Spine Module Notes

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Clinical Pearls

- Test for arterial compression: when the patient holds a deep breath, the radial pulse may disappear in thoracic outlet syndrome, when the arm is abducted and externally rotated (Wright) or extended and turned to the affected side (Adson)
- On the lateral C-spine film, the retropharyngeal space (between the cervical spine and pharynx) increases markedly at the level of C3 on forced expiration (e.g. when crying)

Deformities in the Neck In Children

- Congenital torticollis:
  - Failure of a fibrous sternocleidomastoid muscle to elongate with growth
  - Ischaemia secondary to birth injuries (breech) or distortion in utero
  - Neck lump in first few weeks afterbirth
  - Deformity apparent usually at 1-2 years, with fixed lateral flexion
  - Asymmetrical development of the face = plagiocephaly
  - Need to exclude discitis or lymphadenitis
  - Non-operative – daily muscle stretching (trial for 1 year)
  - Surgical – division of SCM with temporary rigid orthosis to maintain position, followed by strengthening exercises.

- Secondary torticollis
  - Causes: bone anomalies, infection, trauma, juvenile RA, posterior fossa tumours, intra-spinal tumours, dystonias or ocular dysfunction
  - Atlanto-axial rotatory displacement:
    - Inflammation of ligaments and atlanto-axial joint
    - ? recent LRTI
  - Usually treat with soft collar and analgesia ± Halter traction

Scheuermann’s Kyphosis

- Most common cause of thoracic (>45°) or thoracolumbar (>30°) hyperkyphosis in adolescence
  - 4-8% incidence and no gender bias

- Present in late childhood and adolescence with increasing kyphosis
  - Often with compensatory lumbar or cervical “goose-neck” hyper-lordosis

- Symptoms may be minimal and simply a cosmetic issue, or...
  - Dull non-radiating back pain ± local tenderness

- Investigate with supine and hyper-extended x-rays over a bolster to assess mobility. MRI prior to any surgery

- Natural history untreated of curves < 72°:
  - No difference in in types of employment
No difference in pain medications / incidence of back pain
No difference in sick leave
Curves >85° had lower inspiratory capacity, without many major life interference.
Disc degeneration tends to occur at the apex of the curve
Neurological deficits can correlate with shorter-segmented curves

Treatment options:
Physiotherapy – 16-32% reduction in pain
Brace therapy:
  useful if flexible deformity <65°
  early instigation of brace, with an initial correction of >15°
  worn for 16-23 hours a day for 18 months, followed by 18 months of gradual weaning.
  Return of deformity in as much as 30%
Indications for surgery:
  Severe deformities >72°
  Failed conservative management for 6 months
Harrington rods:
  In addition to pre-operative traction and 6-9 months post-operative casting.
  High complication rate: rod breakage, loss of correction, wound infection, junctional kyphosis at adjacent vertebral joints.
  Fusion should extend for T2 to 1st lordotic segment.
  Concurrent anterior release did not improve symptoms or function, but more risks.

Congenital Vertebral Anomalies

Craniovertebral Junction

Occipitalisation of Atlas
  failure of segmentation of the pro-atlas, with fusion to the occiput.
  usually only anterior arch affected (hypochondral bow)
  can compress dura directly, or put increased stress on atlanto-axial joint with delayed instability
  symptoms relate to anterior compression of medulla by an abnormal odontoid, with long tract spasticity.

Basilar coarctation
  congenital invagination of the odontoid into the foramen magnum
  associated with Arnold-Chiari malformations (caudal prolapse of cerebellar tonsils)
  vertebral artery compromise results in ischaemia as a cause of associated syringomyelia (=cyst or syrinx within spinal cord)
  can be asymptomatic either until adolescence or adulthood, or a 2nd hit of trauma.
  symptoms may relate to compression of brainstem, cerebellar tonsils or lower cranial nerves
alternatively vertebral ischaemia can lead to lateral medullary syndrome (posterior inferior cerebellar artery syndrome / Wallenberg syndrome)  
- loss of pain and temperature sensation on the contralateral side of the body and ipsilateral side of the face  
- ataxia  
- dysarthria ± dysphagia  
- treatment centres on decompression ± stabilisation of “tight” foramen magnum

**Basilar Impression**
- **acquired** invagination of the odontoid into the foramen magnum  
  - paget’s disease  
  - rickets & osteomalacia  
  - osteogenesis imperfecta  
  - achondroplasia & hypochondroplasia  
  - neurofibromatosis  
  - rheumatologic disorders; ankylosing spondylitis

Floor of the skull indented by the upper cervical spine ± odontoid impingement on the brainstem.

Often asymptomatic until age 20-30 – present with raised ICP because of blockage of the Aqueduct of Sylvius, or neurological symptoms from cord ischaemia or pressure

Requires surgical stabilisation and decompression.

**Platysbasia**
- flattening of the base of the skull (angle > 143 degrees)  
- symptoms from mechanical occipital pain to medullary compression (dysphagia and loss of cough reflex)  
- treatment reserved for neural compromise – decompression with cervico-occipital stabilisation

**Atlanto-axial Instability**

**Ossiculum terminale**
- failure of fusion of secondary centre at tip of odontoid process  
- usually appears age 2 (delayed appearance) and fuses at puberty

**Os odontoideum**
- failure of fusion of primary apical centre to the axis (normally at age 6)  
- persistent synchondrosis between odontoid base and body of axis  
  - or a nonunion of an unrecognised peg fracture

**Odontoid hypoplasia**
- AVN following prolonged halopelvic traction, or osteomyelitis  
- vertigo or syncope from vertebral artery constriction  
- neck pain to progressive tetra-paresis  
- controversial prophylactic stabilisation or restrict sporting activities as vulnerable to accidental trauma  
- if non-reducible with head positioning or gentle traction under image guidance, may require trans-oral decompression
**Congenital Cruciate ligament laxity**
- instability demonstrated in the absence of any bony anomalies (congenital or traumatic)
- or systemic disease (e.g. rheumatoid disease or infection)
- 1 in 5 *Down's syndrome* children have ligament attenuation or even absence
  - must be screened if undergoing a GA
  - avoid horse riding, cycling and gymnastics, or screen prior to starting

