

Fractures in Abused Children

What tests? When?

VFPMS Seminar

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Background

What do we want to know?

Does bone injury exist?

What pattern/type of injury is this?

Are there other injuries? (Bone / otherwise)

What is the mechanism of injury?
What forces caused it/ contributed?
Timing? How long ago did it happen?

Does the 'explanation offered' account for the injury?
If not, why not? What might better explain it?

How do we know what we know?

- Forensic pathology – child homicides
 - Anatomical pathology and Histology
- Radiology
- Clinical forensic medicine (cause of injury)
- Population Health (epidemiology)
- Orthopaedic surgery
- Research – biomechanics / forces and physics
- Accidental bone injury – patterns of injury and healing
- Metabolic & genetic disease states (abnormal bone)
- The courts – criminal justice system
- The media

There is **MUCH** we still do NOT know

6 favourite references

Bilo RAC, Robben SGF, vanRijn RR Forensic Aspects of Paediatric Fractures Differentiating Accidental Trauma from Child Abuse 2010 (Springer)

Kleinman P., Diagnostic Imaging of Child Abuse 2nd ED 1998 (Mosby)

Offiah and Hall, Radiological Atlas of Child Abuse 2009 (Radcliffe)

WCPSRG Core-Info @ <http://www.core-info.cardiff.ac.uk/fractures/index.htm>

Carole Jenny, Child Abuse and Neglect; Diagnosis Treatment and Evidence

Giardino and Alexander, Child Maltreatment: A Clinical Guide and Reference 3rd ED 2005 (GW Publishing)

Child Homicides

90% CAN fatalities aged < 5
41% CAN fatalities aged < 1
Majority have healing #

Many have NO external signs of injury

Complex skull # (brain injury common
fatal injury in < 2yo)

NB Rib # = common

P. Kleinman

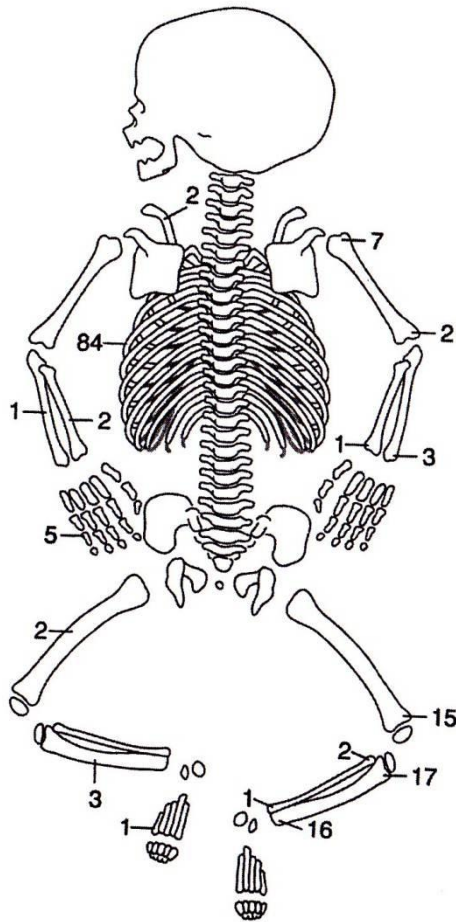


Fig. 2-2 Distribution of 165 inflicted fractures in 31 infant fatalities. Single vertebral fracture is not shown. The skull fractures in 13 infants are not included in the analysis. (From Kleinman PK, Marks SC Jr, Richmond JM, Blackbourne BD: Inflicted skeletal injury: a postmortem radiologic-histopathologic study in 31 infants, *AJR* 165:647-650, 1995.)

Serious Assault (physical abuse)

Some studies of Physically Abused children - 11% - 53% have #

Diaphyseal # > metaphyseal # (4:1)

