## Causes of px arising from shoulder:

- Rotator cuff disorder
- Frozen shoulder
- Instability disorder
- AC joint disorder
- GH joint osteoarthritis
- Inflammatory arthritis
- Septic arthritis

## Causes of px which arise from elsewhere:

- Malignancy
- Referred pain from the neck, heart, or lungs
- Polymyalgia rheumatica

## Red flags on Hx or examination:

- Trauma, pain & weakness, or sudden loss of ability to actively raise the arm (with or without trauma): suspect acute rotator cuff tear
- Any shoulder mass or swelling: suspect malignancy
- Red skin, painful joint, fever, or the person is systemically unwell: suspect septic arthritis
- Trauma leading to loss of rotation & abnormal shape: possible shoulder dislocation
- New Ssx of inflammation in several joint: suspect inflammatory arthritis

## Further investigations:

- **Blood tests:** if malignancy, poly myalgia rheumatica, or inflammatory arthritis are suspected
- Testing for **diabetes** considered if pt with frozen shoulder
- **X-rays:** if Hx of trauma; little improvement with conservative care; Ssx last more than 4 weeks; severe pain or restriction of movement; arthritis suspected

## Treatments:

- Initial management: explanation & education on diagnosis; analgesia if appropriate; MSK treatment
- Corticosteroid injections may be considered, depending on the suspected cause & Ssx severity
- If orthopaedic referral is indicated (suspected septic arthritis / dislocation), should not be delayed
- Referral to 2° care considered if pain & function are not improving following conservative treatment for 3 months
- Earlier referral considered if: pain is having significant impact on ADLs; recurrent shoulder instability; severe post-traumatic pain

## Adhesive capsulitis

- **GREEN or YELLOW**
  - Condition gradually develops
- **Intro:**
  - Also known as *Frozen shoulder*
  - Inflammatory condition causing stiffness & px in the shoulder joint
  - Dx emphasises the gradual development of global limitation of shoulder motion
  - Significant radiographic findings may not be present
  - Assessing passive ROM crucial for Dx

## Adhesive capsulitis (cont)

| | |
|---|---|
| • **Aetiology (risk factors):** | - Prevalence 2-5% in general pop<br>- Common in people starting 55yrs<br>- F>M (1.4:1)<br>- Non-dominant hand often affected<br>- Associated w/ autoimmune comorbidities: thyroid disorders, DM (poorer treatment outcomes)<br>**Form classification:**<br>- **1°:** idiopathic, gradual onset, associated w/ other conditions (e.g. DM, thyroid disease, drugs, hypertriglyceridemia, or Cx spondylosis)<br>- **2°:** result of shoulder trauma (e.g. rotator cuff tears, #, surgery, or prolonged immobilisation) |
| • **Pathophysiology:** | - Not fully understood<br>- Leading hypothesis: inflammation begins in joint capsule & synovial fluid<br>- Subsequent reactive fibrosis & adhesions in synovial lining<br>- Initial inflammation causes px<br>- Fibrosis & adhesions limit ROM |
| • **Clinical presentation:** | - Gradual onset of shoulder px<br>- Worsens over weeks - months<br>- Followed w/ significant limitation in shoulder ROM |
| • **Physical examination:** | - Reduced AROM & PROM<br>- Specifically affected movements: EXT rotation → ABD → INT rotation → forward FX<br>- Severe cases may loose natural arm swing during walking & muscular dystrophy<br>- TTP around the joint<br>- Distal neurology MUST remain intact<br>- RROM elicits px & marked limitation, resembling rotator cuff tear<br>- Apley scratch test: measure INT rotation<br>- +ve special tests: Need, Hawkins, & Speed's (indicating impingement or biceps tendinopathy) |
| • **Diagnosis:** | - Dx based mainly on clinical & physical findings<br>- X-rays considered for alternative Dx or underlying pathology<br>- Injection test can help differentiate adhesive capsulitis from other shoulder pathologies<br>- MRI may reveal characteristic findings: rotator interval synovitis, coracohumeral lig hypertrophy, loss of subcoracoid fat triangle, & thickening of the GH capsule (they're not specific) |

## Adhesive capsulitis (cont)

| | |
|---|---|
| • **Staging:** | **3 clinical phases:**<br>- **Phase 1:** painful phase is characterised by diffuse & disabling shoulder px, initially worse at night, along w/ increasing stiffness, can last 2-9 months<br>- **Phase 2:** frozen or adhesive phase involves a progressive limitation in ROM in all shoulder planes, the intensity of px gradually diminishes during this phase, typically lasts from 4-12 months<br>- **Phase 3:** thawing or regression phase is marked by gradual return of the ROM, recovery of ROM may take 12-24 months for complete restoration |
| • **Complications:** | - Residual shoulder px &/or stiffness<br>- Humeral fracture<br>- Rupture of the biceps & subscapularis tendons<br>- Labral tears<br>- GH dislocation<br>- Rotator cuff tear |
| • **Management:** | - SMT & STW<br>- IASTM/TFM<br>- Spencer technique<br>- NSAIDs<br>- Corticosteroids / steroid injections<br>- Arthroscopic capsular release<br>- Exercises phase 1: Codman pendulum, Cane - FX & ABD, Cross body stretch, Shoulder INT rotation - towel, EXT rotation doorway stretch<br>- Exercises phase 2: Side lying horizontal ABD, resisted shoulder EXT prone, Resisted shoulder FX<br>**Indication for surgery:**<br>- Pt fails a trial of steroids or NSAIDs<br>- No response to GH or SC injections<br>- No response respond to PT<br>**Contraindications for surgery:**<br>- Pt has had an inadequate course of steroids or NSAIDs<br>- Pt has not had any attempt at conservative therapy<br>- Acute infection<br>- Pt has a concomitant malignancy in the shoulder<br>- Pt has a neurological deficit or nerve complaint originating from the Cx spine |

## Adhesive capsulitis (cont)

- **Ddx:**
  - Cx radiculopathy
  - AC arthrosis
  - Bicep tendinopathy
  - GH arthritis
  - Fracture
  - Calcifying tendinitis/synovitis
  - Malignancy
  - Polymyalgia rheumatica
  - Shoulder impingement s.

link text

## AC joint injury

- **GREEN to RED**
  - Sprain degree to torn degree
- **Intro:**
  - Common among athletes & adolescents
  - Around 40% of all shoulder injuries
  - Mild injuries usually don't cause significant morbidity
  - Severe injuries can result in substantial strength loss & function of shoulder
  - AC injuries may be linked to clavicular #
  - They can lead to impingement s.
  - Neurovascular insults are a rare complication associated w/ AC injuries
- **Aetiology (risk factors):**
  - Commonly occur after sporting events, car accidents, & falls
  - Make up about 40% of all shoulder injuries
  - Up to 10% of all injuries in collision sports (e.g. football, lacrosse, & ice hockey)
  - AC joint: lateral process of clavicle meets the acromion process projecting off the scapula
  - Stabilised by AC lig (anterior, posterior, superior, & inferior portion), where superior portion crucial for stability
  - Mild injuries don't cause significant issues, but severe can lead to substantial strength & function loss
  - Linked to clavicular #, impingement s., & occasionally neurovascular problems
- **Pathophysiology:**
  - Most common: direct trauma to lateral aspect of the shoulder or acromion process w/ the arm in ADD
  - Falling on an outstretched hand or elbow may also lead to AC separation

## AC joint injury (cont)

| | |
|---|---|
| • **Clinical presentation:** | - Antero-superior shoulder px<br>**Mechanism of injury:**<br>- Blunt trauma to ABD shoulder<br>- Landing on outstretched arm<br>**Px description:**<br>- Radiating to neck or shoulder<br>- Aggravated by movement<br>- Worse when sleeping on affected shoulder |
| • **Physical examination:** | - Swelling, bruising, or deformity of AC<br>- TTP<br>- Restricted A&PROM due to px<br>- "Piano key sign": palpation of the distal clavicle demonstrates a feeling of "giving way"<br>- Cross-body ADD test<br>- BvR test<br>- Paxino's test<br>- AC differential test |
| • **Diagnosis:** | - X-rays are 1° for Dx<br>- US & MRI may be considered if Dx remains uncertain |
| • **Staging:** | **Rockwood classification** (gold standard):<br>**I:** AC ligament sprain; CC ligament intact; no radiographic abnormalities<br>**II:** AC ligament is torn; CC ligament sprain; clavicle has elevated but is not superior to the border of the acromion, or exhibits a less than 25% increase in the CC interspace compared to the contralateral<br>**III:** AC & CC ligaments are torn; clavicle has elevated above the border of the acromion, or there is an increase of 25-100% in the CC interspace compared to the contralateral<br>**IV:** AC & CC ligaments are torn; posterior displacement of the distal clavicle into the trapezius<br>**V:** AC & CC ligaments are torn; superior displacement of the distal clavicle by more than 100% in the CC interspace compared to the contralateral<br>**VI:** AC & CC ligaments are torn; inferolateral displacement in a subacromial or subcoracoid displacement behind the coracobrachialis or biceps tendon |

