

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

- **Phase 1:** painful phase is characterised by diffuse & disabling shoulder px, initially worse at night, along w/ increasing stiffness, can last 2-9 months
- **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
- **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
- Humeral fracture
- Rupture of the biceps & subscapularis tendons
- Labral tears
- GH dislocation
- Rotator cuff tear

• **Management:**

- SMT & STW
- IASTM/TFM
- Spencer technique
- NSAIDs
- Corticosteroids / steroid injections
- Arthroscopic capsular release
- Exercises phase 1: Codman pendulum, Cane - FX & ABD, Cross body stretch, Shoulder INT rotation - towel, EXT rotation doorway stretch
- Exercises phase 2: Side lying horizontal ABD, resisted shoulder EXT prone, Resisted shoulder FX

Indication for surgery:

- Pt fails a trial of steroids or NSAIDs
- No response to GH or SC injections
- No response respond to PT

Contraindications for surgery:

- Pt has had an inadequate course of steroids or NSAIDs
- Pt has not had any attempt at conservative therapy
- Acute infection
- Pt has a concomitant malignancy in the shoulder
- 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 Mechanism of injury: - Blunt trauma to ABD shoulder - Landing on outstretched arm Px description: - Radiating to neck or shoulder - Aggravated by movement - Worse when sleeping on affected shoulder
• Physical examination:	- Swelling, bruising, or deformity of AC - TTP - Restricted A&PROM due to px - "Piano key sign": palpation of the distal clavicle demonstrates a feeling of "giving way" - Cross-body ADD test - BvR test - Paxino's test - AC differential test
• Diagnosis:	- X-rays are 1° for Dx - US & MRI may be considered if Dx remains uncertain
• Staging:	Rockwood classification (gold standard): I: AC ligament sprain; CC ligament intact; no radiographic abnormalities 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 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 IV: AC & CC ligaments are torn; posterior displacement of the distal clavicle into the trapezius V: AC & CC ligaments are torn; superior displacement of the distal clavicle by more than 100% in the CC interspace compared to the contralateral VI: AC & CC ligaments are torn; inferolateral displacement in a subacromial or subcoracoid displacement behind the coracobrachialis or biceps tendon

AC joint injury (cont)

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

[link text](#)

AC osteoarthritis

- **GREEN**



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AC osteoarthritis (cont)

- **Intro:**
 - Common (spec. in 40 & older) & causes anterior / superior shoulder px
 - Px exacerbated during overhead & cross-body activities
 - 1° affects middle-aged pts due to degeneration of the fibrocartilaginous disc
 - 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
 - Approx 54-57% of elderly pts exhibit x-ray evidence of degenerative changes in AC, though clinically relevant AC OA is less common
 - Approx 20% of all shoulder px
 - Common in 40 & older pts
 - Types of AC arthritis:**
 - **1° OA:** articular degeneration w/o an apparent underlying cause, often occurring due to constant stress from repeated overhead lifting activities
 - **2° OA:** resulting from associated causes such as post-trauma (prevalent) or underlying disease (e.g. RA)
 - Arthritic Ssx have been observed in Grade I & II sprains of the AC
- **Pathophysiology:**
 - AC is a synovial joint connecting the axial skeleton & scapula
 - Limited ROM characterises the AC
 - Articular connection involves the distal clavicle's convex surface & the acromial facet's slight convex surface
 - Fibrocartilage disc exists between the hyaline cartilage covered facets (akin to knee meniscus)
 - Degenerative changes are part of the natural process
 - 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
 - Occupational Hx, e.g. occupation that requires repeated overhead lifting activities
 - Participation in sports that stress / injure AC, e.g. weightlifting, rugby
 - Complaints of px at night during sleeping on affected shoulder
 - Pt may experience *popping, clicking, grinding*, or *catching* sensation w/ movement of the shoulder
 - 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*
 - 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
 - Localised superior shoulder px is common
 - TTP, possible accompanied w/ swelling due to distal clavicle osteolysis
 - 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)
 - 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

Imaging:

 - Plain film & Dx local anaesthetic local injections are essential Dx tools
 - X-ray & MRI provide comprehensive imaging of AC joint pathology
 - US is effective in detecting signs of AC OA & is commonly used for imaging
 - US-guided injections: +ve if Ssx reduction; -ve if persistent px post-injection suggesting alt shoulder pathologies (commonly rotator cuff injury)



AC osteoarthritis (cont)

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

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Tendinopathies*

"**Tendinopathy** is an umbrella term to describe 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

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

Calcific tendonitis (cont)

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

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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