Metaphyseal # around knee and ankles > other limb joints

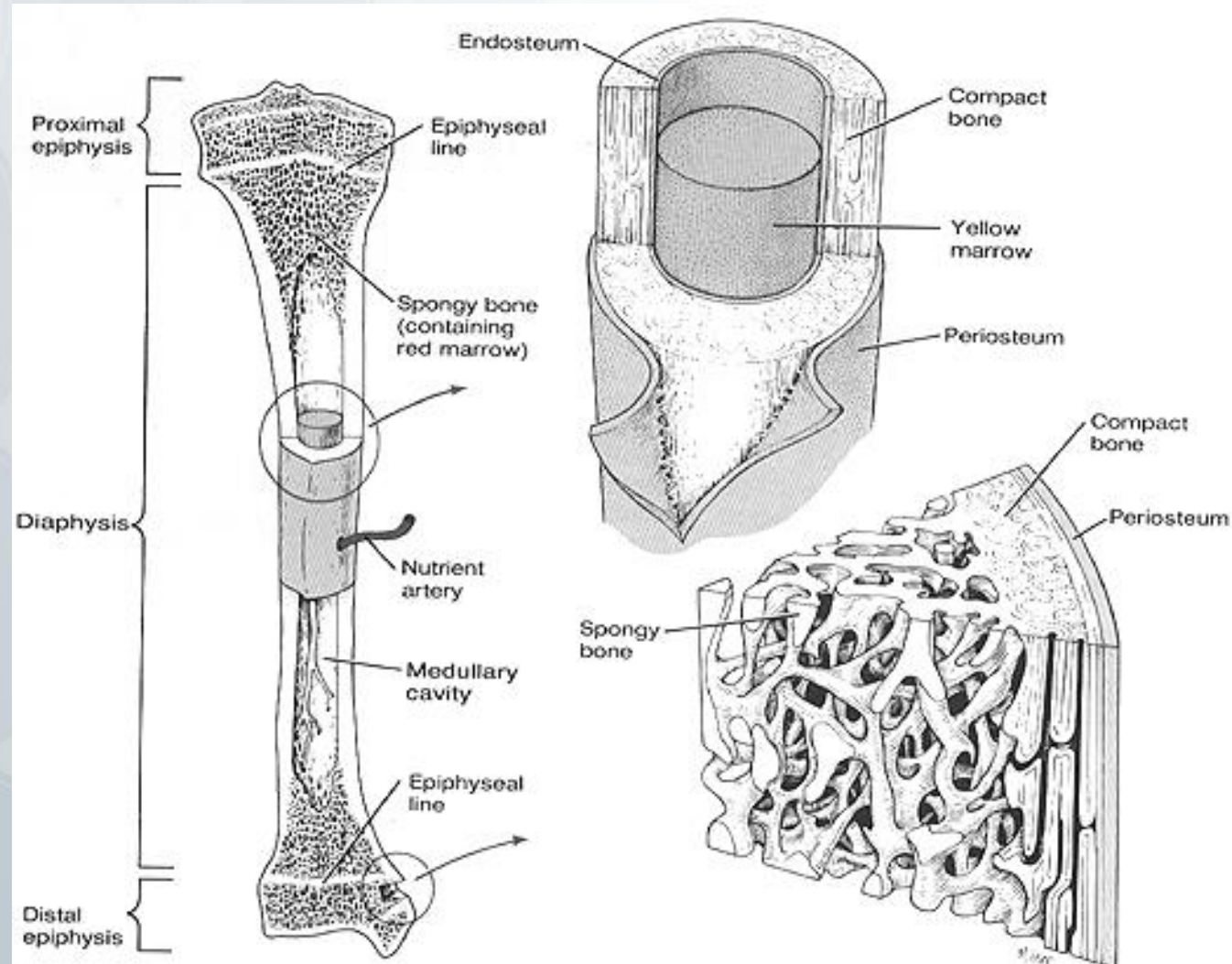
Bruising > isolated diaphyseal # tranverse > spiral

Of Shaft # , middle 1/3 (50%) distal 1/3 (41%)

Most common long bone (tibia, femur, humerus)

Anatomy of Long Bone

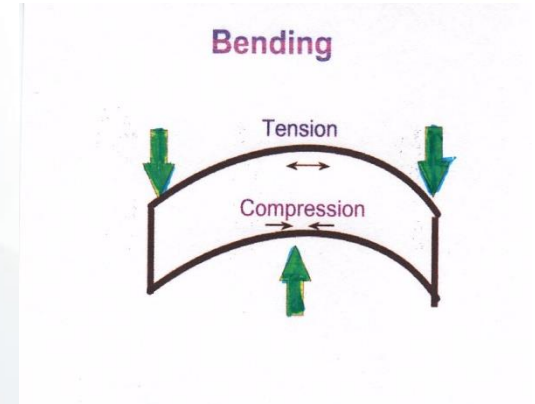
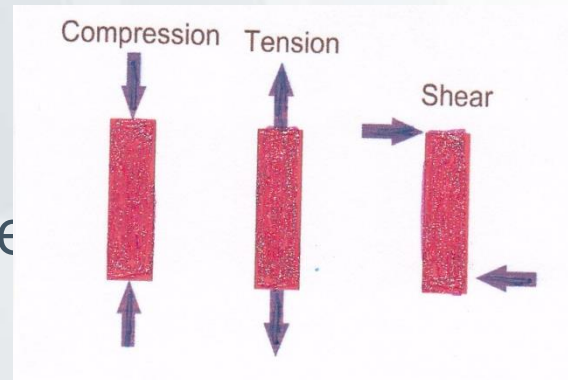
Basic Medical
Anatomy, by Alexander
Spence
(Benjamin/Cummings
1990).



What forces injure bones?

Mechanical forces

- Compression
- Stretching (tensile)
- Shearing
- Direct blow
 - $F=MA$
 - $F/SA \sim$ tissue damage
- Bending
- Rotation / Torsion (twisting)
- Acceleration/decelleration
- Indirect loads via muscles and joints



$$\text{Stress} = F/A$$

$$\text{Strain} = \text{along length}$$

Stress/strain curve ->
Yield point
Stiffness \sim elasticity

DIRECT INJURY MECHANISMS

Eg. long bones (diaphysis)

- Tapping (blow on small S Area)
- Crushing (high force on large area)
- Penetrating (high force on small area)
- Penetrating explosive (high force – lots of tissue damage)

INDIRECT INJURY MECHANISMS

Eg. long bones (diaphysis)

- Transverse fracture - tensile force (eg patella #)
- Oblique - axial compressive force (distal femur #)
- Spiral - torsional force (tibia)
- Spiral with small butterfly - bending force (humerus)
- Transfers oblique with large butterfly - axial compression and bending (tibia)

How do children's bones differ from the bones of adults?

