of how misuse of the medical literature plays out regarding AHT, I am going to provide a few examples.

16.7.1 Hypoxia as a Cause for the Findings in AHT

Since the early 2000s, hypoxia (lack of oxygen in the blood) has been proposed as a potential cause for the finding seen in AHT (particularly subdural haemorrhage, retinal haemorrhage or brain injury; spuriously referred to as 'The Triad'). I would like to trace the origins of this theory and demonstrate how there is no clinical reality to the premise. Due to poor study design and misinterpretation of the literature, it has been given some credibility. In 2000, Geddes and colleagues published a pair of papers which described the central nervous system histologic findings seen in 53 infants and children who were diagnosed with AHT [47, 48]. They reported that these 53 had brain parenchymal findings which were more consistent with hypoxia, as compared to trauma. These papers are often cited in courts as research evidence that infants and children who have died from AHT had in fact died from hypoxia. This is promoted despite the absence of any statistical analysis demonstrating any association between AHT and found hypoxic changes. Of note, in the manuscript itself, the authors themselves report that '(d) istinguishing between terminal hypoxic–ischaemic and traumatic damage to axons…was not always easy' [48]. This challenge is not reported on when the article is used to defend the contention that hypoxia alone can cause the findings associated with AHT.

Shortly after these two papers were published, Geddes and colleagues published a third manuscript which reported to demonstrate that the subdural blood associated with AHT was actually due to hypoxia [49]. In this article, the authors described 50 stillbirth, perinatal, and infant deaths. The authors reported on the presence of intradural haemorrhage (microscopic red cells in dural sections) in non-traumatic deaths. Again, despite the absence of a statistically significant relationship between hypoxia and dural blood, the authors conclude that there was a 'slight indication' of a relationship. Oddly, despite not examining any of the subject's eyes, the authors reported that retinal haemorrhages would occur as well. Geddes and colleagues highlight this study to support the proposition that hypoxia is the underlying mechanism for subdural bleeding in suspected AHT, despite only one foetus having macroscopic subdural blood. Thus, a signature finding of AHT (SDH) was not present in any of the subjects who died from hypoxia.

Together these three papers are commonly referred to as the *Geddes Unified Theory* [47–49]. They are used in court to support the proposition that subdural blood and brain injury associated with AHT are the result of hypoxia absent any trauma. A critical reading of the actual manuscripts, with the appropriate appraisal of study design and analysis, the conclusion that is often touted (that hypoxia can mimic AHT) is simply not contained within the papers cited. To be specific, if the researchers were interested in investigating an association between hypoxia and either parenchymal injury, the correct study design would be case control, which was not done. Without the correct study design, meaningful conclusions would remain unsupported. While hypoxia is likely a part of the secondary injury from AHT, there are no data demonstrating subdural bleeding being the result of a purely hypoxic event. The counterfactual can be easily demonstrated using near-drowning and drowning in children as a model. Rafaat and colleagues evaluated the CT scan of 156 sequential children evaluated for drowning or near-drowning, noting that none of the 156 children had subdural blood on CT scans of the head [50].

As an extension from the *Geddes Unified Theory*, Geddes and Talbert proposed that the actual underlying cause of SDH in AHT was hypoxia, in conjunction with paroxysmal

coughing [51]. They proposed that hypoxia alone was insufficient alone to cause SDH, but that when infants coughed, vomited, or choked they could develop both SDH and RH without trauma. They utilized a computer model to describe how paroxysmal coughing, in conjunction with hypoxia, could raise cerebral venous pressure to the point of vascular rupture. They then propose that this rupture would result in SDH and RH. Their conclusions are based upon their computer model which the authors report 'demonstrates clearly that dangerous rises in intravascular pressures, which may exceed the failure threshold of intracranial veins'. They also note that 'No research specifically addressing the question of stress failure of intracranial veins appears to have been reported in the literature' [51]. Thus they report that the pressure would rise to the point of vascular rupture, but the threshold of that vascular rupture is unknown. By reading the methods section, the reader appreciates that (1) many of the parameters that are put into the model are speculative, and (2) the outcome (blood vessel failure) is unknown. It is easy to be misled by not appreciating the methodological flaws embedded in the study.

