3rd Dr. Sudhakar Shetty Memorial

31st Mangalore Orthopaedic Course

15th & 16 June 2013

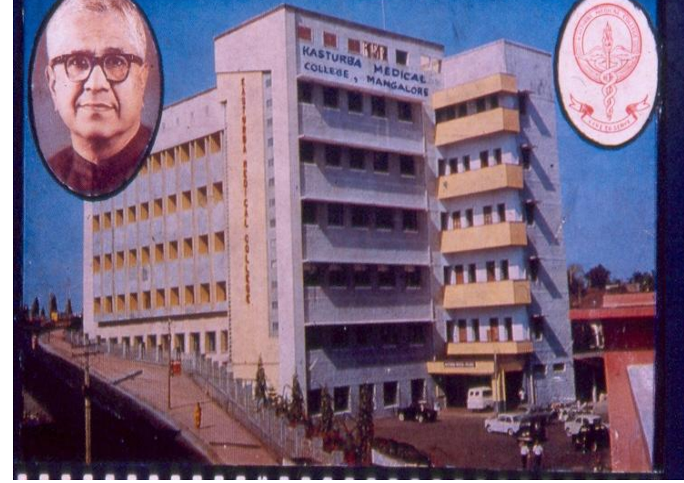
Organised by

CANARA ORTHOPAEDIC SOCIETY (Regd.)

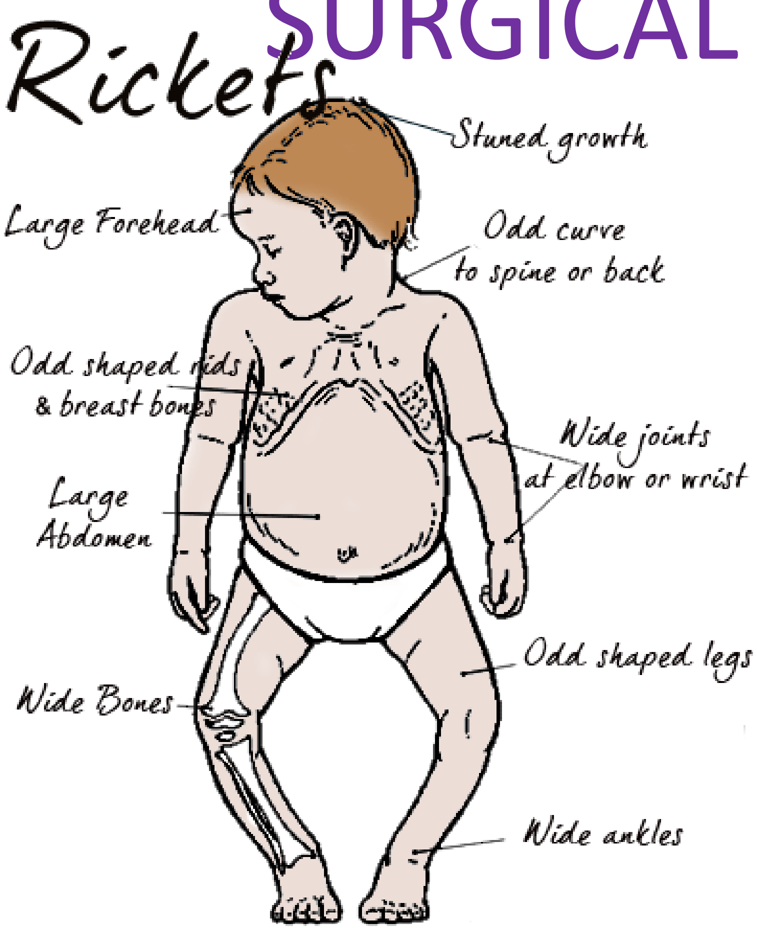
Under the aegis of

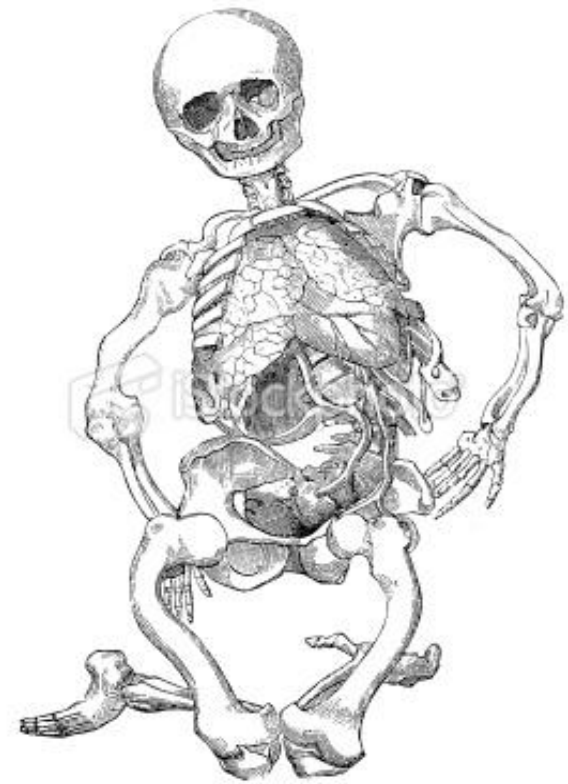
KARNATAKA ORTHOPAEDIC ASSOCIATION & ORTHOPAEDIC ASSOCIATION OF SOUTH INDIAN STATES

Welcome



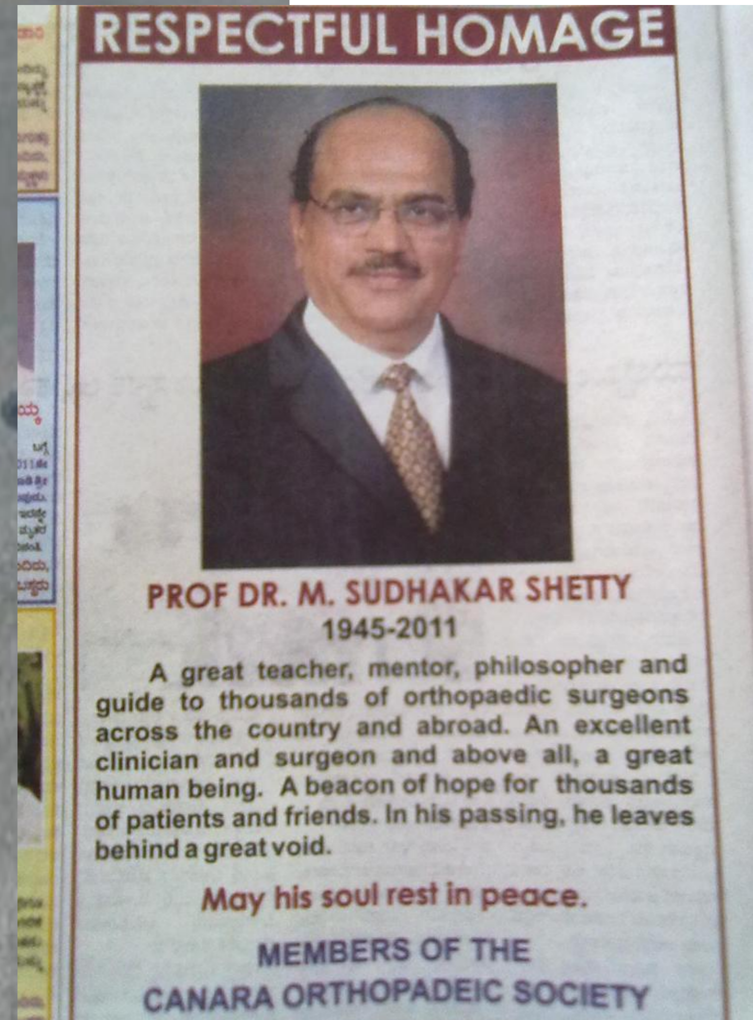
SURGICAL MANAGEMENT OF RICKETS






WELCOME TO
MANGALORE
ಮಂಗಳೂರಿಗೆ
ಸ್ವಾಗತ

to R : Dr. SHIVASHANKAR AITHAL, Dr. R.M. SHENOY, Dr. P.K. USM
SEETHARAMA RAO, Dr. M. SUDHAKAR SHETTY, Dr. P. UMANANDA MAL
JAGANATH KAMATH, Dr. SURENDRA KAMATH, Dr. GHANSHAM KAM



RESPECTFUL HOMAGE

PROF DR. M. SUDHAKAR SHETTY
1945-2011

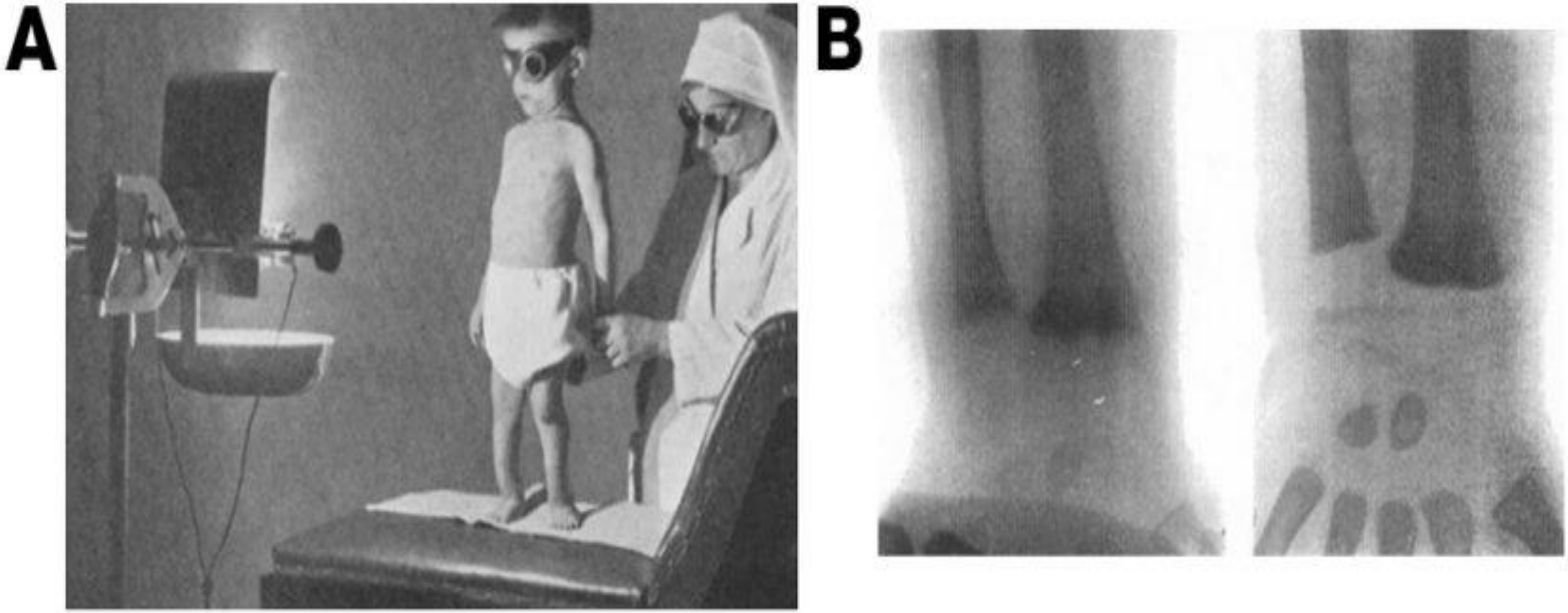
A great teacher, mentor, philosopher and guide to thousands of orthopaedic surgeons across the country and abroad. An excellent clinician and surgeon and above all, a great human being. A beacon of hope for thousands of patients and friends. In his passing, he leaves behind a great void.

May his soul rest in peace.

MEMBERS OF THE
CANARA ORTHOPADEIC SOCIETY


Skeletons affected by Rickets -1749


A
B
leenks.com
SHAIKAT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WHY USE LIGHT
WHEN YOU VE THE SUN SHINING BRIGHT

Rickets making a comeback ?

- Cases of rickets have risen fourfold since studies from the mid-1990s (from 183 to 762).
- Half of Britain's white population, up to 90% of the multiethnic population, and a quarter of children, are suffering from Rickets(the Royal College of Paediatrics and Child Health (RCPCH) .

Rickets is back.....

- Rickets is back with a vengeance(2009)
- Rickets is on the rise once again(2010)
- Rickets epidemic in children(2011)
- Sunscreen causes rickets - new wave of cases in England(2011)

Shock rise in rickets in kids

By STAFF REPORTER

Published: 12 Nov 2010

Add a comment (11)

MORE than one in five kids are showing signs of bone disease rickets as cases explode, say doctors.

Experts were stunned to find 20 per cent of children have the disease and cases of the condition — traditionally linked to poverty — are not concentrated in kids from any particular background.

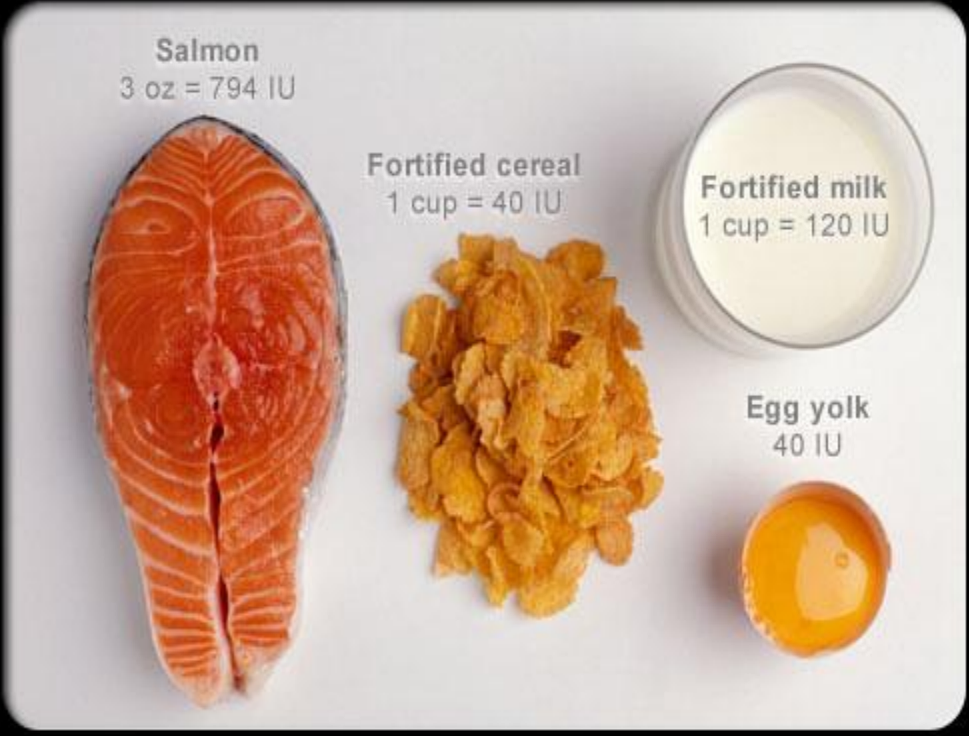
TYPES

- **Nutritional Rickets** — most common type & is caused by a dietary deficiency of vitamin D, calcium, phosphorus, or all three.
- **Vitamin D Resistant Rickets** — also called X-linked hypophosphatemia, a genetic condition thought to be caused by a defect in the kidneys.
- **Vitamin D Dependent Rickets**
- **Congenital Rickets**

OTHER TYPES

- renal osteodystrophy,
- drug-induced rickets,
- hepatobiliary rickets,
- hypervitaminosis D rickets



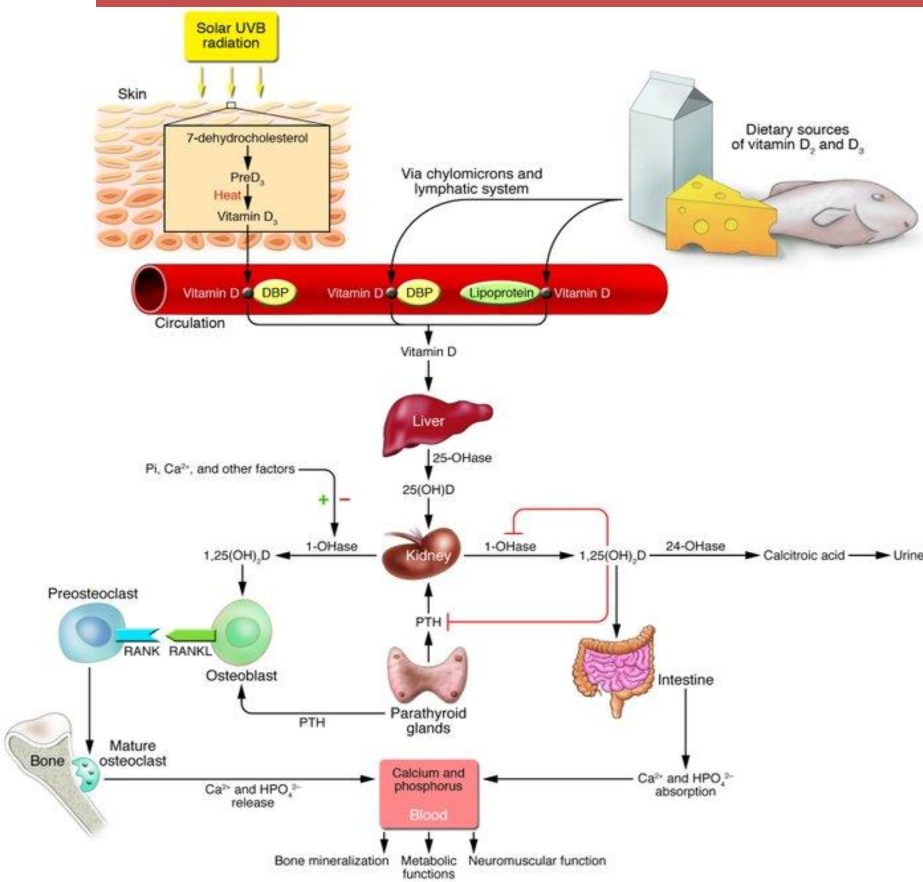

Salmon
3 oz = 794 IU
Fortified cereal
1 cup = 40 IU
Fortified milk
1 cup = 120 IU
Egg yolk
40 IU

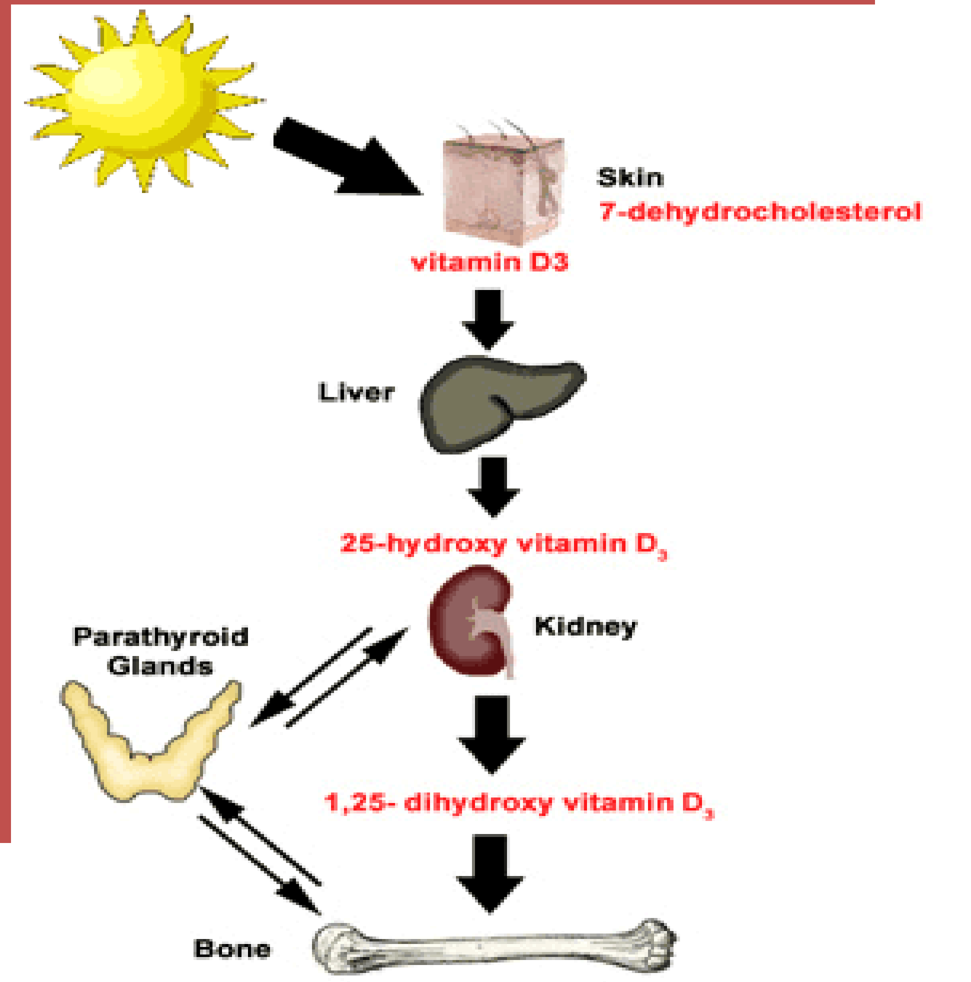
600 IU
(adults under 50)
600 IU
(51–70)
800 IU
(71+)


VAIO

PATHOGENESIS

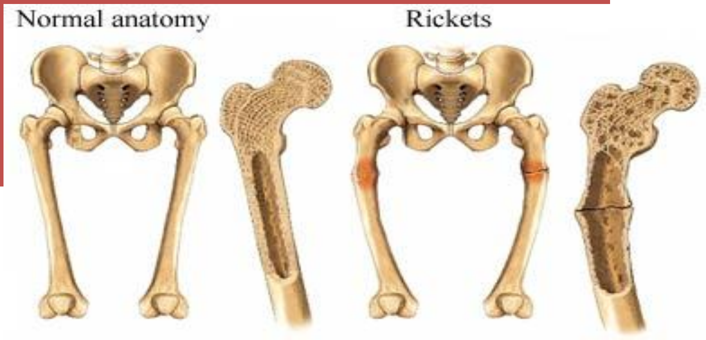
- Failure of osteoid to calcify in a growing person





Histopathology

- Thin cortices with thin and irregular trabeculae
- Widened osteoid seams (unmineralized segments of bone)
- Relatively normal resting and proliferative zone, with a grossly abnormal zone of hypertrophy
- Zone of hypertrophy is widened 5-15 times normal
- Primary spongiosa show only limited bone formation



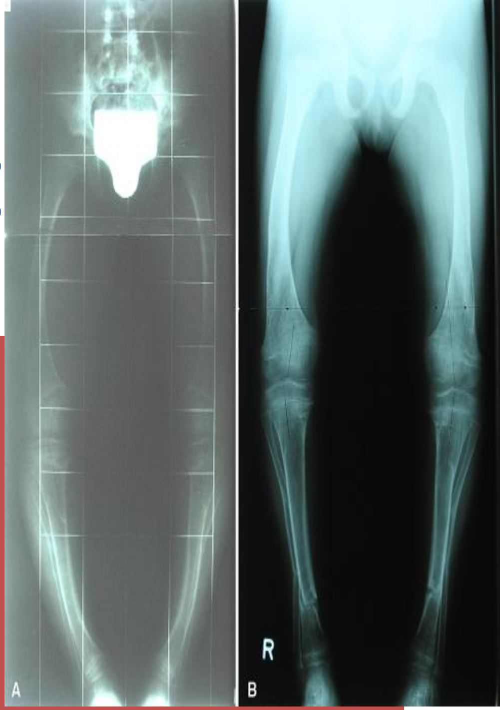
X-rays

- *R*eaction of the periosteum (may occur)
- *I*ndistinct cortex
- *C*oarse trabeculation
- *K*nees, wrists, and ankles affected predominantly
- *E*piphyseal plates, widened and irregular
- *T*remendous metaphysis (cupping, fraying, splaying)
- *S*pur (metaphyseal)

Normal Development

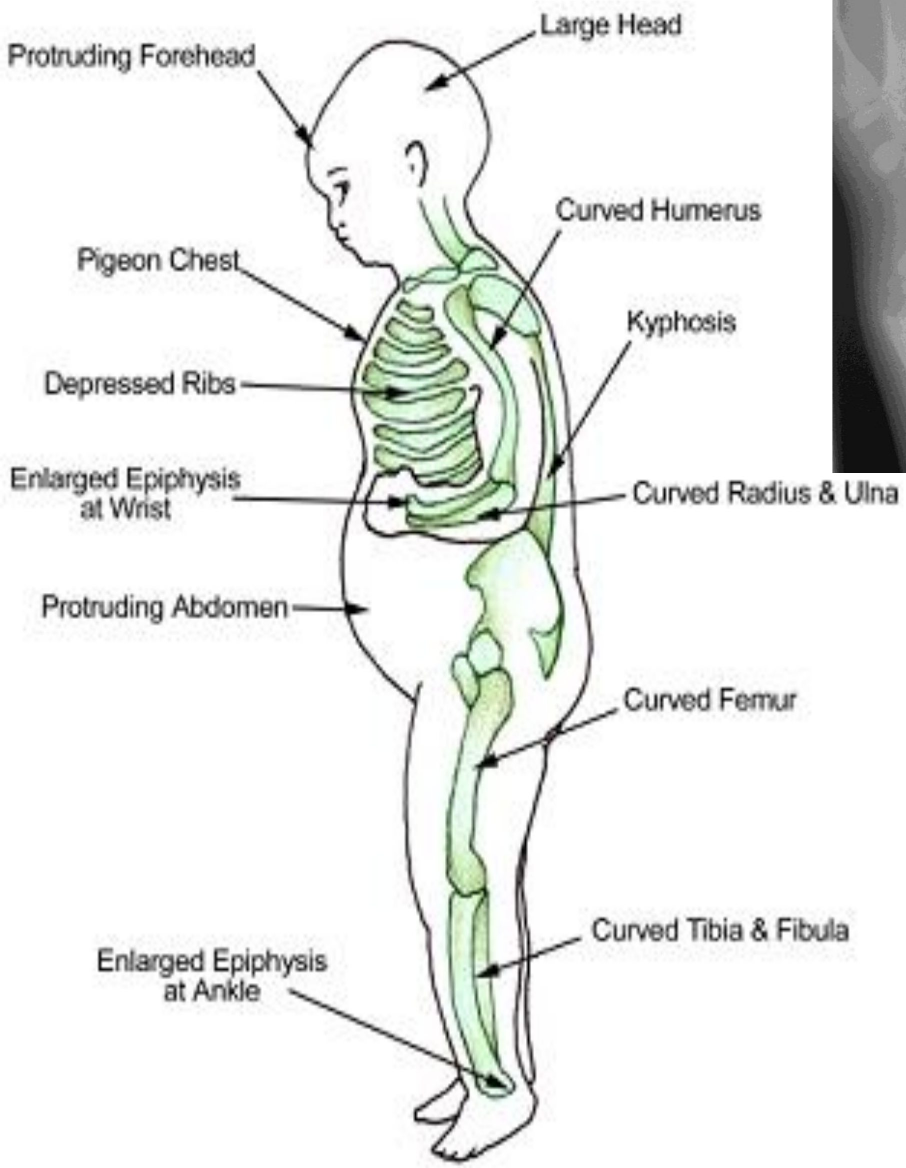
- 1 year: Bow legs / 15° varus
- 2 year: Neutral
- 3 year: Knock knees / 10° valgus
- 6 year: Physiological valgus / 6° valgus
- Note range ~ 15° either way at each age

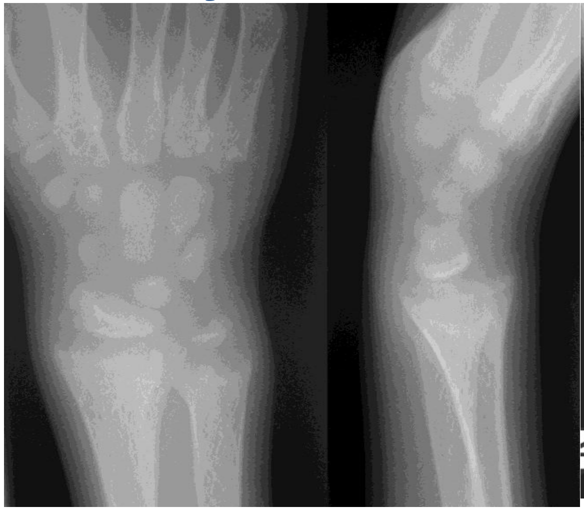
Clinical Assessment

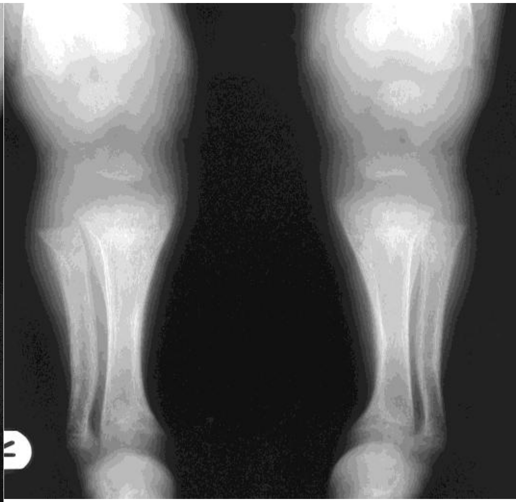


- - Unilateral / bilateral
-   - Angular profile
-   - Femorotibial angle
-   - Inter-malleolar / intercondylar distance (quantify)
-   -LLD / rotational profile / joint laxity
-   - Height vs. Age

X-Rays

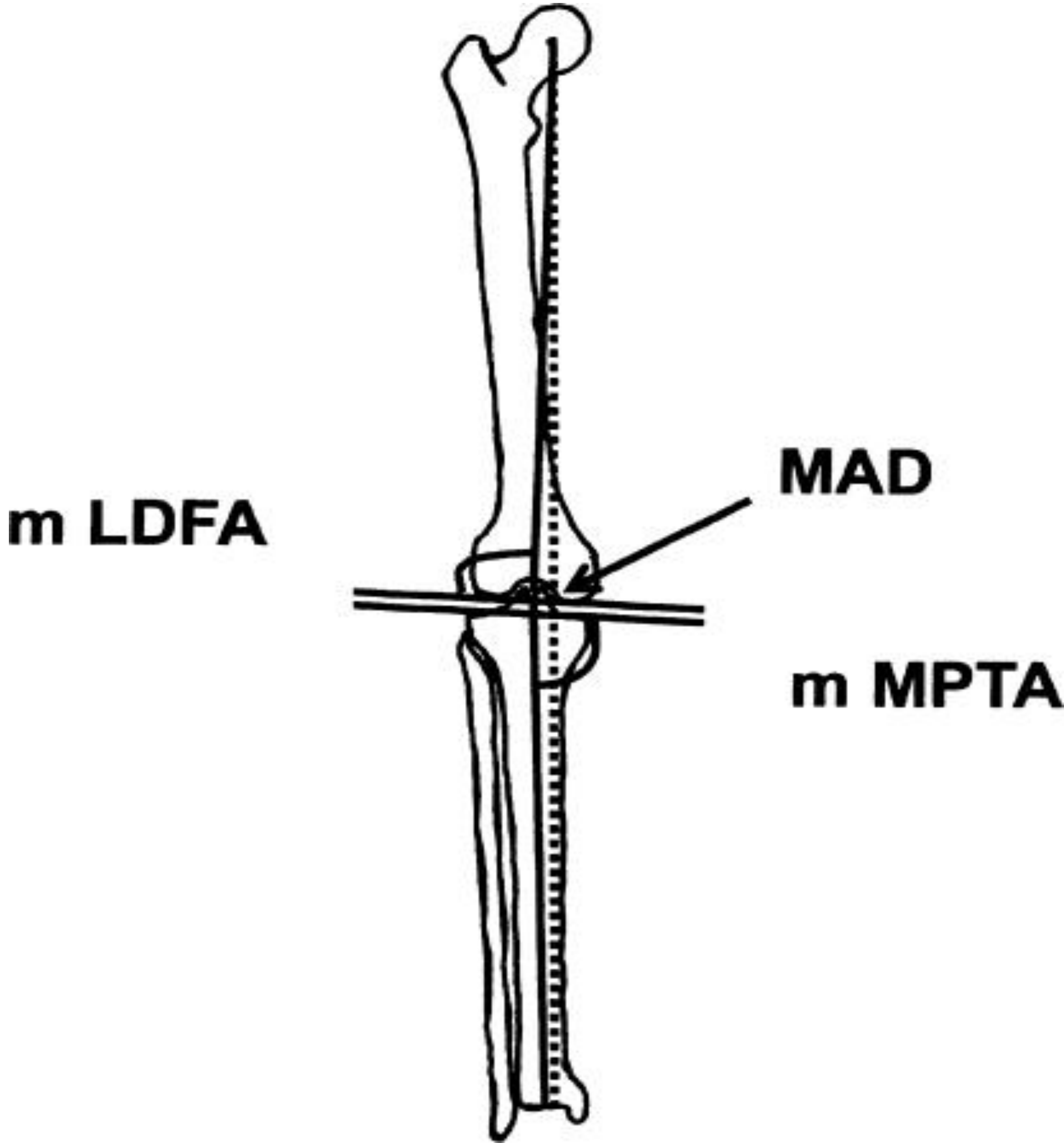

Large Head
Protruding Forehead
Curved Humerus
Pigeon Chest
Kyphosis
Depressed Ribs
Enlarged Epiphysis at Wrist
Curved Radius & Ulna
Protruding Abdomen
Curved Femur
Curved Tibia & Fibula
Enlarged Epiphysis at Ankle



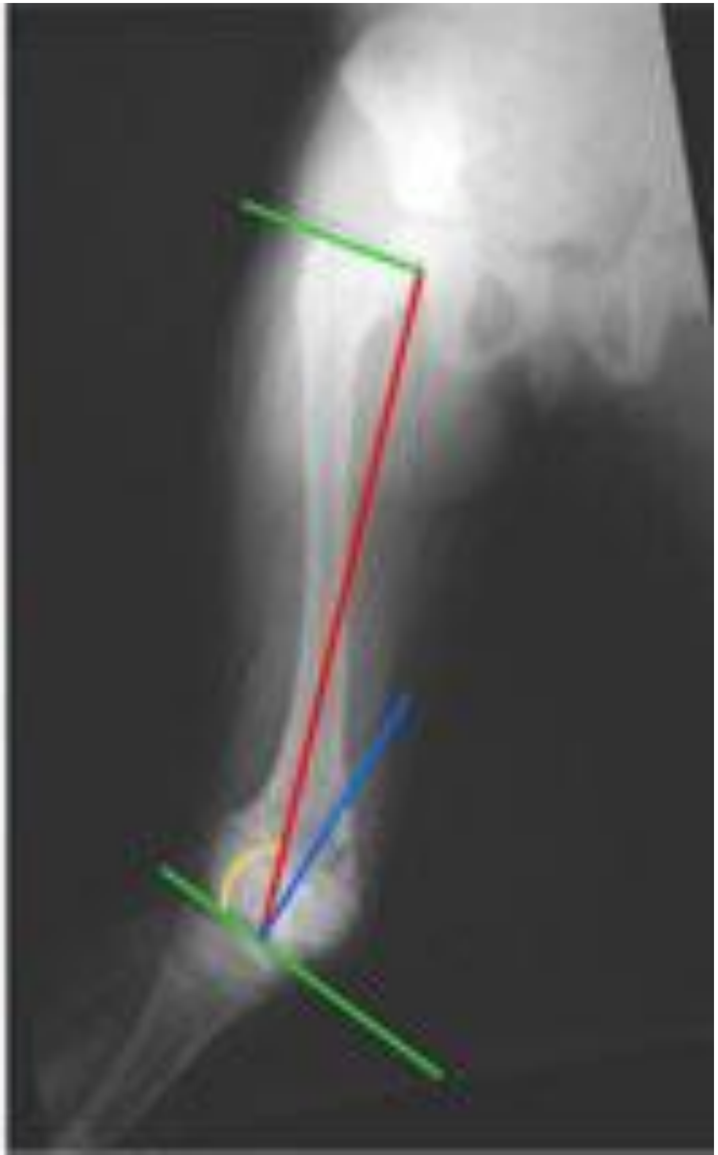


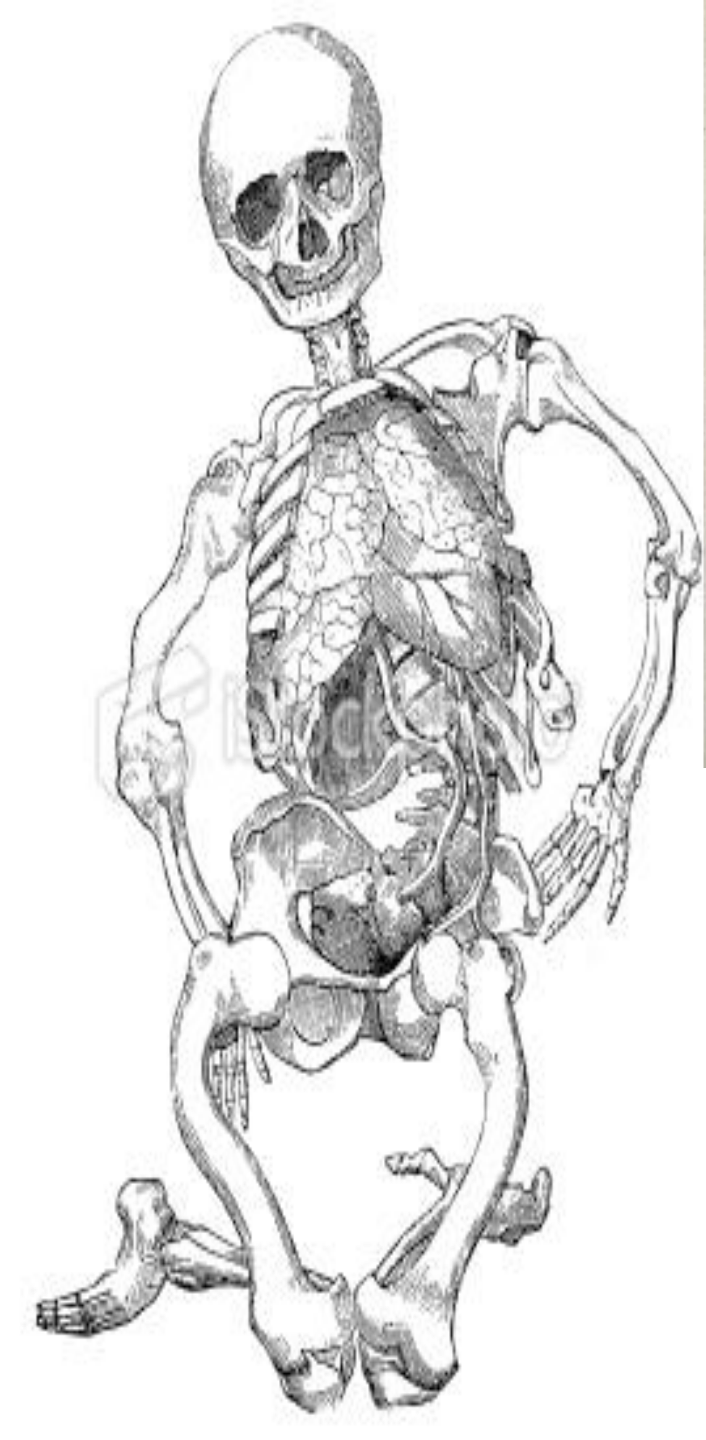
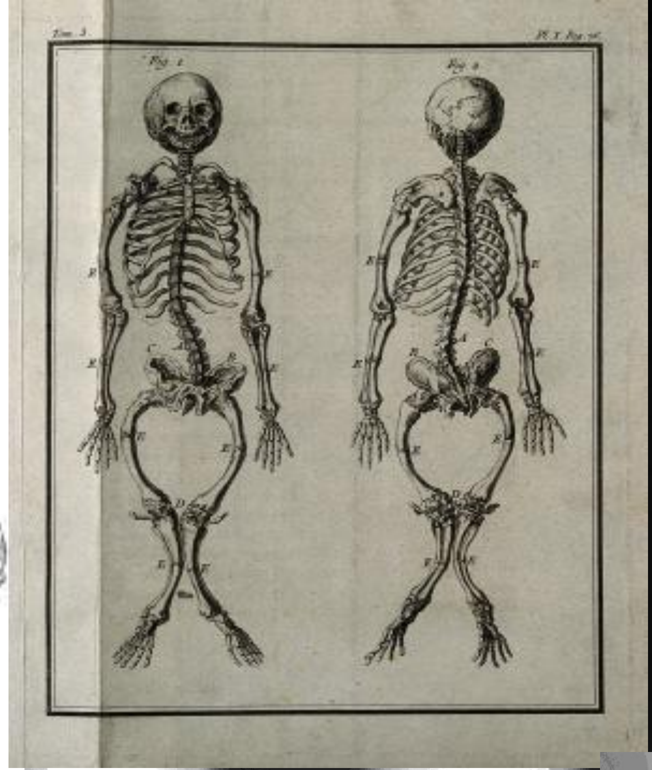