Softer, more elastic

Structure of bone matrix matures with age

Growth plates at ends of long bones

Metabolism differs

More cartilaginous

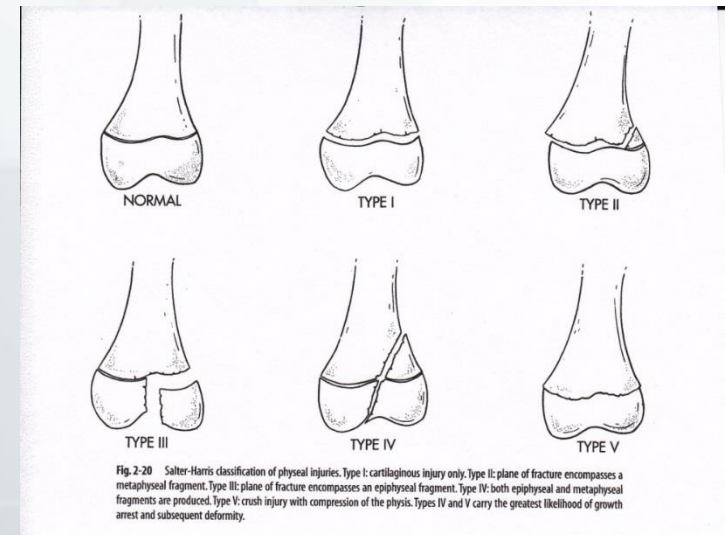
Vascular differences

Poorly attached periosteum along shaft of long bones

Ligamentous laxity (less rigid supports around joints)

Different response to forces

Differing fracture patterns



How do children's bones react to mechanical forces?

Greenstick

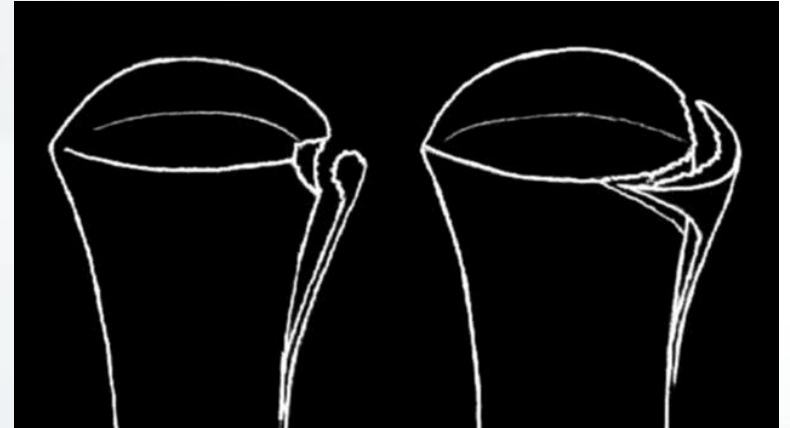
- Torus #, Buckle #

Corner fracture = Bucket handle fracture

- Epiphyseal-metaphyseal #

Periosteum tightly anchored to epiphyseal cartilage

- traction/torsional forces pull the periosteum



What is the Classic Metaphyseal Lesion (CML)? (Kleinman's term)

planar micro-fractures
at the metaphyseal-
epiphyseal regions
in the immature primary
spongiosum layer
disc with thin centre and
thicker outer rim
typically described as
“bucket handle” and
“corner fractures”)

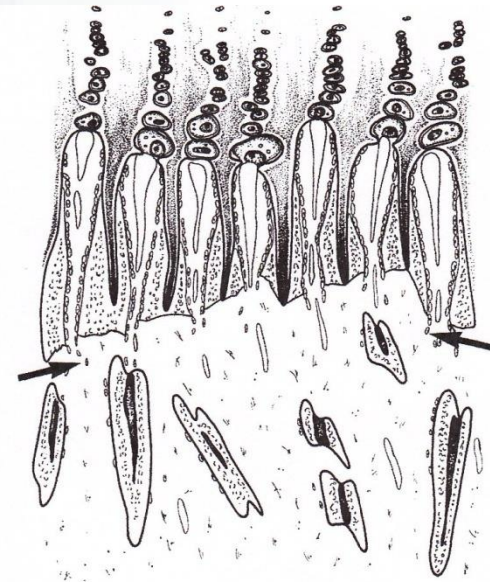


Fig. 2-8 Diagrammatic representation of CML. The fracture is a primarily osseous injury extending in a planar fashion through the primary spongiosa of the metaphysis (arrows). (Compare with normal anatomy in Fig. 1-6.)

Subperiosteal new bone (SPNBF)

Periosteum poorly attached to bone shaft in infants

Strongly attached to cartilage at epiphysis

Promptly clinically obvious at epiphysis??
Silent at diaphysis?

5-14 d thin layer SPNBF – Xray

Nonspecific for abuse

- (breech(Snededecor)
- Infection, trauma, metab, other)

May hint at # (eg tibia)

Forces

Pulling twisting, used as handle for shaking, flailing limbs when shaken + direct blows

