ACUTE DISORDERS OF JOINTS AND BURSAE

MANY MECHANISMS PROVOKE ACUTE JOINT SYMPTOMS → OA, IMMUNE-MEDIATE ARTHRITIS, CRYSTAL ARTHROPATHIES, SERONEGATIVE ARTHRITIS (HLA-B27), VIRAL ARTHRITIS AND BACTERIAL INVASION

THE ABOVE PROCESSES IMPACT JOINT CAPSULES AND SURFACES

SEPTIC ARTHRITIS REFERS TO THE INVASION OF A JOINT BY AN INFECTIOUS AGENT WITH PROLIFERATION AND ASSOCIATED INFLAMMATION

CLINICAL APPROACH TO ACUTE JOINT PAIN

CLINICAL FEATURES AND RISK FACTORS:

- First step in diagnosis is DISTINGUISHING MONOARTICULAR FROM POLYARTICULAR ARTHRITIS AND DETERMINING IF PAIN IS MIGRATORY OR NOT

Table 281-1 Classification of Arthritis by Number of Affected Joints

Number of Joints	Differential Considerations
1 = Monoarthritis	Trauma-induced arthritis
	Nongonococcal septic arthritis
	Gonococcal septic arthritis
	Crystal-induced (gout, pseudogout)
	Osteoarthritis (acute)
	Lyme disease
	Avascular necrosis
	Tumor
2–3 = Oligoarthritis	Lyme disease
	Reactive arthritis (Reiter syndrome)
	Ankylosing spondylitis
	Gonococcal arthritis
	Rheumatic fever
>3 = Polyarthritis	Rheumatoid arthritis
	Systemic lupus erythematosus
	Viral arthritis
	Osteoarthritis (chronic)
	Serum sickness
	Serum sickness–like reactions

Table 281-2 Common Joint Disorders with a Migratory Distribution Pattern

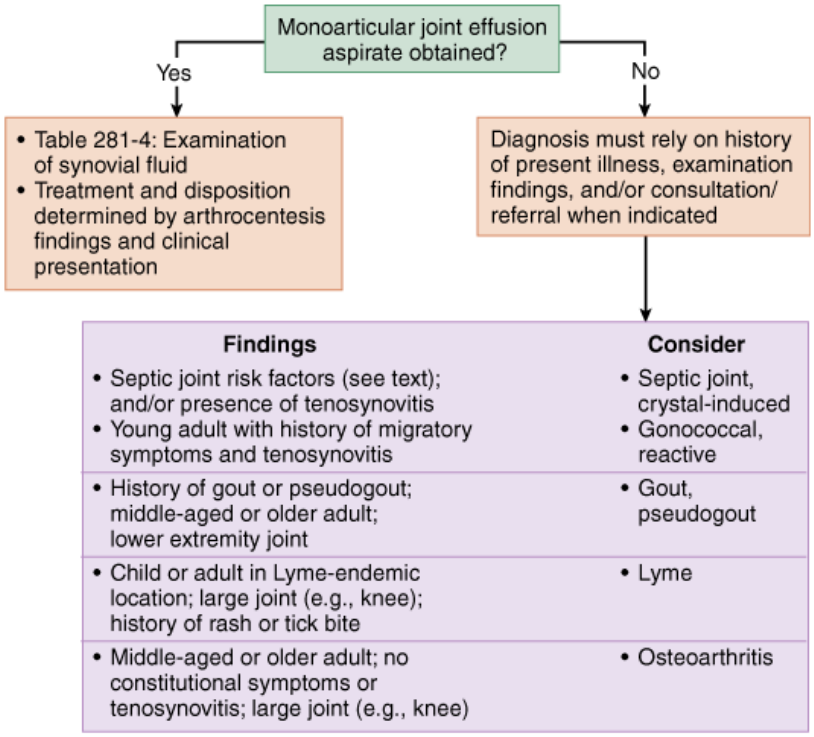
Gonococcal arthritis
Acute rheumatic fever
Lyme disease
Viral arthritis
Systemic lupus erythematosus