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GH dislocation (cont)

• Pathophysiology:

Anterior dislocation:

- Up to 97% of shoulder dislocations
- Mechanism: typically a blow + ABD + EXT rot + EXT
- Exam findings: ABD + EXT rot arm, prominent acromion
- Associated injuries: nerve damage, labrum tears, glenoid fossa or humeral head fractures (up to 40%)

Posterior dislocation:

- 2-4% of shoulder dislocations
- Mechanism: hit to the anterior shoulder, axial loading of ADD + INT rot arm
- Exam findings: arm held in ADD + INT rot, inability to EXT rot
- Higher risk of associated injuries: surgical neck or tuberosity #, reverse Hill-Sachs lesions, labrum or rotator cuff injuries

Inferior dislocation (laxation erecta):

- Least common type (less than 1%)
- Mechanism: hyperABD or axial loading on the ABD arm
- Exam findings: arm held above & behind the head, inability to ADD the arm
- 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
- Ask about PREVIOUS dislocations
- Nerves can get stretched out during shoulder dislocation, some pts may report *stinging & numbness* in the arm at the time of dislocation



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GH dislocation (cont)

- **Physical examination:**
 - ROM diminished & painful
 - **Anterior dislocation:** arm ABD & EXT rot; in thin pts potentially prominent humeral head felt anteriorly, & void can be seen posteriorly
 - **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
 - Neurovascular exam (IMPORTANT): axillary nerve injury (40%)
 - **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
 - Fractures of tuberosity & surgical neck may occur
 - *Bankart lesion:* disruption of glenoid labrum, w/ or w/o avulsed bone fragment
 - *Hill-Sachs deformity:* compression # of postero-lateral humeral head 1° w/ anterior dislocations
 - *Reverse Hill-Sachs deformity:* impaction # of antero-medial aspect of humeral head in posterior dislocations
- **Management:**
 - **Posterior shoulder reduction**
 - **Anterior shoulder reduction:**
 - Scapular manipulation: highest success rate
 - EXT rotation technique
 - Cunningham technique
 - Milch technique
 - Stimson technique
 - Traction-countertraction technique
 - Spaso technique
 - FARES technique
 - Fulcrum technique
- **Ddx:**
 - AC injury
 - Bicipital tendonitis
 - Clavicle fracture
 - RC injury
 - Shoulder dislocation
 - Swimmer's shoulder

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GH instability*

• YELLOW

- **Intro:**
 - Includes dislocation & subluxation events
 - Approx 1-2% of general population experience GH dislocation in their lifetime
 - Shoulder instability events are common among young, active, athletic population
 - Anterior shoulder instability accounts for over 95%

- **Aetiology (risk factors):**
 - **Classification criteria:**
 - Uni- or multidirectional instability
 - Traumatic or atraumatic
 - Presence or absence of accompanying soft-tissue hyperlaxity
 - M>F
 - Rugby & football have particularly high incidence rates
 - Anterior labral tears & Hill-Sachs lesions are frequently observed



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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

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GH instability* (cont)

• Physical examination:

Cx exam:

- Rule out Cx radiculopathy in neck or shoulder pathology
- Evaluate neck posturing, muscular symmetry, palpable tenderness, & ROM
- Conduct *Spurling manoeuvre*, myelopathies testing, reflex testing, & neurovascular exam

Shoulder exam:

- Compare B shoulder girdles for asymmetry, muscle bulk, or atrophic changes
- Check for anterior fullness in chronic anterior instability
- Assess scapulothoracic motion & scapular winging
- AROM & PROM, noting limitation in complex instability cases
- Assess axillary nerve function, supraspinatus muscle, & sensory examination

Provocative tests:

- Assess global tissue laxity, GH translation, & hypermobility
- Anterior apprehension test: reproduce Ssx of anterior instability
- Jobe relocation test: alleviate Ssx
- Load & shift test: assess humeral head translation (Grade 1, 2, or 3)

Other exam considerations:

- Check for posterior & multidirectional instability
- Expect associated shoulder pathologies based on age (e.g. RC injuries in older pts)
- Note weakness or px of specific shoulder injuries (e.g. RC tears or Bankart lesions)

• Diagnosis:

- X-rays for comprehensive evaluation
- MRI & CT for advanced imaging

• Complications:

- Redislocation following surgical fixation
- Nerve injuries (esp. axillary n.)
- Infection (surgery)
- 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

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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
 - TrPs: infraspinatus & teres minor

GIRD Dx criteria:

- At least 20° deficit of IR in dominant arm (compared B)
- TTP in posterior shoulder musculature