R

X-RAYS



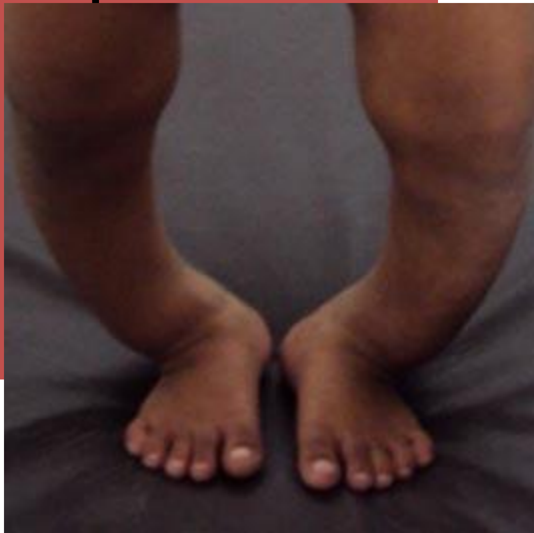
X-Rays



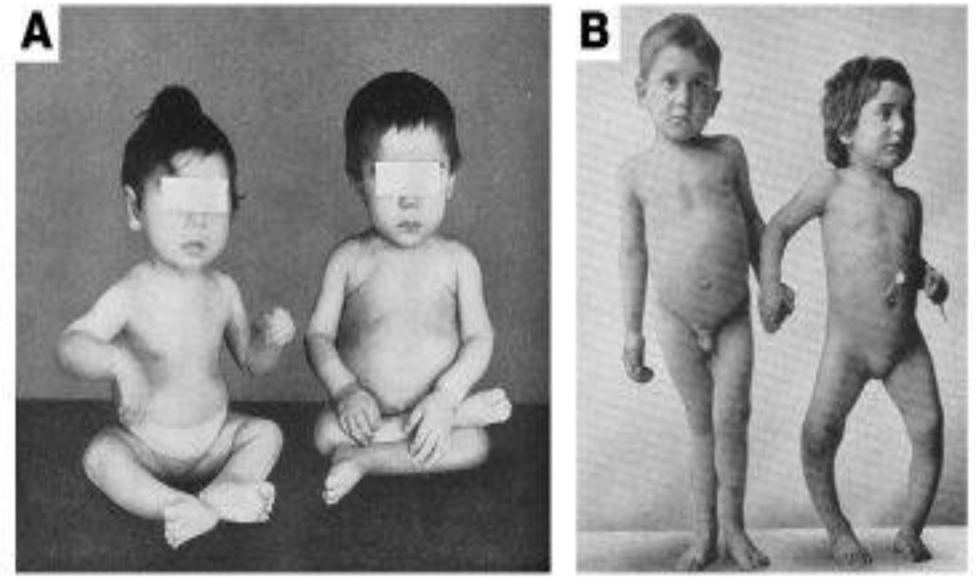

Medical Management

- Vit.D for 6-10 weeks
- X-rays at the end of 24 weeks
- Residual deformity is rare in Nutritional type following medical Management
- Severe deformities seen in Hypophosphatemic type



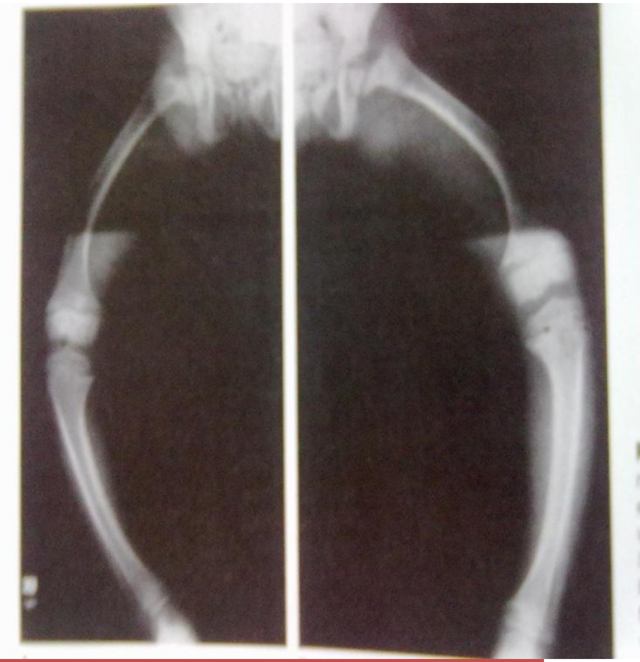
Recommended daily Calcium intake

- 1 to 3 years of age. 500 milligrams (mg) .
- 4 to 8 years of age. 800 mg.
- 9 to 18 years of age. 1,300 mg.
- 19 to 50 years of age. 1,000 mg a day



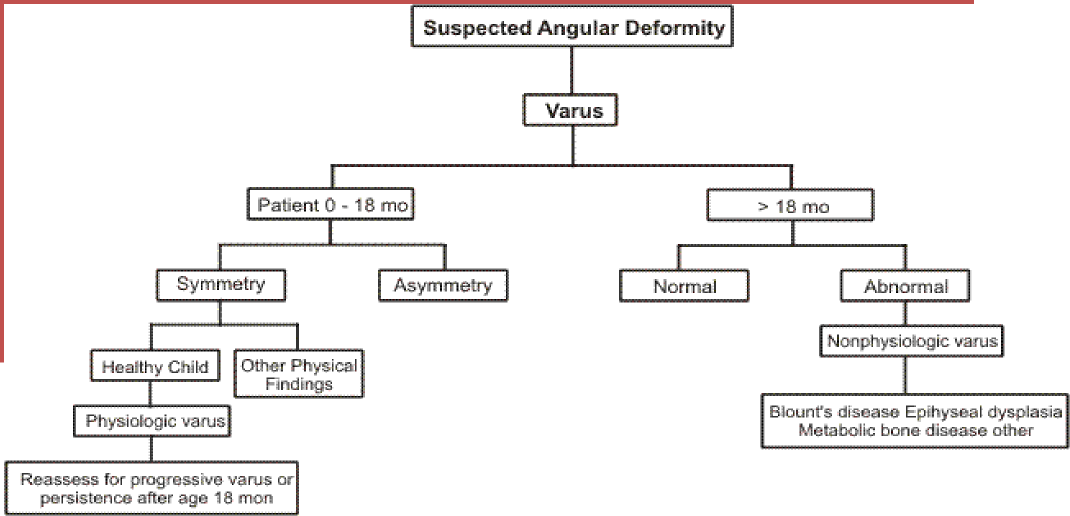
INDICATIONS FOR SURGERY

- - If pathological form suspected
- - Asymmetry
- - < 5th percentile
- - Severe deformity
- - Positive Family history
- Calcitriol & Phosphate for at least 6 months
- Pain, difficulty in walking, compromised gait



Pre-Operative period

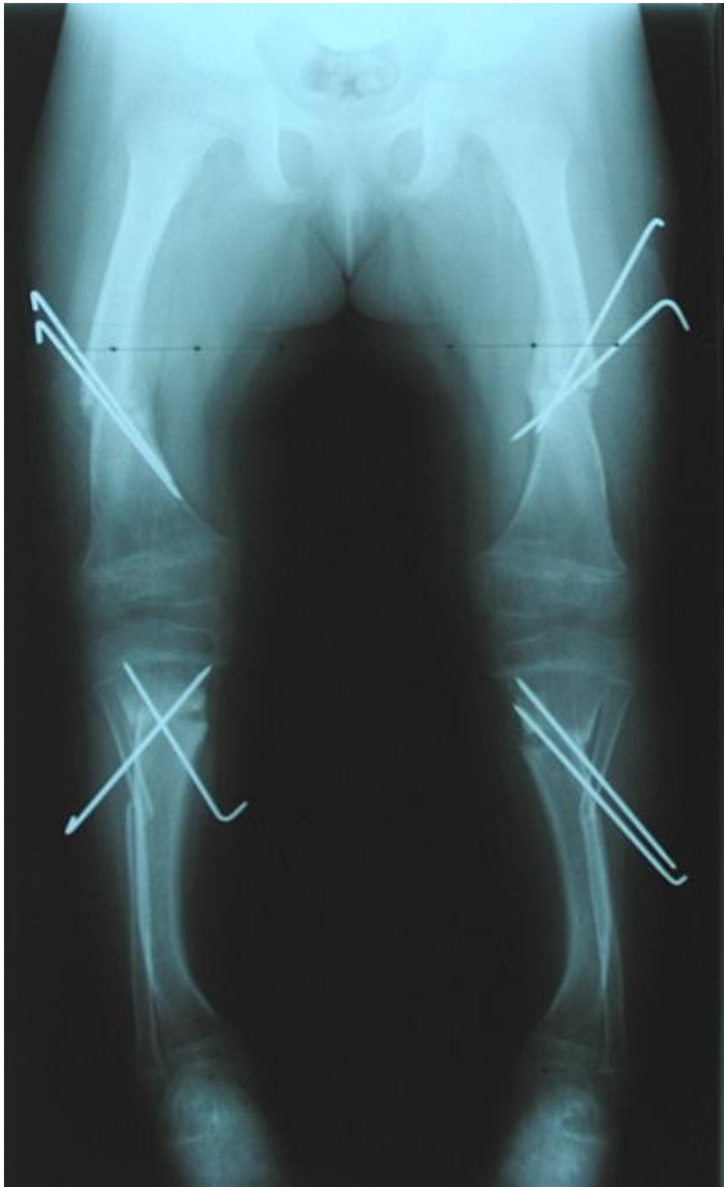
- Lab.values to be checked
- Discontinue Vit.D
- Assess lower limb alignment
- Shoe wedges & bracing ineffective
- Avoid dogmatic predictions



Surgical Treatment

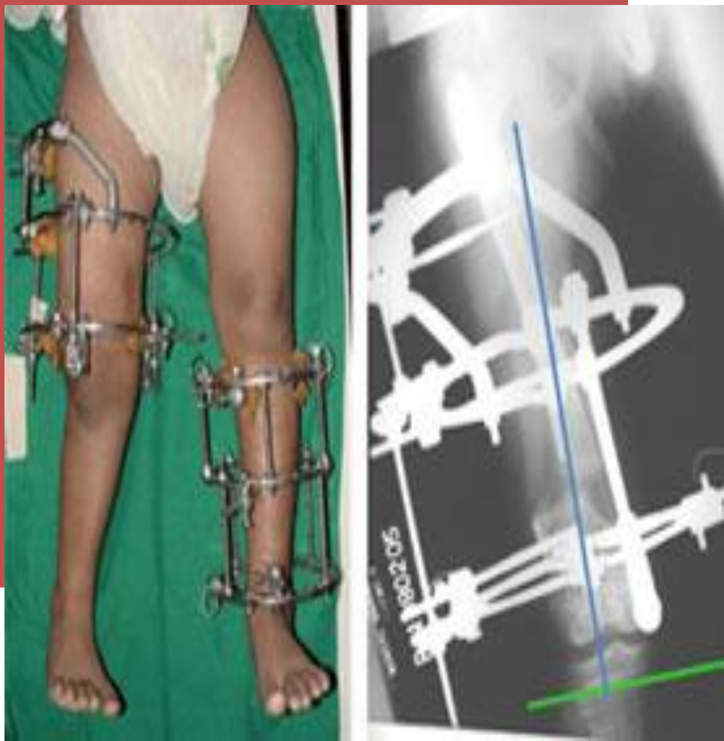
- Multilevel osteotomies
- Mild overcorrection desirable
- Acute/gradual correction of deformity

Corrective osteotomies

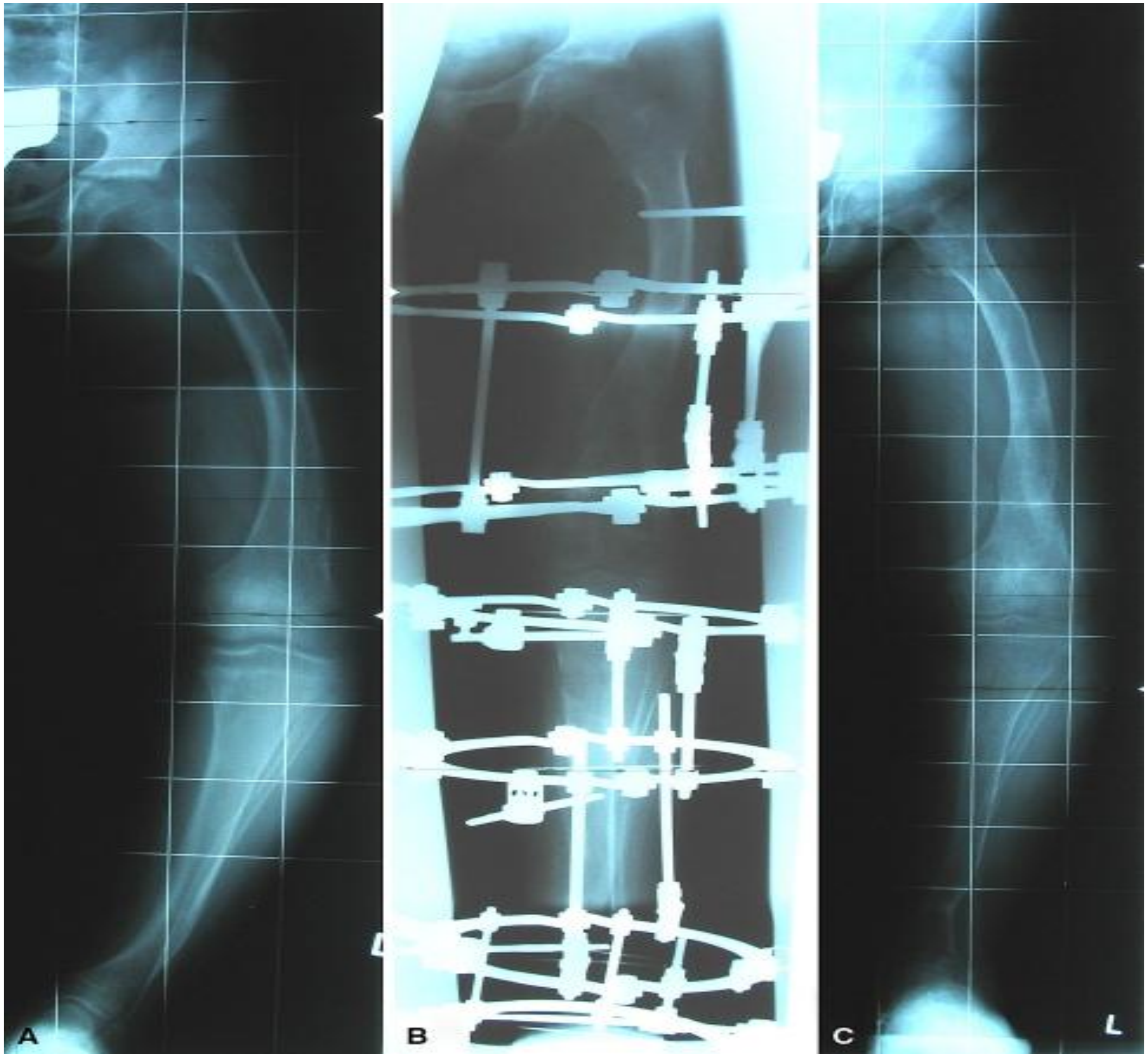


Surgical Methods

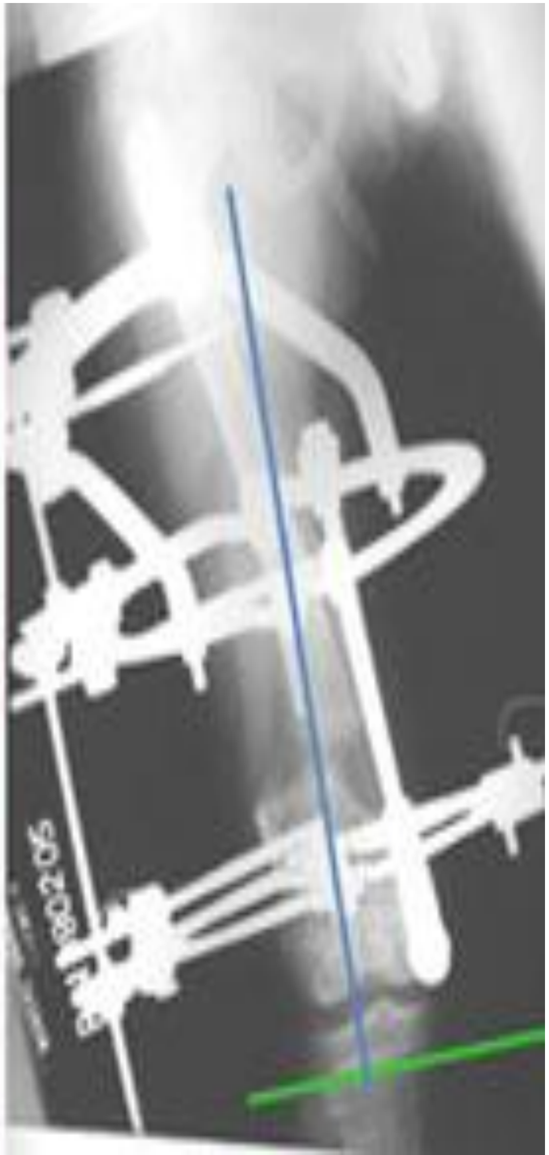
- Ilizarov ring fixator/Unilateral ext.fixator
- ‘Fixator-assisted nailing’
- Orthofix eight-plate – Guided growth plate device
- Ilizarov-Veklich device
- Hemiepiphyseodesis



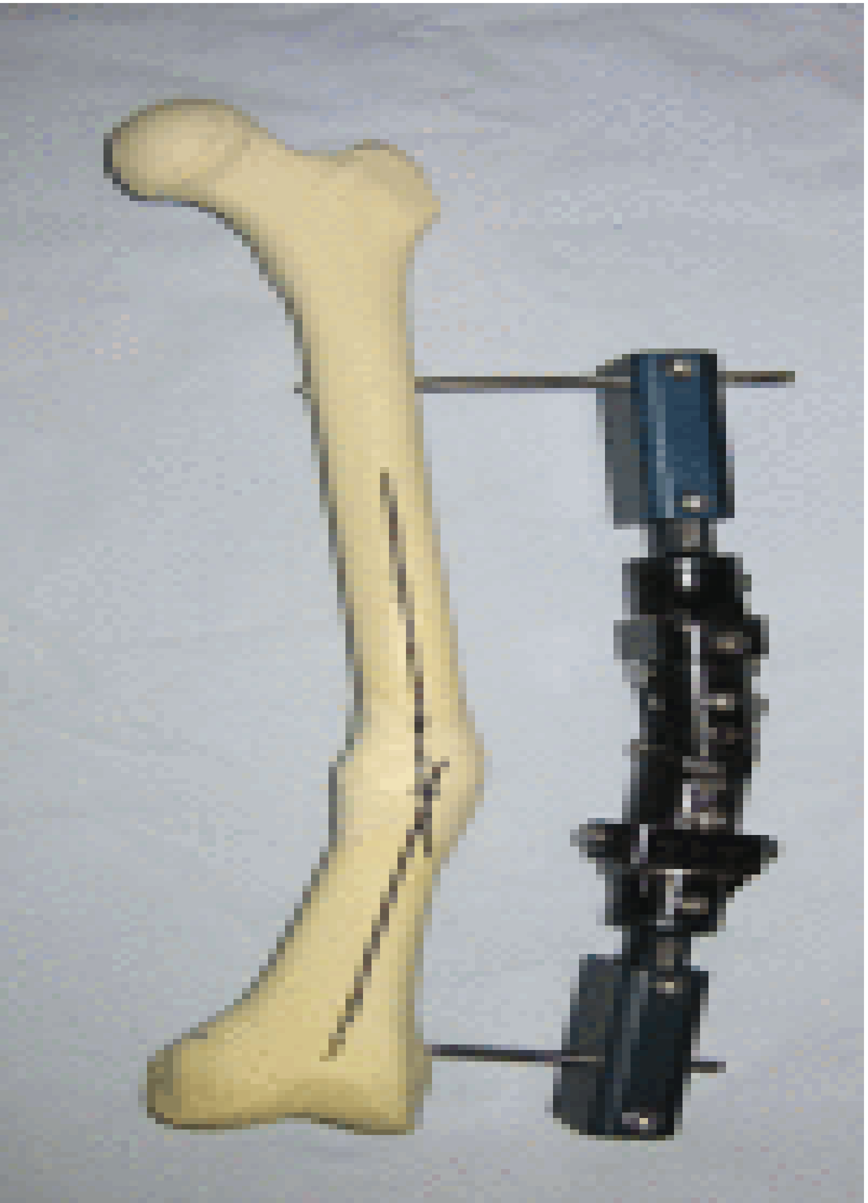
Ilizarov Fixator

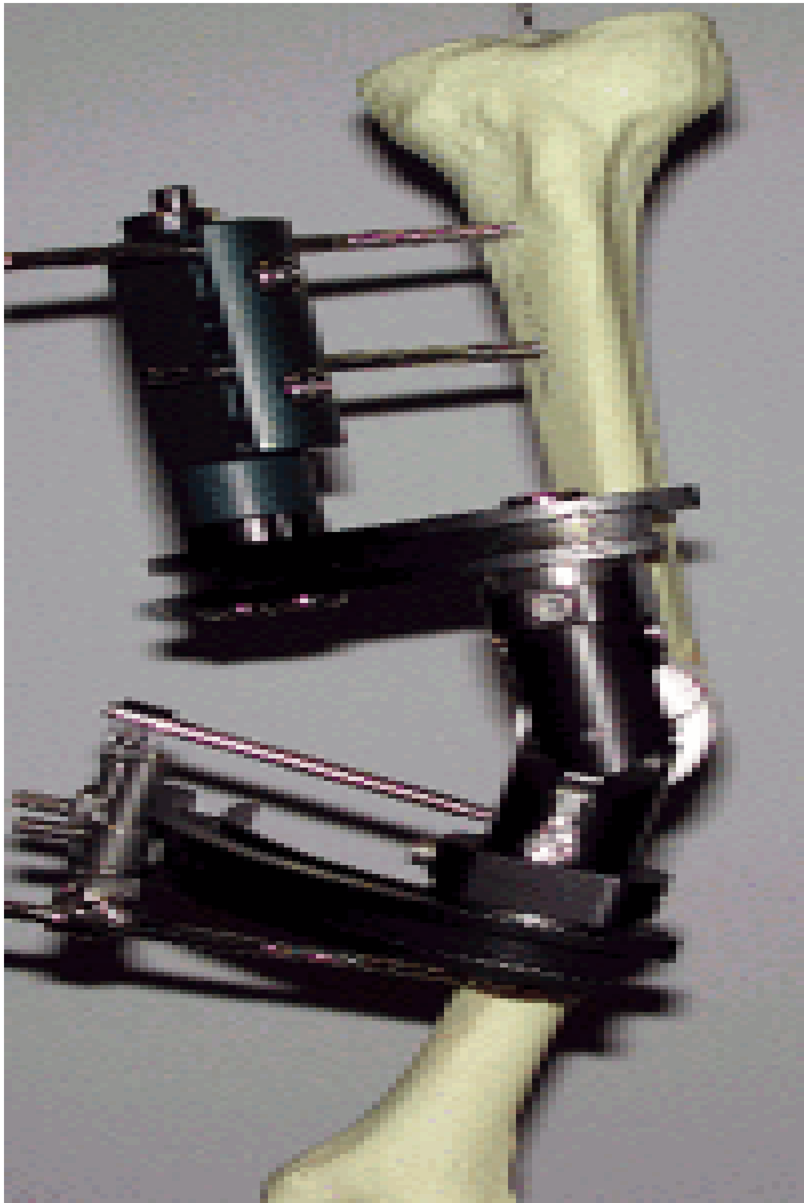


Ilizarov Fixator

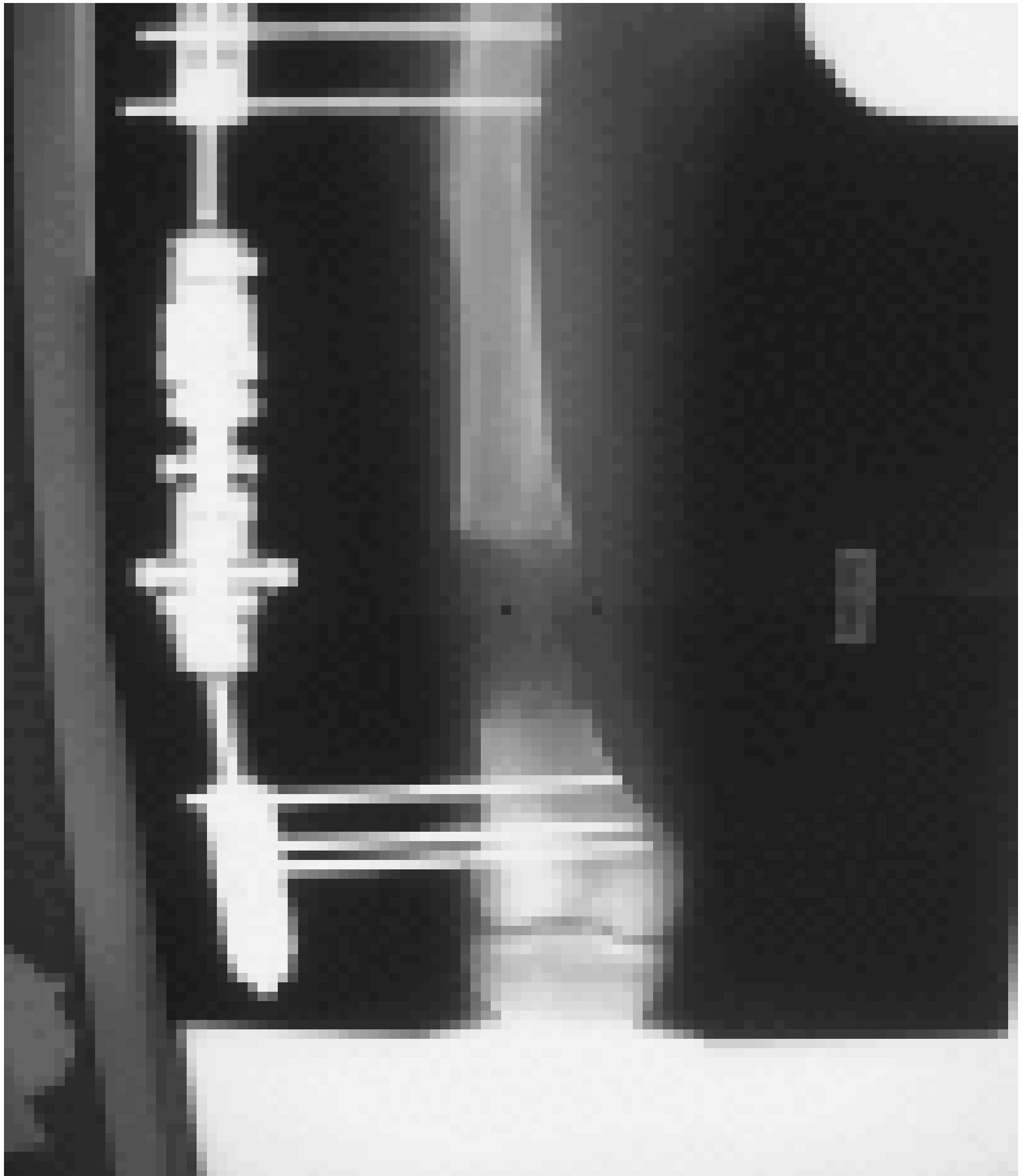


External Fixator



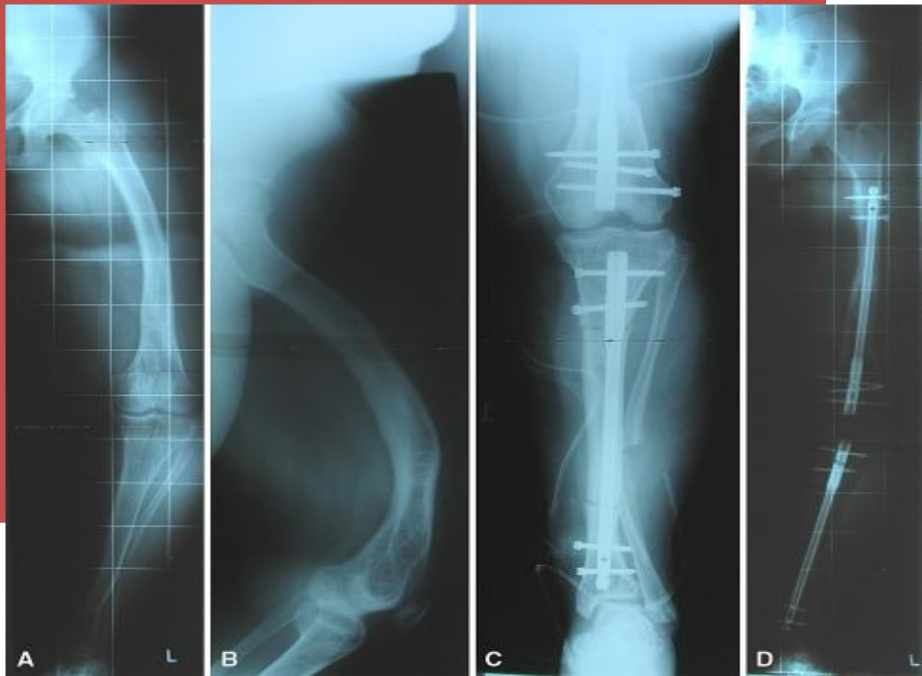


External Fixator



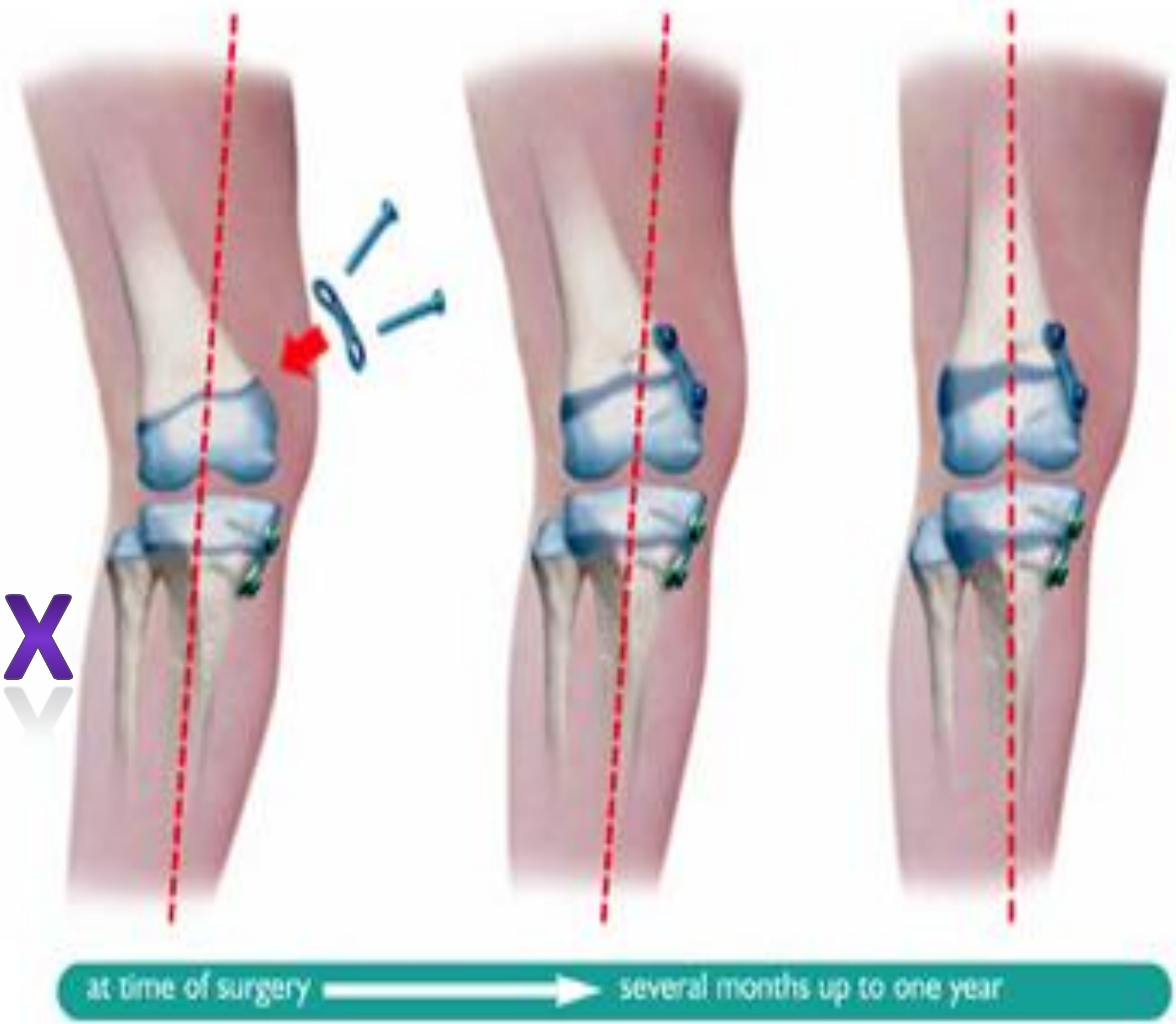
Fixator-assisted Nailing

- Ext.fix.for short/long-term: Deformity correction/Lengthening
- I.M. nail prevents recurrence of deformity and refractures.