## AC joint injury (cont)

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|---|---|
| • **Complications:** | - Residual joint px (30-50% of pts)<br>- AC arthritis (more common in surgical management)<br>- Following fixation: hardware irritation, infection, adhesive capsulitis, coracoid, & clavicular #<br>- Hook plate: acromion irritation, subacromial impingement, & osteolysis |
| • **Management:** | - Generally favourable prognosis<br>- Functional motion regain by 6 weeks & return to normal activity by 12 weeks<br>- Non-operative grade 1, 2 & 3; 3 operative if athlete / > displacement<br>- Acute (within 6 weeks): stabilisation & reduction of Ssx<br>- STW<br>- SMT (not shoulder)<br>- IASTM / TFM<br>- Exercises phase 1: scapular clocks & protraction / retraction<br>- Exercises phase 2: resisted shoulder EXT rotation, cane - FX, low row |
| • **Ddx:** | - AC distal clavicle osteolysis<br>- AC arthritis<br>- Acromion #<br>- Adhesive capsulitis<br>- Anterior humerus subluxation<br>- Complex pain s.<br>- Erb-Duchenne injury<br>- Glenoid labrum tear<br>- Os acromiale<br>- Rotator cuff injury<br>- Superior tabral tear<br>- Septic arthritis<br>- Shoulder dislocation |

link text

## AC osteoarthritis

• **GREEN**

## AC osteoarthritis (cont)

| | |
|---|---|
| • **Intro:** | - Common (spec. in 40 & older) & causes anterior / superior shoulder px<br>- Px exacerbated during overhead & cross-body activities<br>- 1° affects middle-aged pts due to degeneration of the fibrocartilaginous disc<br>- Many pts are asymptomatic, w/ findings often discovered incidentally on shoulder x-ray / MRI |
| • **Aetiology (risk factors):** | - Less common than knee / hip OA, but more common than GH OA<br>- Approx 54-57% of elderly pts exhibit x-ray evidence of degenerative changes in AC, though clinically relevant AC OA is less common<br>- Approx 20% of all shoulder px<br>- Common in 40 & older pts<br>**Types of AC arthritis:**<br>- **1° OA:** articular degeneration w/o an apparent underlying cause, often occurring due to constant stress from repeated overhead lifting activities<br>- **2° OA:** resulting from associated causes such as post-trauma (prevalent) or underlying disease (e.g. RA)<br>- Arthritic Ssx have been observed in Grade I & II sprains of the AC |
| • **Pathophysiology:** | - AC is a synovial joint connecting the axial skeleton & scapula<br>- Limited ROM characterises the AC<br>- Articular connection involves the distal clavicle's convex surface & the acromial facet's slight convex surface<br>- Fibrocartilage disc exists between the hyaline cartilage covered facets (akin to knee meniscus)<br>- Degenerative changes are part of the natural process<br>- In early adulthood, the fibrocartilage disc undergoes degeneration, leaving behind fibrous remnants |

## AC osteoarthritis (cont)

| | |
|---|---|
| • **Clinical presentation:** | - Hx of trauma, e.g. direct impact on the joint or a FOOSH injury<br>- Occupational Hx, e.g. occupation that requires repeated overhead lifting activities<br>- Participation in sports that stress / injure AC, e.g. weightlifting, rugby<br>- Complaints of px at night during sleeping on affected shoulder<br>- Pt may experience *popping*, *clicking*, *grinding*, or *catching* sensation w/ movement of the shoulder<br>- Functional limitations ACJ px include difficulty w/ resisted-training activities that place the GH in an extended position, common in weightlifters AKA *Weightlifter's Shoulder*<br>- Damage to AC can be synchronous w/ damage to the supraspinatus tendon & osteophytes from the arthritic AC joint may contribute to *subacromial impingement* exacerbating & producing further shoulder px |
| • **Physical examination:** | - Pts typically maintain intact ROM, EXCEPT for specific movements: cross-body ADD, behind the back (scratch back), & overhead reaching, which exacerbate px<br>- Localised superior shoulder px is common<br>- TTP, possible accompanied w/ swelling due to distal clavicle osteolysis<br>- Px can be induced in deltoid area through certain movements: forward FX to 90° w/ horizontal ADD (Cross-body test) or straight-ahead pushing (e.g. bench press)<br>- Most sensitive tests: TTP over Acromioclavicular point & Paxino's test, & AC resisted EXT test |
| • **Diagnosis:** | - Dx relies on Hx, physical exam, imaging (x-ray, MRI), & diagnostic local anaesthetic injection<br>**Imaging:**<br>- Plain film & Dx local anaesthetic local injections are essential Dx tools<br>- X-ray & MRI provide comprehensive imaging of AC joint pathology<br>- US is effective in detecting signs of AC OA & is commonly used for imaging<br>- US-guided injections: +ve if Ssx reduction; -ve if persistent px post-injection suggesting alt shoulder pathologies (commonly rotator cuff injury) |

## AC osteoarthritis (cont)

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|---|---|
| • **Management:** | - Activity modification (avoid repetitive & overhead movements), NSAIDs, PT modalities, corticosteroid & local anaesthetic injections<br>- Surgery<br>**Physical therapy:**<br>- Px management using electro-modalities, SMT/STW<br>- Maintaining active ROM & strengthening scapular stabiliser muscles<br>- Rotator cuff strengthening exercises<br>- Postural correction - pec muscle stretching & retractors strengthening |
| • **Ddx:** | - Calcific tendonitis<br>- GH arthritis<br>- Adhesive capsulitis<br>- Rotator cuff impingement s. |

link text

## Tendinopathies*

"**Tendinopathy** is an umbrella term to decribe the tendon px, w/ an unknown cause"

"**Tendinitis** describes a tendon in which inflammatory processes are present. However, studies show that tendons are rather in a degenerative state than in an inflammatory state."

"**Tendinosis** describes the degenerative state of tendons & therefore, this term is more applicable"
• Eccentric exercises major role in treatment
- Promote cross-linking of collagen fibres
- Promote tendon remodelling
• Tendinosis can be described on a continuum

## Calcific tendonitis

• GREEN

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|---|---|
| • **Intro:** | - Self-limiting disorder, identified by calcium deposits in rotator cuff tendons (esp. infra & supraspinatus)<br>- Common & painful condition, that decreases ROM<br>- Visible signs of calcium deposits overlying rotator cuff insertion on shoulder x-rays |
| • **Aetiology (risk factors):** | - Up to 20% of pts are asymptomatic<br>- 40-60% of shoulder pts<br>- 30-60yrs<br>- F>M<br>**Localisation:**<br>- Supraspinatus tendon (80%): critical zone (most common)<br>- Infraspinatus tendon (15%): lower 1/3<br>- Subscapularis tendon (5%): pre-insertional fibres |