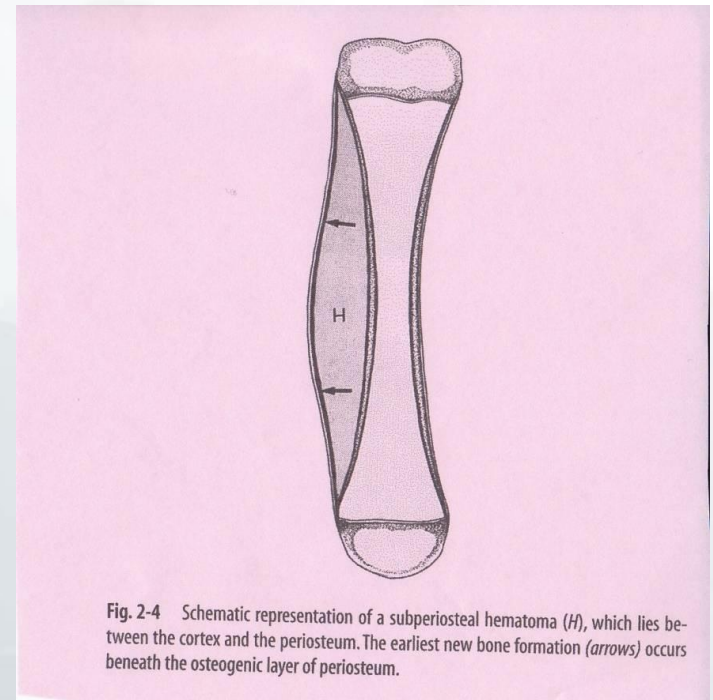


Fig. 2-4 Schematic representation of a subperiosteal hematoma (H), which lies between the cortex and the periosteum. The earliest new bone formation (arrows) occurs beneath the osteogenic layer of periosteum.

Common bone injuries seen in abused children

BEWARE

- Classic Metaphyseal lesions
- Rib fractures, esp posterior
- Rare
 - Scapular
 - Spinous processes
 - Sternal

BE ALERT

- Multiple fractures (esp bilateral)
- Fractures of different ages

Uncommon

- Epiphyseal separations
- Vertebral body #/subluxations
- Digital #
- Complex skull #

Common but Nonspecific (seen after BOTH abuse and accidents)



SPNBF

Clavicle #

Long bone shaft #

Linear skull #

How do children's bones heal?

Non-Osteonal bone healing

- Callus or gap heals with fibrous tissue that differentiates into lamellar or woven bone (UNCOMMON in children)

Osteonal bone healing

- Primary bone healing or primary gap healing (no callus)
- Secondary bone healing with callus formation

Dead bone serves as a mechanical stabiliser until it is remodelled

Remodelling requires weight bearing (Wolff's law)

Spiral and oblique fractures heal more rapidly than transverse fractures (greater surface area of fracture ends / less surrounding soft tissue damage)

Structure and mechanical properties are restored (unlike so skin and tendon)

Local factors influence rate of healing (blood flow coexisting tissue damage)

How do we investigate suspected bone injury in children?

FORENSIC EVALUATION

Build on existing knowledge base

Collate information

- History
- Examination findings
- Radiological investigation results
- Other results

Evaluate the correlation

Test hypotheses

LOGIC & REASONING

“Best fit” and alternative explanations (probability theory)

RADIOLOGICAL INVESTIGATION

- Xray
- Skeletal survey
- CT
- Nuclear medicine scan
- MRI
- Ultrasound



4 phases of bone healing (Radiol)

1. Induction phase

Time of injury to the appearance of new bone at the fracture site.

Inflammatory response may last a few days and reveal itself on x-ray in the form of soft tissue swelling with displacement and obliteration of normal fat and fascial planes.

A fracture line that might initially appear sharp can gradually become less well defined + blurs the fracture margins.

A nuclear medicine scan and MRI scan may detect subperiosteal changes that are not yet evident on x-ray.

4 phases of bone healing

2. soft callous (subperiosteal new bone)

In infants this can occur within approximately 7 to 10 days, later (10 to 14 days) in older children.

By approximately 10 days, a cellular collar surrounds the fracture site.

Woven bone calcifies approximately 10 to 15 days after injury.

Exuberant callous formation can be a sign of fracture instability, and/or repetitive injury.

4 phases of bone healing

3. hard callous

Forms when periosteal and endosteal bone begins to convert to lamellar bone.

This phase begins in infants at 14 to 21 days at the earliest, and peaks at 21 to 42 days.

11-year-old girl with fracture of mid radius. Anteroposterior radiograph shows periosteal new bone (*arrows*) separated from underlying cortex by thin radiolucent line.

4 phases of bone healing

4. Remodelling occurs with gradual correction of deformity.

Begins at approximately 3 months and peaks at 1 to 2 years

Heals completely and appears indistinguishable on x-ray from a bone that has not been injured.

Healing generally occurs more rapidly in younger infants

The rate at which bones heal, and remodelling occurs, varies according to the child's age, the anatomy of the injured bone, the site and nature of fracture (including the degree of angulation and separation of bone segments), and metabolic processes that enable healing of bone injury.

Histological evidence of healing

1. INDUCTION

Lasts ~ 3 weeks from the time of injury.

Osteoblastic activity is stimulated at the area of blood flow disruption. Radiographically, soft-tissue edema and hematoma characterize this stage.

2. INFLAMMATORY phase

- inflammatory exudate
- follows local necrosis and cellular proliferation.

Osteoblasts become active 7 days after injury with bone resorption occurring at areas of necrosis. (Maybe) peaks at 2-3 weeks after injury and is defined by a loss of fracture line definition.

Bone resorption -> widened fracture gap

Histological evidence of healing

3. REPARATIVE phase (Soft callus)

Periosteal and endosteal calcium deposition and the growth of new osteoid tissue.

Calcium deposition begins within a few days of fracture and reaches a peak at several weeks

Lasts ~ 2-6 weeks

Increased density at the fracture margins, (Islam - seen in nearly 90% of fractures at 6 weeks, increase in density at fracture margins was not seen after 11 weeks)

4. CONVERSION (Hard callus)

Conversion of periosteal and endosteal new bone to lamellar bone with bridging of the fracture line.

