The radiological dating of injuries

Children who present after accidental trauma virtually always have a clear history of the events associated with the injuries. When there is no history (from an infant or unhelpful parents) or there are other suspicious circumstances one of the many factors involved in the equation that leads to or excludes a diagnosis of child abuse is the dating of those injuries. The injuries may involve the soft tissues, the skeleton, the internal viscera, or the brain. The colour changes exhibited into more mature sequestra are well known to paediatricians but the dating of injuries to the skeleton, brain and, less commonly the viscera, is a task that usually falls to the radiologist member of the child abuse team. This annotation will centre on the dating of injuries to the skeleton and briefly discuss intracranial injuries.

While there are thousands of published contributions covering the field of fracture repair there is very little on the radiological dating of fractures and even less relating fracture age to child abuse. The most comprehensive review is by O'Connor and Cohen.1

Anatomy and physiology of the developing skeleton2
At birth the diaphyses or shafts of the tubular bones are composed of fetal or woven bone that lacks haversian systems. However, as periosteal mediated appositional bone formation and remodelling enlarge the overall diameter of the shaft and the width of the cortices, mature bone becomes dominant. This early bone is very vascular and more porous than the maturing bone of older children and adults and better able to tolerate deformation. Porous retard progression of a fracture line but increase the likelihood of compression and it is this reason that makes greenstick and buckle, or torus, fractures more common in childhood. Immature periosteum also differs from that in the adult in being thicker and more loosely attached to the underlying cortex. It minimises displacement of diaphyseal fractures and is capable of faster callus and membranous bone formation.3 Towards the end of the bone, adjacent to the growth plate is the metaphysis, a site characterised by decreased cortical bone and increased trabecular bone. The trabecular portion undergoes extensive remodelling as the primary spongiosa is transformed into more mature secondary spongiosa. This area of transformation is structurally weak during the period of rapid growth during infancy.4

Due to the child’s more flexible skeleton and increased amount of stretch allowed by the soft tissues the majority of childhood fractures are undisplaced.

Histological phases of fracture healing
The morphological sequence of fracture repair, namely haematoma, tissue metaplasia, soft callus, hard callus, and remodelled bone dates from the ideas of John Hunter in the 18th century and will be used as the basis for the description here.5 However, because of the limited number of pathological specimens of paediatric trauma that are available for histological examination and because there are so many variables in the healing process such as the severity of the injury, the degree of displacement at the fracture site and the degree of immobilisation, histologists have relied heavily on animal work. Extrapolation from young animals to young humans may not be valid but it is true to say that the repair process proceeds much more quickly in the infant than the child and both are quicker than the adult. This is especially true of the stage of production of fibrocartilaginous matrix in the callus.6

Within hours of a closed shaft fracture of a long bone and the initial haematoma there follows an initial stage of induction, characterised by inflammatory exudate from the ruptured blood vessels of the bone marrow, cortex, periosteum, and surrounding soft tissues. Inflammation is present from a few days to several weeks and is accompanied clinically by soft tissue swelling, pain, and immobility. Osteoclasts are mobilised and become active within four to seven days and with the removal of necrotic bone from the fracture ends the fracture becomes wider and less well defined.

The second stage, or stage of soft callus, overlaps the first and is distinguished by the formation of periosteal new bone and endosteal callus. By the fourth day new osteoid tissue may be seen histologically. Within the next few days periosteal new bone formation is evident and by the end of the first week there is a mass of callus composed of new blood vessels, fibrous tissue, cartilage, and bone. Calcium uptake into the callus begins within a few days of the fracture but does not reach a peak for several weeks.6 The earliest mineralisation is due to calcification of new cartilage, with new bone formation only evident histologically at one week. It is with the progression of these latter two processes that healing becomes evident radiographically and this stage ends when the bone fragments can no longer be easily moved and the fracture line begins to fade.

At the stage of hard callus the fracture is solidly united and periosteal and endosteal new bone are converted to lamellar bone. The final and longest stage of fracture healing is remodelling with a slow change in the shape of the callus and bone if there is residual deformity. Rotational deformities are very little affected by remodelling. The phenomenon of overgrowth is a feature of fractures of the immature skeleton, particularly femoral fractures. The degree of overgrowth is inversely related to age and in infants it is desirable to allow a degree of overriding at the fracture site to compensate for this complication.

