



Current Controversies in Abuse

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Abusive Head Trauma

Biomechanics

The

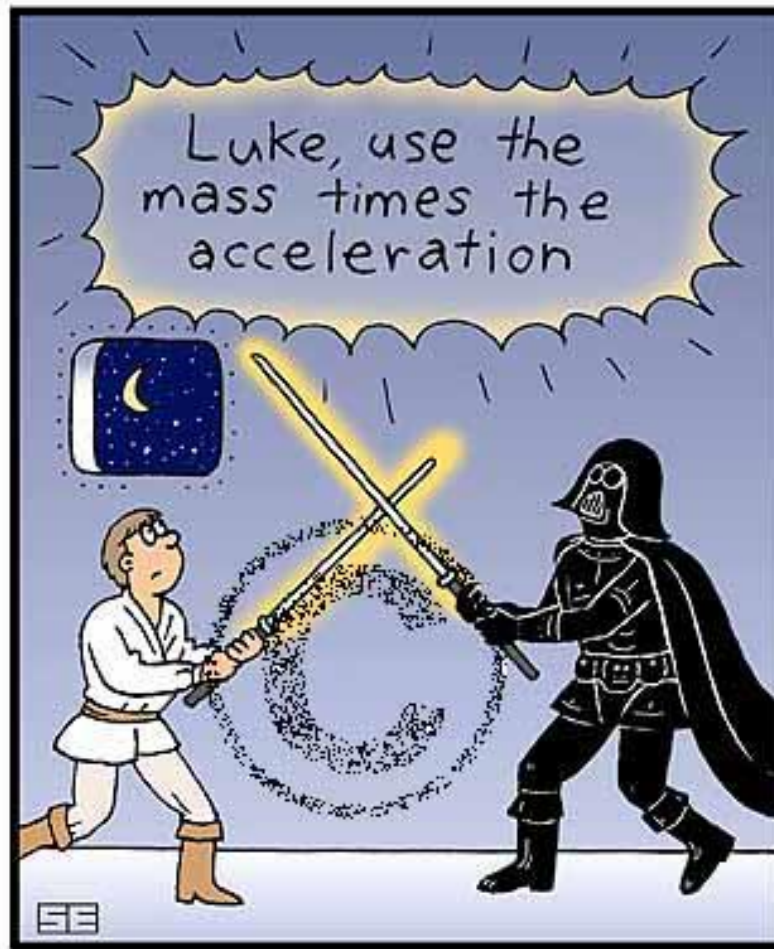
BORDERLINE

By Gabe Martin



As the years drug on, Skywalker began to find Obi-Wan increasingly annoying.

<http://www.cts.com/~borderln/>



$$f = ma$$

Literature Supports

- Primary brain injury from mechanical forces
- Contact forces cause focal injuries
- Translational (non-contact) cause focal injuries
- Rotation can cause diffuse brain injury

Focal Injuries

- Impact – scalp, subgaleal tissue
- Epidural hematoma
- Subdural/subarachnoid hematoma
- Cerebral contusions/lacerations
 - Usually on crest of gyri – gray matter



Diffuse Injury

- Axonal injury
 - acceleration/deceleration leads to pressure gradients/mechanical strains
 - Varies from mild to severe
- Vascular injury
- Ischemic brain injury
- Edema

Neuropathology

- 53 autopsies AHT
- Macroscopic:
 - 27 with extra cranial injury significant
 - 45 with evidence impact to head
 - 43 with SDH – in 34 amt bld trivial
 - 5 with cerebral contusions
- Microscopic:
 - 38/53 had eyes examined:
 - 27 with RH
 - Many with hypoxic-ischemic changes

Neuropathology

- Of 37 children < 9 months with AHT compared with 14 controls:
 - Significant apnea reported in 75%
 - Global hypoxic damage most common
 - Diffuse traumatic axonal injury in 2
 - Focal axonal damage to brainstem/spinal nerve roots in 11 cases and no controls
 - Craniocerebral junction vulnerable – stretch injury from hyperextension/flexion
 - This causes apnea, leading to hypoxia



Abusive Head Trauma

Rebleeds

Rebleed

- Controversies:
 - What causes enlarged subarachnoid spaces?
 - Do large subarachnoid spaces predispose to SDH?
 - Under what circumstances do SDHs rebleed?
 - What is the clinical presentation of subdural rebleeding?

Rebleed

- Enlarged subarachnoid spaces:
 - Benign
 - May be congenital
 - Immature arachnoid villi cause transient communicating or external hydrocephalus
 - SAH
 - Trauma common cause
 - Blood in SA space causes arachnoiditis – impedes absorption CSF in villi
 - Leads to communicating hydrocephalus that appears as big SA spaces

Rebleed

- Enlarged subarachnoid spaces:
 - Associated with overlying acute SDH – impedes flow of CSF into area under bleed
 - Cerebral atrophy
 - Can be difficult to differentiate from communicating hydrocephalus
 - Measure head growth to help determine



Rebleed

- Does a large SA space cause SDH?
 - Benign prominence SA space common
 - Long term studies of infants with benign expansion do not show increased SDH
 - SDH can cause expansion SA space
 - Therefore, answer is probably not
 - Caveat – if large SA space due to atrophy minor trauma can cause SDH but won't have sxs diffuse brain injury

Rebleed

- What causes SDHs?
 - Contact and noncontact forces
 - Subdural hygromas



Rebleeds

- Most chronic SDHs come from SD hygromas:
 - Accumulation CSF in SD space without membrane
 - As SDH resolves, posttraumatic space persists within intradural membrane
 - Effusion created as result of this persistent space
 - Begin as space filling lesion, not mass lesion so usually asymptomatic
 - May form neomembranes over time with neovascularization which can bleed into SD effusion – can occur with little or no trauma
 - Repeated bleeding can turn hygroma into chronic SDH

Rebleed

- What causes SDHs to rebleed?
 - Hygromas can bleed spontaneously
 - Minor trauma
 - Inflicted trauma



Rebleed

- What is the clinical presentation of subdural rebleeding?
 - Small hemorrhages from neomembrane of hygroma may have no sx
 - Acceleration injuries may cause diffuse brain injury

Consequences Rebleeding

- Minimal if bleeding:
 - Microscopic
 - Spontaneous
 - Younger child, elastic skull, open AF, unfused sutures
 - Into hygroma
 - Due to re-injury without diffuse brain injury
- Serious if bleeding:
 - Induced by re-injury
 - Older child, less elastic skull, closed AF, fused sutures
 - Into chronic SDH acting with mass effect
 - Re-injury with primary brain injury



Abusive Head Trauma

Timing and Lucid Intervals

Timing

- Retrospective chart review
- 95 accidental deaths
- Average age – 8.5 years
- 2/95 with lucid interval:
 - 1 with EDH
 - 1 with abdominal exsanguination