**Morquio’s syndrome**
- autosomal recessive disease of metabolism in which the body fails to break down long chains of sugar molecules called glycosaminoglycans
- the syndrome belongs to a group of diseases called mucopolysaccharidoses – specifically known as MPS IV (A or B)
- symptoms:
  - Abnormal development of bones, including the spine
  - Bell-shaped chest with ribs flared out at the bottom
  - Coarse facial features
  - Hypermobile joints
  - Knock-knees
  - Large head (macrocephaly)
  - Short stature with a particularly short trunk
  - Aortic regurgitation & heart failure
- specific spinal dysplasia = odontoid anomalies leading to instability and neurologic injury
- parents advised to be vigilant for early symptoms – fatigue or tingling extremities
- fusion deferred as long as possible, as even by age 8, the surgical field is only 2 cm²

**Lower cervical spine**

**Klippel-Feil Syndrome**
- Failure of segmentation of cervical somites – associated with GU / CVS / CNS anomalies
  - Type 1 = original syndrome of cervical synostosis with triad of: short neck, stiffness and low posterior hairline
  - Type 2 = synostosis of two vertebrae (or more) without clinical features of type I
  - Type 3 = extensive spinal synostosis not limited to neck
- Associated conditions
  - 30% Sprengel deformity of scapula (small, elevated and connected to cervical spine by a bony bar)
  - 30% rib anomalies
  - 60% scoliosis
  - webbing of the neck – may be a part of Turner’s syndrome with gonadal hypoplasia
  - 30% genitourinary anomalies
  - congenital heart defects
Symptoms:
- smaller and rounder intervertebral foramina can result in nerve root compression
- myelopathy from stenosis
- instability at junctional segments are a greater problem than compression, and individuals at risk of trivial injury
- prophylactic fusion abolishes any limited movement, but ensures stability and avoids tetraplegia

Symptomatic children need cervical fusion. Asymptomatic children need to be warned against contact sports, as minor trauma can result in catastrophic neurological compromise.

**Congenital Stenosis**

**Spondylosis and spondylolisthesis**
- failure of fusion between neural arch can occur on one side or both sides
- bilateral pathology risks forward displacement.

**Acute Intervertebral Disc Prolapse**

- Less common in the neck than in the back, but usually secondary to unguarded flexion and rotation affecting the C5/6 or C6/7 levels.

- Original attack usually related to a specific strain episode or trauma (e.g. whiplash)
  - Subsequent attacks usually with trivial precipitant
  - Pain and stiffness
  - Pain and unilateral upper limb paraesthesia (usually C6 or C7)
  - Neck tilted forwards / sideways with tender muscles and reduced ROM

- X-rays frequently show loss of cervical lordosis ± a narrowed disc space. MRI most useful.

- Differentials to exclude:
  - Acute soft tissue strain
  - Neuralgic amyotrophy – *usually no precipitant, with pain over shoulders more than neck, with more than one level affected on careful examination*
  - Infection – *unrelenting pain, x-rays show erosion of end-plates*
  - Tumour – *bone destruction*
  - Rotator cuff lesion

- Treatment principles:
  - REST – soft collar for 1 to 2 weeks
  - REDUCE – traction may enlarge disc space to allow prolapse to subside; 8kg applied to a harness under chin / occiput for no longer than 30 minute periods
Cervical Spondylosis

- Changes of **chronic** intervertebral disc degeneration (C5-7 most common)
  - Discs first flatten and become less elastic
  - Facet and unco-vertebral joints become arthritic
  - Anterior and posterior bony spurs – may cause foraminal encroachment

- Usually age > 40 – Possible symptoms include:
  - Pain and stiffness in the neck
  - Radiations include occiput, shoulders or arms
  - Radiculopathy - root compression / foraminal stenosis
  - Myelopathy – cord compression
  - Usually episodic
  - Muscular tenderness or spasm in neck and across scapulae
  - Reduced / painful ROM

- X-rays may show reduced disc spaces or spur formation, but MRI more reliable to assess if nerve roots affected.

- Differential diagnoses to exclude:
  - Nerve entrapment syndrome – however patients with long-standing cervical spondylosis are more vulnerable to peripheral nerve entrapment
  - Rotator cuff lesions
  - Cervical tumours
  - Thoracic outlet syndrome

- Treatment:
  - Conservative measures should include rest, analgesia, warmth and massage.
  - A collar to restrict movements is most effective during painful attacks
  - Physiotherapy – exercise, gentle passive manipulations and intermittent traction.
  - Surgical options:
    - Foraminal decompression if definitive evidence of root compression
    - Anterior fusion for single level disease that is only relieved by rigid support
    - Long-term results of fusion for multi-level spondylosis is not significantly better than conservative treatment.

Ossification of Posterior Longitudinal Ligament

- Occurs mainly in the cervical spine – reports initially from Japan, but actually widespread.
  - Associated with bone-forming conditions such as diffuse idiopathic skeletal hypertrophy (DISH) and fluorosis
May give rise to spinal stenosis and cervical myelopathy

Usually affects men age 50 to 70; possible symptoms include:
- Pain in neck or arms
- Sensory ± motor weakness in arms
- UMN signs in the legs
- Can lead to muscle wasting and even sphincter dysfunction

X-rays show dense ossification along the back of the vertebral bodies in the mid-cervical spine.

No treatment required if asymptomatic and simply an incidental finding.

Surgical decompression if progressive neurology and severe symptoms
- Posterior laminoplasty with posterior elements being jacked open.

Cervical Stenosis & Myelopathy

A sagittal distance between the posterior vertebral body and the base of the spinous process of < 11 mm is suggestive of stenosis

Neurological compromise can be caused by direct compression or ischaemia of the cord / roots.