Differences between the abused skeleton and the ‘normal’ skeleton
In addition to characteristic fractures, for example metaphyseal fractures and multiplicity of sites and differing ages, there are a number of other differences between fractures in the abused child and ordinary ‘accidental’ fractures that are relevant to fracture healing. Firstly, abuse fractures occur in infancy with very few fractures occurring over the age of 4 years.7-9 Secondly, there may be delay between the episode of trauma and the time medical attention is sought or given.
This may result in poor immobilisation of the fracture or even re-fracture through a partially healed injury. A repetitive injury to the same bone within hours of the first injury will produce yet more bleeding but healing will then proceed normally. A repetitive injury a week or more after the initial event will cause disruption of callus as well as further haemorrhage. The latter scenario, in particular, leads to the appearance of 'exuberant callus', a sign observed not only in child abuse but also in other conditions where fractures may present late, for example congenital insensitivity to pain and osteogenesis imperfecta.

**Growth plate and metaphyseal fractures**

Unlike growth plate fractures in the older child those in the abused child most commonly traverse the most immature primary spongia where columns of calcified cartilage are transformed into metaphyseal trabeculae. Metaphyseal fractures frequently do not disrupt the tightly adherent periosteum and will not, therefore, initiate subperiosteal new bone formation. This creates a particular problem when dating the age of injury. If a periosteal reaction is present it is usually modest in amount and may only produce a haziness of the cortex. When massive periosteal reaction is present it reflects either displacement of the metaphyseal fragment or a shearing injury to the periosteum. During healing, bone resorption will result in indistinctness of the margins of the fracture and endosteal callus will obscure the fracture line and produce a band of sclerosis within the metaphysis but these signs are somewhat subjective. After fracture, discrete radiolucencies extending from the growth plate into the metaphysis have been described and attributed to fracture repair. Trueta and Amato noted an increase in the thickness of the hypertrophic cartilage zone of the cartilaginous growth plate in rabbits with experimental disruption of metaphyseal vasculature, and Kleinman et al have confirmed that these radiolucent extensions of the growth plate into the metaphysis do indeed correlate with cartilage hypertrophy during healing of metaphyseal fractures. They tend to be single and focal with minimal osseous injury and broad and multiple with extensive injury. The depth of penetration of the growth plate into the metaphysis relates to the age of the injury and, although further work needs to be undertaken, it is hoped that by using the known rates of growth of different metaphyses it may be possible in the future to date more accurately a metaphyseal injury from its radiological appearance.

**Radiological changes in the soft tissues**

Haemorrhage and inflammatory exudate begin immediately after the injury. They obliterate the normal lumen fat planes and well defined muscle boundaries around the injured site and are frequently the first and may be the only evidence of a fracture. In the absence of a major underlying fracture these changes persist for a few days only but more severe injuries may be associated with soft tissue changes which persist for longer.

**Response of the periosteum to injury**

Periosteal new bone is laid down in response to a number of types of injuries such as infection and thermal injury as well as physical trauma. Elevated periosteum is not visible radiographically until calcium accumulates in the superperiosteal haematoma and the new bone. The first appearance of periosteal new bone is dependent on the age of the child, the younger the child the earlier it appears. In the infant it can be seen, radiographically, as early as four days after injury but seven to 14 days is more usual. Cumming, in a study of birth related fractures concluded that the absence of periosteal new bone 11 days after birth should suggest the possibility of abuse. Beyond the neonatal period periosteal reaction delayed longer than 21 days may be a consequence of poor nutrition or vitamin D deficiency. The amount of subperiosteal new bone increases with repeated trauma and accumulating subperiosteal haemorrhage, but chronic repetitive trauma may be of such severity as to destroy periosteal new bone as it develops with resultant failure of the usual sequence of changes with healing.

Ultrasoundography has been used to demonstrate subperiosteal haemorrhages, occult long bone fractures, and costochondral injuries before radiographs confirm the presence of bony injury and may be a useful supplementary investigation when attempting to date an acute injury. Radionuclide bone scanning with its low radiation dose has gained increasing popularity in recent years but its role is controversial. Sty and Starshak found a false negative rate of 0-8% for scintigraphy which prompted them to conclude that the test should be the screening procedure of choice. However, Merten et al had a false negative rate of 27% for scintigraphy and advised that it should occupy a secondary role only. From the data of these and others we can conclude that scintigraphy demonstrates superior sensitivity for rib fractures, especially at the costovertebral junction, undisplaced shaft fractures and subperiosteal haemorrhage. Rosenthal et al reported that the earliest fracture demonstrated with radionuclides was seven hours after the injury and that if a bone scan obtained three or more days after injury fails to reveal a focal lesion then a fracture has been excluded. A positive scan steadily becomes less so as the age of a fracture increases but there is no definite time course. The pitfalls of imaging with a bone-seeking isotope are that symmetrical metaphyseal injuries, adjacent to normally ‘hot’ epiphysial growth plates, some vertebral body fractures, and many skull fractures may be undetectable.