Timing

- CPSC data on head injury and playgrounds
- 18 accidental deaths from short falls – many from swings
- Average age – 5.2 years
- 6/18 unwitnessed falls
- 7/18 had no autopsy
- 12/18 with lucid interval based on initial crying
- Most died from mass lesion effects

Timing

- Retrospective review 171 AHT cases
 - 81 cases perpetrator confession
 - Mean age 3.5 months
- Initial symptoms reported by perpetrators:
 - Limpness
 - Seizures
 - Vomiting
 - Lethargy
 - Apnea

Timing

- Mechanism known in 69/81 cases:
 - 20/69 impact only altho some were thrown
 - 32/69 shaking only
 - 17/69 shaking and impact
- Timing ascertained in 57/81:
 - 52/57 immediate sx's per perp
 - 5/57 unclear but in 3 cases child not observed for up to 6 hours after injury

Timing

- Data from Penn Trauma System:
 - Children < 48 months with fatal head injury
- 314 children:
 - 121 (37%) inflicted
 - 40 falls (13%)
 - 153 (49%) MVA
- Defined lucid as GCS 13-15

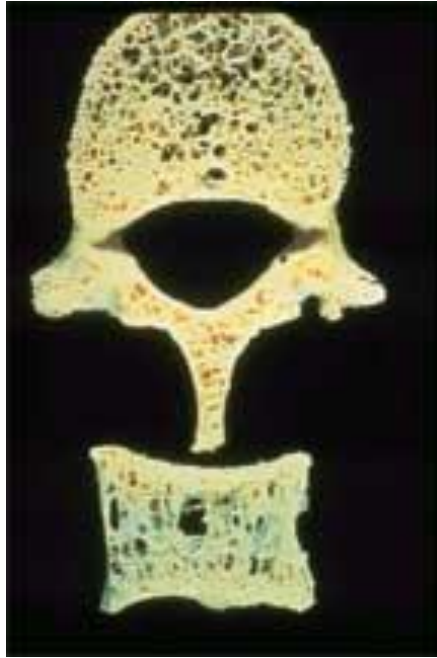
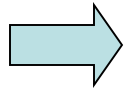
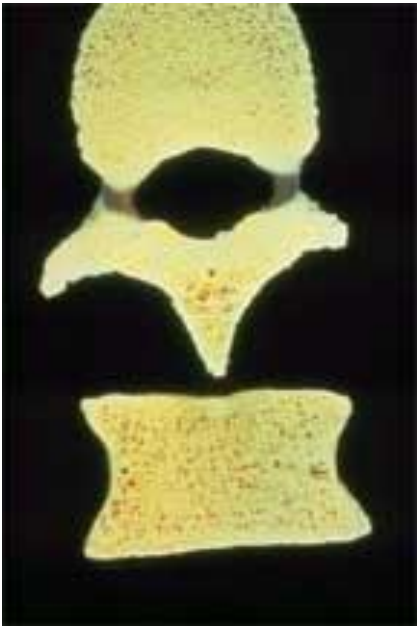
Timing

- Results:
 - 1.9% (6/314) had GCS 13-15
 - 5/6 were < 24 months
 - 3/121 children with AHT had GCS 13-15 – all < 24 months
 - Children < 2 years with AHT > 10 times more likely to have GCS > 7 than those in MVA
 - Caution that GCS 13-15 does not mean children normal or asymptomatic as doesn't address sx's head injury such as vomiting, irritability, etc.



Bone Mineral Density

Pitfalls in pediatric measurements



Osteoporosis

- Disease characterized by low bone mass and micro-architectural deterioration of bone tissue, leading to enhanced bone fragility and a consequent increase in fracture risk



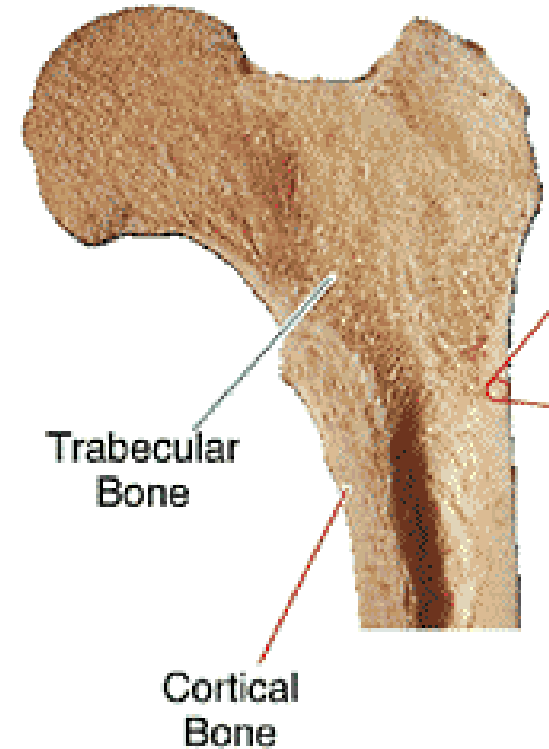
Osteoporosis vs Osteomalacia

- Osteoporosis
 - bones lose an excessive amount of their protein and mineral content, particularly calcium.
- Osteomalacia (rickets)
 - is a type of metabolic bone disease in which there is a lack of available calcium or phosphorus (or both) for mineralization of newly formed osteoid

Bone

- Trabecular bone
 - Metabolically active component
 - Accounts for 20% of the total body bone mass
 - Calcification is primarily responsive to cytokines
- Cortical bone
 - Responsible for the mechanical properties of bone
 - Approximately 80% of the total body bone mass
 - Calcification is controlled by PTH and Vitamin D