Periosteal new bone becoming inseparable from the adjacent cortex and callus density becoming equal to that of adjacent bone.

(Islam - fracture callous density was equal to or greater than that of cortex after 10 weeks (90% of fractures)).

Histological evidence of healing



5. REMODELLING stage.

Changes in callus & bone shape.

(Maybe) lasts from 3 months to 2 years after injury.

(Islam - remodeling beginning as early as 4 weeks with 95% of fractures continuing to show remodeling after 8 weeks.)

Skeletal survey (NO babygram!)

Skull (SXR)

AP and lateral, plus Towne's view for occipital injury.
SXR should be taken with a skeletal survey even if a CT scan has been performed.

Body:

AP/frontal chest (including clavicles)
Oblique views of the ribs (left and right)
AP Abdomen with pelvis and hips

Spine:

Lateral spine - cervical and thoraco-lumbar

Limbs:

AP humeri, AP forearms
AP femurs, AP Tib/fib
PA hands and AP feet
Supplemented by:

- Lateral views of any suspected shaft fracture.
- Lateral coned views of the elbows/wrists/knees/ankles may demonstrate metaphyseal injuries in greater detail than AP views of the limbs alone.

The consultant radiologist should decide this, at the time of checking the films with the radiographers.

Brain imaging:

CT (brain and bone windows) is the method of choice in the acute phase.
A linear skull fracture may not be identified on CT (on bone windows)



PEDIATRICS Vol. 123 No. 5 May 2009, pp. 1430-1435

Bone Scan

“HOT SPOTS”

Subtle bone injury,
Infection,
Inflammation,
Soft tissue trauma,
Growing epiphyses
and other pathological
causes of increased
uptake of radio
nucleotide

Bone scans have little
place in fracture dating
as they become
positive within 7 hours
and can remain
positive for up to one
year

Additional radiology?

Coned views of suspicious sites

Re-examination of hot spots

Repeat Xrays in 2+ weeks (eg to see whether callus has developed at site of suspected fracture)

Occasionally = MRI (spinal injury?) CT?

Very Occasionally = Ultrasound (soft tissue injury DDx #?)

NB CONSIDER AND INVESTIGATE POSSIBLE HEAD TRAUMA

Estimate of time since injury

**TABLE 7-1 TIMETABLE OF RADIOLOGIC CHANGES
IN CHILDREN'S FRACTURES***

Category	Early	Peak	Late
1. Resolution of soft tissues	2–5 days	4–10 days	10–21 days
2. SPNBF	4–10 days	10–14 days	14–21 days
3. Loss of fracture line definition	10–14 days	14–21 days	
4. Soft callus	10–14 days	14–21 days	
5. Hard callus	14–21 days	21–42 days	42–90 days
6. Remodeling	3 mo	1 yr	2 yr to physeal closure

*Repetitive injuries may prolong categories 1, 2, 5, and 6.
SPNBF, Subperiosteal new bone formation.

Kleinman PK. Diagnostic imaging of child abuse.
2nd ed. St Louis:
Mosby, 1998

Systematic review,
3 papers
189 children,
243 #,
56 aged < 5 years

Prosser I., Maguire S., Harrison SK
et al, How old is the fracture?
Radiological dating of fractures in
children: a systematic review *Am
J Roentgenol* 2005; 184: 1282-6

Papers on bone healing in children

INEXACT SCIENCE

Islam O, Sobeleski D, Symons S, Davidson LK, Ashworth MA, Babyn P. Development and duration of radiographic signs of bone healing in children. Am J Roentgenol 2000;175:75-78

Yeo LI, Reed MH. Staging of healing of femoral fractures in children. Can Assoc Radiol J 1994;45:16-19

Cumming WA. Neonatal skeletal fractures. Birth trauma or child abuse? J Can Assoc Radiol 1979;30:30-33

Hard callus and early remodelling is seen at 8 weeks in majority of cases

Early callus (calcified SPNBF) noted as early as 7 days (neonates)

Dating of fractures is an inexact science

The radiological features of bone healing are a continuum, with considerable overlap

Radiological estimates of time of injury are in terms of weeks rather than days. It is vital that all investigating agencies are aware of these broad time frames

Fracture dating :Prosser 2012

228 films of 82 # in 63 children (mean age, 4.8 years)

- Soft-tissue swelling in 59% at days 1-2 after fractures
- Periosteal reaction first seen at day 5 /present in 62% between 15 and 35 days after the fracture
- Soft callus was first seen at day 12 / prevalent in 41% between 22 and 35 days
- Hard callus and bridging began at day 19, increasing to 60% prevalence from 36 days onward
- Remodeling was observed only in fractures 45 days old or more

Halliday – timing of infants' fractures



161 images of 37 long bone fractures in 31 patients aged 0-44 months.
Assessed:

- soft-tissue swelling
- subperiosteal new bone formation (SPNBF)
- definition of fracture line
- presence or absence of callus
- whether callus was well or ill defined
- and the presence of endosteal callus

RESULTS:

“Agreement between observers was only moderate for all discriminators except SPNBF. SPNBF was invariably seen after 11 days but was uncommon before this time even in the very young. In one case SPNBF was seen at 4 days.”

Let's look at some fractures...



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Skull Fractures

When should I worry?