- **The most concerning diagnosis of acute joint pain is septic arthritis due to bacterial invasion, so decision making focuses on its exclusion or inclusion**
- The two most important discriminatory diagnoses are septic (gonococcal or nongonococcal) vs crystal-induced arthropathy (gout and pseudogout)
- Risk factors for septic arthritis are shown below:

Table 281-3 Risk Factors for Nongonococcal and Gonococcal Septic Arthritis

Nongonococcal	Gonococcal
Injection drug use*	Menses
Diabetes mellitus*	Pregnancy
Rheumatoid arthritis*	Complement deficiency
Prosthetic joint, knee,* or hip*	HIV infection*
Immunosuppression, HIV*	Systemic lupus erythematosus
Age: >80 y old*	Injection drug use*
Skin ulceration and/or infection*	
Hemophilia	
Hypogammaglobulinemia	
Malignancy	
Hemodialysis	
Liver disease	
Alcoholism	
Steroid therapy	

- **DIAGNOSTIC ALGORITHM:**



SYNOVIAL FLUID ANALYSIS:

- This is the most useful diagnostic tool for evaluation of acute joint pain, the characteristics of which are outlined below
- Joint fluid should be analysed for culture, gram stain, cell count with differential and wet preparation for crystals

Table 281-4 Examination of Synovial Fluid				
	Normal	**Noninflammatory**	**Inflammatory**	**Septic**
Clarity	Transparent	Transparent	Cloudy	Cloudy
Color	Clear	Yellow	Yellow	Yellow
WBC*/microliter	<200	<200–2000	200–50,000	>25,000
PMNs (%)*	<25	<25	>50	>90
Culture	Negative	Negative	Negative	>50% positive
Crystals	None	None	Multiple or none	None†
Associated conditions	—	Osteoarthritis, trauma, rheumatic fever	Gout, pseudogout, spondyloarthropathies, rheumatoid arthritis, Lyme disease, systemic lupus erythematosus	Nongonococcal or gonococcal septic arthritis

SERUM LABORATORY STUDIES:

- None are particularly helpful in establishing a specific diagnosis in adults → e.g. sensitivity of WBC in adults is only ~60%. Blood cultures should be obtained but are only ~20% sensitive
- Some lab studies should be taken for follow up → lyme titre, Rheumatoid factor, ANA, ANCA, HLA-B27, lupus anticoagulant

IMAGING:

- Radiographic evidence of osteomyelitis can follow symptom onset by 7-14 days but can be useful to detect late OM, occult fracture, avascular necrosis or tumour

<u>ARTHROCENTESIS:</u>

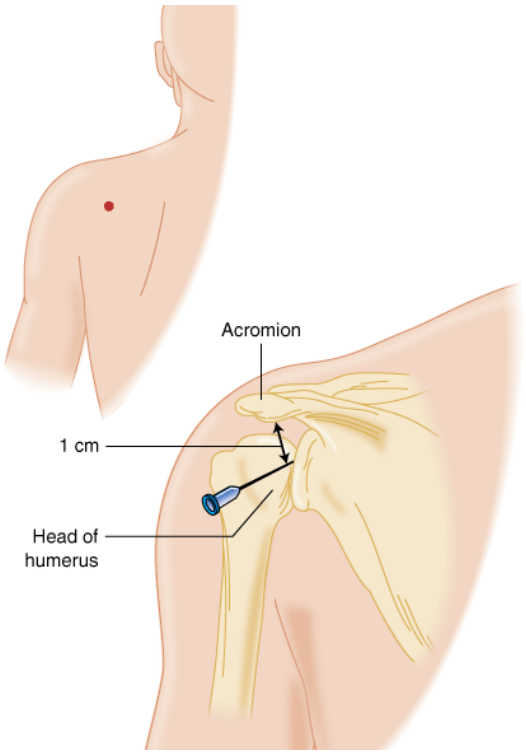
IN PREPARING THE SKIN OVERLYING THE JOINT SPACE TO BE SAMPLED, ENSURE THAT IT IS FREE OF CELLULITIS TO AVOID CONTAMINATION