Guided growth plate device

ORTHOFIX 8 PLATE



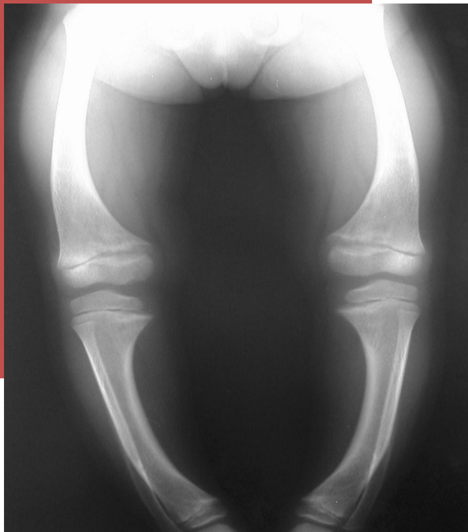
Ilizarov-Veklich device

- Made from titanium
- Strong and safe
- Smaller than other external devices
- Light in weight
- Comfortable
- Leaves minimal scarring

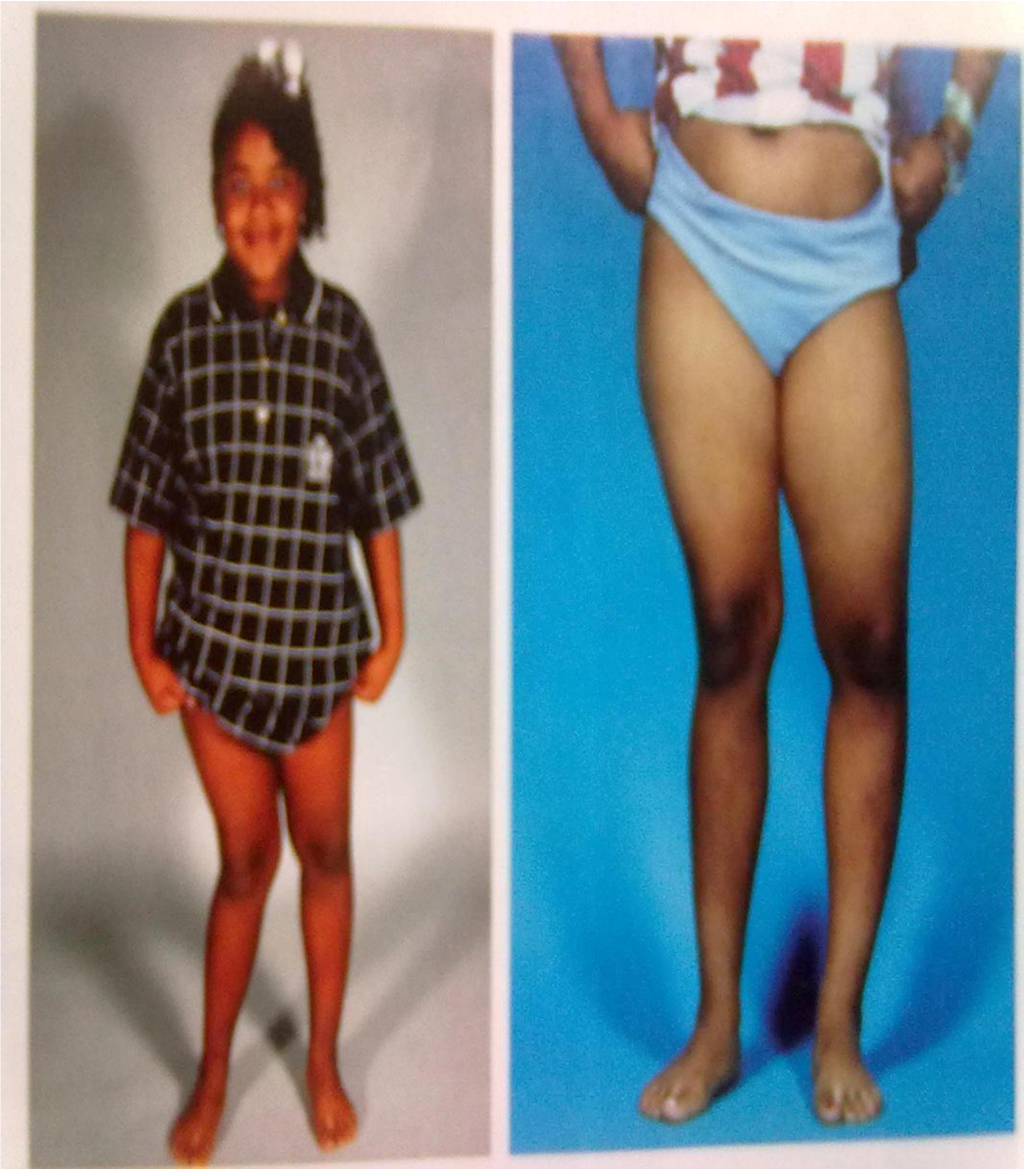
A patient walks before bow legs correction surgery

Hemiepiphyseodesis

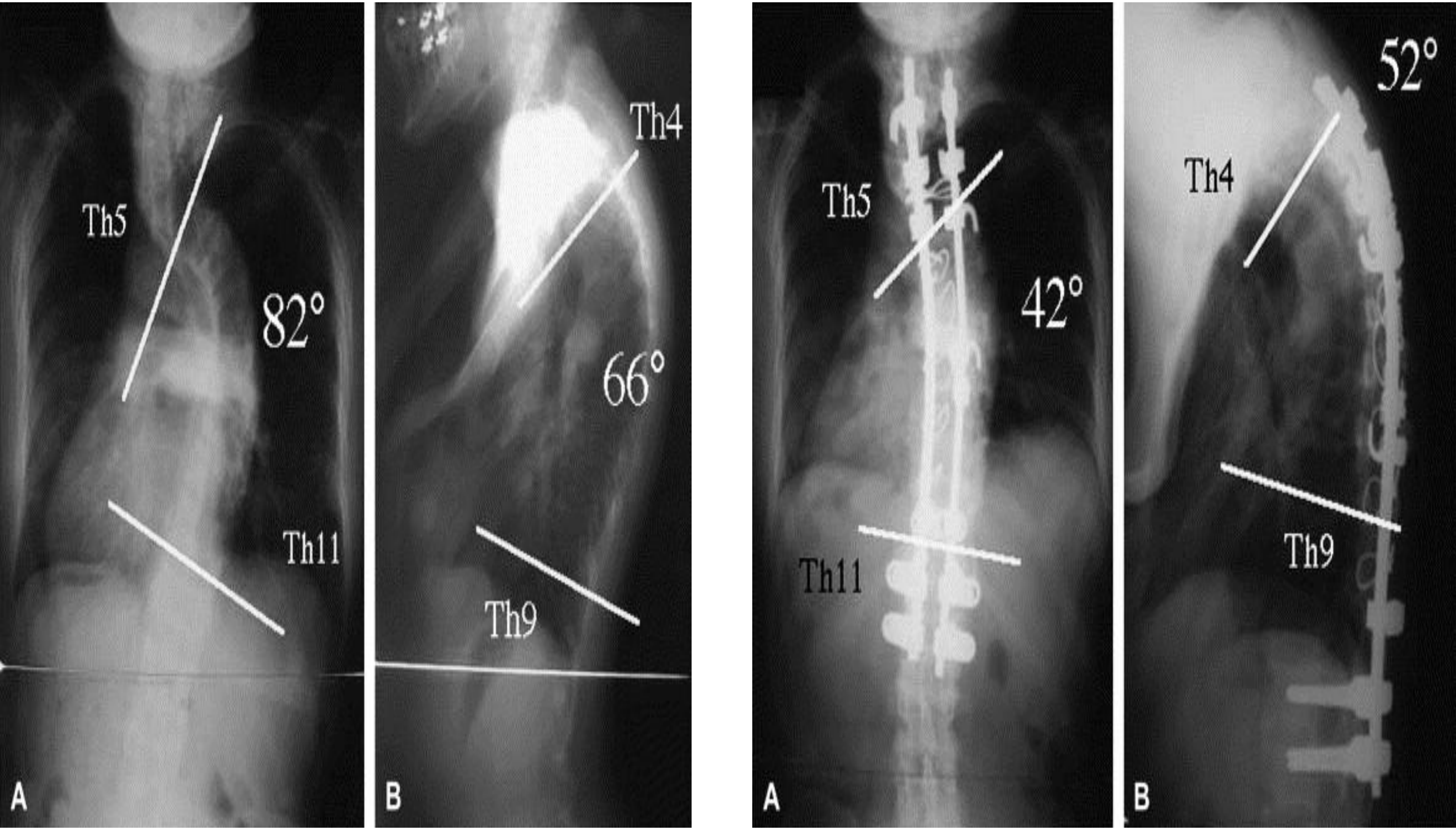
- Produces unilateral physeal inhibition
- Gradual correction of deformity close to the joint
- Lesser surgical procedure than osteotomy
- It is difficult to calculate the exact age for this procedure



Hemiepiphyseodesis



Spinal deformity correction

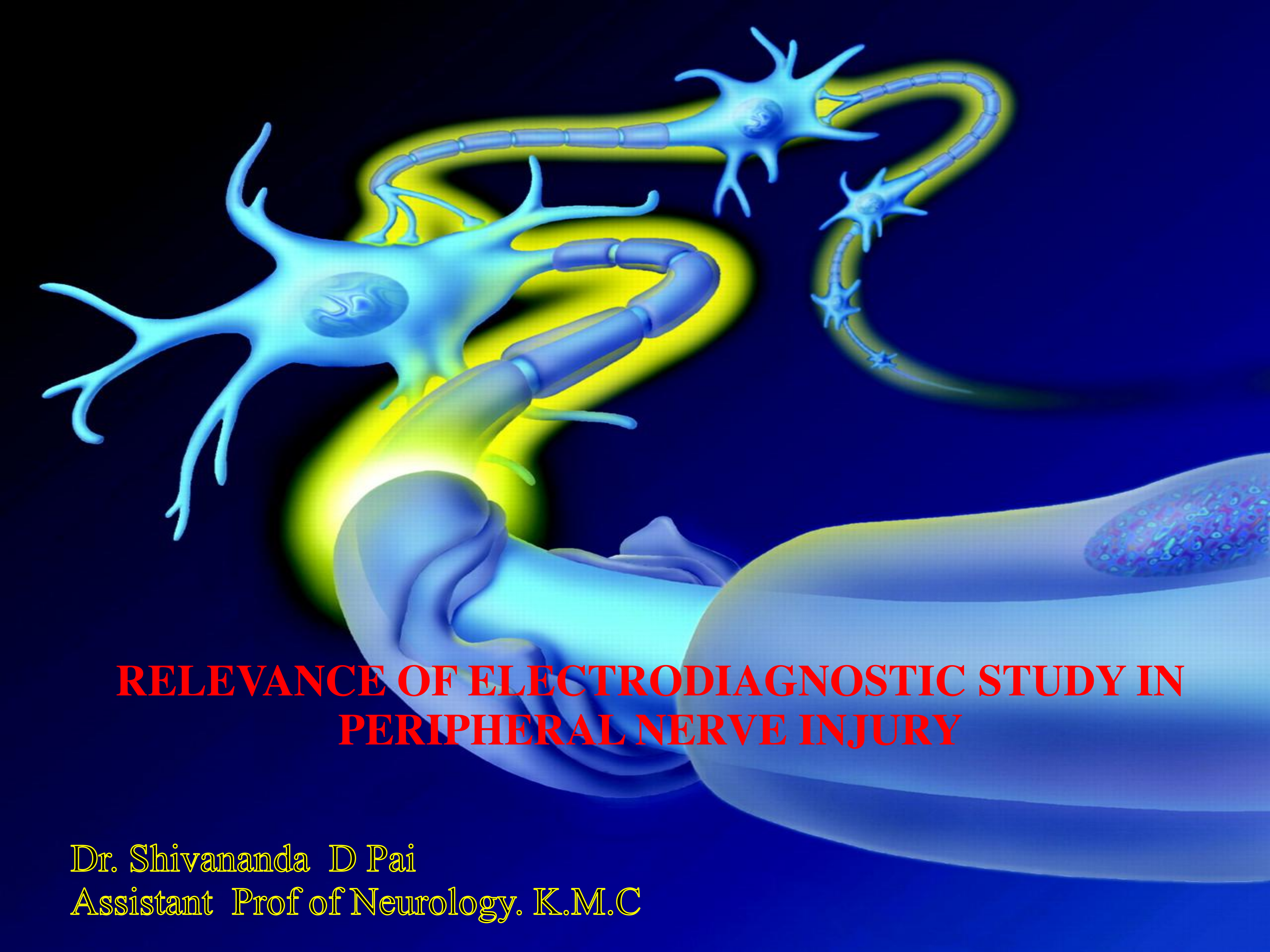


Post-Op.Period

- Avoid "recumbency hypercalcemia"
- Mild deformities should not be overcorrected in young childhood in Hypophosphatemic rickets
- Treat concominant problems in cases of Renal Osteodystrophy

SURGICAL TREATMENT OF RICKETS

- Most deformities resolve with Medical line of management
- Severe deformities in Hypophosphatemic/Renal rickets
- Multiple osteotomies & int./ext.fixn.
- Careful pre-op.planning & post-op.mobilisation
- Spinal deformities need surgical correction



RELEVANCE OF ELECTRODIAGNOSTIC STUDY IN PERIPHERAL NERVE INJURY

Dr. Shivananda D Pai
Assistant Prof of Neurology. K.M.C

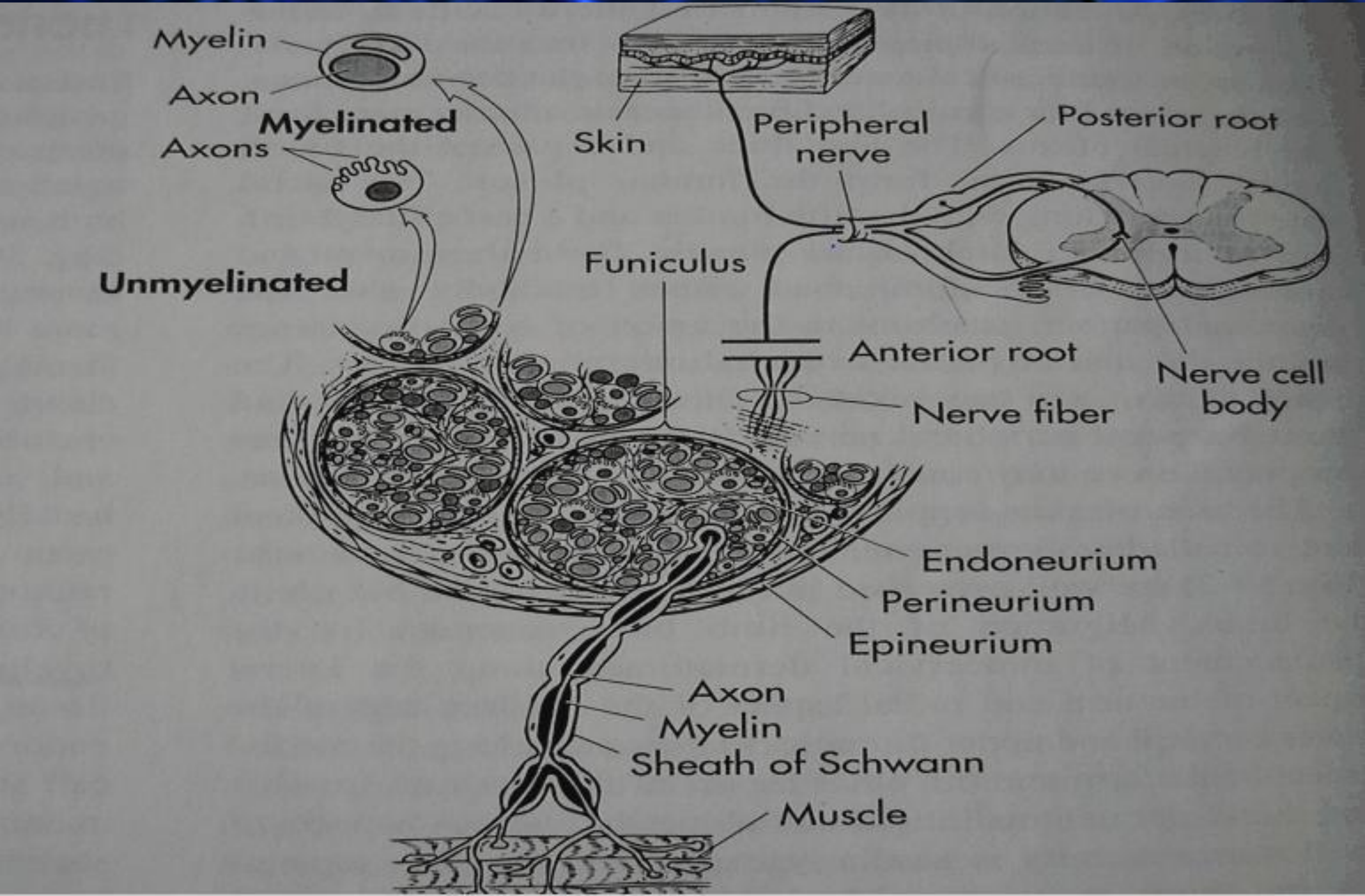
NORMAL NERVE ANATOMY

* Axon

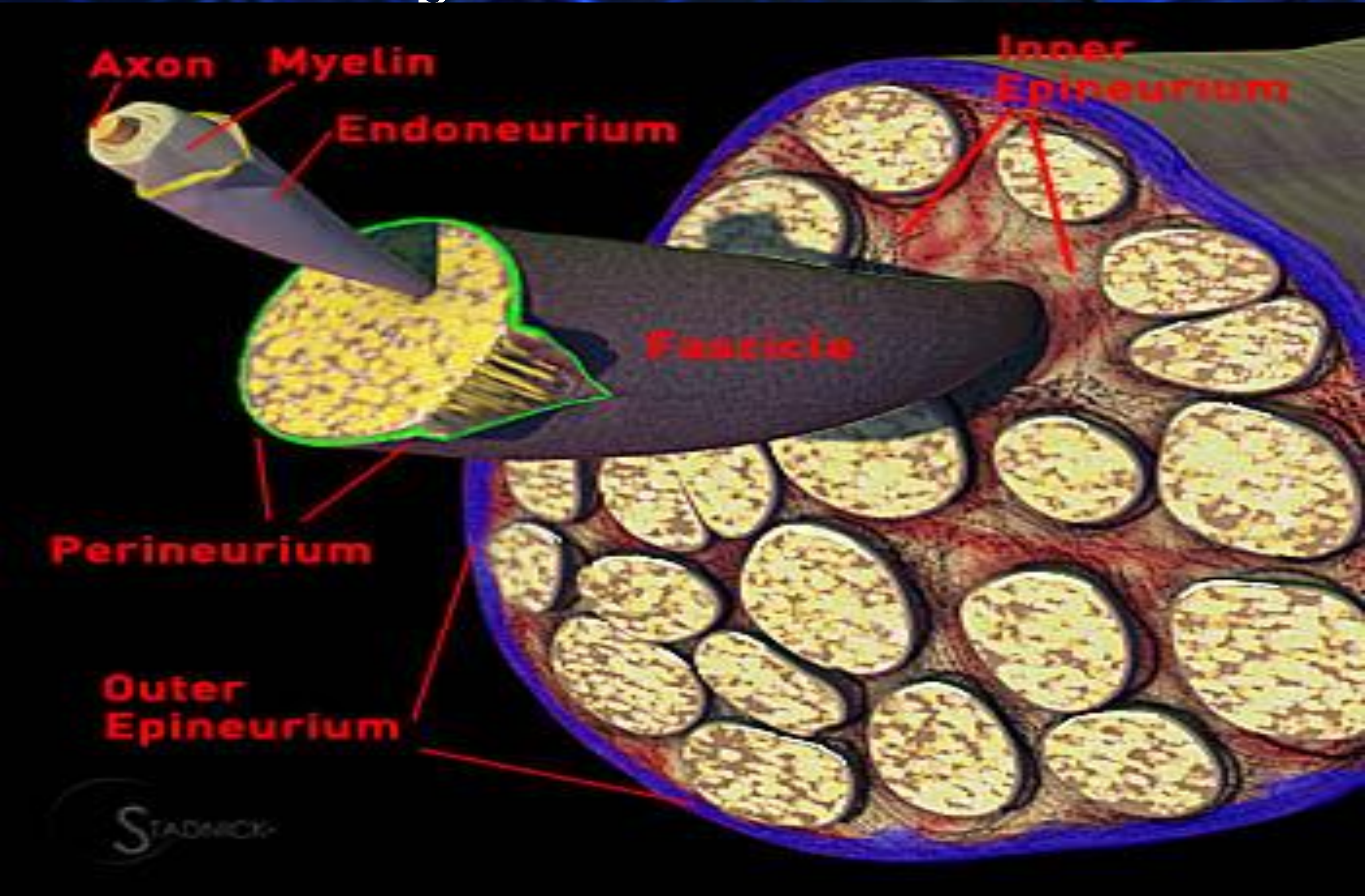
* Myelinsheath with schwan cell
  - Nodes of Ranvier (more Na+channels)
  - Internodal region

* Connective tissue coverings
  - Endoneurium (surrounds nerve axon fibers)
  - Perineurium (surrounds fiber groups to form a fascicle)
  - Epineural (binds fascicles into nerve)

PERIPHERAL NERVE



COVERINGS


Axon
Myelin
Endoneurium
Inner
Epineurium
Fascicle
Perineurium
Outer
Epineurium

Etiology of Peripheral nerve injuries

1. Metabolic or collagen disease
2. Malignancy
3. Endo or exo – toxins
4. Ischaemia
5. Radiation
6. Trauma
    - Thermal
    - Chemical
    - Mechanical
    - Infection : Leprosy

PATHOPHYSIOLOGY NERVE INJURY

1.

- * Ischemia and pressure will decrease intraneural flov

- * Ischemia :
- - 15-45min causes neuropraxia (reversible)
- - >8hrs is not reversible
- * Stretch 5-10% leads to nerve elongation

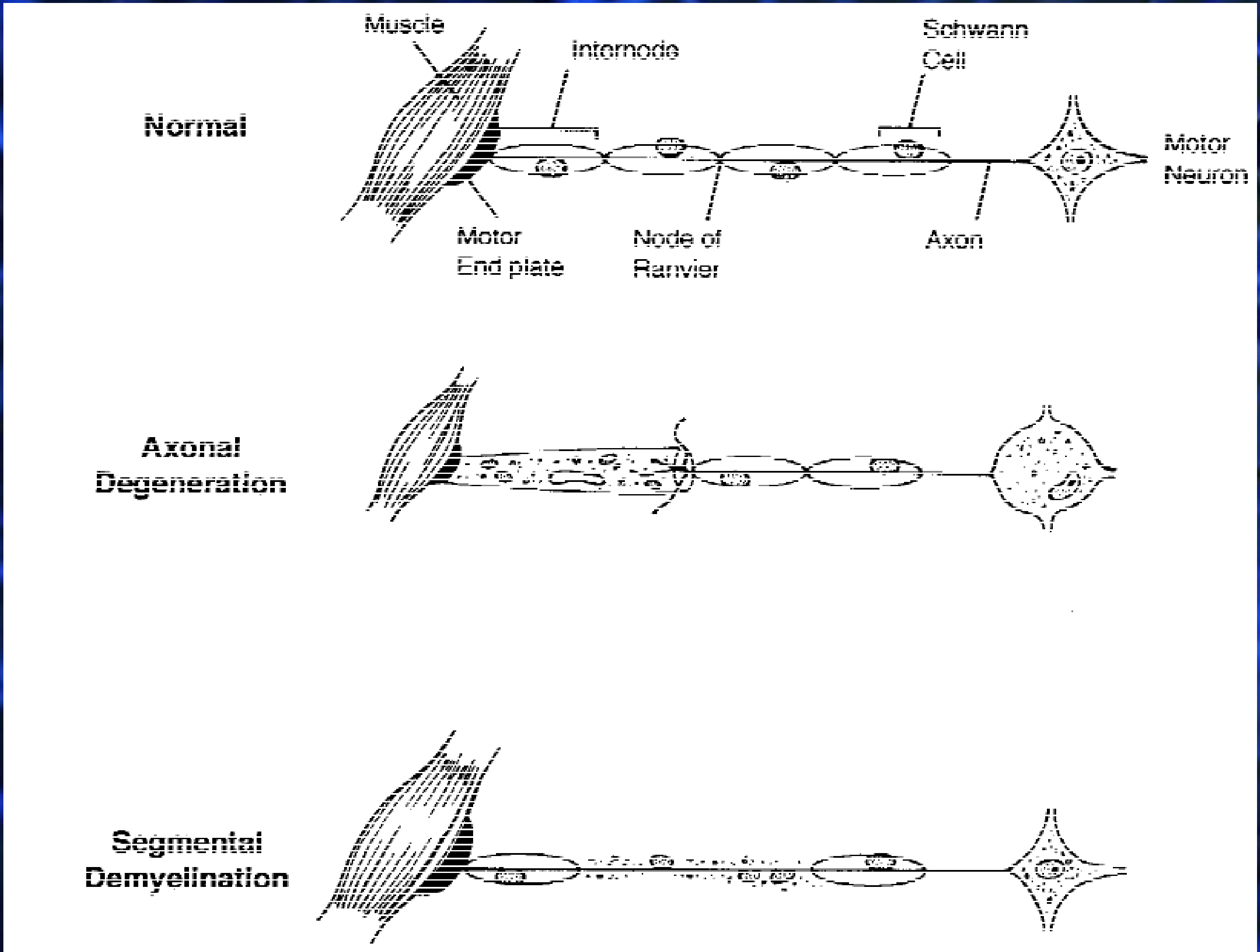
- * Mechanical pressure leads to structural changes
- - largest fibers (motor, vib, proprioception)
- - Peripheral fibers

Pathophysiology (cont)

* Pressure
    - pressure 30mmHg blocks venous blood flow
    - pressure 30-60mmHg blocks axonal transport
    - pressure 60-120mmHg blocks intraneural blood flow

* Chronic pressure leads to perinodal demyelination

Normal peripheral motor nerve anatomy and responses to injury



- Nerve injuries that are associated with focal interruption of the continuity of the axons cause significant changes in the structure of the peripheral nerve distal to the lesion.
- The distal axons undergo a degenerative process, known as *wallerian degeneration*

Consequences of Focal Axonal Injury Distal to the Lesion

- Wallerian (axonal) degeneration
- Myelin breakdown
- Neuromuscular transmission failure
- Endoneurial tube shrinkage
- Fascicular atrophy
- Denervation atrophy of muscles

* Within hours of most nerve injuries, myelin begins to retract from the axons at the nodes of Ranvier. This is followed by swelling of the distal nerve segment , leakage of axoplasm, and subsequently the disappearance of neurofibrils.

* Within days, the axons and myelin fragment, and digestion of nerve components starts. By the end of the first week, the axon and myelin become fully digested and Schwann cells start to bridge the gap between the two nerve segments.

* In chronic nerve lesions, the endoneurial tubes in the distal stump shrink, the nerve fascicles atrophy distal to the lesion, and , in complete nerve transection, the severed ends retract away from each other.

Recovery from peripheral nerve trauma may occur by three mechanisms,

1. Remyelination
2. Collateral sprouting of axons
3. Regeneration from the proximal site of injury.

*Remyelination is the fastest of these reparative processes , occurring over 2-12weeks, depending on the extent of the injury.

* Following degeneration of injured distal axon fragments, collateral sprouts from intact neighboring axons may provide innervation to denervated muscle fibers. This process takes 2-6months

* In cases of severe axonal injury, collateral sprouting is not Sufficient to provide innervation to all muscle fibers. Further Clinical recovery depends on regeneration from the proximal site of injury, which may require up to 18months.

Role of electrodiagnostic studies in Peripheral Nerve injury

1. Localize the site of nerve injury
2. Determine the pathophysiology of the lesion
3. Estimate the severity of the injury
4. Determine the prognosis
5. Assess the progress of remyelination and reinnervation

Demyelination

* It is not uncommon for a disease process to preferentially injure the nerve's myelin
  - chronic ETOH, DM, lead, diptheria, porphyria

* Damaged myelin is removed / replaced (demyelination/ re-myelination)

* In profound demyelinating disease, it is not uncommon to see secondary axonal injury

Axonal Injury

* Walleria degeneration – secondary axon degeneration distal to the site of injury

* Also see some axonal degeneration proximally along with nerve cell body alterations due to edema and blocked axonal flow / transport

Nerve Regeneration

* The timing of recovery depends on the distance of the lesion from the denervated target muscle. Proximal regeneration occurs at a rate of 6-8mm/day.

* Schwann cell tubes remain viable for 18-24months after injury. If the axon does not reach its target muscle within this time, these supporting elements degenerate and effective regeneration cannot occur.

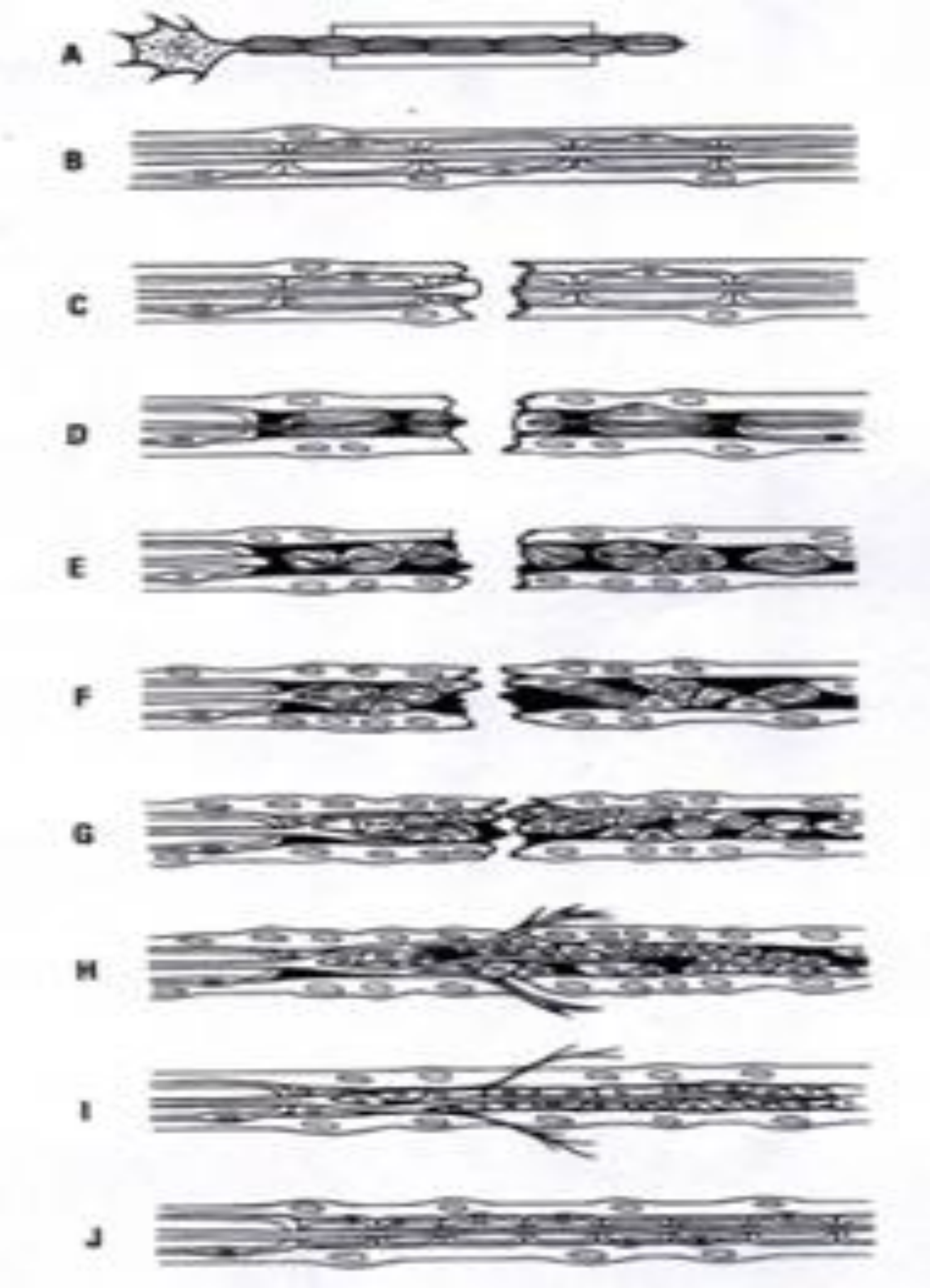
Nerve Re-innervation

* Endoneurial tube (ET)- re-innervation occurs through ET to distal target site.

* Recovery plateaus 18-24months

* Physical separation of ET leads to poor prognosis
- misdirection of nerve sprouting
- less common with compression injuries

* Muscle atrophy seen if not re-innervated by 1-1.5yrs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Categories of Nerve Injury

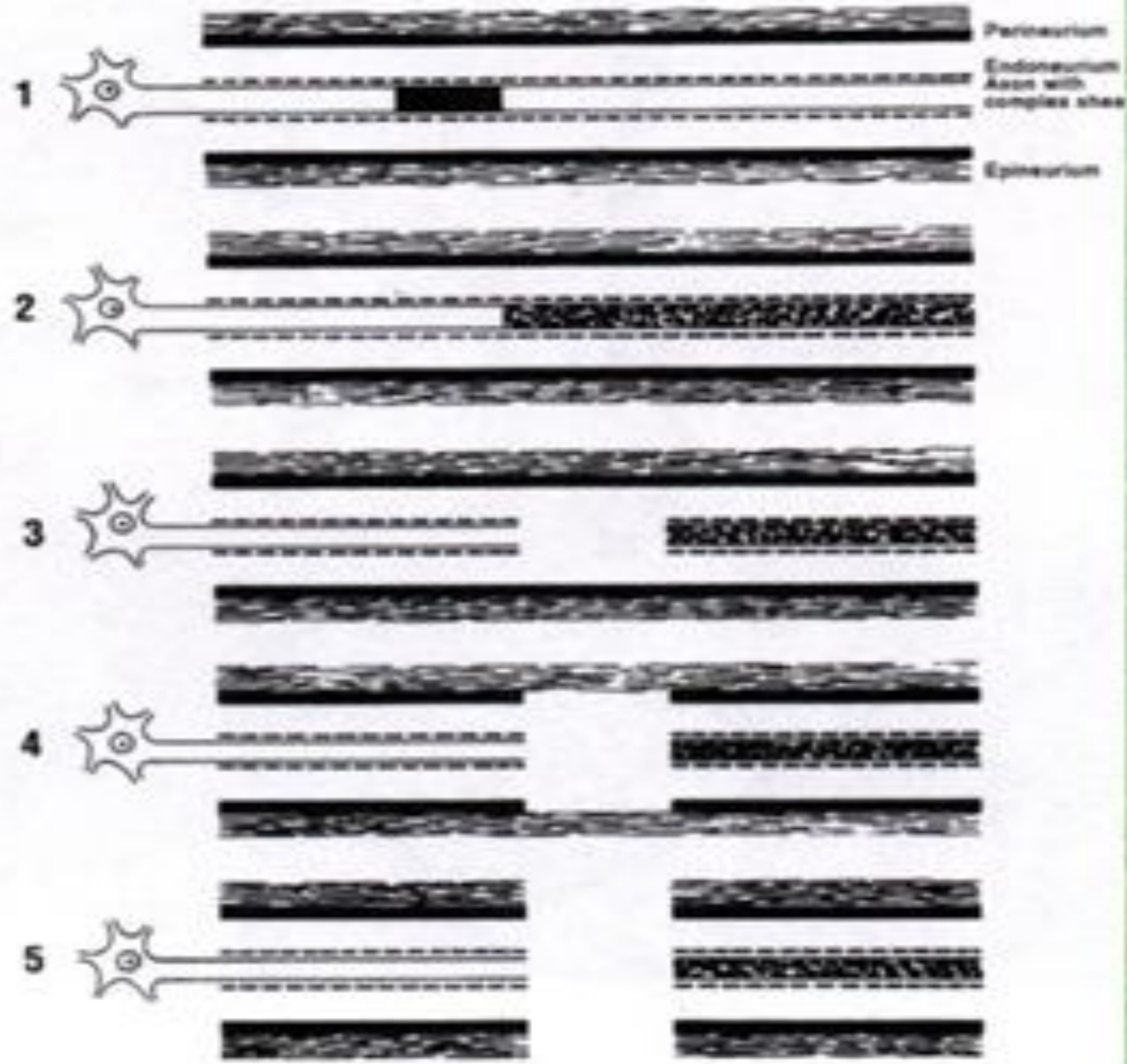
1. Minimal – rapidly reversible conduction block, slowing of nerve conduction (primarily affects FF fibers)

2. Intermediate – focal demyelination w/o axonal damage, prolonged conduction block

3. Severe – Axonal damage with wallerian degeneration

Classification of Nerve Injury
SUNDERLAND CLASSIFICATION

* Each degree of injury suggesting a greater anatomical disruption with its correspondingly altered prognosis
* Anatomically various degrees (1st – 5th) represent injury to
  - - Myelin
  - - Axon
  - - Endoneurial tube and its content
  - - Perineurium
  - - Entire nerve trunk
* Sixth degree (Mackinson) or mixed injuries occur in which a nerve trunk is partially severed and remaining part of trunk sustains 1st to 4th degree injury
* Mixed recovery pattern depending on degree of injury to each portion of nerve