- **Management:**
 - Target improving shoulder ROM (early focus), reduce muscle stiffness, & increase flexibility
 - Stretching targets tightness in posterior capsule & INT rotators - pecs, biceps, subscapularis, infraspinatus, teres minor, & levator
 - Crossbody stretching may be beneficial
 - After pain-free ROM, follow w/ incremental strengthening of GH & scapular stabilisers
 - TrPs like infraspinatus & teres minor (EXT rotators)
 - SMT - IR & inferior glide
 - Rest from throwing & physical therapy for 6 months

- **Ddx:**
 - Shoulder Impingement s.
 - RC s.
 - Biceps tendinopathy
 - Labral lesion

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Glenolabral articular disruption (GLAD)*

• YELLOW or RED

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



Glenolabral articular disruption (GLAD)* (cont)

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



Glenolabral articular disruption (GLAD)* (cont)

- - Imaging, especially MRA, crucial for Dx
- Diagnosis:**
 - Challenging to detect on non-contrast MRI or CTA
 - Findings: superficial tear to the anterior-inferior labrum w/ an underlying glenoid cartilage defect (from superficial to trans-chondral)
 - MRA demonstrates contrast tracking the labral tear & filling into the chondral defect or under a damaged articular flap
- **Complications:**
 - Linked to episodes of anterior shoulder instability
 - Higher failure rates in arthroscopic Bankart repair w/ GLAD lesions
 - Correlation between GLAD lesions & reduced GH stability
 - GLAD lesions as biomechanical risk factor in shoulder instability by reducing joint concavity depth
 - Risk of OA following GLAD injury (hypothesis)
- **Management:**
 - Conservative: time, NSAIDs, & PT (especially for older pts)
 - Incidental findings on imaging may complicate Dx in older pts due to common age-related cartilage & labral degeneration
 - 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*)
 - Anterior-inferior instability lesions that include a glenoid rim # - *bony Bankart lesions*
 - *Perthes lesion*: labral complex injury, but the labrum is still attached to the glenoid via a periosteal sleeve
 - *Anterior ligamentous periosteal sleeve avulsion*: another labral injury, but it displaces medially on the glenoid neck
 - (*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
 - Characterised by px in neck, shoulder, & hip areas
 - Inflammatory condition w/ elevated erythrocyte sedimentation rate (ESR) & C-reactive protein (CRP)
 - Coexistence w/ or development of Giant cell arteritis (GCA) possible
 - Dx challenges inc. distinguishing PMR from other conditions
 - Does not lead to RA development
- **Aetiology (risk factors):**
 - 100,000 / year
 - White >50
 - Second most common inflammatory autoimmune rheumatic disease (after RA)
 - Etiology not well understood
 - Some genetic predisposition
 - Infection contribute: mycoplasma pneumonia, parvovirus B19, & Epstein-Barr virus (EBV)
 - Some connection between PMR & diverticulitis, suggesting a role for changes in microbiota & chronic bowel inflammation
- **Pathophysiology:**
 - Immune-mediated disorder
 - Elevated inflammatory markers are common
 - 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
 - Morning stiffness: worst in the morning, worsens after rest or inactivity
 - Restricted shoulder ROM: common
 - Upper body complaints: px & stiffness in upper arms, hips, thighs, upper & lower back
 - Rapid onset: Ssx develop within day - 2 weeks
 - Impact on quality of life: px impairs sleep & ADLs, e.g. getting out of bed, showering, driving, etc
 - Inflammation sites: GH & hip joint, subacromial, subdeltoid, & trochanteric bursa
 - Systemic Ssx: fatigue, malaise, anorexia, weight loss, low-grade fever (in some cases)
 - Peripheral involvement: arthritis in 1/4 of pts, carpal tunnel s., distal extremity swelling w/ pitting edema, distal tenosynovitis