OTHER RELATIVE CONTRAINDICATIONS:

- Coagulopathy
- Haemarthrosis in haemophiliacs before factor replacement
- Prosthetic joint
    - However, if the index of suspicion is high → still perform arthrocentesis

SHOULDER JOINT ARTHROCENTESIS:

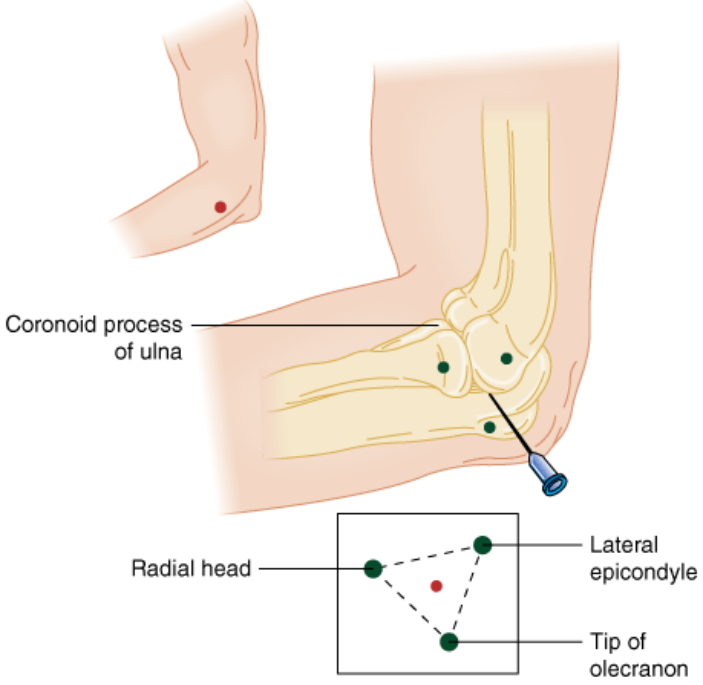
- Posterior approach shown below → find the acromion and identify the posterolateral corner, the point for insertion is 1cm inferior and 1cm medial to the posterolateral corner of the acromion
- Contrast with anterior approach → insert the needle just lateral to the coracoid process, but the posterior approach may be easier



Posterior approach for shoulder arthrocentesis

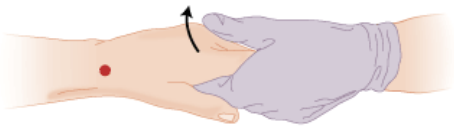
ELBOW JOINT ASPIRATION:

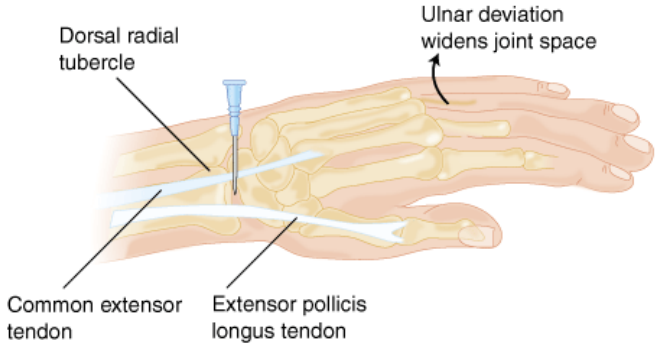
- Use a lateral approach → place in 90 degree flexion, resting on a table with the hand prone to widen the joint space
- Locate radial head, lateral epicondyle of the distal humerus and the lateral aspect of the olecranon → these three landmarks form the ANCONEUS TRIANGLE → the centre of this triangle is the needle entry point