- non-parietal fractures
- multiple and complex fractures
- widely separated and depressed fractures
- fractures associated with significant intracranial injury
- single impact can result in a skull fracture that crosses a suture line.
- single impact may transmit forces such that two skull bones fracture at distant site

Ping-pong fractures and depressed skull fractures can occur as a result of blunt impact with a contoured object (assault and accidental)

Skull fractures in Abused children



What must I know?

Commonest skeletal injury leading to diagnosis of child abuse

Single linear parietal # = most common pattern

Very uncommon in uncomplicated short distance falls

Rib fractures

What must I know

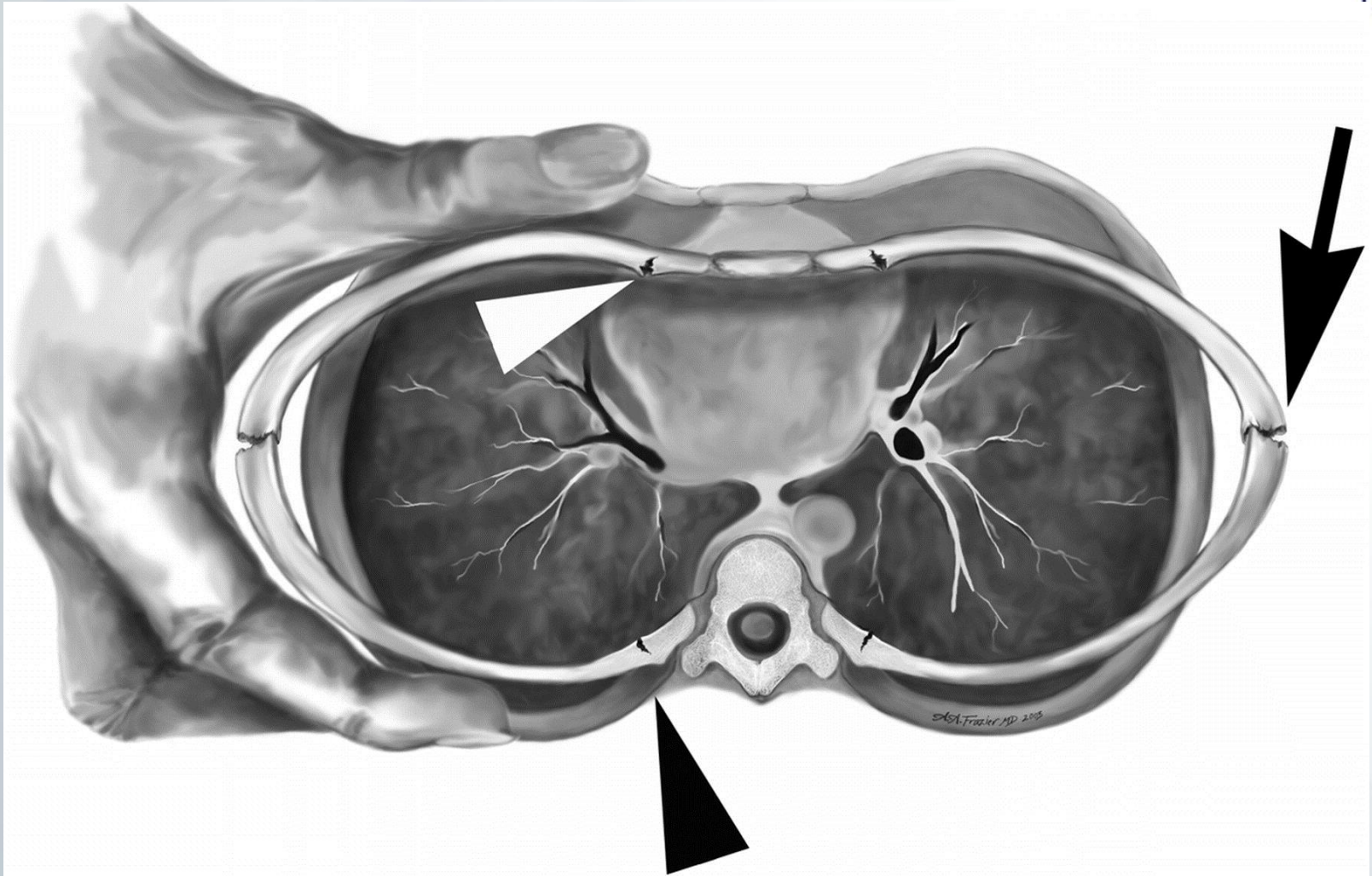
90% in abused children < 2 years

Can be missed on routine Xray

Posterior rib fractures = highly suggestive of abuse

Rib fractures are a rare complication of CPR in children (only 3 out of 923 children) all multiple and anterior, no posterior (WCPSRG)