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-tendinous 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
 - Premature arteriosclerosis due to chronic inflammation is the probable cause of premature coronary artery disease (CAD)
 - Some increased risk of lymphoplasmacytic lymphoma
 - Higher likelihood of developing inflammatory arthritis (factors: small joint synovitis, younger age, & +ve anti-CCP)
- **Management:**
 - Excellent prognosis w/ prompt Dx & appropriate treatment
 - Medication
 - Vitamin D & calcium supplementation for long-term steroids
 - Pt should be educated on temporal/optic arteritis & how to act
- **Ddx:**
 - RA
 - GCA
 - ANCA related vasculitis
 - Inflammatory myositis & statin-induced myopathy
 - Gout & CPPD
 - Fibromyalgia
 - Overuse or degenerative shoulder pathology (e.g. OA, RC tendinitis & tendon tear, adhesive capsulitis)
 - Cx spin disorders (e.g. OA, radiculopathy)
 - Crown dens s.
 - Hypothyroidism
 - Obstructive sleep apnea
 - Depression
 - Viral infections (e.g. EBV, hepatitis, HIV, parvovirus B19)
 - Systemic bacterial infections, septic arthritis
 - Cancer
 - Diabetes
- **Temporal arteritis (TA):**
 - 1 in 5 pts develop TA
 - Systemic inflammatory vasculitis of arteries
 - Scalp is painful to touch (hair brushing)
 - Prominent, hardened & tender superficial temporal artery
 - HA
 - Claudication masticatoria
 - 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
- **Grade 2 (yellow):** several injured muscle fibres w/ muscle px, tenderness, mild swelling, bruising & loss of strength
- **Grade 3 (red):** complete tear of muscle w/ a possible audible sensation & a total loss of muscle function, severe px, bruising & swelling
- Referral depending on grade

• **Intro:**

- RC injuries range from *tendinopathy* to *complete tears*
- Rotator cuff: subscapularis (INT rotator), supraspinatus (ABductor), infraspinatus (EXT rotator), & teres minor (EXT rotator)

• **Aetiology (risk factors):**

- Most common tendon injury in adults
- Approx 30% of adults >60 have a tear, 62% in those >80
- Age is 1^o factor for RC disease, being degenerative & progressive
- Risk factors: smoking (increases severity), family Hx, poor posture (kyphotic-lordotic, flat-back, swayback), trauma, hypercholesterolemia, & overhead activities
- Partial tears are prone to further propagation, factors inc. tear size, Ssx, location, & age
- Larger tears more likely to deteriorate structurally, w/ actively enlarging tears having higher likelihood of developing Ssx
- 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
- **Micro-trauma** causes tendon degeneration, leading to degenerative tears
- Acute tears are typical in younger pts, while degenerative tears occur in older pts
- Sufficient tendon degeneration can make a complete tear possible w/ less force
- Multiple possible mechanisms: chronic degenerative tear, chronic impingement, acute avulsion injuries, iatrogenic injuries



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Rotator cuff injury* (cont)

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



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Rotator cuff tendinopathy* (cont)

• Pathophysiology:

Acute RC tendonitis can be caused by:

- Direct blows to the shoulder
- Poor throwing mechanics in overhead sports
- FOOSH

Tendinopathy develops from repetitive RC injury, leading to:

- Recurrent pathological cycle
- Acute or chronic tendonitis
- Increasing levels of tendinopathy & tendinosis
- Potential progression to PTTs &/or FTTs

Exact pathogenesis of RC tears is controversial, but likely involves:

- Extrinsic impingement from surrounding structures
- Intrinsic degeneration within the tendon itself

• Clinical presentation:

- Acute RC tendonitis: Hx of trauma or acute exacerbation on a chronic condition
- Chronic RC tendinopathy: either acute on chronic Hx/mechanism or a gradual, atraumatic onset
- Ssx may worsen w/ overhead activities
- Px, especially at night, is common

Thorough exam includes:

- Sports participation (including specific position played)
- Occupational Hx & current status
- Hand dominance
- Hx of shoulder &/or neck injury/trauma
- 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 tendinitis 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)



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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



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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 winging (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 dyskinesia*

• GREEN

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



Scapulothoracic dyskinesia* (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

Scapulohoracic dyskinesia* (cont)

- **Physical examination:**
 - Assess AC & SC for instability
 - Infraspinatus strength test
 - Manual resistance of the arm at 130° of FX (for serratus anterior)
 - Manual resistance of the arm at 130-150° of ABD (for lower & middle traps)
 - Extension of the arm at the side (for rhomboids)
 - Low row test
 - Scapulohumeral rhythm test
 - Quadruped rock
 - Lateral scapular slide test
 - Scapular dyskinesia test
 - SICK scapula sign
- **Diagnosis:**

Classification of dyskinesia types:

 - **Type 1:** inferior angle prominence (i.e. anterior tilt of scapula)
 - **Type 2:** medial border prominence (i.e. winging of the scapula)
 - **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
 - 100% of pts w/ shoulder impingement demonstrate dyskinesia
 - 5% of pts w/ dyskinesia have neurologic injury/damage (spinal accessory, long Tx, suprascapular)
 - SD can occur from core & hip ABD weakness
 - SD becomes more apparent w/ dynamic testing, particularly during the lowering phase of arm movement
 - Recognition & rehab should begin independent of (generally absent) Ssx
- **Management:**
 - STW: upper traps, pec minor, biceps
 - SMT: Cx & Tx
 - Scapular mobs
 - Treatment aims at restoration of scapular retraction, posterior tilt & EXT rot
 - Exercises Phase 1: trap stretch - sitting, YTWL scapular depression
 - Exercises Phase 2: low row, burger w/ band