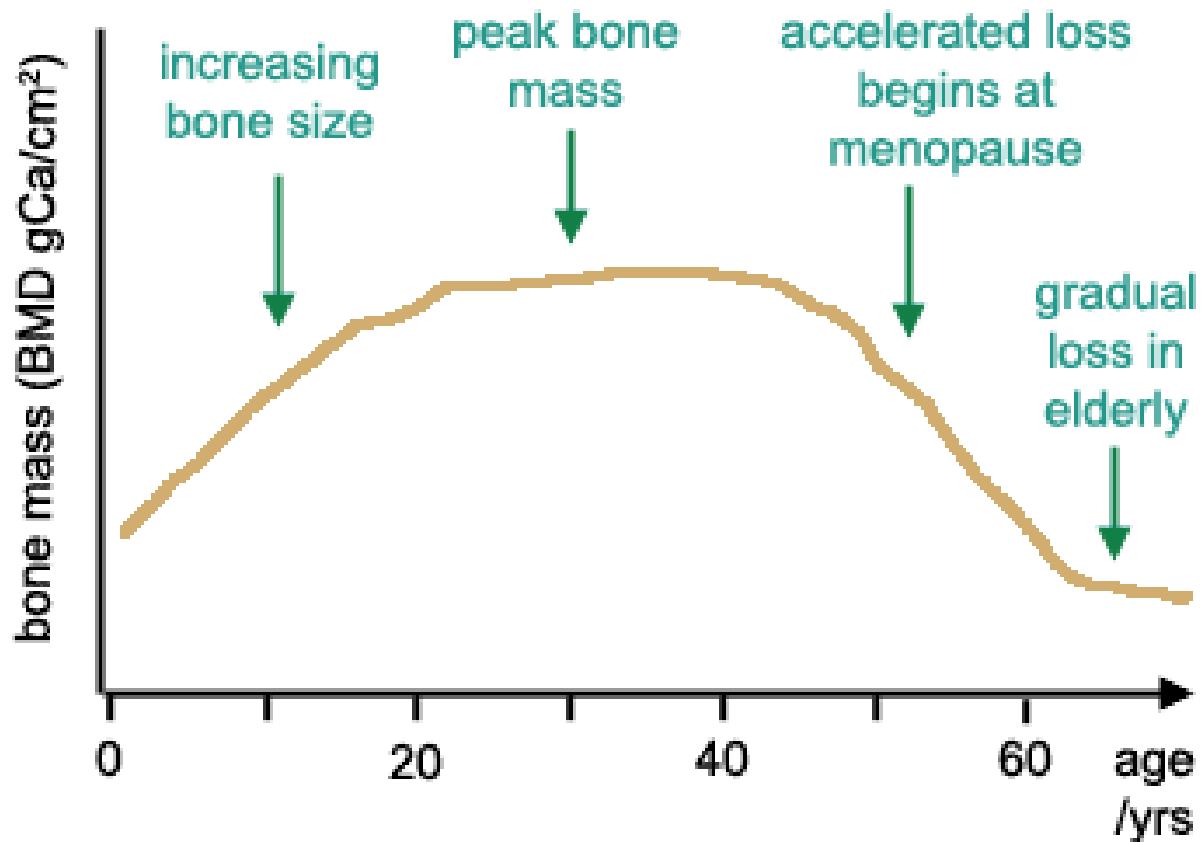
Femur



Bone Mineral Density Measurement

- DXA
 - Dual-energy x-ray absorptiometry
- AXIAL QCT
 - Quantitative computed tomography
- Peripheral QCT
- QUS
 - Quantitative ultrasound
- MRI
 - Magnetic resonance imaging

Bone Mineral Density

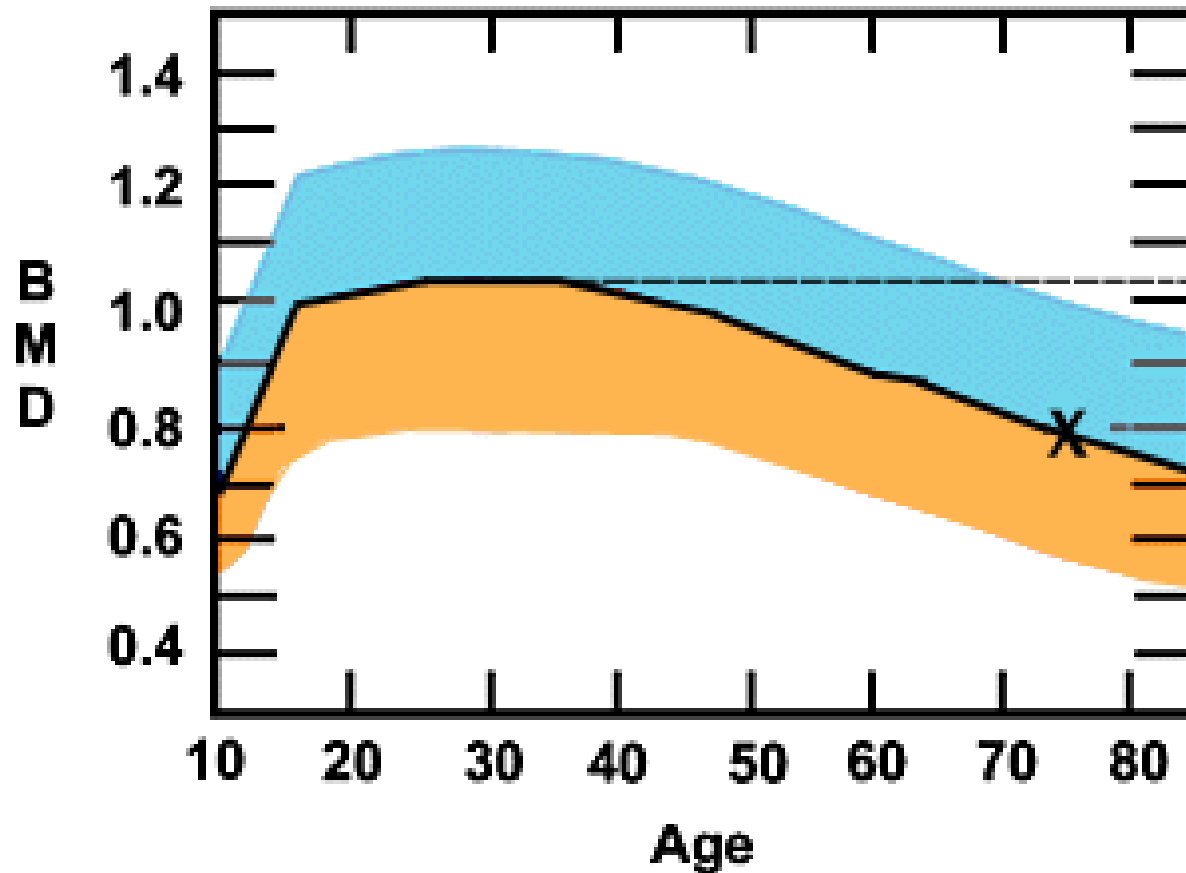


DXA

- Measurement of transmission of x-rays through the area of interest at low and high energies
- Low energy is attenuated by soft tissue
- High energy is attenuated by soft tissue and bone
- Difference = bone