In apparent support of the Geddes and Talbert model, Barnes and colleagues report a case of a 4-month-old infant who they report died as a result of what they called 'dysphagic choking' [52]. They report on an infant who on autopsy was noted to have intracranial injury, extensive SDH and RH, and old and acute rib fractures, and claimed that the infant died as a 'result from a dysphagic choking type of acute life threatening event (ALTE) as consistently described by the caretaker' [52]. It was subsequently reported that the authors failed to report that they were all defense/defence experts for the perpetrator and that they did not include important clinical findings (healing rib fractures) which in review of trial transcripts was clear that they were aware of [53, 54]. Absent this fraudulent case report, there exists no published data supporting SDH and RH would result from either dysphagic choking or paroxysmal coughing. This case report highlights the misuse of a case report to try and demonstrate a 'truth', when it presents suspect data. Even if the data reported in this case report were indeed true, as a single case report it would not meaningfully change the understanding a single case report is, by definition, an outlier.

We see by the sequence of events that published data can be 'spun' to support a hypothesis that is clinically not a consideration (that hypoxia causes all of the findings associated with AHT), how these papers are then used to create an additional clinically irrelevant theory (that dysphagia causes all of the findings associated with AHT), that is then presented in a duplicitous fashion. The hypoxia/dysphagia theory was created with limited data and is now presented in courts around the world as having a number of 'peer review publications' supporting its existence.

16.7.2 The Absence of Any Published Support for AHT

The importance of critically reading the Methods section of a paper cannot be understated. A paper by Donohoe is often cited as evidence that there is not a body of literature supporting the diagnosis of AHT [55]. As Donohoe is routinely cited as 'evidence-based', a detailed analysis of the Methods section will clearly expose its poor quality. While not explicitly claimed in the paper, Donohoe presents itself as, and is often cited as, a Systematic Review, one of the strongest evidence levels in most level of evidence hierarchies (i.e. Level I on the United States Preventative Services Task Force (USPSTF)). A reading of the methods section will readily identify that Donohoe is *not* a Systematic Review, but a Literature Review. While often valuable, Literature Reviews are amongst the weakest levels of evidence (i.e. Level III of the USPSTF).

As noted above, crafting appropriate clinically relevant answerable questions and a comprehensive literature search strategy are the cornerstones of EBM. Donohoe uses the single search term, 'shaken baby syndrome', on the Medline database. The use of a single search term on a single database (Donohoe also notes the use of *Internet Explorer* as a database, which is baffling) is blatantly inadequate. The casual reader may not be aware that while Donohoe searched Medline in 1998, the term 'Shaken Baby Syndrome' was not added as a Medical Subject Heading (MeSH) term until 2002. In this way, Donohoe searched Medline using a term that was unrecognizable to it. This search strategy resulted in identifying 71 initial citations. Donohoe notes that an additional 'important' study by Jayawant et al. [56] was not captured in his search strategy, but about which he was aware. This clearly begs the question about how many additional 'important' papers were not captured by this strategy; about which the author was unaware; the purpose of a Systematic Review. Even if the search was performed currently, the paper would not have been retrieved as Medline does not associate the MeSH term 'shaken baby syndrome' with Jayawant et al. When currently (05/2018) searching Medline using the MeSH term 'Shaken Baby Syndrome', there are 772 citations; 80 if restricted to the time frame in Donohoe. To highlight the importance of the search strategy, repeating the Medline search using the more appropriate MeSH terms associated with Jayawant et al. ('Hematoma, Subdural/aetiology' and 'Infant, Newborn'), restricted to the same time frame as Donohoe, returned 19,863 citations (05/2018). Searching Medline with MeSH terms it recognized resulted in a 280-fold increase in the number of citations returned. This highlights the importance of being an active reader of the literature. Simply accepting the search strategy (and resulting conclusions) as presented in

Donohoe (in a peer reviewed journal), without independent appraisal, would profoundly mislead the reader to the current scope of knowledge.

By way of comparison to a high-quality systematic review, Maguire et al. performed a Systematic Review of published literature to answer the question 'What are the clinical features that distinguish inflicted from non-inflicted brain injury?' [57]. Their comprehensive search strategy involves over 100 keyword combinations and 20 databases. Their search resulted in 1168 articles for full text analysis. They rigorously applied inclusion and exclusion criteria, which resulted in 320 papers which were then analyzed by 2 independent reviewers (with a third reviewer for any discrepancies). It is easy to appreciate that by reading the methods sections of the two papers, and understanding the importance of methodologic rigour, how these two papers (Donohoe and Maguire et al.) resulted in two vastly difference results.