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Subacromial bursitis

• YELLOW

- **Intro:**
 - Bursa is a fluid-filled sac
 - Lubricates joints & body surfaces prone to wear & friction
 - Subacromial bursa is surrounded by the acromion, coracoid, coracoacromial ligament, & deltoid muscle fibres
 - Inflammation of this bursa can lead to subacromial bursitis
- **Aetiology (risk factors):**
 - Around 0.4% of primary care visits
 - F=M
 - Common aetiologies:**
 - Subacromial impingement (especially in older pts)
 - Repetitive overhead activities / overuse (athletes, factory workers, manual labourers)
 - Direct trauma
 - Crystal deposition
 - Subacromial hemorrhage
 - Infection
 - Autoimmune disease (e.g. RA)
- **Pathophysiology:**
 - Aetiologies can cause inflammation of the subacromial bursa, leading to increased fluid & collagen formation
 - Fluid is often rich in fibrin & can become hemorrhagic
 - Bursitis has 3 phases:**
 - **Acute:** marked by local inflammation w/ thickened synovial fluid, resulting in painful movement, especially w/ overhead activities
 - **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
 - **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)

- **Clinical presentation:**
 - Px in the anterolateral aspect of the shoulder
 - Possible causes: trauma (fall w/ direct impact), repetitive overhead activities (sports, lifting)
 - Impingement s. as a common cause
 - Mechanism: decreased subacromial space due to overhead activities
 - Effect of arm ABD: brings humerus closer to acromion, reducing subacromial space
 - Function of subacromial bursa: protects supraspinatus muscle from wear between humeral head & acromion
 - Result of repetitive activity: irritation & inflammation of the bursa
 - Consideration of tendon pathology: supraspinatus tendinitis or tear may coexist w/ impingement s.
- **Physical examination:**
 - TTP at anterolateral aspect of shoulder below acromion
 - Localised px, doesn't usually radiate (if it does, consider Cx pathology)
 - Warm or boggy skin at site, but no erythema typically
 - Px on resisted ABD of arm beyond 75-80°
 - 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)
 - MRI: bursal fluid accumulation visible
 - US: evaluates the thickness of the bursa
- **Complications:**
 - Not associated w/ many complications
 - Repeated steroid injections: theoretical risk of introducing an infection into skin/joint
 - Risk of damaging RC muscles w/ recurrent injections
- **Management:**
 - Good prognosis for pts w/ conservative care, even w/ surgery
 - Rest, NSAIDs, PT, & corticosteroid injections
 - Surgery for pts non responsive to conservative care
- **Ddx:**
 - Impingement syndrome
 - RC tendinitis/tear
 - Biceps tendinitis
 - Adhesive capsulitis
 - 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
 - Pts may report loss of motion, nighttime px, weakness, & stiffness
 - Onset is gradual over weeks to months, w/o a specific traumatic event
 - Px is typically felt over the lateral acromion w/ radiation to the lateral mid-humerus
 - Inquire: onset, quality, exacerbating factors, interventions tried, & prior injuries
 - **Important:** overhead & repetitive activities
 - Relief: rest, NSAIDs, ice
 - Ssx often return w/ activity
- **Physical examination:**
 - Inspection, palpation, A & PROM, & strength testing of neck & shoulder
 - B comparison
 - Common weakness: ABD &/or EXT rotation
 - *Scapular dyskinesis* during arm forward elevation
 - Tenderness over the coracoid process of affected arm
 - Special tests for shoulder impingement:**
 - Hawkins test - subacromial (external)
 - Neer sign - anterior px = subacromial; posterior px = internal
 - Jobe (empty can test)
 - Painful arc of motion
 - Special tests for shoulder instability:**
 - Sulcus sign
 - Anterior apprehension
 - Relocation test - internal
- **Diagnosis:**
 - Dx made from physical exam
 - Imaging used to confirm & rule out other issues
- **Complications:**
 - Due to structural damage within subacromial space
 - Altered biomechanics
 - Avoidance of use w/ subsequent atrophy
 - 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

[link text](#)

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



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Hypermobility syndromes (HMS) (cont)

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



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

Hypermobility syndromes (HMS) (cont)

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

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