## Calcific tendonitis (cont)

| | |
|---|---|
| • **Pathophysiology:** | - Unclear<br>**Hypothesis include:**<br>- Repetitive trauma of tendon → tendon degeneration → calcification<br>- Tendon necrosis → intracellular calcium accumulation<br>- Active process mediated by chondrocytes arising from metaplasia → calcium deposition<br>- Phagocytosis of metaplastic areas reforms normal tendon |
| • **Clinical presentation:** | - Night px, causing loss of sleep<br>- Constant dull ache<br>- Px increases considerably w/ AROM<br>- Decrease in ROM, or complaint of stiffness<br>- Radiating px up into suboccipital region, or down into the fingers |
| • **Physical examination:** | **Cluster (+ve):**<br>- Neer's test<br>- Hawkins-Kennedy test<br>- Drop arm test<br>- Jobe's test |
| • **Staging:** | **Chronic (silent) phase:** presence of the calcific deposit is asymptomatic & may be so for years<br>**Acute painful phase:** severe px, disability, & frequently nocturnal discomfort<br>**Mechanical phase:** tendon impingement being a prominent finding; px of less severe nature than the acute phase |
| • **Diagnosis:** | - Diagnosed through x-rays |
| • **Complications:** | - Adhesive capsulitis<br>- Rotator cuff tear<br>- Ossifying tendinitis |
| • **Management:** | - NSAIDs, PT, stretching & strengthening, steroid injections<br>- ESWT (most useful in refractory calcific tendonitis in the formative & resting phase)<br>- US-guided needle lavage<br>- Surgery (surgical decompression of calcium deposit)<br>**Physical therapy:**<br>- Mobs / drops<br>- ROM exercises to avoid articular stiffness<br>- Strength exercises to restore normal mechanics<br>- Commonly scapular dyskinesia needs to be treated at the same time |

## Calcific tendonitis (cont)

- **Ddx:**
  - Incidental calcification: found in 2.5-20% of 'normal' healthy shoulders
  - Degenerative calcification: found tendons w/ tear Hx; generally smaller; slightly older individuals
  - Loose bodies: associated chondral defect; associated 2° OA

link text

## GH dislocation

- **RED**
- **Intro:**
  - Separation of the humerus from glenoid of scapula at the GH joint
  - 50% of all joint dislocations
  - Anterior dislocation most common
  - Shoulder is an unstable joint due to a shallow glenoid that only articulates w/ a small part of humeral head
- **Aetiology (risk factors):**
  - Directions: anterior, posterior, inferior, or anterior-superior
  - **Risk factors:** Hx of shoulder dislocation, RC tear, Hx of glenoid fracture
  - M>F
  - Younger individuals, likely due to higher activity levels, more prone to redislocation
  - Dislocation occurs due to a strong force or extreme rotation, e.g. blow to the shoulder or trauma from contact sports, motor vehicle accidents, or falls
  - Fibrous tissue connecting the shoulder bones can be stretched or torn during dislocation, complicating injury

## GH dislocation (cont)

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|---|---|
| **• Pathophysiology:** | **Anterior dislocation:**<br>- Up to 97% of shoulder dislocations<br>- Mechanism: typically a blow + ABD + EXT rot + EXT<br>- Exam findings: ABD + EXT rot arm, prominent acromion<br>- Associated injuries: nerve damage, labrum tears, glenoid fossa or humeral head fractures (up to 40%)<br>**Posterior dislocation:**<br>- 2-4% of shoulder dislocations<br>- Mechanism: hit to the anterior shoulder, axial loading of ADD + INT rot arm<br>- Exam findings: arm held in ADD + INT rot, inability to EXT rot<br>- Higher risk of associated injuries: surgical neck or tuberosity #, reverse Hill-Sachs lesions, labrum or rotator cuff injuries<br>**Inferior dislocation (laxation erecta):**<br>- Least common type (less than 1%)<br>- Mechanism: hyperABD or axial loading on the ABD arm<br>- Exam findings: arm held above & behind the head, inability to ADD the arm<br>- Often associated w/: nerve injury, rotator cuff injury, tears in the internal capsule, highest incidence of axillary nerve & artery injury among shoulder injuries |
| **• Clinical presentation:** | - Pts may report: popping sensation, sudden onset of px w/ decreased ROM, sensation of joint rolling out of the socket<br>- Ask about PREVIOUS dislocations<br>- Nerves can get stretched out during shoulder dislocation, some pts may report *stinging* & *numbness* in the arm at the time of dislocation |

## GH dislocation (cont)

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|---|---|
| • **Physical examination:** | - ROM diminished & painfull<br>- **Anterior dislocation:** arm ABD & EXT rot; in thin pts potentially prominent funeral head felt anteriorly, & void can be seen posteriorly<br>- **Posterior dislocation:** easy to miss (pt appears to only guard the extremity) because arm is in INT rot & ADD; in thin pts potentially prominent head can be palpated posteriorly<br>- Neurovascular exam (IMPORTANT): axillary nerve injury (40%)<br>- **Special tests:** apprehension test (anterior & posterior), sulcus sign (inferior instability), load & shift test (anterior & posterior), anterior & posterior drawer test |
| • **Diagnosis:** | - Assess for axillary nerve injury: innervates deltoid & teres minor, & sensation to lateral shoulder<br>- Fractures of tuberosity & surgical neck may occur<br>- *Bankart lesion:* disruption of glenoid labrum, w/ or w/o avulsed bone fragment<br>- *Hill-Sachs deformity:* compression # of postern-lateral humeral head 1° w/ anterior dislocations<br>- *Reverse Hill-Sachs deformity:* impaction # of antero-medial aspect of humeral head in posterior dislocations |
| • **Management:** | - Posterior shoulder reduction<br>**Anterior shoulder reduction:**<br>- Scapular manipulation: highest success rate<br>- EXT rotation technique<br>- Cunningham technique<br>- Milch technique<br>- Stimson technique<br>- Traction-countertraction technique<br>- Spaso technique<br>- FARES technique<br>- Fulcrum technique |
| • **Ddx:** | - AC injury<br>- Bicipital tendonitis<br>- Clavicle fracture<br>- RC injury<br>- Shoulder dislocation<br>- Swimmer's shoulder |

link text

## GH instability*

• **YELLOW**

| | |
|---|---|
| • **Intro:** | - Includes dislocation & subluxation events<br>- Approx 1-2% of general population experience GH dislocation in their lifetime<br>- Shoulder instability events are common among young, active, athletic population<br>- Anterior shoulder instability accounts for over 95% |
| • **Aetiology (risk factors):** | **Classification criteria:**<br>- Uni- or multidirectional instability<br>- Traumatic or atraumatic<br>- Presence or absence of accompanying soft-tissue hyperlaxity<br>- M>F<br>- Rugby & football have particularly high incidence rates<br>- Anterior labral tears & Hill-Sachs lesions are frequently observed |

## GH instability* (cont)

**• Pathophysiology:**

**GH anatomy:**

- Complex, mobile, multiracial ball-and-socket articulation
- Allows motion in frontal, transverse, & sagittal planes
- Glenoid fossa articulates w/ humeral head, allowing 360° circumduction
- Movements at 4 distinct joints: SC, AC, GH, & scapuloTx

**Stabilisers:**

- **Static:** GH articulation, labrum, ligaments, RC interval structures, intra-articular pressure
- **Dynamic:** RC muscles, deltoid, scapular & periscapular stabilisers

**Shoulder instability cascade:**

- Excessive translation of humeral head on glenoid leads to px, weakness, dysfunction
- Anatomic risk factors identified
- Differentiation between joint laxity & instability crucial

**Unidirectional instability:**

- May result from acute trauma or low-energy instability events
- Soft tissue hyperlaxity may accompany
- Hill-Sachs lesion on humeral side common
- Glenoid bone loss prevalent, detected via CT scans
- Blunted osseous defects due to acute or chronic/recurrent processes

**Multidirectional instability:**

- Definition not precise; involves instability in multiple directions
- Often accompanied by capsulolabral injuries
- Soft tissue hyperlaxity associated w/ generalised hyperlaxity

**Long-term implications:**

- Altered biomechanics due to glenoid bone loss
- Scapular dyskinesia common, predisposing to instability
- Recurrent instability possible post non-operative/operative management
- Dislocation arthropathy: degenerative changes following instability events, possibly leading to GH arthritis

## GH instability* (cont)

**• Clinical presentation:**

**1st time dislocations:**

- Recent high-energy trauma or collision is often reported as the cause
- Ask about: degree of trauma, sports activities & positions, discernment between true dislocation & subluxation, & the need for manual reduction

**Chronic cases:**

- Pts often present after ROM limitations impact daily activities significantly
- Detailed Hx of inciting instability events should be gathered
- Initial injury may be overlooked, leading to chronic instability/recurrence
- Heightened clinical suspicion is warranted in cases of seizures, polytrauma, or low-energy, recurrent subluxation