16.8 Summary

Critical literature appraisal is now academic hand-to-hand combat. No longer can articles be read secure in the comfort that the reader is not being led astray. The reader must now be an active participant in reading the medical literature. There are four main take home messages for the reader. First, to be sceptical of everything that you read. Second, read the Materials and Methods section of the paper. This section is often skipped but can no longer be. This section will tell us if we should actually read the paper or not. Third, read the references. It will be a check of the quality of the scholarship of the author, being able to confirm that the references support what the author says they support. Fourth, peer review does not guarantee that the paper is of high quality. While peer review should be minimum criteria for reading a manuscript, in many cases it may only mean that someone else also read the paper.

References

1. Beninger PG, Beal LJ, Shumway SE (2016) Debasing the currency of science: the growing menace of predatory open access journals. J Shellfish Res 35:1–5
2. Fraser AG, Dunstan FD (2010) On the impossibility of being expert. BMJ 341:c6815
3. Guyatt G, Cairns J, Churchill D, Cook D, Haynes B, Hirsh J, Irvine J, Levine M, Levine M, Nishikawa J, Sackett D, Brill-Edwards P, Gerstein H, Gibson J, Jaeschke R, Kerigan A, Neville A, Panju A, Detsky A, Enkin M, Frid P, Gerrity M, Laupacis A, Lawrence V, Menard J, Moyer V, Mulrow C, Links P, Oxman A, Sinclair J, Tugwell P (1992) Evidence-based medicine. A new approach to teaching the practice of medicine. JAMA 268:2420–2425
4. Guyatt G, Cairns J, Churchill D, Cook D, Haynes B, Hirsh J, Irvine J, Levine M, Levine M, Nishikawa J (1992) Evidence-based medicinee. JAMA 268:2420–2425
5. Guyatt G, Rennie D, Hayward R (2002) Users' guides to the medical literature: a manual for evidence-based clinical practice. AMA Press, Chicago
6. Straus SE, Richardson WS, Glasziou P, Haynes RB (2005) Evidence-based medicine: how to practice and teach EBM. Elsevier Churchill Livingstone, Edinburgh
7. Greeley C (2015) Evidence-based radiology and child abuse. In: Kleinman PK (ed) Diagnostic imaging of child abuse. Cambridge University Press, pp 309–323
8. Sackett DL (1997) Evidence-based medicine. Semin Perinatol 21:3–5
9. Wager E, Williams P (2011) Why and how do journals retract articles? An analysis of Medline retractions 1988–2008. J Med Ethics 37:567–570
10. Byard RW (2016) The forensic implications of predatory publishing. Forensic Sci Med Pathol 12:391–393
11. Grudniewicz A, Moher D, Cobey KD, Bryson GL, Cukier S, Allen K, Ardern C, Balcom L, Barros T, Berger M, Ciro JB, Cugusi L, Donaldson MR, Egger M, Graham ID, Hodgkinson M, Khan KM, Mabizela M, Manca A, Milzow K, Mouton J, Muchenje M, Olijhoek T, Ommaya A, Patwardhan B, Poff D, Proulx L, Rodger M, Severin A, Strinzel M, Sylos-Labini M, Tamblyn R, van Niekerk M, Wicherts JM, Lalu MM (2019) Predatory journals: no definition, no defence. Nature 576:210–212
12. Laine C, Winker MA (2017) Identifying predatory or pseudo-journals. Biochem Med (Zagreb) 27:285–291
13. Ferris LE, Winker MA (2017) Ethical issues in publishing in predatory journals. Biochem Med (Zagreb) 27:279–284
14. Wood KE, Krasowski MD (2020) Academic e-mail overload and the burden of "Academic Spam". Acad Pathol 7:2374289519898858
15. Butler D (2013) Investigating journals: the dark side of publishing. Nature 495:433–435
16. Beal LJ (2017) What I learned from predatory publishers. Biochem Med (Zagreb) 27:273–278
17. Bohannon J (2013) Who's afraid of peer review? Science 342:60–65
18. PubMed. https://www.ncbi.nlm.nih.gov/pubmed. Accessed 3 June 2021
19. Think. Check. Submit. https://thinkchecksubmit.org/sample-page/check/. Accessed 5 July 2021
20. Sorokowski P, Kulczycki E, Sorokowska A, Pisanski K (2017) Predatory journals recruit fake editor. Nature 543:481–483
21. Sokal AD (1996) Transgressing the boundaries: toward a transformative hermeneutics of quantum gravity. Social Text 46(47):217–252
22. Soka IAD (1996) A physicist experiments with cultural studies. http://linguafranca.mirror.theinfo.org/9605/sokal.html. Accessed 5 July 2021
23. Seglen PO (1997) Why the impact factor of journals should not be used for evaluating research. BMJ 314:498–502
24. The PLoS Medicine Editors (2006) The impact factor game. It is time to find a better way to assess the scientific literature. PLoS Med 3:e291
25. Kurmis AP (2003) Understanding the limitations of the journal impact factor. J Bone Joint Surg Am 85:2449–2454
26. Wakefield AJ (1999) MMR vaccination and autism. Lancet 354:949–950
27. Dyer C (2010) Lancet retracts Wakefield's MMR paper. BMJ 340:c696
28. Watson JD, Crick FH (1974) Molecular structure of nucleic acids: a structure for deoxyribose nucleic acid. J.D. Watson and F.H.C. Crick. Published in Nature, number 4356 April 25, 1953. Nature 248:765
29. Smith R (2006) Peer review: a flawed process at the heart of science and journals. J R Soc Med 99:178–182
30. Godlee F, Gale CR, Martyn CN (1998) Effect on the quality of peer review of blinding reviewers and asking them to sign their reports: a randomized controlled trial. JAMA 280:237–240

