Musculoskeletal (MSK) Problems: Principles of Management

MSK symptoms
Pain – site, radiation (get radiation distally, more common with more proximal joints e.g. shoulder and knee), quality (shooting with nerve entrapment). Onset. With usage (mechanical), at rest (inflammatory) or at night (if no relation to movement concern about bony mets).
Bony pain – well localised, doesn’t radiate, progressive, night predominant, not related to movement, due to fractures, neoplasia, Paget’s disease, osteomalacia, osteomyelitis, osteonecrosis.
Stress pain – in tight pack positions, if in all directions suggests synovitis.
Stiffness – prolonged early morning and inactivity imply inflammatory cause.
Swelling and deformity.
Instability
Locking – sudden inability to move joint, usually painful but transient, post unlocking discomfort.
Affects hinge joints (knee, elbow, TMJ). Mechanical problem e.g. synovium, cartilage, split meniscus.
Loss of function, disability and handicap.
Systemic illness – night sweats, malaise, fatigue, weight loss suggest inflammatory process.
Sleep disturbance.

Red flags: progressive, well localised progressive night-predominant pain (mets), red hot joint (sepsis), sequential joint involvement (spreading sepsis).

History – need to determine if traumatic or non-traumatic, ask about dominant hand.
Trauma – time, place, mechanism (twist, blow, crush), magnitude of force, direction of force, first aid, immediate symptoms and progression, previous injuries.
Non-trauma – rule out (RESTIT) Referred pain, Embolus, Sepsis, Tumour, Ischaemia, Thrombosis.
Systems enquiry for non MSK causes, associated features, drug toxicity, pre-op assessment and involvement of other organs. Especially general malaise (anaemia), hot and shivery (infection), skin (rashes, psoriasis, Raynaud’s), eyes (iritis, scleritis).

MSK signs
Attitude – hold joints in loose-pack position so capsule minimally stretched and can accommodate oedema.
Deformity – correctable or fixed, can be due to soft tissue (scarring, swelling or overgrowth), bone or joints. Malalignment, varus (distal part medially), valgus (distal part laterally), fixed flexion, subluxation (slip from normal alignment but still in contact), dislocation.
Skin changes – red with sepsis, crystals, also rheumatism, reactive arthropathy. Psoriasis.
Muscle bulk and strength.
Swelling – can be fluid (effusion, haemarthrosis if very quick after trauma), soft tissue or bone. If moves with tendons (tuck sign) implies tenosynovitis.
Nodules – rheumatoid nodules, gout tophi, hyperlipidaemia xanthomata (also lupus, rheumatic fever, histiocytosis, polyarteritis nodosa, sarcoidosis).
Signs of effusion – small bulge sign, moderate - , large – balloon sign.
Tenderness.
Range of movement – restricted, hypermobile. Stress pain is increasing pain towards extremes of movements – if universal then synovitis, if selective then localised lesion in or around joint. May be most painful in tight pack position – capsule at tightest so can’t accommodate pressure rise. Loss of movement may due to loss of muscle power, damage to joint, reflex inhibition of muscle, protective muscle spasm.
Creptitus – fine is due to tendon sheath or bursa, coarse conducts through bone and can be heard.
Warmth.
Stability.
Differentiating arthropathy and periarticular lesion:

**Arthropathy** – joint line tenderness, active and passive movement equally tender and restricted, capsular swelling, diffuse warmth, coarse crepitus, pain in several directions of movement.

**Periarticular lesion** – periarticular tenderness, active movement more painful and restricted than passive, pain with resisted active movement, localised swelling and warmth, fine crepitus, usually pain only with one direction of use. Divide into single regional (injury, overuse) or multiple regional (fibromyalgia).

Differentiating synovitis and inflammation from mechanical joint damage:

**Synovitis** – lots of stiffness, warmth, stress pain, soft tissue swelling, large effusion. No crepitus, deformity or instability.

**Joint damage** – no or minimal stiffness, warmth, stress pain, soft tissue swelling and effusion. Crepitus, deformity and instability.

**Investigations**
Aim to support diagnosis or monitor disease.
Screening blood tests – FBC, U&Es (renal involvement, drugs).