## GH instability* (cont)

| | |
|---|---|
| • **Physical examination:** | **Cx exam:**<br>- Rule out Cx radiculopathy in neck or shoulder pathology<br>- Evaluate neck posturing, muscular symmetry, palpable tenderness, & ROM<br>- Conduct *Spurling manoeuvre*, myelopathies testing, reflex testing, & neurovascular exam<br>**Shoulder exam:**<br>- Compare B shoulder girdles for asymmetry, muscle bulk, or atrophic changes<br>- Check for anterior fullness in chronic anterior instability<br>- Assess scapulothoracic motion & scapular winging<br>- AROM & PROM, noting limitation in complex instability cases<br>- Assess axillary nerve function, supraspinatus muscle, & sensory examination<br>**Provocative tests:**<br>- Assess global tissue laxity, GH translation, & hypermobility<br>- Anterior apprehension test: reproduce Ssx of anterior instability<br>- Jobe relocation test: alleviate Ssx<br>- Load & shift test: assess humeral head translation (Grade 1, 2, or 3)<br>**Other exam considerations:**<br>- Check for posterior & multidirectional instability<br>- Expect associated shoulder pathologies based on age (e.g. RC injuries in older pts)<br>- Note weakness or px of specific shoulder injuries (e.g. RC tears or Bankart lesions) |
| • **Diagnosis:** | - X-rays for comprehensive evaluation<br>- MRI & CT for advanced imaging |
| • **Complications:** | - Redislocation following surgical fixation<br>- Nerve injuries (esp. axillary n.)<br>- Infection (surgery)<br>- Implant-related problems |

## GH instability* (cont)

**• Prognosis:**

- Depends on various factors
- Instability severity index score (ISIS) to guide shoulder instability management
- Risk factors for recurrence: age, gender, joint hyperlaxity, sport participation level/type, Hx of instability, & osseous lesions (10-point scoring of ISIS)

**5-year overall success rates:**

- 94% w/ 1-2 risk factors (ISIS score ≤ 3)
- 85% w/ ISIS score of 4-6
- 55% w/ ISIS score >6

**• Management:**

- Rehab program aim: enhance scapular stability; correct postural or functional deficits; increase RC function; improve proprioception
- Closed-chain exercises help stability w/o increasing shear force
- **Phase 1 (rehab):** decrease px, regain ROM, improve functional coordination
- **Phase 2 (exercises):** improve strength, coordination, proprioception
- Derby shoulder instability programme for recurrent *posterior* instability: stepwise exercise progression
- Scapular stability exercises focus on improving retraction & EXT rotation
- RC deficits, especially subscapularis, are crucial to address
- *Forward shoulder posture* may benefit from SMT in EXT rotation
- *Controllable* functional instability usually managed conservatively; *non-controllable* cases may need surgical repair
- Six-month conservative care trial appropriate before surgical intervention for non-traumatic posterior instability

## GH instability* (cont)

- **Ddx:**
  - Labral defect
  - SLAP lesion
  - Bankart lesion
  - Hill-Sachs lesion
  - Fracture
  - Inflammatory arthropathy
  - Shoulder impingement
  - RC tendinopathy
  - Biceps tendinopathy
  - Suprascapular n. entrapment
  - Quadrilateral space s.
  - Cx spine referral
  - Radiculopathy

link text

## GH internal rotation deficit (GIRD)

- **GREEN**
- **Intro:**
  - Commonly results from repetitive over-head throwing
  - Results in loss of IR
  - Functional deficit, not a specific injury
- **Aetiology (risk factors):**
  - Throwing motion ABD + ER + EXT w/ high velocities
  - High amount of stress on static & dynamic stabilisers of shoulder
  - Throwers often have a component of pathologic laxity or micro-instability (deposition for injuries)
- **Pathophysiology:**
  - Chronic tensile loading of posterior capsule leads to micro-tears & scarring
  - Resultant tissue changes contribute to loss of INT GH rotation
  - Limitation contributes to various shoulder, elbow, & wrist conditions
  - Sequellae inc: scapular dyskinesia, anterior shoulder impingement, RC s., & labral lesions
  - Limited shoulder ROM can also result from these conditions
- **Clinical presentation:**
  - Vague posterior shoulder px
  - Need for prolonged warm-up due to shoulder stiffness
  - Loss of throwing velocity, described as dead arm
  - Ssx exacerbated in the late cocking phase of throwing, typically localised to the posterior shoulder
  - Rare radiation of discomfort extending into the arm

## GH internal rotation deficit (GIRD) (cont)

| | |
|---|---|
| • **Physical examination:** | - Increased EXT rotation & decreased INT rotation - NOT related to MSK injuries or px in overhead throwing athletes<br>- TrPs: infraspinatus & teres minor<br>**GIRD Dx criteria:**<br>- At least 20* deficit of IR in dominant arm (compared B)<br>- TTP in posterior shoulder musculature |
| • **Management:** | - Target improving shoulder ROM (early focus), reduce muscle stiffness, & increase flexibility<br>- Stretching targets tightness in posterior capsule & INT rotators - pecs, biceps, subscapularis, infraspinatus, teres minor, & levator<br>- Crossbody stretching may be beneficial<br>- After pain-free ROM, follow w/ incremental strengthening of GH & scapular stabilisers<br>- TrPs like infraspinatus & teres minor (EXT rotators)<br>- SMT - IR & inferior glide<br>- Rest from throwing & physical therapy for 6 months |
| • **Ddx:** | - Shoulder Impingement s.<br>- RC s.<br>- Biceps tendinopathy<br>- Labral lesion |

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## Glenolabral articular disruption (GLAD)*

• **YELLOW or RED**

| | |
|---|---|
| • **Intro:** | - Soft tissue shoulder injury subtype<br>- Involves a tear to anterior-inferior labrum & adjacent glenoid articular cartilage damage<br>- Uncommon but established post-trauma cause of shoulder px<br>- Associated w/ stable GH joint; full ROM w/o apprehension or subluxation<br>- GLAD lesions seen in isolated or recurrent dislocations, challenging clinical Dx<br>- Imaging required for confirmation |

## Glenolabral articular disruption (GLAD)* (cont)

| | |
|---|---|
| • **Aetiology (risk factors):** | - Rare condition<br>- Est. 1.5-2.9% of cases of traumatic labral tears<br>- Younger M, consistent w/ general traumatic labral pathology<br>- Result from shoulder joint trauma, often involving forced ADD from a position of ABD + EXT rot, e.g. *FOOSH*<br>- Injury mechanism also inv. forceful ADD from throwing<br>- Anterior GH instability is a common injury mechanism associated w/ GLAD |
| • **Pathophysiology:** | - Affects the labrum & underlying glenoid cartilage in the GH joint<br>- GH: synovial ball & socket joint formed by the humeral head & glenoid fossa of the scapula<br>- Labrum function: adds depth to fossa & attachment point for long head of biceps tendon & GH ligaments<br>- Anterior labroligamentous complex: anterior-inferior GH ligament & labrum<br>- Function: prevents anterior dislocation & maintaining shoulder stability<br>- Injury mechanism: forceful ADD of the humeral head against the glenoid fossa, potentially accompanied by shear force, resulting in tears to the labrum & varying degrees of cartilage damage<br>- Despite the damage, the anterior labroligamentous complex often remains intact → shoulder joint remains stable in GLAD lesions<br>- Association between GLAD lesions & anterior shoulder instability |
| • **Clinical presentation:** | - Younger male, w/ clear onset of px after the event<br>- Potentially anteriorly, possibly diffusely<br>- Pt may localise px to deep-seated anterior joint<br>- Clear Hx of FOOSH, mechanism ADD force onto an ABD + EXT rot shoulder |
| • **Physical examination:** | - Px may be elicited on ABD & EXT rot<br>- Force ADD may produce *'popping'* sensation<br>- **Special tests:** Crank test; O'Briens test; Anterior Apprehension test; Passive Compression test<br>- High association between GLAD & anterior shoulder instability |