31. Ray M (2016) An expanded approach to evaluating open access journals. J Scholar Publish 47:307–327
32. Greenhalgh T (1997) How to read a paper. Getting your bearings (deciding what the paper is about). BMJ 315:243–246
33. Vandenbroucke JP (2001) In defense of case reports and case series. Ann Intern Med 134:330–334
34. Love JC, Derrick SM, Wiersema JM, Pinto DC, Greeley C, Donaruma-Kwoh M, Bista B (2013) Novel classification system of rib fractures observed in infants. J Forensic Sci 58:330–335
35. Piantadosi S, Byar DP, Green SB (1988) The ecological fallacy. Am J Epidemiol 127:893–904
36. Price EA, Rush LR, Perper JA, Bell MD (2000) Cardiopulmonary resuscitation-related injuries and homicidal blunt abdominal trauma in children. Am J Forensic Med Pathol 21:307–310
37. Matshes EW, Evans RM, Pinckard JK, Joseph JT, Lew EO (2011) Shaken infants die of neck trauma, not of brain trauma. Acad For Path 1:82–91
38. Cook DJ, Mulrow CD, Haynes RB (1997) Systematic reviews: synthesis of best evidence for clinical decisions. Ann Intern Med 126:376–380
39. Alper BS, Haynes RB (2016) EBHC pyramid 5.0 for accessing preappraised evidence and guidance. Evid Based Med 21:123–125
40. Pannucci CJ, Wilkins EG (2010) Identifying and avoiding bias in research. Plast Reconstr Surg 126:619–625
41. Sica GT (2006) Bias in research studies. Radiology 238:780–789
42. Hahn U, Oaksford M (2007) The rationality of informal argumentation: a Bayesian approach to reasoning fallacies. Psychol Rev 114:704–732
43. Casaulta C, Stirnimann A, Schoeni MH, Barben J (2008) Sweat test in patients with glucose-6-phosphate-1-dehydrogenase deficiency. Arch Dis Child 93:878–879
44. Hill AB (2015) The environment and disease: association or causation? 1965. J R Soc Med 108:32–37
45. Willoughby RE Jr, Tieves KS, Hoffman GM, Ghanayem NS, Amlie-Lefond CM, Schwabe MJ, Chusid MJ, Rupprecht CE (2005) Survival after treatment of rabies with induction of coma. N Engl J Med 352:2508–2514
46. Talisse R, Aikin SF (2006) Two forms of the straw man. Argumentation 20:345–352
47. 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
48. 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
49. Geddes JF, Tasker RR, Hackshaw CD, Adams GGW, Whitwell HL, Scheimberg I (2003) Dural haemorrhage in non-traumatic infant deaths: does it explain the bleeding in 'shaken baby syndrome'? Neuropathol Appl Neurobiol 29:14–22
50. Rafaat KT, Spear RM, Kuelbs C, Parsapour K, Peterson B (2008) Cranial computed tomographic findings in a large group of children with drowning: diagnostic, prognostic, and forensic implications. Pediatr Crit Care Med 9:567–572
51. Geddes JF, Talbert DG (2006) Paroxysmal coughing, subdural and retinal bleeding: a computer modelling approach. Neuropathol Appl Neurobiol 32:625–634
52. Barnes PD, Galaznik J, Gardner H, Shuman M (2010) Infant acute life-threatening event–dysphagic choking versus nonaccidental injury. Semin Pediatr Neurol 17:7–11
53. Greeley CS (2010) Letter to the editor – Infant acute life-threatening event – dysphagic chocking versus nonaccidental injury. Semin Pediatr Neurol 17:275–278
54. Edwards GA (2015) Mimics of child abuse: Can choking explain abusive head trauma? J Forensic Legal Med 35:33–37
55. Donohoe M (2003) Evidence-based medicine and shaken baby syndrome: part I: literature review, 1966–1998. Am J Forensic Med Pathol 24:239–242
56. Jayawant S, Rawlinson A, Gibbon F, Price J, Schulte J, Sharples P, Sibert JR, Kemp AM (1998) Subdural haemorrhages in infants: population based study. BMJ 317:1558–1561
57. Maguire S, Pickerd N, Farewell D, Mann M, Tempest V, Kemp AM (2009) Which clinical features distinguish inflicted from non-inflicted brain injury? A systematic review. Arch Dis Child 94:860–867