**Inflammatory markers**
**ESR** - normal 0-10 in males, 0-20 in females but increases with age.
**CRP** – quickest to change.

**Synovial fluid analysis**
**Indications** – to diagnose acute bacterial sepsis and crystal associated disease if acute monoarthritis. Both of these are curable.
To confirm a joint is not infected and differentiate inflammatory (raised cell count) and non-inflammatory effusions.
If suspect sepsis then need to do gram stain and culture. If suspect crystals do compensated polarised light microscopy.

**Normal synovial fluid** – low volume, low cell count, clear, straw coloured, high viscosity, GAGs.
Synovial fluid in inflammation – becomes opaque (neutrophils), becomes less viscous, increased volume.

**Blood stained** – traumatic aspiration (non-uniform), haemarthritis (uniform – due to trauma, severe inflammation, bleeding diathesis, rare tumours, subchondral fracture if lipid layer on surface).

**Pus** (pyoarthrosis) – very high concentration of neutrophils due to sepsis or crystals.

**Synovial biopsy**
To diagnose chronic monoarthritis.
Can see chronic infection e.g. TB, foreign body, sarcoidosis, amyloid.

**Anaemia**
**Chronic disease** – normochromic, normocytic.
**Occult bleeding** – NSAID treatment, increased TIBC.
**Bone marrow suppression** – methotrexate.
**Haemolytic** – SLE.
Remember ferritin is an acute phase protein so increased if inflammation.

**Autoantibodies**
**Rheumatoid factor:** Positive in 70% RA. Usually higher titres with more severe disease, nodules, vasculitis and lung disease. Also in lupus, scleroderma, other CTDs, chronic infection, viruses (rubella, CMV), parasites, health people.
Antinuclear factor:
Positive in 100% SLE – good screening test but poor specificity.
Also in RA, Felty’s, scleroderma, autoimmune thyroid disease, liver disease, juvenile chronic arthritis, normal subjects.
Includes various subgroups. Anti double-stranded DNA is more specific for lupus and a poor prognostic factor. Anti-histone with drug induced lupus. Also Ro, La, Sm, Scl 70 and centromere.

ANCA:
Antineutrophil cytoplasmic antibody.
cANCA – specific to Wegener’s.
pANCA – less specific, other vasculitides.

Antiphospholipid:
In antiphospholipid syndrome.

CPK
Elevated with inflammatory myopathy, vasculitis, muscular dystrophy, MND, alcohol, drugs, trauma, strenuous exercise, MI, hypothyroidism, metabolic myopathy.

Imaging
Xrays – bone, fractures.
CT – bony structures, complex bones e.g. ankle.
MRI – discs in spine, cord compression, knee menisci and ligaments.
Isotope bone scanning – show metastatic bone disease, not myeloma.
DEXA – bone density.
Bone biopsy to diagnose osteomalacia, renal osteodystrophy, osteoporosis in young pts.

Arthroscopy
Especially for knee.
Evaluate mechanical problems e.g. menisci, take synovial biopsy (esp if concerned about TB), diagnose septic arthritis.

Nerve conduction studies
Have orthodromic (same way as nerve) and antidromic (opposite way) conduction.
Give stimulus and look for when it reaches probe. Do from proximal to carpal tunnel and distal to carpal tunnel. Look for drop in velocity as go through carpal tunnel/cubital tunnel etc.
Need to lose most of fibres before slowing apparent.

Management
To educate patient, treat pain, restore function and modify disease process.

Specific medical or surgical treatment - to relieve pain, correct deformities, prevent arthritis, allow mobilisation.
Pain relief and symptoms control - need to do this for humane reasons and also to allow rehabilitation and movement.
Rehabilitation.

Types of surgery – arthroplasty (new joint), excision arthroplasty (remove all or part of joint), arthrodensis (stiffening of joint), arthroscopy (opening joints, usually for access for arthroscopy), osteotomy (cutting bone), fixation of bone, soft tissue release (tight tendons, soft tissue contracture, decompression of nerve or muscle), synovectomy (may help with pain relief), tendon transfers or repairs, amputation.
Main aim of surgery is pain relief. This may lead to improvements in function and quality of life.
Management of pain
Education, exercise, reduce adverse mechanical factors, weight reduction, simple analgesia.
If these fail – other analgesia, DMARDs, corticosteroids, local injections, heat and cold, surgery, coping strategies.