WRIST JOINT ASPIRATION:

- Landmarks are radial tubercle of the distal radius, the anatomic snuffbox, extensor pollicis longus tendon and common extensor tendon of the index finger → insert needle perpendicular to the skin just ULNAR TO THE RADIAL TUBERCLE and the anatomic snuffbox



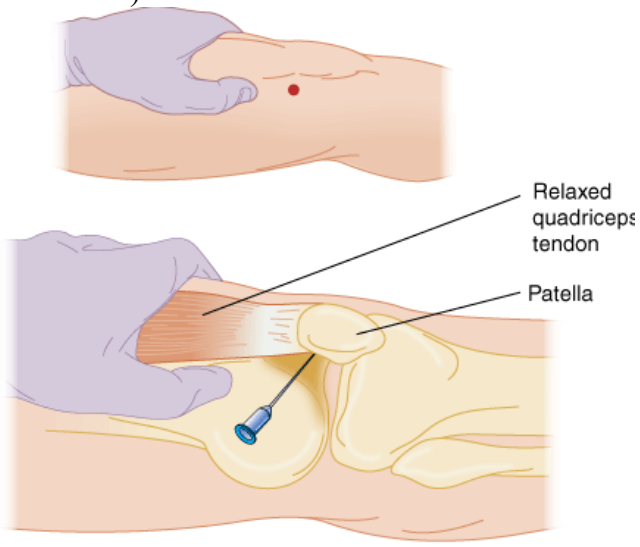


HIP JOINT ASPIRATION:

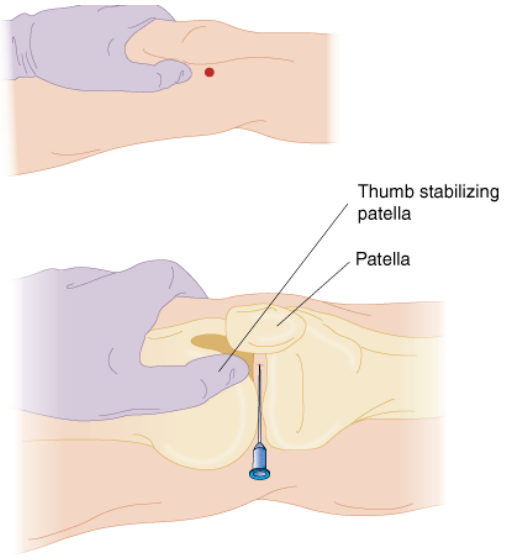
- Performed by anterior or medial approach, but normally performed in conjunction with orthopaedics with view to surgical drainage

KNEE JOINT ASPIRATION:

- Can be entered via medial or lateral to the patella
- Fully extend the knee and make sure quadriceps is relaxed
- Identify the midpoint of the patella → insertion point is 1cm inferior to the patellar edge either lateral or medial to the middle of the patella
    - Direct the needle posterior to patella and horizontal to the joint space (see below)