Bayesian Interpretation of Paediatric Fractures

17

Wouter A. Karst and Hubert G. T. Nijs

Contents

17.1 Introduction

Paediatric fractures have an important role in the evaluation of child physical abuse. As explained throughout this book, some fractures are highly specific for child abuse whereas other fractures are less specific for child abuse. But how should medical professionals express a diagnostic value of fractures? Correct interpretation of paediatric fractures is crucial, as wrong interpretations might lead to incorrect accusations or to ongoing risks for the child and for other children.

Medical professionals who are involved in evaluating children for possible abuse, should be aware of several sources of bias, like cognitive bias, contextual bias, and base rate bias [1]. Unawareness of bias leads to diagnostic errors in general medicine, and certainly in paediatric radiology and child abuse paediatrics as well [2–5]. An example of unconscious bias is the impact of risk factors on decision-making, which is often larger than can be substantiated.

A probabilistic approach can be useful to separate the diagnostic value of paediatric fractures from all other information. A probabilistic approach is a way to deal with several sources of bias, but it is also a way to force medical professionals to stay within their field of expertise. Relevant information in a case could be beyond medical expertise, forcing to express the medical findings in a way that is supportive to others. In forensics, the diagnostic value of findings is reported to the trier of fact, i.e. a person (judge), or group of persons (jury), who determines facts in a legal proceeding [6]. But also outside the forensic field, one should appreciate that reporting the diagnostic value of medical findings is a logic way to formulate expert opinions [7].

Even if medical professionals need to make decisions by themselves, one should still separate the diagnostic value of the fracture from all other information that might have an effect on the probability of abuse, but not on the probability of the fracture. This separation is needed to prevent that risk factors (or protective factors), prevalence of abuse, the value of the history provided, and other factors will have an effect on the diagnostic value of the fracture. In this chapter a Bayesian approach will be presented as a way to separate the diagnostic value, also called the evidential power, from the value of other factors.

17.2 Bayesian Statistics

In Bayesian statistics, named after Thomas Bayes, an English reverend and statistician from the eighteenth century, the probability of an event is described (Fig. 17.1). His rule

W. A. Karst (✉) · H. G. T. Nijs
Department of Forensic Medicine, Netherlands Forensic Institute, The Hague, The Netherlands

R. A. C. Bilo et al. (eds.), *Forensic Aspects of Paediatric Fractures*, https://doi.org/10.1007/978-3-031-12041-1_17

Fig. 17.1 Reverend Thomas Bayes

describes how a degree of belief changes as new evidence is presented. The new evidence, which is the diagnostic value, will update a prior belief. A well-known derivation of Bayes' rule considers the ratio of the probabilities of two hypotheses:

Prior odds times *likelihood ratio* equals *posterior odds.*

This equation will be explained using an example of a 10-month-old healthy infant who presents with a humeral fracture, resulting in a consideration of abuse as a cause of that fracture.