Figure 8. Rib fracture mechanism in tight squeezing



Lonergan G J et al. Radiographics 2003;23:811-845

Compression & rib fractures

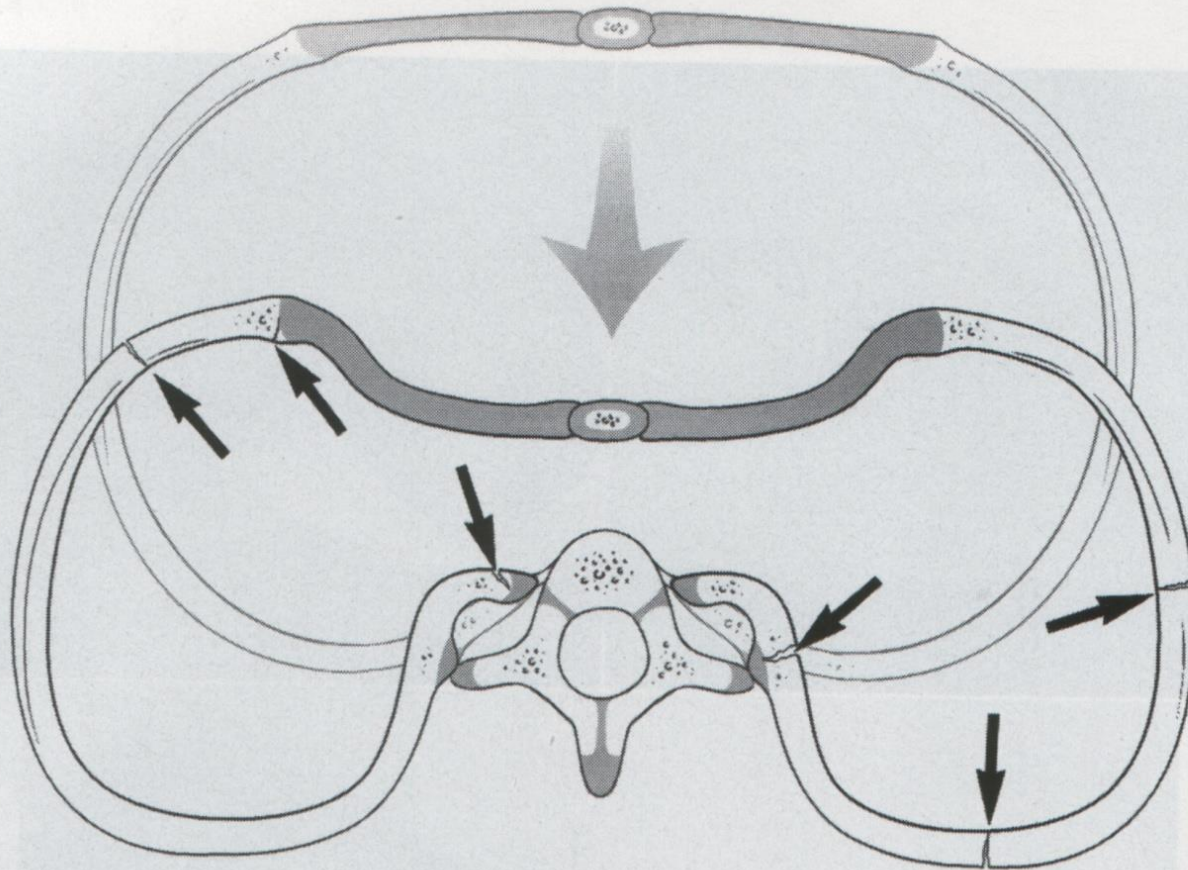


Fig. 5-7 Mechanism of injury, diagram. With anteroposterior compression of the chest, there is excessive leverage of the posterior ribs over the fulcrum of the transverse processes. This places tension along the inner aspects of the rib head and neck regions, resulting in fractures at these sites (*arrows*). This mechanism is also consistent with the morphologic patterns of injury occurring at other sites along the rib arcs and at the costochondral junctions (*arrows*).

Femoral fractures

What must I know?

Abusive femoral fractures occur predominantly in infants ([evidence level IIb](#))

Significantly more abusive femoral fractures arise in children who are not yet walking ([evidence level IIb](#))

Mid shaft fracture is the commonest fracture in abuse and non abuse (analysed for all age groups)([evidence level IIa](#))

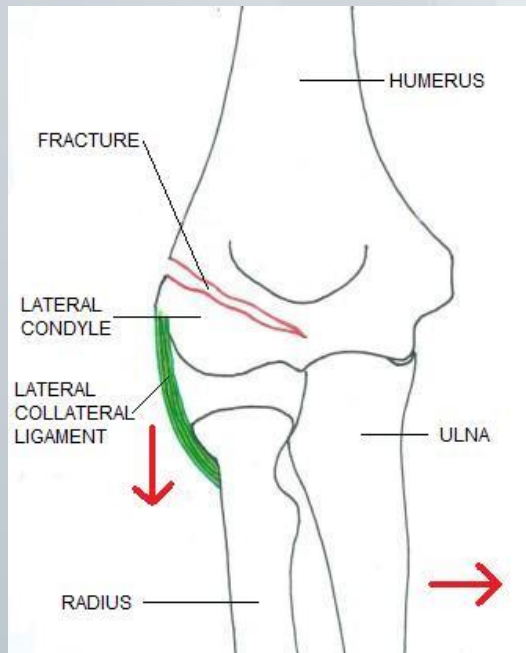
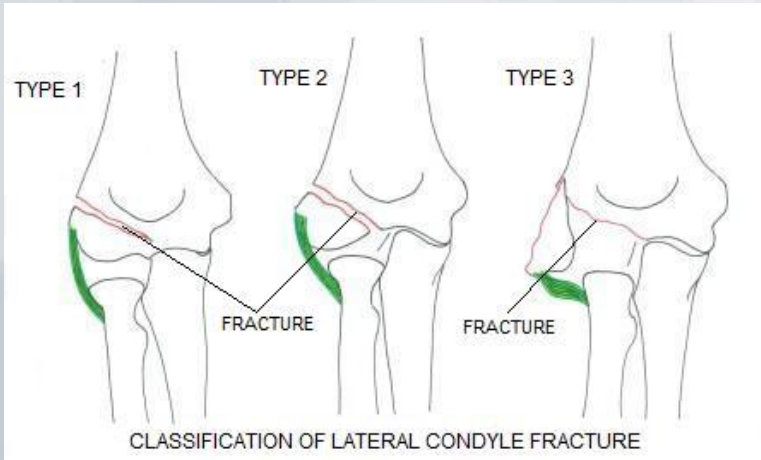
Under fifteen months of age a spiral fracture is the commonest abusive femoral fracture $p=0.05$ ([evidence level IIb](#))

Humeral fractures

What must I know?