Clinical features:
- neck pain and brachialgia
- paraesthesias, numbness, weakness and clumsiness in arms and legs
- increasingly unsteady gait
- may present acutely after a hyper-extension injury
- severe cases can result in sphincter disturbance

The condition is usually slowly progressive, but occasionally can deteriorate rapidly warranting urgent treatment.

Imaging:
- Sagittal (AP) canal diameter < 10 mm (12-13 at risk, and >17mm normal)
- Pavlov ratio < 0.8 = canal/body widths at level
- MRI is now the standard investigation, but CT myelography is superior in demonstrating bony detail

Differential diagnoses:
- Multiple sclerosis – episodic symptoms
- Amyotrophic lateral sclerosis (MND) – pure motor symptoms
- Syringomyelia
- Spinal cord tumours

Treatment:
- Conservative: analgesia, isometric exercises, collar, gait training
- Avoid manipulation or traction
- Surgical decompression for acute deterioration
Anterior for discogenic causes, osteophytosis and PLL ossification
Posterior for flavum ossification and other bone disorders

Pyogenic Infection

- Often missed in the early stages when responsive to antibiotic treatment
  - Initially confined to the intervertebral disc and adjacent body end plates
  - Later abscess formation with pus in the spinal canal or soft tissue planes
- Symptoms of severe neck pain with stiffness and muscle spasm
  - Systemic features often mild
  - But look for a raised WCC & ESR
- Treatment by antibiotics and rest
  - Traction immobilisation or a simple collar may be used
  - As infection is treated with antibiotics, the disc is destroyed
  - The vertebral space is obliterated as the bodies gradually fuse
  - Frank pus / abscess requires drainage

Tuberculosis

- Young patient usually complains of pain and stiffness
  - Neglected cases may present with a retropharyngeal abscess ± swelling ± dysphagia
  - Alternatively, the neck may be in kyphosis from disc & adjacent body collapse
- X-rays will show narrowing of the disc space and erosion of adjacent bodies
  - MRI will confirm this along with any paravertebral soft tissue abscess
- Treatment:
  - Anti-tuberculous drugs (minimum 6 months)
  - Immobilisation with a collar / brace
  - Surgery reserved for: abscess drainage, decompression of a threatened cord and to address any instability.

Rheumatoid Arthritis

- Affects cervical spine in 30% if RA patients:
  - erosion of atlanto-axial joints and transverse ligament
  - erosion of atlanto-occipital articulation, with cranial sinkage
  - mid-cervical facet joint erosion
  - osteoporosis either from disease progression or steroid use
- Neurological signs surprisingly uncommon, but can be caused by:
  - Direct mechanical compression
  - Local granulation tissue
Vertebral artery thrombosis

- Usually present after long history of RA
  - Neck pain and marked restriction of movement
  - Upper limb neurology more frequent than lower limb
  - Vertebro-basilar insufficiency (tinnitus, vertigo, visual disturbances)
  - Lehrmitte’s sign – paraesthesia in the legs on neck flexion
  - General debility and peripheral joint pathology may mask neurology

X-rays:
- Flexion-extension views show > 5mm gap between back of anterior arch and odontoid peg on flexion, with correction of the subluxation on extension.
- Lateral film shows the tip of the peg should be within 5 mm below McGregor’s line (a line from posterior edge of hard palate to the lowest point of the occiput; in atlanto-occipital erosion, the peg may be up to 10 mm above the line.

Indications for stabilisation:
- Unremitting pain
- Signs of nerve root or cord compression
- Fusion may need to be initially supported with a brace or even halo-jacket
- High morbidity and mortality is an argument for earlier intervention in instability with a threatened cord.

Ankylosing Spondylitis

- Most common seronegative arthropathy to affect the neck.
  - Chronic pain and stiffness over several years
  - Develops kyphosis
  - Chin-on chest deformity results in loss of upward gaze

A known AS-patient with new or increased neck pain is assumed to have a fracture until proven otherwise.

Displaced fractures need careful closed reduction and halo traction followed by halo-vest immobilisation, as surgery carries high risks.

Spasmodic Torticollis

- Most common form of focal dystonia – involuntary twisting or clonic neck movements
  - Spasms can be triggered by emotional disturbance or attempts to correct
  - Cause is principally idiopathic / unknown
  - Occasional association with basal ganglia lesions

Anti-cholinergics and other drugs tried with little success
- Botox-injections into the sternocleidomastoid have been tried.
The Back

Scoliosis
- Postural or structural
  - Postural (or irritative) represents compensatory deformity to a condition outside the spine (e.g. leg length discrepancy, pelvic tilt, or muscle spasm after a prolapsed disc).
  - Structural scoliosis is non-correctible lateral curvature + rotation
    - Spinous process rotate towards the concave side
    - The transverse process of the convex side rotates posteriorly
    - The ribs on the convex side stand out producing a rib deformity
  - Rare hysterical scoliosis
- Deformity increases during the growth period of the individual, but curves > 50-60° continue to worsen at a rate of 1° per year
- TB is worldwide commonest cause of kyphosis, while polio is worldwide commonest cause of scoliosis
- Causes:
  - Idiopathic (most common) – 80%
  - Osteopathic (congenital bony anomalies)
  - Neuropathic
  - Myopathic – arthrogryposis, muscular dystrophy
  - Metabolic – rickets, Osteogenesis Imperfecta
  - Syndromic – Marfan’s, Ehlers-Danlos
- Clinical history:
  - Deformity is usually the presenting complaint
  - Rib hump
  - Pain is rare – need to exclude neural tumour
  - Commonest symptoms is cosmesis
  - Birth details and milestones
  - Parents or others noticed progression
  - Limitations – academic / sporting usually placed on them by others
  - Age at menarche
  - Adults may complain of back ache
  - Family hx – abnormalities in pregnancy or developmental milestones
- Assessment
  - Examine for skin pigmentation, sacral dimples or hair tufts (neurofibromatosis, spina bifida)
  - Previous scars from thoracotomy or repair of cord anomaly
  - Assess for compensatory curves or balanced curves – occiput remains midline
Diagnostic feature of structural scoliosis is that forward bending makes the curve more prominent.