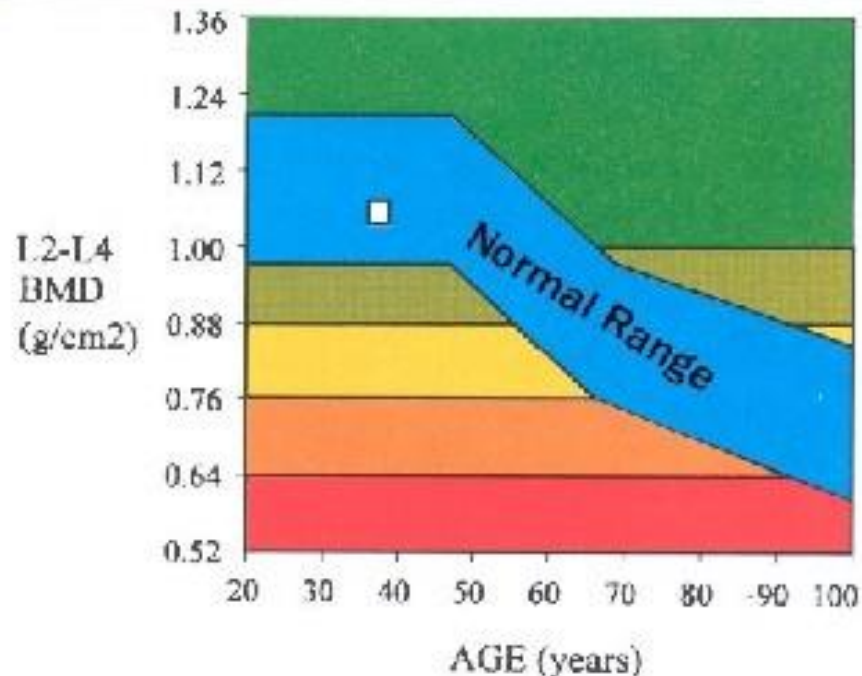


Bone Mineral Density



DXA scan

- Z score
 - Patient's value compared to the standard deviation for patient's age
- T score
 - Patient's value compared to the standard deviation for a young adult



Region	BMD ¹ (g/cm ²)	Young-Adult ²		Age-Matched ³	
		%	T	%	Z
L1	0.930	88	-1.1	91	-0.7
L2	0.985	88	-1.1	91	-0.8
L3	1.117	100	0.0	104	+0.3
L4	1.045	93	-0.6	97	-0.3
L2-L4	1.052	94	-0.6	98	-0.2

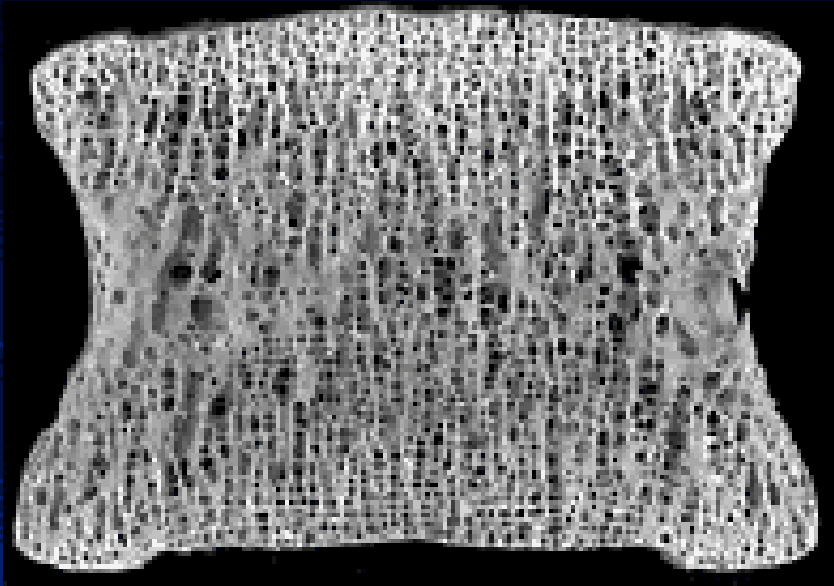
1-Statistically 68% of repeat scans fall within 1SD (+ 0.010) for L2-L4.

2-China AP Spine Female Reference Population, Ages 20-40.

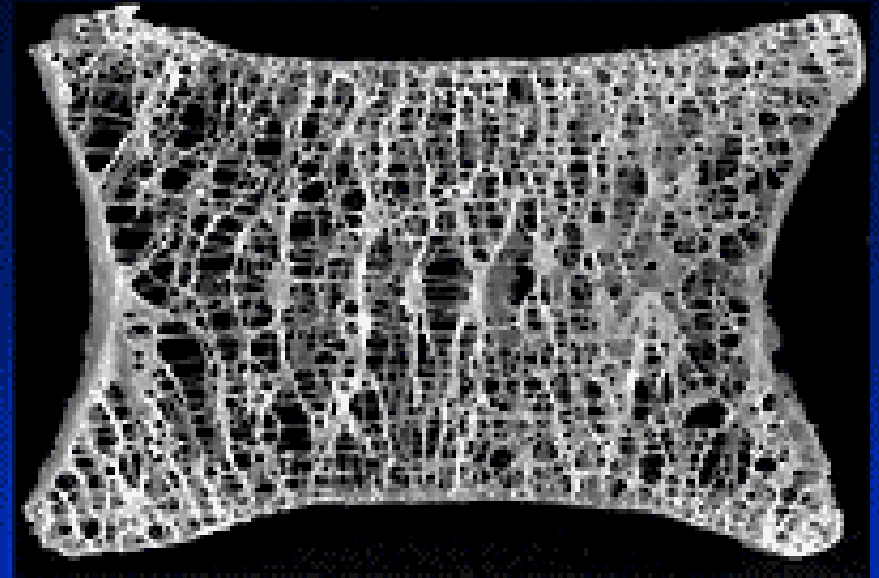
3-Matched for age, weight (females 25-100kg) and ethnicity

The area in blue is the range of normal bone density:

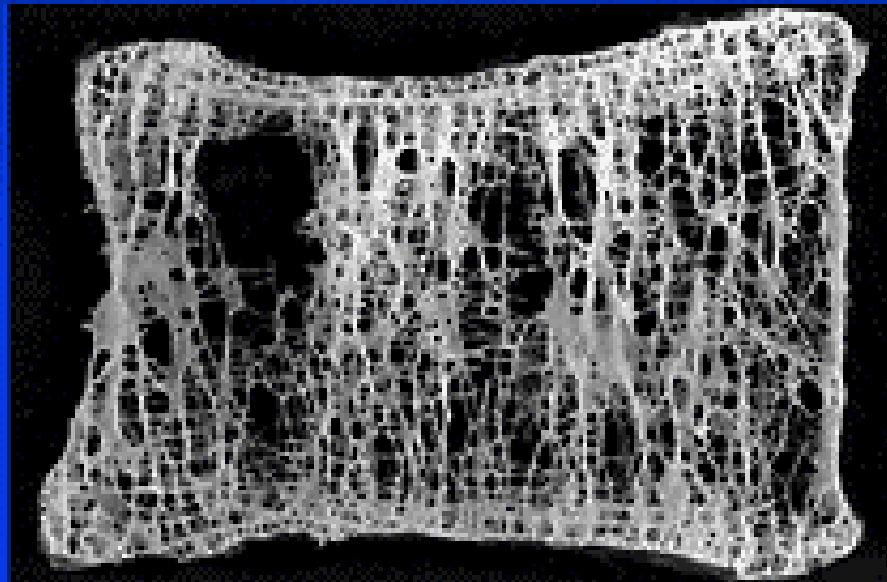
Normal



Moderate Osteoporosis

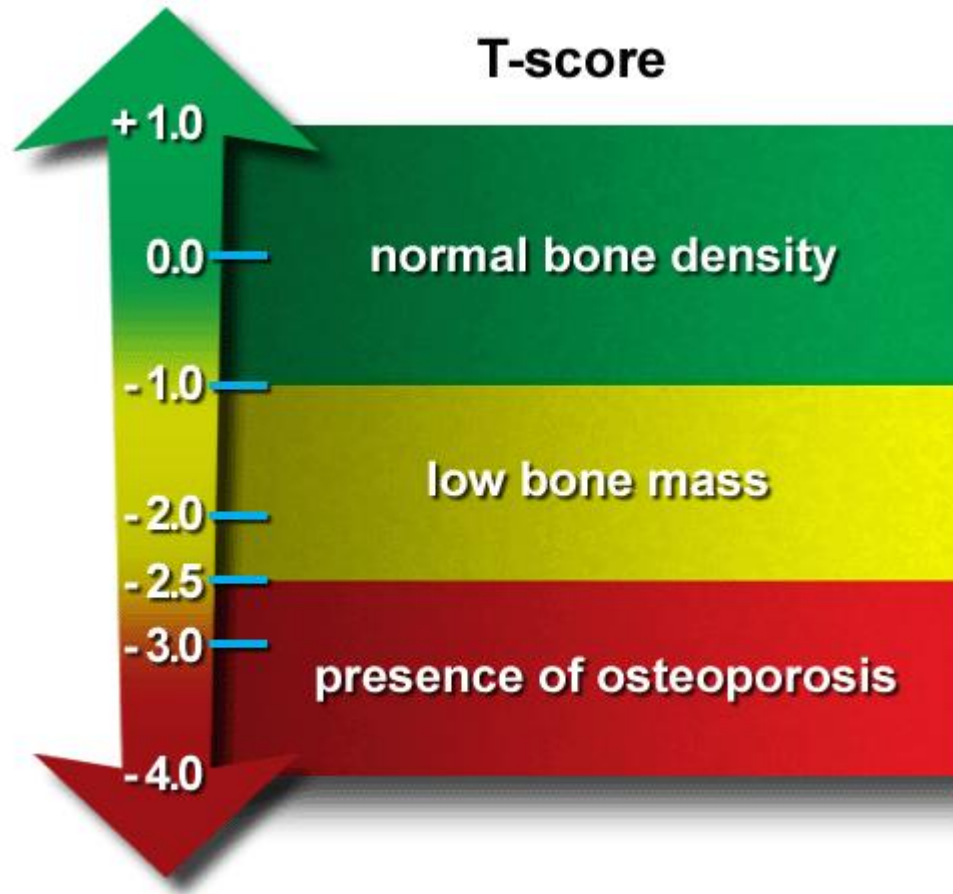


Severe Osteoporosis



Courtesy of Dr. A. Boyde.

T-Score



WHO Classification of Osteoporosis

- Normal
 - T score BMC or BMD not less than -1
- Osteopenia
 - T score BMC or BMD -1 to -2.5
- Osteoporosis
 - T score BMC or BMD lower than – 2.5
- Severe or established osteoporosis
 - T score BMC or BMD lower than – 2.5 and one or more fragility fractures

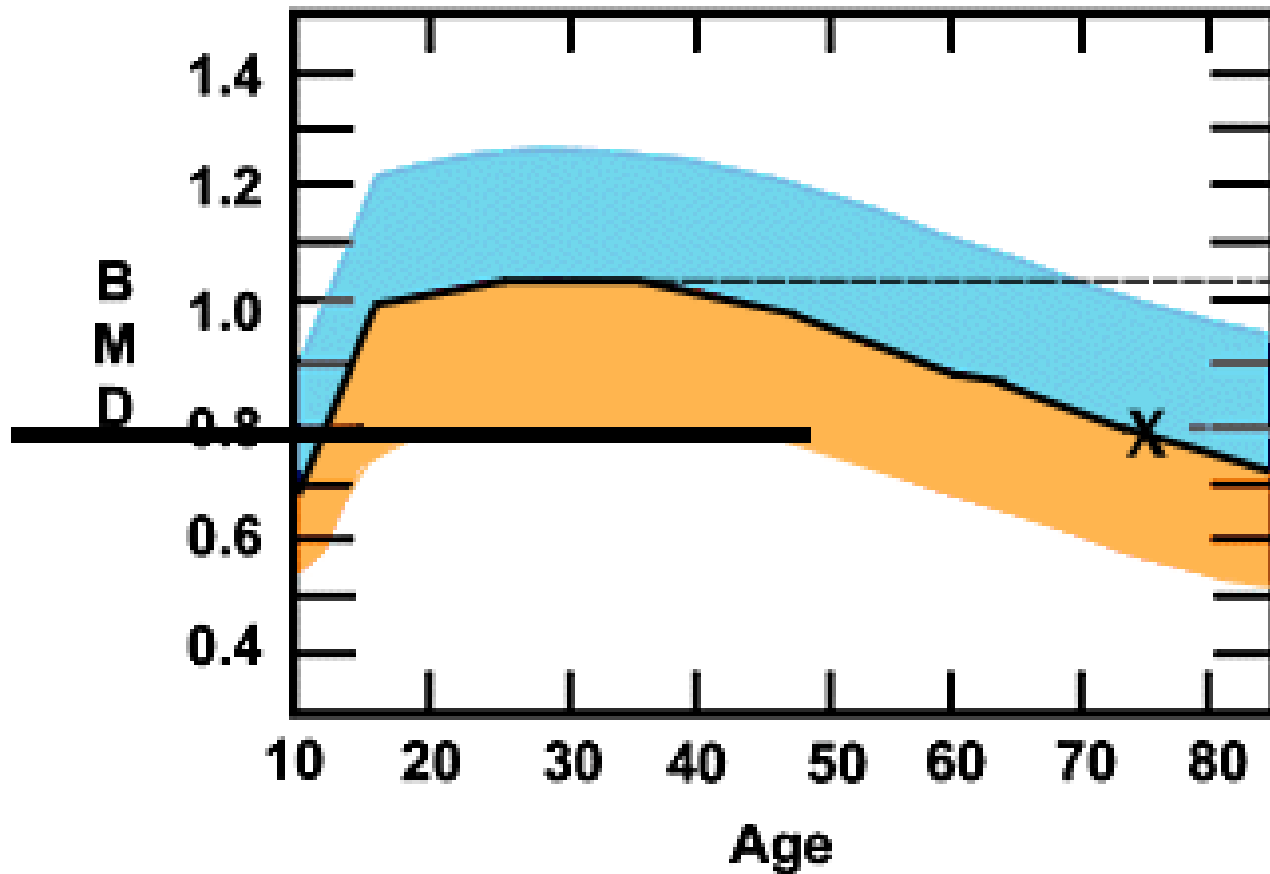
WHO Classification for Osteoporosis

- Sensitivity of predicting vertebral fractures in adults is only 65%
- No longitudinal epidemiologic studies in children and adolescents to determine the predictive value