| Glenolabral articular disruption (GLAD)* (cont) | |
|---|---|
| • **Diagnosis:** | - Imaging, especially MRA, crucial for Dx<br>- Challenging to detect on non-contrast MRI or CTA<br>- Findings: superficial tear to the anterior-inferior labrum w/ an underlying glenoid cartilage defect (from superficial to trans-chondral)<br>- MRA demonstrates contrast tracking the labral tear & filling into the chondral defect or under a damaged articular flap |
| • **Compli-cations:** | - Linked to episodes of anterior shoulder instability<br>- Higher failure rates in arthroscopic Bankart repair w/ GLAD lesions<br>- Correlation between GLAD lesions & reduced GH stability<br>- GLAD lesions as biomechanical risk factor in shoulder instability by reducing joint concavity depth<br>- Risk of OA following GLAD injury (hypothesis) |
| • **Manage-ment:** | - Conservative: time, NSAIDs, & PT (especially for older pts)<br>- Incidental findings on imaging may complicate Dx in older pts due to common age-related cartilage & labral degeneration<br>- Treatment approach depends on the size & nature of the chondral defect & labral injury |
| • **Ddx:** | - Common traumatic labral tears, tearing of the labrum & associated ligaments partially or completely off the glenoid, most commonly the anterior-inferior labrum *(Bankart lesions)*<br>- Anterior-inferior instability lesions that include a glenoid rim # - *bony Bankart lesions*<br>- *Perthes lesion*: labral complex injury, but the labrum is still attached to the glenoid via a periosteal sleeve<br>- *Anterior ligamentous periosteal sleeve avulsion*: another labral injury, but it displaces medially on the glenoid neck<br>- *(HAGL)* or *Bony HAGL*: this time, the anterior-inferior GH ligament is avulsed from the humeral rather than labral attachment |

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## Polymyalgia rheumatica (PMR)*

| | |
|---|---|
| • **YELLOW or RED** | - Red if signs of vascular arteritis |
| • **Intro:** | - Rheumatic disorder 1° affecting white adults >50<br>- Characterised by px in neck, shoulder, & hip areas<br>- Inflammatory condition w/ elevated erythrocyte sedimentation rate (ESR) & C-reactive protein (CRP)<br>- Coexistence w/ or development of Giant cell arteritis (GCA) possible<br>- Dx challenges inc. distinguishing PMR from other conditions<br>- Does not lead to RA development |
| • **Aetiology (risk factors):** | - 100,000 / year<br>- White >50<br>- Second most common inflammatory autoimmune rheumatic disease (after RA)<br>- Etiology not well understood<br>- Some genetic predisposition<br>- Infection contribute: mycoplasma pneumonia, parvovirus B19, & Epstein-Barr virus (EBV)<br>- Some connection between PMR & diverticulitis, suggesting a role for changes in microbiota & chronic bowel inflammation |
| • **Pathophysiology:** | - Immune-mediated disorder<br>- Elevated inflammatory markers are common<br>- PMR pts have decreased number of circulating B cells (correlates w/ ESR & CRP) compared to healthy pts |
| • **Clinical presentation:** | - Symmetrical px & stiffness: affects shoulders, neck, & hip girdle<br>- Morning stiffness: worst in the morning, worsens after rest or inactivity<br>- Restricted shoulder ROM: common<br>- Upper body complaints: px & stiffness in upper arms, hips, thighs, upper & lower back<br>- Rapid onset: Ssx develop within day - 2 weeks<br>- Impact on quality of life: px impairs sleep & ADLs, e.g. getting out of bed, showering, driving, etc<br>- Inflammation sites: GH & hip joint, subacromial, subdeltoid, & trochanteric bursa<br>- Systemic Ssx: fatigue, malaise, anorexia, weight loss, low-grade fever (in some cases)<br>- Peripheral involvement: arthritis in 1/4 of pts, carpal tunnel s., distal extremity swelling w/ pitting edema, distal tenosynovitis |

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## Polymyalgia rheumatica (PMR)* (cont)

**• Physical examination:**

- Diffuse tenderness over shoulder
- Restricted AROM
- Normal PROM
- Restricted Cx & hip movements
- Muscle tenderness: neck, arms, & thigh
- Intact muscle strength despite complaints of weakness
- Normal sensory & reflexes (helps rule out mimicking conditions, e.g. peripheral neuropathy)
- Gait changes due to px & stiffness, e.g. shortened stride length, slow gait speed, stiffness, difficulty initiating movement, antalgic gait, decreased arm swing, & trunk lean

**• Diagnosis:**

**Labs:**

- Elevated ESR (>40mm)
- Elevated CRP
- Liver enzymes, especially alkaline phosphate, occasional elevated
- Serologic test (ANA, RF, Anti-CCP AB) negative
- CPK value within normal range

**Imaging:**

- US: assess subacromial/subdeltoid bursitis, biceps tenosynovitis, & GH synovitis
- MRI: depicts bursitis, synovitis, & tenosynovitis, more sensitive for hip & pelvic girdle findings; pelvic MRI often shows B peri-t-endinous enhancement of pelvic girdle tendons & occasional low-grade hip synovitis
- PET: shows FDG uptake in shoulders, ischial tuberosities, greater trochanters, GH, & SC joints

**Provisional classification criteria for PMR:**

Age 50 or older w/ B shoulder aching & abnormal CRP/ESR, + specific points from:

- Morning stiffness >45 min duration
- Hip px or restricted ROM
- Absence of rheumatoid factor or anti-citrullinated protein antibodies
- Absence of other joint involvement
- US findings (if available)

## Polymyalgia rheumatica (PMR)* (cont)

| | |
|---|---|
| • **Complications:** | - PMR pts have an increased risk of CV diseases<br>- Premature arteriosclerosis due to chronic inflammation is the probable cause of premature coronary artery disease (CAD)<br>- Some increased risk of lymphoplasmacytic lymphoma<br>- Higher likelihood of developing inflammatory arthritis (factors: small joint synovitis, younger age, & +ve anti-CCP) |
| • **Management:** | - Excellent prognosis w/ prompt Dx & appropriate treatment<br>- Medication<br>- Vitamin D & calcium supplementation for long-term steroids<br>- Pt should be educated on temporal/optic arteritis & how to act |
| • **Ddx:** | - RA<br>- GCA<br>- ANCA related vasculitis<br>- Inflammatory myositis & statin-induced myopathy<br>- Gout & CPPD<br>- Fibromyalgia<br>- Overuse or degenerative shoulder pathology (e.g. OA, RC tendinitis & tendon tear, adhesive capsulitis)<br>- Cx spin disorders (e.g. OA, radiculopathy)<br>- Crown dens s.<br>- Hypothyroidism<br>- Obstructive sleep apnea<br>- Depression<br>- Viral infections (e.g. EBV, hepatitis, HIV, parvovirus B19)<br>- Systemic bacterial infections, septic arthritis<br>- Cancer<br>- Diabetes |
| • **Temporal arteritis (TA):** | - 1 in 5 pts develop TA<br>- Systemic inflammatory vasculitis of arteries<br>- Scalp is painful to touch (hair brushing)<br>- Prominent, hardened & tender superficial temporal artery<br>- HA<br>- Claudication masticatoria<br>- Preliminary stage to optic arteritis (threat to visual ability) |

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| Rotator cuff injury* | |
|---|---|
| • **GREEN to RED** | - Grade 1, 2, 3 |
| • **Classification of strains:** | - **Grade 1 (green):** few torn/stretch muscle fibres w/ normal strength<br>- **Grade 2 (yellow):** several injured muscle fibres w/ muscle px, tenderness, mild swelling, bruising & loss of strength<br>- **Grade 3 (red):** complete tear of muscle w/ a possible audible sensation & a total loss of muscle function, severe px, bruising & swelling<br>- Referral depending on grade |
| • **Intro:** | - RC injuries range from *tendinopathy* to *complete tears*<br>- Rotator cuff: subscapularis (INT rotator), supraspinatus (ABductor), infraspinatus (EXT rotator), & teres minor (EXT rotator) |
| • **Aetiology (risk factors):** | - Most common tendon injury in adults<br>- Approx 30% of adults >60 have a tear, 62% in those >80<br>- Age is 1° factor for RC disease, being degenerative & progressive<br>- Risk factors: smoking (increases severity), family Hx, poor posture (kyphotic-lordotic, flat-back, swayback), trauma, hypercholesterolemia, & overhead activities<br>- Partial tears are prone to further propagation, factors inc. tear size, Ssx, location, & age<br>- Larger tears more likely to deteriorate structurally, w/ actively enlarging tears having higher likelihood of developing Ssx<br>- Anterior tears are more likely to progress to cuff degeneration |
| • **Pathophysiology:** | - **Macro**-trauma leads to acute tears, commonly in younger pts, resulting in complete tears<br>- **Micro**-trauma causes tendon degeneration, leading to degenerative tears<br>- Acute tears are typical in younger pts, while degenerative tears occur in older pts<br>- Sufficient tendon degeneration can make a complete tear possible w/ less force<br>- Multiple possible mechanisms: chronic degenerative tear, chronic impingement, acute avulsion injuries, iatrogenic injuries |