- Assess degree of mobility remaining by lateral bending
- Coronal and sagittal balance
- Symmetry – shoulder or waist balance
- Local tenderness – costo-iliac impingement
- Neurological examination / history
  - Including abdominal reflexes – 80% association of abnormal cord with absent abdominal reflexes
- Leg length
- Calf circumference for muscular dystrophy
- Chest height < 18 cm at maturity → associated with cardiopulmonary insufficiency
  - Mismatch in skeletal growth and chest height, resulting in early scoliosis causing reduction in vertical chest height growth which normally still has 50% to go during adolescence
  - Alveoli increase in number from birth to age 5 (20 million to 300 million)
  - Alveoli increase in size until chest wall growth is complete

**Imaging:**
- Erect PA x-ray of whole spine – which include the iliac crests to assess skeletal maturity from Risser’s sign
- Cobb angle – angle subtended by lines parallel to upper border of most cranial vertebra and lower border of most caudal vertebra within curve
- Lateral bending views – to assess correctibility
- Erect lateral whole spine – to assess sagittal balance
- MRI of whole spine to rule out any cord abnormalities
- Lung function tests

**Idiopathic**
- Often familial
- Incidence of curves > 30° and needing treatment is 0.3%
- 2-3% overall prevalence for smaller curves < 10° with less female dominance
- More trivial curves more common
- Can be divided into adolescent, juvenile and infantile

**Adolescent (age 10+):**
- 90% in girls
- usually primary right thoracic curves, with lumbar curves to the left
- curves under 20° usually remain static or resolve spontaneously
- once progression begins → usually continues until growth period over
- predictors of progression:
  - very young age
  - marked curvature
  - incomplete Risser sign

- treatment aims to prevent a mild deformity from becoming severe, and to correct cosmetic appearance which can have psychosocial implications to the patient as well as future cardiorespiratory issues.
Most referrals are initially monitored to gauge progression with 4-monthly x-rays of the standing whole spine.

Well balanced curves under 30° can be monitored.

If progression seen for curves between 20° and 30°, then consider bracing to prevent further deterioration, but be aware bracing does not achieve any reduction. Milwaukee or Boston brace worn for 23 hours of the day, and does not preclude mild exercise.

Surgical correction reserved for curves > 30° that are likely to progress or are that are cosmetically unacceptable. Double major balances curves corrected if > 60°

**Harrington rods** – placed posteriorly on concave side of curve, attaching to upper and lower ribs to distract the curve if flexible. Fusion then performed along length of curve to hold in place. Does not correct rotation and consequent rib hump.

**Luque rod and sublaminar wires** – sublaminar wires added in this modification of Harrington system to provide extra support along curve and try to pull backwards on each vertebra to reduce rotation. However risk of neurological injury increased.

**Cotrél-Dubousset system** – double rods to distract concavity and compress convexity. Rigid enough to not need post-operative bracing.

Current practice is to consider growing rods (Isola or VEPTR) to halt curve progression in growing children, lengthening every 6 months to reduce shortened stature, and performing a final fusion after skeletally mature. Alternatively, for untreated significant curves in a skeletally mature patient use posterior double rod instrumentation (e.g. DePuy Expedium or K2M), and consider anterior release and discectomies over apex of curve for better correction and to shorten anterior segment to reduce any distraction injury of cord during straightening. Neurophysiological cord monitoring mandatory.

**Juvenile (age 4-9):**
- Uncommon but with worse prognosis
- Surgical correction may be necessary before puberty
- However a brace may hold the curve until age 10, for a final fusion

**Infantile (age < 3):**
- Rare and reducing prevalence
- More common in boys – with left thoracic curves
- 90% can resolve spontaneously, but those with **rib-vertebra angles which differ by >20° between sides will progress** and deteriorate severely with cardiopulmonary sequelae.

**Treatment:**
- Serial elongation-derotation-flexion plaster casting under GA.
- Once age 4+ should be able to fit and comply with a brace.
- If bracing fails to control curve progression, then surgical intervention needed to control curve using growing rods.
- Avoid posterior fusion to allow as much height as possible.
ICEOS 2012 Conference:
- New classification system or early onset (EOS) and late onset scoliosis.
- Cut off age is 7 years
- Reflects importance of alveolar hyperplasia during the first 5 years following birth (rise from 20 million to 300 million)
- 35° Cobb angle is a limit for moderate scoliosis under which there is a role for bracing – this can control or improve Cobb angle, or at the very least delay the need for surgery and allow pedicles to grow enough to accommodate screws.
- Treatment stratification:
  - Bracing
  - Growing rods - manual lengthening, controlled self lengthening (SHILLA) or external lengthening (MAGEC)
  - Vertical expanding prosthetic rib (VEPTR) better for primarily chest wall problems.
  - Growing rods susceptible to law of diminishing returns where most only grow for 5 years.
  - Risk of auto-fusion over the years, but always need assessment for definitive surgical fusion (with or without instrumentation) on completion of majority of growth.

Congenital
- Assoications:
  - Klippel-Feil 25%
  - Renal 30%
  - Cardiac 10%
  - Neural axis 15%
  - 25% static, 50% moderate progression, 25% severe progression

Failure of formation
- Block vertebra – often multiple and stunt trunk growth, but progress at a slow rate as themselves have little growth potential
- Wedge Vertebra – usually in lower thoracic and lumbar regions, again progressing slowly at 1-2° per year
- Hemivertebra (unsegmented) – little growth potential and slow growth, usually <30° at maturity
- Multiple or segmented hemivertebra – as the site of the anomaly goes down the side, the progression rate increases.
- Sacral agenesis – in diabetic mothers

Failure of separation / segmentation
- Block vertebra
- Unilateral unsegmented bar – progression rate increases further down the spine, and tend to result in trunk imbalance, pelvic obliquity and large secondary curves.
  - Resection not effective because of re-fusion, but will respond to distraction (e.g. VEPTR – vertical expanding titanium rib)

Mixed defects:
- Potential for rapid deterioration in excess of 14° per year.
- Complex / unidentifiable pattern
Other causes:
- Absent or fused ribs
- Fractures from rickets or osteogenesis imperfecta

Rate of progression based on type and site:
- Failure of formation – 1 to 2° per year upper thoracic, 2 to 3° in lower thoracic, >3° per year in thoracolumbar region.
- Failure of segmentation – 2 to 6° per year in thoracic region, 6 to 9° in thoracolumbar region, and about 5° per year in the lumbar region.

