Anatomy

- There are 2 lordotic segments:
  - Cervical: .......................................................25° cervical lordosis
  - Lumbar .......................................................50°
- And 2 kyphotic segments:
  - Thoracic ......................................................35°
  - Sacrococcygeal
- These allow for the head to be directly over the pelvis \( WB \) line just anterior to S1
- Cross sectional anatomy of the spine reveal 3 columns
  - Anterior column:
    - Anterior longitudinal lig
    - Anterior ½ of body
    - Annulus fibrosus
    - Disc
  - Middle column
    - Posterior ½ of body
    - Annulus
    - Disc
    - Posterior longitudinal lig
  - Posterior column
    - Facets
    - Neural arches
    - Ligamentum flavum
    - Interspinous lig
- Fracture instability is a big issue in spine surgery as it affects the plan for treatment
  - Compression fractures that affects the anterior and middle columns \( \rightarrow \) stable
  - Distraction and fracture dislocations are unstable
  - Gains Classification scores the degree of comminution, displacement, & kyphosis
  - Fractures that score > 7 points are unstable and need anterior and posterior fixation
  - Sometimes the answer for the Q of instability is difficult and need extensive clinical and dynamic radiologic studies
**Osseous anatomy:**
- Cervical spine protect brain stem & sp cord and suppor the head
- 50-60% of flexion-extension occur between base of skull and C1
- 50-60% of axial rotation occurs at the level of C1-C2
- Remaining motion occur between C2 and the CxTh segment
- Cup and ball type of articulation between C0-C1 allows coronal and sagittal rotation
- Horixontal & flattened orientation of C1-C2 allow predominantly axial rotation
- Insipite of this unstable osseous anatomy that permits wide range of flexion extension & rotation stability comes from ligamentous structures
- The subaxial spine contributes approximately half of the flexion-extension and rotation of the cervical spine.
- The orientation of the posterior facet joints (45-degree angle in the coronal plane) allows for more mobility than is possible in the other spinal regions.
- Motion at the facet joints is also complemented by concomitant motion between vertebral bodies through the intervertebral disks.

**Biomechanics:**
- Four smooth curves causes flexion, extension, bending to occur together in harmony by the so coupled movements
- The mobile cx & lumbar areas are separated by rigid thoracic vertebral segment; this creates stress risers at cervico-thoracic & thoraco-lumbar junctions
- Ideally, centre of gravity passes through cx vertebral bodies → anterior to Thx vertebrae → intersecting ant corner of sacrum
- So most of spinal column experience compressive forces anteriorly and tensile posteriorly; except in lumbar lordosis this KB reversed
- Try WB component in compression is cancellous bone → adapted for this
- While cortical bone is responsible only for 10% of compressive strength
- Marrow element has viscous property → hydraulic system provide both strength and dampening effect (energy absorption)
- Posterior column → less massive osseous elements → designed to attachment for lig & tendons are mostly collagenous & extremely strong to tension (most stabilizing element of posterior column)
- Lig attachment at a distance form instantaneous axis of rotation gibes them excellent mechanical attachment
- Discs act as force transmission & dampening unit for ant column
- Disc nucleus transmit axial load from body to body& transfer compressive force tension force in the annulus
- Outer layer of annulus → important for rotational stability
- Annulus thick ant & lateral; and thin posterior & postero-lateral corner → stress risers this is a common site for disc herniation
### Instantaneous axis of rotation:

- Axis around $\Omega$ relative motion of an object occurs from one position to another
- It is a geometric concept that locate the line around $\Omega$ vertebral body rotate
- It is not necessary to be contained in the vertebral body
- Its position is affected by:
  - Degenerative changes
  - Loss of anatomic stabilizers
  - Anatomic destruction
- All these causes shift IAR towards uninjured segment (but in certain limits)
- IAR is important to know; to put any construct in mechanically favorable position far distant from IAR $\rightarrow$ moment arm of the implant $\rightarrow$ mechanical advantage
- Atlanto-Occipital IAR (lateral bend) = 2-3 mm above the dense apex in the middle line
- Atlanto-axial IAR (flexion & extension) is in the dense
- Cervical IAR for flexion $\rightarrow$ anterior body; while for lateral bending is centre of the body
- Lumbar IAR for rotation $\rightarrow$ near the posterior annulus; if destroyed $\rightarrow$ IAR migrates posterior; if posterior is destroyed $\rightarrow$ migrates anterior
- Structures far from the IAR are responsible for constraining the motion; e.g. anterior longitudinal lig (ALL) & anterior annulus are the structures most important in rotation control in intact spine
- If anterior column is injured $\rightarrow$ rotational instability
- Lumbar IAR for flexion is the within nucleus pulposus
- In flexion if Ant column is destroyed IAR moves inf & post; if Ant & Middle columns are destroyed $\rightarrow$ IAR further back and inferior
- In extension if Posterior column (facetal joint) is destroyed $\rightarrow$ ant & inferior

![Diagram of Spinal Structures](image-url)
Circulation Of Spinal Cord

- **Three Main Trunks:**
  1. **Anterior Median Longitudinal Arterial Trunk** → is the main supply and are responsible for the more vascularity of the ant part of the body at the ALL
  2. Pair of **Posterolateral Trunks** near the posterior nerve rootlets.

1. The source of these trunks are:
   a. In the neck they come from ................ vertebrala, costocervical & thyrocervical trunks (In 60% additional source arises from the ascending pharyngeal of ext.carotid artery)
   b. In thoracic and lumbar areas from ...... Aorta
   c. In sacral area from .................. lat sacral, mid-sacral, 5th lumbar, & iliolumbar

2. Direction of flow in the blood vessels of the spinal cord. The three longitudinal arterial channels permit reversal of flow and alterations in the volume of blood flow in response to metabolic demands. Relative demands of gray matter > white matter and these arterial trunks are largest in the cervical and lumbar near the girdle plexus.

3. Trunks connect: by mean of segmental vessels

4. **Segmental Arteries Of The Spine:** at every vertebral level a pair of segmental arteries that run to reach the intervertebral foramen (External Distribution Point) and give rise to many branches:
   a. Vertebral branch: richly supply the anterior aspect of the body and ALL (This is the most vascular point of the vertebra)
   b. Dorsal branch: supply the arch
   c. Spinal branch

5. **Spinal Branches** enters the intervertebral foramen and give rise to superior, inferior, transverse branches that anastomose the above, below, other side artery → transverse arterial rings (Vertebral Arterial Plexus):
   a. Internal arterial circle of the cord → Radicular Vessels to the cord
   b. Extradural arterial circle: gives 3-4 Vertebral Nutrient aa. enters the body via posterior nutrient foramen. These are of two types:
      o End arterioles that reach the metaphysis
      o Anastomotic arterioles that anastomose with there counterpart from segmental a.

6. **Artery Of Adamkiewicz.** Is the main feeder of lumbar cord; arise at T9 to 11 on the left side, the most important feeder is the anterior longitudinal arterial channel of the cord; but all are important and must be preserved. Spinal blood supply is very variable

**Venous Drainage:** Metaphyseal minute tributaries drain into:
1. centre of the body → large valveless Basivertebral v. → emerges from nutrient foramen → anterior internal plexus
2. Via emissary viens to anterior external plexus
3. From posterior arch tributaries drain into the posterior internal and external venous plexuses
4. All these plexuses form an extensive network called Batson’s vertebral plexus that drain into the segmental vein
Embryology

- A typical vertebra is ossified from three primary centres: 9-12 wk
  1- 2 at the side of vertebral arch → transverse process, lamina, spine, & pedicles
  2- One in the centrum. The body's major part, the centrum, ossifies from a primary centre dorsal to the notochord. (Centra are occasionally ossified from bilateral centres which may fail to unite.
- During early postnatal years the centrum is connected to each half neural arch by a synchondrosis or neurocentral joint.
- During the first year the arches unite behind each other, first in the lumbar → thoracic → cervical
- Centra unite with arches about the third year, the lumbar being the last at 6th year
- Until puberty the upper & lower surfaces of bodies + apix of transverse + apix of spinous processes are cartilaginous = five secondary centres appear:
  1- 2 at the apex of each transverse
  2- Apix of spinous process
  3- 2 annular epiphyseal 'rings' for circumferential parts of upper and lower surfaces of the body
- Costal articular facets are extensions of the annular epiphyses.
- 2ry centres fuse at ~ 25 years. In bifid cervical spinous processes there are two secondary centres. The annular 'epiphyses' of vertebrae probably cannot be equated with epiphyses of long bones. In most mammals they are complete osseous discs.
Allen Classification Of Cervical Fractures

Compression Flexion

Vertical Compression

Distraction Flexion

Compression Extension

Destruction Extension

Lateral Flexion

Stage I

Stage II

Stage III

Stage IV

Stage V
Prevalence
1. 60-80% of people will have LBP sometime in their lives.
2. 90% LBP resolves in 6w, 75% may experience symptoms & disability 1y after

Types of Back Pain:

1. Discogenic Back Pain
   - Pain from innervated ligamentous layer of the annulus & LDP
   - It is midline & worse with lordotic postures, bending & lifting

2. Facetal Joint Pain:
   - Each facet is enervated by 2 nerves (non specific and difficult to localize)
   - 2 types of pain:
     - Arthritic Pain
     - Root compression & lateral recess stenosis

3. Sacro-Iliac Pain:
   - Also may cause back aches: usually due to inflammatory arthritis e.g. Ank Sp

4. Radicular Back Pain: has a Dermatomal Pattern
   - External pressure from a facet, hypertrophied LF, NPH, Pseudoarthrosis of lytic Spondylolitis
   - Ischemia of blood flow
   - Inflammation around e.g. TB, pyogenic osteomyelitis, discitis

5. Referred Back Pain: No dermatomal pattern (sclerotomal distribution)
   - Aortic Aneurysm
   - Visceral (DU, GB, Pancreatic disease, endometriosis, pleural disease)
   - Infection: UTI, PID
   - Hip Arthritis

6. Musculogenic Pain:
   - Inflammatory: myofacial $ or fibromyalgia rheumatica
   - Vascular: lumbar paraspinal ms compartmental $
   - Exertional

7. Iatrogenic Back Pain
   - Dural adherions
   - Post surgical instability
   - Post operative discitis; arachnoiditis

8. Ganglionic Pain:
   - Compression by an abscess or tumor → edema → ® the release of substance P & CGRP (calcitonin gene related peptide) → ® the nociceptors → pain

9. Psychogenic Back Pain
   - Must exclude organic pathology
   - Waddell’s inappropriate signs often present:
     - a. Painful Jackson's Test (axial skull compression)
     - b. Painful Pelvic rotation: passive rotation of shoulders and pelvis together
     - c. Resisted hip flexion
     - d. Non-dermatomal sensory loss
     - e. 'Cogwheel' (give-way) weakness
     - f. Inconsistent SLR & a clinical Dx
     - g. Widespread tenderness
     - h. Overreaction - disproportionate expressions, or tremor during examination
   - >3 present = be wary of operating ≈ nonorganic features
**Discogenic Pain**

- Normal discs have sensory nerve endings of the sinovertebral nerve (recurrent br of the spinal n Ω supply the dura, PLL, annulus) in the outer 1/3 of the annulus
- Disc stimulation studies (using either hypertonic saline or contrast media) showed normal discs do not cause pain
- Pain correlate with the degree of disc fissuring & not with disc degeneration
- Discogenic pain can result from infection (discitis), a torsional injury (circumferential tear of the annulus) and internal disc disruption
- Internal disc disruption cannot be diagnosed clinically but only after post-discography CT
- The correlation between reproduction of pain and a grade 3 tear is very strong

<table>
<thead>
<tr>
<th>Grade</th>
<th>Disruption</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grade 0</td>
<td>No disruption evident</td>
</tr>
<tr>
<td>Grade 1</td>
<td>Disruption to inner 1/3</td>
</tr>
<tr>
<td>Grade 2</td>
<td>Disruption to middle 1/3</td>
</tr>
<tr>
<td>Grade 3</td>
<td>Disruption to outer 1/3</td>
</tr>
</tbody>
</table>

**Xeroradiography:**

- Still it is an ionizing radiation (electro-magnetic field), but it is received on a special photoconductor plate that enhances images

**Sprung back**

- Pain from upper sacrum to knees
- Due to PLL tear + posterior annulus
- Tenderness, depression may be felt
- ✴ flexion, otherwise is normal, SLR painful > 100
- ttt: fusion if resistant

**Osteitis Condensans Ilii**

- Sclerosis of one or both sacro-iliac joints
- Unknown etiology
- More in young females
- PXR is diagnostic

**Lumbago** (fibromyocitis of lumbar muscles)

- Acute painful spasm and tenderness of lumbar ms
- Uncertain etiology & pathology
- Tender nodules may be felt
- Usually attributed to cold exposure
- ttt: rest, NSAIDS, warmth, local Novocain inj

**Fibro-Myalgia**

- Regional myofascial pain
- ACR Criteria for diagnosis:
  1. Widespread pain
     - ≥ 3 months:
     - Pain is bilateral
     - Pain is above and below the waist
     - Pain is axial (neck, mid-back, low back)
  2. Pain must be in ≥ 11 of 18 fibromyalgia tender points
     - These points must have marked tenderness to palpation
     - ± refer pain, and not just “tender”
     - Pain is elicited é = 4 kg of pressure
Disc Degeneration

Definition:
- Altered intervertebral disc structure & production of pain especially at L4-5, L5-S1

Aetiology
- It is not exactly clear why discs degrade, but it is not related to aging
- Theories put forward include:
  1. End plate fracture which then causes disruption of the delicate nuclear homeostasis
  2. Autoimmune process
- Intervertebral discs are able to withstand large forces without herniation:
  1. Load to failure = 10,000N (vertebral end plate fail first)
  2. Intrinsic cohesiveness; even if an annular tear, herniation occur only to degraded disc
- Predisposing factors for degradation:
  1. Smoking (↓ O2 tension at the annulus)
  2. Chronic cough and constipation
  3. Bad postural habits in work, office, driving,…
  4. Repetitive trauma
  5. Obesity

Pathology:

Hill Degenerative Cascade:

1. Stage of Dysfunction:
   - Minor disc tears, Facetal synovitis, & ms sprain
   - Axial dull aching ms pains & trigger points

2. Stage of Degeneration:
   - Proteoglycans disturbance (↑ chondroitin sulphate) + ↓ water content
   - ↓ collagen content of the nucleus bulbus
   - Nucleus appear dry and brown & less gelatinous (Black disc $ on T1)
   - All lead to inelastic nucleus & less stress sharing function
   - This leads to stresses over:
     1. Annulus fibrosis → fissuring // to end plate → herniation
     2. End plate → failure
     3. Facetal joints

3. Stage of Spondylosis:
   - Reactive Bone Formation:
     1. Around the end plate herniation → Schmorl’s nodes
     2. Around protruded disc δ periosteal elevation → marginal osteophytes
     3. Around the facets → osteophytes & OA
   - Flattening of the disc

4. Stage of Stabilization:
   - As the reactive bone formation continue & more osteophytes formation → stabilization of the adjacent vertebrae


**Acute Disc Prolapse**

**1. Lumbar Disc Prolapse**

**Definition:**
- Acute disc herniation that produce Neurologic compressive disorders and pain

**Epidemiology**

**3 Ages**
- Mostly in **Middle Age**: very young and very old seldom have acute disc prolapse
- In adolescents look for infection, benign tumours and spondylolisthesis
- In the elderly look for vertebral compression fracture and malignancy

**3 Warnings**
- **Sciatica** is referred pain ( \( \approx \) prolapse). It can also come from facet, SI joints or infection
- Maximum two **Levels**, if multiple levels \( \rightarrow \) suspect neurological cause
- Severe, **Unrelenting Pain** is not a feature of disc prolapse; suspect tumour or infection

**3 Major DDx**
- **Inflammatory** disorders such as infection, ankylosing spondylitis
- **Vertebral Tumours**: cause severe pain and spasm
- **Nerve Tumours** such as neurofibromata of cauda equina \( \rightarrow \) sciatica é continuous pain

**Pathology:**
- The condition occur ð:
  a. Physical stress: combination of flexion + compression (mainly on L4,5 or L5,1) where stress is more severe
  b. Disturbance of hydrophilic properties of the nucleus
- At first: there is posterior bulge of the disc é out rupture
- Eventually: the annulus will rupture usually postero-lateral, but may occur central
- Neurological manifestation occur due to:
  o Compression of the roots of the level below é posterolateral bulge (90%)
  o Compression of the root of the same vert above é far lateral bulge
  o Compression of the multiple roots centrally (Cauda Equina) é central bulge
  o Compression of the cord (Conus Medullaris) é central bulge at \( T_{12}L_{1} \)

**Nerve Root Affected**

<table>
<thead>
<tr>
<th>Disc</th>
<th>Post Lat</th>
<th>Motor</th>
<th>Sensory</th>
<th>Reflex</th>
<th>Far Lat root</th>
<th>Central</th>
</tr>
</thead>
<tbody>
<tr>
<td>L2/3</td>
<td>L3</td>
<td>knee extension</td>
<td>Ant knee</td>
<td>Knee jerk</td>
<td>L2 ( { ) hip flexion ( } )</td>
<td>Cauda</td>
</tr>
<tr>
<td>L3/4</td>
<td>L4</td>
<td>Dorsiflexion</td>
<td>Medial leg</td>
<td></td>
<td>L3</td>
<td></td>
</tr>
<tr>
<td>L4/5</td>
<td>L5</td>
<td>Hallux extension</td>
<td>Lat leg</td>
<td>Medial hams</td>
<td>L4</td>
<td></td>
</tr>
<tr>
<td>L5/S1</td>
<td>S1</td>
<td>Plantar flexion</td>
<td>Lat foot</td>
<td>Ankle jerk</td>
<td>L5</td>
<td></td>
</tr>
<tr>
<td>S1/S2</td>
<td>S2</td>
<td>knee flexion</td>
<td>Back leg</td>
<td>Lat hams</td>
<td>S1</td>
<td></td>
</tr>
</tbody>
</table>

Pain | Back & leg | Leg | Back
Clinically:
- C/O:
  1. Sudden Severe **Backache** é lifting a heavy object → inability to straighten up
  2. After few days symptoms of nerve irritation **Sciatica** appear:
     1. Referred to buttocks, back of thigh, & leg more to one side
     2. \( \uparrow \) é cough and strain
  3. Few days later: symptoms of n compression appear **Radiculopathy** more to one side
     1. Sensory symptoms; hyposthesia, paraesthesia
     2. Motor weakness
  4. If central compression occur **Cauda Equina** manifestation occurs:
     1. Bilateral LMN weakness in the legs
     2. Loss of perianal sensation **Saddle Anaesthesia**
     3. Insensinate UB, painless retention é overflow: urinary incontinence
     4. Fecal incontinence
  5. If central compression at a higher level D12L1 (not common) **Conus Medullaris** lesion:
     1. Bilateral LMN weakness at L1 + Bilateral **UMNL** motor weakness below
     2. L1 **Sensory Level**
     3. Fecal urinary incontinence (insensinate UB, painless retention é overflow)
- O/E:
  1. Standing: ............................................................(postural changes)
     1. Inspection:
        [1]. **Sciatic List** (scoliosis): pt bend to one side \( \delta \) ms spasm
        [2]. **Flexed Knee**: pt bend it to \( \downarrow \) tension on the sciatic n
     2. Palpation: Back **Tenderness** max on lower vertebrae
     3. Movements: Limited all back movements +VE **Schober Test** 3 points in posterior midline are marked and the pt is asked to bend & straight up & measure the diff
  2. Supine: ..............................................................(stretch signs)
     1. **Lasegue's**: SLR \( \rightarrow \) pain at 70° at buttocks and calf (rather than thigh and back) \( \rightarrow \) slow drop of leg \( \rightarrow \downarrow \) pain \( \rightarrow \) passive dorsiflexion \( \rightarrow \) reappearance of pain
     2. **Brudzinsky's**: also passive neck flexion \( \rightarrow \) reappearance of pain
     3. **Kernig's** sign: Flexion of the knee \( \rightarrow \) relief of pain (if persist = +ve **Buttock** problem test)
     4. **Bowstring** sign: SLR + flex the knee 20° + press ! lat popliteal n \( \rightarrow \) pain reappear
     5. **Fajersztajn** sign: well leg raise \( \rightarrow \) pain on the affected leg (=Cross over sign) and indicates large axillary disc protrusion of the other side
     6. **Bilateral SLR**: pain after 70° raising
     7. **Hoover** test for malingering: if pt can not do active SLR \( \rightarrow \) put 2 hands below each heels and feel if he is trying (you feel a push over the other palm) or he is not trying
  8. **Prone Knee Bending** sign: max flexion of knee in prone position \( \rightarrow \) unilat. Lumbar pain (femoral stretch test) = L2,3 irritation

<table>
<thead>
<tr>
<th>Postive degree</th>
<th>Lasegue</th>
<th>Bilateral SLR</th>
<th>Brudzinsky</th>
</tr>
</thead>
<tbody>
<tr>
<td>30°-70°</td>
<td>Hip</td>
<td>Sacroiliac</td>
<td>if (+ve) Lasegue + (-ve)</td>
</tr>
<tr>
<td>70°</td>
<td>Sciatic stretch</td>
<td>Lumbar</td>
<td>Brudzinsky sign ( \rightarrow ) hamstring tightness</td>
</tr>
<tr>
<td>&gt;70°</td>
<td>Sacroiliac or facetal</td>
<td>Lumbar</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>L5 impairment</th>
<th>S1 impairment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Motor weakness</td>
<td>EHL &amp; ST</td>
</tr>
<tr>
<td>Sensory</td>
<td>Outer leg &amp; dorsum foot</td>
</tr>
<tr>
<td>Reflex</td>
<td>Brisk knee (weak ST) ( \downarrow ) ankle jerk</td>
</tr>
<tr>
<td>Sensory</td>
<td>Outer foot &amp; dorsum 5th toe</td>
</tr>
<tr>
<td>Motor weakness</td>
<td>TP &amp; TAc &amp; peronei</td>
</tr>
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</table>
Radiological

1- **PXR:**
   - [1]. Narrow disc space
   - [2]. Traction spur
   - [3]. Facetal arthropathy
   - [4]. Straightening of the spine (ms spasm)

2- **CT:** More accurate and reliable

3- **Myelography:** to exclude intrathecal tumor & confirm disc (limited value after MRI)

4- **MRI:**
   - [1]. Assess the cord and root condition
   - [2]. Confirm the disc & its extent & size
   - [3]. The study of choice

Natural History

- After:
  - 1st episode 90% ................................................. improve and do not relapse
  - 2nd episode 90% ................................................. improve and 50% relapse
  - 3rd episode 90% ................................................. improve and 100% relapse
- Regardless of treatment, impaired motor function had a good prognosis whereas sensory deficits remained in almost one half of all the patients.