17.2.1 Prior Probability of Abuse

In Bayesian statistics, the prior belief in two competing hypotheses needs to be determined before the impact of new (medical) findings can be calculated. In our example, the probability of abuse needs to be determined before the diagnostic value of a humeral fracture in a 10-month-old infant can be added. If abuse is the hypothesis that we would like to consider, then we need a competing hypothesis to be able to calculate prior odds of abuse. Examples of other possible causes for a fracture are medical diseases, birth and accidental trauma (see Chaps. 13 and 14). In our example, the competing hypothesis is every cause other than abuse. As mentioned before, the prior probability of abuse cannot always be determined by the medical provider. That will be discussed in more detail in Sect. 17.3.

In our example, the prevalence of abuse might be a good way to estimate the prior probability of abuse. A relevant prevalence of abuse is the prevalence in 10-month-old infants. An even better way to determine the prevalence of abuse in our case might be to look at the prevalence of abuse in 10-month-old infants who undergo radiologic examination because of a concern of a fracture. Even certain risk factors and protective factors could change the prior probability of abuse, as well as information not or not yet known to the medical provider, or information outside the field of expertise of the medical provider.

In our example, data from a study in which the prevalence of abuse was determined in young children with a fracture, with exclusion of children with head trauma, is used [8]. Using a large American database, Leventhal et al. evaluated data from 4248 infants less than 12 months old who had presented in a hospital with a fracture. Of these children, 868 children were diagnosed with abuse. This results in a prevalence of abuse of 20.4%. This 20.4% can be considered as the prior probability of abuse in children who presented with a fracture. Compared with the other 79.6% of children in the same group who were not diagnosed with abuse, the prior odds of abuse in this study were 0.256 (20.4% over 79.6%).

17.2.2 Diagnostic Value

The 10-month-old infant in our case had a humeral fracture, without any details provided. In real cases, there are more details available, e.g. from the history provided, from the physical examination, from laboratory testing, and from other radiologic examinations.

The fracture itself could change the prior belief of abuse. The diagnostic value, or evidential power of the humeral fracture, can be determined by looking at the frequency of a humeral fracture at this age in abused children, compared with non-abused children. If the fracture is more common in abused children, than there is some evidential power in favour of the hypothesis abuse. Also, if the fracture is more common in non-abused children, than there is some evidential power in favour of the hypothesis non-abuse.

The ratio between the frequency of the fracture in abused children and the frequency of the fracture in non-abused children is called the likelihood ratio. The likelihood ratio is the diagnostic value or the evidential power of the medical finding. Note that it is not relevant how common the two groups are in which the prevalence of the medical findings needs to be determined. If a certain fracture is common in abused children in a population in which child abuse is uncommon, then the number of children with these fractures might be larger in the non-abused group. Despite that, this certain fracture still provides evidential power towards child abuse.

We could look at a study in which the authors looked at both abused and non-abused children who presented with a fracture, to find out how common fractures on specific locations were [9]. In children less than 18 months old, 37 out of 377 abused children (9.8%) had a humeral fracture (regardless of type), compared to 19 out of 425 non-abused children (4.5%). Based on these data, it was 2.2 times more likely to have a humeral fracture if the child was abused, compared to non-abused children. The likelihood ratio or the diagnostic value therefore is 2.2.

17.2.3 Posterior Probability of Abuse

The updated prior probability by adding the diagnostic value of abuse results in a new probability of abuse. Looking at just a humeral fracture without any details in our example, the prior odds of 0.256 can be multiplied by the likelihood ratio of 2.2. The result is posterior odds of 0.563. This can be converted into posterior probability by dividing 0.563 by the total of (0.563 + 1), resulting in a probability of 36.0%. So, the prior probability of abuse of 20.4% was updated by the diagnostic value of a humeral fracture, resulting in a posterior probability of 36.0%.

The posterior probability of abuse can also be found in scientific papers. An example is a study in which the authors determined how many children with certain fractures were eventually diagnosed with abuse [10]. From 518 infants less than 12 months of age with a humeral fracture, 223 (43.1%) were diagnosed with abuse. This closely resembles the above calculated probability of abuse of 36.0%.

17.3 Diagnostic Value

So now it is explained that the diagnostic value of medical findings (the likelihood ratio) updates a prior belief of the ratio of two hypotheses (the prior odds). Many medical professionals find it difficult to interpret the value of the likelihood ratio. A nomogram as shown in Fig. 17.2 can be helpful. A line from the pre-test probability (not the pre-test odds) through the likelihood ratio will end in the post-test probability (not the post-test odds).