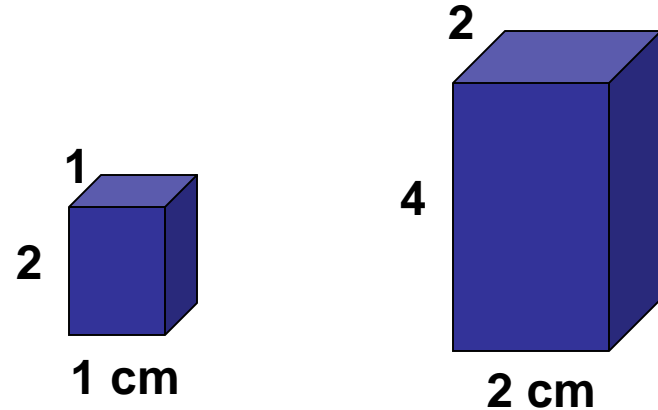
DXA Scan

- Not a measure of the true bone mineral density BMD
- Measure the bone mineral content BMC and the area of bone
- *Area* BMD is calculated by dividing the BMC the bone area gm/cm^2

Bone Mineral Density



Areal BMD Measurements Are Influenced by Bone Size



Volumetric bone density (g/cm³)	1.0	1.0
Projected area (cm²)	2.0	8.0
Volume (cm³)	2.0	16.0
BMC (g)	2.0	16.0
BMD (g/cm²)	1.0	2.0

The International Society for Clinical Densitometry Guidelines

Fracture Prediction and Definition of Osteoporosis

- The diagnosis of osteoporosis in children and adolescents should NOT be made on the basis of densitometric criteria alone.
- The diagnosis of osteoporosis requires the presence of both a clinically significant fracture history and low bone mineral content or bone mineral density.



The International Society for Clinical Densitometry Guidelines

- A clinically significant fracture history is one or more of the following:
 - Long bone fracture of the lower extremities
 - Vertebral compression fracture
 - Two or more long-bone fractures of the upper extremities
- Low bone mineral content or bone mineral density is defined as a BMC or areal BMD Z-score that is less than or equal to -2.0, adjusted for age, gender and body size, as appropriate.

The International Society for Clinical Densitometry Guidelines

DXA Interpretation and Reporting in Children and Adolescents

- ❑ The hip (including total hip and proximal femur) is not a reliable site for measurement in growing children due to significant variability in skeletal development and lack of reproducible Region Of Interest.
- ❑ In children with linear growth or maturational delay, spine and TBLH BMC and areal BMD results should be adjusted for absolute height or height age, or compared to pediatric reference data that provide age-, gender-, and height-specific Z-scores.

DXA Interpretation and Reporting in Children and Adolescents

- ❑ Baseline DXA reports should contain the following information:
 - ❖ DXA manufacturer, model, and software version
 - ❖ Referring physician
 - ❖ Patient age, gender, race/ethnicity, weight, and height
 - ❖ Relevant medical history including previous fractures
 - ❖ Indication for study

DXA Interpretation and Reporting in Children and Adolescents

- Indications for follow-up scan
- Comparability of studies
- Interval changes in height and weight
- BMC and areal BMD Z-scores adjusted or unadjusted for height or other adjustments
- Percent change in BMC and areal BMD and interval change in Z-scores
- Recommendations for the necessity and timing of the next BMD study are optional

DXA Interpretation and Reporting in Children and Adolescents

- ❑ **The term “osteoporosis” should not appear in pediatric DXA reports without knowledge of clinically significant fracture history.**
- ❑ **“Low bone mineral content or bone mineral density for chronologic age” is the preferred term when BMC or BMD Z-scores are less than or equal to -2.0.**

DXA Measures Bone in Two Dimensions Whereas pQCT Provides 3 Dimensions

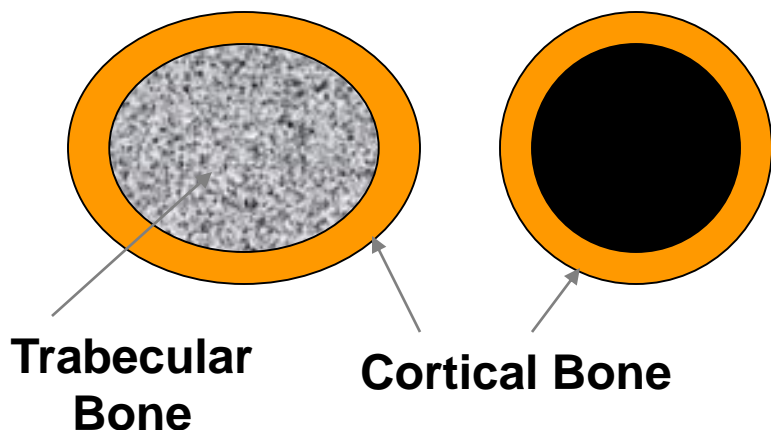
DXA (2-dimensional projection):