The majority of accidental humeral fractures in children are supracondylar and the commonest abusive humeral fracture in children <5 years are spiral or oblique

Welsh Child Protection Systematic Review Group



Other bones

BEWARE

Uncommonly injured bones

- Scapula
- Spine
- pelvis

Vertebral, pelvic, hands, feet and sternal fractures occur in physical abuse, appropriate radiology is required for detection (grade C)

Vertebral fractures may be unstable, early identification is important (grade C)

Welsh Child Protection Systematic Review Group

BE Alert

Uncommon presentations of commonly injured bones

Unusual skull #

Ends of clavicle #

Unusual fracture
patterns when long
bones injured

Brittle bones

Transient - TBBD

A unique hypothesis (still has proponents) Transient copper defect? No scientific validity?

Deregistration of Dr. Colin Paterson from the Medical Practitioners Board of Scotland

Carol Jenny " *Although frequently offered in court cases as a cause of multiple infant fractures, there is no evidence that this condition actually exists.*"

Jenny C; Committee on Child Abuse and Neglect. Evaluating infants and young children with multiple fractures *Pediatrics*. 2006 Sep;118(3):1299-303

Medical causes

- Vit D deficiency
- Rickets (renal / other)
- OI
- Menkes / Copper abn
- Scurvey, Vit A toxicosis
- Hyper PTH (osteoclasts++)
- Hypocalcemia (exprem / metabolic)
- Osteopenia (disuse / other)
- Malignancy (leukemia, N'blastoma, histiocytosis)
- Infection – osteomyelitis
- Infantile cortical hyperostosis
- OTHERS

Vit D defic rickets in Aust children



APSU Jan 06-Jul 07

398 children ≤ 15 with vitamin D deficiency (55% male; median 6.3 years [range, 0.2-15 years]).

- 4.9/100 000/year.
- All had a low 25OHD level (median, 28 nmol/L [range, 5-50 nmol])
- All elevated alkaline phosphatase level (median, 407 IU/L [range, 229-5443 IU/L])
- 48 (12%) were hypocalcaemic.
- 95 had wrist x-rays, 67 (71%) had rachitic changes.

98% had dark or intermediate skin colour and 18% of girls were partially or completely veiled.

Most children were born in Africa (252; 63%) 75% were refugees.

Duration of exclusive breastfeeding was inversely related to serum vitamin D levels in children < 3 years of age. Empirical vitamin D treatment was given to 4% of children before diagnosis.

Schilling: Vit D & Fractures under 2s



118 subjects, 8% had deficient vit D (20 ng/mL), 31% (20–30 ng/mL) and 61% were sufficient (30 ng/mL)

Lower vit D associated with higher incidences of hypocalcemia *and elevated alkaline phosphatase* but not hypophosphatemia

The majority of children sustained

- Accidental fractures (60%)
- Nonaccidental 31%
- Indeterminate 9%

There was no association between vitamin D levels and any of the following outcomes: child abuse diagnosis, *multiple fractures, rib fractures or metaphyseal fractures*

CONCLUSIONS: Vitamin D insufficiency was common in young children with fractures but was not more common than in previously studied healthy children. Vitamin D insufficiency was not associated with multiple fractures or diagnosis of child abuse.

Nonaccidental trauma remains the most common cause of multiple fractures in young children

How do I assess bone strength?

VERY TRICKY+++

Metabolic tests

- Calcium
- Phosphate
- LFT (proteins and AlkPhos)
- Vit D
- U&E, Creat
- FBE
- Other (Mg, PTH, UMS...
- Tests for OI – collagen and genetic

Radiological tests

Xray osteopenia = very late sign?

Xray signs of other disease (rickets, scurvy, bone dysplasia, OI)

Bone densitometry not clinically useful (forensically) but increasing use in AN, other medical diseases in children

Metabolic and genetic bone disease

ALWAYS test basic bone metabolism

- Calcium
- Phosphate
- Alkphos
- Proteins
- U&E Creat
- Vit D.

MAYBE

- Mg
- PTH
- UMS
- Cu
- VDRL....
- COL1A..... (fibroblast culture)
- Genetic tests....
- BMA, Biopsy? Other.....

International OI Nomenclature 2010



Syndrome Names	Equivalent Numerical Type	Sub Types
Classic Non-deforming OI with Blue Sclerae	I	2
Common Variable OI with Normal Sclerae	IV	2
OI with Calcification in Interosseous Membranes	V	1
Progressively Deforming OI with Normal Sclerae	III	9
Perinatally lethal OI	II	8
Bruck Syndromes		2
Cole-Carpenter syndrome		1

Don't forget to examine teeth!



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