Assess for clues such as tufts of excessive hair, skin dimples, naevi.
Spina bifida may be associated along with visceral anomalies
MRI to assess for Arnold-Chiari malformations, diastematomyelia, cord tethering, syrinx or conditions that put the cord at risk during correction.
Often require staged anterior release and posterior correction to reduce the risk of cord traction injuries.
No increased risk of next child having a congenital scoliosis – reassure parents.

**Neuropathic & Myopathic**

Deformity tends to be progressive even after skeletal maturity, with inadequate compensatory curves producing a loss of balance
- Typical paralytic curve is convex towards the side of the weaker spinal / thoracic / abdominal musculature
- Can result in imbalance and instability to the spine, which may make sitting or hygiene problematic (costo-iliac impingement).

Treatment poses ethical dilemmas in terms of consent and balancing the prognosis of the underlying pathology with the risks of surgery.
- Mild curves may require no treatment.
- Moderate curves can be monitored initially and then braced either externally or internally.
- Progressive or severe curves may simply be managed with sitting orthoses, but may require staged surgical release and stabilisation.
- May not help with respiratory or GI problems, which are contributed to from reflux, and the curve tends to be below the diaphragm.
- More risks and complications:
  - Poorer bone quality for screw fixation
  - Soft tissues oedematous and non-compliant
  - Respiratory problems and cardiomyopathies common
  - Nutritional status poor – check for albumin or lymphocyte deficiency
  - Uncooperative, epilepsy or other involuntary movements may increase risks of non-union
  - Higher infection rates (up to 10%)

Classification:
- Neuropathic
- Upper motor neurone
  - Cerebral palsy
  - Spino-cerebellar degeneration – CP, Friedreich’s, CMT
  - Syringomyelia
  - Spinal cord tumour / trauma
- Lower motor neurone
  - Poliomyelitis
  - Other viral myelitis
  - Traumatic
  - Spinal muscular atrophy

- Myopathic
  - Arthrogryposis
  - Muscular dystrophies – Duchenne’s
  - Congential hypotonia
  - Myotonia dystrophica

- Treatment principles are to restore function, taking into account any likely neurological progression from the primary disease, and the patient’s mobility and prognosis
  - Bracing is used to hold a curve static to allow younger patients some growth before definitive surgery.
  - Recently the VEPTR (vertical expanding titanium rib) can allow indirect internal control.
  - Wheelchair adaptations can achieve a similar effect to a brace without restricting chest movements
  - Physiotherapy with regular stretching important but will not alter curve progression.

- Surgery tends to involve long posterior corrections to prevent junctional recurrence above or below the fused segments. Anterior releases at the apex of very severe curves, and anterior surgery reserved for children under 10, where further anterior growth in the presence of posterior fusion would otherwise result in crankshaft failure.

- Cerebral Palsy – 25% have spinal deformity, frequency increases with level of disability.
  - Group 1 have balanced curves – treated surgically when curve has reached 60°, and usually does not need to be extended to pelvis
  - Group 2 have unbalanced curves with pelvic obliquity
    - Usually more severely disabled with stiff curves needing anterior release and posterior correction from T2-T3 all the way to pelvis.

- Neurofibromatosis:
  - Scoliosis can affect up to 33% of patients with neurofibromatosis
  - Usually a short, sharp curve
Managed as described above, but there is a higher rate of graft dissolution and pseudoarthrosis to be considered when planning final surgery.

BEWARE paralysis from dislocated rib head in the foramen, and intradural neurofibromas which tether cord

Ehlers-Danlos – avoid anterior surgery, due to friable anterior vessels.

Duchenne’s Muscular Dystrophy = X-linked affecting 1 in 3500 males.

- Progressive scoliosis in 95%, with initial hyperlordosis in 5%
- Usually have problems in teenage years, when fail from standing and go into a chair.
  - Muscle weakness more progressive at this stage, and lose compensatory hyperlordosis when walking – which tensions the ALL to stabilise the spine
- Beware: dilated cardiomyopathy and reduced lung function → plan surgery when curve is around 20° (i.e. quite early)
- But normally eat by equal movements or arm to mouth, and flexing spine down to arm → spinal fusion can mean they lose this “last independent action” of self feeding
- Usually only posterior correction to avoid further chest function impairment

Marfan’s

- Aortic defects mean anaesthetic risk
- Dural ectasia results in dilated dura and scalloped pedicles – difficult to place screws.

Kyphosis

Postural kyphosis

- Usually associated with other postural defects such as flat feet, or compensatory to hyper-lordosis elsewhere.
- Treatment consists of posture training and exercises

Structural kyphosis

- Children – congenital causes, achondroplasia, osteogenesis imperfect
- Adolescence – commonest cause is Scheuermann’s disease
- Adults – trauma, ankylosing spondylitis or TB
- Elderly – osteoporosis or simple degeneration

Congenital:

- Type 1 = failure of formation of the centrum in the body
  - Short, angular kyphosis, and progress most rapidly (2.5 to 5° per year)
  - Children with curves < 40° → posterior spinal fusion
  - Older or curves > 40° → combined anterior & posterior fusion
  - Combined with decompression if any neurological compromise
  - Untreated are most likely to result in paraplegia (10%) during preadolescent growth spurt
- Type 2 = failure of segmentation
  - Long, sweeping curves that progressed least rapidly
- Usually in the form of an anterior intervertebral bar limiting growth
- Posterior part continues to grow causing kyphosis
- Neurological compromise less frequent, but curve progression still warrants posterior fusion
  - Type 3 = mixed
  - Tend to occur in thoracolumbar area and progression rate was in between types 1 and 2.