Perineurium
Endoneurium
Axon with
complex shea
Epineurium
1
2
3
4
5

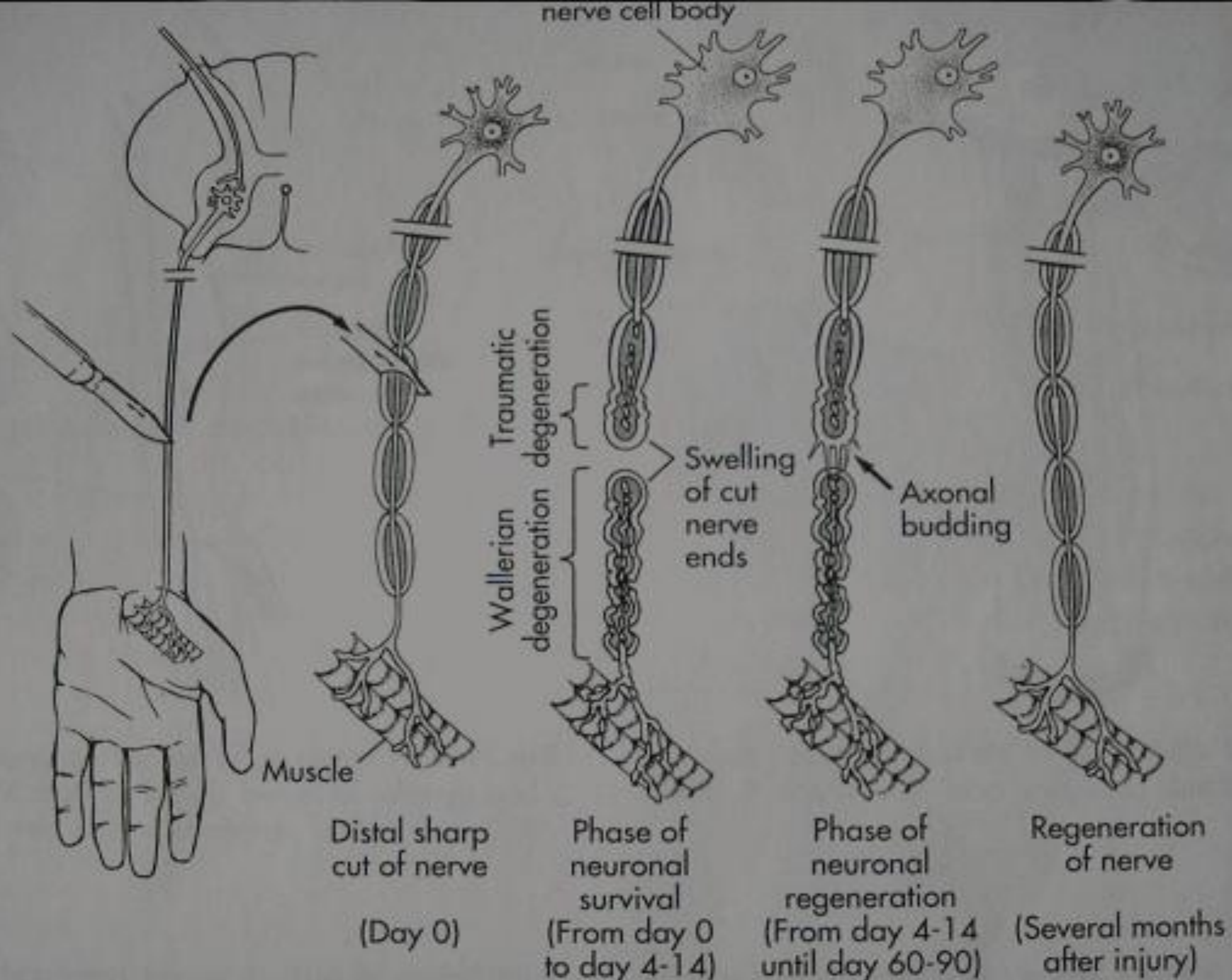
Neuronal degeneration and regeneration

-> Any part of neuron detached from its nucleus, degenerates and is destroyed
by phagocytosis
* Distal - Secondary/ wallerian degeneration
* Proximal- Primary/ Traumatic/ Retrograde degeneration

-> Time required for degeneration varies between sensory and motor fibers and is also related to size and myelination of fibers

-> *Advancing Tinel sign* and presence of *motor march phenomena* are signs of regeneration

Physiological changes in regeneration of Peripheral motor nerve axon after division with sharp object



<u>Seddon classification</u> - based on amount of nerve injury

1. Neuropraxia (mild conduction block)
2. Axonotmesis (axon disruption with intact endoneurium
3. Neurotmesis (axon disruption with loss of endoneurium)

1. Neuropraxia

-“ Conduction block”
- No axonal degeneration
- Large myelinated fibers more susceptible to compression, ischemia (motor)
  . Nerve conduction is normal distally, but altered <u>across</u> “ injury” site
  . Needle EMG shows <u>decreased recruitment</u>, but no abmormal spontaneous potentials. Normal conduction returns in days /weeks (due to re-myelination of damaged segment)

Axonotmesis

* Axon damage with preservation of endoneurium , Perineurium and epineurium
  - Etiology – compression, traction

* Wallerian degeneration of axon
  - motor NCS lost day 4-7 (NMJ fragmentation)
  - sensory NCS lost day 8-10

* Preservation of endoneurium allows for regeneration with re-innervation
  - Recovery time dependent on distance for re- innervation

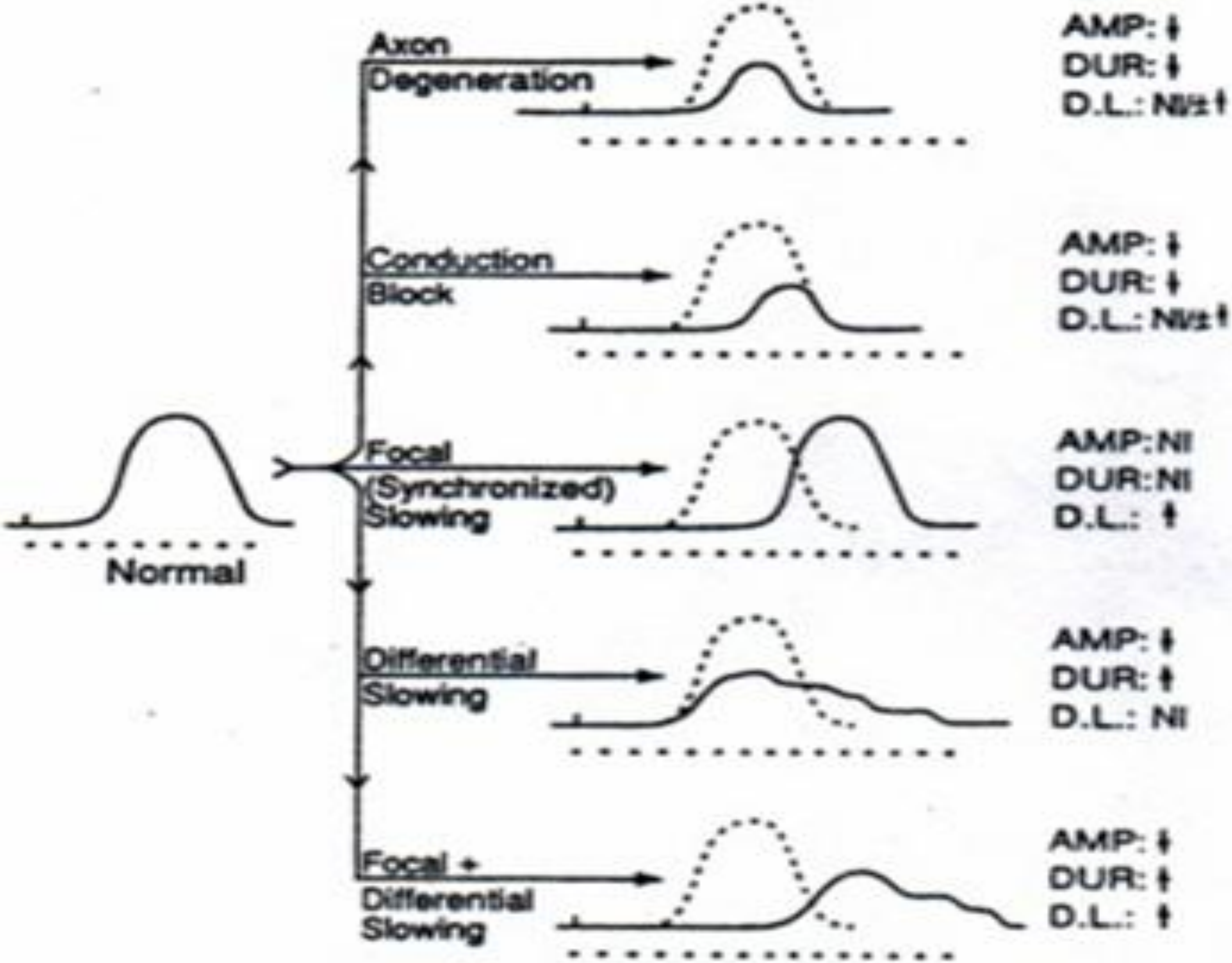
* Axonotmesis

- Day 0-3 : same as Np
- Day 4-7 : decreased motor amplitude
- Day 8-10 : decreased sensory amplitude
- Day 10-14: abnormal spontaneous potentials on EMG (PSW, Fibs)
- Month 6-12: " nascent potentials (S > M) & "jitter"

Neurotmesis

* Disruption of axon, endoneurium and connective tissue (perineurium & epineurium)

*Poor prognosis for re-innervation


Axon
Degeneration
AMP: ↓
DUR: ↓
D.L.: Nl/± ↑
Conduction
Block
AMP: ↓
DUR: ↓
D.L.: Nl/± ↑
Focal
(Synchronized)
Slowing
AMP: Nl
DUR: Nl
D.L.: ↑
Normal
Differential
Slowing
AMP: ↓
DUR: ↑
D.L.: Nl
Focal +
Differential
Slowing
AMP: ↓
DUR: ↑
D.L.: ↑

EDX findings (cont)

- * Performing EDX too early may lead to misleading
- information (wait 2-4 weeks)
- * An early sign of axonotmesis is decreased CMAP
- amplitude
- \- > 30-40% lower than contra lateral side
- * Repeat in 2 weeks

DIAGNOSIS OF PERIPHERAL NERVE INJURIES

* HISTORY

- Which nerve ?
- What level ?
- What is the cause?
- What degree of injury?
- Old or fresh injury?

DIAGNOSIS OF PERIPHERAL NERVE INJURIES

1. Motor

- * All muscles distal to the injury-paralyzed and atonic
- * atrophy: 50-70% in 1st two months
- * striations & motor end plate configurations retained for 12-18months (*Critical limit of delay)*

DIAGNOSIS OF PERIPHERAL NERVE INJURIES

2. SENSORY

* Sensory loss usually follows a definite anatomical pattern, although factor of overlap from adjacent nerves may be present

* ***Autonomous zone***

* Weber 2 point discrimination test

* ***Tinel's sign***

3) REFLEX

* Abolishes all reflexes transmitted by that nerve, either afferent or efferent arc.
* Complete and incomplete lesion. So, not a reliable guide to injury severity.

4) Autonomic

* Loss of sweating
* Loss of pilomotor response and
* Vasomotor paralysis in autonomous zone

5) Others:

* Trophic changes
  - Esp. hand and feet
  - skin -thin, glistening, breaks easily to form ulcers that heal slowly

* Finger nails
  - Ridged, distorted and brittle

* Osteoporosis (Reflex sympathetic dystrophy)

Electrodiagnostic Evaluation of Brachial Plexus Injuries

BP lesion localization

* Know clinical ANATOMY!!!
  -Root/trunk/division/cord/branch (RTDCB)
  -Motor/sensory innervation
* Comprehensive Edx eval
  - NCS & needle EMG
  - Consider less common motor/sensory NCS

Edx eval of BP Injury

* Nerve Conduction Studies (NCS)
    common (median, ulnar)
        . (evaluates lower trunk & medial cord)
    less common (radial, MC, Axillary, SS)
    proximal NCS (C5-6, Erbs point)
        .(technically possible, difficult, uncomfortable)

* Needle EMG (recruitment, abnormal spontaneous potentials)

* Late-responses (H-reflex, F wave)- may be abnormal but ? less useful

Motor/Sensory NCS

* Distal latency & NCV are not helpful
* <u>Amplitude</u> is "key" parameter
- remains NL(on distal stim) if no axonal loss (cond block, demyelination) or with preganglionic BPI (SNAP NL)
- look for decreased side-side > 50%
  motor day 4-7 (NMJ fragmentation)
  sensory day 8-10

Localizing NCS involement

* Terminal branches of Brachial Plexus
- Median, Ulnar, Radial, Axillary, MC
- sensory & motor

* travel to and from the CNS thru the various roots, trunks, divisions & cords in a fairly consistent "pattern"

Sensory NCS Localization

Nerve	Cord	Trunk
Musculocut.	Lateral	Upper
Median (1)	Lateral	Upper
Median (2-3)	Lateral	Middle
Radial	Posterior	Upper
Ulnar	Medial	Lower

Motor NCS Localization

Nerve	Cord	Trunk
Musculocutan	lateral	upper
Axillary	posterior	upper
Suprascapular	---	upper
Radial	posterior	middle
Median	medial	low
Ulnar	medial	lower

Needle EMG

* Abnormal spontaneous potentials
    - positive sharp waves, fibrillations
    - 7-10 days (paraspinal), 2-4 weeks (distal m's)
    - Important: follow "pattern" of BP innervation
* Paraspinal M's WNL! (distal to Post rami)
* Decreased recruitment (voluntary MUAP)

Adjunctive tests

* Xrays (C-spine, clavicle, humerus, 1st rib)
* Myelography - w/i 2-3 weeks, <u>nerve root avulsion</u> forms diverticulum c/w SA space
* MRI (>CT)

Somatosensory Evoked Potential (SSEP)

* Supraclav. Fossa / Erbs pt. (N9) / cervical spine (N13) / contra somatosensory cortex (N19)
* Sensory fibers / post column / thalamus
* Considerations (less than ideal agreement)
  -Postganglionic-N9 Abnl (> 30% side-side diff.)
  -Preganglionic- Nl N9 w/ Abnl N13

Axon reflex testing

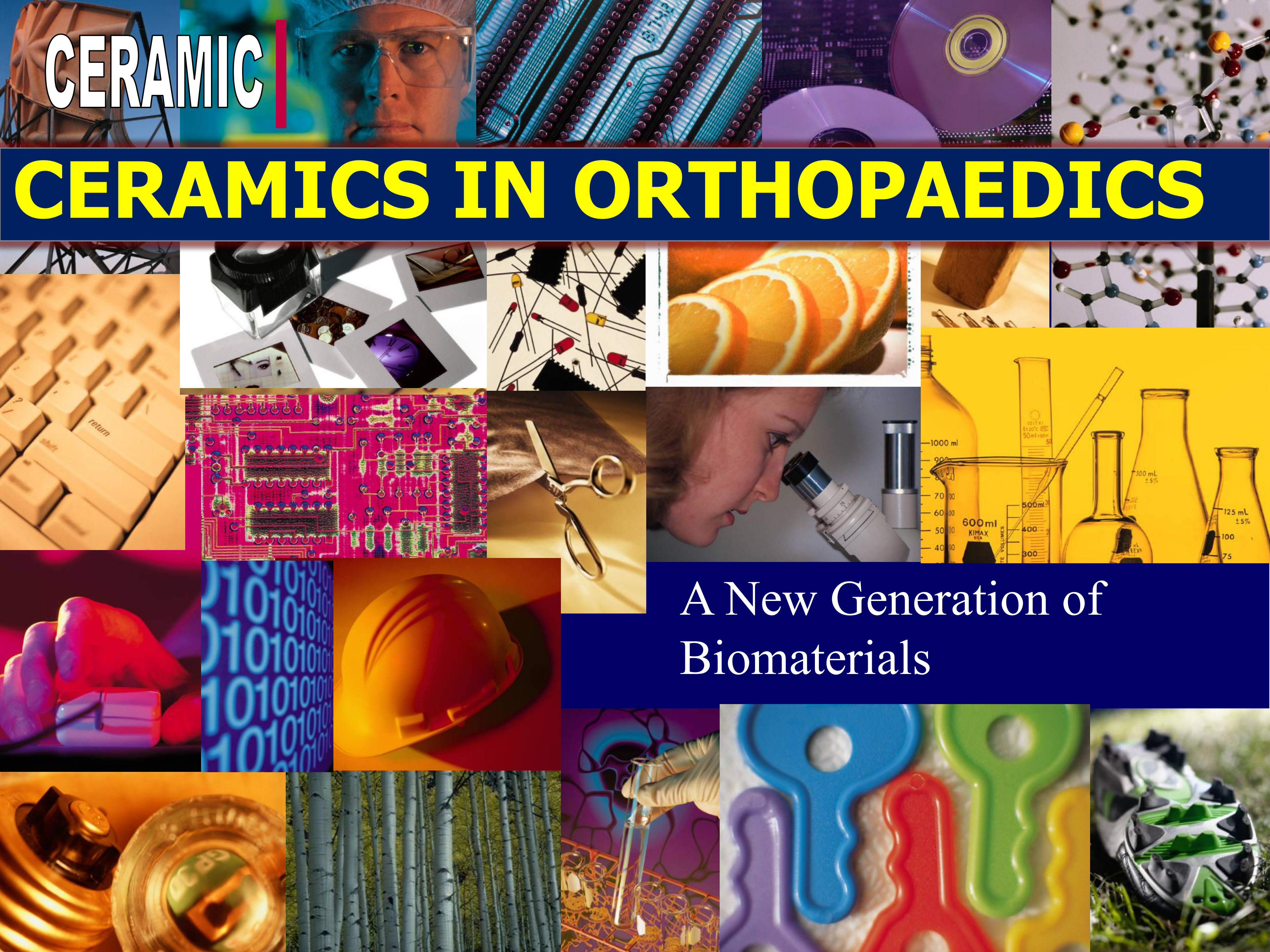
* To evaluate pre vs post ganglionic lesion
* 1% SQ histamine normally leads to a vasodilation, wheal & flare due to reflex between DRG & cutaneous receptors
  - "Triple response" in light of clinical picture c/w BPI = lesion proximal to DRG (ie: preganglionic root avulsion & poor prognosis)
  - Loss of flare = postganglionic (better prognosis)

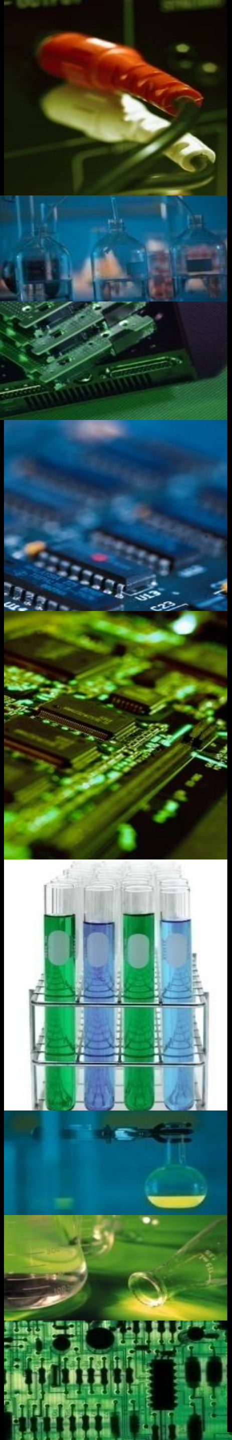
Brachial Plexus Injuries (Summary)

* Know your ANATOMY!!!

* Needle EMG:
  - localizing pattern of involement
  - paraspinal m's WNL (unless preganglionic)

*NCS:
  - localizing pattern of involvement
  - amplitudes often most affected


CERAMIC
CERAMICS IN ORTHOPAEDICS
A New Generation of Biomaterials



CERAMICS IN ORTHOPAEDICS

Prof M. Shantharam Shetty

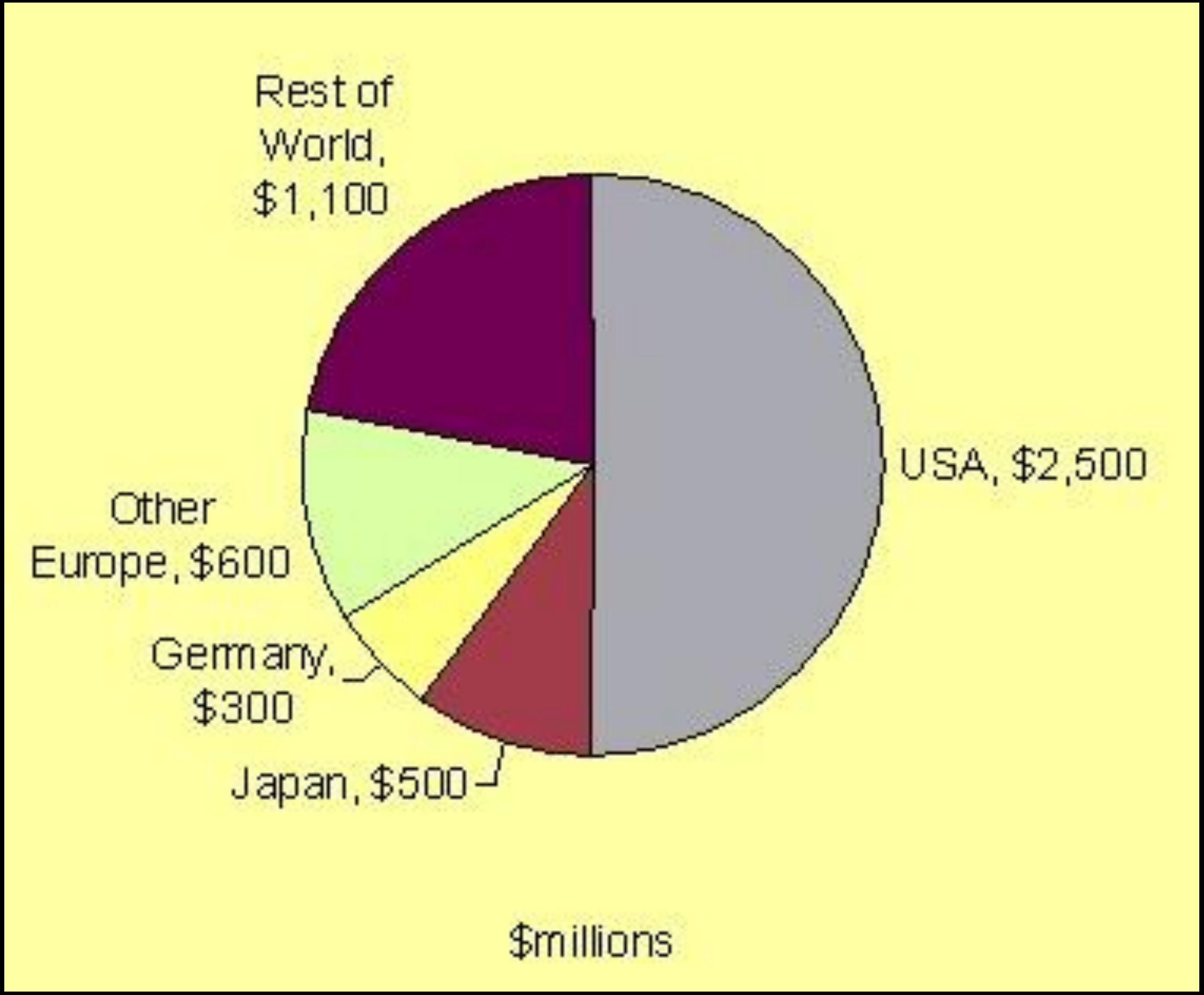
Pro Chancellor, NITTE UNIVERSITY

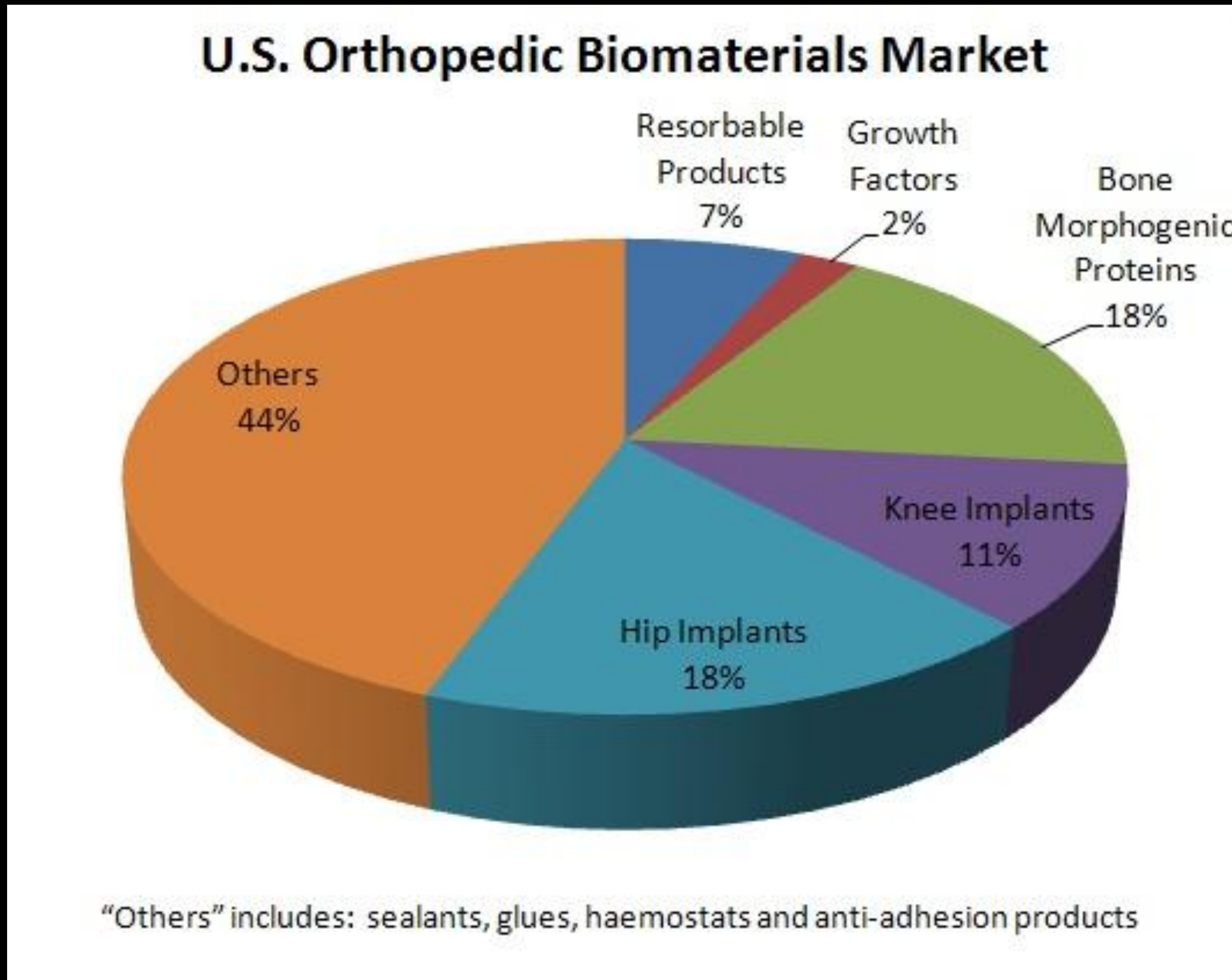
&

Chairman ,Tejasvini Hospital & SSIOT

Mangalore

Mangalore Orthopaedic Course – 15th June 2013

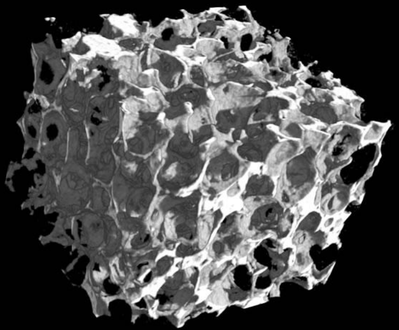

Rest of World, $1,100
USA, $2,500
Other Europe, $600
Germany, $300
Japan, $500
$millions


U.S. Orthopedic Biomaterials Market
Resorbable Products 7%
Growth Factors 2%
Bone Morphogenic Proteins 18%
Others 44%
Knee Implants 11%
Hip Implants 18%
"Others" includes: sealants, glues, haemostats and anti-adhesion products

Biomaterials in orthopaedics

Material that interacts with human tissue and body fluids to treat, improve, or replace anatomical element(s) of the human body.

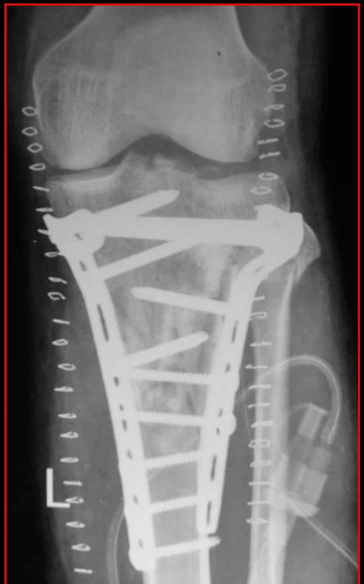
Biomaterial devices used in orthopaedics are commonly called *implants*.



Biomaterial Types

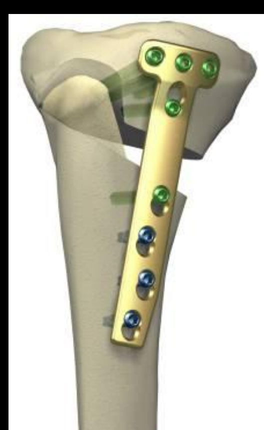
Two main groups:

Metals and Non-metals



Metals

- **Low carbon grade austenitic stainless steels: 316L**
- **Titanium and titanium-base alloys: commercially pure titanium (CP Ti), Ti-6Al-4V, and other.**
- **Cobalt alloys: Co-Cr-Mo, and other.**



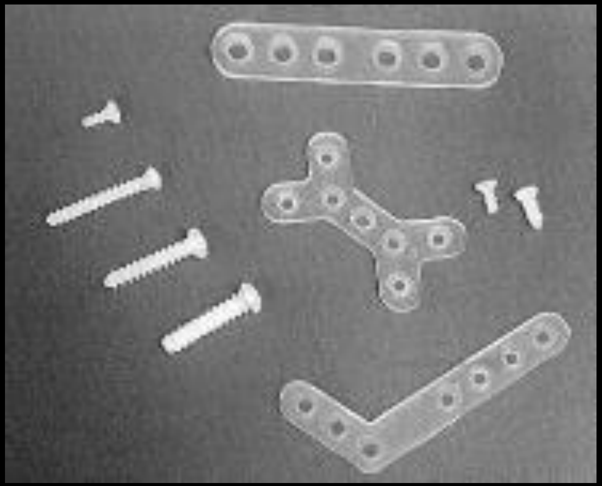
Nonmetals

Three main subgroups :

polymers, ceramics, and composites.

Polymers

- Ultrahigh molecular weight polyethylene (UHMWPE)
- Acrylic bone cements
- Thermoplastic polyether ether ketone (PEEK)
- Bioabsorbables



Composites

Filler(reinforcement) added to a matrix material in order to obtain properties that improve every one of the components.

May have several phases.

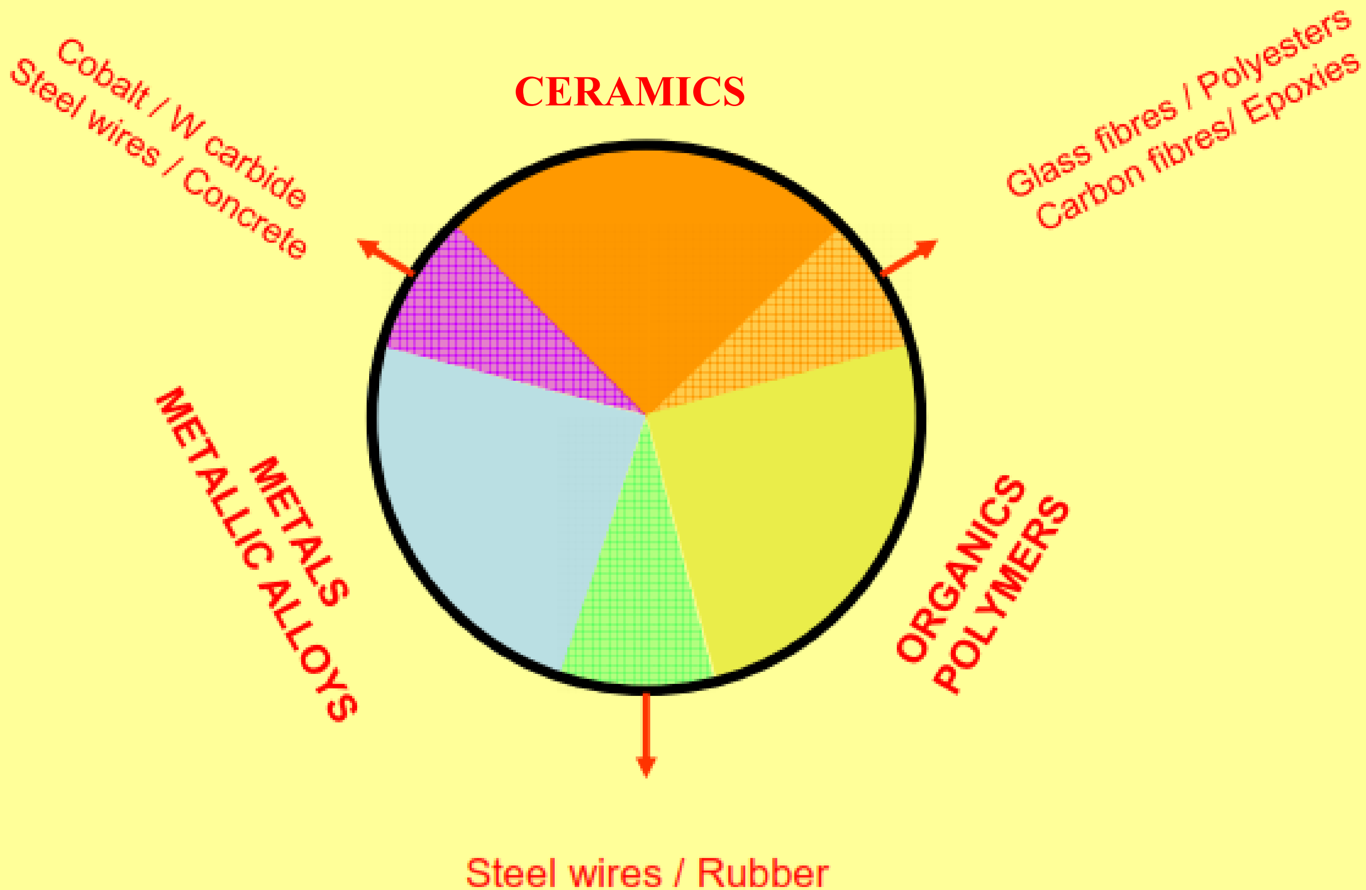
Polymers containing particulate fillers are known as particulate composites.

Ceramics

(keramikos- pottery in Greek)

ALMOST ALL CIVILISATIONS IN WORLD HAD CERAMICS IN THEIR ARMAMENTARIUM

3 classes of materials



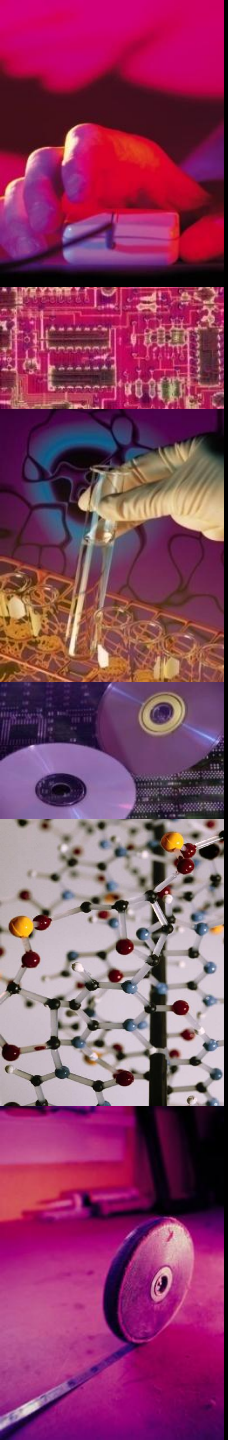
- **“Total hip replacement is the most rewarding operation in the history of medicine. Keep it safe, effective and durable” - 2010**

– Richard H. Rothman, MD, PhD(USA)

- **Bearing Couples – The Key to Success?!-2011**

- **“The Ceramic bearings have superb outcomes in 10-year studies, and the results are better than any other bearings in young patients” - 2012**

– Kyung-Hoi Koo (Korea)



- “The advent of multi-bearing cup and questions regarding the long-term durability of highly cross-linked polyethylene will continue to keep ceramic-on-ceramic bearings as a very viable option for younger and active patients” -2012

  – Jonathan P. Garino (USA)

- “The current study shows that ceramic-on-ceramic THAs in the young patient population have a very low revision rate with absence of wear or osteolysis for uncemented stems”.