DD:

- **Inflammatory condition:**
  - o  ♦ stiffness
  - o  ♦ ESR
  - o  Erosive on PXR

- **Vertebral tumor**
  - o  Severe pain (osteoid osteoma, osteoblastoma)
  - o  Marked spasm
  - o  PXR lesion

- **Nerve tumor:**
  - o  Sciatica
  - o  Continuous pain
  - o  MRI

Red Flags for Back Pain:

1- Fever
2- Deformity
3- Loss of wt
4- Neurological Acute
5- Past history of malignancy
6- Bladder
Treatment (Majority require no surgical intervention)

Non-operative ..................................................90% effective
1- Bed rest in Fowler position & knee flexed ± Traction for 2 weeks
2- Pelvic corset
3- NSAID's
4- Physiotherapy: Back classes helpful
5- Epidural injections of LA ± steroid 80-120mg depo medrol
6- If all failed chemonucleolysis by chemopapain (dangerous & less effective than surgery)

Operative indication
1- **Cauda** equina syndrome is considered an emergency
2- **Persistent** leg pain despite adequate conservative measures >3wk
3- Neurological **Deterioration** in spite of conservative ttt

Standard operative treatment
1- **Intervertebral discetomy** (Fenestration of LF + Partial Laminectomy): for central discs
2- **Inter Transverse discetomy** for far foramenal disc
3- **Micro-discetomy**: under microscopic magnification
   o Shorter stay, mini incision
   o Need experience + intraop PXR
   o More complication (Bleeding, infection, limited field)
   o Dural tear: headache + soaked wound & brown halo
      - +ve ß 2 transferin
      - Small ..................nothing to be done → bed rest
      - Medium ................interrupted water tight sutures
      - Large ....................autogenous fat graft or gel foam or advac-L
4- **Percutaneous suction discetomy** (Automated Percut Lumbar Discetomy) APLD:
   o Rotatory shaver probe is inserted into the disc under PXR guidance
   o The probe cut the disc and then sucked via the same probe
5- **Percutaneous Laser discetomy** using the YAG or KTP laser beam
6- **Percutaneous Endoscopic discetomy**:
   o Series of dilators are introduced to the bone followed by insertion of wide cannula
   o Special endoscopic instruments are used to retract, cut, & excise the disc
7- **Percutaneous disc radio-ablation**:
   o Evaporization of the nucleus pulposus using the radiofrequency
   - Excellent treatment for eradicating leg pain but not for back pain
   - The addition of spinal fusion at the same time as discetomy has not been proven to be superior to simple discetomy and adds considerable morbidity
8- **Disc replacement surgery**: see later

Persistent pain after surgery:
- Residual disc material
- Disc prolapse at another level
- Root compression ð:
  o Facetal OA
  o Narrow lateral recess
- Late ð post-lamenectomy Instability **NEVER REMOVE > 1/3 THE FACET**
Rational for Disc Prosthesis:
- Replace the degenerative painful disc by a mechanically sound prosthesis
- It restores the height
- It restores the motion
- Regain the physiologic stiffness in all planes of motion plus axial compression
- With stand the physiologic stress and transmit it to the next level

Types:
[1]. Screw fixation
[2]. Staple fixation
[3]. Teeth fixation
[4]. Porous coated prosthesis
[5]. Macrotexure surface prosthesis
[6]. Hydrogel prostheses: replace of the NP only & retain the AF. Consist of hydrogel core constrained in a woven polyethylene jacket

Indications:
1. Diagnosis of Degenerative Disc Disease at the L4/L5 or L5/S1 level
2. At least six months of conservative treatment
3. Still under trial for cervical and thoracic disc prolapse

Precautions
1. Should be place centrally not to shift axial load to the facets
2. Avoid the destruction of facets and ligaments.
3. An artificial disc must exhibit tremendous endurance.
4. The intervertebral disc prosthesis ideally would replicate normal range of motion

Contraindications:
1. Previous back surgery (except discectomy, laminotomy or nucleolysis at the same level)
2. Multiple level degeneration, ligamentous laxity, Spondylolisthesis, or Scoliosis
4. Facetal pain
3. Osteoporosis, steroid ttt, metabolic bone disease, or autoimmune disorder
5. Morbid obesity

Advantages:
4. The device maintain the proper intervertebral spacing
5. Provide stability
6. Restore the normal shock absorbing mechanism of the spine
7. Less morbidity than the standard fusion techniques
8. Better functional out come
5. Percutaneous placement could be done é nuclear hyrdogel replacements

Complications:
1. Biomechanical problems:
   1. Bone resorption
   2. End plate failure
   3. Prosthesis failure
   4. Facetal over load and degeneration
2. Surgical complications:
   5. Neurological complications
   6. Vascular complications
   7. Visceral complications
3. Biological complications:
   8. Abnormal bone deposition
   9. Infections
2. Thoracic Disc Prolapse

- Very rare .................................................0.5% of all prolapsed discs (75% > T8 & very rare < T4)
- Cord compression and thoracic back pain from malignancy more common
- Most common in the 4th decade
- Thoracic back pain radiating round the chest with UMN signs in the lower limbs
- Investigate with MR scan
- Treatment
  1. Posterior surgery with laminotomy possible but the cord does not tolerate retraction
  2. The disc can be approached via a costotransversectomy, or transthoracic approach with fusion of the affected level with rib strut graft

Age Related Changes in the Intervertebral Disc

- **Proteoglycan synthesis decreases** with age thus the concentration of proteoglycans will diminish with age from 65% in early adult life to 30% by the age of 60 years
- The proteoglycans that persist are smaller in size and the concentration of **chondroitin sulphate falls** (keratan sulphate concentrations remain constant)
- This causes a **drop in the water content** from 88% at birth to 65% by age 75.
- However most of this dehydration occurs in childhood and early adolescence, with a decrease of only 6% from early adult life to old age.
- **Collagen content** in both the nucleus and the annulus but the concentration of **elastin**
- This loss of water and increase in collagen content causes the discs to become stiffer and less resistant to deformation and also less able to recover from creep deformation
- As the nucleus dries out and the becomes more fibrous it is less able to transmit forces to the annulus and the annulus then has to play a greater role in load transmission therefore subjecting it to greater stresses
- Disc height does not decrease with age but stays constant, with any loss of disc height representing a degenerative process as opposed to an age related change
3. Cervical Disc

Categorization By Odom

<table>
<thead>
<tr>
<th>Unilateral soft disc protrusion</th>
<th>Nerve root compression / radiculopathy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Osteophytes / hard disc</td>
<td>Nerve root compression / radiculopathy</td>
</tr>
<tr>
<td>Medial soft disc protrusion</td>
<td>Spinal Cord compression / myelopathy</td>
</tr>
<tr>
<td>Cervical spondylosis</td>
<td>Spinal Cord compression / myelopathy</td>
</tr>
</tbody>
</table>

Cervical Radiculopathy

Definition
- A condition caused by compression of a nerve root in the cervical spine

Incidence
- <1:1000
- Age: ........................................................... 40s and 50s.
- Cervical degeneration begin 10 years later than lumbar spine degeneration
- Sex - ♂ : ♀ .............................................. 3:2

Pathology
- Lateral Disc herniation ................. 50% (soft disc)
- Osteophyte formation in Lushka .......... 50% (hard disc)
- Developmental spinal stenosis .......... 5% also have (AP dr of spinal canal > 13)
- Compression site.............................. Entrance or Mid zones & occasionally at exit zone
- Frequency of root compressed .......... C7>C6>C8>C5

Symptoms
1- 1st symptom is **UNILATERAL NECK PAIN**: nape, supraescapular, scapular or interscapular regions
2- **RADIATING ARM PAIN**
3- Finger **PARASTHESIA**
4- **WEAKNESS**

Signs
1- Motor + Sensory + Reflexes
2- **SPURLINGS NECK COMPRESSION TEST** : Downward pressure to head ė the neck ext & tilt to the painful side ė pain. Usually positive but may be negative in chronic cases

<table>
<thead>
<tr>
<th></th>
<th>C5 (C4-5)</th>
<th>C6 (C5-6)</th>
<th>C7 (C6-7)</th>
<th>C8 (C7-T1)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neck pain</td>
<td>Suprascapular</td>
<td>Scapular / interscapular</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Arm pain</td>
<td>lat upper arm</td>
<td>Lateral arm</td>
<td>Posterior arm</td>
<td>Medial arm</td>
</tr>
<tr>
<td>Paresis</td>
<td>Deltoid (biceps)</td>
<td>Biceps (Deltoid)</td>
<td>Triceps</td>
<td>Intrinsics (triceps)</td>
</tr>
<tr>
<td>Sensory</td>
<td>none</td>
<td>Thumb</td>
<td>Index/long</td>
<td>little</td>
</tr>
<tr>
<td>Reflexes</td>
<td>Biceps</td>
<td>Triceps</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Differential diagnosis
- DDx is usually achieved by: neck pain < other symptoms, Spurling sign, Radiculopathy
1- Shoulder pathology
2- Thoracic outlet syndrome
3- Cubital tunnel syndrome
4- Tennis elbow
5- De Quervains disease
**Investigations**
- In contrast to the spinal cord in myelopathy, compressed root can rarely be depicted even with MRI

**Plain Xrays**
- Spurs of the superior articular process
- Spurs of the Luschka joint.
- Disc joint spaces at levels of herniation are usually preserved.

**CT scanning**
1. Most useful for detecting bony spondylitic spurs.
2. The slice just cranial to the to the disc space is the most informative slice
3. CT myelogram with low dose water soluble contrast media

**MRI** is the most useful for herniated disc.
- However NPH are seen in 20% of asymptomatic pts 45-54yrs old. 57% in those over 64.

**Conservative treatment** .............................................. up to 90% resolve
- More effective with acute cases
- Collar, NSAIDS, bed rest ± Halter
- Poor man’s traction - with head (5kg) hanging over the end of the bed (prone in flexion/ supine in extension) for several minutes several times a day.
- Cervical epidural injection
- Direct cervical nerve root block

**Indications for Surgery**
1. Failure of conservative treatment
2. Increasing neurological deficit
3. Unbearable pain

**Surgical Procedures**
- Approach should be determined by the position and type of lesion
- Soft lateral discs easily removed by posterior approach
- Spurs and more paramedian discs, via an anterior approach

**Approach:**

**Posterior approach:**
- Positioning- Prone with face in a headrest, neck flexed. Shoulders retracted inferiorly é tape
- Incision- midline 2.5 cm lower than the interspace to be explored
- Division of ligamentum nuchae
- Blunt dissection of paraspinal muscles
- Fenestration of ligamentum flavum, identify nerve root, osteophytes, disc excision
- tissue through a cruciate incision in posterior longitudinal ligament

**Anterior approach:**
- Recurrent laryngeal nerve is the most important structure at risk
- Positioning: Supine 30º elevation (.choice bleeding) é neck ext & use head ring & NG tube
- Incision: Make a oblique skin crease incision at level of the pathology using landmarks described in the table. Extend it from the midline to post border of sternocleidomastoid

<table>
<thead>
<tr>
<th>Landmarks</th>
<th>Vertebral level</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hard palate</td>
<td>Arch of atlas</td>
</tr>
<tr>
<td>Lower border of mandible</td>
<td>C2-3</td>
</tr>
<tr>
<td>Hyoid bone</td>
<td>C3</td>
</tr>
<tr>
<td>Thyroid cartilage</td>
<td>C5</td>
</tr>
<tr>
<td>Cricoid cartilage</td>
<td>C6</td>
</tr>
</tbody>
</table>
• **Superficial**
  - Incise fascia over platysma in line with skin incision
  - Split platysma with fingers in the line of fibres or cut in line of incision.
  - Identify the anterior border of sternocleidomastoid and incise the fascia immediately anterior to it

• **Deep**
  - Using fingers retract sternocleidomastoid laterally and sternohyoid and sternothyroid strap muscles, with trachea and oesophagus medially
  - Divide omohyoid (Omohyoid lies over carotid sheath + Transverse cervical artery are landmarks to brachial plexus)
  - Palpate the carotid artery, develop a plane between the medial edge of the carotid sheath and the midline structures (thyroid, trachea and oesophagus), cutting through pretracheal fascia

• The superior & inferior thyroid aa may limit the exposure but can be divided
• Recurrent laryngeal n between the two forks of the inferior thyroid a (on the right mainly)
• Vertebrae covered by longus colli → enter this ‘valley’ between longus colli muscles.
• ALL is in the midline, cut here, as Sympathetic chain is on longus colli just lateral to body

• **Structures at Risk**
  - Main dangers are recurrent laryngeal nerve and sympathetic chain
  - (Thoracic duct on the left)

• Excise the anterior annulus fibrosis & as much disc material as can be seen
• Insert an interspace spreader
• Remove the posterior annulus and the extruded disc and or any associated bone spurs
• PLL can be removed for a better view of the dura and nerve roots
• Interbody grafting then performed using:
  - Smith Robinson fusion
  - Cloward fusion
  - Bailey Badgely fusion
  - Bloom Raney modification of the Smith Robinson fusion
  - Moss cage
  - Moss cage & Plate

**Surgical results**

• No difference in results reported for anterior or posterior approaches
• No significant differences between the results of surgery for soft disc or for osteophytes
<table>
<thead>
<tr>
<th>Level</th>
<th>Dimension</th>
<th>Inclination</th>
</tr>
</thead>
<tbody>
<tr>
<td>T₁</td>
<td>0.7 mm</td>
<td>35º medial</td>
</tr>
<tr>
<td>T₃</td>
<td>0.6 mm</td>
<td>20º medial</td>
</tr>
<tr>
<td>T₄,₅</td>
<td>0.5 mm (narrowest)</td>
<td>5-10º medial</td>
</tr>
<tr>
<td>T₁₂</td>
<td>0.7 mm</td>
<td>5-10º medial</td>
</tr>
<tr>
<td>L₃</td>
<td>1.3 mm</td>
<td>25º medial</td>
</tr>
<tr>
<td>L₄</td>
<td>1.5 mm</td>
<td>30º medial &amp; 10º caudal</td>
</tr>
<tr>
<td>L₅</td>
<td>1.7 mm (widest)</td>
<td>35º medial &amp; 20º caudal</td>
</tr>
<tr>
<td>S₁</td>
<td></td>
<td>35º medial &amp; 30º caudal</td>
</tr>
</tbody>
</table>

Costotransversectomy. 

A. Straight longitudinal incision about 2.5 inches (6.3 cm) lateral to spinous processes, centered over level of vertebral dissection. 

B. Resection of costotransverse articulation.

Sinuvertebral nerve

A: The sinuvertebral nerve shown on a cutaway drawing. 

B: The branches of the invertebral nerve shown on a lateral view of an intact spine. 

a: Dorsal root ganglion. 
b: Rami communicantes. 
c: Autonomic ganglion. 
d: Sinuvertebral nerve. 
e: Terminal branches of the nerve (may ascend or descend one or two vertebral levels).

Pedicle Entrance point in spine at intersection of lines drawn through middle of inferior articular facet and middle of insertion of transverse processes (1 mm below facet joint). 

A, Anteroposterior view. 

B, Lateral view.

Entry points of pedicular screws

- **Thoracic**: base of transverse process near superior facet
- **Lumbar**: meeting of tr process é superior facet lateral to pars
- **Sacral**: base of S₁ facet in line é the neural foramena
Cervical Myelopathy

Incidence
- 6: 100 000
- Age .......................................................... 50s or 60s
- Sex M:F ....................................................... 2:1

Pathology:
- Compressive Factors:
  1. Anterior cord compression by ........... Disc / Posterior Spur / OPLL (ossification of PLL)
  2. Anterolateral compression by ............ Joints Of Luschka
  3. Lateral compression by ................ Facets
  4. Posterior compression by ................... hypertrophied or calcified Ligamentum Flavum
  5. Developmental Stenosis ..................... ≤12mm
  6. Dynamic Stenosis - Penning's Jaw Diameter: distance from postero-inferior corner of the body, to the lamina below .................. ≤12mm + 2mm of retrolisthesis with extended neck

- Site:
  1. Most commonly affects C5-6 > C4-5 > C3-4 > C6-7 (radiculopathy common at C6-7). As cervical enlargement of the spinal cord is at C4-5 & C5-6 levels.
  2. Retrolisthesis on extension (dynamic instability) is rare at C7 & its anterior tilt
  3. Grey matter is more vulnerable to compression than white matter

Symptoms In order of appearance:
  1. Electric shock in neck extension indicating an early stage to the disease 30%
  2. PAIN - characterized by central burning and stinging
  3. Parasthesia of the fingers
  4. Paresis: clumsiness of the hands and heavy writing
  5. Tightness, hot or cold sensations in the trunk
  6. Tingling in legs
  7. Ataxic broad based shuffling gait & spasticity
  8. Urinary disturbance

Signs:

Upper Extremity: - UMN & LMNL
  1. Hoffman's Sign: pinching distal phx of middle finger → flexion of other fingers IP joints
  2. Finger Escape Sign: small finger spontaneously abducts & weak intrinsics
  3. Inverted Radial Reflex: (C5 - C6) tapping brachiorad tendon → reflex fingers flexion
  4. Biceps Reflex primarily indicates neurologic integrity of C5 ± C6 component

Lower Extremity - UMN
  1. Decline in ability to walk, Apparent Ataxia
  2. Hyperreflexia and frank Clonus
  3. Proprioception
  4. Lhermite's Sign: Electric shock of leg in neck flexion
  5. Babinski's Sign: +ve in severe myelopathy

Jaw jerk (trigeminal function) ......................... rules out pathology above the foramen magnum
- Tapping on the slightly opened jaw → contraction of the masseter effectively

Central Classification of Symptomatology -

<table>
<thead>
<tr>
<th>Central Cord Syndrome</th>
<th>Symptoms And Signs In UL &gt; LL</th>
</tr>
</thead>
<tbody>
<tr>
<td>1- Transverse lesion syndrome</td>
<td>Symptoms and signs in upper and lower extremities</td>
</tr>
<tr>
<td>2- Brown-Sequard syndrome</td>
<td>Ipsilateral motor &amp; DC loss; contralateral pain &amp; temp loss</td>
</tr>
<tr>
<td>3- Motor syndrome &amp; otodynia</td>
<td></td>
</tr>
<tr>
<td>4- Brachialgia + cord $ = radiculomyelopathy</td>
<td>UL pain + Pyramidal tract involvement</td>
</tr>
</tbody>
</table>
Spinal cord syndrome

- Complete ............................................................................. Cord transection
- Incomplete:
  1- Central: [extension injury é OA] .......................... Disc + buckled LF
  2- Hemi: Brown Sequard $ .............................. $ + DC + contralate sp thalamic
  3- SCIWORA ............................................................. sudden traction in a child
- Root:
  1- Cauda equina [Lesion below T12-L1] ............. LMNL + radiculopathy
  2- Conus medullaris [Lesion at T12-L1] ............. UMNL + saddle + sphincters
- Vascular:
  1- Anterior cord syndrome: ........................................... $ + AHC + spinothalamic
  2- Posterior cord syndrome: .................................. Lemniscal + dorsal horn
- Neurologic
  1- Transverse myelitis .......................................................... All
  2- Syringomyelia (central) ............................................ Crossing sp. thalamic
  3- Tabes dorsalis (dorsal column) .......................... Lemniscal
  4- Friedreich's Ataxia ...................................................... $ + Lemniscal + cerebellum
  5- Subacute combined degeneration ................ $ + Lemniscal
  6- Amyotrophic lateral sclerosis ........................................ $ + AHC
  7- Familial spastic paresis ...................................................
  8- Poliomyelitis & Peroneal ms atrophy ...... $ + AHC

Investigations
- Plain Xray, for stenosis
- MR scan, shows cervical disc prolapse well. Demonstrates spinal cord well. High intensity signal can be found in spinal cord on T2 weighted images, representing necrosis/cavity formation. Not able to detect compression of spinal cord on extension.
- CT scan, shows OPLL and bone spurs best

Treatment

Conservative
- In early phases, try NSAIDS, collar, muscle relaxants

Surgical
- Absolute indication: ............................................... Progressive Neurological Deficit
  1. Anterior Decompression And Fusion:
     - If disc herniation or posterior spur causing compression at 1-2 levels
     - If there is kyphotic deformity, so that correction can be achieved
  2. Posterior Decompression
     - If compression > 3 levels
     - In developmental stenosis
     - Calcification of LF
  3. Laminectomy
  5. Kirta’s bilateral open door laminotomy [elevated to $ spinal canal diameter] ± BG

Prognosis
- Herniation shows ............................................... better improvement after surgery
- Older patients & dynamic stenosis ........... less improvement
- Most get worse ............................................. if not treated
CERVICAL DISC PROSTHESIS

Rationale
- A new technique which has been available in Europe for the past 2 years is replacing the disc with an artificial disc that has many benefits:
  1. Regain the normal motion is maintained
  2. Regain the physiologic stresses and no adjacent segment disease in the future.
  3. Regain the physiologic stiffness in all planes of motion plus axial compression
  4. Regain the physiologic height and no fear of root compression
  5. Replace the degenerative painful disc by a mechanically sound prosthesis
  6. Withstand the physiologic stress and transmit it to the next level
- The idea of spinal disc replacement is not new. It was first attempted 40 years ago when implanted stainless steel balls were implanted into the disc spaces of over 100 patients.