- Scheuermann’s disease:
  - Defect in ossification of the ring epiphyses of vertebral end plates, with normal thoracic curve placing most pressure anteriorly where greater damage occurs and limits growth.
  - Affects boys > girls; starting at puberty
  - Increasing thoracic curvature noted by parent, with complaints from the child about back ache.
  - Severe deformity can obviously result in cardiopulmonary compromise
  - Compensatory hyper-lordosis predisposes to lower back pain later in life
  - X-ray – irregular vertebral adjacent end plates (T6 – T10) and wedge shapes bodies ± Schmorl’s nodes
  - Symptoms can be quite painful during adolescence, but usually subside after a few years

  - Treatment:
    - <40°: postural training and back exercises
    - 40-60°: brace to hold lumbar spine flat and thoracic spine in “extension”
    - 60-70°: posterior instrumented correction and fusion
    - >70°: anterior release and posterior fusion

- Degenerative kyphosis:
  - Usually caused by degeneration and narrowing of intervertebral discs
  - Gradually increasing stoop seen
  - Painless unless co-existent facet joint OA.

**Osteoporotic Vertebral Compression Fractures (VCF)**

- Osteoporosis is the most common cause of vertebral compression fractures in the elderly.
  - a reduction in thickness of trabeculae
  - reduced ratio of the vertical:horizontal trabeculae. (normal 2:1)
  - reduced density and increased porosity
  - failure strength falls linearly with density, and so suddenly normal physiological loading (up to 7000N) can exceed this.

  - The superior end plate is naturally thinner and more prone to fracture, perhaps as a result of pedicular shielding, and therefore results in characteristic kyphotic pattern of wedge fracture.

  - Note in the skeleton, trabeculae bone comprises 25% of all bone. However in the vertebrae, 75% of the bone is trabeculae.
Pain settles within 12 weeks in 85%, and percutaneous vertebroplasty (PVP) reserved for remaining 15% with chronic debilitating pain and focal tenderness at the level of the fracture (Lee, *Spine* 2009)
- Effect plateau’s within a few days
- Can also be used vs. AVN, myeloma or bone metastases
- Relief and mobility achieved in 80%
- Persistent kyphosis and inadequate height reduction – extrapedicular approach improves height restoration, but is harder to perform.
- Thermal necrosis minimal, as in vivo studies have shown that temperature only rises to 44°C within the vertebrae (and temperature rise is even less at the dura).

Complication rate between 1.5 to 3%
- Leakage of bone cement – limit by using more viscous cement
- Embolic events (marrow)
- Pneumothorax
- Paravertebral haematoma
- Infection
- 2nd level vertebral fracture prevalence up to 50%, with 70% of these 2nd fractures adjacent to the treated level.
  - Natural result of kyphosis is to add a sheer factor to adjacent level in addition to natural compressive forces.
  - Cement filling will increase the modulus above natural bone, and this gradient in sheer allows increased strain at these adjacent levels predisposing to 2nd fracture.

Mechanism initially thought to stop vertebral micro/macro-movements, but now unknown.

Occasionally, prophylaxis with 20% cement filling of adjacent vertebrae performed to reduce risk of adjacent level fracture.

**INVEST trial (2009)**
- Double blind RCT comparing PVP vs. a sham procedure of local anaesthetic
- Assessed disability at 30 days and 1 year
- Clinical improvement in pain in both groups, but no significant difference
- But used different clinical indications:
  - did not specify that there must be focal tenderness correlating to level of fracture, and evidence of oedema on MRI.
  - Disregarded clinical examination findings leading to inclusion of patients with other causes of mechanical back pain.
  - 32% of fractures acute – and results confounded by natural history of condition where up to 85% would resolve regardless of treatment.

**Klazen (*Lancet*, 2010)**
- Showed that using standard inclusion criteria that are currently employed in clinical practice, there was an immediate benefit with PVP over continuing conservative treatment.
Osteomyelitis and Discitis

- Two most common causes are direct inoculation after invasive procedures, or haematogenous spread, with *Staph aureus* being more common organism in 80% of cases.
- Usually starts in the vertebra and spreads into the disc and adjacent vertebra via the anterior intervertebral ligament.
- Occasionally a paravertebral abscess may track down to point into the groin.

 Symptoms & investigation
- Intense, unremitting localised pain
- Point tenderness of palpation
- Pyrexia and tachycardia may not be present
- Raised WCC and CRP
- Look for loss of disc space, with end plate erosion, and new bone formation
- MRI shows increased signal on T2 images, with irregularity of the end plates and oedema of the adjacent bone, with progressive destruction and disc space narrowing.
  - Gadolinium will distinguish between pus and solid inflammatory tissue, as there is no contrast uptake into pus. Can also highlight avascular bone in AVN.
  - Degenerative disc space narrowing with end-plate Modic type 1 changes are very similar – distinguish by the fact that this disc is dehydrated in degenerative disease, but oedematous in infection.
- Infection rarely involves the posterior elements, and therefore involvement of the pedicles is an ominous sign of malignancy or TB.
- Delay antibiotics under blood cultures and biopsy have been taken if possible

 Treatment:
- 4-6 weeks targeted IV antibiotics
- Once inflammatory markers normalising, move to oral antibiotics for further 6-8 weeks, and mobilise in brace.

 Surgical debridement if:
- Unresponsive to antibiotics
- Neurological symptoms develop
- Drainage of a soft tissue abscess required
- Grafts may be needed to fill any debrided bone defects, or posterior stabilisation.
- Non-iatrogenic causes of discitis is more common in children, where they often describe a recent flu-like illness followed by back pain, local tenderness, muscle spasm and severe restriction in movement.

 Metastatic disease or tumour usually does not spread to the relatively avascular disc $\rightarrow$ good disc, bad news & bad disc, good news.