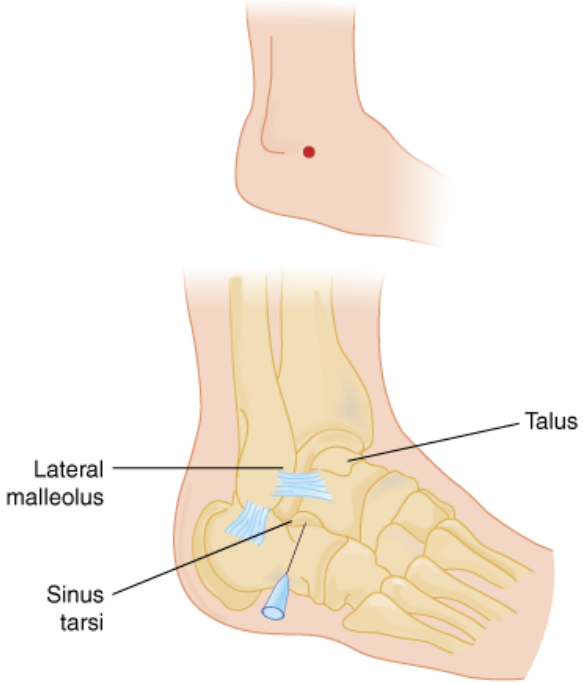
Lateral approach



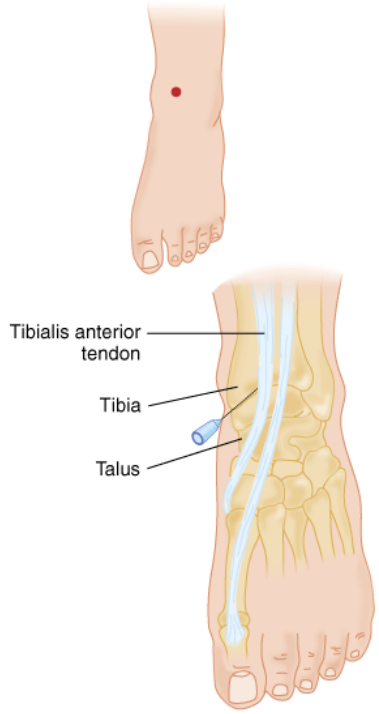
Medial approach

ANKLE JOINT ASPIRATION:

- Performed either at the subtalar approach (lateral) or at the tibiotalar joint (medial)



Lateral → insert needle just below tip of lateral malleolus medially toward joint space



Medial approach → find a sulcus lateral to the medial malleolus and medial to tibialis anterior → plantarflex the foot with the needle entering the skin overlying the sulcus

SEPTIC ARTHRITIS:

- THE MOST IMPORTANT DIAGNOSTIC CONSIDERATION IN ACUTE JOINT PAIN BECAUSE IF IT GOES UNRECOGNISED, THEN IT CAN DESTROY A JOINT IN DAYS
- Risk factors are listed previously
- In infants and children, typical infecting organisms vary with age group, whereas when adolescents become sexually active, they increase their risk for gonococcal arthritis

Table 281-5 Commonly Encountered Organisms in Septic Arthritis in Adolescents and Adults*

Patient/Condition	Expected Organisms	Antibiotic Considerations
Older children and healthy adults, or patients with risk factors for *Neisseria gonorrhoeae*	*Staphylococcus*, *N. gonorrhoeae*, *Streptococcus*, gram-negative bacteria	Vancomycin, 15 milligrams/kg IV load–if Gram stain reveals gram-positive organisms in clusters; ceftriaxone, 1 gram IV, or imipenem, 500 milligrams IV, should be used/added if either gram-negative organisms are present or no organisms present on Gram stain and *N. gonorrhoeae* suspected (also culture urethra, cervix, or anal canal as indicated).
Adults with comorbid disease (rheumatoid arthritis, human immunodeficiency virus, cancer) or injection drug users	*Staphylococcus*, gram-negative bacilli	Vancomycin, 15 milligrams/kg IV load, plus ceftriaxone, 1 gram IV, or ciprofloxacin, 400 milligrams IV; imipenem, 500 milligrams IV, may be used as an alternative agent.
Sickle-cell patients	*Salmonella* (increasingly *Staphylococcus)*	Vancomycin, 15 milligrams/kg IV load, plus ciprofloxacin, 400 milligrams IV; imipenem, 500 milligrams IV, may be used as an alternative agent.