Indications
1. Disc Prolapse: one or two levels anterior compression
2. Degenerative cervical disease (spondylosis) persistent pain, radiculopathy or myelopathy.
3. Focal compressive lesion should be documented by Myelo-CT or MRI.
4. Above six weeks failed conservative treatment

Contra-Indications
1. Neck or arm pain of unknown etiology.
2. Advanced mechanical problems:
   - Marked instability (Translation > 3mm)
   - Severe spondylosis with absence of movement (e.g. Total loss of disc height)
   - Severe facet joint disease.
   - Cervical spinal stenosis (AP diameter < 10mm).
   - Ossification of PLL
3. Abnormal bone stalk:
   - Post-traumatic affected vertebral bodies.
   - Iatrogenic: failed previous decompressive / fusion surgery
   - Neoplastic: destroyed vertebral body
   - Infectious: destroyed vertebral body
   - Metabolic bone disease: Osteoporosis/Osteomalacia/Pagets/Ankylosing Spondylitis
   - Drugs interfering with bone or soft tissue healing
4. Systemic disease
   - Infections
   - HIV / AIDS
   - Rheumatoid Arthritis
   - Hepatitis - Morbid Obesity (BMI > 35)
5. Component allergies (Titanium / Cobalt / Polyethylene)

Types:

According To Fixation
1. Screw fixation
2. Keel fixation
3. Teeth fixation
4. Porous coated prosthesis
5. Macroteture surface prosthesis
6. Combined

According to bearing surfaces
1. Metal on Metal
2. Metal on polyethylene
3. Hydrogel prostheses: replace of the NP only & retain the AF. Consist of hydrogel core constrained in a woven polyethylene jacket
Advantages:
1. The device maintains the proper intervertebral spacing
2. Provides stability
3. Restores the normal shock absorbing mechanism of the spine
4. Less morbidity than the standard fusion techniques
5. Better functional outcome
6. Percutaneous placement could be done with nuclear hydrolgel replacements

Precautions
1. Should be placed centrally not to shift axial load to the facets
2. Avoid the destruction of facets and ligaments.
3. An artificial disc must exhibit tremendous endurance.
4. The intervertebral disc prosthesis ideally would replicate normal range of motion

Operative details
- The table is placed in a slight reverse Trendelenburg position ± adhesive pull of shoulders
- The skin incision on the right or left side may be transverse or obliquely vertical along the anterior border of the sterno-mastoid. Transverse incisions are better cosmetically & vertical incision allows greater exposure
- Platysma is retracted & hemostasis is obtained
- The superficial & deep cervical fascia around sterno-mastoid muscle is sharply divided along its medial edge
- Divide the omohyoid muscle (runs obliquely across the field at the level of the C6)
- Palpate the carotid artery & retracted laterally with the sternocleidomastoid muscle.
- The short strap muscles, trachea, and esophagus are retracted medially
- Divide the pretracheal fascia & scissors. The anterior longitudinal ligament, however, should not be disturbed until the operative disc level is positively identified.
- Spinal needle is carefully inserted into the disc space & checked fluoroscopically
- Once the operative disc space is identified reflect Longus Colli musculature from the anterior lateral edges of the vertebral body, so the entire anterior surface of the disc space.
- Remove ALL and Anterior Annular fibers are removed with scalp dissection.
- Remove as much as easily accessible from Disc space & Osteophytes with a small curette or rongeur
- Disc space distraction is applied gently using a lamina spreader rather than by halter traction
- Apply the prosthesis & test for its stability under fluoroscopy
- Put a drain, close in layers & apply a cervical collar

Complications:
1- Biomechanical problems:
   1. Bone resorption
   2. End plate failure
   3. Prosthesis failure
   4. Facet over load and degeneration
2- Surgical complications:
   5. Neurological complications:
      ▪ Rt. recurrent laryngeal n.
      ▪ Superior laryngeal n.
      ▪ Sympathetic chain (3 ganglia; superior at C3, middle at C6, cervicothoracic at C7-T1) → Horner’s syndrome
6- Vascular complications
   ▪ Carotid a
   ▪ Internal juglar vein
   ▪ Vertebral artery if anomalously enters into a higher level than C6
   ▪ Thyroid arteries; inferior & superior
7- Visceral complications:
   ▪ Trachea
   ▪ Oesophagus
   ▪ Thoracic duct in left exposures
3- Biological complications:
   8. Abnormal bone deposition
   9. Infections
Facet Joint Dysfunction
Spondylosis & Segmental Instability

Definition:
- Degenerative disease characterized by flattening of the IV disc → posterior displacement of the facet joint → disturbed movement of the vertebrae (segmental instability)

Pathology: Hill Degenerative Cascade:
1. Stage of Dysfunction: Minor disc tears, Facetal synovitis, & ms sprain
2. Stage of Degeneration:
   - Inelastic nucleus bulbosus → ↓ stress sharing → ↑ stresses over:
     1. Annulus fibrosis → fissuring // to end plate → herniation
     2. End plate → failure
     3. Facetal joints
3. Stage of Spondylosis:
   - ↑ reactive bone formation → End plate Schmorl’s nodes/marginal osteophytes/facetal hypertrophy
   - Flattening of the disc → posterior displacement of the facet → ↑ OA
   - During flexion & ext → disturbed movement → segmental instability
4. Stage of Stabilization: → ↑ osteophytes → stabilization of the adjacent vertebrae

Clinically:
- Stage of dysfunction: Axial dull aching ms pains é trigger points
- Stage of degeneration: Sharp back pain → sciatica → radiculopathy (disc prolapse)
- Stage of Spondylosis: Intermittent back pain referred to buttocks & GT (≠ sciatica)
  - ↑ é work, walk, or prolonged sitting & ↓ é lying down
- Stage of Stabilization: Constant but less intense back pain
  - ↓ by physiotherapy and local warmth

Examination  Male 70y
- Inspection: NAD
- Palpation: poorly localized tenderness over the buttocks & lower lumbar spine
- Movements: painful limited ± catching during straightening
- Residual signs: ↓ ankle jerk / Spinal stenosis

PXR

<table>
<thead>
<tr>
<th>Early</th>
<th>Late</th>
</tr>
</thead>
<tbody>
<tr>
<td>Narrowing of the disc space</td>
<td>Flattening + vacuum sign (black deg disc)</td>
</tr>
<tr>
<td>Retrolithesis (L4-5 &amp; L5-S1)</td>
<td>Degenerative spondylolisthesis</td>
</tr>
<tr>
<td>Anterior end plate sclerosis (abnormal loading)</td>
<td>Marginal osteophytes</td>
</tr>
</tbody>
</table>

White & panjabi ccc of degenerative lumbar instability:
1. >4.5 mm of translation
2. >22º relative sagittal angulation
3. >15º-25º of angular motion bet adjacent segments on flexion–extension PXR
Discography
- Not routinely done as it has controversial value

Tc
- hot spot at the disc space

MRI:
- Asymmetrical facetal joint
- OA of facetal joint
- Advanced disc degeneration
- Modic Classification: as the annulus tear into end plate
  - GI: oedema ↑ T1 (low T2)
  - GII: fatty inf ↑ T2 (T1 turns low)
  - GIII: fibrosis both T1 and T2 ↓

DD:
- Forestier’s disease: usually multiple spurs at multiple levels more on rt side
- Ochronosis: intervertebral calcification

Treatment
- Conservative as usually the joint will stabilize itself
  1. Physiotherapy strengthening of the back muscles & abdominal muscles
  2. Corset: relief pain especially in obese patients
  3. Facetal injection: If the lesion is localized to one or two levels
     - Position: prone on fluoroscopy under local anaesthesia
     - 20 gauge needle introduced ⊥ to skin 2-3 cm from the middle line at spinous process
     - Pt rolled into oblique position and the needle is adjusted under image
     - 2 ml LA + steroid
  4. Modification of activity
  5. NSAIDs mild
  6. Psycho-therapy
- Surgical spinal fusion
- Exhaust every mean to avoid surgery as the results are usually unsatisfactory
- Indications:
  - Failure of conservative measures
  - Intractable pain
- Precautions:
  - Careful repeated examination to detect any treatable pathology
  - There should be some response to medical otherwise pt will not benefit from fusion
  - There should be unequivocal proof for OA or spondylosis at a specific level
  - Pt should be emotionally stable & entailed that there is failure rate 10-20%
  - And that this pathology may occur at a different level after fusion upto 40%
Spondylosis & Spondylolisthesis

SPONDYLOLYSIS

- caused by a defect in the pars interarticularis
- usually a fatigue fracture from repetitive hyperextension stresses (gymnasts)
- most common cause of LBP in adolescents
- Radiology
  - plain x-rays demonstrate 80% of lesions
  - oblique views - additional 15% picked up - 'Scottie dog' sign (Lachapelle)
- CT - may miss fracture
- Te - uptake indicates an acute lesion & will probably heal
- Non-union is common

SPONDYLOLolisthesis

Definition

- "spondy" refers to the vertebrae and "listhesis" means "to slip"
- It is the forward "anterolithesis" or backward "Retrolithesis"
- Normally laminae & facets have locking mechanism preventing forward slippage
- usually L4/5 and L5/S1 sagittal facet orientation

Newman Classification

1. Dysplastic (20%)
   S1 Superior articular facets are dysplastic ± sp.bifida occulta:
   1. Axial malalignment
   2. Coronal malalignment

2. Isthmic (50%)
   more in children:
   - lordosis
   - thin pars
   Spondylosis ......................... (L5/S1) neural arch still in place
   a. Lytic–fatigue fracture of the pars interarticularis
   b. Pars Elongation
   c. Pars fracture

3. Degenerative (25%)
   Degenerate facet joints .......... (L4/L5) more in W>30° gpqr cwug tumble OA, gout. Neural arch slip back; but usually <30% displacement

4. Post-traumatic
   Acute fracture of pedicle, facet that allows a slip to occur
   pars is intact here

5. Pathological
   Tumours, paget's

6. Post-operative
   Postlamenectomy instability

- Effect of spondylolisthesis = CAUSES OF NEUROLOGICAL symptoms:
  1- Pressure on the roots ..... Radiculopathy & pars pseudoarthrosis, LF, facet, spurs
  2- Pressure on the dura ...... by lamina above or body below
  3- Pressure on the cord ...... Cauda equina by NPH
  4- Lumbar disc prolapse

Meyerding Severity of Slip:

1. Percentage of slip
   distance to AP diameter of the vertebra below:
   - Grade I ............................................... < 25%
   - Grade II ........................................... 25-50%
   - Grade III ......................................... 50-75%
   - Grade IV .......................................... 75-100%
   - Grade V ......................................... >100% (spondyloptosis) = L5 below the promontory line

2. Slip Angle
   - normal = > 0° .................................. >55° is progressive
### Clinical
- usually asymptomatic esp in children → discovered only incidentally on x-rays
- Begins during the second or third decade & injury may aggravate any symptoms
- **1-** **Insidious Backache** (↓ with walking and standing ↑ by rest)
- **2-** **Leg Pain** (Radiculopathy L5)
- **3-** **Flattening** of the back & **Lost Waist**
- **4-** **Spinous process Step-Off** can be felt at L5S1 if significant displacement
- **5-** **+Ve Schober Test** (limited Lumbosacral mobility)
- **6-** **Compensatory Lordosis** at the segment above
- **7-** **Heart Shaped Buttocks** δ sacral prominence & vertical orientation
- **8-** **Claudication** may signal the development of lateral stenosis
- **9-** **Scoliosis** μβ associated: idiopathic, lithetic, sciatic
- **10-** **+Ve Tripod Sign**

### PXR

1- **Napoleon Hat** ....................... appearance of lamina of severely slipped vertebra in AP view
2- **Scotty Dog** ......................... in spondylolysis before slippage in oblique view only
3- **Meyerding Slip %** .................... lateral view (slip displacement / AP diameter of vert below)
4- **Slip Angle:** ......................... between the upper border of L5 and sacral line
5- **Sacro-inclination:** ................. to the vertical on standing lateral view
6- **Sacro-horizontal Angle:** ........... same but to horizontal line (↑ in sacral kyphosis = dysplastic)
7- **Dome Sacrum & Trapezoid LS** .. (δ resorbed ant & post edges of the sacrum)
8- **Lumbar Index:** ...................... measure the trapezoidal wedging of L5

### CT:
- Detect the pars defect in lytic type
- Detect facet orientation in dysplastic type

### TG:
- ↑ uptake = acute pars lesion ↓ will probably heal

### MRI:
- In degenerative SL é neurologic complications
- May show Modic changes

### SPECT (Single Photon Emission Tomography)
- Done for lytic defects:
  - If hot = recent defect → put him in a brace for 6mo
  - If cold = old defect → physiotherapy till operation

### Treatment
**Non-operative: (Gl – GII)**
- reduce sports
- adolescents - x-rays every 6 mo until maturity

### Signs of Progression:
1. **Clinical:**
   - 1- Young
   - 2- ♀
   - 3- Postural & gait abnormality
   - 4- Episodes of backache
2. **Radiological**
   - 5- Meyerding G III, IV, V
   - 6- >40º Slip angle or progress
   - 7- >30º inclined sacrum
   - 8- >25º Angular rotation
   - 9- Dome shape sacrum
Operative:

**Indications:**
1. slip >50%
2. **Progressing** in adolescents …. explain
3. **Pain Unresponsive** to non-operative treatment
4. functionally significant neurological **Deficit**

**Type of surgery**
1. Grade II ................................................. in situ **Pedicular fixation ± Decompression**
2. Grade III, IV & V ................................. in situ **Pedicular fixation + Decompression**
3. Elderly degenerative cases .............. **Decompression** without fusion
4. Lytic lesions & children even if low grade:
   [1]. **Posterolateral Fusion** without Instrumentation for L1-L4 SL
   [2]. **Pars Repair** for L5-S1 defects:
       o Buck: screw fixation
       o Scott: wire fixation
       o Combined: tension wire bet t.process or spinous process & a screw

5. **Anterior Interbody Fusion** - for failed posterior fusions or some times in combination
6. **Mini ALIF, PLIF, XLIF** can be used as a limited approach in combination é posterior fusion

7. **Reduction** is debatable:
   • ↑ stability
   • ↓ progression
   • Better cosmetically
   • But time consuming
   • Does not give better clinical result than in situ fusion

---

**DD between degenerative & lytic:**

<table>
<thead>
<tr>
<th></th>
<th>Degenerative</th>
<th>Lytic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>Old</td>
<td>Young</td>
</tr>
<tr>
<td>Level</td>
<td>L4-5 or L3-4</td>
<td>L5-S1</td>
</tr>
<tr>
<td>Canal</td>
<td>Compromised</td>
<td>Not compromised</td>
</tr>
<tr>
<td>Progression</td>
<td>Stable</td>
<td>Progressive</td>
</tr>
<tr>
<td>T</td>
<td>Facetal OA</td>
<td>Facetal malalignment</td>
</tr>
</tbody>
</table>

**Pearl:**

- To determine the appropriate surgical procedure to be done first know your pathology:
  I. Coronal balance: ......................... (scoliosis) 10°/25°/35°/45°
  II. Sagittal balance: ...................... (kyphosis) -60/-40/ -20 / 0
  III. Width: ................................. (Stenosis) 11.5/10/8/6
  IV. Translation ......................... (lithesis) 1 mm → 10 mm
- 4 groups:
  1. Conserve
  2. Decompression
  3. + fusion ± instrumentation
  4. + anterior surgery
Lee Zones of lateral canal

**ENTRANCE zone (1)** antero-medial to the inferior facet

**Mid-ZONE (2)** between the body anterior and the pars posterior; medial boundary is open to spinal canal.

**EXIT ZONE (3)** is formed by intervertebral foramen.
Spinal Stenosis

- Is the narrowing of the spinal canal at its central, lateral recess, or lateral foramen that production of symptoms.
- It is one manifestation of the general process of spinal degeneration that occurs with aging, and often becomes symptomatic in the 7th & 8th decades of life.

**Epidemiology**

- The L4-L5 segment is the most commonly affected, followed by the L3-L4.
- **Men** are more commonly affected, because their canals are narrower at the L3-L5 levels.

**Anatomy**

- In most individuals, the spinal cord ends by the L1 level; below this level the remaining nerve roots travel as the **Cauda Equina**.
- The nerve roots are more tolerant of chronic pressure than the spinal cord, which is why an individual may tolerate a higher stenosis degree in the lumbar spine than in the cervical or thoracic spine.
- The boundaries of the central canal vary with the level:
  - **At body**: (most affected by congenital short pedicle)
    - Anterior border is formed by the body
    - Lateral border by the pedicles
    - Posterior border by the laminae posteriorly.
  - **At the level disc** (most affected by degenerative)
    - Anterior border is formed by annulus
    - Posterolateral borders by the facet joints
    - Posterior border by ligamentum flavum.

**Aetiology**

**Arnoldi Classification**

1. Congenital
   - [1]. Idiopathic
   - [2]. Achondroplasia

2. Acquired
   - [1]. Degenerative
     - Central
     - Peripheral (lat recess, foramen)
   - [2]. Combined degenerative + congenital
   - [3]. Trauma
   - [4]. Iatrogenic (laminectomy, fusion, chemonucleolysis)
   - [5]. Infection (TB)
   - [6]. Spinal tumour
   - [7]. Spondylolytic & spondylolithetic
   - [8]. Metabolic: Paget's & Fluorosis

**Diameter of spinal canal:**

<table>
<thead>
<tr>
<th></th>
<th>Normal</th>
<th>Relative</th>
<th>Stenosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Midsagittal (Lat)</td>
<td>&gt;11.5</td>
<td>10 - 11.5</td>
<td>&lt;10mm</td>
</tr>
<tr>
<td>Interpedicular (AP)</td>
<td>&gt; 20</td>
<td>16 - 20</td>
<td>&lt;16mm</td>
</tr>
<tr>
<td>Cross section area</td>
<td>1.45 cm²</td>
<td>1 - 1.45</td>
<td>&lt;1 cm²</td>
</tr>
</tbody>
</table>
Pathological Classification by site:
A. **Central Stenosis** ................................................................. affect the Thecal Sac & Cauda Equina
   - caused by:
     1- Arthritic facets with medial encroachment
     2- Congenitally narrow canal ('Trefoil' shape)
     3- Spondylolisthesis
     4- Central disc herniation
     5- Posttraumatic; Post-surgical → fibrosis

B. **Lateral Stenosis** ................................................................. affect the Root at **Lee Zones** Of Lat Canal
   1- Entrance Zone............................................................... affects the traversing (lower) nerve root
   2- Mid & foraminal Zones .................................................... affects the exiting (upper) nerve root
   3- Extra-foraminal (far-out): ............................................. sacral ala, tr. process affect the dorsal root
   - Caused by:
     o Superior articular facet enlargement
     o Lateral disc herniation
     o Ligamentum flavum hypertrophy
     o Uncinate spurring

Factors affecting the Dimensions of the canal
[1]. Dynamic factors
[2]. Level of the canal: The foramina ‡ size while the nerve roots ‡ in diameter as you move down the spine. Thus the lumbar spine is most commonly affected.
[3]. Postural factors:
   [1]. Axial loads ‡ bulge of the disc → ‡ root compression
   [2]. when standing ‡ lumbar lordosis → ‡ the stenosis → subarachnoid obstruction → ‡ diffusion of fluid from endoneurium to subarachnoid → ‡ endoneurial pressure → 'COMPARTMENT SYNDROME' in the nerve roots → ‡ conduction

Clinical
- Virtually all individuals at 8th decade have some degree of spinal stenosis
  1- **Central stenosis:**
     a. **NEUROGENIC CLAUDICATION.** (ð cauda equina compression = Pseudo-claudication)
     b. ↑ **IMBALANCE & PARASTHESIA**
     c. feeling better if they walk **STOOPED** forward (sitting, bending, squatting)
     d. Rarely - urinary incontinence & cauda equina syndrome
  
  2- **Foramenal stenosis:**
     e. **RADICULOPATHY** from narrowing of the lateral recess or the neural foramen.
     f. **BROAD-BASED GAIT + FORWARD STOOP** posture
     g. **CHECK HIP**
     h. **CHECK DISTAL PULSES** to **SCREEN** for vascular causes of claudication.
     i. Stress Test = walk until symptoms occur then
     j. Stoop test = continue walk ë stoop → pain ‡ (if persist = vascular or hip)

<table>
<thead>
<tr>
<th>effect of ... on</th>
<th>Neurogenic Claudication</th>
<th>Vascular claudication</th>
</tr>
</thead>
<tbody>
<tr>
<td>Walking</td>
<td>Proximal bilateral thigh pain</td>
<td>Distal-proximal pain; calf pain</td>
</tr>
<tr>
<td>Uphill Walking</td>
<td>late pain</td>
<td>++</td>
</tr>
<tr>
<td>Rest</td>
<td>relief with sitting or bending</td>
<td>relief with standing</td>
</tr>
<tr>
<td>Bicycling</td>
<td>-</td>
<td>++</td>
</tr>
<tr>
<td>Lying Flat</td>
<td>++</td>
<td>-</td>
</tr>
</tbody>
</table>
In **Investigations**

1. **X-rays:**
   1. Uncinate spurs
   2. Disc height
   3. Facet hypertrophy in older patients
   4. Calcified ligament
   5. Mid sagittal diameter .......... <10mm
   6. Interpedicular distance .......... <16mm
   7. **Hardley’s line** is broken: it is a smooth S line that extends from the inferior border of the transverse process to the lateral contour of superior facet of the vertebra below

2. **CT:**
   1. More accurate than PXR for bony anatomy
   2. Less accurate than MRI for soft tissue compromise
   3. AP Diameter < 10mm .......... Absolute Stenosis

3. **MRI:** (without gadolinium) is the “gold standard” in the evaluation of central stenosis.
   1. T1 ................... absence of the foraminal fat around the root
   2. T2 ................... condition of the cord

4. **Epiradicular Nerve root block:**
   - Improvement of radiculopathy after LA injection is suggestive of lateral stenosis.

5. **Myelography** è water soluble metrizamide: it can be useful in selected cases

**Natural History**

- Symptoms unchanged .......... 60-70%
- Worse .......................... 15-20%
- Improved ....................... 15-20%

<table>
<thead>
<tr>
<th>DD</th>
<th>1- Spinal canal Stenosis</th>
<th>LDP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age &amp; sex</td>
<td>&gt;50 ♂</td>
<td>&lt;50 ♂</td>
</tr>
<tr>
<td>Onset</td>
<td>Chronic</td>
<td>Acute</td>
</tr>
<tr>
<td>Pain</td>
<td>Referred</td>
<td>Radicular</td>
</tr>
<tr>
<td>Weakness</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>Sensory</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>Lasegue &amp; neuro</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>Provocation Position</td>
<td>Walking</td>
<td>Sitting</td>
</tr>
<tr>
<td>Palliation Position</td>
<td>sitting</td>
<td>Standing or Lying</td>
</tr>
</tbody>
</table>

2. **Diabetic Neuropathy:**
   - Glove and stocking
   - First to vibration

3. **Facetal Synovial Cyst:**
   - May rupture into the dura and compress the root

4. **Forestier’s Hyperostosis**
Treatment

Non-Operative:
1- NSAIDs
2- Muscle relaxants
3- Antidepressants for chronic radicular pain
4- Calcitonin in paget’s
5- **Epidural Root Injection**: long-term relief in foramenal or lateral recess stenosis
6- Flexion **Brace** (Williams brace) - prevents lordosis. Not well tolerated.
7- **Physiotherapy** (with massage, ultrasound, TENS, braces or supports, acupuncture, biofeedback, hot or cold packs, traction, or manipulation) can offer symptomatic relief of radicular or low back pain, but not for claudicant symptoms.