  – Steppacher et al (USA) in Semin Arthro 2012:22:252

Bearing Surfaces

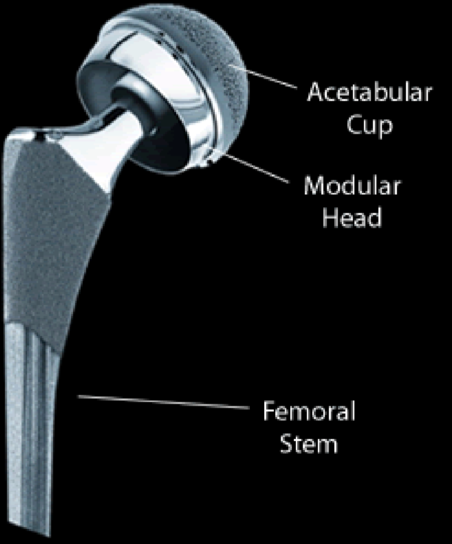
Metal on Polyethylene

- metal (cobalt-chrome) femoral head on polyethylene acetabular liner
- benefits
  - longest track record of bearing surfaces
  - lowest cost
  - most modularity
- disadvantages
  - higher wear and osteolysis rates compared to metal-on-metal and ceramics
  - smaller head (compared to metal-on-metal) leads to higher risk of impingement (smaller head:neck ratio)

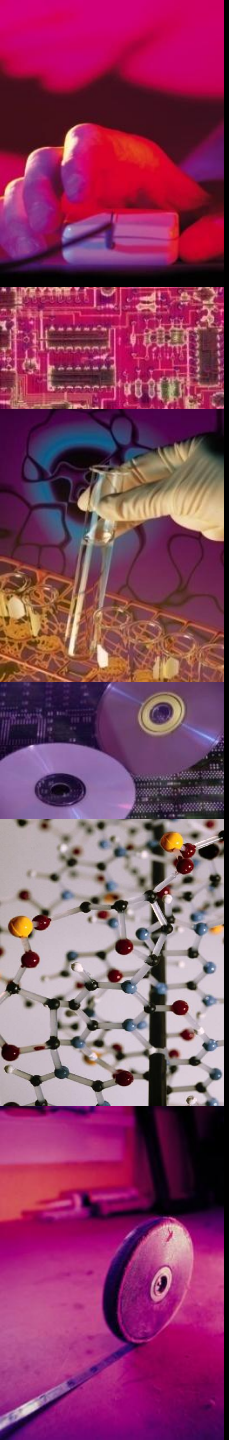
Metal-on-Metal

- **Benefits**
  - better wear properties (lower linear wear rate and volume of particles) than metal on poly,
  - larger head allows for increased ROM before impingement (large head:neck ratio)

Metal-on-Metal

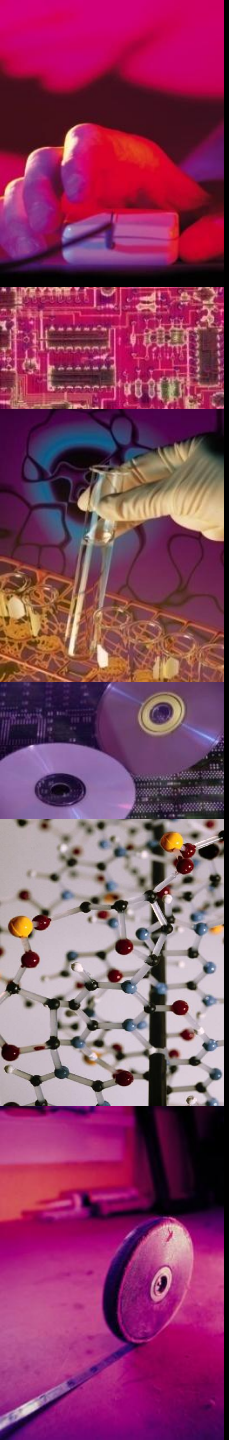


- **Disadvantages**
  - more expensive than
  - metal-on-poly
  - increased metal ions in serum and urine (5-10x normal)
    - serum metal ion concentration highest at 12-24 months
      - correlates with the initial "wear in" or "run-in" phase of increased particle generation, but then followed by a "steady state" phase of decreased particle generation
    - no proven cancer link
  - contraindicated in pregnant women, persons with renal disease, and those with metal hypersensitivity due to metal ions
  - formation of pseudotumor



Ceramic on Ceramic

- Benefits
  - best wear properties of all bearing surfaces
  - lowest coefficient of friction of all bearing surfaces
  - inert particles, no concern for cancer risk



Ceramic on Ceramic

- Disadvantages
  - more expensive than metal-on-poly
  - worst mechanical properties (brittle)
  - reports of squeaking in certain types of ceramic THA
  - less modularity (fewer neck length options)
  - stripe wear - caused by contact between the femoral head and rim of the cup during partial subluxation, results in a crescent shaped line on the femoral head

First introduced in orthopaedics by Pierre Boutin in early 1970s

CERAMIC

Linear wear rate 4000 times lower than that of metal-on-polyethylene

Excellent frictional characteristics

5 types

- Glass
- Plasma sprayed polycrystalline ceramic
- vitrified ceramic
- Solid state sintered ceramic
- polycrystalline glass-ceramic

Ceramic is processed by mixing particulates of material together with water and organic binder

Then mould desired shape, dried to evaporate water and binder burned out by thermal treatment

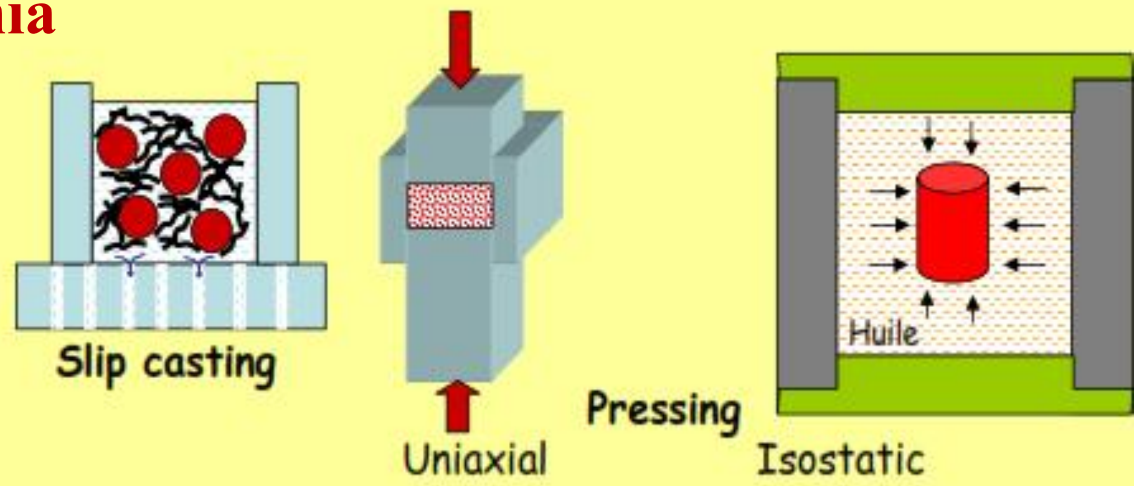
Firing or sintering at a very much higher temperature then densifies the residual material.

What is a ceramic ? Processing

Alumina – Zirconia

Ceramic powder

↓ Compaction:



Green ceramic

↓ Thermal treatments:

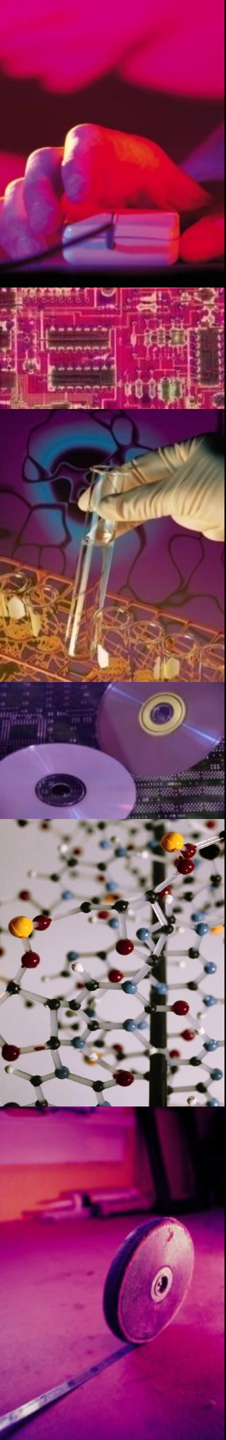
Debinding → Burn organic compounds

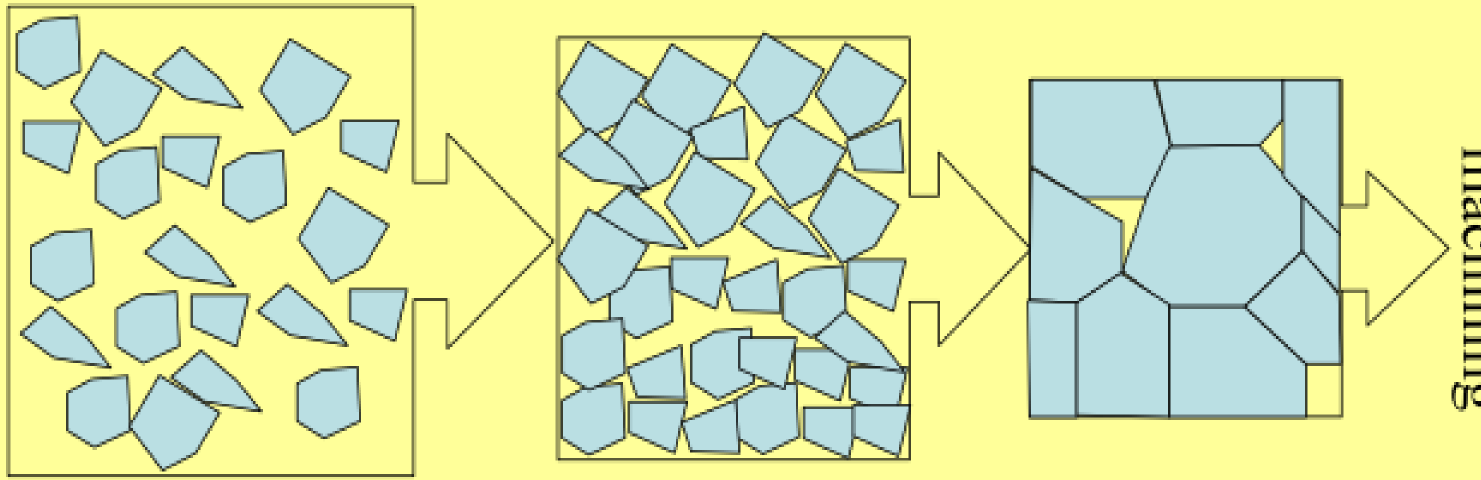
Sintering → Densification, Grain growth

Sintered ceramic

↓ Machining , polishing

Final piece





Powder (suspension).
synthetic (tech. c.)
Natural ore (trad. c.)

Green product
(after pressing)
Weak bonds
between powder
grains.

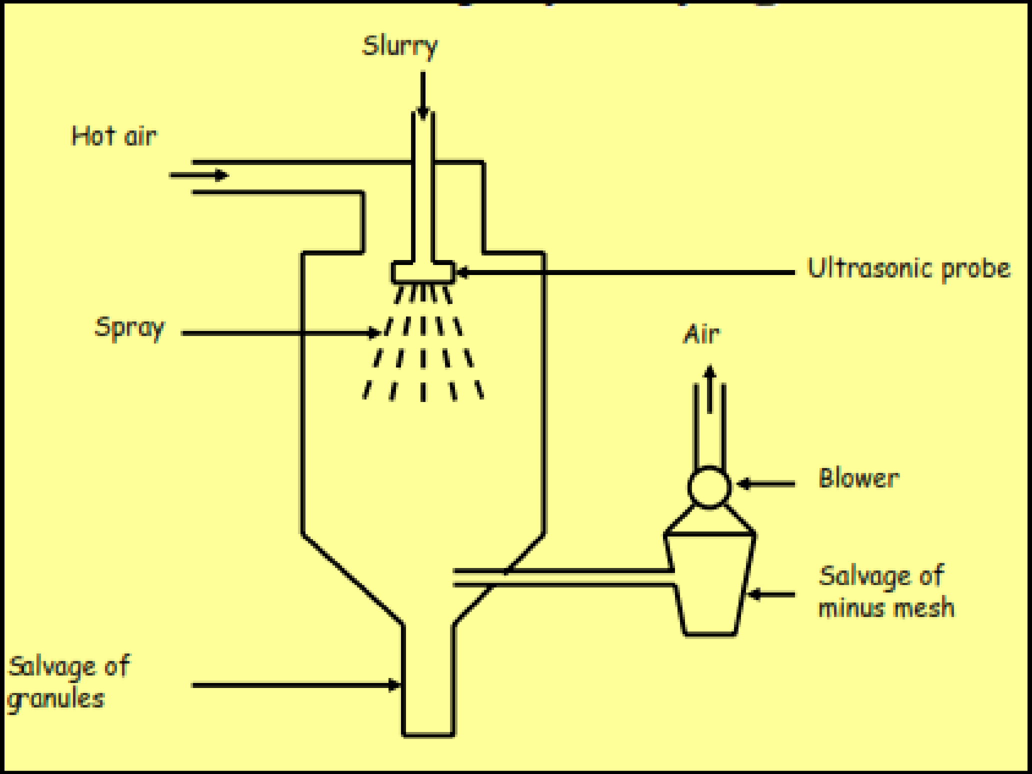
Sintered piece
diffusion in solid
state (~ 1500°C)

Example: Processing of alumina-zirconia composite femoral heads ….

- *Powder preparation*

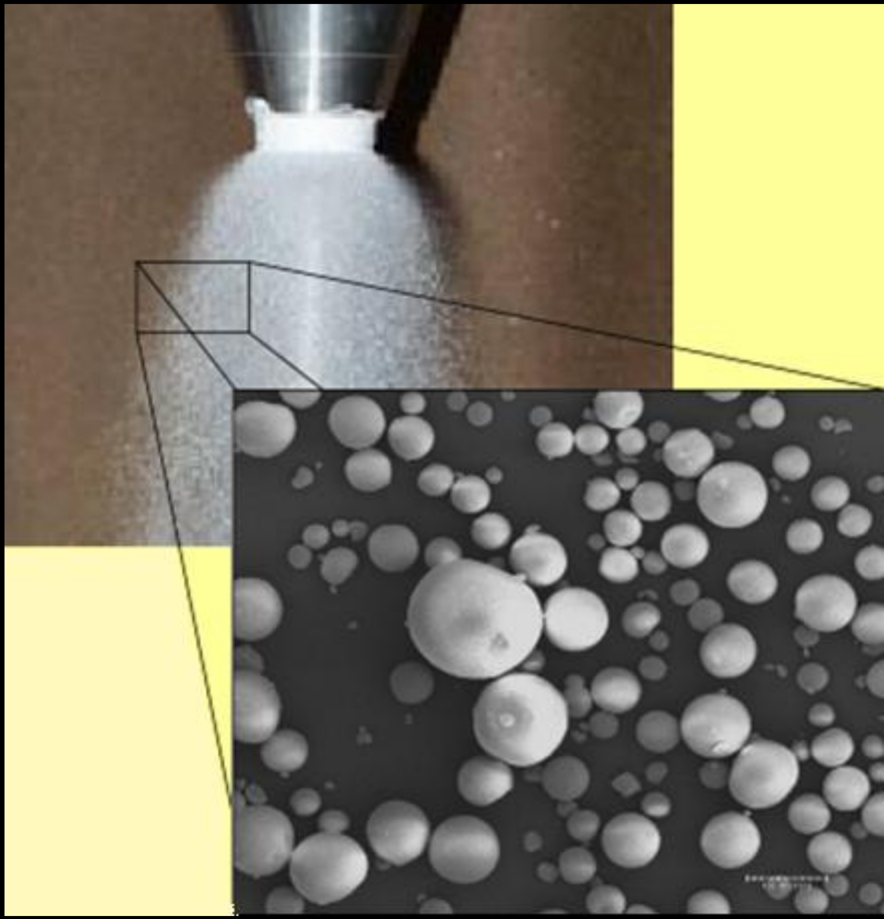
- *Slurry Preparation*

Spray-drying of Ceramic Powders



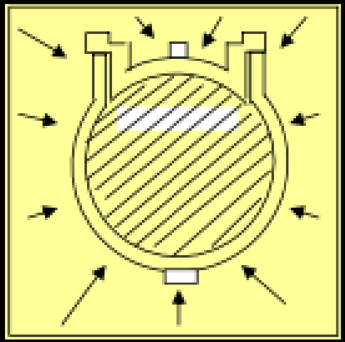
Powder Preparation

Spray Drying



Forming and Sintering

Cold isostatic Pressing



Sintering

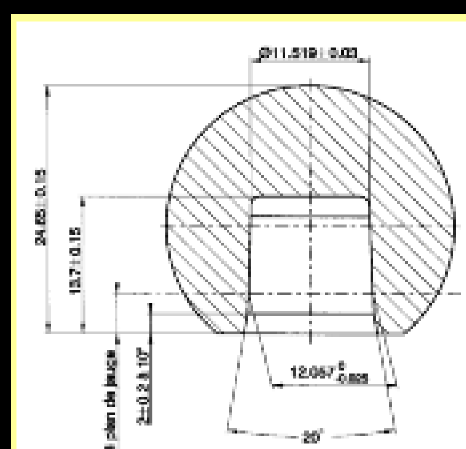
Hot Isostatic Pressing and Whitening

HIP, Whitening

Polishing and machining

Polishing

Grinding of the cone



Wear is the main issue in Orthopaedics

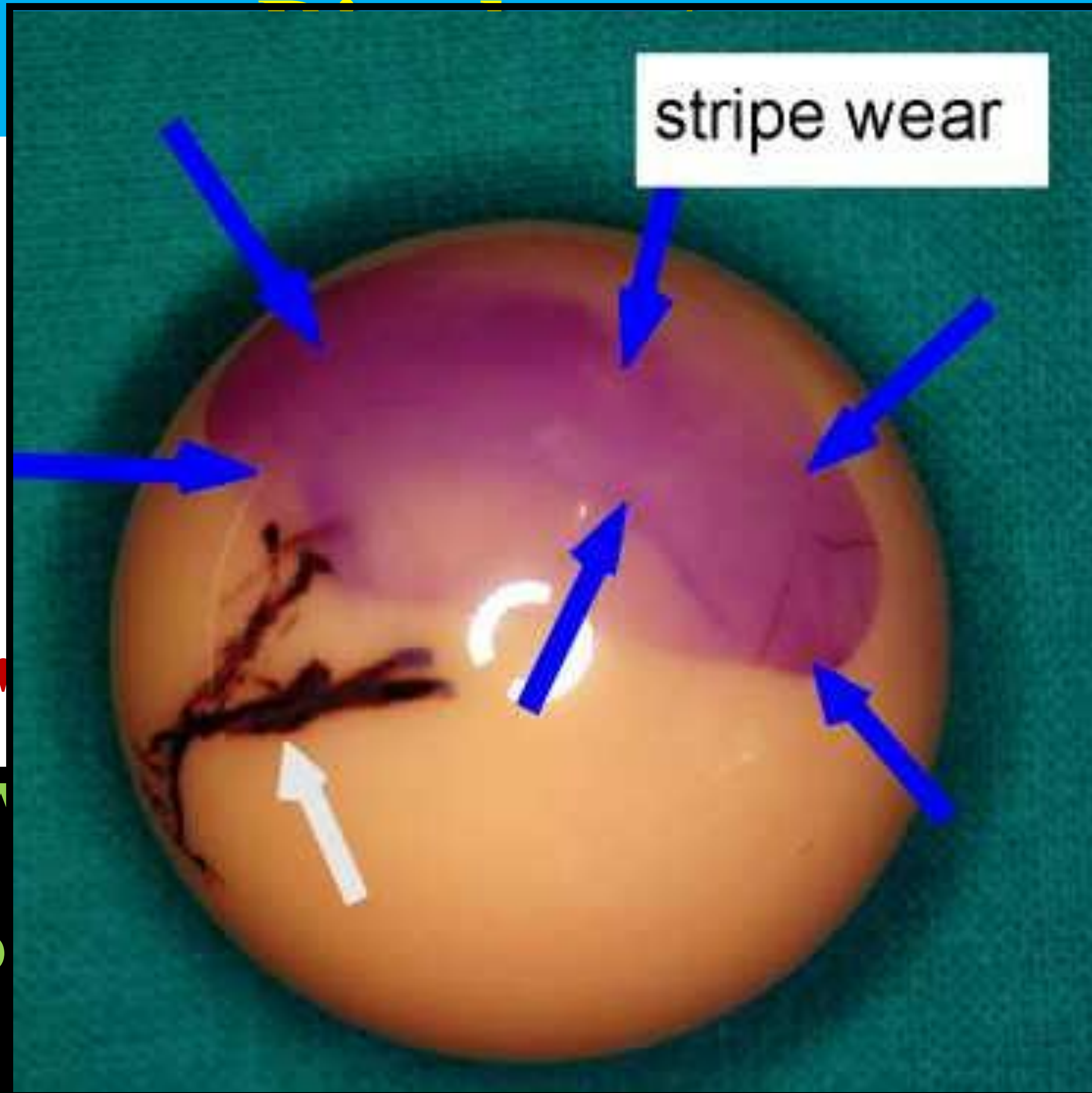
The major advantage of Ceramics: low wear debris generation

PROPERTIES

- Refractory polycrystalline

Best wear characteristics with PE (0.5 to 2.5 μ per component per year)

- Good aesthetic appearance



" ... gs

– Low

– Poo

Implant Couple	Squeak Prevalence (%)
Trident cup with Omnifit stem	
Walter et al[5]	0.54
Capello et al[16]	0.75
Manley[17]	0.46
Trident cup with Accolade stem	
Restrepo et al[18]	2.70
Jarrett et al[1]	11.00
Christensen & Jacobs[7]	7.70

The major drawback of Ceramics: Risk of Fracture in vivo

Reminder: failure in vivo is not acceptable

In Vivo ceramic head fracture

Aluminum Oxide (Al_2O_3)

- The Most Well-known Oxide Ceramic Material

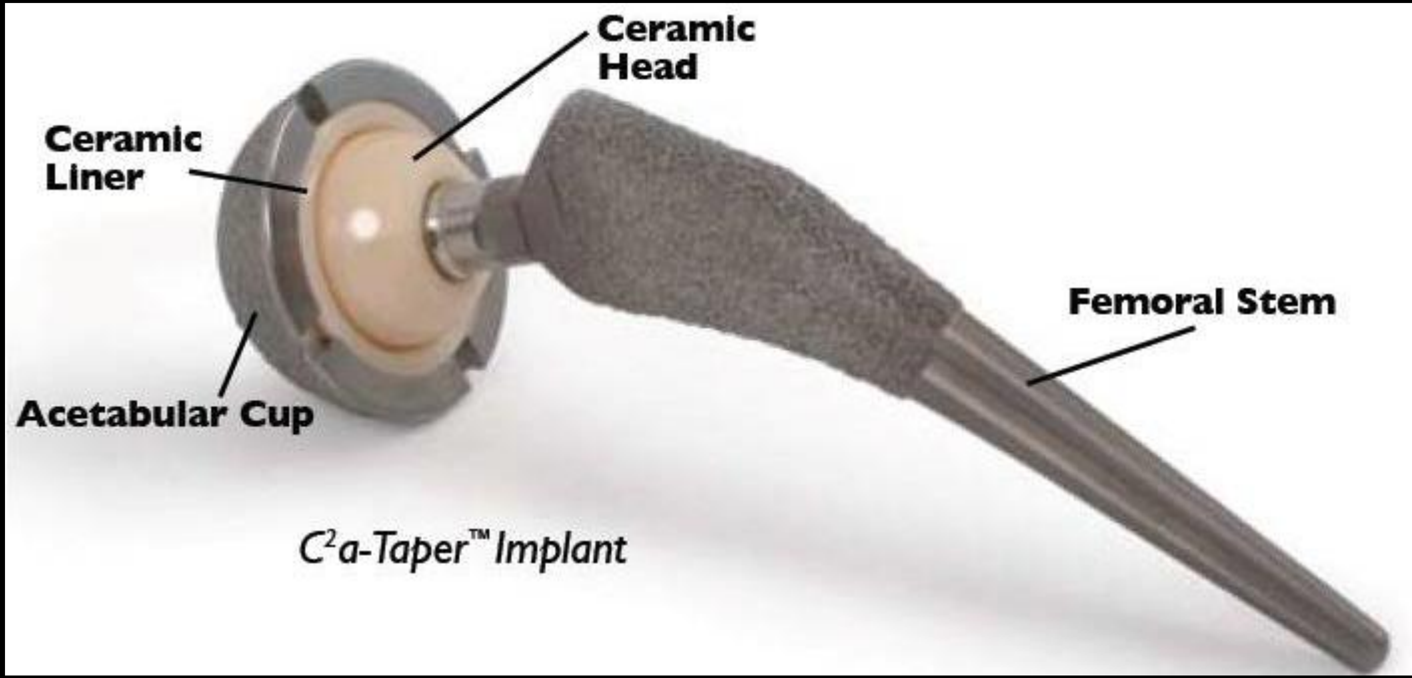
Alumina ceramic

Obtained by sintering alumina powder at temperatures between 1600 and 1800°C

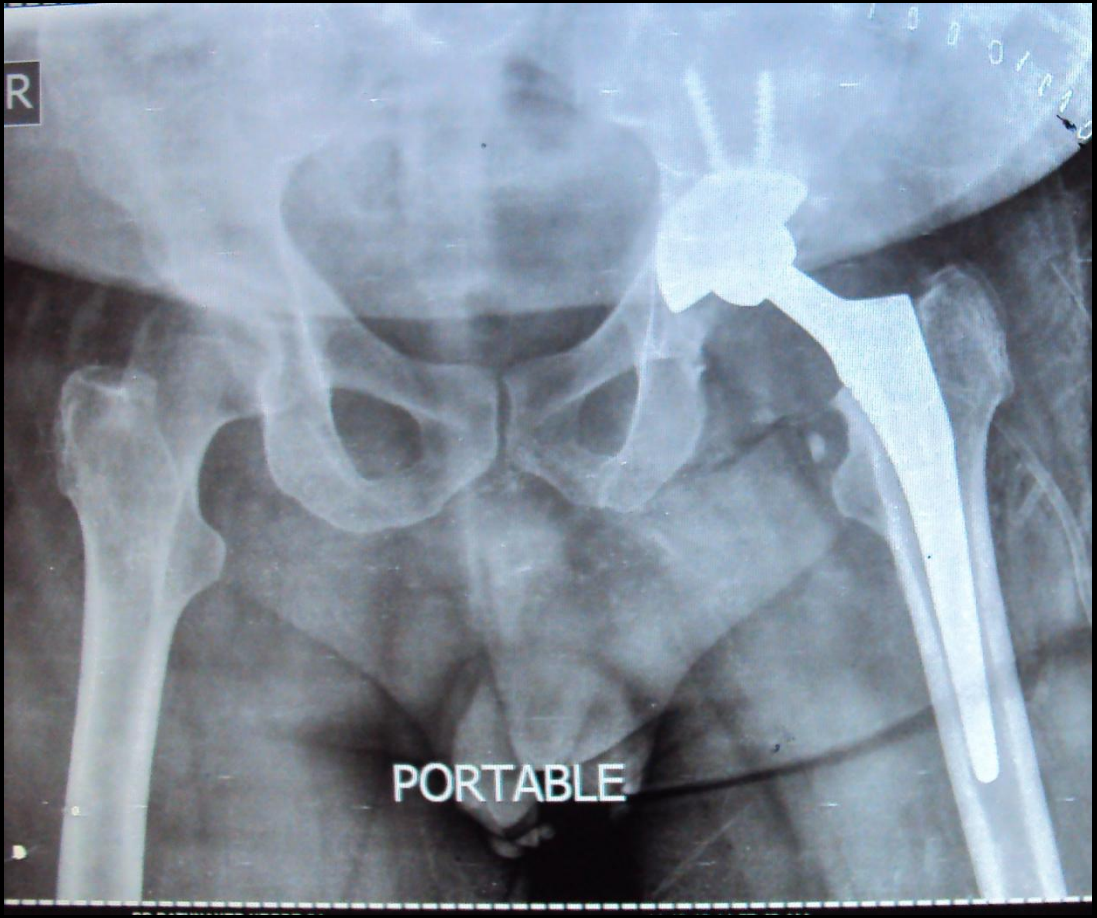
Young's modulus is 300 times greater than that of cancellous bone, and 190 times higher than polymethylmethacrylate (PMMA)

Alumina ceramic

Standardised material since 1984


Ceramic
Head
Ceramic
Liner
Acetabular Cup
Femoral Stem
C2a-Taper™ Implant


R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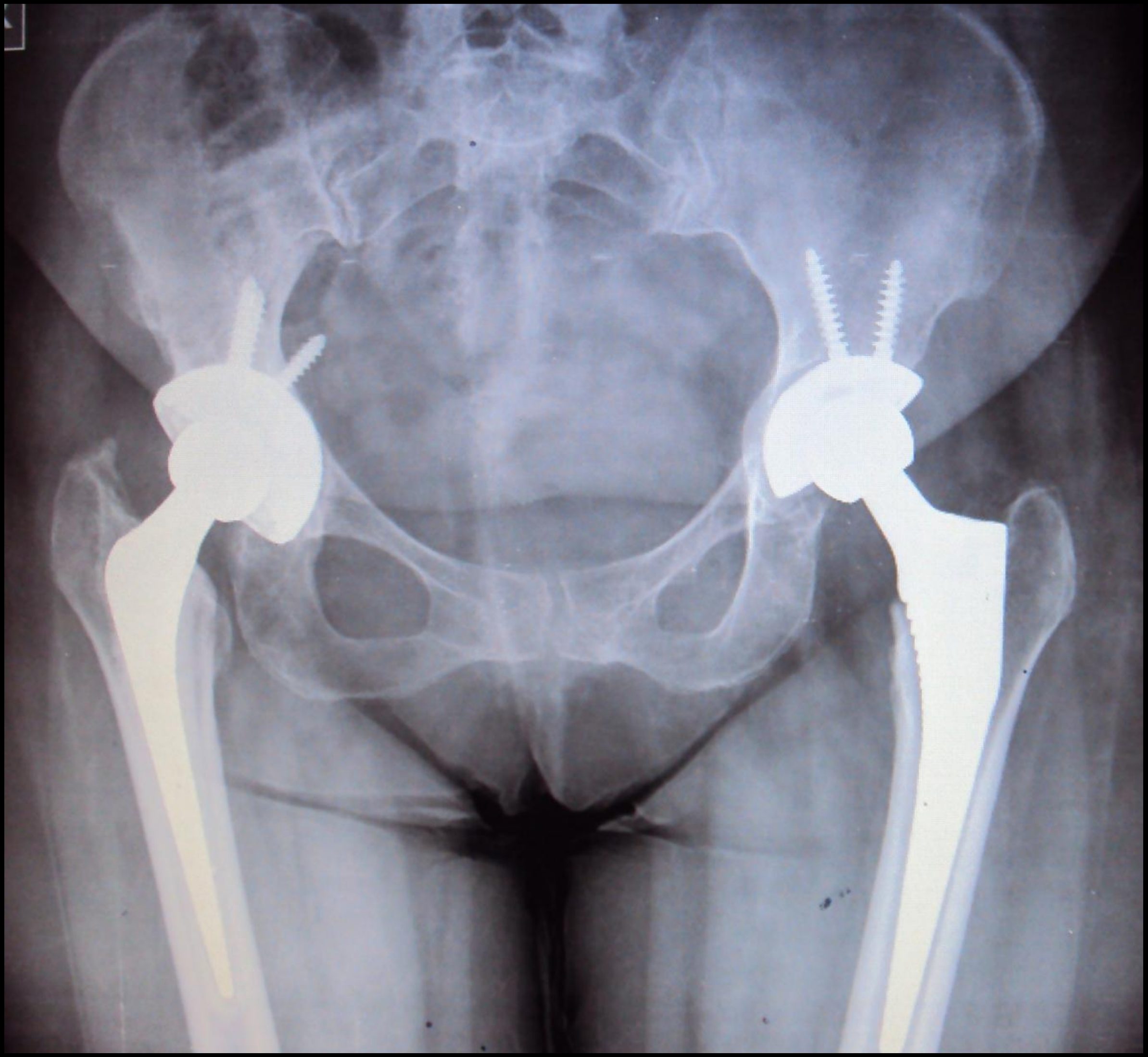
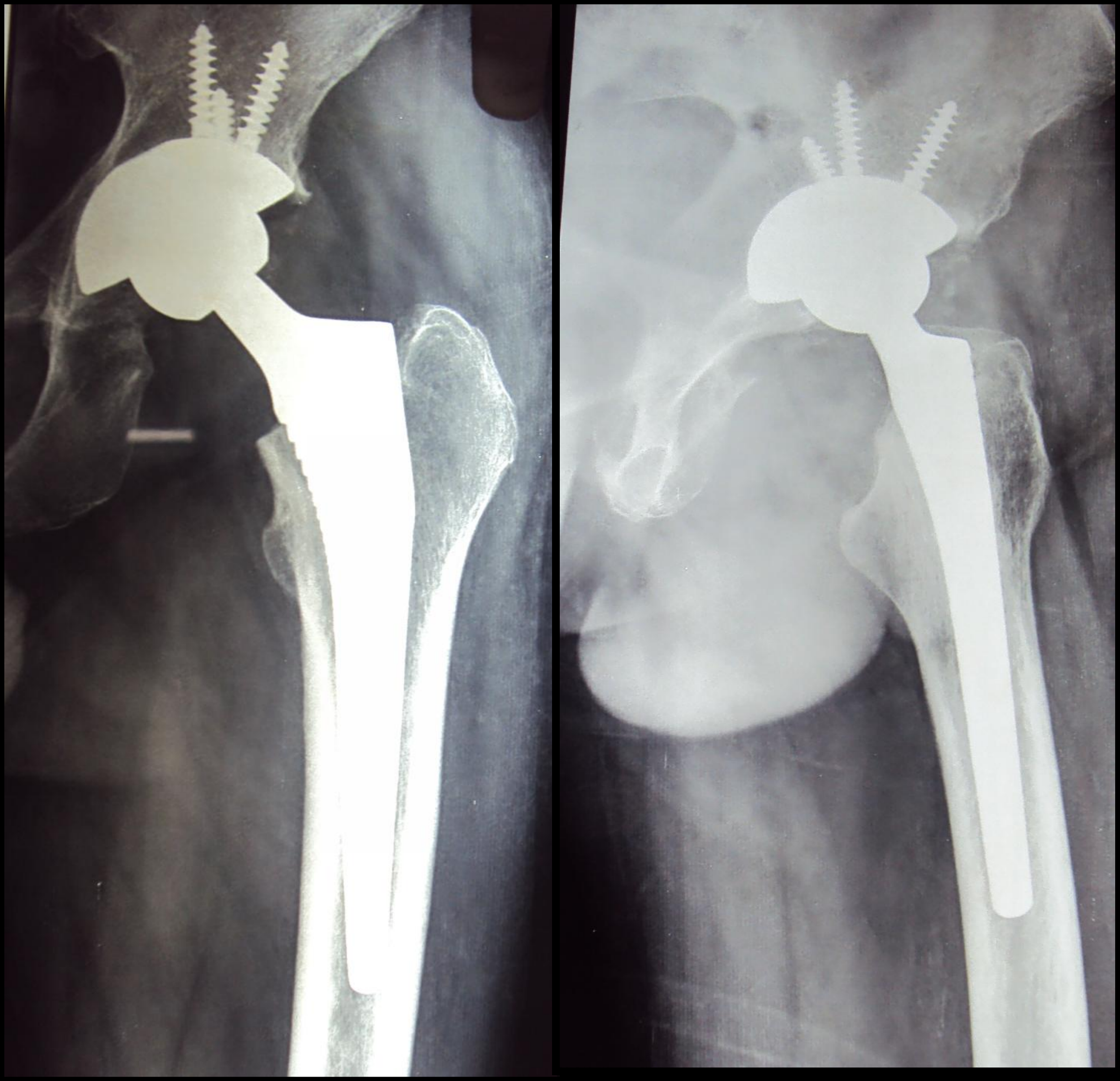
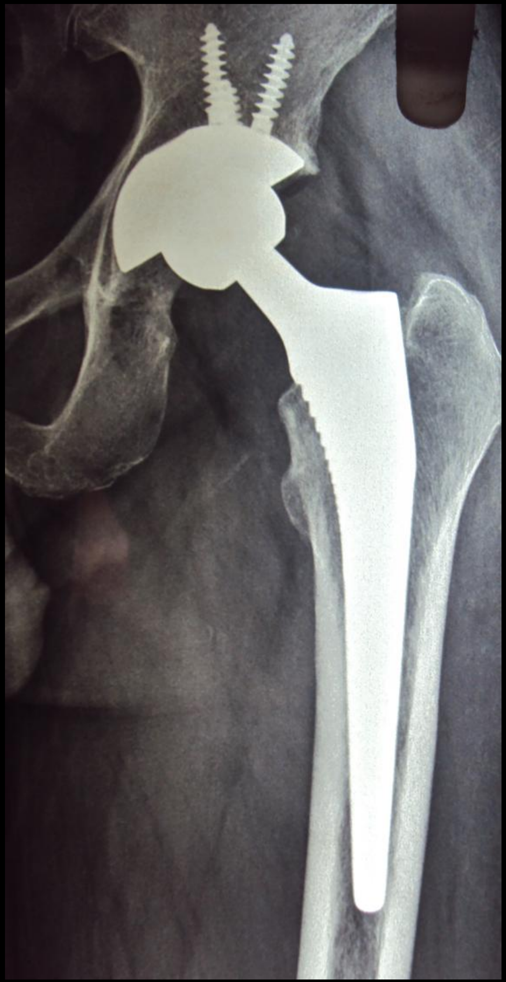
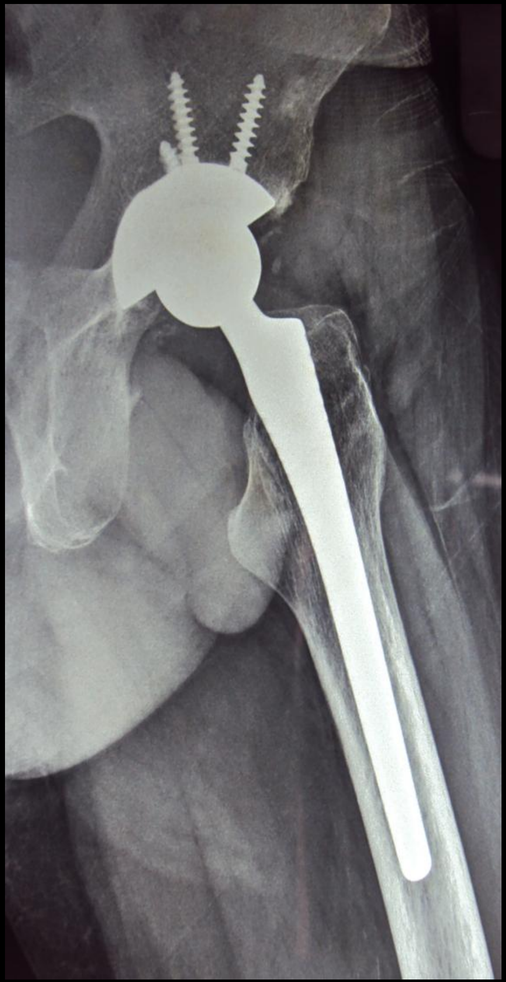
PORTABLE






PORTABLE
15-03-13 11:18:19 AM
Pelvis Both Hips
DR. AJITH KUMAR
TEJASVINI HOSPITAL , SSIOT . MANGALORE .