## Rotator cuff injury* (cont)

| | |
|---|---|
| • **Clinical presentation:** | - Typically begins w/ px, which can be acute or gradual<br>- Athletes often adapt biomechanics until they can no longer do their sport w/o px<br>- Pts may experience increasing px & difficulty w/ overhead activities & lifting heavy objects<br>- Px can radiate into the deltoid muscle area & may be felt when lying on the affected side<br>- Younger pts often have overuse tendinopathy<br>- Older pts may have OA contributing to the condition |
| • **Physical examination:** | - Tenderness at muscle insertion<br>- Muscle atrophy<br>- Abnormal scapular motion<br>- **Special tests:** Jobe (empty can) test, resisted EXT rotation, belly press test, drop arm test, & EXT & INT rotation lag sign |
| • **Diagnosis:** | - Plain radiography<br>- US - good for evaluating RC<br>- MRI - gold standard |
| • **Complications:** | - Retearing the cuff repair<br>- Adhesive capsulitis<br>- Inability to regain motion<br>- Cuff strength |
| • **Management:** | - Surgical & conservative treatment largest improvement at 12 months<br>- Surgery generally recommended for complete tears in pts <40, followed by rehab<br>- Conservative: PT, NSAIDs, subacromial corticosteroid injections<br>- STW<br>- SMT Cx & Tx<br>- GH mobs<br>- Nerve floss - brachial plexus<br>- Exercise phase 1: Codman pendulum, YTWL scapular depression, GH INT rotation, corner pectoral stretch<br>- Exercises phase 2: low row, eccentric supraspinatus, eccentric scapular stabilisers, eccentric shoulder ER's |

## Rotator cuff injury* (cont)

- **Ddx:**
  - SLAP or other labral tears
  - Subacromial impingement from bursitis, os acromiale, bone spurs
  - AC OA
  - Biceps tendinitis
  - Cx radiculopathy

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## Rotator cuff tendinopathy*

- **YELLOW**
- **Intro:**
  - RC injuries vary from minor contusions & tendonitis to chronic tendinopathy, partial tears (PTTs), & full-thickness tears (FTTs)
  - They can impact diverse pt groups, from recreational athletes (weekend warriors) to elite athletes
  - RC pathology is observed across all age demographics
- **Aetiology (risk factors):**
  - Subacromial impingement s. (SIS) is the most common cause of shoulder px, RC tendonitis is often seen associated
  - Occur acutely due to trauma or chronically from repetitive overuse activities
  - 5-10% in pts <20, & over 60% in pts >80
  - **Acute RC tendonitis** often affects athletes due to direct trauma, poor throwing mechanics, or FOOSH
  - **Chronic RC tendinopathy** can result from *extrinsic* compression (mechanical impingement) or *intrinsic* mechanisms (cuff degeneration)
  - Extrinsic compression can be caused by degenerative bursa, acromial spurring, or presupposing acromial morphologies
  - Intrinsic degenerative theories suggests cuff degeneration compromises joint stability, making the cuff susceptible to extrinsic compressive forces
  - Risk factors: vascular changes, age, sex, & genetics

## Rotator cuff tendinopathy* (cont)

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|---|---|
| • **Pathophysiology:** | **Acute RC tendonitis can be caused by:**<br>- Direct blows to the shoulder<br>- Poor throwing mechanics in overhead sports<br>- FOOSH<br>**Tendinopathy develops from repetitive RC injury, leading to:**<br>- Recurrent pathological cycle<br>- Acute or chronic tendonitis<br>- Increasing levels of tendinopathy & tendinosis<br>- Potential progression to PTTs &/or FTTs<br>**Exact pathogenesis of RC tears is controversial, but likely involves:**<br>- Extrinsic impingement from surrounding structures<br>- Intrinsic degeneration within the tendon itself |
| • **Clinical presentation:** | - Acute RC tendonitis: Hx of trauma or acute exacerbation on a chronic condition<br>- Chronic RC tendinopathy: either acute on chronic Hx/mechanism or a gradual, atraumatic onset<br>- Ssx may worsen w/ overhead activities<br>- Px, especially at night, is common<br>**Thorough exam includes:**<br>- Sports participation (including specific position played)<br>- Occupational Hx & current status<br>- Hand dominance<br>- Hx of shoulder &/or neck injury/trauma<br>- Relevant surgical Hx |

## Rotator cuff tendinopathy* (cont)

**• Physical examination:**

**Cx exam:**

- Rule out Cx radiculopathy (Spurling's test)
- Evaluate neck posturing, muscular symmetry, tenderness, & ROM
- **Special tests:** Spurling's, sensation testing, reflex testing, & neurovascular exam (7 P's)

**Shoulder exam:**

- Shoulder girdle symmetry, posturing, & muscle bulk
- Check for scapular winging & skin abnormalities
- Palpate for tenderness
- AROM & PROM
- Consider *RC tendonitis* w/ anterolateral tenderness
- Test motor strength C5-T1

**Special tests:**

- **Supraspinatus (SS):** Jobe's & drop arm test
- **Infraspinatus (IS):** Strength test & EXT rotation lag sign
- **Teres minor (TM):** strength test & Hornblower's sign
- **Subscapularis (SubSc):** IR lag sign, passive ER ROM, lift-off test, & belly press
- **EXT / subacromial impingement:** Neer impingement sign, Near impingement test, & Hawkin-Kennedy test
- **Internal impingement:** pt supine, shoulder brought into terminal ABD & EXT rotation; +ve if px reproduced

**• Diagnosis:**

- Imaging should be obtained in all pts w/ acute or chronic shoulder px
- Plain radiographs
- US (should be used more due to their specificity)
- MRI (provides more accurate tear details)

## Rotator cuff tendinopathy* (cont)

**• Complications:**

**Non-operative:**

- Persistent px / recurrent Ssx
- Setting of PTTs: risk of tear propagation, lack of healing, fatty infiltration, atrophy, & retraction
- Risks for tear progression: initial presence of FTT, medium-sized cuff tears (1-3cm), smoking
- Setting of chronic/atrophic tears: DJD & RC atrophy

**Surgical:**

- Most effective for pts who failed 4-6 months of conservative care
- Risks of surgery: recurrent px/Ssx, infection, stiffness, neurovascular injury, & risks associated w/ anaesthetic use
- Subacromial decompression/acromioplasty: deltoid dysfunction or anterosuperior escape

**• Management:**

- Majority of pts w/o FTTs improve w/ non-operative management
- NSAIDs, rest/activity modification, cortisone injections
- STW RC muscles
- Cx & Tx SMT
- GH mobs
- Nerve floss - brachial plexus
- Exercises Phase 1: Codman pendulum, YTWL scapular depression, GH IR, & Corner pec stretch
- Exercises Phase 2: low row, eccentric supraspinatus, eccentric scapular stabilisers, eccentric shoulder ER's
- Surgery

## Rotator cuff tendinopathy* (cont)

• **Ddx:**

**Impingement:**
- External / subacromial
- Subcoracoid
- Calcific tendonitis
- Internal (inc. SLAP, GIRD, little League shoulder, posterior labral tears)

**RC pathology:**
- Tendonitis (acute), Tendinopathy (chronic or acute on chronic)
- PTTs vs FTTs
- RC arthropathy

**Degenerative:**
- Advanced DJD (often associated w/ RC arthropathy)
- GH arthritis
- Adhesive capsulitis
- AVN
- Scapulothoracic crepitus

**Proximal biceps:**
- Subluxation (associated w/ subscapularis injuries)
- Tendonitis & tendinopathy

**AC joint conditions:**
- AC separation
- Distal clavicle osteolysis
- AC arthritis

**Instability:**
- Unidirectional instability - seen in association w/ an inciting event/dislocation (anterior, posterior, inferior)
- Multidirectional instability (MDI)
- Associated labral injuries/pathology

**Neurovascular conditions:**
- Suprascapular neuropathy (can be associated w/ paralabral cyst at the spinoglenoid notch)
- Scapular wining (medial or lateral)
- TOS
- Quadrilateral space s.