Operative
- **Indications**: 
  1. Severe neurological symptoms
  2. Failed conservative treatment
  3. Impaired ADL (activities of daily living)

- **Modalities**: 
  1- Cervical anterior decompression
  2- Laminaplasty: in which the lamina is cut completely from one side, and partially from the other side to the level it could be opened like a book while it is in place, then BG is placed to elevate the lamina, then the lamina is sutured in the new position. This technique is useful for cervical stenosis where the stenosis is rather central more than lateral recess or foramenal stenosis
  3- Lumbar foramenotomy éout fusion
  4- Lumbar lamenectomy + Facetectomy (<1/3 of medial facet) + Fusion if:
     i. Pt < 60 y with 2 facetectomies
     ii. Pt < 55y with spondylosis
     iii. Pt < 50y with isthmic spondylolitheis
Rheumatoid Arthritis

- Affects 3% of women & 1% of men
- Hand > Knee > hip > cervical spine

**Diagnosis:**

**Ara Criteria** (American Rheumatism Association):

1. **Morning Stiffness:** Lasting at least 1 hour before maximal improvement.
2. **Arthritis Of 3 Or More Joint Areas**
   - At least 3 joint areas simultaneously have had soft tissue swelling or fluid (not bony overgrowth alone) observed by a physician; the 14 possible joint areas are right or left proximal interphalangeal (PIP) joints, metacarpophalangeal (MCP) joints, wrist, elbow, knee, ankle, and metatarsophalangeal (MTP) joints.
3. **Arthritis Of Hand Joints**
   - At least 1 area swollen (as defined above) in a wrist, MCP or PIP joint.
4. **Symmetric Arthritis**
5. **Rheumatoid Nodules**
   - Over bony prominences, or extensor surfaces, or in juxta-articular regions
6. **Rheumatoid Factor +Ve**
7. **Radiographic Changes**

- At Least 4 Of 7 Criteria.
- Criteria 1 Through 4 (At Least 6 Weeks).
- **Clinical Staging:**
  - 7 ccc .........................Classic
  - 5 ccc .........................Definite
  - 3 ccc .........................Probable
  - 2 ccc ..........................Possible

**Aetiology:**

- Genetic susceptibility: RA is common in first degree relatives of RA patients and twins
- Immunological process: HLA-DR4 & DW4 encoded on chromosome 6; and is found on the surface of APC (antigen presenting cells); & when interact é the antigen (some times the antigen with the HLA form the activating complex) → autoimmune response
- When APC and T-cells interact → cell proliferation + cytokines secretion → © phagocytes & B-cells
- Rheumatoid factor: Anti-IgG auto antibodies which is detected in the serum of the patient
Pathology:
Affected joints:
1. Atlantoaxial joint + transverse ligament ........................................... Atlanto-Axial Instability
2. Atlanto-occipital → dens project into f.magnum .................... Cranial Settling = Basilar Invagination
3. Facetal joints of the mid cervical region ........................................... Subaxial Subluxation

Stage 1: Synovitis
• vascular congestion & effusion → Osteopenia (also from the steroid ttt)
• synoviocyte proliferation → Villous formation
• infiltration of subsynovial layers by PMNs, lymphocytes & plasma cells

Stage 2: Destruction
• a Pannus of granulation tissue creeps over the articular surface eroding cartilage & bone
• cartilage destruction occur partly by proteolytic enzymes & vascular tissue
• bone destruction occur partly by proteolytic enz, & osteoclastic activity
• direct invasion occurs at the margins of the joint
• similar changes occur in tendon sheaths → rupture

Stage 3: Deformity & Complications:
• acute inflammation subsided
• deformities occur δ: articular destruction / capsular stretching / tendon ruptures
• Neurological manifestation occur δ:
  o Different Instabilities
  o Mechanical compression of the cord by a pannus
  o Compression of the vertebral artery
  o Peripheral neuritis from the disease itself
  o DMAR drugs

Extra-articular Manifestations: Felty’s, SJÖRGEN, CAPLAN $, PERICARDITIS, VASCULITIS, NODES
Clinically: Palendromic, Systemic, Monoarticular, Myalgic
• 40 ☟ may be é positive family history
• Early: usually no cervical manifestations:
  o Painful swollen Hands + morning stiffness
  o Painful, Swollen, Stiff, Cracking Joints
  o loss of Weight, weakness
• Late:
  o Deformity: F Add ER of the hip
  o Path #: from the disease and drugs (usually neck femur)
  o Bouchard’s nodules, Swan neck, Boutonniere, Z-thumb, fingers ulnar deviation, wrist radio-volar deviation, valgus knee, valgus feet, clawed toes
  o Atlanto-Axial Subluxation: 20% of patients
    1- Headache (C2 nerve compression) & Black out spells
    2- Pain, stiffness, rarely parasthesia
    3- Severe neck tenderness & ♦ ROM
    4- LMNL in UL + UMNL in LL
    5- Lhermitte’s sign: sudden shocking leg parasthesia é neck flexion

Ranawat Neurologic Classification

<table>
<thead>
<tr>
<th>Class</th>
<th>Motor habilitation</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Motor ambulatory</td>
</tr>
<tr>
<td>II</td>
<td>Parasthesia &amp; hyper-reflexia</td>
</tr>
<tr>
<td>IIIa</td>
<td>Motor ambulatory</td>
</tr>
<tr>
<td>IIIb</td>
<td>Motor non-ambulatory</td>
</tr>
</tbody>
</table>
**Laboratory Findings:**
- ↑ ESR, CRP
- RF +ve in 80%, ANA 30%
- ACCP (anti Cyclic Cetrolinated Peptide): 97% early +ve in RA even in seronegative RA
- anemia: abnormal erythropoiesis, and chronic blood loss from analgesic gastritis
- WBC: Normal or ↓ if ↓ suspect Felty
- Complement

**Synovial Biopsy & Fluid:**
- Biopsy: is non specific to RA
- Fluid: ↓ ptn, C, glucose / poor clot / ↑ RA cells & PNL

**Larsen = Dale Radiologic Index**
1. Stage I: juxta-articular Osteopenia
2. Stage II: Narrow joint space (usually bilat, symmetrical, concentric ± protrosisio)
3. Stage III: Bone Erosion + bone Cysts
4. Stage IV: Deformity
- Usually no sclerosis nor osteophytes (except if 2ry OA)

**White Criteria** of basilar invagination on PXR cervical lateral view:
1. PADI (Posterior Atlanto-Dental Interval) <13mm = BI
2. AADI (Anterior Atlanto-Dental Interval) >4mm = BI
3. DBI (Dens-Basion Interval) <4mm = BI
4. Wackenheim’s clivus tangent line should tangent the dens not cut it
5. Chamberlain’s line from f.magnum to hard palate <3mm cut to dens
6. McGregor’s line from occiput to hard palate <5mm cutting to dens
7. McRae’s line bet basion & foramen magnum dens should never pass it
8. Ranawat’s from C2 pedicle to transverse plane of C1 15-17mm
9. Redlund-Johnell from axis base to McGregor’s line 29-34mm

**NB** The Atlas sublux anteriorly (commonest) on the axis with flexion & reduce è extension (AADI change > 3mm) this is the old method

**DDx:**
- Seronegative: SLE, Still’s
- AS: spondarthropathy
- Reiter’s: Conjunctivits, urethritis, Arthritis
- Gout & CPPD: crystals
- OA: DIP affection, osteophytes
- Polymyalgia rheumatica: pelvic, and pectoral weakness, and aching, +ve steroid test
- Sarcoidosis: Erythema nodosum, Hilar LN, +ve Kveim test
**Management Principles:**

- **Stop the Synovitis**
  - Rest
  - **DMAR** Drugs (Disease Modifying Anti-Rheumatic) - Pyramid Approach = NSAID- Antimalarials - Sulphasalazine – Gold – MTX – D-penicillamine - Azathioprine – Leflunomide (Avara) + low dose steroids
  - Synovectomy - chemical, irradiation, surgical

- **Prevent Deformity**
  - Splintage $\rightarrow$ hard collar $\rightarrow$ Traction $\rightarrow$ surgery
  - Physiotherapy
  - Tendon repairs & joint stabilisation

- **Surgical (start $\rightarrow$ knee if $> 45^\circ$ flexion deformity)**
  - Arthroplasty is the gold standard
  - Osteotomy not done:
    - Doesn’t remove the cartilage $\omega$ is a source of inflammation
    - RA is concentric & no healthy cartilage
  - Arthrodesis not to be done (bilateral)
  - Cervical fusion:
    - **Indications:**
      - A. Severe pain
      - B. Neurologic signs
      - C. PADI $< 13$mm
    - **Procedure:**
      1. $C_0$$-$$C_2$ fusion by BG & wiring ± C1 laminectomy or odontoidectomy
      2. Hartshill-Ransford loop + sublaminar wire (if atlanto-axial + subaxial)
      3. $C_0$$-$$C_2$ fusion by plating ± BG
      4. Inter laminar clamps + BG
      5. Posterior atlantoaxial transarticular screws
      6. $C_0$$-$$C_2$ fusion by BG followed by Halo traction

- **Rehabilitate & keep moving**
  - Cervical collar for 3-6 mo
  - Physiotherapy

**At Onset:** NSAID, exercise

**Early:** NSAID, Steroids, DMD, local injections, physiotherapy, Rest and splintage

**Erosive:** DMD, splintage, operative (synovectomy, tendon repair, joint stabilization)

**Late:** Reconstructive arthroplasty
### Drugs Details:

<table>
<thead>
<tr>
<th>Drug</th>
<th>Mechanism</th>
<th>A/E</th>
</tr>
</thead>
<tbody>
<tr>
<td>NSAIDs</td>
<td>⊖ PG synthesis → ◄ pain and inflammation</td>
<td>Gastric Upset</td>
</tr>
<tr>
<td>Antimalaria</td>
<td>⊖ PG &amp; phagocytic activity of PNL</td>
<td>Lucoma</td>
</tr>
<tr>
<td>Sulphaslsazine</td>
<td>Anti-inflammatory</td>
<td>Megaloblastic anemia</td>
</tr>
<tr>
<td>Gold</td>
<td>Alters the function of macrophages and complement</td>
<td>Thrombocytopenia</td>
</tr>
<tr>
<td>Methotrexate</td>
<td>Immune suppression</td>
<td>Liver toxicity</td>
</tr>
<tr>
<td>D-Penicillamine</td>
<td>Dissolve RF complexes in joints to be excreted</td>
<td>Late resp.&amp; nephrotic</td>
</tr>
<tr>
<td>Azathioprine</td>
<td>Immuno suppression</td>
<td>Liver toxicity</td>
</tr>
<tr>
<td>Leflunomide</td>
<td>⊖ DiHydro-Orotate Dehydrogenase → ◄ T-cell prolif</td>
<td></td>
</tr>
</tbody>
</table>

### Complications:
- Fixed Deformities
- Joint Rupture
- Infection
- Spinal cord compression
- PN compression
- Vasculitis
- Amyloidosis, proteinuria
- progressive RF

### Poor Prognostic Signs:
- Very high RF
- Peri-articular erosions
- Nodules
- Muscle wasting
- Joint contractures
- Vasculitis
- PADI < 13mm

### Prognosis:
- 10% improve after first attack of synovitis
- 60% have remissions & exacerbations
- 20% have severe joint erosions requiring multiple operations
- 10% become completely disabled
Some Abnormal Gait Patterns

- Weak Quad
- Stiff knee + Equinus
  Back knee
- Flexed Knee + calcaneus
  Crouch
- Ataxic Gait
- Weak Glutei
- High steppage gait
**Ankylosing Spondylitis**

**Aetiology:**
- Affects spine & sacroiliac joints primarily

**Prevalence**
- 0.2%
- Males > females & Familial involvement
- HLA-B27 marker

**Pathology:**

I. **Affected Joints**

1. **Plane synovial joint:** 1st **Sacroiliac** → 2nd **Facet** (L, Thx, & Cx) → 4th **COSTOVERTEBRAL**
2. **Syndesmosis & symphyses:** IV discs, sacroiliac ligaments, symphysis pubis
3. Cervical spine:
   a. Atlanto-occipital erosion
   b. Subaxial subluxation and kyphosis
   c. Atlanto-axial subluxation δ:
      1. rigid spine → C1-2 stresses
      2. Transverse ligament dysfuction

II. **Stages:**

1. **Inflammation & erosion:**
   - Round cell infiltration, *granulation* tissue
   - *Erosion* of anterior edge of the vertebra → *Squaring* of the vertebra
   - *Repair* process starts → prominence of edges + *Osteopenic Midsection*
2. **Fibrosis:**
   - *Replacement* of granulation tissue with fibrous tissue
3. **Ossification of:**
   - A. Fibrous tissue
   - B. ALL
   - C. Annulus → vertical marginal osteophytes “**Syndesmophytes**”
4. **Evolution:**
   - A. reformation of the anterior concavity of the body
   - B. Symmetrical syndesmophytes → pathognomonic **Bamboo** appearance
   - C. Kyphosis deformity measured by the chin-brow to vertical angle:
     - Acute Type
     - Chronic type kyphosis deformity

III. **Extraskeletal:**

1. Prostatitis
2. Conjunctivitis & uveitis in 20%
3. Carditis, AS, Pulmonary fibrosis
Clinical:
1. Insidious Pain & stiffness & ADL limitation
2. CHIN TO CHEST deformity
3. WALL TEST: pt can not stand with heal, occiput, buttock touching the wall
4. ENTHESOPATHY of tendo-achillies
5. † spinal motion (+VE Schober’s)
6. † Chest expansion < 7cm
7. † Hip motion (+VE Thomas TEST)
8. +VE SACROILIAC TESTS
9. Difficult cervical spine fractures

Radiology:
1. EROSIVE arthritis
2. SQUARING of vertebral bodies
3. Patchy periarticular OSTEOPENIA
4. SYNDENSMOPHYES
5. BAMBOO spine
6. CHIN-BROW to vertical angle

Laboratory:
1. ESR ................................ +
2. HLA-B27 ........................ +ve 90%
3. RF ................................. -ve

Management:
[I]. Early: NSAIDs (phenylbutazone) + Postural management + activity modification
II. Late: corrective ttt:
1. Acute kyphosis:
   • Halo traction in the line of deformity é gradual correction
   • Followed by Halo cast brace for 12 mo
2. Chronic kyphosis:
   • Extension closing wedge under LA setting position (only GA during the osteodasis only) at C7-T1; why?
      ▪ Wide Canal
      ▪ C8 is flexible and less important
      ▪ Vertebral artery passes anterior to the C7 → less liable to be injured
   • Lumbar closing wedge osteotomy at L2: but
      ▪ Aortic disruption
      ▪ Superior mesenteric $ 
III. Latest: reconstructive operations
   3. THR
   4. Sacroiliac fusion: using screw fixation, transsacral rods
   5. Atlanto-Axial fusion

DDx:
1. OSTEITIS CONDENSANS Ilii: pregnant ♀ é sclerotic △ medial ilial aspect of the SI joint
2. FIBRODYSPLASIA OSSIFICANS PROGRESSIVA: generalized heterotopic ossification & fused SI
3. DIFFUSE IDEOPATHIC SKELETAL HYPEROSTOSIS (FORESTIER’S $)
   i) Old ♂
   ii) Right side
   iii) Ossification ALL & sharpee’s fibers at vertebral waist
   iv) Non marginal syndesmophytes
   v) No erosive arthropathy, squaring, nor narrowing
   vi) Normal ESR
Spinal Injury

Spinal cord syndrome

**COMPLICATIONS OF SPINAL INJURY**

1. Neurological damage
2. Damage to vertebral column causing deformity and pain

**Stable injuries**
- Vertebral components won't be displaced by normal movement.
- An undamaged spinal cord is not in danger.
- There is no development of incapacitating deformity or pain.

**Unstable injuries**
- Further displacement of the injury may occur.
- Loss of 50% of vertebral height.
- Angulation of thoracolumbar junction of > 20 degrees.
- Failure of at least 2 of Denis’s 3 columns.
- Compression # of three sequential vertebrae can lead to posttraumatic kyphosis.

**Anatomical considerations**
- The upper Thx spine (T1-T10) is protected by ribs, sternum and the facet joint orientation.
- At thoracolumbar junction there is a fulcrum of increased motion → risk of injury.
- The middle thoracic spine is a vascular ‘watershed’, vascular insult can cause cord ischaemia (Artery of Adamkiewicz).
- Cauda equina begins at L1-L2. Lesions below L1 have a better prognosis as nerve roots, not cord are affected.
**Mechanisms of injury**

1. Hyperextension - common in the neck. Anterior ligaments and disc may be damaged.
2. Flexion - If posterior ligaments intact, wedging of vertebral body occurs. If posterior ligaments torn, may cause subluxation
3. Axial compression - Causes burst #s. Bony fragments may be pushed into spinal canal
4. Flexion, with posterior distraction - May disrupt middle and posterior columns.
5. Flexion with rotation - Causes dislocation with or without #.
6. Shear.

**SPINAL SHOCK**

1. Diaphragmatic breathing
2. Flaccid limbs
3. warm periphery
4. decr. pain sensation
5. reduced reflexes
6. erection
7. Urinary retention
8. decr. BP + decr. pulse rate = Neurogenic
9. decr. body temp.
10. Return of bulbocavernos reflex at 24hrs= end of Spinal Shock

**INITIAL MANAGEMENT OF SPINAL INJURY PATIENTS**

ABC, Spinal board, hard collar

**History**

- Strongly suspect spinal injury if any major accident, unconscious patient, fall from a height, sudden jerk of neck after rear end car collision, facial injuries or head injury
- Ask about neck or back pain, numbness, tingling, weakness, ability to pass urine

**Examination**

- Logroll - look for bruising, palpate for a step, tenderness
- Repeated neurological examination to determine neurological damage and its progression/resolution
- Thorough overall examination for fractures etc as patient may not feel pain

**Imaging**

- Xrays-Cervical spine AP, Lateral including C7/T1, open mouth view of odontoid. Swimmers view or pull arms down. AP and lateral view of other tender areas of spine
- CT scan shows bony injury
- MR scan shows soft tissue involvement

**If neurological damage**

1. Catheterise
2. Note reduced BP and bradycardia due to neurogenic shock (temporary generalised sympathtectomy). Rule out hypotension due to haemorrhage elsewhere. May need treatment with vasopressors, not fluid resuscitation.
3. Invasive monitoring required
4. Give methylprednisolone IV 30mg/kg over 15 mins then 5.4mg/kg/hr for next 23 hours. Needs to be given within 8 hrs. Discuss with the spinal team.
5. Attend to skin by turning
DEFINITIVE MANAGEMENT

Objectives
- Preserve neurological function
- Relieve reversible nerve or cord compression
- Stabilise the spine
- Rehabilitate patient

With no neurological deficit
- If stable spinal injury- pain relief, collar or brace. Exception can be a burst #, ? operatively stabilise
- If unstable injury, reduce and hold secure until bone / ligts heal with ORIF or traction in tongs, halo vest

With complete sensory and motor loss
- Usually an unstable injury
- Only consider conservative management for high thoracic injuries
- Early operative stabilisation- to help with nursing, prevent spinal deformity and pain. Speeds up rehab

With Incomplete neurological loss
- Stable injury- conservative bed rest, brace. Exception can be burst # ?operatively stabilise
- Unstable injury- early reduction and stabilisation
NEUROLOGICAL ASSESSMENT IN SPINAL INJURIES

Aims
- To determine the level of the lesion - counted as the lowest level at which neurological function is intact bilaterally
- To determine whether damage is complete/ incomplete
- To determine prognosis
- May be difficult until period of spinal shock (flaccidity, areflexia) is over (24-48 hrs after injury)

1. Tone
2. Power

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diaphragm</td>
<td>C3-4-5</td>
</tr>
<tr>
<td>Shrugging shoulders</td>
<td>C4</td>
</tr>
<tr>
<td>Flex elbows</td>
<td>C5,6</td>
</tr>
<tr>
<td>Extend elbows</td>
<td>C7</td>
</tr>
<tr>
<td>Abduct fingers</td>
<td>C8</td>
</tr>
<tr>
<td>Active chest expansion</td>
<td>T1-T12</td>
</tr>
<tr>
<td>Hip flexion</td>
<td>L2</td>
</tr>
<tr>
<td>Knee extension</td>
<td>L3-4</td>
</tr>
<tr>
<td>Ankle dorsiflexion</td>
<td>L5-S1</td>
</tr>
<tr>
<td>Ankle plantar flexion</td>
<td>S1-S2</td>
</tr>
<tr>
<td>Eversion of foot</td>
<td>L5</td>
</tr>
<tr>
<td>Inversion of foot</td>
<td>L4</td>
</tr>
</tbody>
</table>

3. Reflexes

<table>
<thead>
<tr>
<th>Reflex</th>
<th>Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>Biceps</td>
<td>C5-6</td>
</tr>
<tr>
<td>Triceps</td>
<td>C6-7</td>
</tr>
<tr>
<td>Supinator</td>
<td>C5-6</td>
</tr>
<tr>
<td>Knee jerk</td>
<td>L3-4</td>
</tr>
<tr>
<td>Ankle jerk</td>
<td>L5-S1</td>
</tr>
<tr>
<td>Plantar response</td>
<td>If upgoing = UMN lesion</td>
</tr>
<tr>
<td>Abdo cutaneous</td>
<td>If lost = UMN lesion</td>
</tr>
<tr>
<td>Bulbo cavernosis</td>
<td>Pull penis, causes anal sphincter tightening If returned, period of spinal shock is over</td>
</tr>
</tbody>
</table>

4. Sensation

5. PR
1. Always perform this to assess S2, 3, 4
INCOMPLETE CORD INJURIES

<table>
<thead>
<tr>
<th>Syndrome</th>
<th>Injury/pathology</th>
<th>Features</th>
<th>Prognosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>B.Sequard</td>
<td>Penetrating trauma</td>
<td>Loss of ipsilateral motor function and vibration and position sense, contralateral loss of pain &amp; temp</td>
<td>Best</td>
</tr>
<tr>
<td>Ant cord</td>
<td>Flex.compression</td>
<td>Motor loss, dorsal columns spared</td>
<td>Poor</td>
</tr>
<tr>
<td>Central cord</td>
<td>Age&gt;50 ext.injuries</td>
<td>Affects UL&gt;LL, motor and sensory loss</td>
<td>Fair</td>
</tr>
</tbody>
</table>

LONG TERM CARE OF NEUROLOGICALLY INJURED SPINAL PATIENTS

Frankel grade- Useful in monitoring functional improvement from spinal cord injury

<table>
<thead>
<tr>
<th>Frankel grade</th>
<th>Function</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Complete paralysis</td>
</tr>
<tr>
<td>B</td>
<td>Sensory function only below injury level</td>
</tr>
<tr>
<td>C</td>
<td>Incomplete motor function below injury level (1or 2/5)</td>
</tr>
<tr>
<td>D</td>
<td>Fair to good motor function (3-4/5) below injury level</td>
</tr>
<tr>
<td>E</td>
<td>Normal function below injury level</td>
</tr>
</tbody>
</table>

Highest mortality in the first 2 weeks

Most common causes of morbidity and mortality:

1. **Respiratory insufficiency** - atelectasis and pneumonia. Any deficit proximal to T10 causes increased reliance on the diaphragm. Treat with IPPV, physio, tracheostomy and suctions, repeated bronchoscopy
2. **Paralytic ileus** - Keep NBM initially for 24 hours, IV fluids
3. **GI bleeds** from haemorrhagic gastritis - Due to unopposed parasympathetic activity. Give H2 antagonists
4. **Urological** complications - UMN lesion of bladder, catheterise intermittently to stop urinary stasis and infection. LMN, may manage with suprapubic pressure to relieve bladder
5. **Pressure sores** - Turn every 2 hrs, teach to relieve pressure later
6. **Joint contractures** - Passive movements +/- splintage 2x per day
7. **Psychological** withdrawal - multi disciplinary approach.