R
THASLEEMA 22 year(s) Female
17-04-13 4:28:02 PM
Pelvis with Both Hip Joints
DR.AJITH KUMAR
TEJASVINI HOSPITAL & SSIOT, MANGALORE
AGFA

Zirconium Oxide (ZrO_2)

Zirconia ceramic

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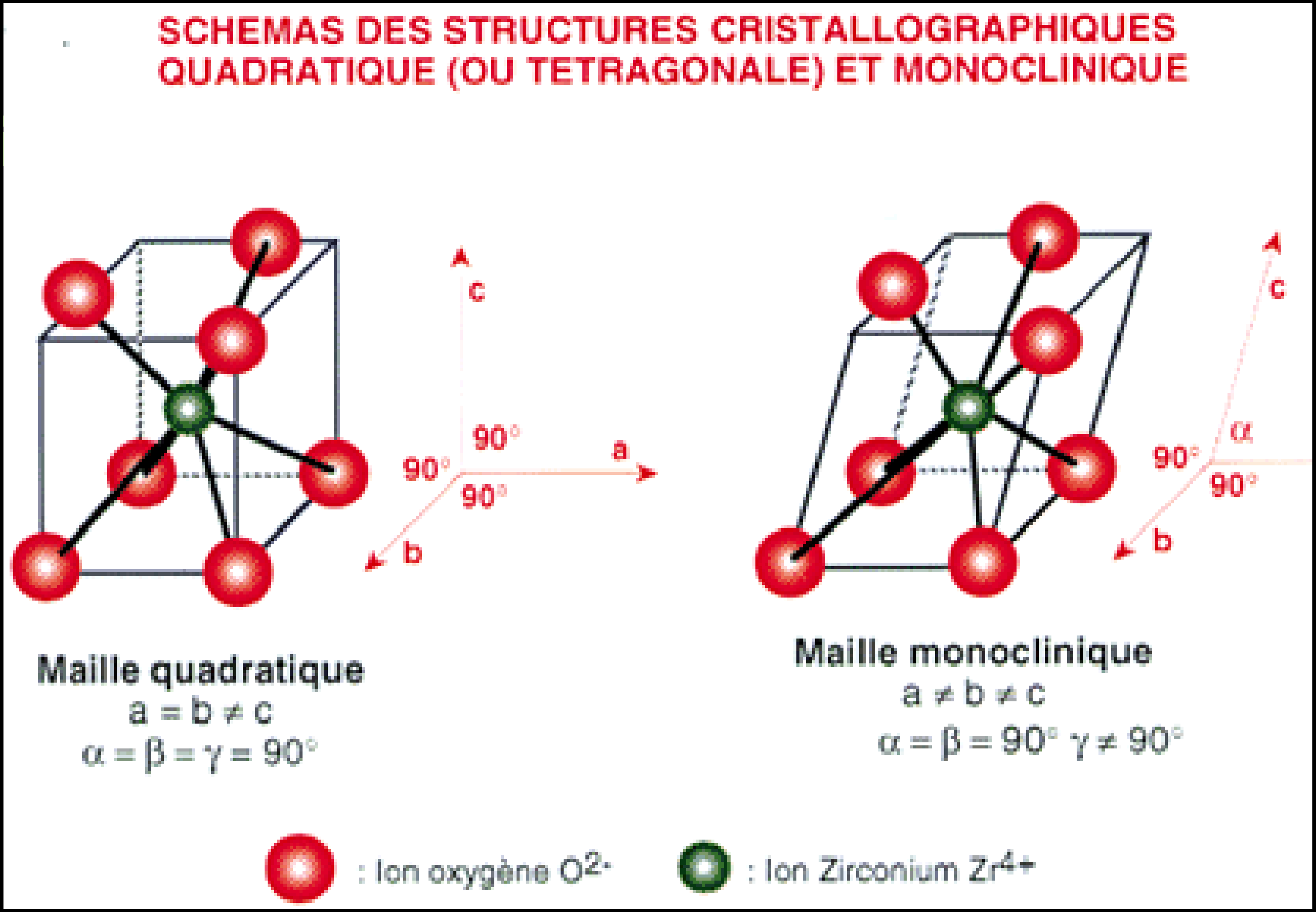


Zirconia ceramic

Yttrium-stabilised tetragonal polycrystalline zirconia (Y-TZP) offers the best mechanical properties.

Zirconia ceramic

Standardised in 1997 (International Standard Organisation, ISO 13356)

Zirconia ceramic

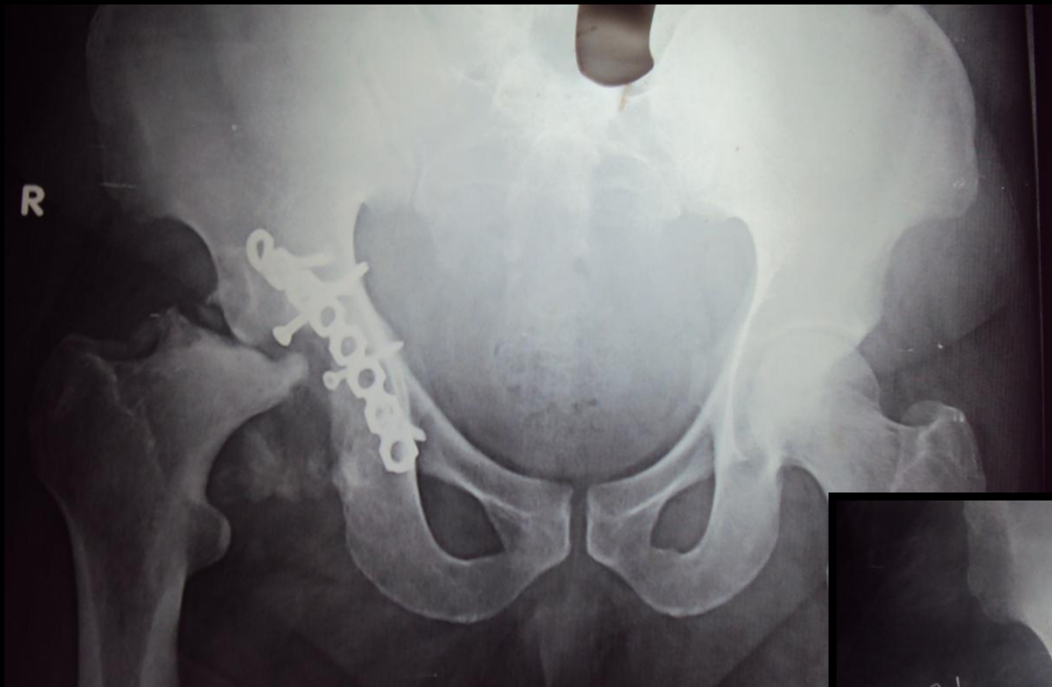
Zirconia femoral heads should articulate only against polyethylene sockets

Zirconia ceramic

Long-term performance may be altered by degradation in vivo with transformation of the material into its monoclinic unstable phase.

- **Very high resistance to crack propagation**
- **Very high thermal expansion, therefore often the material of choice for joining ceramic and steel**
- **Significantly more expensive than alumina ceramics**

Zirconia ceramic



R

Oxinium

Oxinium

- 97.5% zirconium and 2.5% niobium — two most biocompatible metals known —with proprietary process,extreme heat and oxygenation.

- This process yields a 5-micron thick ceramic surface on a core of metal — providing OXINIUM material

Oxinium

- 4900 times more resistant to abrasion than cobalt chrome
- 160 times smoother than cobalt chrome

Oxinium

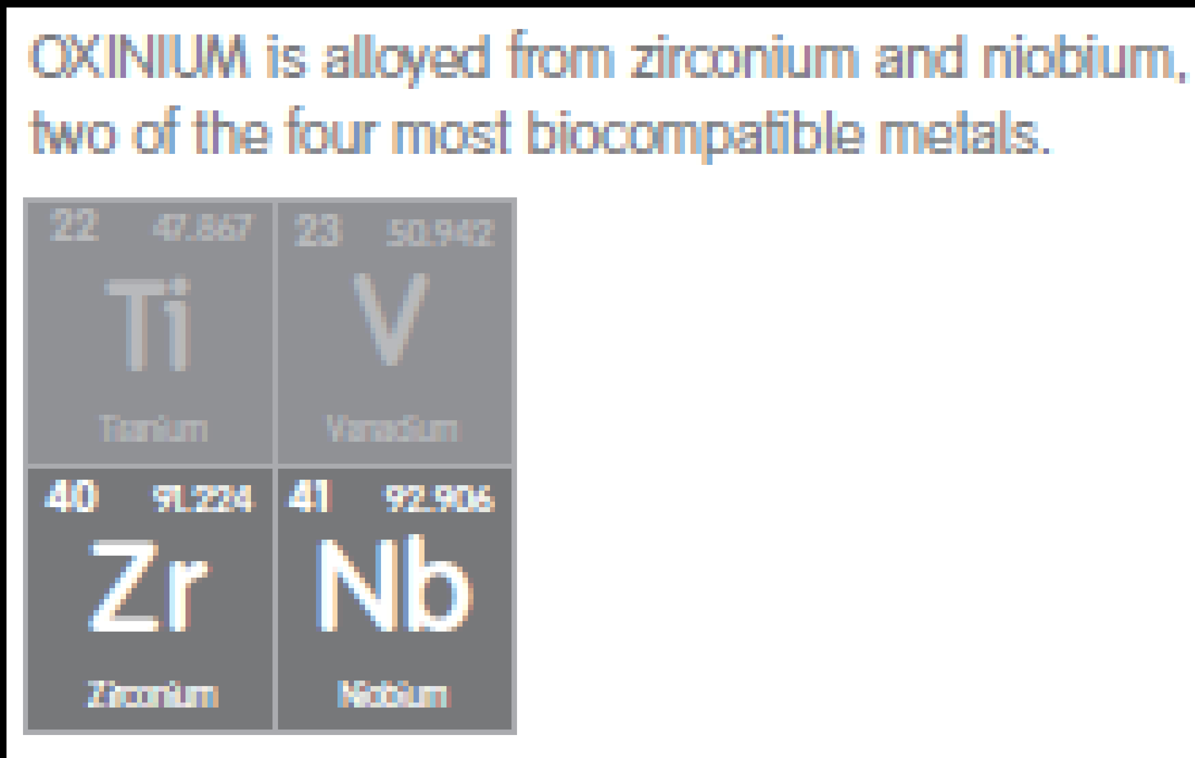
Oxidiz

Surfac

Suitabl

Abrasi ace

Long t bility



- **Surface hardness twice that of cobalt-chrome**
- **Avoids risk of brittle fracture that can occur with ceramic implants**
- **20% lighter than cobalt-chrome**
- **Contains no detectable nickel, the leading cause of negative reactions in patients with metal allergies.**


Oxinium Femoral
Head Component

Silicon Nitride (Si_3N_4)

Unique property

Ability to be formulated into a porous substrate as well as a hard glassy bearing surface.

Only ceramic that addresses possibility of monolithic implants, capable of an articulating smooth surface on one side, with a porous ingrowth surface fabricated on the opposing side of the same implant


1

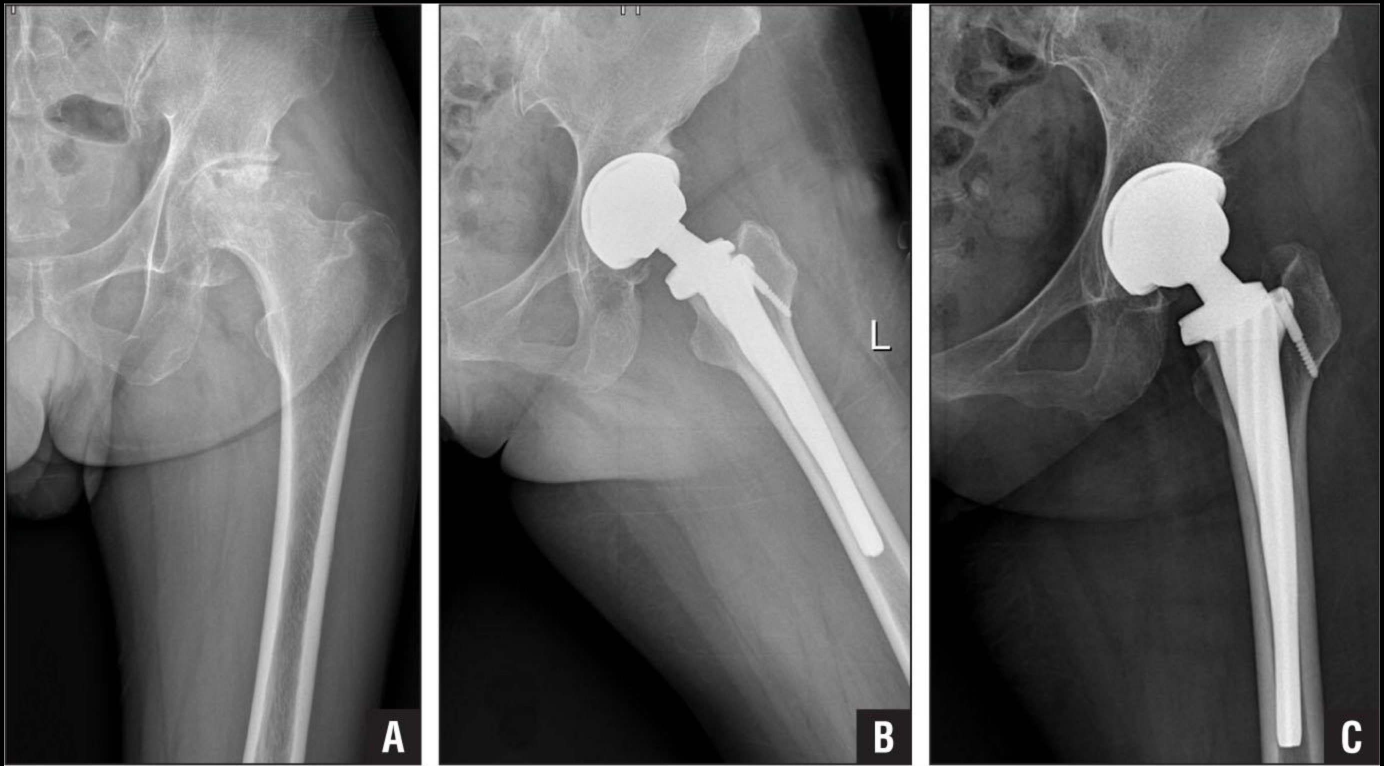
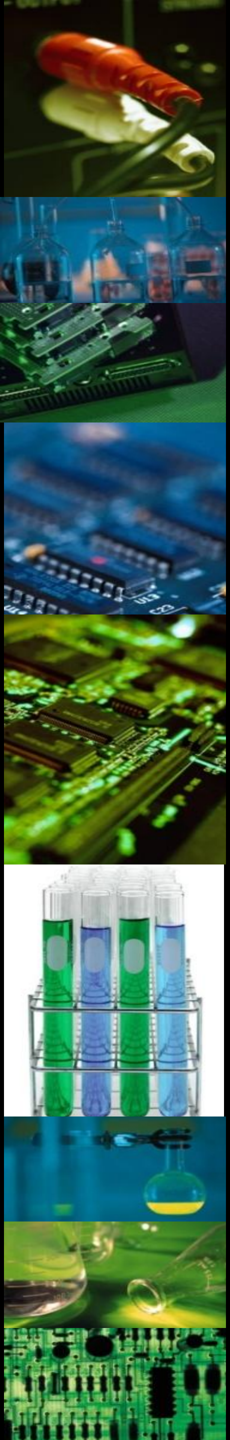

A
L
B
C

Table 1: Comparative Physical Properties of Alumina and Zirconia ceramics of Surgical grade

Property	Alumina	Zirconia
Purity (%)	>99.8	97.0
Density (g/cm^3)	3.98	6.05
Grain size (µm)	3.6	0.2 to 0.4
Surface Finish (Ra. µm)	0.02	0.008
Bending Strength (MPa)	595	1000
Compressive Strength (MPa)	4250	2000
Young's modulus (GPa)	380	210
Hardness (Vickers hardness number)	2000	1200
Fracture toughness K_{IC} (MN/m$^{2/3}$)	5	7



Mechanical Properties of Comparison

Property	Highest	Intermediate	Lowest
Tensile Modulus	Ceramic	Metals	Polymers
Yield strength	Metals	--	Polymers
Ultimate strength	Ceramics	Metals	Polymers

The newer Ceramics

Mixed-oxide/Dispersion ceramics

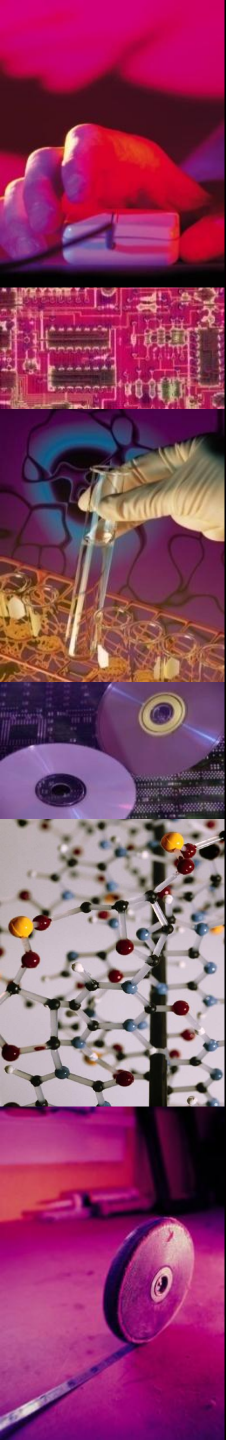
- New class of materials developed recently to combine the tribological properties of alumina and the mechanical characteristics of yttrium stabilised zirconia

Mixed-oxide ceramics

Bioactive ceramics

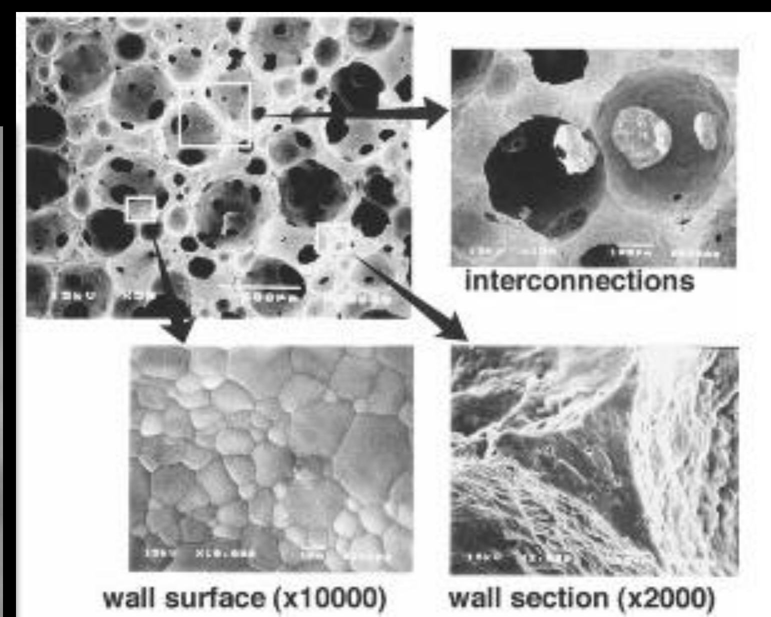
- Osteoconductive, acting as a scaffold to enhance bone formation on their surface, and are used either as a coating on various substrates or to fill bone defects

Calcium phosphate ceramics

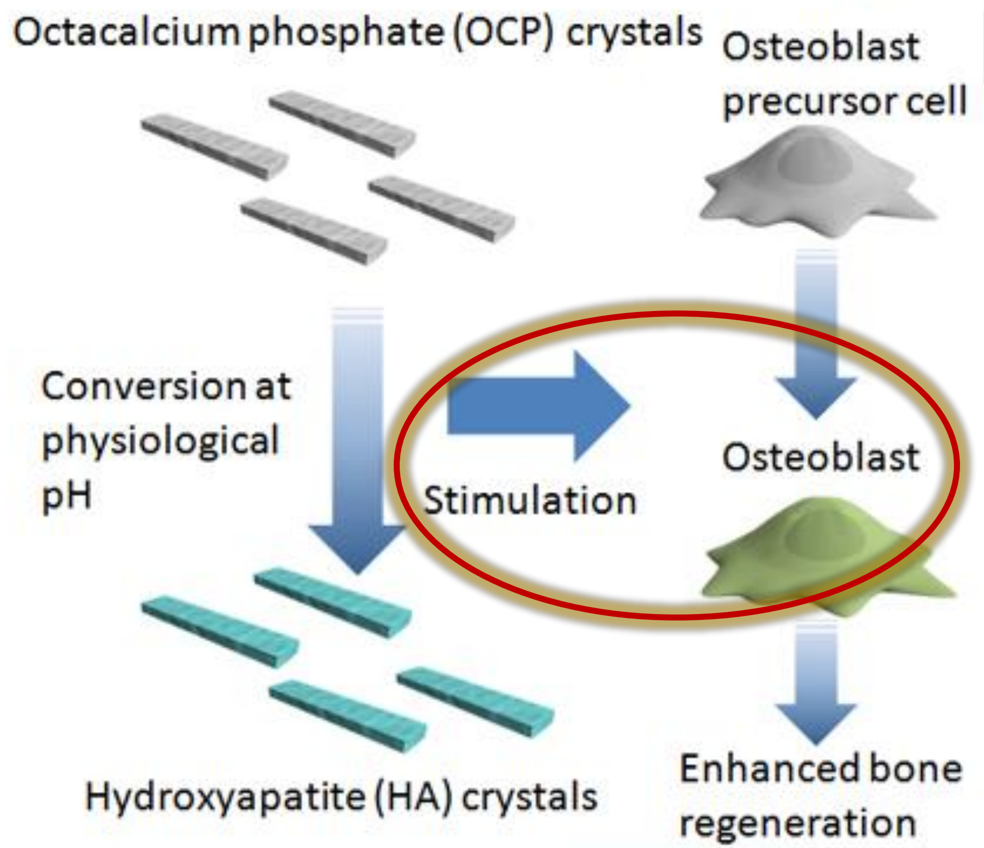


Calcium phosphate ceramics

- **Biological HA, however, is Cadeficient and a carbonated apatite.**
- **The bonding mechanism of HA to bone, seems to be due to the attachment at the surface of the HA of osteogenically-competent cells which differentiate into osteoblasts**



An example of a developed material stimulating cells



Suzuki O et al., Biomaterials 27:2671 (2006)
Suzuki O. Acta Biomater 6:3379 (2010)

Bone substitute materials developed

Calcium phosphate ceramics

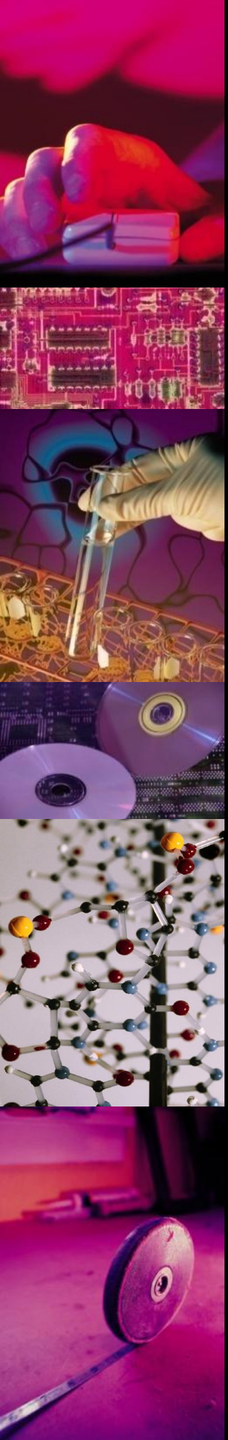
- HA … n femoral
  pros… a means
  of fi…
  com… use of
  PM… by
  plas…

Bioactive glasses

- The one common feature of these materials is the formation of a hydroxycarbonate apatite (HCA) surface layer

- Have a vitreous structure and bond chemically to bone

Legendary Professor Larry Hench discovered Bioglass®, the first man-made material to bond to living tissues in 1969



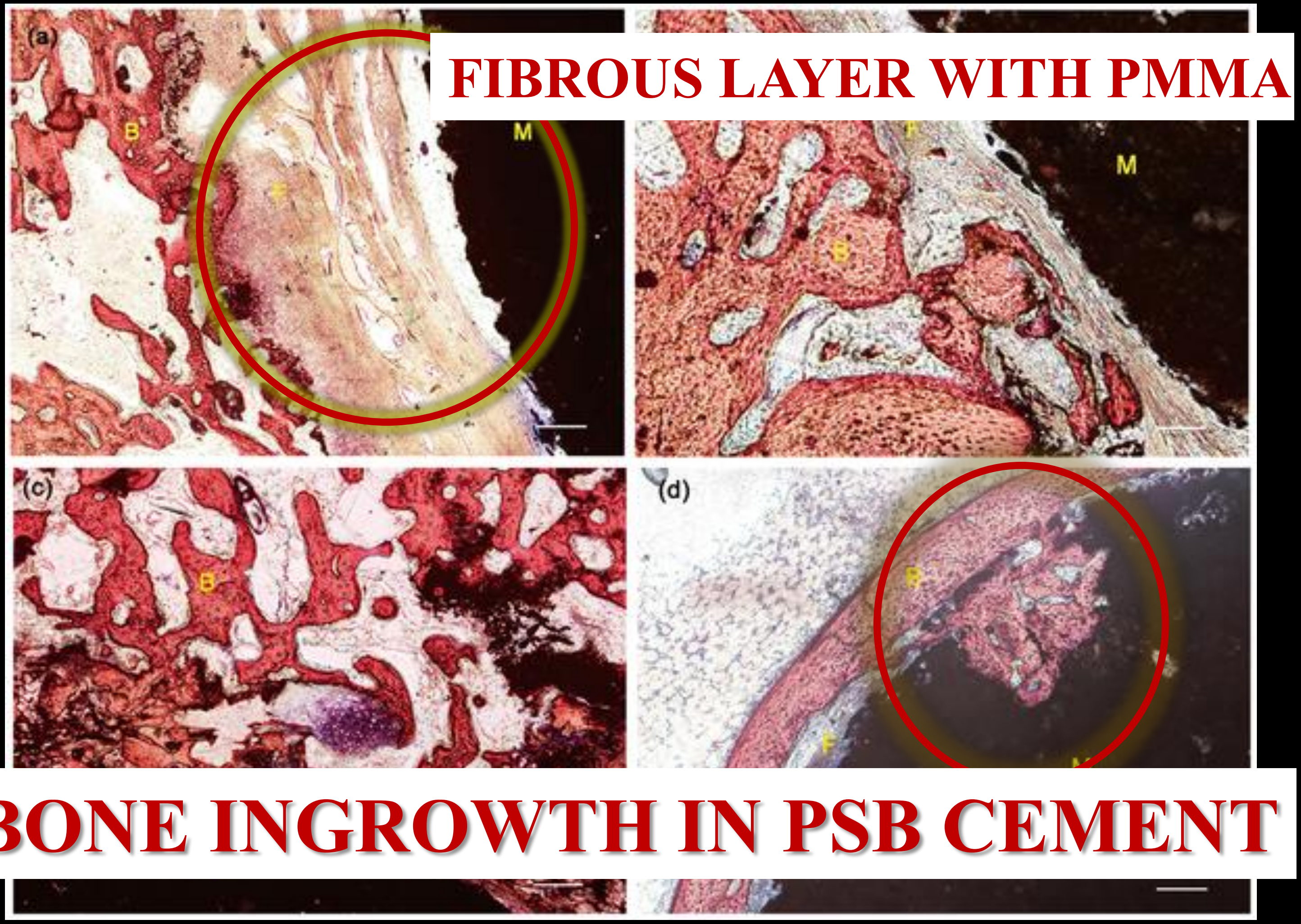
Bioactive bone cements

- Higher compressive, bending, and tensile strengths than PMMA cement and have a character of bonding directly with bone in 4-8 weeks in vivo

Bioactive bone cements

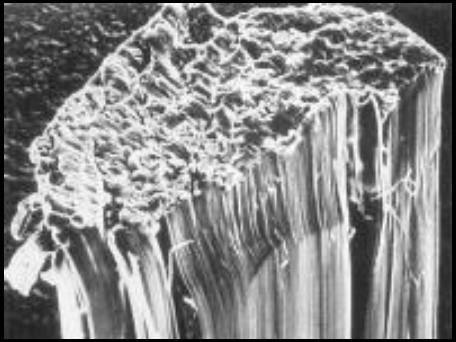
- **Porous polymethylmethacrylate can be achieved with the addition of carboxymethylcellulose, alginate and gelatin microparticles to promote bone ingrowth.**
- **Porous Surface modified Bioactive Bone Cement (PSB CEMENT)**

Histological morphologies of the interface between bone tissue and cement.



He Q, Chen H, Huang L, Dong J, et al. (2012) Porous Surface Modified Bioactive Bone Cement for Enhanced Bone Bonding

Bioabsorbable Devices in CMF Surgery



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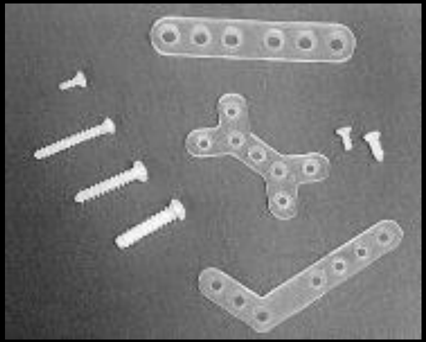

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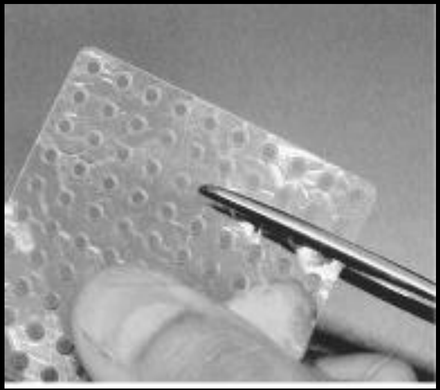

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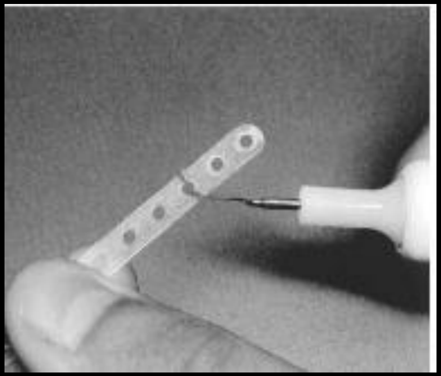

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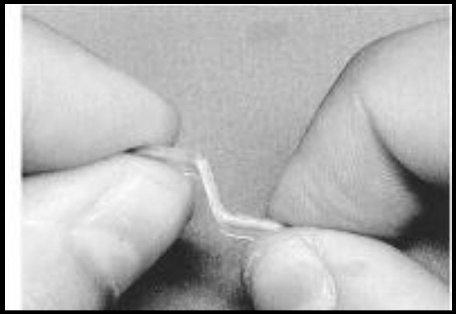
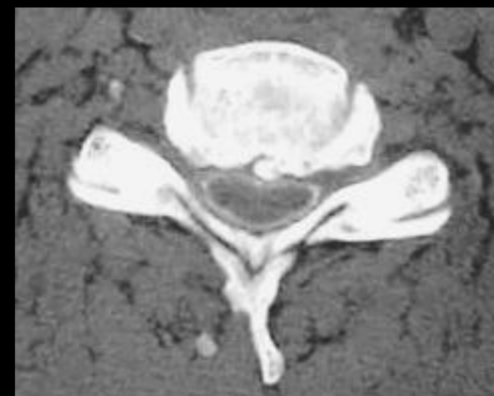
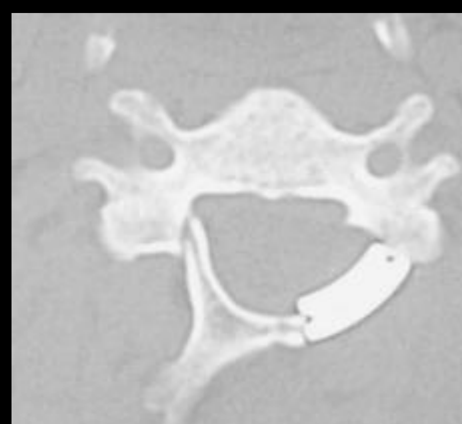


E

CERAMIC SPACER FOR CERVICAL LAMINOPLASTY





To Conclude

- Of all the Biomaterials in Orthopaedics today, Ceramics has stood the test of time for the last 30 years, specially in total hip replacement because of its tensile modulus, superior yield strength and the ultimate strength as a whole

To Conclude

- **As bearing and bonding surfaces, newer class of ceramics like silicon nitride, bioactive ceramics, calcium Phosphate ceramics and bioactive glasses have opened new vistas not only for joint replacements, but also as a bioactive bonding agent.**

Oxide Ceramics

Aluminum Oxide (Al_2O_3)

Aluminum Titanate (Al_2TiO_5)

Mixed/Dispersion Ceramics

Piezo-ceramics

Silicate Ceramics

Zirconium Oxide (ZrO_2)

Non-oxide Ceramics

Aluminum Nitride (AlN)

Silicon Carbide (SiSiC / SSiC)

Silicon Nitride (Si_3N_4)

SiAlONs

Ceramic Composite

Metal Matrix Composite (MMC)
Metal/Ceramic Composites

CLUB FOOT CURRENT CONCEPTS

Dr. Gopakumar T.S
Prof & Head of Orthopaedics
Medical College
Trivandrum

- Common congenital anomaly in the lower limb
- Challenging problem to manage

EPIDEMIOLOGY

- Incidence – 0.6-8/1000 live births (average 1.2/1000)
- Prevalence – increased in developing countries
- Global epidemiology –
  - approximately 200,000 new cases per year
  - 80% in low and middle income countries
  - Est 50,000 new cases per year in India
- More common in boys: 2:1 male: female
- 40% of cases bilateral

SPECTRUM OF CLUBFOOT DEFORMITY

CLASSIFICATION

Based on cause	Based on treatment
– Idiopathic	– Corrected
– Secondary	– Uncorrected
– Postural	• Resistant
	• Recurrent
	• Neglected

IDIOPATHIC

- Isolated Clubfoot, otherwise 'normal' child

SECONDARY

- **Neuropathic**
  - Spina bifida
  - Other neurological deficits
- **Myopathic**
- -Arthrogryposis
- **Osteopathic**
  - Cong absence of tibia

- **Syndromic**
- Larsen's syndrome
- Whistling face syndrome
- Constriction band syndrome

SECONDARY CLUBFOOT

RESISTANT CLUBFOOT

- Clubfoot that is difficult to completely correct with the conservative technique

- Hindfoot and/or midfoot contractures persist

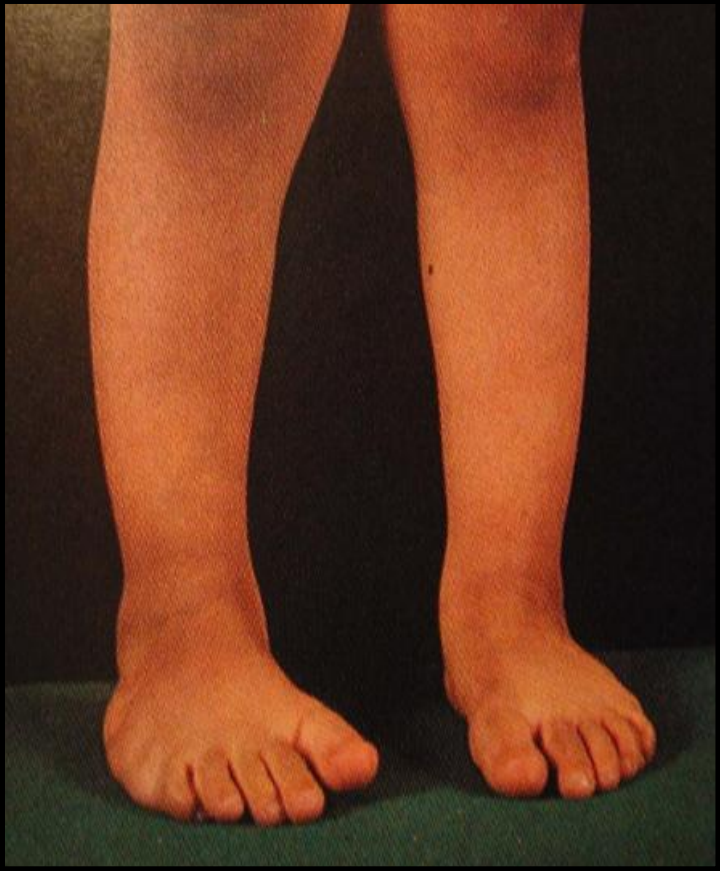
- Often seen as part of a syndrome or arthogryposis

ATYPICAL CLUBFOOT

- Short, cocked up great toe
- Transverse crease
- Plantaris
- Correction stops after 3-4 casts
- Casts fall off

RECURRENT CLUBFOOT

- A corrected clubfoot that later develops recurrence
  - Walking on the lateral border of the foot
  - Equinus and/or Varus of Hindfoot
  - Dynamic Supination of foot
    - (Overactive Tibialis Anterior)



NEGLECTED CLUBFOOT

- Clubfoot that has not been corrected before the child starts walking
- May present with severe contractures and bony deformity

COMPLEX CLUBFOOT

- Clubfoot that has been treated with methods other than the Ponseti technique
- May have other deformities or scarring
- Treatment must be individualised

ETIOLOGY

- Unknown
- Germ plasm defect
- Retracting fibrosis
- Neuromuscular
- Genetic
- Environmental

RECENT ADVANCES IN ETIOLOGY

- Several chromosomal deletion regions, including 2q31-33 are associated with talipes equinovarus and may harbor genes that contribute to the idiopathic talipes equinovarus phenotype

Heck et al (2005)

ANTENATAL DIAGNOSIS

- Can be diagnosed antenatally using ultrasound.
- Positive predictive value of 83% with a false positive rate of 17%
- False-positive rate was higher for unilateral (29%) than for bilateral clubfoot (7%).
- Scans at 20 to 24 weeks may be more reliable for the diagnosis than those taken earlier. (Bar Hava et a)

ANTENATAL DIAGNOSIS

- Degree of deformity was difficult to assess before birth.
- At birth, 26% were found to require no treatment, while 61% needed treatment
- Important implications for prenatal counseling.