**Other conditions:**
- Scapulothoracic dyskinesia
- Os acromiale
- Muscle ruptures (pec major, deltoid, lat dorsi)
- Fracture (acute injury or px resulting from long-standing deformity, malunion, or nonunion)

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## Scapulothoracic dyskinesis*

• GREEN

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| • **Intro:** | - Altered position & motion of the scapula<br>- Also known as *dysrhythmia*, *dyskinesia*,or *SICK scapula syndrome*<br>- Scapular wining, exists but denoted a distinct condition typically following Tx or spinal accessory n. injury<br>- Observed in overhead athletes & pts w/ shoulder issues like RC disease, GH instability, impingement s., & labral tears, as well as in healthy pts<br>- No clear relationship between SD & shoulder px, even though some pts present w/ shoulder px<br>- Theory: SD might predict future shoulders even in the absence of current Ssx |
| • **Aetiology (risk factors):** | **Shoulder-related:**<br>- Shoulder pathologies associated w/ SD *(AC instability, shoulder impingement, RC injuries, glenoid labrum injuries, clavicle #)*<br>- Inflexibility of the pec minor & short head of biceps<br>- Stiffness of posterior GH capsule<br>**Neck-related:**<br>- Mechanical neck px s.<br>- Cx n. root-related s.<br>**Posture-related:**<br>- Excessive Tx kyphosis & Cx lordosis<br>- Athletes show these are more related causes SD |

## Scapulothoracic dyskinesis* (cont)

**• Pathophysiology:**

**Scapular motions:**
- Upward/downward rotation
- Internal/external rotation
- Anterior/posterior tilt

**Scapular translation:**
- Upward/downward sliding on the Tx
- Medial/lateral sliding around the curvature of Tx

**Common scapular patterns:**
- Scapular retraction: EXT rot + posterior tilt + upward rot + medial translation
- Protraction: INT rot + anterior tilt + downward rot + lateral translation
- Shrug: upward translation + anterior tilt + INT rot

**Normal overhead elevation:**
- Minimal INT/EXT rot until 100°
- 1° scapular motion: upward rot
- 2° scapular motion: posterior tilt

**Scapulohumeral rhythm:**
- Coordinated movement between scapula & humerus for efficient arm movement
- 2:1 ratio between GH elevation & scapular upward rot
- Consistent pattern during scapular plane elevation: upward rot + posterior tilt + EXT rot + clavicular elevation + retraction

**Altered mechanics in SD:**
- Increased scapular anterior tilt
- Increased scapular INT rot
- Altered scapular upward rot

**• Clinical presentation:**

- Pts w/ SD can be symptomatic or asymptomatic

*Ssx can be one or combination of the following:*
- Anterior shoulder px
- Posterosuperior scapular px (may radiate into ipsilateral para spinous Cx region or radicular/thoracic outlet-type Ssx in the affected UL)
- Superior shoulder px
- Proximal lateral arm px

## Scapulothoracic dyskinesis* (cont)

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|---|---|
| • **Physical examination:** | - Assess AC & SC for instability<br>- Infraspinatus strength test<br>- Manual resistance of the arm at 130° of FX (for serratus anterior)<br>- Manual resistance of the arm at 130-150° of ABD (for lower & middle traps)<br>- Extension of the arm at the side (for rhomboids)<br>- Low row test<br>- Scapulohumeral rhythm test<br>- Quadruped rock<br>- Lateral scapular slide test<br>- Scapular dyskinesia test<br>- SICK scapula sign |
| • **Diagnosis:** | **Classification of dyskinesia types:**<br>- **Type 1:** inferior angle prominence (i.e. anterior tilt of scapula)<br>- **Type 2:** medial border prominence (i.e. winging of the scapula)<br>- **Type 3:** early scapular elevation or excessive/insufficient upward rot during arm elevation |
| • **Complications:** | - SD diminishes subacromial space & leads to decreased RC strength, impingement Ssx, & eventual RC damage<br>- 100% of pts w/ shoulder impingement demonstrate dyskinesia<br>- 5% of pts w/ dyskinesia have neurologic injury/damage (spinal accessory, long Tx, suprascapular)<br>- SD can occur from core & hip ABD weakness<br>- SD becomes more apparent w/ dynamic testing, particularly during the lowering phase of arm movement<br>- Recognition & rehab should begin independent of (generally absent) Ssx |
| • **Management:** | - STW: upper traps, pec minor, biceps<br>- SMT: Cx & Tx<br>- Scapular mobs<br>- Treatment aims at restoration of scapular retraction, posterior tilt & EXT rot<br>- Exercises Phase 1: trap stretch - sitting, YTWL scapular depression<br>- Exercises Phase 2: low row, burger w/ band |

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## Subacromial bursitis

• **YELLOW**

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|---|---|
| • **Intro:** | - Bursa is a fluid-filled sac<br>- Lubricatesjoints & body surfaces prone to wear & friction<br>- Subacromial bursa is surrounded by the acromion, coracoid, coracoacromial ligament, & deltoid muscle fibres<br>- Inflammation of this bursa can lead to subacromial bursitis |
| • **Aetiology (risk factors):** | - Around 0.4% of primary care visits<br>- F=M<br>**Common aetiologies:**<br>- Subacromial impingement (especially in older pts)<br>- Repetitive overhead activities / overuse (athletes, factory workers, manual labourers)<br>- Direct trauma<br>- Crystal deposition<br>- Subacromial hemmorhage<br>- Infection<br>- Autoimmune disease (e.g. RA) |
| • **Pathophysiology:** | - Aetiologies can cause inflammation of the subacromial bursa, leading to increased fluid & collagen formation<br>- Fluid is often rich in fibrin & can become hemorrhagic<br>**Bursitis has 3 phases:**<br>- **Acute:** marked by local inflammation w/ thickened synovial fluid, resulting in painful movement, especially w/ overhead activities<br>- **Chronic:** constant px due to a chronic inflammatory process, which can weaken & eventually rupture surrounding ligaments & tendons. Require attention to tendinitis as they may coexist<br>- **Recurring:** can result from repetitive trauma or routine overhead activities, & it may also be seen in pts w/ inflammatory conditions (e.g. RA) |

## Subacromial bursitis (cont)

| | |
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| • **Clinical presentation:** | - Px in the anterolateral aspect of the shoulder<br>- Possible causes: trauma (fall w/ direct impact), repetitive overhead activities (sports, lifting)<br>- Impingement s. as a common cause<br>- Mechanism: decreased subacromial space due to overhead activities<br>- Effect of arm ABD: brings humerus closer to acromion, reducing subacromial space<br>- Function of subacromial bursa: protects supraspinatus muscle from wear between humeral head & acromion<br>- Result of repetitive activity: irritation & inflammation of the bursa<br>- Consideration of tendon pathology: supraspinatus tendinitis or tear may coexist w/ impingement s. |
| • **Physical examination:** | - TTP at anterolateral aspect of shoulder below acromion<br>- Localised px, doesn't usually radiate (if it does, consider Cx pathology)<br>- Warm or boggy skin at site, but no erythema typically<br>- Px on resisted ABD of arm beyond 75-80°<br>- Compression of subacromial bursa at undersurface of acromion during motion |
| • **Diagnosis:** | - X-rays may be used to rule out other pathologies (e.g. fractures, dislocations, OA, etc)<br>- MRI: burial fluid accumulation visible<br>- US: evaluates the thickness of the bursa |
| • **Complications:** | - Not associated w/ many complications<br>- Repeated steroid injections: theoretical risk of introducing an infection into skin/joint<br>- Risk of damaging RC muscles w/ recurrent injections |
| • **Management:** | - Good prognosis for pts w/ conservative care, even w/ surgery<br>- Rest, NSAIDs, PT, & corticosteroid injections<br>- Surgery for pts non responsive to conservative care |
| • **Ddx:** | - Impingement syndrome<br>- RC tendinitis/tear<br>- Biceps tendinitis<br>- Adhesive capsulitis<br>- AC joint OA |

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## Subacromial impingement syndrome (SIS)*

• GREEN

**• Intro:**

- SIS is the inflammation, irritation, degradation in subacromial space structures
- Shoulder impingement s. is considered most common cause of shoulder px
- Shoulder px often persists or recurs
- 54% of pts experience persistent Ssx after 3 years