**Fracture line definition**

Fresh fractures, including metaphyseal fractures, have sharply defined margins. With the development of an osteoclastic response to necrotic bone the fracture ends become less well defined and the fracture widens. There are no objective criteria to estimate fracture healing using this sign but it is not apparent radiologically before one week and reaches a peak at two to three weeks.

**Callus formation**

With the production of osteoid and its subsequent calcification and ultimate transformation to bone there is an increase in density along the fracture line. With impacted fractures and other fractures where the periosteum remains intact or little disturbed this may be the only evidence of injury and it is, therefore, a less valuable sign that periosteal new bone and fracture line clarity. Again, movement may inhibit or destroy endosteal callus and a fracture line may remain clearly visible after a bone has united by periosteal new bone. Endosteal soft callus is first visualised as an ill defined increase in density and begins to form soon after the first appearance of periosteal new bone, that is at about 10 to 14 days. When lamellar bone bridges the fracture, about a week later, the stage of hard callus has begun. The chronology of callus formation and fracture consolidation is dependent on age. A birth related fracture may be united at one month but a similar fracture in an 8 year old will be united at eight weeks.
Remodelling

Estimation of the age of a fracture by this criterion is very difficult because initial deformity, the amount of callus produced, and age of the child are major variables. In the young child with a stable, undisplaced fracture the remodelling process may be complete at three months while the older child with an angular deformity or a markedly displaced fracture may continue to remodel for two years.

Intracranial haemorrhage

Computed tomography is the primary diagnostic method for the evaluation of head trauma and its value in the management of the abused child is well documented.25–29 The appearance of haematoma on computed tomography changes slowly with time and is well understood.30 Freshly clotted blood is of higher attenuation (whiter) than brain, becoming the same density as brain from two to four weeks, and less dense than brain after this time, approaching the density of cerebrospinal fluid by six weeks. Chronic subdural haematoma associated with ventricular dilatation may be difficult to distinguish from cerebral atrophy on computed tomography but magnetic resonance imaging (MRI) readily identifies it as chronic haematoma. MRI has been shown to be superior to computed tomography in the investigation of subdural or chronic head injury but the MRI appearances during acute haemorrhage are more complex, change rapidly during the first week, and have a wide differential diagnosis.32 Nevertheless, once haemorrhage has been identified on computed tomography, MRI can be used to assess its age more accurately if that is appropriate.

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Fungal skin infections

Fungal skin infections or superficial mycoses are common causes of skin disease in most age groups. They include three main diseases—dermatophyte or ringworm infections, candidosis, and pityriasis versicolor. There are significant differences, though in the epidemiology and clinical behaviour of these infections when they present in childhood. This is in part due to variations in rates of exposure in the case of dermatophytosis but in other examples may reflect real differences in the expression of host resistance.

Resistance to skin infection

The factors that determine the outcome of skin infection are divided into innate mechanisms and those that require the expression of immunological memory.4–7 Innate mechanisms include the capacity for epidermal cells to respond to damage to the stratum corneum by increased proliferation. Although scientific proof is lacking there is no reason to suppose that this capacity varies with age, except perhaps in photoaged skin. Likewise fungal infection due to reversible binding by unsaturated transferrin,3 present in sweat and serum, is not known to be affected by age. However the presence of free fatty acids (FFAs) on the skin surface is known to affect fungi as those of medium chain length inhibit the growth of dermatophytes while facilitating the growth of pityrosporum yeasts. The composition of sebum containing these FFAs changes at puberty when the inhibitory moieties of medium chain length come to dominate. This is believed to be the explanation of the comparative rarity of scalp ringworm, tinea capitis, in postpubertal children whereas pityrosporum infections, such as pityriasis versicolor, are seen more frequently. FFAs appear to act by direct inhibition of fungal growth but they may also interfere with adhesion,4 the interaction between fungal cell wall receptors with keratinocytes, which is essential for subsequent penetration of the skin.

The other main processes determining the outcome of fungal invasion are antigen reception by Langerhan’s cells,5 the accumulation of effector cells, principally neutrophils, the site of infection via the production of chemotactic factors by the infected tissue, and the immune response of T lymphocytes, predominantly helper cells.6,7 The means by which the latter effect fungal clearance is also not understood. Production of cytokines which amplify neutrophil killing or

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