In forensic standards and guidelines from both Europe and the United States, it is stated that the strengths of evidence need to be expressed by a value of the likelihood ratio or using a verbal scale related to the value of the likelihood ratio [6, 11–13]. Interpreting fractures or other medical findings by medical professionals should be of the same level as instructed by forensic standards and guidelines. Not only is estimating the probability of abuse in some way a legal decision, it is also important to understand that medical professionals often are not able to determine the prior odds of abuse. And therefore, the final probability of abuse can most often only be guessed.

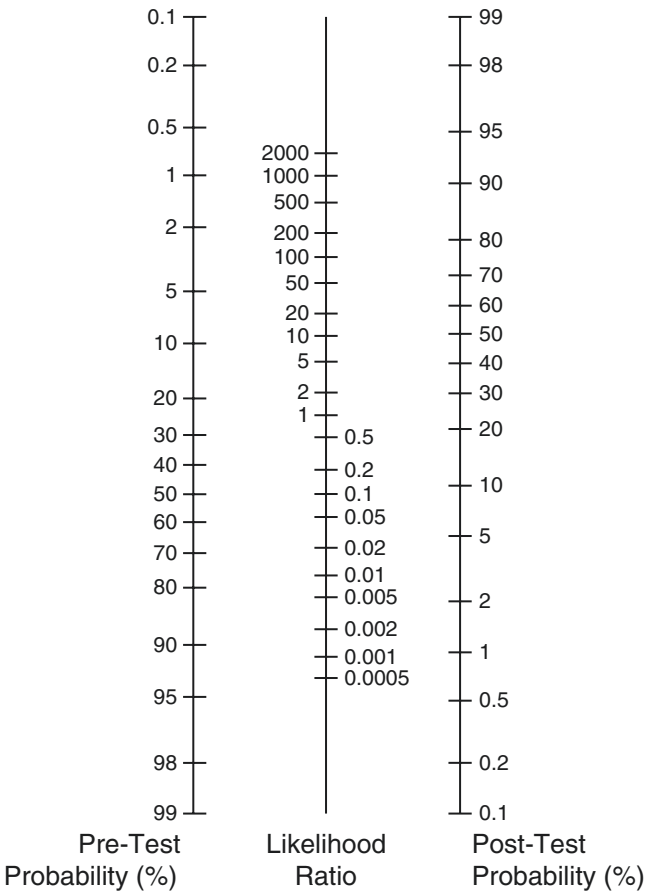


Fig. 17.2 Nomogram for calculating post-test probabilities from pre-test probabilities and likelihood ratios

The main reasons why medical professionals are not able to determine the prior odds of abuse are:

- Prior odds of abuse are dependent of the prevalence of abuse. In some situations, like at level-III hospital in an urban region, the prevalence of abuse might be different than in other situations, like at a small hospital in a thinly populated area. We never know if the prevalence of abuse in certain research settings as described in the literature is comparable to the prevalence of abuse in the setting of the medical professional that is evaluating abuse because of a fracture.
- While the prevalence of abuse is based on a group of people, individuals might have factors that increase or decrease 'their' prevalence of abuse. Risk factors or protective factors are important to consider. For some specific factors, their influence on the prior odds of abuse is in some way known. But most often not all risk factors or protective factors are known, and most often their exact level of influence is unknown, and never it is known if a

specific child deserved the known update of the prior odds, based upon group levels.

- As mentioned before, often information is lacking to determine definite prior odds of abuse. For example, a caregiver might have confessed certain actions to a relative or to a friend. If the medical professional is not aware of this confession, the probability of abuse will be estimated wrongly. And even if the medical professional has all information available, then certain information might be beyond the expertise of the medical professional. For example, determining the veracity of the history provided, is within the judicial field of expertise.

So, the diagnostic values of medical findings need to be expressed as likelihood ratios. Likelihood ratios can be compared with weights on a scale. The scale is pointing towards abuse or non-abuse (or whatever which hypotheses are considered). The position of the scale before the medical input, is the prior probability of abuse. The likelihood ratio is the weight that the medical professional is adding to the scale. A light weight reflects a small likelihood ratio or little diagnostic value. A heavy weight reflects a high likelihood ratio or much diagnostic value. The new position of the scale, after the medical professional added the weight, reflects the posterior probability of abuse.

Remember that the diagnostic value refers to the probability of the medical findings, and does not refer to the probability of abuse. If you add a heavy weight to the scale, then that often leads to a new position of the scale pointing towards the direction of the weight. But that is not often true: it might be that the weights that were already on the scale (based upon information unknown to the medical professional, for example) were on the opposite direction. Therefore, the probability of abuse is most often impossible to determine by the medical professional alone. The medical professional can determine the probability of medical findings if the child was abused, and relate that to the probability of the medical findings if the child was not abused. This is the diagnostic value or the likelihood ratio.