Conus medullaris syndrome

Injury of the sacral cord (conus) and lumbar nerve roots within the spinal canal, usually results in areflexic bladder, bowel, and lower extremities. Most of these injuries occur between T11 and L2 and result in flaccid paralysis in the perineum and loss of all bladder and perianal muscle control. The irreversible nature of this injury to the sacral segments is evidenced by the absence of the bulbocavernosus reflex and the perianal wink. Motor function in the lower extremities between L1 and L4 may be present if nerve root sparing occurs.

Cauda equina syndrome

Injury between the conus and the lumbosacral nerve roots within the spinal canal, also results in areflexic bladder, bowel, and lower limbs. With a complete cauda equina injury, all peripheral nerves to the bowel, bladder, perianal area, and lower extremities are lost, and the bulbocavernosus reflex, anal wink, and all reflex activity in the lower extremities are absent, indicating absence of any function in the cauda equina. It is important to remember that the cauda equina functions as the peripheral nervous system, and there is a possibility of return of function of the nerve rootlets if they have not been completely transected or destroyed. Most often the cauda equina syndrome presents as a neurologically incomplete lesion.
**Aetiology**

**Risk factors:**
- older debilitated & immunocompromised patient
- IV drug addicts (pseudomonas)
- history of pneumonia, UTI, skin infection
- 70% arise from UTI, chronically ill, elderly adults via **Batson’s venous plexus**
- Sickle cell anemia

**Organisms:**
- Staph aureus ......................................................... 60% & MRSA is on the increase
- Gram negatives (E coli, Pseudomonas, Proteus) & anaerobes are on the increase
- Strep viridans
- brucellae, candidae, coccidiomycosis (in immunocompromised)
- tuberculosis (commonest site = T10)
- Superinfection on TB may occur during surgery

**Route of infection:**
1. **Hematogenous** spread (commonest): can affect any part of the spine; body, arch, facet, odontoid,…Baston has attributed this to the presence of the large valveless vein in the anterior body, but Trueta proved the arterial route being the principle way for infection
2. Direct spread from near structure: colon, subphrenic abscess, abdominal abscess
3. Direct inoculation: postop either after open surgery, nucleolysis, or discography, laser

**Site of infection:**
- lumbar is the commonest ........................................ (60%) éout paralysis
- thoracic & cervical .................................................. higher incidence of paralysis

**Pathophysiology**

1- Haematogenous spread via arterial route (Trueta) rather than the old theory of (Batson’s venous complex); and goes via the end arterioles where it stop:
   - In children: the disc ......................................... (still vascular)
   - In adults: the anterior metaphysis ............ (disc is avascular)

2- toxins cause septic thrombosis, infarct, abscess, blocks nutrition

3- Infection **Spread** locally via:
   - A. Intermetaphyseal anastomosis by passing the segmental artery
   - B. Along the ALL
   - C. posteriorly ➔ epidural abscess .............. Early paralysis δ abscess & oedema
   - D. Anteriorly ➔ paravertebral abscess ........ Late paralysis δ kyphosis

4- **Paravertebral Abscess** may tickle down:
   - A. Cervical: Inter costal ➔ Retropharyngeal, supraclavicular / Brachial ➔ Axilla
   - B. Dorsal: Inter costal ➔ paraspinal / Paravertebral ➔ paranephric, or psoas abscess
   - C. Lumbar: Psoas & Femoral ➔ psoas, femoral / / ➔ Gluteal, ischiorectal, Petits’s △

5- weakening of the bone may cause vertebral **Collapse**.

6- **Paralysis** either early or late ☞ by age, level, DM, staph.aureus; and of 2 types:
   - o Central cord $ ..........................................................66%
   - o Anterior cord $ ..........................................................33%
**Clinical**

- Often a significant **delay** in diagnosis (6-12 weeks)

**Symptoms**

- **Triad** = back pain + fever + tenderness (consider **bacterial endocarditis**)
  1. Pain: .................................................is the hallmark symptom
     - Insidious course, developing over 1-3 mo
     - ↑ activity & ↓ rest
     - May reach to be pain at rest & even nocturnal pain
  2. Fever: .............................................only 50%
  3. Neurology: ...............................only 10%

**Signs:**

1. Local tenderness
2. Ms spasm ....................................... back motion and torticollis ↑ **VE COIN TEST**
3. Deformity ................................. **GIBBUS**
4. ↑ SLR ........................................... **LAEGUE**, Brudziniske, Kernig’s, …
5. Neurological signs ..........................Motor, sensory, reflexes
6. Meningeal signs ............................. **LHERMITE**

**Radiology**

**DRES OFF**

**X-Rays:** (lateral cervical & AP lumbar, dorsal)

- 2 weeks ........................................ disc space **NARROWING** (two bodies + disc affected)
- 4 weeks ........................................ **RAREFACTION**
- 6 weeks ........................................ **ENDPLATE EROSION**: osteolysis
- 8 weeks ........................................ reactive **SCLEROSIS** δ trabecular collapse
- 12 weeks ....................................... **NFB**
- 6-12 months ................................... intervertebral **FUSION** - usually signifies resolution
- Examine paravertebral soft tissue mass - retropharangeal & psoas contours.

**Disc destruction is atypical of neoplasms**

**CT:**

- Can diagnose more early
- Show the signs of rarefaction, erosion, lysis + reactive sclerosis
- Can be combined with myelography:
  - More accurate for cord condition
  - Can with draw CSF for analysis

**MRI**

- Very sensitive & specific & accurate (for differentiating from tumour)
  1. ↑ T1 (loss of distinction bet disc & body)
  2. ↑ T2 é streaky linear appearance + absent nuclear cleft
  3. Gadolinium enhances sensitivity
  4. STIR (short T1; inversion recovery=Fat suppresion); → high contrast bet norm & abnorm

**Nuclear studies:**

- high sensitivity & fair specificity; but all are less accurate than the MRI
  1. **Tc**
     - Flow phase ↓ .....................detect perfusion abnormality
     - Blood Pool phase ↑ .............detect hyperemia
     - Static bone phase ↑ ..........detect osteoblastic activity (absorbed by HA)
  2. **Ga** is more specific for infection the substance is taken by the leucocytes
  3. **In** labelled WBC accuracy is 66% & can not differentiate bet acute and chronic
  4. **SPECT** (single photon emission CT) simply is a CT + radionuclide (best in osteoid osteoma)
  5. Recent: **Strontium** 100% specific
Investigations

1- Leucocytosis ........................................30% increase
2- ESR ..................................................30% > 100, 70% > 50
3- CRP ................................................30% positive
4- blood cultures ..............................30% positive
5- CT guided FNAC ...............................68-86% +ve; but sacrum are not safe for needle aspiration; do stains (Gram & AFB & fungi), Cultures, histopathologic examination
6- Specific for organisms:
   o ASOT
   o anti-staph. titres
   o Tuberculin skin tests for TB
   o IFAT, IHAT for brucellosis and salmonellosis

Treatment

Goals: (of any ttt):

- ttt the disease
- relief the symptoms
- prevent complications: collapse, deformity, neurological symptoms

Non Operative

- IV AB for 6w — Ord — 6wk.
- LSO / TLSO / CO / CTO / CTLSO (Milwaukee)
- Follow up by: CP, PXR, MRI, ESR an other lab

Operative

- Indications
  - failure of response to medical treatment
  - Progression of the disease
  - severe pain and neurological symptoms
  - Complications: instability, deformity, abscess
  - need for open biopsy
- Objectives:
  1- Debridement
  2- Decompression of the cord
  3- Realignment
  4- Stabilization
- Approaches:
  o **Anterior Approach** provides better exposure + draining abscesses
  o Ant. decompression & strut **Graft**. (for all levels cervical, thoracic, and lumbar)
  o Role of spinal **Institution** is controversial - with a large deformity, posterior instrumentation may be indicated
Tuberculosis

- Tuberculosis is common throughout the world
- Usually due to Mycobacterium tuberculosis or Mycobacterium bovis infection
- Spine is the most common site of skeletal TB

Pathology:

1\text{st} lesion:
- Site:
  - Lung usually (sub-pleural Ghon’s Focus + mediastinal lymphadenopathy)
  - Pharynx & Gut
- Changes:
  - Local inflammatory focus \rightarrow Lymphangitis \rightarrow Lymphadenitis
- Seculae:
  - TB bacilli remain dormant in LN (intra macrophage)
  - Body is sensitized to toxin (Type IV delayed hypersensitivity)

2\text{nd} lesion:
- Due to reactivation, repeat exposure, \Diamond immunity (e.g. drugs or HIV infection)
- Results in more significant symptoms as it spreads to:
  - Lung .................................. military TB, TB bronchopeumonia
  - Meninges:................................

3\text{rd} lesions (10% affect the musculoskeletal system)
- \textbf{Tuberculoma} formation:
  - Central \textbf{CASEATION} necrosis (coagulation necrosis)
  - Surrounded by \textbf{EPITHELIOID} cells, \textbf{LANGERHANS} giant cells, \textbf{LYMPHOCYTES}
  - They tend to coalesce to form a wide area of caseation necrosis
- \textbf{TB Arthritis} (hip, knee, ankle, shoulder, then wrist)
  - Synovium is \textbf{THICKENED} \& Cell rich \textbf{EFFUSION}
  - Granulomatous \textbf{PANNUS} may form \& creaps on the cartilage & bone
  - Cartilage & bone \textbf{EROSION} (peripherally at synovial reflection)
  - Juxta articular \textbf{OSTEOPENIA} \& hyperaemia
- \textbf{Appendicular skeleton}:
  - Metaphyseal bone destruction (no sclerosis, no periosteal reaction)
- \textbf{TB Dactylitis = Spina Ventosa} (middle and distal phalanx)
  - Digit is swollen spindle shape \& little pain
  - Starts diaphyseal \& bone rarefaction + PNBF + soft tissue swelling
  - PXR: Spina \{spindle shaped digit\} Ventosa \{full of are i.e. rarefied\}
- \textbf{Cold Abscess}:
  - Infected LN may \textbf{COALASE} together to form big area of caseation
  - Caseation spread via soft tissue planes i.e. through skin or along ms fascia or a bundle
  - May burst to skin to form a sinus
- \textbf{TB Spondylitis}:
  - Blood borne - settles in vertebral body anteriorly
  - Thoracic is commonest \pm skip lesions \& multiple level (lumbar for OM \& discitis)
  - \textbf{POTT’S PARAPLEGIA}

Healing:
- \textbf{Resolution}
- \textbf{Fibrous Ankylosis}
- \textbf{Dormant} bacilli
**TB Spondylitis:**
- Sites of vertebral affection respectively:
  1. Anterior in the body the most vascular part of the body
  2. Paradiscal & Central are less common
  3. Synovial affection of the facetal joint is rare
  4. Posterior elements are recorded as well
- Thoracic is commonest ± skip lesions (lumbar for OM & discitis)
  1. May be **MULTIPLE LEVEL** form the start
  2. may have **‘SKIP LESIONS’**
  3. **SPREAD** locally:
    - [A]. Extensive destruction, more sequestra, gaseous pus > pyogenic
    - [B]. IV Discs preserved until late when break via the end plate
  4. **COLD ABSCESS** formation more common than pyogenic OM:
    - [C]. Cervical: —— Retropharyngeal, supraclavicular / —— Axilla
    - [D]. Dorsal: Inter costal → paraspinal / —— paraneural. Fascia
    - [E]. Lumbar: —— psoas & femoral. Axillai → Gluteal, ischiorectal, Petit’s △
  5. **COLLAPSE** of anterior vertebral body → sharp kyphosis if progressive →
  6. **POTT’S PARAPLEGIA:**
    1. **EARLY** Pott’s paraplegia:
      - [F]. Progress then subside: paraplegia ð abscess & **OEDema**
      - [G]. Progressive ð mech pressure by **SEQUESTRA**, disc, or bone bridge
      - [H]. Acute paraplegia ð **THROMBOSIS** or concertina collapse of Seddon
      - [I]. Compression paraplegia ð **TB OF LAMINA** or encysted TB →
    2. **LATE** Pott’s paraplegia:
      - [A]. Soon after cure: ð **REACTIVATION** of the disease
      - [B]. Very late: ð **CORDOMALACIA** & friction against kyphotic canal

**Hodgson Classification of Pott’s Paraplegia:**

<table>
<thead>
<tr>
<th>Group A</th>
<th>Active disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>External pressure on the cord by an abscess or laminar TB</td>
</tr>
<tr>
<td>2</td>
<td>Penetration of dura by infection</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Group B</th>
<th>Healed Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Cord Transection by the kyphotic bone</td>
</tr>
<tr>
<td>2</td>
<td>Constriction of cord by granulation &amp; fibrosis</td>
</tr>
</tbody>
</table>

**Clinically:**
- General: Night sweat, night fever, Loss of weight, loss of appetite
- Local:
  - **NIGHT CRIES**: ð  † ms spasm → stretch of the damaged tissue → pain
  - Marked **MS WASTING**
  - **TB arthritis:** **SYNOVIAL THICKENING, STIFFNESS, DEFORMITY**
  - **POTT’S PARAPLEGIA:** (4 Grades)

<table>
<thead>
<tr>
<th>Paraplegia</th>
<th>Motor</th>
<th>Sensory</th>
<th>Jerk</th>
<th>Clonus &amp; Babinski</th>
<th>Sphincters</th>
</tr>
</thead>
<tbody>
<tr>
<td>1- Mild</td>
<td></td>
<td>Intact</td>
<td>Slight increase</td>
<td>-ve both</td>
<td>Intact</td>
</tr>
<tr>
<td>2- In extension</td>
<td>Pyramidal weakness of flexors</td>
<td>+</td>
<td>Brisk extensor</td>
<td>+ve both</td>
<td>Intact</td>
</tr>
<tr>
<td>3- In Flexion</td>
<td>Complete motor paralysis of both</td>
<td>+</td>
<td>Brisk flexors</td>
<td>-ve clonus</td>
<td>Intact</td>
</tr>
<tr>
<td>4- Complete</td>
<td>Same</td>
<td>Lost</td>
<td>Lost both</td>
<td>Same</td>
<td>Lost</td>
</tr>
</tbody>
</table>
1. **Juxtap Articular Osteopenia** → washed out bone ends
2. **Concentric Joint Space** (child epiphysis → δ) → **Phemister Triad** (hyperaemia)
3. Peripheral bone **erosion** and **cystic** subchondral
4. **No** periosteal reaction
5. **No Sclerosis**
6. Erosions in **Babcock’s Δ, Sup. Acetabulum** → **Wandering Acetabulum**
7. **Pott’s** disease of the spine

   [1]. Earliest sign is **Osteopenia** of two adjacent vertebrae
   [2]. **Erosion:**
      - **Multisegmental**
      - **Skip** lesions
      - **Scalloping** of the anterior border of the vertebra
      - **Disc Sparing** (*pyogenic*)
   [3]. **Soft Tissue Mass** .......... Paraspinal abscess
   [4]. **Collapse:**..........................**Kyphosis / Rib Sun Ray Appearance**
   [5]. **Healing:**............................**Bone Density** & paravertebral abscesses may **Calcify**
   [6]. **Konstam angle:** predict the development of kyphosis ê 90% \( \gamma = a + bx \)

   \[ \gamma = \frac{5.5 + 30.5 \times \text{Konstam at presentation}}{\text{No of Vertebrae}} \times \frac{1}{10} \]

   - a and b are constants = 5.5 & 30.5, respectively

**MRI**
- No ↑ of disc space signal δ granulation (*pyogenic*)
- Extent of the destruction and soft tissue mass
- Cord condition e.g. cordomalacia, compression ...

**Bone Scans**
- high false -ve rate with Tc & Ga

**DD**
- Other infections
- Traumatic paraplegia
- Tumors
- Rheumatoid & AS
- Sheurmann

**Investigation**
- Blood:
  1. ↑ ESR
  2. Leucopenia é relative lymphocytosis
  3. ↑ Lymphocyte/monocyte ratio ≈ 1
- Immunological:
  1. +ve PCR
  2. +ve Mantoux test
- Aspirate fluid:
  1. Physical: ......................↓ viscosity
  2. Chemical: ......................↓ ptn / ↓ glucose / poor mucin clot
  3. Bacteriological: .............. Red acid-alcohol fast **INTRACELLULAR** bacilli é **Zeal Nelsen** ..........20%
  4. Cultivation: ..................... **Lowenstein Jensen** media or Dorset egg .........................80%
     - Concentrated centrifuged decontaminated sample (Petroff method)
     - Keep 35° for 6 wk
  5. Organisms also **Fluoresce With Auramine** staining
  6. Guinea pig inoculation
- biopsy:
  - Granulomatous reaction (caseation + Langerhans + epitheliod + lymphocytes)
  - Characteristic evidence of a delayed hypersensitivity reaction
- TB tend to give negative results

**Pyogenic DD:**
- Pyogenic chronic
- Single vertebral focus Multisegment
- Symmetrical collapse Kyphosis
- Spreads intrasosseously Spread é fascial planes
- Disk destroyed ↑ T2 Disk sequestered ↓ T2
- Ant-column involvement 3 columns involvement
- Epidural abscess Paravertebral abscess
### Skin tests
- Delayed hypersensitivity reaction used to diagnose tuberculosis
- The two commonest tests are the Mantoux and Heaf test
  **Mantoux test:**
  - 0.1 ml of purified protein derivative is injected intradermally
  - +ve if > 5 mm papule at 72 hours
  **Heaf test**
  - PPD is inoculated into the skin using a gun to produce multiple punctures
  - +ve if > 4 papules at puncture sites at 72 hours
- Positive skin test are indicative of active infection or previous BCG vaccination

### Treatment

1. **Rest**: (in Acute stage)
   - Bed rest + Spinal orthosis (prevent deformity and ms spasm) till:
     1. Clinically: No fever, no wt loss, no spasm
     2. **PXR:** ↑ calcification
     3. **Lab:** ESR, lymph/monocyte ratio > 5
   - Then motion is encouraged é the orthosis on 18mo → gradual weaning & if pain & spasm return → resume orthosis

2. **Chemotherapy**: (at lease 9-12mo)
   - RIPES - Rifampicin, Isoniazid, Pyrazinamide, Ethambutol, Spectinomycin
   - Rifampicin + Isoniazid 6-8 mo give 80% recovery of the infection
   - Ethambutol (or pyrazinamide, spectinomycin) for the initial 8wk
   - Streptomycin is toxic

3. **Operative**
   - Indications:
     1. Large abscess
     2. Instability
     3. Neurology
     4. Progressive impairment to pulmonary function
     5. No response to medical therapy
     6. Young pt ê Konstam angle > 150º (↑ risk for kyphosis progression)
   - Adjuvant chemotherapy beginning 10 days before surgery is essential

I. **Hong Kong Procedure**:
   - Radical anterior debridement
   - Anterior fusion iliac strut grating (allows better correction of the kyphosis)
   - Posterior instrumentation

II. **Costotransversectomy**:
   - Excision of the transverse process + medial 4 cm of the rib
   - Evacuation of the abscess and closure

III. **Lateral Rhachotomy**:
   - As costotransversectomy + removal of the ant and side wall of the canal
   - Laminae, pedicles, and facets are preserved

IV. **Laminectomy**:
   ±
   - Cold abscess calls for urgent drainage
   - GT bursitis Bursectomy
   - Painful destroyed joint then arthroplasty
**Discitis (Juvenile)**

**Definition**
- Inflammation of the intervertebral disc or end plate in young children, probably an autoimmune condition, no sepsis to be found.

**Epidemiology:**
- 2-6 y = 30%
- Self-limiting condition = 30%
- No organism in culture = 75%
- L4, L5 commonest = 75%

**Pathology:**
- Inflammation of the IV disc or end plate.
- It may represent extension of subacute vertebral endplate osteomyelitis which did not progress to give vertebral osteomyelitis.

**Clinical**
1. Children are typically afebrile or mild fever (≠ osteomyelitis)
2. Back pain
3. Child refuses to stand, walk, or flex the spine
4. May also complain of hip or abdominal pain
5. Tenderness, paravertebral spasm, loss of normal lumbar lordosis, ↓ ROM

**Investigations**
- WBC is usually normal
- ESR elevated > 40
- When a causative organism can be identified, it is most commonly S. aureus.
- Biopsy is indicated if failed response or if an organism is suspected.

**Radiology**
- X-rays:
  - May appear normal early on.
  - Disc-space NARROWING
  - Loss Of Distinction between adjacent vertebral end-plates (never severe destruction).
  - Later - in adult disc space usually FUSE, while in child usually is RESTORED.
- Bone Scan:
  - Tc uptake of isotope in infected disc space - may be useful in early diagnosis of discitis.
- MRI:
  - MRI is more sensitive than bone scans in early discitis.
  - T1 and T2

**Treatment**
- Bedrest + TLSO (avoid traction)
- Empirical systemic antibiotics (probably make no difference)
- Never surgery.
Epidural Abscess

- Uncommon devastating condition 2:10000
- Mortality rate = 12%

**Aetiology:**
1. Same org as pyogenic
2. Same predisposing factors
3. more common in immunocompromised, malignancy, DM, alcohol abuse, invasive procedures, vertebral fractures

**Pathology:**
- **Level:** thoracic spine most common
  - Followed by lumbar then lastly the cervical as it has little epidural fat
  - Epidural abscess tend to span over 2-3 levels; but may spans the whole spine
- **Site:**
  - Dorsal abscess is the most common in thoracic, lumbar & following surgery
  - Ventral abscess is the most common in cervical region & discitis or osteomyelitis
  - Severe necrosis, suppuration

**Clinically:** Triad Of Fever, Leukocytosis, ESR, Neurological Signs
- Early: Unexplainable Axial Pain + Fever
- 3d Later: Rapid Deterioration E RADICULAR PAIN, NEURAL DEFICIT, and MENINGEAL SIGNS
- Must be kept in mind when investigating a patient for spinal infection or tumour

**Investigations:**
- ↑↑↑ ESR
- ↑↑ Leukocytosis

**DD**
1. Other infection
2. Tumors
3. Epidural hematoma
4. Acute transverse myelitis
5. Meningitis

**Treatment:**
1. it is a medical & surgical EMERGENCY :
2. Immediate DECOMPRESSIION: - laminectomy usually is sufficient as the lesion tend to be thoracic and dorsal
3. Ventral cervical abscesses require anterior approach
4. Immediate empirical broad spectrum Ab that cross the BBB
5. Stabilization + BG

**Poor prognostic factors:**
- rapidly progressing paralysis
- complete paralysis
- neurological deficits for > 36 hours
- immunocompromised, DM, elderly
Post-Operative Infections

**Incidence:**
- Discectomy ........................................ 0.5%
- fusion éout instrumentation .............. 2%
- fusion é instrumentation .......................... 5%

Post-op disciitis may be difficult to diagnose - keep in mind.