BACTERIAL, NON-GONOCOCCAL SEPTIC ARTHRITIS:

- Non clinical pattern is diagnostic, but general observations are helpful:
  - Joint pain is present in 85% casees
  - Fever in 57%
  - Swelling in 78%
    - These are the only findings that occur in greater than 50% cases
- The involved joint can become exquisitely painful over a few hours
- Resistance to passive and active movement and limitation of full joint movement is a notable finding
- An acute hot, swollen and tender joint (or joints) with restriction of movement is bacterial non-gonococcal septic arthritis UNTIL PROVEN OTHERWISE
- Although joint aspiration and analysis is essential → the commonly quoted cutoff of 50,000 White cells per mm cubed HAS A SENSITIVITY OF ONLY 64%
- If septic arthritis cannot be reliably excluded after clinical evaluation, admit the patient for pain control, IV antibiotics until synovial fluid culture results are available → consult orthopaedics for possible joint irrigation if indicated
- Antibiotic choice is identical to that for osteomyelitis:

For empirical therapy of osteomyelitis, use:

di/flucloxacillin 2 g (child: 50 mg/kg up to 2 g) IV, 6-hourly.

For patients hypersensitive to penicillin (excluding immediate hypersensitivity, see Table 2.2), use:

cephazolin 2 g (child: 50 mg/kg up to 2 g) IV, 8-hourly.

For patients with immediate penicillin hypersensitivity (see Table 2.2), use initially:

vancomycin 1.5 g (child less than 12 years: 30 mg/kg up to 1.5 g) IV, 12-hourly (adjust dosage for renal function and monitor blood concentrations, see Dosing and monitoring of vancomycin; slow infusion required).

GONOCOCCAL SEPTIC ARTHRITIS:

- The most common cause of septic arthritis in adolescents and young adults
- Typically joint infection is preceded by a prodromal phase of migratory arthritis and tenosynovitis predominate before pain and swelling settle on one or more joints → ESPECIALLY THE FINGERS
- Synovial fluid cultures are often negative → only 25-50% of cases yielding positive ID of the organism → recommend culture of posterior pharynx, urethra, cervix and rectum before antibiotic treatment
- Same general principles of management apply, but JOINT DESTRUCTION IS NOT AS PREVALENT, and thus surgical intervention is rarely needed
- THIRD GENERATION CEPHALOSPORIN IS AGENT OF CHOICE

CRYSTAL-INDUCED SYNOVITIS (GOUT AND PSEUDOGOUT):

- Primarily an illness of middle-aged and elderly adults
- URIC ACID (gout) and CALCIUM PYROPHOSPHATE (pseudogout) are the two most common crystalline agents
- Classic description is monoarthritis involving the great toe or knee joint in a man >40

    - Women are spared onset of gout until older age, but are MORE PRONE TO POLYARTICULAR INVOLVEMENT
- Joint pain develops over hours → often following trauma, surgery, significant illness or change in medication
- Gout results from precipitation of uric acid crystals in joints (pseudogout from calcium pyrophosphate) → no joint is the exclusive site of involvement for either crystal
    - DIAGNOSIS IS BY SYNOVIAL FLUID ANALYSIS under a light microscope → NEGATIVE BIREFRINGENCE for uric acid, POSITIVE BIREFRINGENCE for calcium pyrophosphate
- Serum uric acid levels generally NOT USEFUL → up to 30% of people with gout will have normal levels during acute attacks
- WBC on joint aspirate is frequent → but absence of bacteria, presence of crystals and dramatic response to NSAIDs clarify the diagnosis
    - BEWARE → concordant septic joint can exist but is uncommon → if in doubt, IV antibiotics and admit
- For patients with normal renal function → standard of care is NSAIDS → indomethacin 50mg tds for three days
- DO NOT GIVE NSAIDS TO PATIENTS WITH RENAL INSUFFICIENCY
    - Colchicine (0.5mg bd) in patients with normal renal and hepatic function → risk of bone marrow suppression, neuropathy, myopathy and death
- PROPHYLAXIS → allopurinol or probenicid for gout, none available for pseudogout

Acute gout

Initially, gout may subside spontaneously in less than a week, but the patient will usually seek help. The acute treatment should focus on reducing inflammation and usually has no effect on modifying the plasma urate concentration. Nonsteroidal anti-inflammatory drugs (NSAIDs) at the maximum recommended doses reduce inflammation and pain relatively quickly. An attack will be aborted using:

1 **an NSAID orally, at the upper dosing range until symptoms abate (typically 3 to 5 days), then reduce the dose until signs of joint inflammation have abated, and then cease (see Table 12.1)**

OR

2 **colchicine 500 micrograms orally, 6- to 8-hourly until the attack has abated (maximum 6 mg over 4 days) and then cease. Higher doses are not justified; adjust dose in renal impairment (see Colchicine)**

OR

2 **prednis(ol)one 15 to 20 mg orally, daily until symptoms abate (typically 3 to 5 days) and then cease.**

VIRAL ARTHRITIS:

- **Most common causes of viral arthritis are PARVOVIRUS B-19, RUBELLA AND HEPATITIS B**

Table 281-6 Common Causes of Viral Arthritis				
Virus	**Prevalence of Arthritis**	**Findings**	**Duration**	**Additional Features**
Parvovirus B19	Children 10% Adults 50%–70%	Polyarticular Symmetric	2–8 wk or chronic	Causes erythema infectiosum in children, rarely causes aplastic crisis
Rubella	Adults 50%	Polyarticular	5–7 d	Relapse
Epstein-Barr virus	1%–5%	Poly or monoarticular	1–12 wk	Autoantibodies
Hepatitis B	10%–25%	Migratory	1–3 wk	Vasculitis
Hepatitis C	10%	Polyarticular	Chronic	Vasculitis
HIV	10%–50%	Mono- or oligoarticular	Chronic	Viral load >10,000 copies of HIV RNA, CD4 count <350 cells
Alphaviruses*	>50%	Oligoarticular	1–4 wk	More common in Asia, Africa; fever, myalgias

LYME DISEASE:

- Rare in Australia, suspect if there is a recent visit to an endemic area with history of tick bite
- Large joints preferentially
- Treat with doxycycline, penicillin G, amoxicillin or ceftriaxone

HAEMARTHROSIS:

- **SPONTANEOUS:**
  - Indicates underlying systemic illness and should prompt consideration for primary or secondary coagulopathies → HAEMOPHILIACS SHOULD RECEIVE FACTOR REPLACEMENT AS TREATMENT FOR AND PRIOR TO JOINT ASPIRATION
- **TRAUMATIC:**
  - High association with ligamentous injury or intra-articular fracture
  - Treatment → immobilisation, ice and elevation with appropriate follow up for ligamentous and articular injuries

RHEUMATOID ARTHRITIS:

- Typically a progressive disease, with polyarticular involvement of symmetric joints and sparing of DIPJ → women 3-4x more likely to be affected
- Salicylates or other NSAIDs are cornerstone of treatment
  - Steroids for brief periods
  - Long-term agent s→ methotrexate, leflunomide, antimalarials, biologic agents
- Consider septic arthritis in those with acute episode of arthritis in those who are also receiving immunosuppressive agents

OSTEOARTHRITIS:

- Distinguished from RA by lack of constitutional symptoms and/or multisystem involvement
- May involve DIPJ
- Treatemt is rest of affected joints, NSAIDS/paracetamol
- No role for steroids

- Orthopaedic referral in longer term for joint replacement

REACTIVE ARTHRITIS:

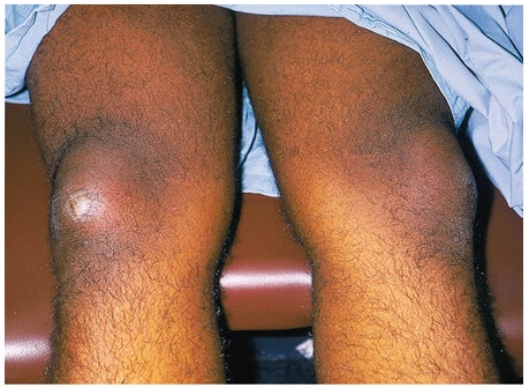
- Formerly known as REITER SYNDROME → a seronegative spondyloarthropathy characterised by acute, asymmetric oligoarthritis occurring 2-6 weeks after an infectious illness
- CLASSIC TRIAD NOT NEEDED FOR DIAGNOSIS BUT INCLUDES:
    - Arthritis
    - Urethritis
    - Conjunctivitis
- Common inciting agents → Chlamydia, ureaplasma, or post-dysentery (Salmonella, Shigella, Yersinia, Campylobacter)
- Joint involvement typically involves lower extremities → heels and feet in 70%

ANKYLOSING SPONDYLITIS:

- Another of the seronegative spondyloarthropathies → arthritic predilection for spine and pelvis
- Rheumatoid factor negative
- Suspected in individuals <40 who note insidious onset of symptoms that improve with exercise, are associated with morning stiffness and last >3 months
- Classic x-ray → bamboo spine (squaring of vertebral bodies)
- Pain control with NSAIDS
- BIOLOGIC DMARDS playing increasing role

BURSITIS:

- **NON-SEPTIC BURSITIS:**
    - An inflammatory process involving one of the >150 bursae in the body
        - Most commonly overlying elbow or knee
    - Affected bursa is easily palpable but not significantly tender and is not erythematous
    - If it is acute → consider septic bursitis
    - NO LIMITATION OF OR PAIN UPON JOINT MOVEMENT
    - Treatment is NSAIDs
- **SEPTIC BURSITIS:**
    - Characterised by acute pain, tenderness, erythema of the affected bursa with overlying warmth
    - Most common sites → prepatellar bursa (50-53%) and olecranon bursa (40-45%



- Fever in <50%
- Pain can be mild but is often severe
- BURSAL ASPIRATION CAN BE BOTH DIAGNOSTIC AND THERAPEUTIC

Table 281-7 Characteristics of Bursal Fluid in Patients with Septic and Nonseptic Olecranon and Prepatellar Bursitis

	Septic	Traumatic and Idiopathic	Crystal Induced
Appearance	Purulent; may be straw colored or serosanguineous	Straw colored, serosanguineous, or bloody	Straw colored to bloody
Leukocytes/microliter	1500–300,000; mean, 75,000, typically >30,000	50–11,000; mean 1100, typically <28,000	1000–6000; mean, 2900
Differential count	Predominantly polymorphonuclear leukocytes	Predominantly mononuclear	Highly variable
Ratio bursal fluid to serum glucose	<50% in 90% of cases	>50%, 70%–80% in 98% of cases	Unknown
Gram stain	Positive in 70%	Negative	Negative
Crystals present	No*	No	Yes
Culture results	Positive	Negative	Negative

- Majority of infections due to STAPHYLOCOCCUS AUREUS, but S epidermidis and Strep species also encountered
- Generally responds to PO antibiotics (CLINDAMICIN OR CEPHALEXIN, FLUCLOXACILLIN)
- CONSIDER IV ANTIOBTICS AND I&D if:
  - Extensive purulent bursitis
  - Extensive surrounding cellulitis
  - Suspected joint involvement
  - Immunocompromise
  - Failure to respond to a course of oral antibiotics

di/flucloxacillin 2 g (child: 50 mg/kg up to 2 g) IV, 6-hourly.

For patients hypersensitive to penicillin (excluding immediate hypersensitivity, see Table 2.2), use:

cephazolin 2 g (child: 50 mg/kg up to 2 g) IV, 8-hourly.

For patients with immediate penicillin hypersensitivity (see Table 2.2), use initially:

vancomycin 1.5 g (child less than 12 years: 30 mg/kg up to 1.5 g) IV, 12-hourly (adjust dosage for renal function and monitor blood concentrations, see Dosing and monitoring of vancomycin; slow infusion required).

Adjust therapy according to culture and susceptibility results.