**• Aetiology (risk factors):**

- Common in overhead sports (handball, volleyball), & manual labourers
- Incidence rises w/ age (especially 60s)
- Shoulder *external* impingement distinguished from *internal* impingement by RC anatomy

**Extrinsic risk factors:**

- Heavy loads
- Infection
- Smoking

**• Pathophysiology:**

- Normal shoulder movement narrows subacromial space, causing px
- Unclear whether tendon damage or narrowed space causes impingement
- Described by location (external/internal) & cause (1°/2°)

**Anatomic borders:**

- Acromion
- Coracoacromial ligament
- AC joint
- Humeral head

**External (subacromial) impingement:**

- Mechanical encroachment of soft tissue in subacromial space
- **1° impingement:** structural narrowing (e.g. abnormal acromion)
- **2° impingement:** onset during motion due to RC weakness

**• Staging:**

**Neer's classification:**

- **Stage 1:** edema, haemorrhage from overuse
- **Stage 2:** fibrosis, irreversible tendon changes
- **Stage 3:** tendon rupture/tear due to chronic fibrosis

## Subacromial impingement syndrome (SIS)* (cont)

| | |
|---|---|
| • **Clinical presentation:** | - Px upon lifting the arm or lying on the affected side<br>- Pts may report loss of motion, nighttime px, weakness, & stiffness<br>- Onset is gradual over weeks to months, w/o a specific traumatic event<br>- Px is typically felt over the lateral acromion w/ radiation to the lateral mid-humerus<br>- Inquire: onset, quality, exacerbating factors, interventions tried, & prior injuries<br>- **Important:** overhead & repetitive activities<br>- Relief: rest, NSAIDs, ice<br>- Ssx often return w/ activity |
| • **Physical examination:** | - Inspection, palpation, A & PROM, & strength testing of neck & shoulder<br>- B comparison<br>- Common weakness: ABD &/or EXT rotation<br>- *Scapular dyskinesis* during arm forward elevation<br>- Tenderness over the coracoid process of affected arm<br>**Special tests for shoulder impingement:**<br>- Hawkins test - subacromial (external)<br>- Neer sign - anterior px = subacromial; posterior px = internal<br>- Jobe (empty can test)<br>- Painful arc of motion<br>**Special tests for shoulder instability:**<br>- Sulcus sign<br>- Anterior apprehension<br>- Relocation test - internal |
| • **Diagnosis:** | - Dx made from physical exam<br>- Imaging used to confirm & rule out other issues |
| • **Complications:** | - Due to structural damage within subacromial space<br>- Altered biomechanics<br>- Avoidance of use w/ subsequent atrophy<br>- Potential pathologies that may result: RC tendonitis/tear, bicipital tendonitis/tear, or adhesive capsulitis |

## Subacromial impingement syndrome (SIS)* (cont)

- **Management:**
  - Most pts resolve within 2 yrs w/ conservative care (initial approach before considering surgery)
  - Restoring ROM is crucial, avoid aggravating movements e.g. elevation & INT rotation
  - Tape used enhance recovery & decrease px
  - Steroid injections
  - Surgery
  - STW (RC), SMT (Cx/Tx), GH mobs, nerve floss (brachial plexus)
  - Exercises Phase 1: Codman pendulum, YTWL scapular depression, GH INT rotation, Corner pec stretch
  - Exercises Phase 2: low row, Brugger w/ band
- **Ddx:**
  - Adhesive capsulitis
  - RC tear
  - AC OA
  - AC sprain
  - Trapezius muscle spasm
  - Biceps tendonitis
  - Biceps tendon rupture
  - Calcific tendonitis
  - GH arthritis
  - Distal clavicle osteolysis
  - Cx radiculopathy
  - TOS

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## Hypermobility syndromes (HMS)

- **GREEN**

- **Intro:**
  - Generalised articular hypermobility, w/ or w/o subluxation or dislocation
  - Also known as *joint hypermobility s.* & *benign hypermobility joint s.*
  - Primary Ssx: excessive laxity of multiple joints
  - Differs from localised joint hypermobility & other disorders e.g. Ehlers-Danlos s, RA, lupus, & Marfan s.
  - May occur in chromosomal & genetic disorders like Down syndrome, & metabolic disorders e.g. homocystinuria & hyperlysinemia
  - Lab tests used to exclude other systemic disorders when HMS is suspected

## Hypermobility syndromes (HMS) (cont)

| | |
|---|---|
| • **Aetiology (risk factors):** | - Most prevalent in children & tends to decrease w/ age<br>- Joint mobility is at its highest at birth, decreasing in children around 9-12 yrs<br>- Adolescent girls hypermobility peak at 15, decrease after, influenced by hormonal changes<br>- F>M<br>- More prevalent in ASIA, Africa, & Middle East |
| • **Pathophysiology:** | - Involves systemic collagen abnormality<br>- Joint hypermobility & tissue laxity are linked to abnormal collagen ratios<br>- Collagen types I, II, & III are decreased in the skin<br>- Dx criteria include joint abnormality<br>- Affects cardiac tissue, smooth muscle in female genital system, & GI system<br>- Impairs joint position sense |

## Hypermobility syndromes (HMS) (cont)

• **Clinical presentation:**

**Joint instability & trauma:**
- Recurrent ankle sprains
- Meniscus tears
- Acute or recurrent dislocations or subluxations of various joints (shoulder, patella, MCP joints, TMJ)
- Traumatic arthritis
- Bruising
- Fractures (chronic or non-traumatic)
- Chondromalacia

**Soft tissue disorders:**
- Tendinitis
- Epicondylitis
- RC syndrome
- Synovitis
- Juvenile episodic synovitis
- Bursitis

**MSK conditions:**
- Scoiliosis
- OA
- Congenital hip dislocation
- Delayed motor development
- Flat feet & sequelae

**Neurological Ssx:**
- Nerve compression disorders - carpal tunnel, tarsal tunnel, TOS
- Raynaud s.
- Clumsiness
- Chronic HA

**Px & sleep issues:**
- Exercise-related / post-exercise-related px
- Nocturnal leg px
- Low nocturnal sleep quality
- Joint swelling
- Back px
- Unspecified arthralgia or effusion of affected joint

**Other systemic effects:**
- Fibromyalgia
- Chronic fatigue s.
- Functional GI disorders
- Immune system dysregulation
- Pelvic dysfunction
- CV dysautonomia
- Exocrine glands dysfunction
- Little changes of the skin
- Greater risk of failures in tendon, ligament, bone, skin, & cartilage
- Enhanced flexibility
- Ankylosing spondylitis (axial spondyloarthritis)

## Hypermobility syndromes (HMS) (cont)

| | |
|---|---|
| • **Physical examination:** | - ROM<br>- End feel<br>- Beighton score<br>- Paradoxical breathing evaluation |
| • **Diagnosis:** | **Major criteria:**<br>- Beighton score of ≧4/9<br>- Arthralgia for >3 months in >4 joints<br>**Minor criteria:**<br>- Beighton score of 1-3<br>- Arthralgia in 1-3 joints<br>- Hx of joint dislocation<br>- Soft tissue lesions >3<br>- Marfan-like habitus<br>- Skin striae, hyperextensibilty or scarring<br>- Eye signs, lid laxity<br>- Hx of varicose veins, hernia, visceral prolapse<br>**Requirement for Dx of HMS:**<br>- 2 major criteria<br>- 1 major criteria + 2 minor criteria<br>- 4 minor criteria<br>- 2 minor criteria & unequivocally affected 1st-degree relative in FHx |
| • **Complications:** | - Px & stiffness<br>- Clicking<br>- Dislocations<br>- Recurrent injuries<br>- Digestive problems<br>- Dizziness & fainting<br>- Fatigue |
| • **Management:** | - Education<br>- Abdominal brace exercise<br>- Active mobs exercises<br>- Strengthening exercises - muscle surrounding the joint<br>- Proprioceptive exercises<br>- Control neutral joint position<br>- Re-train dynamic control<br>- Motion control<br>- NSAIDs for px management |

## Hypermobility syndromes (HMS) (cont)

- **Ddx:**
  - Ehlers-Danlos syndrome
  - Fibromyalgia
  - Chronic fatigue syndrome
  - Depression

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