Bone Mineral Content
Bone Area
aBMD

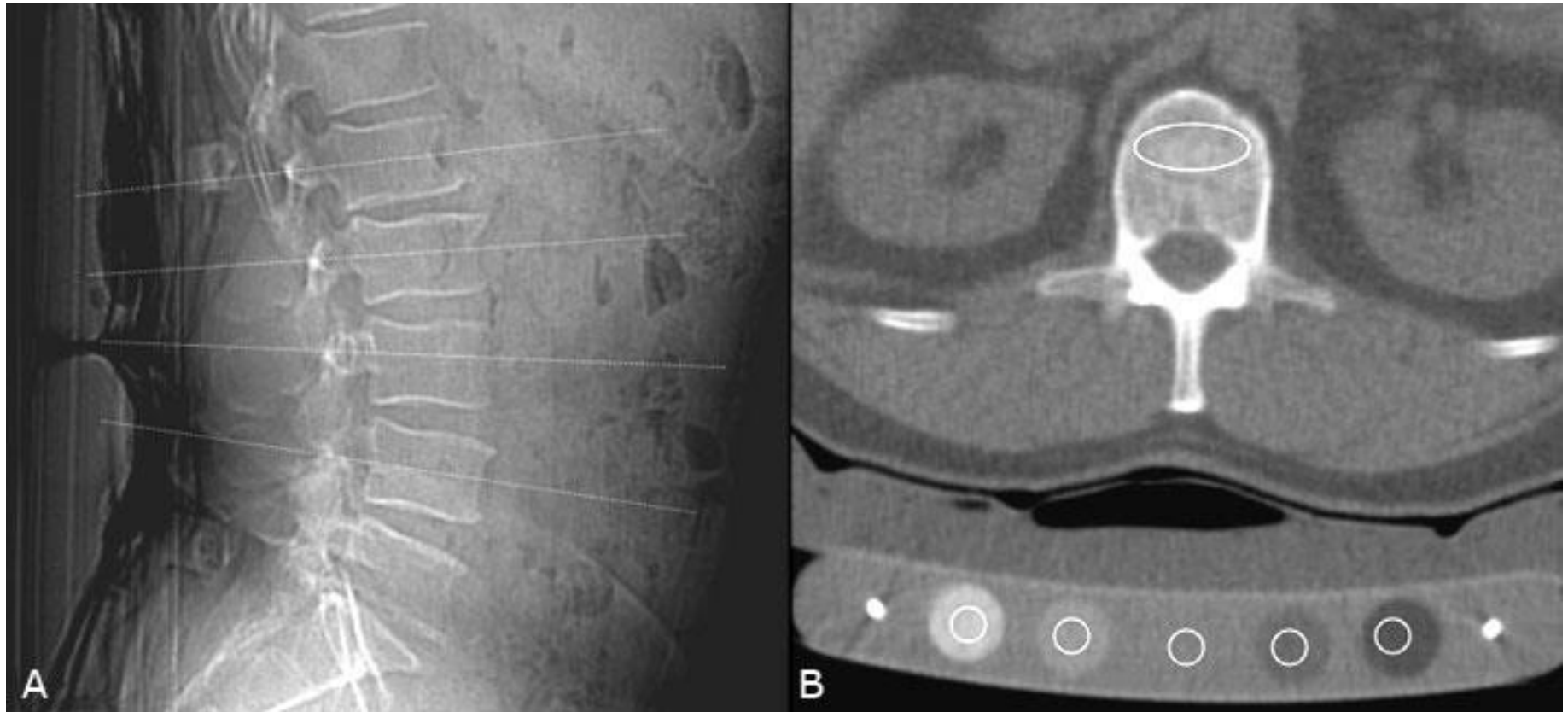


pQCT (3-dimensional slices):

Total Bone Area
Cortical Bone Area
Periosteal & Endosteal Circumferences
Cortical Thickness
Cortical vBMD
Trabecular vBMD



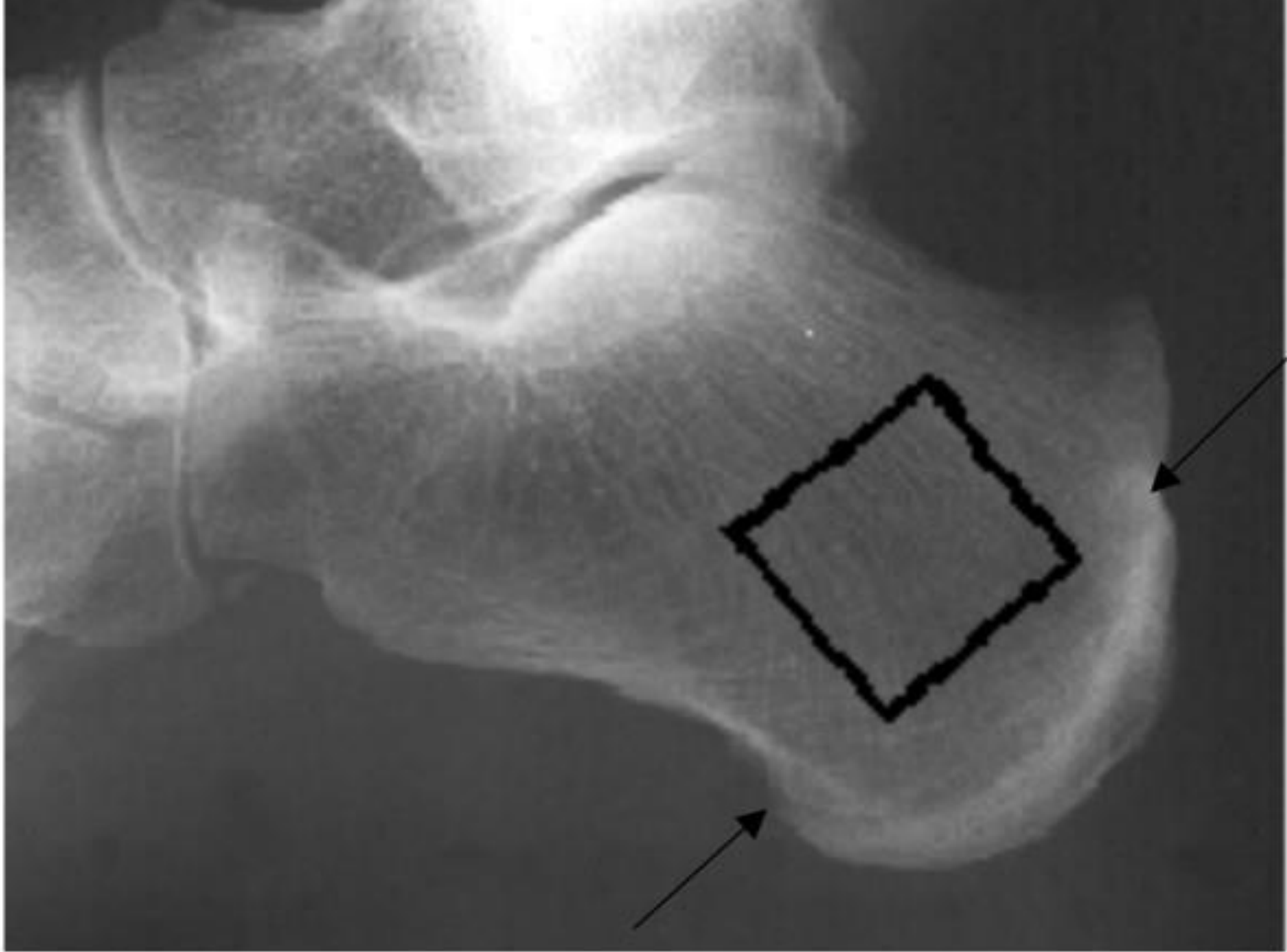
QCT



Quantitative Ultrasound

- Based on the attenuation of the ultrasound beam as it passes through the area of interest
- Measurement is related to BMD and parameters of bone quality and strength





Quantitative Ultrasound Pediatric Limitations

- Foot wells are designed for adult size feet
- Lack of normative pediatric age values
- Calcaneus is primarily trabecular bone



Comparison of Bone Densitometry Techniques

Technique	Site	Radiation dose* (μSv)	Precision (CV %)
DXA	Lumbar spine	0.4-4	<1
	Total body	0.02-5	1-2
	Proximal Femur	.15-5.4	0.8-1.5
AXIAL CT	Spine	55	0.8-1.5
QUS	Calcaneus	None	1.6-5