Tuberculosis of the Spine

- Begins in vertebral body adjacent to this disc, resulting in bone destruction and caseation. Spreads across disc space to the adjacent vertebral end plate.
  - Bony collapse and kyphosis may occur
With healing, the vertebrae recalcify and fusion may occur.
Progressive kyphosis may occur if the spine has been destabilised

Pott's paraplegia
- Early onset paresis in spinal TB due to compression from abscess, caseous material or bony sequestrum
- Late onset paresis is caused by increasing deformity or vascular insufficiency
- Lower limb weakness, UMN signs, and sensory signs present

X-ray signs – local osteoporosis of two adjacent vertebral end plates with narrowing of this disc space. Followed by bone destruction and collapse.
- May see paraspinal soft-tissue shadows ± calcification if chronic

Mantoux may be positive, and ESR raised. If any doubt proceed to needle biopsy of a targeted lesion

Differential – bacterial infection (disc space collapse) and malignancy (discs preserved)

Goals of treatment:
- Eradicate or arrest disease
- Prevent or correct deformity
- Prevent or treat major neurological complications
- Ambulant chemotherapy for early or limited disease with no abscess formation – continue for 6-12 months. Consider a brace.
- Bed rest and chemo therapy for more advanced disease provided there is no abscess to be drained. If skills, facilities or patient fitness not suitable for radical surgery.
- Surgical debridement if any abscess that can be easily drained, or for an unstable spine. Usually an anterior approach is used with debridement of all necrotic material and stabilisation with a tricortical strut graft. If multi-level involvement, then may need augmentation with posterior instrumentation and fusion. Anti-tuberculous drugs still required.

Disc Degeneration & Prolapse

- Disc degeneration – age related changes of the nucleosus pulposus from a gelatinous bulb to a brown, dessicated structure. Accompanied by fissuring of the annulus fibrosus which allows nuclear material to squeeze through.
- Spondylosis – flattening of discs with marginal osteophytes, and Schmorl's nodes where protruded disc perforates the vertebral end plates.
- Reduction in disc space results in eventual disruption of normal facet joint mechanics, leading to osteoarthritis at these joints and narrowing of the
lateral recesses, along with “crumpling”/”thickening” of the flavum. The net result may be canal stenosis.

- Acute disc herniation results in a failure of the annulus to contain the nucleus material from the physical stresses experienced in flexion and compression. The disc usually bulges to one side of the posterior longitudinal ligament.
  - With complete rupture, disc material may sequestrate and lie free within the canal.
  - Local inflammation can aggravate nerve root compression
  - Pain from:
    - Disruption of the outermost fibres of the annulus
    - Stretching or tearing of the PLL
    - Pressure on the dura or covering of the nerve root
    (direct pressure on the nerve root itself causes paraesthesias and weakness)

- Usually affects young, fit adults age 20 to 45
  - Typically patient will recall a lifting or stooping and twisting event.
  - Usually leg pain develops after 48 hours
  - Coughing / straining can exacerbate pain
  - Paraesthesias and weakness may develop later
  - Cauda equina is rare, but presents with sphincter control problems
  - May stand slightly to one side (sciatic scoliosis) – with the ipsilateral knee slightly flexed, and all back movements reduced.
  - Symptoms are usually episodic, with intervals of normality. Severe, unrelenting pain is more typical of infection or tumour.

- Signs on examination:
  - Reduced straight leg raise
  - Sciatic stretch test provocation
  - Bow string of the lateral peroneal nerve
  - Crossed sciatic tension
  - Femoral nerve stretch test with a high or mid-lumbar prolapse.
  - Peripheral neurological deficits
  - Depressed ankle jerk with paradoxical increased patella reflex due to quadriceps action and weakness in antagonist muscle group (L5)

- MRI is the investigation of choice. Gadolinium contrast MRI reserved for recurrent prolapses, where scar tissue needs to be differentiated
  - Myelography useful for confirming the pathology site as the pain generator, but can be distressing to the patient and has side effects of headache (30%), nausea and dizziness.

- Treatment:
  - Rest
    - Bed rest ± 10kg traction to the pelvis ± NSAIDs
    - Successful in 90% within 2 weeks
  - Reduction
    - Steroid injection may dampen inflammatory response
  - Removal
- Cauda equine compression syndrome
- Neurological deterioration while under conservative management
- Persistent pain and signs of sciatic tension after 6 weeks of treatment
- Confirmed disc pathology on MRI at the same level as clinical signs.

- Rehabilitation
  - Isometric exercises
  - “Back school” education programme (how to sit, bend and lift)

- Arachnoiditis
  - Can occur after epidural injections or surgery or myelography
  - Diffuse back pain with vague lower limb symptoms
  - Cramps, burning, irritability
  - Sphincter dysfunction and male impotence may occur
  - Dural scarring may obliterate the subarachnoid space, and seen on MRI

- Treatment:
  - Steroid injection is only a temporary solution
  - Further surgery may worsen the condition
  - Pain clinic, psychological support and graduated activity programme is the only current option.
Segmental Instability and Facet Joint Dysfunction

- Prevalence of spondylolysis in young adult age group up to 7 to 10%

- Facet joint abnormalities include:
  - Anatomical variation that limits movement
  - Anatomical variation that permits excessive movement
  - Mal-opposition of the joint surfaces from loss of disc height
  - Loose bodies
  - Synovial thickening
  - Softening / fibrillation of articular cartilage
  - Classic changes of OA

- Clinical features:
  - Particular bad episode of back pain accompanied by referred leg pain
  - No true neurological symptoms
  - Usually intermittent episodes relating to heavy activity
  - Pattern of movement affected instead of range – with flexion easy, but straightening difficult
  - Muscle spasm can give a “locked back”

- X-ray changes:
  - Assymetry of disc space on AP view
  - Narrowing of disc space with “vacuum” sign (abnormal lucency)
  - Transverse anterior traction spurs
  - Spondylolisthesis or retrolisthesis