**Clinical**
- ↑ Back pain not coinciding with the wound appearance
- May be considered as malingering or hysterical
- Tenderness, paravertebral spasm
- ↑ SLR
- ± neurological signs

**Investigations:**
- ↑ ESR ± normal leucocytes
- PXR & Tc are not conclusive
- MRI is of choice → **BODEN TRIAD** = ↑ T1 / ↑ T2 at disc / ↑ ↑ ↑ T1 gadolinium or BM & disc

**Treatment:**

**Prevention:**
- Prophylactic antibiotic
- Delicate handling of the tissues
- Meticulous hemostasis
- Debridement of jeopardized tissues before closure

**Active ttt:**
1. Open the wound in toto
2. Debride the necrotic & pus
3. Irrigate with 8-10 L of Ringer’s
4. Remove the instrumentation
5. Closure é suction irrigation for 5 days
6. Dressing UGA is done after the 5 days

---

**COMPARISON TABLE**

<table>
<thead>
<tr>
<th></th>
<th>Osteomyelits</th>
<th>Discitis</th>
<th>TB</th>
<th>Tumour</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age</strong></td>
<td>adult</td>
<td>child</td>
<td>any</td>
<td>any</td>
</tr>
<tr>
<td><strong>Region</strong></td>
<td>lumbar</td>
<td>lumbar</td>
<td>thoracic</td>
<td>lumbar</td>
</tr>
<tr>
<td><strong>Site</strong></td>
<td>metaphysis</td>
<td>end plate,disc</td>
<td>anterior body</td>
<td>posterior body</td>
</tr>
<tr>
<td><strong>Disc involvement</strong></td>
<td>yes</td>
<td>yes</td>
<td>no</td>
<td>no (except lymphoma &amp; myeloma)</td>
</tr>
<tr>
<td><strong>ESR</strong></td>
<td>high</td>
<td>high</td>
<td>high</td>
<td>normal</td>
</tr>
<tr>
<td><strong>WBC</strong></td>
<td>high</td>
<td>N</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td><strong>Biopsy</strong></td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>+</td>
</tr>
</tbody>
</table>
Brucellosis

_Etiology:_
Organism:
- Non motile, non spore forming, acid fact, gram –ve _coccobacilli_
- Route of infection ingestion of milk product of infected cattles

_Pathology:_
- Usually affect the spine, lumbar spine 15% of all brucella infection
- Granulomatous lesions with giant cell caseating lesions are present
- Spinal cord compromise occur in 12%

_Clinically:_
- Fever, anorexia, headache, malaise, night sweat
- Polyarthralgia

_PXR:_
- Step like spinal erosion at the anterior margin of the vertebra
- MRI show a picture like that of TB

_Lab:_
- Brucella titre: 1:80 or more

_Treatment:_
- AB: Deoxyycyclin, Rifampicin, Vibramycin for 4 mo may be sufficient
- Surgery: is rarely indicated and if so it follows the rules for the pyogenic surgery

Fungal Infection

- Usually it is opportunistic infections that affect patients with bad general condition
- They are slow infections, less painful, é similar PXR and lab finding as pyogenic infection
- DD: TB & tumors
- Dx: is made absolutely by isolation of the organism by biopsy and direct cultivation

- Aspergillus:
  - Is an opportunistic infection esp in cardiac and renal transplanted pt
  - Spinal affection .....................65%
  - ttt: amphotericin B ± surgery only if neurologic compression

- Cryptococcal infection:
  - In leukemia & sarcoidosis
  - ttt: Amphotericin B

- Candidiasis
- Actinomycosis
- Blastomycosis
- Coccidioidomycosis

_Echinococcus Granulosus_

- 1-2% may affect the bone; è the spine being 50% of these cases
- Space occupying lesion that may enlarge → weaken the vertebra & collapse
- CP: mainly pain, swelling, and deformity
- PXR: large lytic expanding lesion (like gct, or fibrous dysplasia)
- Dx: _casoni_ test
- Ttt: mebendazole + surgery
Paediatric Spinal Deformities

Biomechanics
- Kyphosis = failure in 1 plane. **Scoliosis** = failure in 3 planes (sagittal, rotation & lateral wedging)
- **Heuter-Volkman Law** = Pressure on epiphysis $\downarrow$ rate of growth; whilst tension $\uparrow$ the rate. Thus the 'leading edge' of a deformity grows more rapidly than the 'trailing edge' $\rightarrow$ $\uparrow$ the rate of progression. (eg. Scoliosis)

Scoliosis Classification (International Scoliosis Society):
1- Idiopathic: ................................................................. 65%
   1- Infantile <3y:  ▪ Regressive ...... 85%
   ▪ Progressive .... 15% (worst)
2- Juvenile 3-8y
3- Adolescent >8y

2- Congenital: ................................................................. 15%
   o Posterior Failed Formation: wedge vertebra, or Hemivertebra
   o Posterior Failed Segmentation: Unsegmented Bar, Block vertebra, Unfused Ribs
   o Anterior Open: spina bifida / Anterior Closed: Diastomato-myelina

3- Neuromuscular ....................................................... 10%
   o Neuropathic
      ▪ UMNL - CP, Spinocerebellar degeneration, cord tumor, cord trauma, other
      ▪ LMNL - polio, trauma, spinal muscular atrophy, myelomeningocele
   o Myopathic - dystrophies (Duchenne, LGD, FSHD), Arthrogryphosis, Congenital hypotonia, myotonia, myasthenia

4- Neurofibromatosis (Kyphoscoliosis) .... 5%
   o +ve family history, 5 café au lait patches, pachydermatocoele, pseudoarthrosis

5- Others: ................................................................. 5%
   1- Dysplasias - Achondroplasia, SED, diastrophic dwarfism, MPS
   2- Traumatic Fracture/dislocation, irradiation, burns
   3- Rheumatoid
   4- Tumours
   5- Metabolic - Rickets, OI, Marfans, homocystenuris, Ehlers-Danlos
   6- Functional - postural, leg length, ms spasm, Hysterical it not a structural scoliosis:
      ▪ Correct by leaning forward or lying down
      ▪ Coleman blocks
Congenital Scoliosis

- Congenital scoliosis is a developmental curvature of the spine due to vertebral anomalies

**Classification:**

1. Failure of Formation - Partial Unilateral (wedge vertebra), Complete Unilateral (Hemivertebra)
2. Failure of Segmentation - Unilateral (Unilateral unsegmented bar), Bilateral (Block vertebra)
3. Mixed

**Assessment:**

1. Severe spinal deformity
2. Overlying skin shows: angioma, tuft of hair, nevus, fat pad
3. Radiography: must be extensive to diagnose the type of the malformation
   a. PA, Lat, lateral bending, Risser Cortel, Ferguson, Stagnara
   b. (MRI) or myelography should be performed to see if there are any associated intraspinal anomalies. Should an anomaly, such as *diastematomyelia*, be discovered, it should be resected before correction of the scoliotic curve.

**Treatment** (staged operation)

- Resection of the curve apex
- Anterior and posterior spinal fusion

---

NeuroMuscular Scoliosis

- **Etiology:** as before
- **Pathology:**
  1. Long severe curve convexity to the paralyzed side
  2. Marked instability
  3. In severe there is pelvic obliquity, sitting imbalance
  4. Loss of sensibility
- **PXR:** in traction to assess the correctability
- **Rx:**
  1. Mild: .......................................................... No ttt
  2. Moderate: .................................................. as idiopathic
  3. Severe: ................................................... anterior and posterior fusion

---

Scoliosis in Neurofibromatosis

- **CP:**
  1. +ve family history
  2. >2 Skin multiple neurofibromatosis
  3. >6 Café au lait patches
  4. Optic glioma
  5. Iris lisch nodules on slit lamp
  6. Pseudoarthrosis
  7. Scoliosis .................................(30%)
     - Short sharp curve
     - Mild to severe in degree
- **Rx:**
  1. Mild: .................................................. as idiopathic
  2. Moderate: ................................. Combined anterior and posterior fusion
Idiopathic Scoliosis

Aetiological theories:
1. **Endocrine Theory**: Patients with idiopathic scoliosis often taller with high somatomedin levels.
2. **Equilibrium Theory**: Abnormalities in the vestibular system. Scoliosis was induced in rats by destruction of brain stem.
3. **Neurotransmitter**: Removal of pineal gland in chickens → SCL.
4. **Genetics**: Multifactorial mode of inheritance ( Particularly degree relatives having scoliosis).

**Epidemiology**:
- Overall prevalence is 25:1000.
- Small curves are more common.
- Right-sided prevalence of spine asymmetry probably due to descending aorta on left.
- Increased incidence in girls explained by normal flattening of thoracic kyphosis at age 12, which corresponds to female growth spurt.

**Pathogenesis**:
- **LORDOSIS**: May be the initiator of deformity as it shifts the normal axis of rotation backward.
- Pt tends to flex the trunk to correct the deformity, and this causes rotation of lordotic section to convexity to one side (usually right). Vertebral changes occur after the rotation.
- **Spinous Process**: Rotate to concavity & the rib hump to convexity.
- At first the deformity is correctable then as it exceeds the limit of stability spine buckle & rotate into a fixed position. Usually this is accompanied by appearance of a 2ry compensatory curve Ω is less marked and easily correctable for a while. The process deformity continue to a rate that may reach 1º/y (if the curve >50º); & at maturity

**Pathology**
1. **IV Disc**: GAG & collagen content.
2. **Body**: Concave side are hypoplastic & convex side hypertrophied (Heuter Volkmann law).
3. Paravertebral **Musculature**: Diff ms fibres on either side of curve.
4. **Ligaments** and tendons: PLL is thickened.
5. **Curve Patterns**

<table>
<thead>
<tr>
<th>Curve</th>
<th>Region</th>
<th>Start</th>
<th>End</th>
<th>Apex</th>
<th>Prevalence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rt Dorsal</td>
<td>Dorsal</td>
<td>D5</td>
<td>L1</td>
<td>D9</td>
<td>85%</td>
</tr>
<tr>
<td>Lt Lumbar</td>
<td>Lumbar</td>
<td>D12</td>
<td>L4</td>
<td>L2</td>
<td></td>
</tr>
<tr>
<td>Rt Dorsolumbar</td>
<td>both</td>
<td>D9</td>
<td>L2</td>
<td>D12</td>
<td></td>
</tr>
<tr>
<td>Double major</td>
<td>Rt dorsal</td>
<td>Lt lumbar</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Double dorsal major</td>
<td>Lt upper</td>
<td>Rt lower</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Classifications**:
- Classified according to time of onset (old SRS)
  1. Infantile: < 3y
  2. Juvenile: 3-8y
  3. Adolescent: 8-20y
- According to curve pattern.

**King Moe Classification**: see later.

![Diagram of Spine Patterns](image)
Assessment

Diagnosis holds 4 parameters:

1. **ÆTILOGY**
2. **REGION**
3. Convexity towards which **SIDE**
4. **COMPENSATED** or not

Complaint:

1- Usually progressive deformity
2- Pain is an unusual symptom and must rule out other causes
3- Neurological or cardiopulmonary complications

General examination:

1- Cardiopulmonary status
2- Skin: café au lait patches
3- Associated congenital malformation: CVS, CNS, GUS
4- Delayed 2ry sexual characters + family history of late menarche (both ♀ the progression)
5- LL examination:
   - LLD correctable or not **COLEMAN TEST**
   - Pelvic obliquity corrected or not **PILOL TEST**

Local examination

- Complete trunk exposure: and the pt is examined from back, forward, and sides

1- Assess the **CURVE** region & convexity e.g rt dorsal curve & rib hump

2- Assess if it is **COMPENSATED** or not:

   ![Diagram](image)

   [1]. **PLUMB LINE** form C7 vertically down
      - and note its distance to the natal cleft
   [2]. Shoulder level
   [3]. Scapular level
   [4]. Arm Torso Distance
   [5]. Asymmetrical loin creases

3- Assess **CORRECTABILITY**:
   1- Lean forward → ♀ or ♂ the rib hump (**ADAM’S TEST**)  
   2- Traction
   3- Lt & Rt bending

4- Assess pelvic **OBLIQUITY**:
   1- Block test of **COLEMAN**
   2- **PILOL** test: pt is seated on a pillow over the lower pelvis only and see if it corrects

5- **NEUROLOGICAL** examination is essential

6- **ROM**
Radiological

1. **View:**
   - First: **Pelvis AP & LT Wrist** to assess the skeletal maturity
   - **PA:** (辐射 to breast & ovary): **Supine, standing, Rt & Lt bending, in traction**
   - **Lateral:** supine and standing (flexion, extension)
   - **Stagnara Derotation View:** cassette lie // to medial aspect of the rib hump & ! beam
   - **Ferguson View:** which is taken perpendicular to the plane of the L5/S1 disc, is used to look for abnormalities at the lumbosacral junction

2. **Changes:** Ætiology, Dx of curve, Describe, Complication
   - Congenital malformation, dysplasia, tumor, … etc
   - Bodies, pedicles & discs narrow to concavity
   - Curve side, region, & Rotation
   - End vertebra most tilted
   - Apical vertebra ...... at centre of curve
   - Stable vertebra ...... bisected by mid-sacral line
   - Neutral Vertebra .... see both pedicles equally
   - Compensatory changes: e.g. pelvic obliquity
   - Completely correctable by bending or not

3. **Measurements:**
   - **Cobb Angle:**
     - Line drawn along upper end plate of upper end vertebra and lower end plate of lower end vertebra. Perpendiculars drawn from these lines. Angle of intersection measured.
     - For double curve, one vertebra is lower end vertebra for upper curve and upper end vertebra for lower curve (transitional curve). Only one line drawn on this vertebra.
     - Future, measurement should always be from same vertebrae.
     - True size of curve can be measured by Stagnara derotation views that measure both lat curves of scoliosis & sagittal curves of kyphosis for more accurate figures and isolation of a particular curve

   - **Mehta Rib-Vertebral Angle Difference (RVAD):**
     - Is the difference bet the angle formed by a vertical line through the apical vertebra & rib lines on either side
     - **RVAD > 20°** or overlap of the head of the rib over the vertebra = progressive infantile ⇒ will

   - **Risser’s staging** - Indicates skeletal maturity and physiological age. Based on ossification of the iliac crest apophysis & graded 0-5 from anterior to posterior. The iliac crest is divided into quarters & ossification graded according to the number of quarters with ossified apophysis (from ant to post), with Stage 5 = fusion of the ossified apophysis.
Moe Method - measures the apical vertebra rotation:
- Draw the Dattum line and divide the area from it to the lateral border into 3 zones; and grades are according to the position of the convex side pedicles in relation to these zones:
  - Grade I: slight asymmetry but still in zone 1
  - Grade II: convex side pedicle entered the 2nd zone
  - Grade III: entered the middle zone
  - Grade IV: passed the Dattum line
- Traction radiographs RISER-COTREL frame are used to assess torso balance and pelvic leveling, which decides between a one-stage PSF or 2-stage APSF approach for neuromuscular scoliosis
- ISIS (integrated shape imaging system) true 3d computerized image of the spine provide true sagittal profile no need for multiple PXR

Other investigations:
- A painful functional scoliosis Tc to exclude tumour or infection
- Pulmonary function test before the operation
- MRI - If intraspinal pathology suspected; e.g. Left-sided, Male, Painful, Rapidly progressive, Neurological abnormality.
- 20% of right curves have pathology, 80% of left curves have pathology

Progression:
- Progression is defined as: > 5º of change on 2 sequential x-rays. Not all curves progress. The larger the curve at presentation the more likely it is to progress.
- Risk Factors for progression:
  1. Female: Incidence is relatively equal; but larger curves are more common in ♀
  2. Young age at Diagnosis: < 12y has 3 x greater chance for progression
  3. Single thoracic curve
  4. Sexually immature (premenarche)
  5. Skeletally immature - Risser 3-5 < 20% Risser < 2 ∼ 50% /
  6. Nash & Moe GIII, GIV
  7. Cobb angle > 50º
  8. Mehta RVAD > 30º
  9. Lonstein Progression factor > 1.5

Lonstein progression factor [Pf]
- Pf = (Cobbº - 3 x Risser) / chronological age
- Pf 1 = 25% risk / Pf 1.5 = 50% risk / Pf 2 = 75% risk / Pf 2.5 = 100% risk
- Growth potential evaluated by a number of factors- Historical, Age, Menarche, Growth spurt (outgrowing shoes and clothes), Height, Tanner’s sign (breasts and pubic hair), Risser stage, Hand (Gruber & Pyle)
- Risk factors in thoracic curves = Vertebral rotation (RVAD > 30º, apical rotation > 30º)
- Risk factors in lumbar spine = Vertebral rotation, Direction of curve (right), Position of L5 (not below intercrest line)
- 70% of curves progress after skeletal maturity & progress an average of 20º
- Curves < 30º not progress
- Curves 50º-75º progress; esp. thoracic curves by a rate of 1º/y
**Classification - King-Moe**

- Type I - lumbar dominant (10%) - S-curve, Both thoracic > lumbar curves cross midline
- Type II - thoracic dominant (33%) - S-curve, Both thoracic > lumbar curves cross midline
- Type III - thoracic (33%) - Thoracic curve, Lumbar curve does not cross midline
- Type IV - long thoracic (10%) - Long thoracic curve, L5 over sacrum, L4 tilted into curve
- Type V - double thoracic (10%) - Double thoracic curve, T1 tilted into upper curve

![Diagram of spinal curves](image)

**Treatment**

Depends on

1. Cobb angle
2. Growth potential:
   - Premenarche (girls) and axillary hair (boys) = .... Rapid growth
   - Risser 2 = ............................................................. Rapid growth

<table>
<thead>
<tr>
<th>Curve</th>
<th>Rapid growth</th>
<th>Decreased growth</th>
<th>Adult</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt;40º</td>
<td>brace</td>
<td>Observation</td>
<td>Observation</td>
</tr>
<tr>
<td>40-60º</td>
<td>surgical</td>
<td>Surgical or observation</td>
<td>Observation</td>
</tr>
<tr>
<td>&gt;60º</td>
<td>surgical</td>
<td>Surgical</td>
<td>Observation</td>
</tr>
</tbody>
</table>

**Non-surgical treatment**

- **Milwaukee (CTLSO)** if apex above T8: Provides passive correction by pressure on convex side and active correction by muscle contraction pulling body away from pads.
- **Boston (TLSO)** if apex of curve below T8
- Brace worn 23 hours a day. Allowed out to play sport. Patient then seen every 3-6 months till maturity if curve progresses > 45º → surgery indicated.

**Surgical treatment**

**Rationale & Goals**

1. **Correct** the deformity (the rotation and the rib hump)
2. Prevent **Respiratory** insufficiency
3. **Rigid Fusion Of Structural Curve** only and not compensatory curve (é bending films)
4. Avoid fusion < the measured curve and usually more
5. Avoid fusion to L5 (L4 or sacrum)
6. Avoid fusion above T1
7. Fuse down to **Neutral Stable Vertebra**
8. Fuse **Level Above & Level Below** measured curve

**Plan:** Anterior or posterior:

- If correctable (é Risser Cortel) < 50% → **Ant Release** → fusion + BG
- If correctable > 50% → **Post Fusion** direct, and be ware of the crankshaft phenomenon
- Costoplasty: may be done for correction of rib hump

If documented progression
Availability of the brace
Compliance of pt
Young Female
Level of Fusion

Treatment according to king Moe classification

<table>
<thead>
<tr>
<th>King</th>
<th>Description</th>
<th>Limits</th>
</tr>
</thead>
<tbody>
<tr>
<td>King I</td>
<td>Fuse both curves to lower vert</td>
<td>No lower than L4</td>
</tr>
<tr>
<td>King II</td>
<td>Selectively fuse thoracic curve only</td>
<td>Lower level at stable vert (rather than neutral)</td>
</tr>
<tr>
<td>King III</td>
<td>Fuse measured thoracic curve</td>
<td>Lower level at first stable vertebra</td>
</tr>
<tr>
<td>King IV</td>
<td>As for king III</td>
<td>Usually stop at L4</td>
</tr>
<tr>
<td>King V</td>
<td>Fuse both thoracic curves</td>
<td>Lower level at stable vertebra</td>
</tr>
</tbody>
</table>

Instrumentation:
- **Posterior**: Complete excision of facet + laminae + transverse processes + BG
- **Harrington rod and hook**: No longer used
- **Wisconsin**: instrumentation wires passed through spinous processes. No longer used
- **Luque**: rigid & control some rotation but wires endanger the dura
- **Cotrel-Dubousset**:
  - 2 Rods & multihooks (1 rod on convex for compression, 1 on concave for distraction)
  - Initial distraction and subsequent rotation.
  - Rods cross-linked with transverse linkage bars → Rigid fixation so no TLSO needed
  - Technically more difficult & very expensive.
- **Anterior**: release of rigid anterior structures as ALL, disc, PLL then fuse

1. Indications – rigid thoraco-lumbar curve in young pt to avoid **CRANKSHAFT PHENOMENON**
   (posterior fusion →↑ lordosis)
2. Advantages - Less levels instrumented & Better rotational correction
3. Instrumentation - **Dwyer** system → now **Zielke** system

Early complications

1. Neurological injury during surgery 1% (use intraoperative SEP)
2. Blood loss → Hypotensive anaesthesia, Autotransfusion
3. Wound infection - Prophylactic antibiotics indicated
4. Pneumothorax in anterior approach
5. Dural tear - During ligamentum flavum removal or hook or wire insertion
6. Inadvertent 'flat-back' alignment
7. Incorrect fusion levels
8. Inappropriate ADH secretion

Late complications

1. Pseudarthrosis ~ 1-5% with fusion to sacrum. Solid fusion should occur by 6 months
2. Rod or wire breakage - Due to pseudarthrosis or fatigue failure.
3. Back pain - Appears to be due to Fusion below L4 , Loss of lumbar lordosis

**Juvenile Idiopathic Scoliosis**
- Worse prognosis than the adolescent
- Usually progress
  - ttt: brace till 10y then surgery

**Infantile Idiopathic Scoliosis**
- ♂ left thoracic curve
- 85% regressive
- 15% progressive (worst prognosis): RVDA > 20° & cardiopulmonary complication
  - ttt: serial EDF POP (elongation derotation flexion) if progressive (15%) Anterior Zielke
**Etiology:**

1- Postural:
   - 2ry to flat foot or 2ry to exaggerated lumbar lordosis
   - Disappear é back stretch + shoulder retraction

2- Scheuermann’s disease

3- Congenital
   - Defect of segmentation
   - Defect of formation
   - Mixed é rotary instability