In the example used in this chapter, the only medical finding to consider was the humeral fracture. However, most often more medical findings need to be interpreted. The more findings that need to be considered, the more complex the interpretation is. Likelihood ratios from medical findings which are independent from each other, can be multiplied. But the level of dependency needs to be considered to know what fraction of the likelihood ratio of new findings can be added to likelihood ratios of other findings. Sometimes the level of dependency is known from the medical literature, for example from clinical prediction rules. But more often the level of dependency needs to be estimated. In those cases, it is not possible to calculate an exact likelihood ratio. A verbal scale related to the value of the likelihood ratio can be used in those cases. The relation to the range of values of the likelihood ratio is important, so that we all speak the same language when using verbal scales. In Table 17.1, the verbal scale used by the European Network of Forensic Science Institutes is presented [12].

Table 17.1 Verbal scale of values of likelihood ratio, in an example of the hypotheses abuse versus non-abuse

LR	Verbal equivalent
1–2	The findings provide no assistance in addressing the issue
2–10	The findings are slightly more probable given (abuse) relative to (non-abuse)
10–100	The findings are more probable given (abuse) than (non-abuse)
100–1000	The findings are appreciably more probable given (abuse) than (non-abuse)
1000–10,000	The findings are much more probable given (abuse) than (non-abuse)
10,000–1,000,000	The findings are far more probable given (abuse) than (non-abuse)
1,000,000 and above	The findings are exceedingly more probable given (abuse) than (non-abuse)

17.4 Conclusion

Interpreting fractures in children is challenging, especially if abuse is being considered as a cause of fractures. It is in everyone's interest that medical providers give an accurate and solid interpretation of the medical findings, nothing more and nothing less. The diagnostic value of medical findings needs to be expressed as likelihood ratio. This ratio reflects the evidential power of the medical findings. In this chapter, it is explained how a likelihood ratio can be used to determine the probability that a child was abused.

References

1. Dror IE (2020) Cognitive and human factors in expert decision making: six fallacies and the eight sources of bias. Anal Chem 92:7998–8004
2. Lane WG, Dubowitz H (2007) What factors affect the identification and reporting of child abuse-related fractures? Clin Orthop Relat Res 461:219–225
3. Skellern C (2015) Minimising bias in the forensic evaluation of suspicious paediatric injury. J Forensic Leg Med 34:11–16
4. Laskey AL, Stump TE, Perkins SM, Zimet GD, Sherman SJ, Downs SM (2012) Influence of race and socioeconomic status on the diagnosis of child abuse: a randomized study. J Pediatr 160(1003–1008):e1001
5. Loos MHJ, Allema WM, Bakx R, Stoel RD, van Rijn RR, Karst WA (2020) Paediatric femur fractures-the value of contextual information on judgement in possible child abuse cases: are we bias? Eur J Pediatr 180(1):81–90
6. Cole SA, Barno M (2020) Probabilistic reporting in criminal cases in the United States: a baseline study. Sci Justice 60:406–414

7. Buiskool M, Nijs HG, Karst WA, Berger CE (2016) More on the strength of evidence in forensic pathology. Forensic Sci Med Pathol 12:238–239
8. Leventhal JM, Martin KD, Asnes AG (2010) Fractures and traumatic brain injuries: abuse versus accidents in a US database of hospitalized children. Pediatrics 126:e104–e115
9. Pandya NK, Baldwin K, Wolfgruber H, Christian CW, Drummond DS, Hosalkar HS (2009) Child abuse and orthopaedic injury patterns: analysis at a level I pediatric trauma center. J Pediatr Orthop 29:618–625
10. Leventhal JM, Martin KD, Asnes AG (2008) Incidence of fractures attributable to abuse in young hospitalized children: results from analysis of a United States database. Pediatrics 122:599–604
11. Association of Forensic Science P (2009) Standards for the formulation of evaluative forensic science expert opinion. Sci Justice 49:161–164
12. European Network of Forensic Science Institutes (2015) Guideline for evaluative reporting in forensic science – approved version 3.0. European Network of Forensic Science Institutes, Wiesbaden
13. National Research Council (2009) Strengthening forensic science in the United States: a path forward. The National Academies Press, Washington, DC