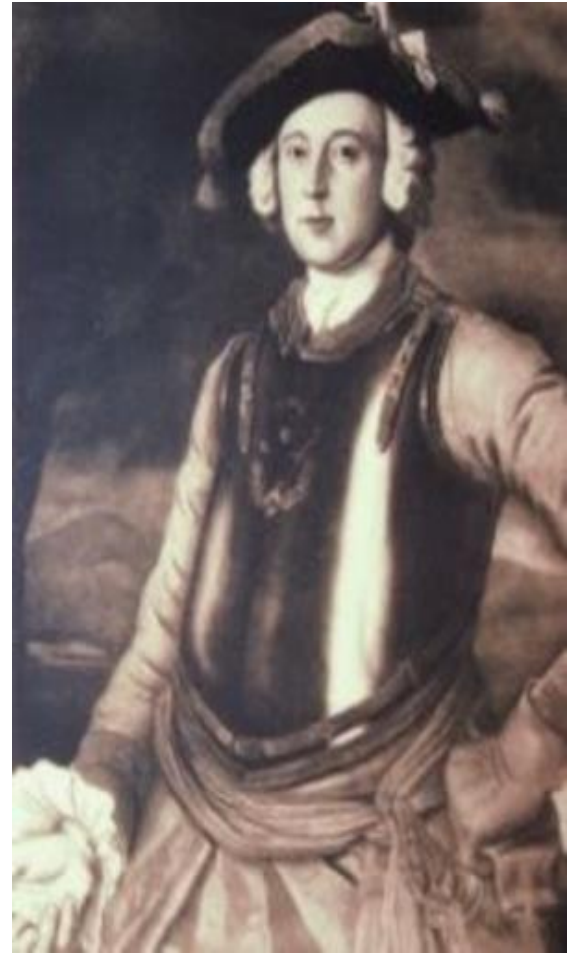
* Chest Radiograph 12-20 μSv , Plain lumbar spine 700 μSv



Munchausen Syndrome by Proxy

Who is Munchhausen anyway?

- Born in Germany 1720
- Enjoyed telling stories of travel/adventure
- Rudolf Erik Raspe wrote “Baron von Munchhausen’s Narrative of his Marvellous Travels and Campaigns in Russia”
- Honorable man who enjoyed telling stories for his friends



Who is Munchhausen anyway?

- Asher in Lancet 1951:
 - "Here is described a common syndrome which most doctors have seen, but about which little has been written"
 - Published description and case histories of 3 patients who fabricated illnesses
 - Moved from hospital to hospital seeking admission
- Chose to name Munchausen Syndrome as:
 - "like the famous Baron von Munchausen, the persons affected have always travelled widely, and their stories, like those attributed to him, are both dramatic and untruthful"

Why Munchausen by Proxy?

- The Hinterland of child abuse
- Case reports of 2 children:
 - Induced UTIs
 - Salt poisoning
- Mothers gave deliberate, false histories and altered specimens
- Mothers were pleasant, cooperative, appreciative
- Fabricated stories shared features of Munchausen syndrome but were by proxy to the children

Meadow, R, The Lancet, 1977

Why Munchausen by Proxy?

- Cases are a reminder that at times physicians must treat history and laboratory specimens with skepticism
- “This paper is dedicated to the many caring and conscientious doctors who tried to help these families, and who, although deceived, will rightly continue to believe what most parents say about their children, most of the time.”



Definition

Or does motivation really matter?

Definition

- Pediatric condition falsification:
 - Abusive act
 - Adult falsifies symptoms in a child causing others to believe the child is ill
- Factitious disorder by proxy:
 - Motivation
 - Person who intentionally falsifies history, signs, symptoms in another in order to meet his/her own needs

Definition

- MSBP:
 - Collection of acts
 - Intent is not observable
- Focus should be on identifying and minimizing harm to child regardless of motivation of the caregiver
- Harm caused by a caregiver who exaggerates, fabricates or induces symptoms may be called ***child abuse in a medical setting***

Definition

- Form of child abuse
- Involves repeated fabrication of illness in a child by an adult caretaker, usually the mother
- Illness may be simulated or induced
- Caretaker denies knowledge of the etiology of the problem resulting in the child being subjected to unnecessary medical tests, procedures, surgery

Definition

- Misinterpret/exaggerate illness
- Fabrication of symptoms
- Inducement of symptoms
- Common:
 - Caregivers' insistence something is wrong
 - Absence of findings to support diagnosis
 - Harm to child



Diagnosis

*How do I know it's not something
really rare?*

Diagnosis

- Caregiver fabricates impression of illness in a child sometimes deliberately causing harm to the child
- Persistent fabrication by one individual of illness in another
- Diagnosis depends on:
 - Harm or potential harm
 - Caregiver causing it to happen

Diagnosis

- Diagnostic criteria don't capture all about a disorder:
 - Smallest set of findings that must be present to make a diagnosis
 - Each criterion must be present in order to make a diagnosis
 - Each must be pivotal – presence is required for and absence precludes the diagnosis – medical training
 - Each criterion must be credibly observable - intent
 - Observations must be replicable
 - Don't tell everything there is to know about the disorder

Diagnosis

- Diagnosis by inclusion:
 - Supported by incontrovertible evidence of commission
 - Example: Parent caught smothering child on video
- Diagnosis by exclusion:
 - All other possible explanations for the condition have been considered and excluded
 - Example: Parent claims child has OI but no fractures on imaging studies

Diagnosis

- Child abuse is a diagnosis that describes what is happening to a child
- Diagnosis of MSBP is difficult:
 - Signs and symptoms undetectable or inconsistent
 - Action must be determined by harm or potential harm to child

Diagnosis

- When to suspect:
 - Persistent/recurrent symptoms for which cause cannot be found.
 - Discrepancies between history and physical findings.
 - Difference between reported history and what is seen
 - Problem does not respond to treatment as expected.
 - Problem appears to originate only in association with suspected perpetrator's presence.
 - Symptoms and signs abate when the child is not with the caretaker.
 - Problem resumes after caregiver told child has recovered

Diagnosis

- If suspect falsification pursue the diagnosis
- Gather information from all involved
- Falsification of a medical condition is a medical diagnosis
- Covert surveillance:
 - Capture parent's misbehavior
 - Fail to confirm reported symptoms
 - Exonerate a suspected caregiver

Treatment

- Current safety
- Future safety
- Occur in least restrictive setting
- Involve the multidisciplinary team



Interventions

- Individual and/or family therapy with primary care physician as “gatekeeper” for medical care utilization
- Monitor medical care usage by involving people or institutions outside the medical practice to alert the physician gatekeeper about health care issues – e.g. schools, insurance company

Interventions

- Admit the child where actual signs/symptoms can be monitored
- Involve CPS to obtain dependency, to control overuse of medical resources/gradually reintroduce child to home while monitoring child's safety
- Place child permanently
- Prosecute the caregiver