4- Skeletal dysplasias
   - Achondroplasia
   - Mucopolysaccharidoses
   - Other

5- Neuromuscular

6- Myelomeningocele

7- Posttraumatic
   - Acute
   - Congenital

8- Postsurgical
   - Postlaminectomy
   - Following excision of vertebral body

9- Postirradiation

10- Inflammatory

11- Collagen disease: e.g Ankylosing spondylitis

12- Tumor

13- Metabolic
   - Osteoporosis Dowager’s Hump
   - Osteogenesis imperfecta
   - Other

**Normal regional alignment:**

1- Cervical ....................-30°

2- Thoracic ....................45°

3- Lumbar ....................-60°

**SVA (sagittal vertical axis)**

1- Axis from centre of C7 body vertically downward → posterior to L5 disc

2- Most children stand in slight –ve balance
Congenital Kyphosis

- Deformity is characterized by severe angular deformity with a prominent gibbus at the apex of the curve;

**Etiology**

- **Defect of Formation (Type 1):** - failure of formation of the anterior elements:
  - The worst prognosis
  - **Sequelae:** - paraplegia commonly results if untreated; & compression of viscera; - impairment of pulmonary function; - poor sitting posture;

- **Defect of Segmentation (Type 2):**
  - Slightly better prognosis; - produces a more rounded kyphotic shape
  - Deformity progresses more slowly and paraplegia is uncommon;

**Treatment**

1. **Posterior Fusion:** - in children <5 yrs with curves <55 deg, only an situ posterior arthrodesis is required since some spontaneous correction of kyphosis will occur with continued growth; - posterior fusion may have wider indications with kyphosis due to failure of segmentation (as opposed to failure of formation); - posterior kyphectomy & arthrodesis involve meticulous care of tissues; - resection of the non-functioning cord at apex of the deformity; - water-tight dural closure, with care being taken not to occlude terminal end of normal spinal cord at site of transection of cord; - bivalve total-contact orthosis is used to support trunk until fusion is solid;

2. **Anterior & Posterior Fusion:** - combined anterior & posterior fusion is indicated for children >5 years, curves >55 deg, and neurological deficits;

---

### Defects of Vertebral Body Segmentation

<table>
<thead>
<tr>
<th>Defects of Vertebral Body Segmentation</th>
<th>Defects of Vertebral Body Formation</th>
<th>Mixed Anomalies</th>
</tr>
</thead>
<tbody>
<tr>
<td>Partial</td>
<td>Anterior and Unilateral Aplasia</td>
<td></td>
</tr>
<tr>
<td>Anterior Unsegmented Bar</td>
<td>Anterior and Median Aplasia</td>
<td></td>
</tr>
<tr>
<td>Postrolateral Quadrant Vertebra</td>
<td>Butterfly Vertebra</td>
<td></td>
</tr>
<tr>
<td>Complete</td>
<td>Anterior Aplasia</td>
<td>Anterolateral Bar and Contralateral Quadrant Vertebra</td>
</tr>
<tr>
<td>Block Vertebra</td>
<td>Anterior Hypoplasia</td>
<td></td>
</tr>
<tr>
<td>Posterior Hemivertebra</td>
<td>Wedged Vertebra</td>
<td></td>
</tr>
</tbody>
</table>
Sheuermann's Disease

**Definition** = Spontaneous Wedging of 3 adjacent vertebrae of at least 5 degrees.

**Aetiology:**
- Unknown; although
- **Strong HEREDITARY** tendency (may be AD)
- **OSTEOCHONDRODITIS** of the end plate Apophysis is claimed as an aetiology
- Primary **COLLAGEN DISORDER** theory
- **ABNORMAL ENDOCHONDAL OSSIFICATION**
- Growth deficiency δ mechanical stresses on the anterior column

**Epidemiology:**
- Most common cause of structural kyphosis of thoracic & thoracolumbar spine
- Skeletally immature ♂

**Pathology:**
- Most commonly affect the thoracic spine
- Weakness of upper and lower end plates occur → with activity IVD are compressed against the weak end plates → vertebral collapse → wedging with anterior marginal detachments
- 3 Forms:
  1. Thoracic type ..................... apex T7-9 (commonest)
  2. Thoracolumbar type ........ apex T10-L1
  3. Athletic lumbar hypolordosis

**Associated pathologies:**
- Spondylolysis: ♠ lordosis strains L5 pars interarticularis
- Scoliosis: ......................... 25%

**Clinical Findings:**
- Teenage male +ve family history
- Deformity:
  1. Progressive till the maturity
  2. Rigid: doesn't reverse with hyperextension
  3. Rounded thoracic kyphosis
  4. Compensatory lumbar lordosis is common & Scoliosis may occur
  5. Shoulders are prominent
- Backaches:
  - lower lumbar ms sprain (é progressive compensatory lordosis)
  - Facetal dysfunction 2ry to chronic lordosis
  - Scheuermann’s disease of the lumbar spine itself is painful
  - May occur over the apex of kyphosis
- Neurologic manifestations rarely δ Scheuermann’s but 2ry to:
  - UMN paresis ....................... by NPH or compression 
  - SLR δ spastic hamstring (+ve Tripod, popliteal angle)
- Thoracic Scheuerman's is not usually painful, lumbar Scheurman’s is often symptomatic

**PXR: SORENSEN CRITERIA**

- **Thoracic kyphosis** ........................... >40º (25º-40º being normal)
- **Thoracolumbar kyphosis** .............. >30 deg (thoracolumbar spine is normally straight)
- **Wedgeing** .................................... >5º of 3 adjacent vertebrae (clusitng & striations are normal)
- Irregular end plates ....................... (normally in children are straight)
- Loss of disc space height
- **Schmorl’s nodes** are constant finding that reflect high pressures at the disc end plate interface
- Angel is measured in the Lateral view (≠ scoliosis)
DD:
- Postural kyphosis: painless, correctable, no PXR changes
- Discitic & OM: severe pain, PXR show erosions, soft tissue mass
- SED: affection of the whole body joints

Non Operative Treatment:
- <60º adolescent the ttt is follow up
- 60-75º bracing (CTLSO) if correctable or hyperextension cast if fixed, for 1-2 y
- pain usually subsides at end of growth unless deformity is severe;

Operative Treatment:
Indications:
1. Thoracic kyphosis .................... >75º
2. Rigid kyphosis .......................... > 55º
3. persistent back pain that is unresponsive to non operative treatment;

Technique
- Anterior release (leave the PLL intact) + interbody fusion followed by PSF é compression instrumentation (in the same setting)
- Cortel-Dubousett is the system of choice

Post-Laminectomy Kyphosis
- Occur when multiply level laminectomy is performed in children

Predisposing factors:
- Young age
- Cervical and upper thoracic laminectomy

Mechanism:
- Lack of posterior support
- Abnormal anterior compressive forces
- Hypermobility of the segment

Prevention:
- Avoid facetectomy
- Surgical fusion by BG or instrumental would prevent kyphosis
- Postoperative bracing
- Laminaplasty: is a technique could be used in children to avoid laminectomy, in ω the lamina is removed in toto then replaced and fixed inplace

Active ttt:
- Cervical.............................. Anterior interbody fusion
- Thoracic................................. Anterior release & fusion  → posterior fusion
- Postoperative bracing
CERVICAL SPINE DISORDERS

Corticollis

**Definition:**
- It is the head (up) tilt and rotation to one side at or shortly after birth

**Congenital**
1. Congenital Muscular Torticollis (see below)
2. Odontoid hypoplasia
3. Klippel-Feil syndrome

**Acquired:**
1. **Osseus**
   - Trauma - Atlantoaxial rotary instability
   - Infections
     - tuberculosis
     - pyogenic infections
     - Grisel's syndrome - follows upper respiratory tract infection
   - Tumours
     - Osteoid osteoma
   - Inflammatory
     - ankylosing spondylitis
     - Rheumatoid arthritis
2. **Non-osseus**
   - Neck burn contractures
   - traumatic: Prolapsed disc
   - Infections: retropharyngeal abscess
   - Tumours: intraspinal or intracranial - posterior fossa e.g medulloblastomas
   - ocular - with a superior oblique muscle paresis → compensatory head tilt
1- Congenital Muscular Torticollis

**Pathology:**
- the sternomastoid on one side is fibrous & fails to elongate → progressive deformity
- Head tilt towards the ms affected & chin rotate to the other side

**Aetiology:** unknown
1. may be due to **ISCHAEMIA** of the muscle from a distorted position in-utero
2. Local **COMPRESS**ion on the soft tissue of the neck → fibrosis of the sternomastoid
   - associated with:
     - Breech
     - DDH
     - Difficult labour

**Clinical**
1. lump may be noticed in first few weeks of life
2- **TORTICOLLIS & ↑ ROM**
3- sternomastoid feels **TIGHT** & hard
4- **ASYMMETRICAL FACIAL DEVELOPMENT**
5- DDH may be associated

**PXR**
- To exclude other pathology but here it is usually negative
- Hip PXR & US → DDH

**Treatment**
1- Start by **STRETCHING** exercises & physiotherapy
2- After 1 year:
   - **Z PLASTY** of the sternal end ± division at upper end as well
   - immobilise with a collar
   - then stretching exercises

2- Odontoid Hypoplasia

**Pathology:**
- Types:
  1- Aplasia
  2- Hypoplasia
  3- Os Odontoidium: unfused ossicle odontoid tip may confuse é # but sclerotic border
  4- Ossiculum terminale (unfused odontoid tip eventually may separate & fuse é C1

- Complication: predisposes to atlantoaxial instability

**Clinically:**
- discovered **INCIDENTALLY** usually
- **NEUROLOGICAL** deficits as cervical disc
- **TORTICOLLIS**

**Associated with:**
- Morquio's Disease –
- Down's Syndrome
- Klippel Feil $
- SED

**Treatment**
- **C1-C2 FUSION** advised if marked discomfort or neurological symptoms
I. Short neck  
II. Low posterior hair line  
III. Neck ROM (fusion of at least two cervical segments)

Etiology is unknown.

- It is a failure of the normal segmentation of cervical during the 3rd & 6th wks of gestation.

Pathology:

- Vertebrae are fused and may encroach on the canal or root → neurological symptoms

Classification:
1. Type I: C2-C3 fusion with occipitalization of the atlas
2. Type II: Long fusion below C2
3. Type III: Single open space between two fused segments

Associated Anomalies:
1. Springle deformity,  
2. Cervical rib & disrafism  
3. Diastematomyelia & syringomyelia  
4. Arnold Chiari I malformation  
5. Cleft palate, Syndactyl, supernumerary digits  
6. VSD, renal, respiratory

Clinical Findings:

Triad of ...  
1. Torticollis: Flexion and extension are better than bending and rotation.  
2. Neck Webbing [Prominent trapezius]  
3. Low Anterior Hair Line

Plus ...  
1. Compensatory Hypermobile at the unfused segments → instability and pain  
2. Neurologic: Root irritation & cord compression may reach paraplegia & death  
3. Facial Asymmetry.  
4. Facial N Palsy  
5. Abducant Palsy (lateral rectus palsy) ± Ptosis of the eye

Radiographic Findings:

1. Flat fused vertebrae  
2. Hemivertebrae or block vertebrae  
3. Intersegment instability  
4. Wasp-Waist Sign: Indentation at the site of open space between the fused vertebrae.

MRI access cord compression along with cord anomalies.

Treatment:

- Minimally involved patients lead normal lives with only minor restrictions.  
- Avoid contact sports that place neck at risk.
1. Cervical Collar, NSAIDS, ± traction  
2. Posterior Fusion of the irritation segment of root or cord ± Decompression if stenosis  
3. Dislocations and basilar invagination → traction → posterior fusion.
4. Basilar Impression

- It is the malposition of the odontoid being more cephalad than normal

**Pathology**

- When occur it compresses:
  1. Cord
  2. Vertebral a
  3. CSF flow
  4. Cranial Nerve

- It may confuse with:
  1. Posterior fossa tumor
  2. Polio bulbar palsy

**Etiology:**

- Associated é:
  1. Klippel Feil
  2. Arnold Chiari $\$
  3. Odontoid malformation
  4. Bifid posterior atlas arch

- May be 2ry to:
  1. OI III, IV
  2. RA, Ank Sp, Paget’s
  3. NF
  4. Osteomalacia, Rickets

**Clinically:**

1. Pure UMNL
2. In Arnold Chiari $\$: Dizziness, ataxia, nystagmus
3. Cranial N. affection (CN that come out form the f.magnum); 5, 9, 10, 11
4. Vertebral artery: dizziness & syncopal attacks

**PXR**

White Criteria of basilar invagination on PXR cervical lateral view:

1. Padi (Posterior Atlanto-Dental Interval) .........................<13mm = BI
2. AADI (Anterior Atlanto-Dental Interval) .......................>4mm = BI
3. DBI (Dens-Basion Interval) ..........................<4mm = BI
4. Wackenheim’s clivus tangent line ..................should tangent the dens not cut it
5. Chamberlain’s line from f.magnum to hard palate ................<3mm cut to dens
6. McGregor’s line from occiput to hard palate ...............<5mm cutting to dens
7. McRae’s line bet basion & foramen magnum .............dens should never pass it
8. Ranawat’s from C2 pedicle to transverse plane of C1 .... 15-17mm
9. Redlund-Johnell from axis base to McGregor’s line ......29-34mm
10. Fischgold & Metzger digastic line in AP at lower edge of mastoid → should be tangent to dens
11. Powers Ratio: .........................................................>1 anterior translarion / < 0.55 post

**Treatment:**

1. Splintage ......hard collar ......If Cranial Settling ......Traction ......If fail ......surgery
2. Cervical fusion: C0-C2 by Wires, Luque, BG, Plates, Screws
5- **Occipitalization of the Atlas**

- refers to failure of segmentation between occipital and spinal sclerotome that ranges between synostosis to syndesmosis

**Pathology:**
- Axis and occiput are fused together → ♦ mobility at the atlanto-axial joint
- Normally movement at C0-1 is flexion extension & at C1-2 is rotation mainly
- When occipitalization occur → C1-2 show ♦ abnormal motion → degeneration and instability and pain
- Basilar invagination may occur
- Associated condition:
  1- C2-3 fusion
  2- Kyphosis & scoliosis
  3- C1-2 instability
  4- Cervical rib, cleft palate,

**Clinically:**
- As the abnormal motion segment starts to develop OA & basilar impression
- **NEUROLOGICAL** symptom δ basilar impression, or irritation of the cord
  1- Anteriorly: → pyramidal tract
  2- Posteriorly: → lemniscus system
  3- Basilar: → nystagmus, vertigo, cranial nerve
  
  **TORTICOLLIS**

**Radiological:**
- **PA/DI** <13mm
- **MCRAE** line
- **CT** is the best as it shows the coalition segment at C0-1

**Treatment:**
1- Minor symptoms: → conservative
2- Major symptoms:
   o Anterior: traction then fusion
   o Posterior: → MRI and according to the tethering structure
     ▪ Suboccipital craniotomy ± fusion
     ▪ Excision of atlas arch ± fusion
6- Atlanto-Axial Rotary Instability

- refers to loss of ligamentous stability between atlas and axis, occurs most often in older children and adolescents

**Pathology:** unknown

- The basic pathology is dysfunction of transverse ligament or the one of the alar ligaments either due to trauma or spontaneous
- This leads to instability at C₁₋₂ → Rotation + lateral tilt of the neck → compression of the cord + vertebro-basilar insufficiency
- approx 50 % of cervical rotation takes place between atlas and axis, slightly anterior dens → lateral wall of atlas when rotates it encroach the canal → physiologically ↓ canal diameter
- Spinal canal of the atlas is large compared with that of other segments,

**Steele’s Rule of Thirds:** - canal of atlas is about 3 cm in its AP diameter; - spinal cord, odontoid process, and free space for cord are each about 1 cm in diameter

**Classification:**

1- AARI
2- AARI with anterior shift of C1 < 5mm
3- AARI with anterior shift of C1 > 5mm
4- AARI with posterior shift

**Associated Conditions**

1- Grisel's &: - AARI after torticollis ð pharyngeal infection & hyperemia → demineralization of attachment of transverse lig to ant arch of atlas, ë subsequent rotary subluxation of atlas on axis or anterior atlantoaxial subluxation
2- Morquio syndrome
3- Down syndrome (25% of patients)
4- Klippel Feil
5- SED, Achondroplasia, Larsen's syndrome
6- Rheumatiod Arthritis (adults)

**Clinically:**

1- Torticollis “Cock Robin” poition:
   o Head tilt to one side and rotate to the other
   o Sternomastoid of the long side is spastic
   o Neck is slightly flexed
2- Occipital neuralgia
3- Occasionally vertebrobasilar artery insufficiency

**Radiographs:**  
* Lateral View in flexion
  - ADI < 3.5mm........... transverse ligament is intact
  - ADI 3-5mm ............. transverse ligament is insufficient = type II injury
  - ADI > 5mm............. failure of the alar ligaments = type III rotatory subluxation
* AP open mouth view
  - Asymmetrical distance between both facets and the dens
  - Cine radiography is a dynamic roentographic imaging technique

**Treatment:**

1- <1wk .................... soft collar, NSAIDs
2- >1wk ..................... Halter
3- If failed reduction C₁₋₂ fusion
Diastematomyelia

- Fibrous, cartilagenous or bony bar creating a longitudinal cleft in the spinal cord.
- usually in lumbar spine
- can cause tethering of the cord with neurological deficits
- X-rays: widened interpedicular distance
- MRI makes the diagnosis

**Treatment:**
No spinal deformity or neurology .......... observe
Spinal deformity ± neurology ..................... resect bar before correcting deformity.

Sacral Agenesis

- = partial or complete absence of the sacrum & lower lumbar spine.
- associated with maternal Diabetes
- accompanied with GI, GU & cardiac abnormalities

**Clinically:**
- prominent lower lumbar spine & atrophic legs; sit in ‘Buddha’ position
- motor deficit below level of agenesis; sensory spared

**Treatment:**
- amputation or spinal-pelvic fusion.

Arnold Chiari Malformation

**Description:**
- Protrusion of the inferior poles of cerebellum + medulla oblongata through foramen magnum into the spinal canal, without displacing the lower brain stem.

**Aetiology:**
- Deformity Of Occipital Bone & Upper Cervical Spine.
- ± stenosis of the aqueduct of Sylvius → hydrocephalus → atrophy of the brain tissue.

**Clinical**
1. Hydrocephalus ± Mental dullness
2. Spina bifida and meningomyelocele
3. Headache, vomiting
4. Visual disturbances, diplopia
5. Paralysis of extremities
6. Cerebellar ataxia

**Chiari’s classification:**
- Type I: .............................................. herniation of cerebellum-tonsils
- Type ii: ............................................. herniation of vermis and pons + dilated 4th ventricle.
- Type iii: .......................................... high cervical hernia containing cerebellum-tissue.
- Type iv: ........................................... hypoplasia of cerebellum ± encephalocele.
Spine Tumors

Benign Tumors

Incidence
- They are the 2nd most common cause of pain
- Night pain is exclusively suggestive of osteoid osteoma (OsOs) & osteoblastoma (OB)
- Usually ......................... 3rd decade
- Sacral tumors occur in adults; if occur in child it is mostly malignant

Pathology
- Types:
  1- Most common ....................... GCT
  2- OsOs ........................................ 50%
  3- OB
  4- Hemangioma
  5- Osteochondroma
  6- ABC
  7- EG
- Sites:
  o GCT, Hemangioma, Osteochondroma, ABC, EG in the body
  o OsOs & OB ................................. in the arch
- Classification
  o Latent ...................................... Osteochondroma & Hemangioma  → no ttt
  o Active ..................................... OsOs, ABC, EG
  o Aggressive .............................. GCT, OB

Diagnosis
Symptoms
1- Pain:
   o  night
   o Radicular pain may occur .......... OsOs & OB
   o Dull aching pain ..................... GCT, ABC, H, EG
2- Painful scoliosis
3- Myelopathic symptoms ..................... more in thoracic and cervical vertebral tumors

Signs
1- Myelopathic signs: as in cervical ...
2- Decompensate curve usually
3-  ROM

PXR
- No rotation (≠ idiopathic)
- No wedging (≠ idiopathic)
- OsOs & OB ............................... sclerotic pedicle & usually found in the concave side. And usually interpreted as –ve PXR
- GCT & ABC ............................ Lytic lesion
- Hemangioma ............................. vertical striation
- EG ........................................................ Vertebra plana

Tc
- Of choice in painful scoliosis ............ mostly OsOs, OB; Tc scan is very sensitive to them
- False –ve in ................................ hemangioma

CT
- To detect the bony extent
- CT myelogram may be very beneficial in some cases of cord compression
MRI
- To detect soft tissue shadow and involvement
- Sometimes surrounding edema may be marked to over estimate the tumor size

**Treatment**

1. Marginal excision for latent, active, or cases è neurological compression
2. Wide excision for Grade III aggressive tr:
   - Avoid complete laminectomy
   - Partial laminectomy
   - >50% of the facet to be removed
   - If complete laminectomy posterolateral fusion & BG is performed
   - Complete facetectomy posterolateral fusion & BG
   - Total segmental resection posterolateral fusion & BG + Anterior structural BG
3. Adjuvant Cryotherapy is preferred than, PMMA, or radiation
4. Stabilization + fusion is necessary esp. after complete laminectomy in Cx&Thx
5. Orthosis postop. Is mandatory as well
6. Reconstruction:
   - Autograft Iliac & fibula
   - Allograft
   - Composite Hollow titanium cages + BG

**Precautions**

1. Approach in thorax should be anterior approach not via limited costotransversectomy
2. Sacrum is very vascular and embedded in a dense venous plexus → profuse bleeding
3. Sacrum is in close contact to bladder and rectal innervation
4. If dura is adherent to a tumor excise the stuck part and then graft it

**Complications:**

1. Kyphosis especially after laminectomy in cervical and thoracic
2. Wound problems:
   - Infection: leave a drain, meticulous closure
   - Wound dehiscence

**Radiation:**

- Role of radiation is mainly in ABC & hemangioma
- ABC 30Gy
- Hemangioma 40Gy
- GCT may be sensitive but its use must be limited to:
  - Complicated cases
  - Unresectable
- Complications:
  - Scoliosis 30%
  - Late sarcomatous change after several years
Malignant Tumors

Incidence
- More common in adults
- Metastatic carcinoma is the majority
- Spine metastasis ...........................................50% of all bone metastasis

Pathology
- Site:
  - 75% .................................................. in thoracic
  - 75% .................................................. in body
- Secondaries .............................................. is the majority
  - Breast, Lung
  - Prostate, renal
  - GIT, Lymphoma
  - Thyroid
- Primary sarcoma ........................................... Less common
  - Multiple Myeloma ..................................... is the commonest primary
  - Chordoma .............................................. in the sacrum
  - Osteosarcoma .......................................... less common