- Treatment options:
  - General care and attention – supra-tentorial reassurance
  - Physiotherapy
  - Facet joint injections
  - Spinal support – pulls their centre of mass closer to reduce moment arm and opposing posterior muscular forces.
  - Analgesia
  - Psychological support
  - Spinal fusion
    - Failed trial of conservative options
    - Should have had some benefit with conservative options
    - Proven facet joint arthropathy at the level fusion is proposed
    - Emotionally stable patient with no exaggeration or Waddell’s signs
    - Avoid in multi-level pathology, as cannot fuse the whole spine
    - Warned of failure rate up to 20%, and 40% risk of subsequent instability at another level
**Spondylololisthesis**

- **Dysplastic (20%)**
  - Congenital abnormality of superior sacral facets leads to an L5/S1 slip
  - Associated with spina bifida
  - Can lead to severe slip and neurologic compromise

- **Lytic / isthmic (50%) – spondylolysis**
  - Defects in pars interarticularis
  - Repeated trauma and healing can lead to elongation of pars
  - Often runs in families; affects Eskimos
  - Increased incidence with age – possible stress fracture
  - Competitive gymnasts, weight lifters, fast bowlers
  - A slip of up to 10%
    - rarely progresses after adulthood (5%)
    - does not predispose to future problems
    - not a contradiction to strenuous work (Wiltse, 1990)
  - A slip of over 25% gives an increased risk of back pain later in life

- **Degenerative (25%)**
  - Facet joint instability and arthritis, along with degenerative disc pathology can permit forwards slip – usually L4/L5
  - Rare below the age of 50
  - Slow progression and seldom exceeds 30% displacement

- **Other causes (5%)**
  - Post-traumatic
  - Post-operative
  - Pathological

- Can affect the dura, cauda equina or emerging foraminal nerve roots

- **Clinical features:**
  - Asymptomatic in children, with parents noticing abdomen is protruding forwards and child has a peculiar stance
  - Back pain and sciatica may affect adolescents and adults
  - Elderly with degenerative changes may present as above or with stenotic symptoms

- **Visible signs:**
  - Flat buttocks with transverse loin crease
  - Forward tilting of the pelvis
  - A “step” on palpation of the spinous processes

- **Management:**
  - Initial 6-12 months of conservative treatment considered the gold standard regardless of whether unilateral or bilateral
  - Reduction of activity and relative immobilisation ± modified Boston brace
Stretching of glutei and hamstrings, with a core strength training programme.

Local anaesthetic injection into the pars defect worth considering to establish this as the pain generator, before proceeding to surgery.

Surgical options include fixation or fusion:
- Buck repair has the most evidence with good outcome
- Altaf (JBJS, 2011) reports new repair with impacted bone graft before direct fixation with a pedicle screw and U-shaped connector underneath the spinous processes to maintain posterior structures – 60% union, 90% ODI and pain score improvement at 4 years.

Predictors of outcome:
- Age < 25 years
- Lysis < 4mm
- Absence of degenerative changes
- Good pain relief with pars block
- Grade 0 to 1 slips only

Spinal Stenosis

Natural history – 70% symptoms unchanged at 4 years, 15% improve and 15% deteriorate (Johnson, 1993)

Conservative measures rarely improve condition on a longterm (Postacchini, 1983)
- Activity modification (lifting, bending, twisting)
- Aerobic exercise – improves endorphins and circulation
- Physiotherapy – strengthening abdominals
- Short term use of corset
- NSAIDs, epidural injections
- Pain management

Indications for surgery:
- Failure of conservative measures to maintain symptoms static
- Intractable leg pain
- Progressive neurological deterioration
- Progressive functional impairment

Causes:
- Congenital vertebral dysplasia (e.g. achondroplasia)
- Chronic disc protrusion with peridiscal fibrosis/ossification
- Facet joint OA / displacement
- Hypertrophy of the ligamentum flavum
- Spondylolisthesis

CT defined measurements are 11mm AP-diameter and 16mm transverse diameter.

Symptoms:
- Aching, numbness, paraesthesias in thighs and legs
- Onset with standing or walking for 5-10 minutes
Relieved by sitting, squatting or leaning forwards
(in contrast to vascular claudication where rest relieves symptoms)
Walking downhill is harder than uphill

Conservative management simply includes postural changes.

Surgery involved a wide decompressive laminectomy and usually combined with instrumented stabilisation in patients over the age of 60

Surgical results (Iguchi + Kleeman, Spine, 2000)
- 82% 5 year success and 57% 10 year success with laminectomy (midline structures removed) → risk of instability and lumbar back pain
- highly selective decompression (midline preserved) → 96% improvement in leg pain, 80% improvement in back pain.
- Early surgery, use of a post-operative corset and physiotherapy improve prognosis.
- Predominant back pain, multiple levels of stenosis and significant neurological deficit are negative prognostic predictors.

Approaches to Low Back Pain

Transient backache following muscular activity
- More common in patients with FFD of hip or thoracic kyphosis, who tend to stand in hyper-lordosis
- Short period of rest followed by increasing exercise

Acute sciatica
- Exclude spondylolisthesis and infection
- Young adult – acute disc prolapse
- Elderly – osteoporotic fractures common, but exclude myeloma and metastatic disease

Intermittent low back pain after exertion
- Facet joint dysfunction
- Disc degeneration and segmental instability
- Exclude ankylosing spondylitis, chronic infection and myeloma

Back pain and pseudo-claudication
- Usually age > 50 → stenosis

Severe and constant pain localised to a particular site
- Compression fracture
- Paget’s disease
- Tumour
- Infection
Wadell signs (1984) are non-organic physical signs:
- Pain and tenderness in a bizarre distribution or degree
- Pain on performing impressive, non-stressful manoeuvres (e.g. pressing on head or rotating trunk)
- Deliberate SLR tender, but not on distraction
- Non-anatomic / physiologic patterns of sensory or motor abnormalities
- Overdetermined behaviours – trembling, sweating, hyperventilating, inability to move, exaggerated withdrawal.