ASSESSING, MEASURING AND RECORDING THE CLUBFOOT DEFORMITY

PIRANI SCORE

- Scoring system developed by Dr Shafique Pirani
- Tests 6 different components of deformity
- Assigns each a severity score of 0, 0.5 or 1
- A valid, reliable method of measuring and recording deformity
- Good intra/inter-observer reliability

PIRANI SCORE

- Midfoot contracture score (MCFS)
  - Curved lateral border
  - Medial crease
  - Lateral head of talus
- Hindfoot contracture score (HFCS)
  - Posterior crease
  - Rigid equinus
  - Empty heel
- Total score (TS)

MIDFOOT CONTRACTURE:

CURVED LATERAL BORDER

MIDFOOT CONTRACTURE:

MEDIAL CREASE

MIDFOOT CONTRACTURE:

LATERAL HEAD OF TALUS

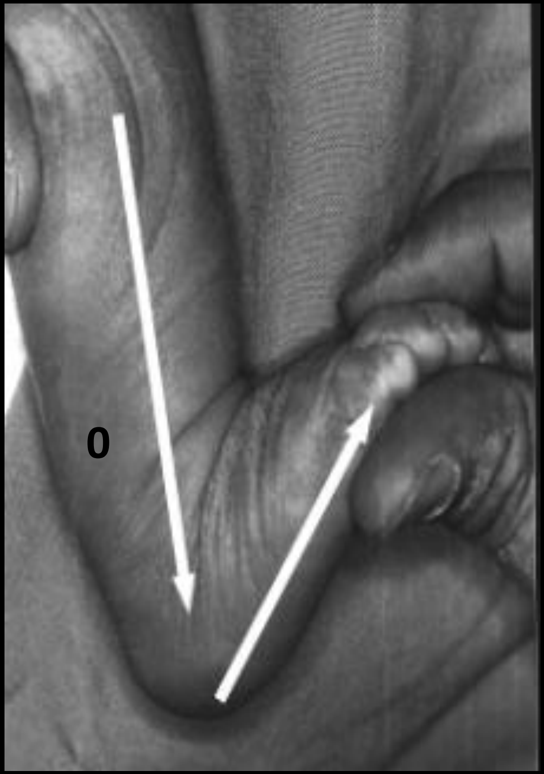


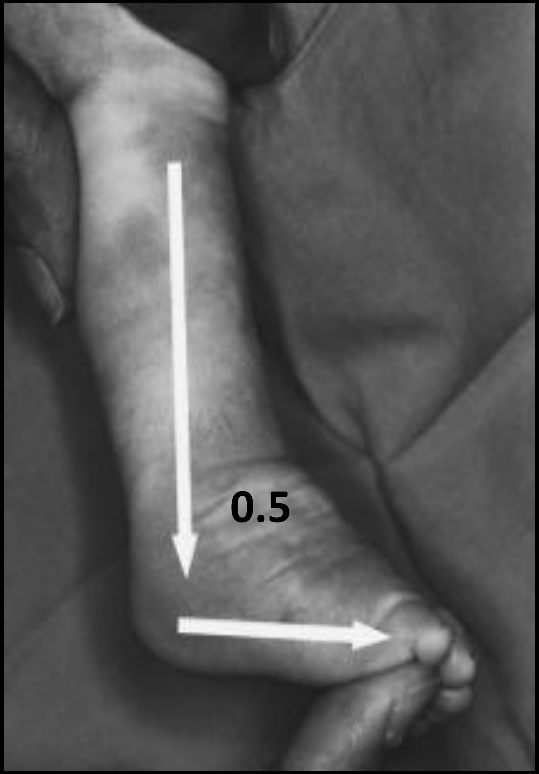
HINDFOOT CONTRACTURE:

POSTERIOR CREASE

HINDFOOT CONTRACTURE:

RIGID EQUINUS

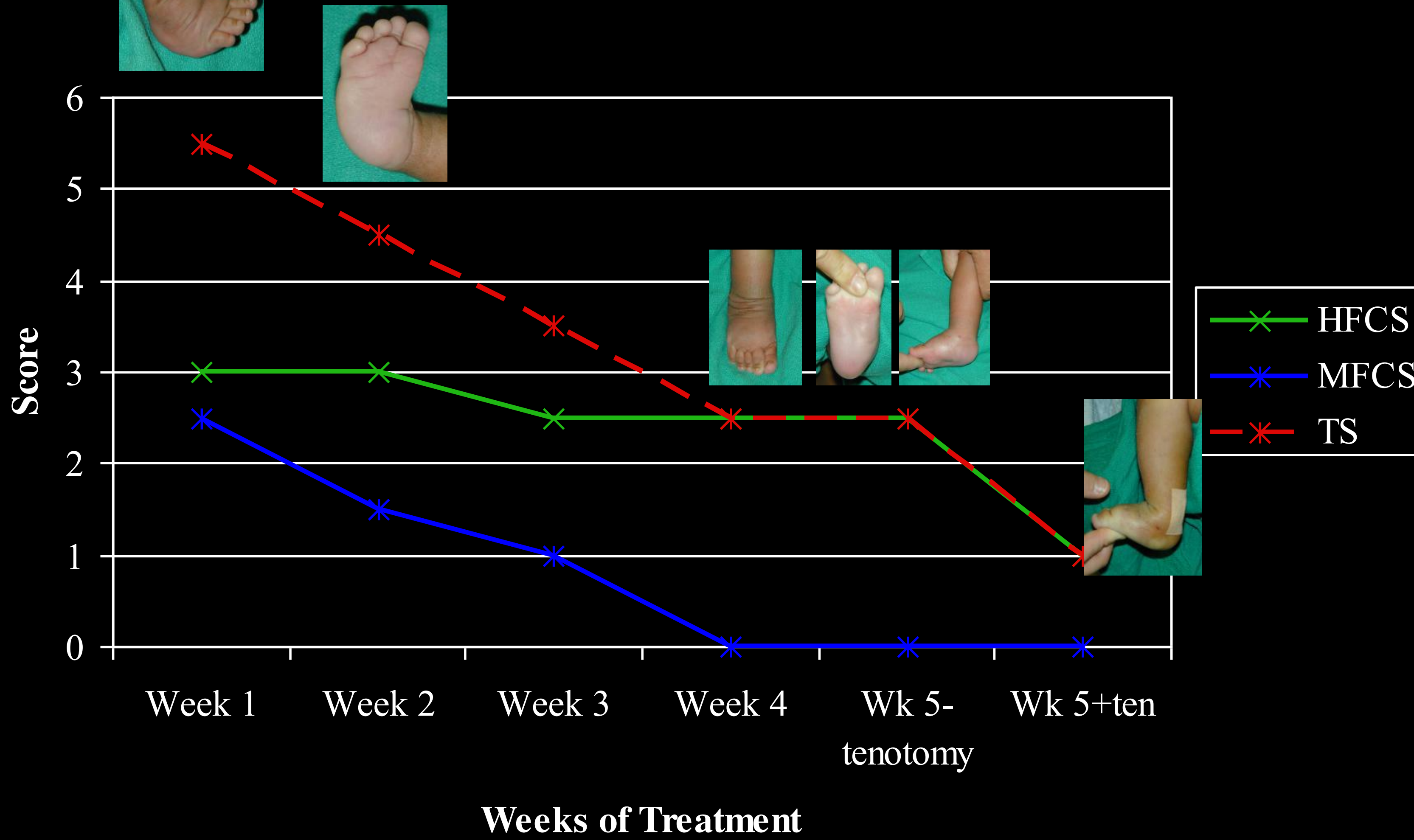




HINDFOOT CONTRACTURE:

EMPTY HEEL

Clubfoot Scores with Ponseti Treatment



AIM OF TREATMENT

- To achieve concentric reduction of the talocalcaneonavicular joint
- To maintain reduction
- To restore normal articular alignment of the tarsus and the ankle
- To establish muscle balance between evertors and invertors, dorsiflexors and plantarflexors
- To provide mobile foot with normal function and weight bearing

TREAMENT

- CONSERVATIVE
  KITE
  PONSETI
  BENSAHEL

- SURGICAL
  ‘one-size fits all’ Turco
  ‘a la carte’ Bensahal

- EXTERNAL FIXATION
  JESS
  Ilizarov

SURGICAL TREATMENT

SURGICAL

'one-size fits all' Turco

'a la carte' - Bensahal

Long-term results of soft-tissue correction of CTEV (Dobbs, Nunley and Schoenecker)

- 73 feet in 45 patients
- Minimum follow-up of 25 years.
- Turco style release and 87% had more than one operation, the second usually in adolescence.
- The Laaveg and Ponseti scores revealed 0% excellent, 33% good, 20% fair and 47% poor results.

LONG TERM FOLLOW UP SURGICAL TREATMENT

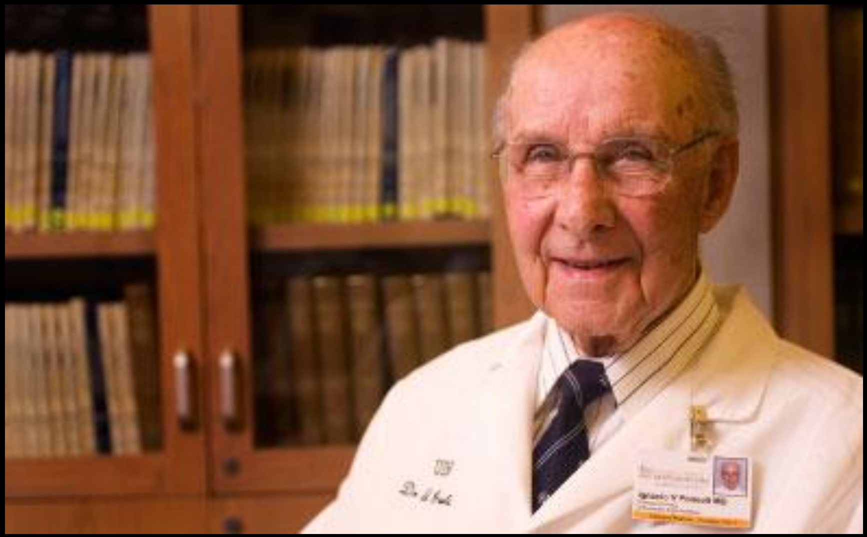
- Conspicuous anatomical changes, significant muscle weakness,and insufficient ankle range of motion
- Functional results have been reported to be better if extensive surgery could be avoided

Reoperations required in more than half of the patients,

Risk of serious complications which are sometimes more difficult to treat

TREATMENT OF CLUB FOOT

Shift from surgical to conservative treatment



Ignacio Ponseti, MD
Professor
Department of Orthopaedic Surgery
The University of Iowa

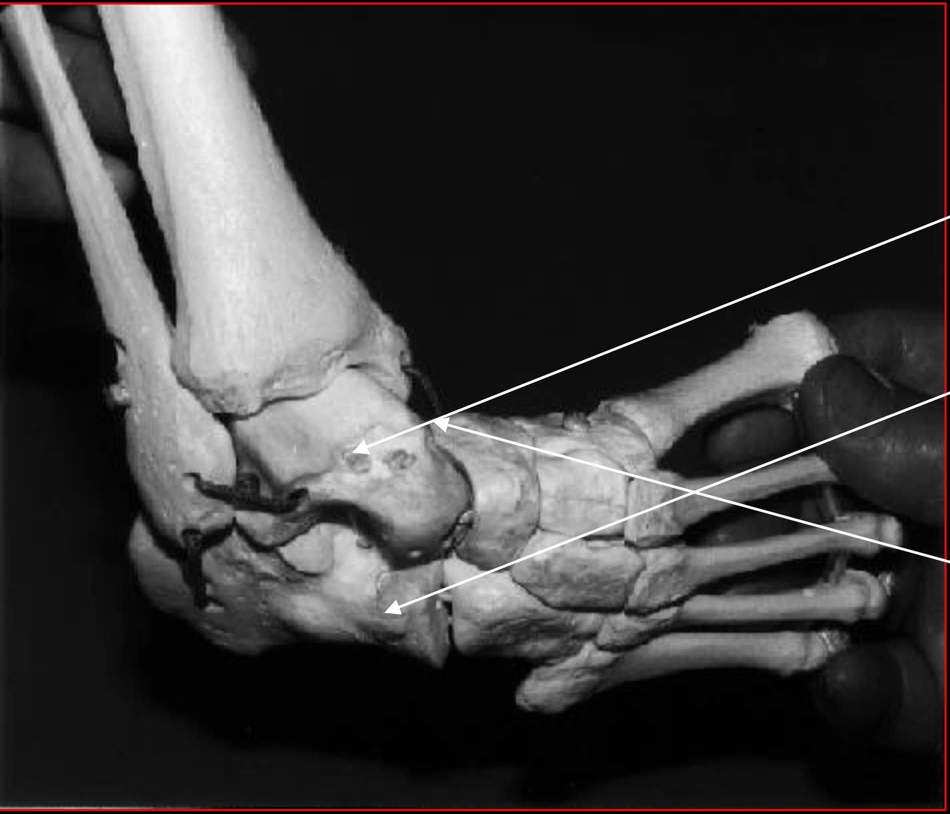
‘our patients treated 25 to 42 years ago it was found that although the treated clubfeet were less supple than the normal foot, there were no significant difference in function or performance compared to a population of a similar age born with normal feet.’

March 1996

PONSETI TECHNIQUE

- Serial casting of the lower limb using a strictly defined technique
- Tenotomy of the tendo Achillis at ‘hindfoot stall’.
- Once the foot is corrected FAO up to the age of four years.
- TA transfer in recurrence of deformity after two and a half years of age.

PONSETI - PATHOANATOMY



The talus and calcaneus are in severe flexion.

The calcaneus, navicular and the cuboid are adducted and inverted.

The navicular tuberosity is close to the medial malleolus.

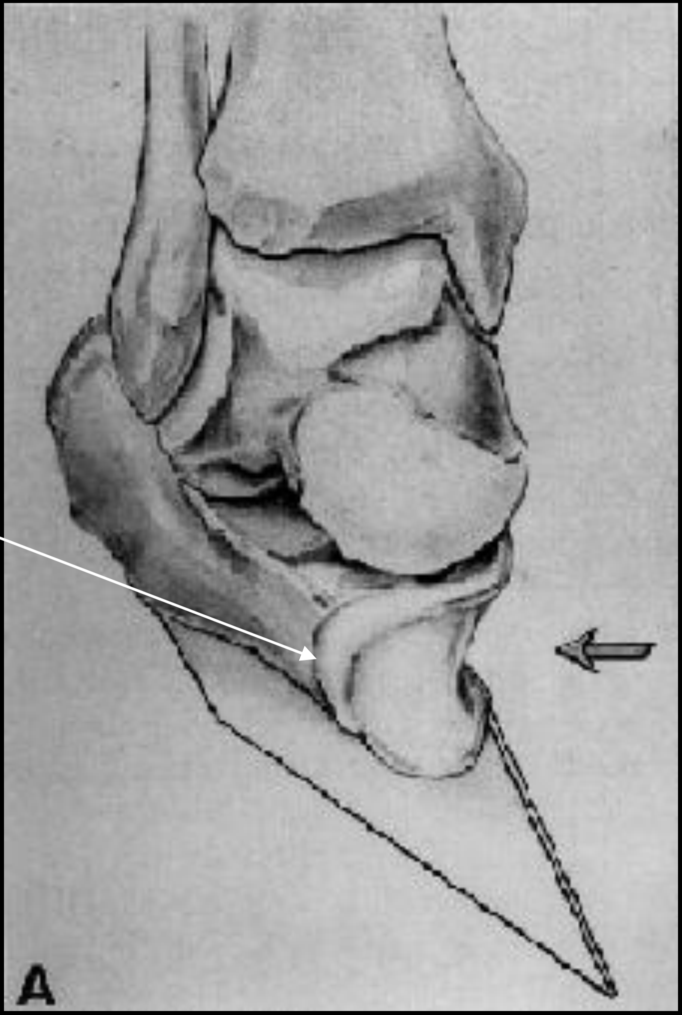
.

PATHOANATOMY

The first metatarsal is in more flexion than the other metatarsals, thus causing the cavus.

BIOMECHANICS

- Clubfoot deformity mimics extreme position of subtalar flexion, adduction, and inversion.
- Anterior end of the calcaneus is beneath the talar head, which results in an equinus and cause varus deformity of the heel.



CALCANEAL-PEDAL BLOCK

- The forefoot moves as a unit with the calcaneus, and this unit is termed the calcaneal-pedal block.
- The calcaneal-pedal block moves around the talus and motion of the intertarsal joints and the tarsometatarsal joints can be disregarded.
- This permits simultaneous rotation at the talocalcaneal and the talonavicular joints.

BIOMECHANICS

- Correction of clubfoot deformity can be accomplished by abducting the forefoot while blocking the talus in the ankle joint.

- This brings the foot from adduction, inversion, and flexion (supination) to abduction, eversion, and extension (pronation)

PONSETI - STEPS

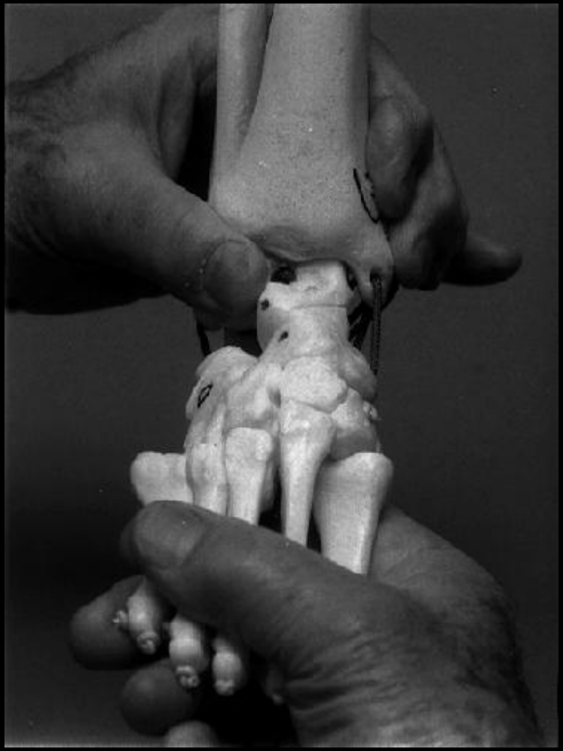
The first metatarsal is in more flexion than the other metatarsals, thus causing the cavus.

The cavus is corrected by extending the first metatarsal and supinating the forefoot.

PONSETI - STEPS

In this position the foot can be abducted under the talus.
Counterpressure is applied on the lateral aspect of the head of the talus.
The heel is not touched.

PONSETI - STEPS



The lower part of the tibia is grasped by one hand with the index and middle fingers

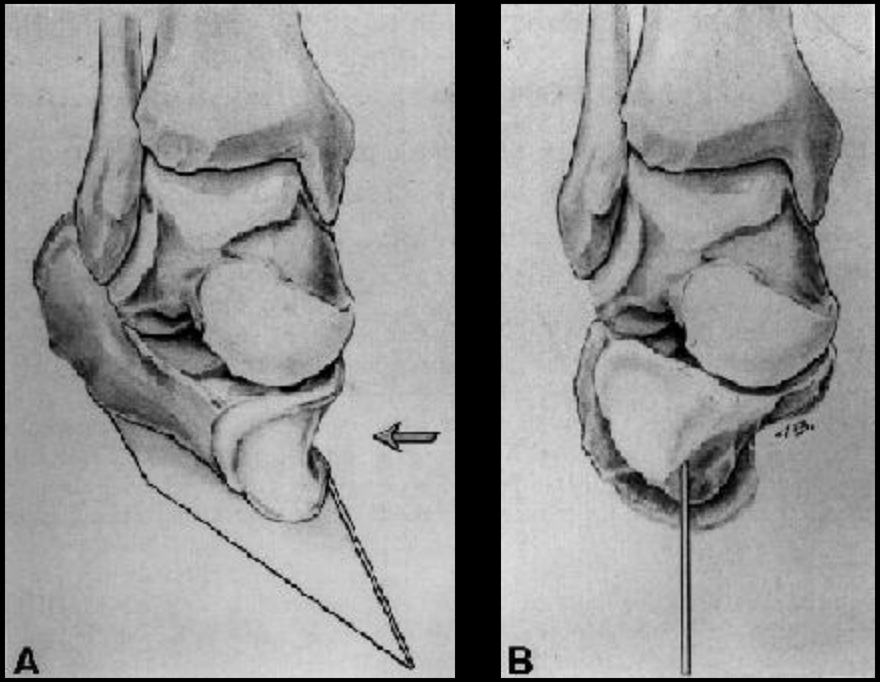
The thumb rests on the lateral aspect of the head of the talus.

The other hand grasps the forefoot and midfoot in slight supination.

PONSETI - STEPS

Complete correction of the clubfoot requires severe abduction of the midfoot and forefoot to stretch the tight medial tarsal ligaments.

PONSETI - STEPS



When the calcaneus abducts it simultaneously extends and everts to its normal and neutral position under the talus.

THE COMMON ERRORS IN THE TREATMENT

- Remove the plaster cast at home the day before the cast change.

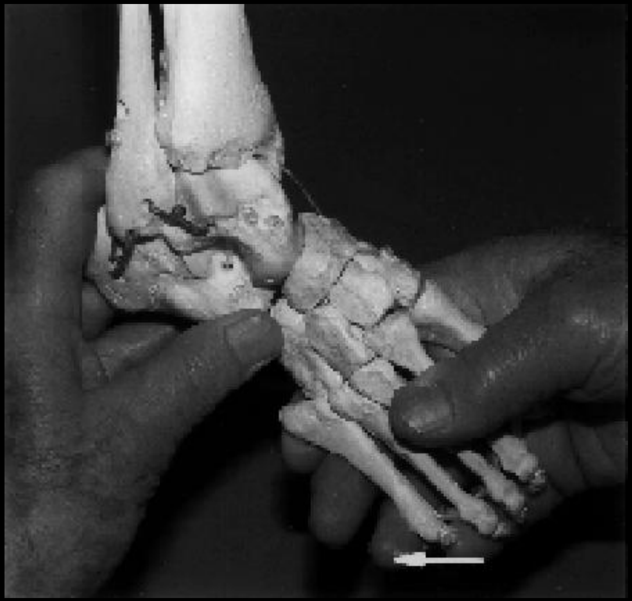
- Much correction is lost while the foot is out of the cast.

- The cast should not be removed more than an hour before the new cast is applied.

THE COMMON ERRORS IN THE TREATMENT

Pronating the supinated forefoot is incorrect because it increases the cavus deformity and locks the midtarsal joint

PRESSURE OVER CALCANEOCUBOID JOINT



Arching the foot as if to straighten a bent wire with pressure applied near the calcuneocuboid joint is a major error

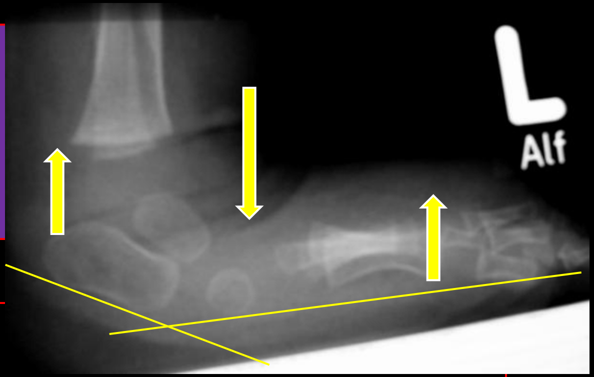
Medial pressure at the calcaneocuboid joint prevents the calcaneus from abducting since the calcaneus can evert only when it is abducted

KITE' ERROR

- Kite wrongly believed that the heel varus would correct simply by everting the calcaneus.

- He did not realize that the calcaneus can evert only when it is abducted

EQUINUS CORRECTION



- In 90% of cases, it is necessary to perform an Achilles tenotomy to correct the residual equinus deformity
- Tenotomy should be performed only after 70 degrees of foot abduction is achieved and the heel is palpated to be in a valgus position

EQUINUS CORRECTION

- An Achilles tenotomy is performed on infants as old as six months with local anesthesia in an office setting.

- For infants older than six months GA may be needed

EQUINUS CORRECTION

- After a percutaneous Achilles tenotomy is performed, the LLC is applied for 3 weeks to allow the severed Achilles tendon to heal.
- The cast is applied with the foot in 70 degrees of external rotation and maximum dorsiflexion (approximately 10 degrees to 20 degrees).

HEEL VARUS

The heel is in varus when the foot and calcaneus are adducted.

The heel varus is corrected by abducting the foot.

BRACING PHASE

- Foot abduction orthosis set at 45 degrees of external rotation for the normal foot and 70 degrees of external rotation for the clubfoot.
- For bilateral cases, both feet are set at 70 degrees of external rotation
- **23 hours per day for the first three months and then at night only for two to four years**

COMPLIANCE WITH THE BRACING PROTOCOL

- 17 of 157 patients (10.8%) who had undergone Ponseti treatment had a recurrence of clubfoot. Of the 17 patients, 15 were noncompliant and two were compliant.
-
- A noncompliant patient was 17 times more likely to experience a recurrence than a compliant patient.
- Most challenging aspect of the Ponseti method is to maintain compliance with the bracing protocol.

(Morcuende et al)

RELAPSE

- Infants with rigid feet, especially those with short, fat feet
- Early relapse is heralded by supination and varus deformities of the forefoot and heel.
- Treatment for relapse is repeat casting to regain correction and then continued bracing
- Surgery in < 3% of cases

TIBIALIS ANTERIOR TENDON TRANSFER

- Dynamic supination occurs secondary to weak peroneal tendons and strong the tibialis anterior muscle.
- Tibialis anterior tendon transfer to the lateral cuneiform might be necessary in children older than 24 months when the lateral cuneiform is ossified

RESULTS

	Excellent and good results
Ponseti and Smoley	89%
Morcuende JA, Dolan LA, Dietz FR, Ponseti IV	98%
Cooper DM, Dietz FR (30 year follow up)	62%
Radler et al 2006	93%
Abdelgawad etal	93%
Shack etal	97.5%
Atul Bhaskar etal	80%

OUR EXPERIENCE

- 240 Children (August 2011)
- Complete data 80 children 120 feet
- 95% good and results
- 4 cases of relapse



BINCY ATYPICAL



PONSETI METHOD

- Ponseti method has revolutionized treatment of club foot
- Can correct club foot in 90% of cases
- Avoid extensive surgical procedures
- Long term result of surgical treatment is disappointing
- Ponseti method can be used in older children
- It may be successful even in relapsed and neglected club foot

BIOMECHANICS

- Internal rotation (adduction) of the calcaneus about the subtalar axis occurs only with inversion and plantar flexion of the ankle joint. The resulting clinical motion is defined as supination.
-
- External rotation (abduction) of the calcaneus about the subtalar axis occurs only with eversion and some dorsiflexion of the ankle joint. The resulting motion is defined as pronation.

PONSETI - STEPS

.

The medial tarsal ligaments are stretched allowing the calcaneus to abduct with the foot and the anterior tuberosity of the calcaneus is disengaged from its position under the head of the talus.

CLUBFOOT IN OLDER INFANTS.

- Children who were first seen after the age of three months in whom conservative treatment had failed. After a period of 24 months, only one of 36 feet (2.8%) required extensive surgery.
- **Bor N, Herzenberg JE, Frick SL.** Ponseti management of clubfoot in older infants. *Clin Orthop* 2006;444:224–8.[

CLUBFOOT IN OLDER INFANTS.

.

- Ponseti technique is reproducible and effective in children at least up to 12 months of age.

Ponseti technique for the correction of idiopathic clubfeet presenting up to 1 year of age

Arch Orthop Trauma Surg (2006) 126: 15–21

NEGLECTED CLUB FOOT

- A painless plantigrade foot was obtained in 16/24 feet without the need for extensive soft-tissue release and/or bony procedures.

- Ponseti method is a safe, effective and low-cost treatment for neglected idiopathic club foot presenting after walking age.

- **Correction of neglected idiopathic club foot by the Ponseti method.**
  J Bone Joint Surg Br. 2007 Mar;89(3):378-81.
  Lourenço AF, Morcuende JA.

Cooper DM, Dietz FR. Treatment Idiopathic clubfoot: a thirty-year follow-up note. *J Bone Joint Surg [Am]* 1995;77-A:1477–89.

- 45 adults, with 71 clubfeet, who had been managed with the Ponseti method, 30 years after treatment.
- The results showed 62% excellent. Radiographs showed that the feet were not completely corrected, but functioned well despite this.

Morcuende JA, Dolan LA, Dietz FR, Ponseti IV. Radical reduction in the rate of extensive corrective surgery for clubfoot using the Ponseti method. *Pediatrics* 2004;113:376–80

- .
- Short-term results of a more recent series of 256 feet. Correction was obtained in 98% of the patients with between one and seven casts.
- Percutaneous tenotomy of the tendo Achillis was performed in 86% of the cases.
- The mean angle of dorsiflexion of the ankle after tenotomy was 20° (0° to 35°).
- Minor complications from the cast were encountered in 8% of patients
- 2.5% required extensive corrective surgery.
- The rate of relapse after initial successful treatment was 10%.

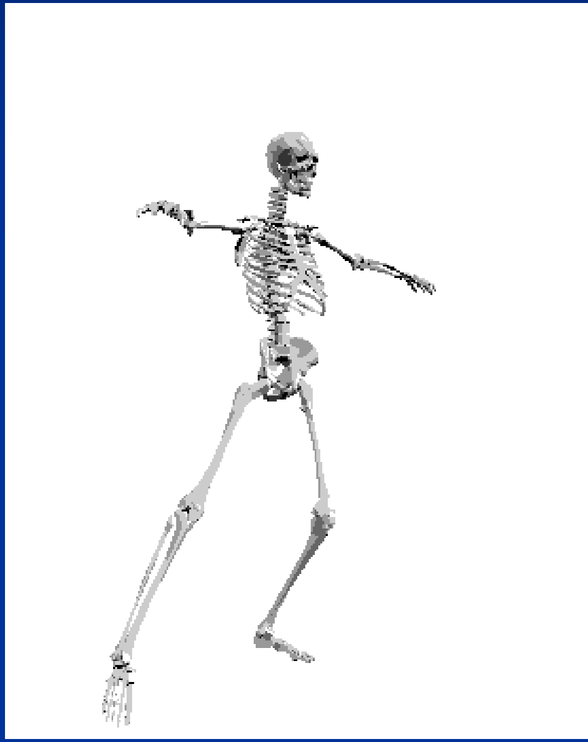
Results of Ponseti method

- Ponseti and Smoley reported that open surgery was avoided in 89%

- Posteromedial soft-tissue release was avoided in 81% -Changulani, M

- Good results in 80% of cases- Atul Bhaskar etal

Tumor markers in orthopedic pathology



Dr. Muktha R. Pai

Professor of Pathology,

AJ Institute of Medical Sciences,Mangalore

Introduction

- Bone tumors are very diverse in morphology and biological potential
- Most bone tumors are benign lesions
- Most benign lesions are seen <30 years of age
- **A new bone tumor in the elderly is more likely to be malignant**
- Benign lesions typically present as incidental finding
- **Pathological fracture can be the first sign of malignant tumor**

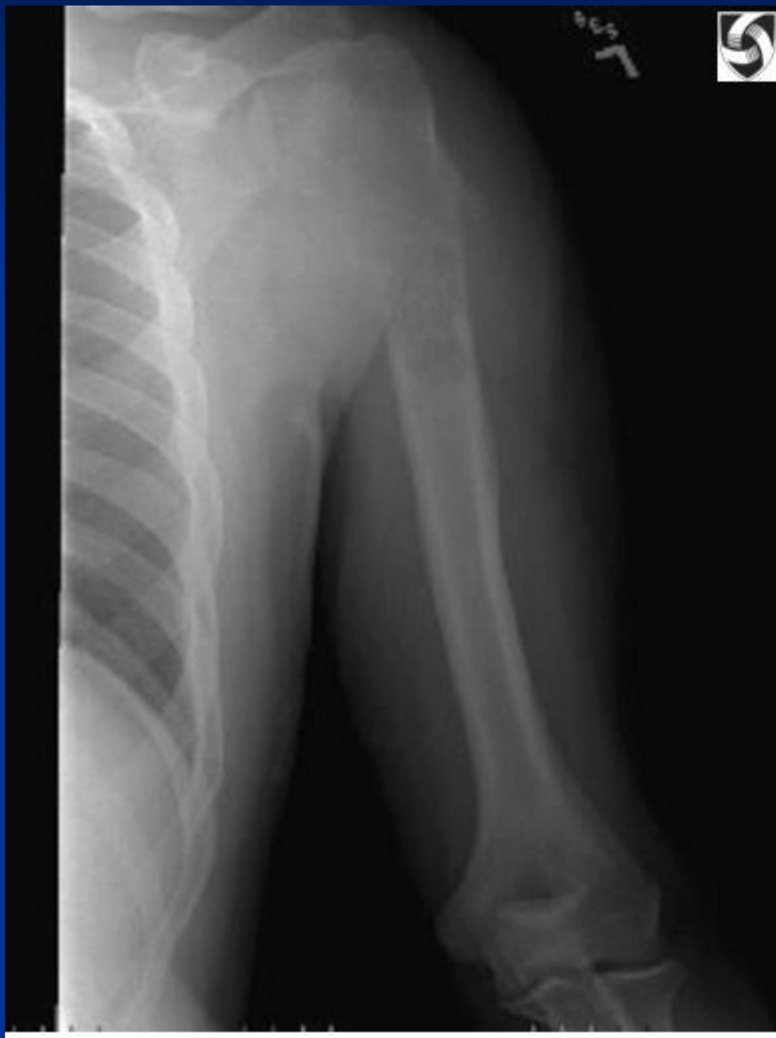
Introduction

- Multidisciplinary approach is mandatory for Bone tumor diagnosis- **orthopedician, radiologist & pathologist**
- why is radiology important?
  - Exact location of lesion Extent of growth/metastasis
  - Aggressiveness
- Best test for Dx= X-ray
- Best test for staging= CT or MRI
- **Quick shout out to the pathologists– histologic grade is the most imp.prognostic feature of bone sarcomas**
- **essential for staging the bone tumor types.**

Case I

- 16 yr old white male with pain in his left upper arm.
  - Mild swelling and tenderness
  - Pain progressively getting worse for ~ 3 months
  - Recent onset of mild fever
  - Lab tests-   ESR ,Leucocytosis

Imaging:



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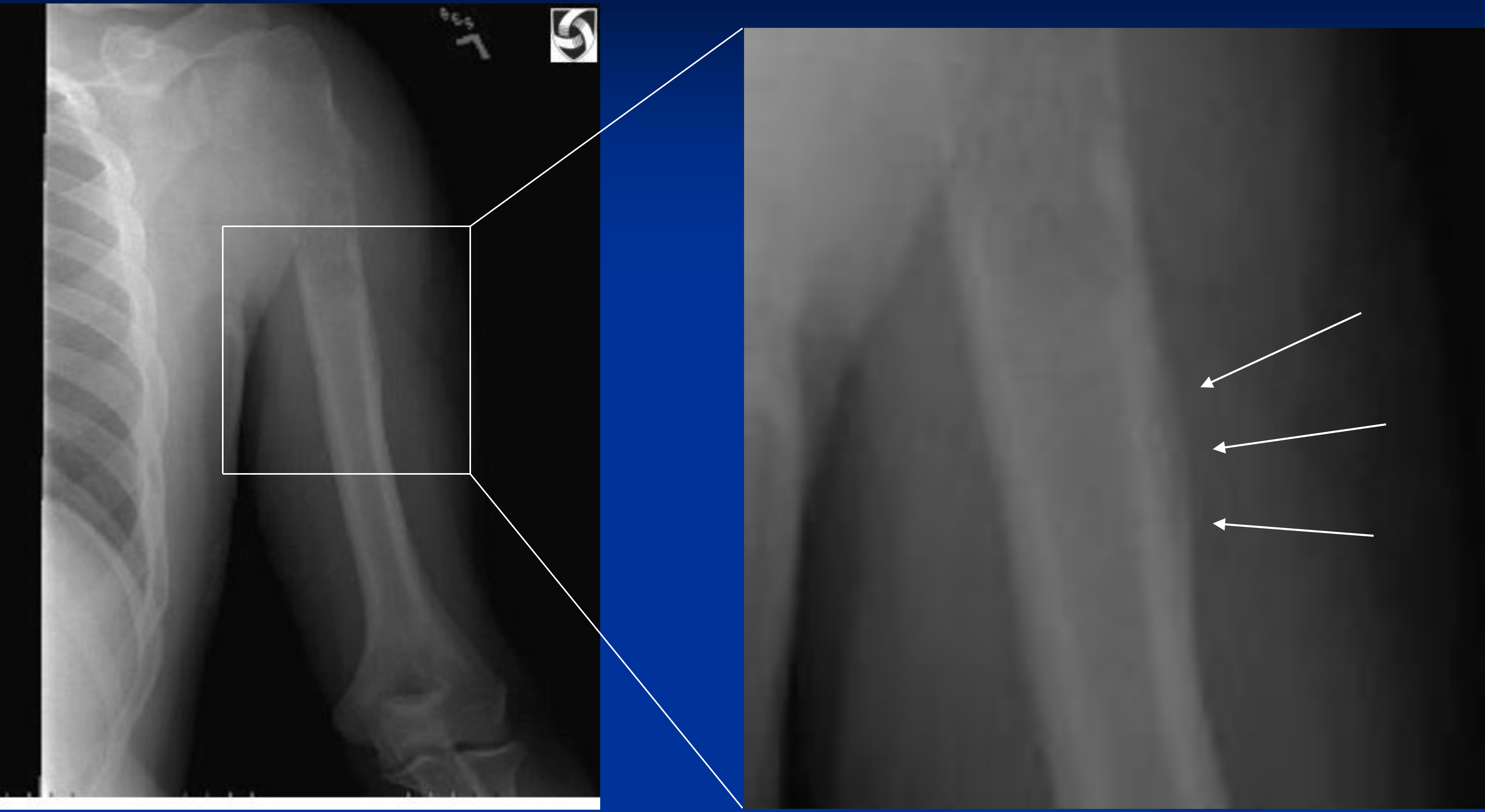
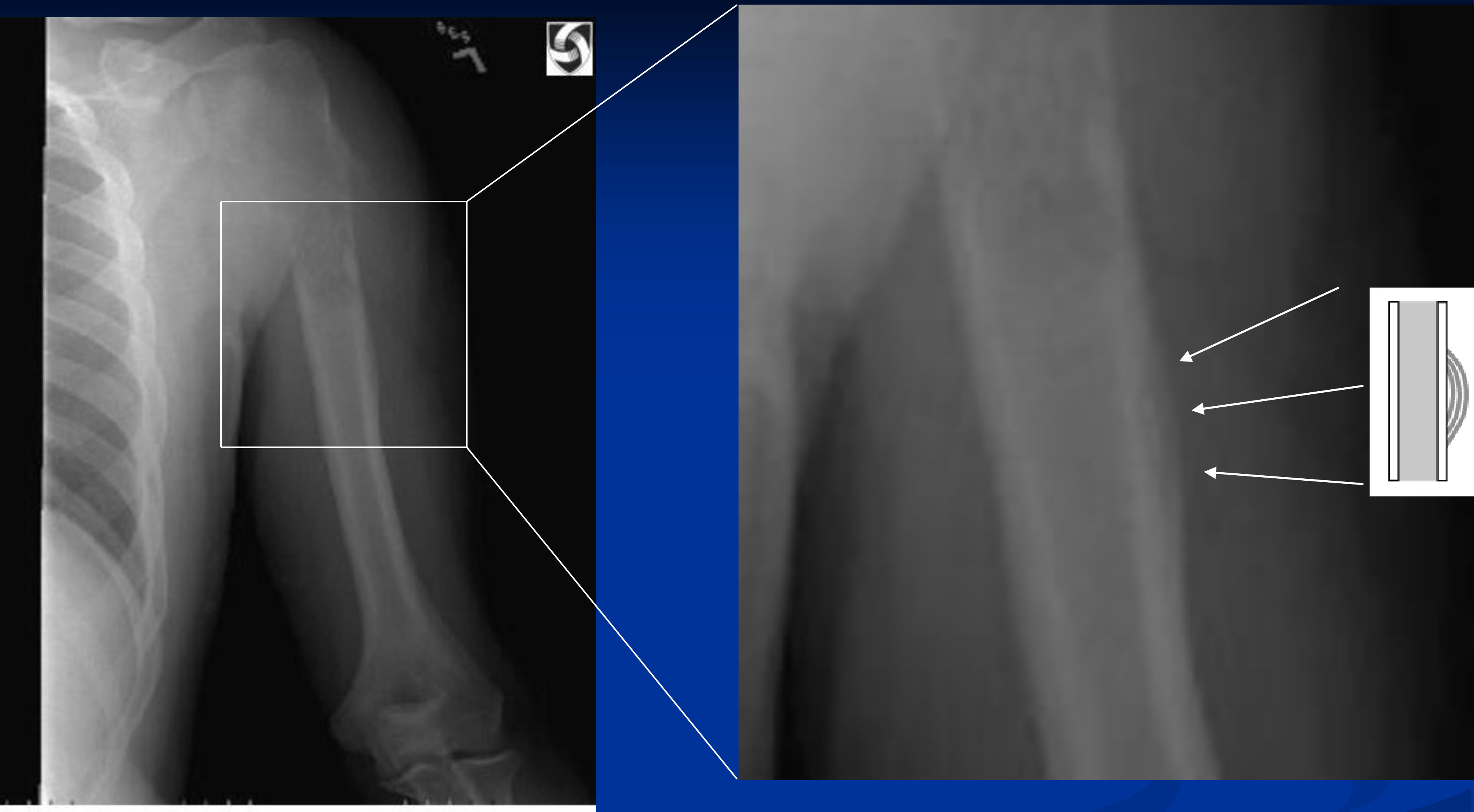
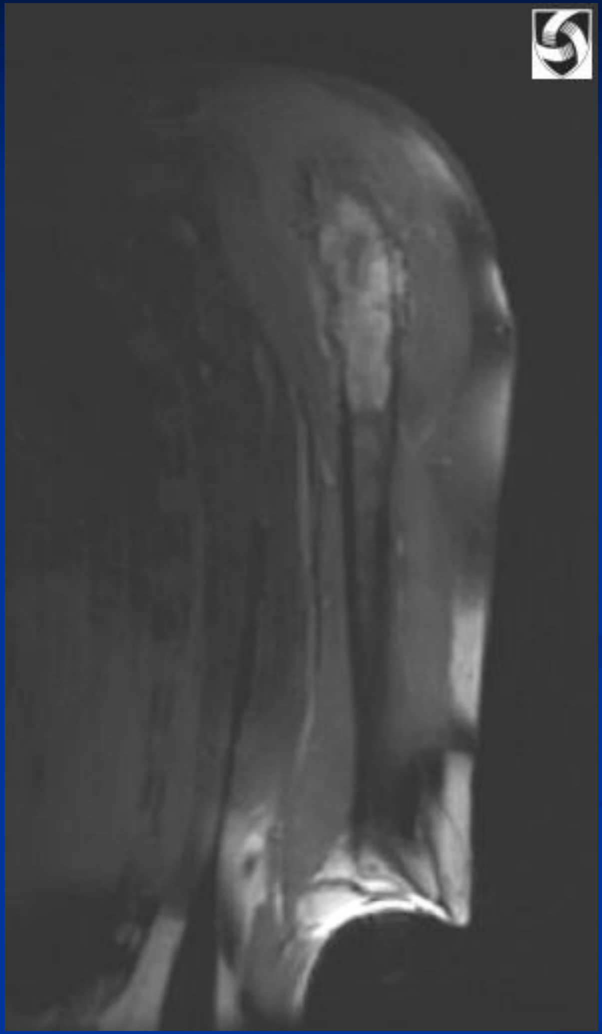
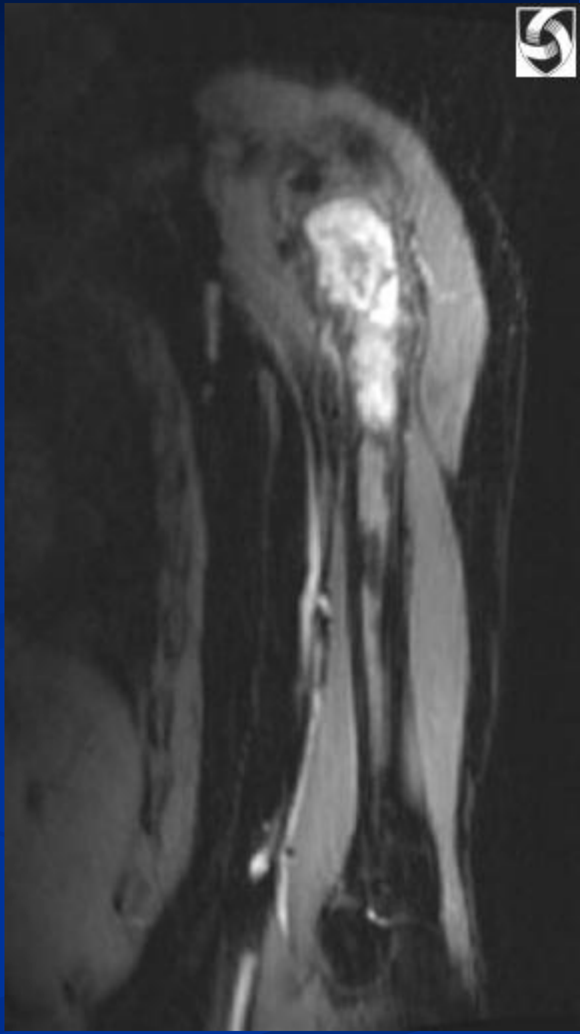
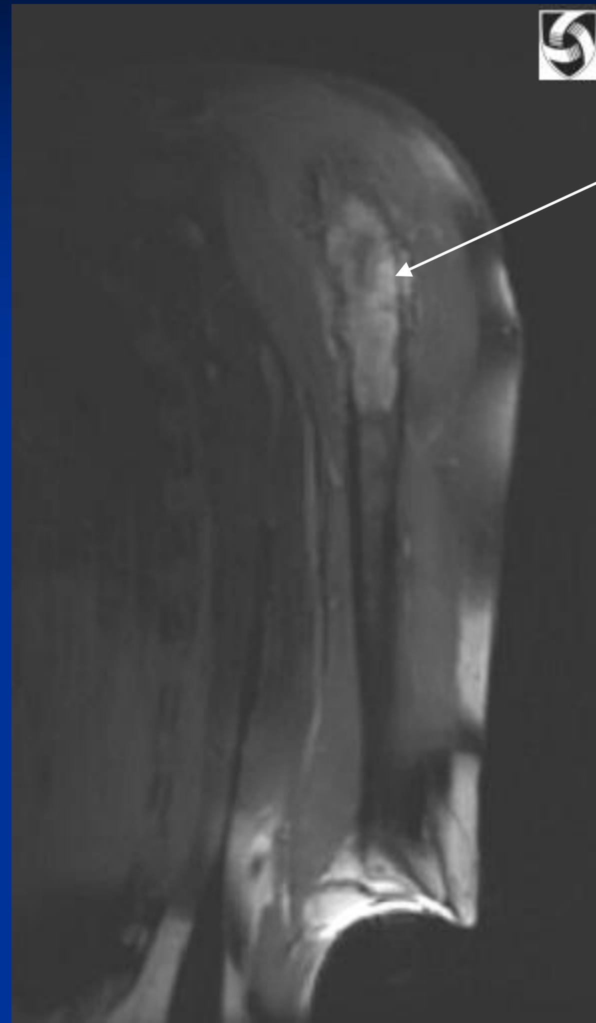
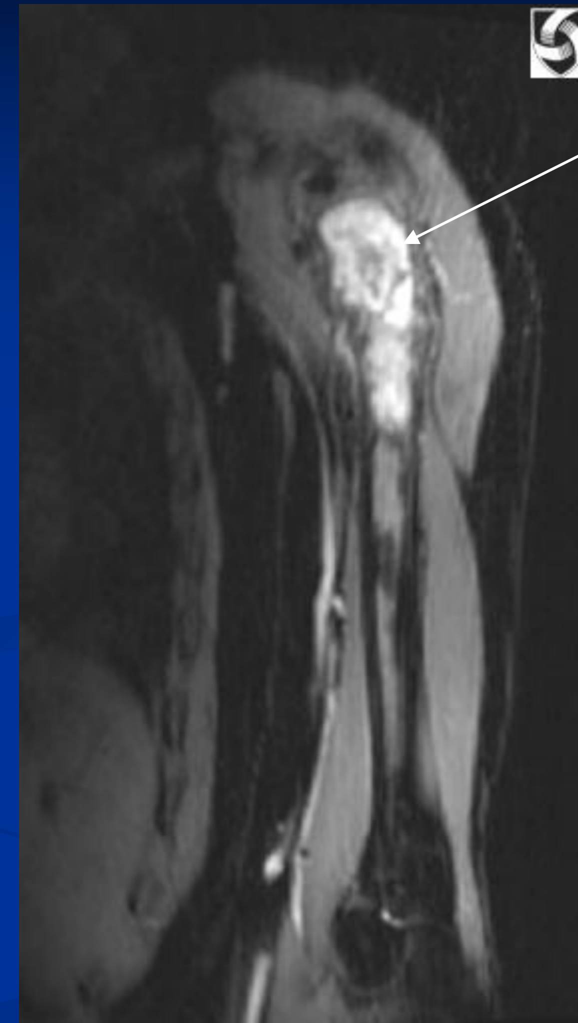




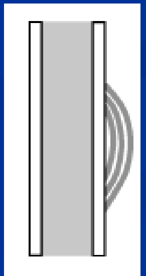
Another most excellent example of "onion-skinning"

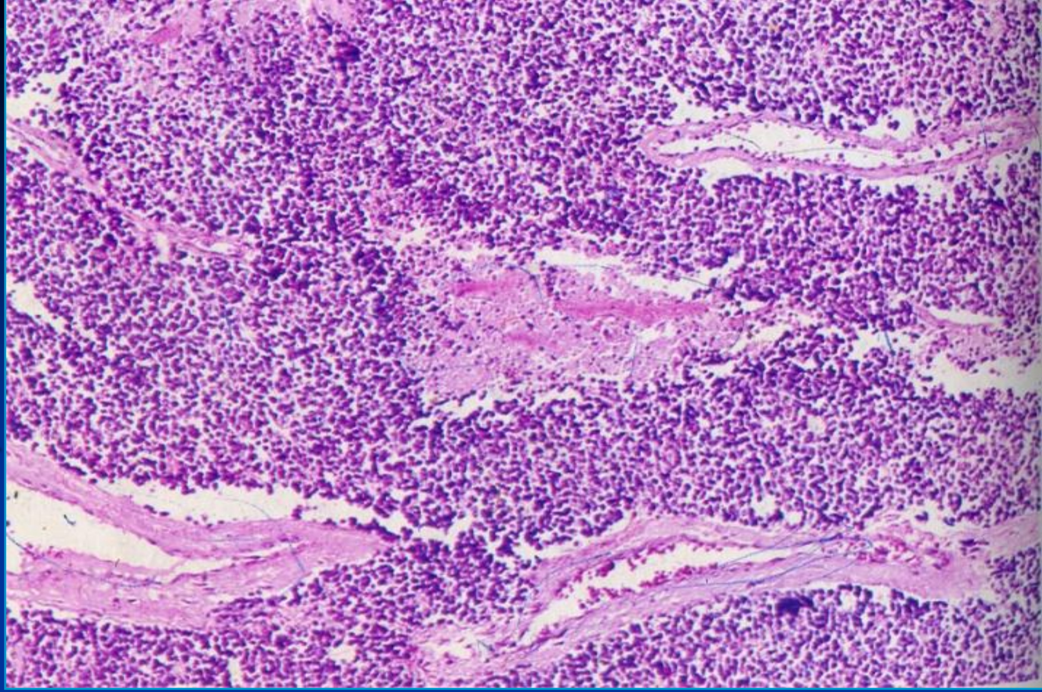




Dx: Ewing's Sarcoma (or PNET)

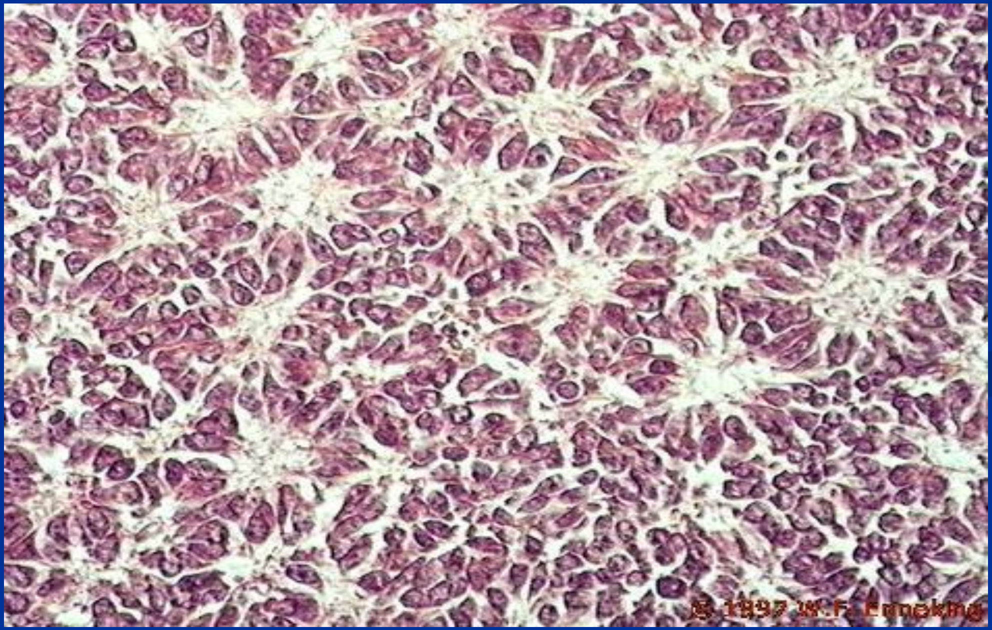
- #2 primary bone malignancy in children (5-15) is most common age group(Ewing family)
- common in diaphysis of long tubular bones or in large flat bone
- Lytic tumor with permeative margins extending into the soft tissue
- Periostial rxn creates sheets of reactive bone in an **<u>onion-skin</u>** fashion

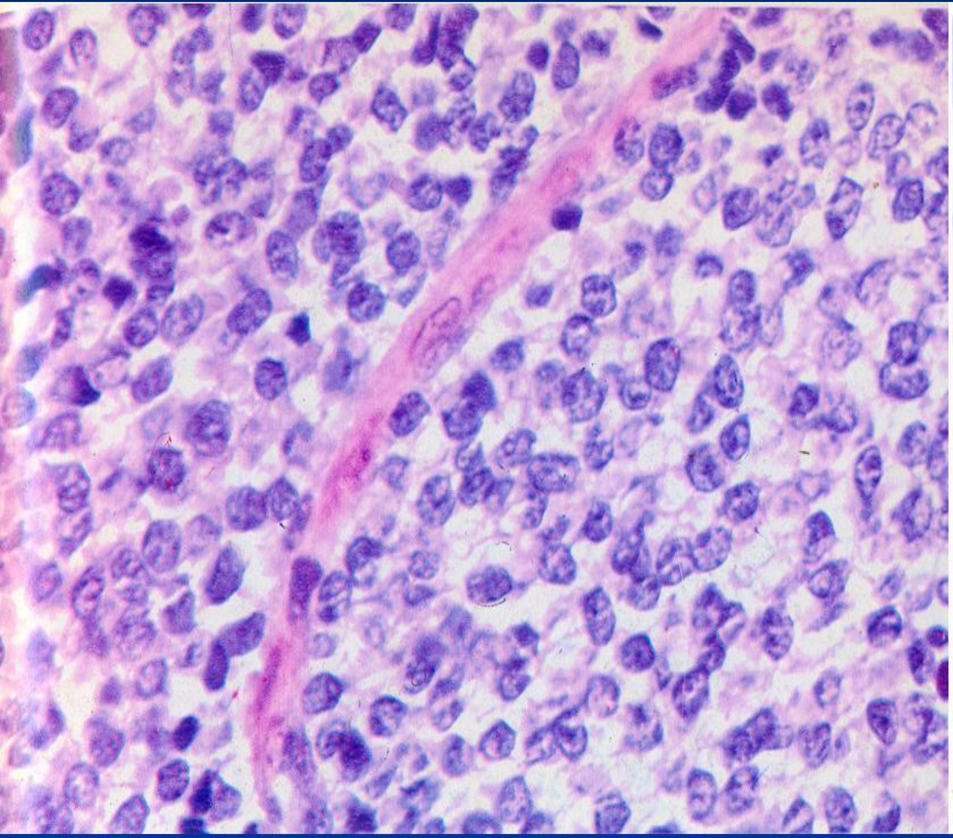


Microscopy Ewing sarcoma

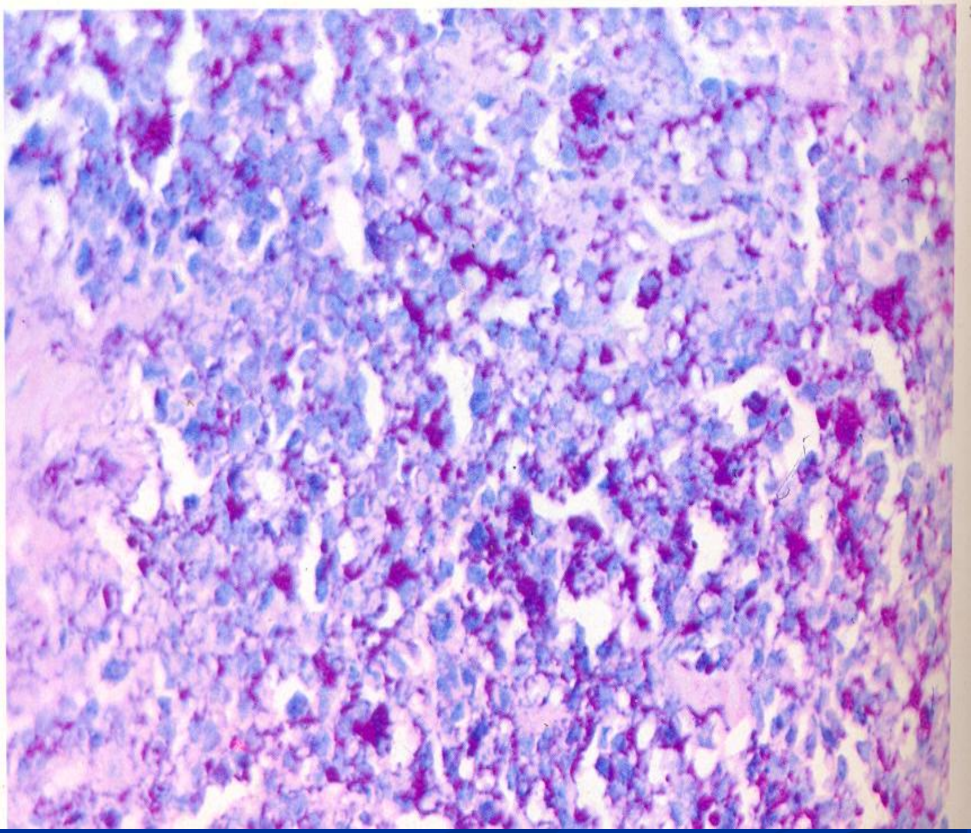


- Sheets of uniform, small round cells (slightly > lymphocytes)
- Large nuclei, scanty clear cytoplasm
- Homer Wright rosettes
- Necrosis +++
- Cells PAS + (Glycogen content)





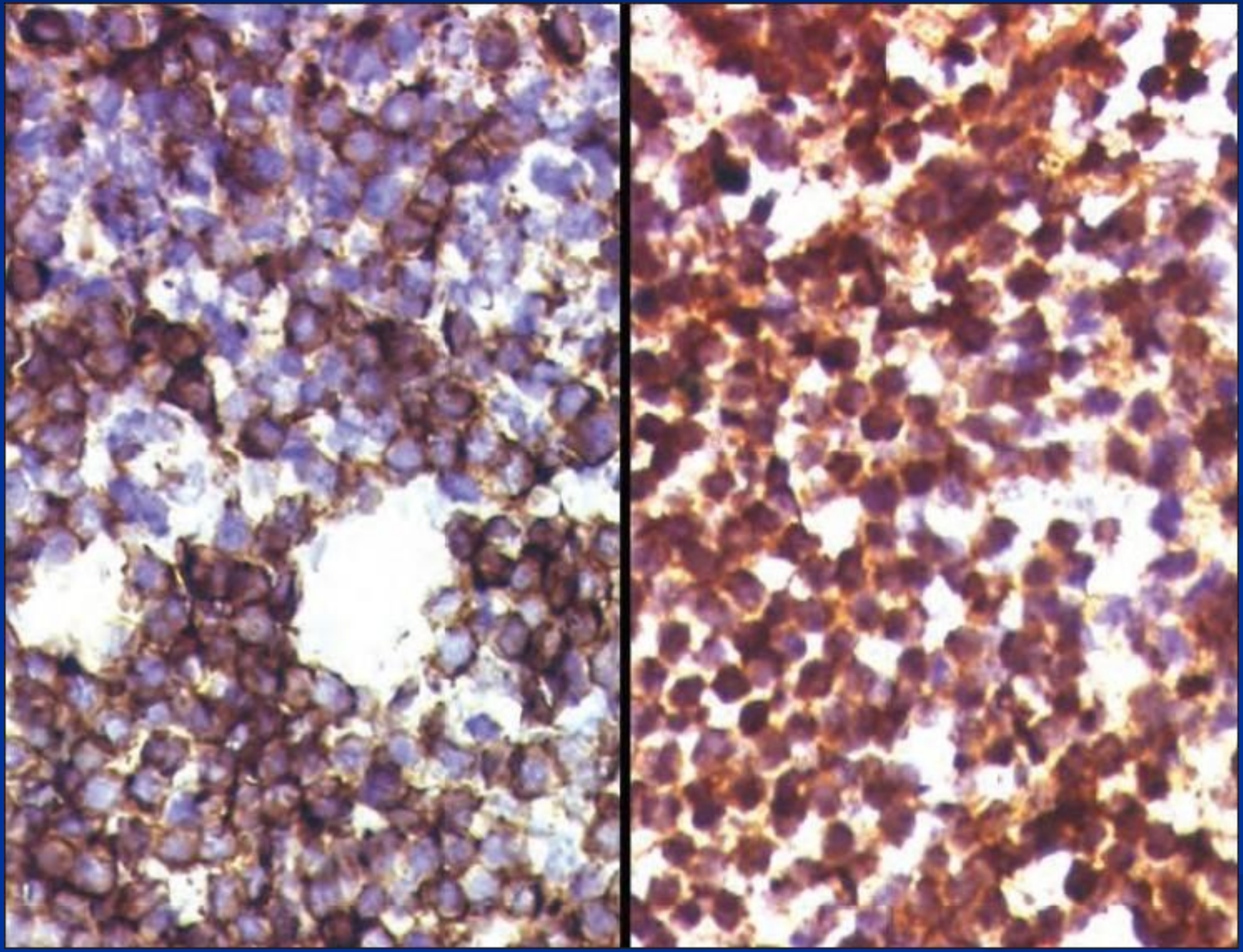
Small round cells



Pas +

- D/D Small blue cell tumor
- Ewing sarcoma/PNET
- Neuroblastoma
- Lymphoma
- Rhabdomyosarcoma

CD99 FLI1 (MIC-2)



Ewing sarcoma Immunohistochemistry

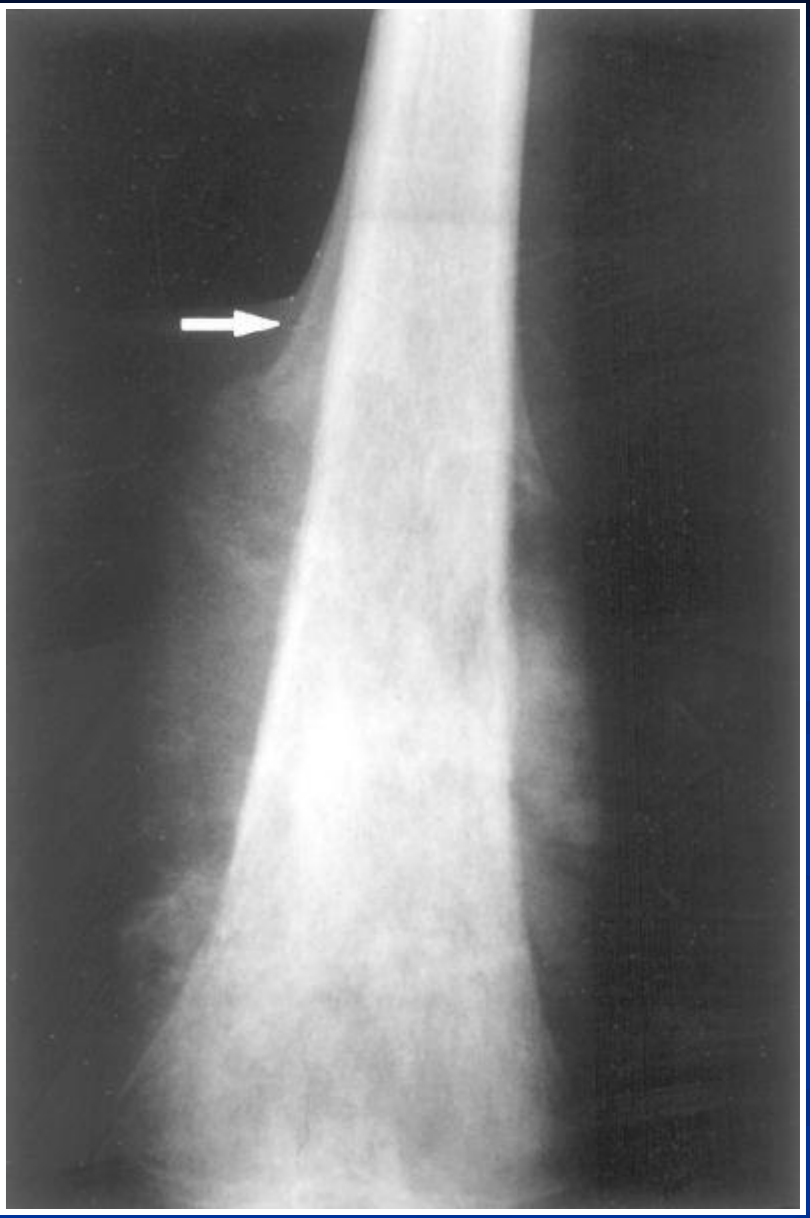
Cytogenetics-Ewing sarcoma t(11;22)

EWS gene 22 q 12

- **FL I1q24**
  - **t (11:22) (q24:q12)**
  - **EWS/FL I1**
  - **(Most common Fusion gene-Oncogene)**
  - **85%**
- **ERG 21q22**
  - **t (21:22) (q22:q12)**
  - **EWS/ERG**
  - **5-10%**
- **ETV 7p22**
  - **t(7:22) q(22:12)**
  - **EWS/ETV**
  - **< 1%**

Case II

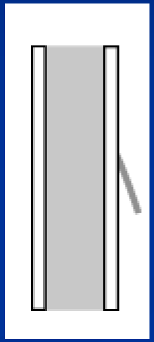
- 21 yr old male with
  - an ongoing aching leg pain for the past 6 months which he has put off seeing a doctor for.



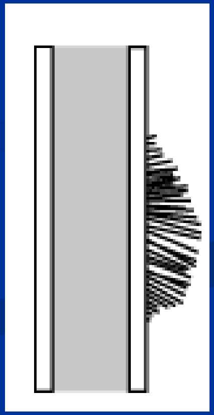
Dx: The dreaded Osteosarcoma

- #1 primary bone malignancy
- Associated with RB1 and p53 gene mutations
  - 1000x greater risk w/ Hx of hereditary retinoblastoma
  - Member of Li-Fraumeni Syndrome family
- Bimodal age spike: young and elderly
  - 75% <age 20
  - Osteosarcoma in elderly usually - secondary
    - Paget Dz, bone infarcts, history of radiation, etc

- Metaphysial tumor
- 60% at the knee (distal femur or prox tibia)
- Radiographic terms to know:
  - Codman’s Triangle:

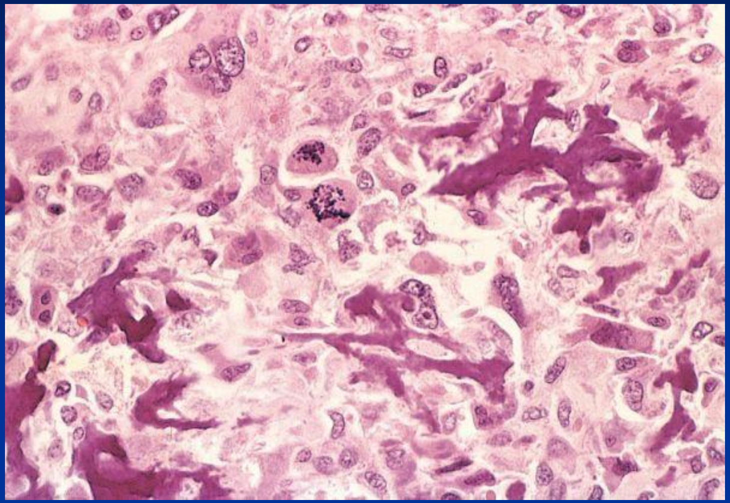


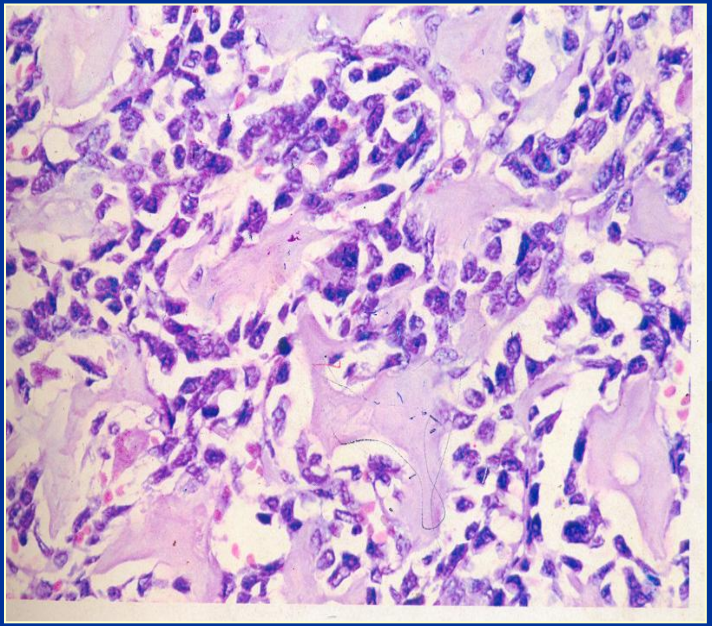
  - “Sunburst” periostial formation:
  - “Hair on end”



Microscopy

- Hyperchromatic spindle cells or polygonal
- Osteoid production by the tumour cells
- Reactive bone formation
- Bizarre Tumour giant cells





Case III

A 65 year old man comes with pathologic fracture of humerus

Metastatic Lesion

- Most common malignant lesion of bone
- Bone is 3rd on the list of favorite places for mobile Ca
- Typically multifocal BUT **renal and thyroid carcinomas are notorious for producing only a solitary lesion**
- Can be lytic, blastic, or both:
  - **L**ung is **L**ytic, **P**rostate **P**roduces, **B**reast does **B**oth

Mets (cont)

- Adults
  - Lung
  - Prostate
  - Breast
  - Kidney
- Children
  - NB
  - Wilm's
  - OS
  - Ewing's
  - Rhabdo

    myosarcoma

Intraoperative procedures frozen Section

separate bit should be reserved for frozen sections

Inherent problems- Decalcification not possible

- Adequacy of the Bx can be studied
- **Indications - To differentiate**
  - B. tumors from inflamm. lesions
- Non neoplastic conditions like osteomyelitis
- Mets , Lymphohematologic malignancy

Cryostat

Tumor Markers

- are substances, usually proteins, produced by the body in response to **cancer growth or by the cancer tissue itself**
- that may be detected in **blood, urine, or tissue samples.**
- Some tumor markers are specific for a particular type of cancer.
- These markers may also be elevated in non-cancerous conditions.

Tumor Markers

Glycoconjugates on *the* cell surface

- involved in adhesion, motility, metastasis
- can induce immune response
- expressed early in malignancy

  shed by cancer cells into serum

Tumor Markers

Monoclonal antibodies can be developed and used to detect these antigens on the cancer cell surface or in the serum

Tumor Markers

Pathologists can now help with diagnostic dilemmas
Eg: Unknown primary- Adenoca profile: TTF1, CK-7, CK20

Mets from…..

- Focus on

  Serum Tumor Markers

  esp: CEA

  CA 125

  CA 19.9

  also AFP, BHCG, PSA, CA15-3

Tumor markers- Key features

- Lack of specificity
- Cancer heterogeneity
- False negatives
- Benign diseases positive CA 125 or CEA
- Smokers have raised CEA
- Many men (20-40% !?) die with,
  not from, prostate ca.