Diagnosis
  Clinically
  1- Pain:
    - Relentless pain
    - Awakening pain
    - Night pain
    - Radicular pain may occur
    - Dull aching pain
  2- Myelopathic manifestation ..................... 20%
  3- Survival ................................................. <1y

PXR
- Unfortunately > 50% of the body must be destroyed before the tr being visible
- Osteoporosis may harden the job more
- Unilateral absent pedicle ......................... Awl eye appearance
- Usually confined to 1 vertebra ............... respect the disc
- Pathological fracture

MRI
- Detect spinal canal compromise
- Detect paraspinal extension
- Less helpful incase of pathological # ... ♦♦ surrounding edema & confusion

CT
- May be more helpful in case of pathological #
- For CT guided biopsy

Staging investigations

Preoperative investigations
Treatment

- Unfortunately surgery is not the line of choice in ttt of spine malignancy
- Contraindications to surgery:
  1. Secondary tumors
  2. Multiple Myeloma
  3. <2mo survival is expected after surgery
  4. Bad general condition
- Other modalities:
  1. Radiotherapy
  2. Chemotherapy
  3. Hormonal therapy
  4. Angiographic embolization for thyroid and renal secondaries (very vascular)
- Indications for surgery:
  1. Pathological #
  2. Neurological compromise é radioreistance
  3. Painful kyphosis
- Rational of surgery:
  1. ttt of complications
  2. regain pt to fair functional status
  3. Remove as much tumor as could be
  4. Restore stability
  5. Improve the quality of remaining life
  6. Laminectomy is abandoned
- Approaches: Anterior approaches are better than the posterior approaches
  1. Transthoracic
  2. Retroperitoneal
  3. Thoracolumbar
  4. Lumbar exposures
  5. Posterior approach:
     • posterolateral extracavitary decompression
     • Transpedicular decompression
     • Total spondylectomy
     • Laminectomy?
  6. Sternal splitting ......................... T1-4
  7. Cervical anterior approach......... for cervical tr
  8. Posterior approach ..................... for sacral tumors; after coccygectomy, develop the retrorectal plane and sacrum is excised
- Technique:
  1. Anterior corpectomy
  2. Total spondylectomy (via anterior fusion + PSF)
  3. Excision to the level of structurally sound vertebra above and below
  4. Excision up to total vertebrectomy + decompression + fusion + instrumentation
  5. Extensive lesion + bad condition \(\rightarrow\) Posterior decompression + BG + fusion
  6. Laminectomy is done only if the tumor originate from the lamina
- Reconstruction:
  1. Steinmann pins (or Harrington) + cement
  2. Wide sheet of gel foam to protect the dura
  3. Split fibula or double fibula ........ if survivor is expected to be > 2y
  4. Combined BG + cement

Postoperative

- Radio & chemotherapy is used but should be delayed 6wk if BG is used
- Postoperative TLSO
Results of surgery

- 70-90% ............................................. effective in pain and neurological control
- Anterior approach results are too much superior to posterior
- Laminectomy give bad results .................. 40% = the results of the radiation therapy alone

DDx of osteochondroses

<table>
<thead>
<tr>
<th>Osteochondrosis</th>
<th>Disease</th>
<th>Site</th>
</tr>
</thead>
<tbody>
<tr>
<td>AVN</td>
<td>Legg–Clave-Perthes' disease</td>
<td>Upper Femoral Epiphysis</td>
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<tr>
<td>Crushing</td>
<td>Kienbock's disease</td>
<td>Lunate</td>
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<td></td>
<td>Preiser's disease</td>
<td>Scaphoid</td>
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<td></td>
<td>Panner's disease</td>
<td>Capitulum</td>
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<td></td>
<td>Scheuermann's disease</td>
<td>Vertebral Bodies</td>
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<tr>
<td></td>
<td>Kohler's disease</td>
<td>Tarsal Navicular</td>
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<tr>
<td></td>
<td>Freiberg's disease</td>
<td>2nd Or 3rd Metatarsal Head</td>
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<tr>
<td></td>
<td>Blount's</td>
<td>Knee ?</td>
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<tr>
<td></td>
<td>Theimann's disease</td>
<td>Multiple Phalanges</td>
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<td></td>
<td>Friedrich's disease</td>
<td>Sternal End Of The Clavicle</td>
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<tr>
<td>OCD</td>
<td>OCD</td>
<td>Knee</td>
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<tr>
<td></td>
<td>OCD</td>
<td>Talus</td>
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<tr>
<td></td>
<td>OCD</td>
<td>Patella</td>
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<tr>
<td></td>
<td>OCD</td>
<td>1st MT Head</td>
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<tr>
<td></td>
<td>Buschke's disease</td>
<td>Medial Cuneiform</td>
</tr>
<tr>
<td>Traction apophysitis</td>
<td>Osgood–Schlatter's disease</td>
<td>Tibial Tuberosity</td>
</tr>
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<td>Johansson-Larsen syndrome</td>
<td>Patella</td>
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<td></td>
<td>severs disease</td>
<td>Calcaneus</td>
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<tr>
<td></td>
<td>Iselin's disease</td>
<td>Tuberosity Of 5th MT</td>
</tr>
<tr>
<td></td>
<td>Mandl's disease</td>
<td>Greater Trochanter</td>
</tr>
</tbody>
</table>
Mini Invasive Spine Surgery

**Advantages:**
1. ↓ surgical complications
2. ↓ surgical blood loss
3. ↓ use of postop narcotic pain medicines
4. ↓ hospital stay
5. ↑ speed of functional return to daily activities
6. Better exposure

**Classification**

I. **Based on spine regions:**
   1. Minimal invasive surgery Cervical
   2. Minimal invasive surgery Thoracic spine
   3. Minimal invasive surgery Lumbar spine

II. **Based on body plains**
   1. Anterior
   2. Posterior
   3. Postrolateral

III. **Based on technique**
   1. Microscopic techniques: e.g. anterior cervical microforaminotomy
   2. Endoscopic techniques

**In Cervical spine:**
1. **Anterior:**
   a. **Endoscopic foraminotomy** for cervical disc herniation
   b. **Percutaneous endoscopic** anterior cervical discectomy
2. **Posterior:**
   a. **Endoscopic foraminotomy**
   b. **Cervical laminaplasty**
3. **Posterolateral** endoscopic approach for tumors

**In thoracic:**
1. Anterior Transthoracic endoscopic surgery
2. Posterior Transpedicular endoscopic approach

**In lumbar:**
1. Endoscopic Surgeries:
   a. Anterior endoscopic lumbar approaches
   b. Posterior **Endoscopic Microforaminotomy** e.g. Lumbar Stenosis
   c. Posterior **Micro Endoscopic Discectomy**
   d. Postero-lateral endoscopic lumbar approaches
2. **Mini invasive lumbar fusion approaches:**
   a. **PLIF**: Posterior Lumbar Interbody Fusion
   b. **ALIF**: Antero-Lumbar Interbody Fusion
   c. **TLIF**: Transforaminal Lumbar Interbody; i.e. postero-lateral + facetal amputation
   d. **XLIF**: Extreme Lat Interbody, i.e. extra-peritoneally via lat approach under fluoroscopy
   e. **Mini ALIF**: reach the disc extra-peritoneally via lat approach under fluoroscopy
2. **Mini invasive aiding devices:**
   a. Laser
   b. Motorized Shaver & Suction
   c. Thermal: disc ablation, also controls pain
   d. Radiofrequency Neucloplasty
3. **Intervention spine radiology:**
   a. Kyphoplasty
   e. Vertebroplasty
Back Pain in a child

**Differential Diagnosis:**

- **Congenital:**
  1. **Klippel-Feil $\ddagger$:** pain is usually due to hypermobility or instability of adjacent vertebral segment or to degenerative osteoarthrosis;
  2. **Diastematomyelia:** frequently associated with a cutaneous malformation overlying defect, is more likely to present with neurological abnormalities involving lower extremities, such as unilateral cavus foot or calf atrophy, rather than with back pain;
  3. **Scheuermann’s Kyphosis** - is the commonest cause of pain in thoracolumbar regions

- **Traumatic:**
  4. Herniated **Disc** in the Child
  5. **Slipped Vertebral Apophysis:**
     - Posterior displacement of the ring apophysis & adjacent disc into vertebral canal
     - $\delta$ incomplete or delayed fusion between vertebral ring apophysis & central cartilage
     - Occur in lumbar region mostly
     - Cause both discogenic and radicular pain
  6. **Spondylolisthesis**

- **Infections:**
  7. **Osteomyelitis** of the Spine
  8. **Tuberculous** Spondylitis
  9. **DISCITIS**

- **Tumors**
  10. Spinal **Cord** Tumors
  11. Primary **Bone** Tumors: EG, OB, OsOs, ABC
  12. Secondary tumors: metastatic neuroblastoma

**Crankshaft Phenomenon**

- in skeletally immature pt, isolated posterior arthrodesis with instrumentation of a lordotic curve may act as a posterior tethering bar, producing lordosis & bending of the fusion mass as the unfused anterior vertebral bodies continue to grow

**Risk Factors:**

1. open triradiate cartilages
2. physiologic youth: - girls younger than 11 years; - boys younger than 13 years
3. Risser grade 0 or 1
4. juvenile scoliosis > congenital scoliosis (abnormal anterior growth plates)

**Radiographs:**

- >10º progression of the Cobb angle or RVAD (assuming that other causes of curve progression such as pseudoarthrosis is not present)

**Prevention:**

- Anterior and posterior arthrodesis for risk group
### Definition
- Sciatica is a symptom commonly used to describe symptoms of pain radiating downward from the buttock over the posterior or lateral side of the lower limb. It is usually assumed to be caused by compression of a nerve roots as they emerge from the spine; LDP, spurs, fibrosis, ...
- Pseudo-sciatica is the same but occurs 2ry non spinal causes

### Aetiology
1. Piriformis S
2. Psoas S
3. Hamstring S
4. Gluteus minimus $\$
5. ITB S
6. Fibromyalgia

### Pathogenesis
- Pseudo-sciatica pain arises from:
  1. Entrapment of: ........ • Sciatic n.
     • Posterior cutaneous n. of thigh
  2. Trigger points in the soft tissue $\alpha$ can be elicited by manual palpation of the musculature associated with the hip if the pain is not to be misdiagnosed.
- Piriformis syndrome: The piriformis muscle is in such a state of constant tension and irritates the sciatic nerve.
- Iliopsoas $\$$ (mischief maker): No other muscle has so many functions & cause so much pain & is so difficult to palpate. Iliacus is active in running & posas is active during the last 60° while sitting
- Fibromyalgia: chronic lumbar & gluteal fatigue $\rightarrow$ sciatic nerve irritation
- ITB $\$$: Runners who run on only one side of a slanted track can irritate ITB $\rightarrow$ ⊕ sciatic n.
- Sciatic entrapment causes pain to be referred to its autonomous cutaneous areas, back of thigh, lateral leg, & foot

### Clinically
- Pain:
  1. Low back radiating into one buttock and down the leg
  2. May be severe enough that may lock back motion
  3. ⊕ by sitting, standing, certain positions
  4. ⊕ by cough, sneeze
- Numbness & ⊘ Reflexes
- **TRIGGER POINTS** = area of extreme tenderness & irritability that when compressed ⊕ nerve along its pathway, or ⊘ of an irritated muscle

### Differentials

<table>
<thead>
<tr>
<th>Muscles</th>
<th>Cause</th>
<th>Pain</th>
<th>Triggers</th>
<th>TTT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Piriformis</td>
<td>Fall on butt</td>
<td>Back thigh</td>
<td>IR (by Morton’s neuroma)</td>
<td>Injection</td>
</tr>
<tr>
<td>Psoas</td>
<td>Psoas tension</td>
<td>Front of thigh</td>
<td>Long upright standing</td>
<td>Physiotherapy</td>
</tr>
<tr>
<td>Hamstring</td>
<td>Pressure on hamstrings</td>
<td>Upper thigh</td>
<td>Up from crossed leg sitting</td>
<td>Injection</td>
</tr>
<tr>
<td>Glut Minimus</td>
<td>Overuse</td>
<td>Back pain to calf</td>
<td>Sleeping on the affected side</td>
<td>Physiotherapy</td>
</tr>
<tr>
<td>ITB</td>
<td>Overuse</td>
<td>Lat knee &amp; back</td>
<td>Long uphill running &amp; cycling</td>
<td>US &amp; Release</td>
</tr>
<tr>
<td>Fibromyalgia</td>
<td>Overuse</td>
<td>Back pain</td>
<td>Long standing &amp; sitting</td>
<td>Rest &amp; injection</td>
</tr>
</tbody>
</table>

- Pain referred from trigger points in gluteus medius less likely to involve the thigh
- Pain referred from trigger points in gluteus maximus ⊗ flexion
- Pain referred from trigger points in piriformis ⊗ IR
**Coccydynia**

**Definition:**
It is a painful coccyx

**Anatomy:**
- Triangular piece of bone that originally formed of Four Pieces that fuse at skeletal maturity
- It may fuse as well with the sacrum
- It has 2 upper **Corinialae** & 2 lateral **Transverse Processes**
- Two intercornual ligmaments are attached to the cornu
- 2 lateral coccygeal ligaments are attached to the transverse processes
- The gluteus maximus and the sacrotuberous ligament are attached to the posterior surface
- **Ganglion Impar** (end of the sympathetic chain) is situated at the ventral surface
- **Ano-Coccygeal Body** situated at the tip where ano-coccygeal lig & coccygius ms attach

**Types:**
- Type I: .................................................. slightly curved anterior
- Type II: .................................................. markedly curved anterior at S5C1
- Type III: .................................................. markedly curved anterior at C1C2
- Type IV: ................................................. Subluxed anterior

**Ætiology:**
1. Congenital types II, III, IV
2. Traumatic:
   - Fracture (uncommon)
   - Sprains
   - Post-traumatic OA
3. Inflammatory:
   - Adventitious Bursitis
4. Tumors:
   - Perianal fistulae
   - Chordoma of the sacrum
   - Glomus tumor: middle scaral A-V fistula
   - Tumors of the cauda
5. Degenerative disc
6. Psychogenic

**Assessment:**
- usually female middle age
- Symptoms:
  - Duration of the symptoms ....................... acute (traumatic) or chronic (tumor or bursa)
  - Related to certain position ..................... (traumatic)
  - Is the pain is continuous .......................... tumors
  - Is there dyschasia, constipation ............... perianal fistulae
  - Note the attitude of the pt ..................... hysterical pt
  - Examination:
    - Tenderness, hotness, warm .................... bursitis & trauma
    - Sinuses & fistulae ............................... perianal fistula
  - PR:
    - Attitude of the coccyx ....................... Abnormal types
    - Mobility ....................................... hypermobile (fractures)
    - Size ........................................... enlarged if there is a tumor
    - Tenderness ................................. if not tender the pt KB hysterical
  - PXR:
    - Abnormal type & size
    - Fractures
    - Chordomal of the sacrum
  - MRI: May be needed to evaluate the presence of a tumor

**Treatment**
- Conservative: NSAID, Donut pillow, hot packs, local inj, mobilization UGA
- Surgical coccygectomy:
  - Prone & through a curved incision at the lateral border of the coccyx
  - Divide the structures attached to the tip first
  - Subperiosteal dissection till it is freed totally + local lidocain
  - Total coccygectomy may endanger the rectum as it lies just in front the S5C1
The general classification of joints:

1. Synarthroses: Fixed or rigid joints.
2. Amphiarthroses: Slightly movable joints.

According to type of tissue that characterizes the junctional area:

1. Fibrous articulations: Bony surfaces are fastened by fibrous tissue.
2. Cartilaginous articulations: Connected by cartilaginous tissue.
3. Synovial articulations: Apposed bony surfaces are separated by an articular cavity that is lined by synovial membrane.

Fibrous joints: (Articulationes Fibrosae)

In most instances fibrous joints consist of predominantly collagenous junctions between bones but in a minority of situations fibro-elastic tissue predominates. Three main groups of fibrous articulation are generally recognized, namely:

1. Sutures: Limited to the skull where bone ends are separated only by connective tissue, and covered by a layer of osteogenic cells (the ‘cambial’ layer), then capsular lamella of fibrous tissue corresponds to and continues with periosteum. Between the two layers of sutural periosteum is a central stratum of loose fibrous connective tissue that contains blood vessels which communicate with diploic vessels, intracranial venous sinuses and external veins in the scalp. When cranial growth ends, osteogenic cells start complete ossification of sutural ligaments, ultimately leading to obliteration and rigid synostosis.

2. Schindylesis: Is a specialized suture where a ridged bone fits into a groove on a neighbouring element, e.g., the cleft between the alae of the vomer that receives the rostrum of the sphenoid.

3. Gomphoses: A peg-and-socket joint (articulatio dentoalveolaris) is a specialized fibrous articulation for fixation of teeth in alveolar sockets in the mandible and maxillae.

4. Syndesmoses: Is a fibrous articulation in which bony surfaces are bound together by an interosseous ligament as in the Posterior Sacroiliac joint & Tibio-fibular joint.
Cartilaginous Joints

Cartilaginous Joints (Articulation Cartilaginaeae), or synarthroses, bone junctions bonded by solid connective tissue, Cartilaginous joints are classified into two groups:

1- **S**ynchondroses *(primary cartilaginous joints)*: These articulations occur where originally separate, but adjacent, centres of ossification appear within a continuous mass of hyaline cartilage. As ossification spreads it invades the actively **GROWING ZONE OF CARTILAGE** occupying the interval between the contiguous osseous surfaces. Functionally, synchondroses are primarily growth mechanisms and, although contributing slightly to the more flexible skeleton of youth, their growth potential is combined with the ability to successfully resist forces, whether of compression, tension, shear or torsion. Later endochondral ossification ceases and entirely replaced by complete bony union between the originally separate osseous surfaces, forming a synostosis, losing its cartilaginous growth potential and mechanical properties, but acquiring the maximal rigidity of bone.

2- **S**ymphyses *(secondary cartilaginous joints)*: is another variety of cartilaginous synarthrosis having many features in common with other arthroses, Topographically, all symphyses are median and, with one exception, are confined to the axial skeleton; as manubriosternalis, **INTERVERTEBRALIS**, symphysis menti, and **SYMPHYSES PUBIS**. Symphysis consists of two surface areas of articulating endochondral bones; the osseous surfaces varying from a few millimetres to over a centimetre apart being bound together by strong, tightly adherent, solid connective tissues. Each bony surface is firmly attached to a thin lamina of hyaline cartilage, which in turn blends with the surface of a thick, strong, but deformable pad (or disc) of fibrocartilage. Collagenous ligaments extend from the periostea across the symphysis and blend with the hyaline and fibrocartilaginous perichondria. Usually unlike synchondroses they are permenant.
Synovial Joints

1. **Plane Joints**
   These are appositions of almost flat surfaces (e.g. Intermetatarsal, Intercarpal, Facetal, Anterior Sacroiliac, Patello-Femoral), movements being considered pure translations or sliding between bones.

2. **Ginglymi (Hinge Joints)**
   These resemble hinges and are shaped to restrict movement to one plane, i.e. they are uniaxial. They have strong collateral ligaments; Interphalangeal and HumeroUlnar joints

3. **Trochoid (Pivot) Joints**
   Also uniaxial, they have an osseous pivot in an osteoligamentous ring, allowing rotation only around the pivot’s axis, as the Head Of The Radius Rotates Within The Annular Ligament, and the atlas (with its transverse ligament) rotates around the dens of the axis.

4. **Bicondylar Joints**
   Largely uniaxial, with a main movement in one plane, they also have limited rotation about a second axis. The rotation is of two varieties:
   - Conjunct, with the main movement.
   - Adjunct which can occur independently and may or may not accompany the principal movement. They have two convex condyles (knuckles) articulating with concave surfaces. Condyles may be almost parallel (e.g. Knee) with a common fibrous capsule

5. **Ellipsoid Joints**
   These are biaxial, with an oval, convex surface apposed to an elliptical concavity, as in Radiocarpal and Metacarpophalangeal joints. Primary movements are about two orthogonal axes (e.g. flexion–extension, abduction–adduction), which may be combined as circumduction; rotation around the third axis is largely prevented by shape.

6. **Sellar (Saddle) Joints**
   Also biaxial, these have concavoconvex surfaces; each is most convex in a particular direction but at right angles to this they are maximally concave. The convexity of the larger is apposed to the concavity of the smaller surface and vice versa. Primary movements occur in two orthogonal planes but articular shape causes axial rotation of the moving bone. Such conjunct rotation, as mentioned above, is never independent and is not simply a by-product of ‘imperfect’ mechanics but is functionally significant in habitual positioning and limitation of movement. The most familiar sellar joint is the carpometacarpal joint of the thumb; others include the Ankle and Calcaneocuboid joints.

7. **Spheroidal Joints (‘Ball-and-socket’)**
   Formed by reception of a globoid ‘head’ into an opposing cup, e.g. Hip & Shoulder joints, they are multiaxial, with three degrees of freedom. Their surfaces, although resembling parts of spheres, are not strictly spherical but slightly ovoid. (Articulatio ovoidalis is an accepted alternative.) Consequently, in most positions congruence is not perfect, occurring only in one position, at the end of the commonest movement (see below).
**Incidences** = Rate of occurrence of a New disease in a population previously free of the disease.

- = [No. of new cases in study period]/[No. at risk at the beginning of study period]

**Prevalence** = Frequency at any given time

- = [No. of patients with the disease]/[No. of patients with the disease + those at risk]

**Sensitivity**

- Ability to exclude false negatives
- = True positive / all with condition (all positives)

**Specificity**

- Ability to exclude false positives
- = True negatives / all without condition (All negatives)

**Positive Predictive Value**

- Probability that a subject who tests positive is truly positive

**Negative Predictive Value**

- Probability that a subject who tests negative is truly negative.

**Accuracy**

- how often is a test correct?

<table>
<thead>
<tr>
<th></th>
<th>Disease Positive</th>
<th>Disease Negative</th>
<th>Totals</th>
</tr>
</thead>
<tbody>
<tr>
<td>Test Positive</td>
<td>a (TP)</td>
<td>b (FP)</td>
<td>a + b</td>
</tr>
<tr>
<td>Test Negative</td>
<td>c (FN)</td>
<td>d (TN)</td>
<td>c + d</td>
</tr>
<tr>
<td>Totals</td>
<td>a + c</td>
<td>b + d</td>
<td>a + b + c + d</td>
</tr>
</tbody>
</table>

**Sensitivity** = \( \frac{a}{a + c} \)

**Specificity** = \( \frac{d}{b + d} \)

**Positive Predictive Value (PPV)** = \( \frac{a}{a + b} \)

**Negative Predictive Value (NPV)** = \( \frac{d}{c + d} \)

**Accuracy** = \( \frac{a + d}{a + b + c + d} \)