Analgesics
Conventional analgesics.
Capsaicin – chilli extract, initially stimulates nociceptive receptors (discharge of substance P) then leads to a reduction in nociceptive responses. Can be used to treat neuropathic pain.
NSAID creams and gels – can penetrate to superficial tissues and capsule.
Amitriptyline – for neuropathic pain. Side effects - sedation and atropine-like effects.

Corticosteroids
Rapid potent anti-inflammatory action. Not clear whether they have disease-modifying effects.
Dose and duration dependent side effects so use smallest dose for shortest time.

Systemic
Indications – rapid, short term control of flares of disease while waiting effect of slow-acting antirheumatic drug, life or organ threatening inflammatory disease e.g. CTD or vasculitis, primary treatment of polymyalgia rheumatica, control of inflammatory disease during pregnancy.

Oral – mainly prednisolone, give in morning to try and avoid suppression of HPA axis. Start from 10 up to 60mg. Aim for maintenance dose of 7.5g or less. Need to increase dose if ill.
IV – hydrocortisone, short-term control.
IM – methylprednisolone, can repeat at intervals to avoid some from problems of oral. Risk of scarring at injection site.

Local
Intraarticular injection – long acting steroid. Useful if only one joint flaring up. Pain relief for arthritis for about 2m, but may get post injection flare. Risk of septic arthritis. If repeated problems with joint tissue atrophy and Cushing’s syndrome.
Periarticular injection – rapid pain relief (does not speed healing) for bursitis, tenosynovitis and enthesopathy. Injecting near to tendons has risk of rupture.
Nerve blocks – steroid and LA, control resistant pain.

Side effects
Redistribution of fat – buffalo hump, moon face, truncal obesity.
Metabolic – hypertension, increased appetite, muscle wasting and proximal myopathy, hyperglycaemia, susceptibility to infection (esp chicken pox, check immunity), adrenal atrophy and suppression.
Bone – avascular necrosis, osteoporosis (need bone protection with bisphosphonates if in long term steroids), pathological fractures.
Skin – thinning, bruising, poor healing, striae, cataracts, acne, plethora.
Psychological – euphoria, depression, psychosis.

With intra-articular or peri-articular routes – haemarthrosis, flare, iatrogenic infection, joint tissue, skin and fat atrophy, tendon rupture.

Interactions:
Rate of metabolism increased (efficacy decreased) by – carbamazepine, phenytoin, rifampicin.
Reduce efficacy of hypoglycaemics and antihypertensives.
Non-pharmacological interventions

**Education**
Reduction and disability as well as health care costs.
By one to one discussion, written literature, group classes etc.

**Exercise**

**Aerobic fitness** – long term reduction, encourages restorative sleep, benefits common comorbidities.
**Local strengthening exercises** – improve muscle strength, proprioception, coordination and balance.

**Avoidance of triggers**
Stop excessive impact loading or adverse repetitive use e.g. pacing of activities.
Shock-absorbing footwear, walking stick on contralateral side.
Weight loss – aggravates pain as mechanical strain, speeds progression of joint damage in arthritis.

**Physical treatments**

**Treatments to relax muscles** – heat, cold, hydrotherapy.
**Splints** – temporary rest and support, maintain posture.
**Orthoses** – more permanent appliances to reduce instability e.g. working wrist splints. Esp in severely disabled patients who are unsuitable for surgery.

**Coping with chronic pain**
Reassure patient and provide explanation for pain.
Increase self management.
Recognise negative aspects of psyche and change attitude and behaviour e.g. relaxation techniques, avoid stress, alter beliefs, reduce maladaptive pain behaviour etc.

**Nutri-pharmaceuticals**
A wide variety of compounds are available e.g. normal constituents of cartilage (glucosamine sulphate, chondroitin sulphate) or trace elements.
Limited evidence from clinical trials, may produce some pain relief in knee OA and slow further structural damage.

**Periarticular Pain**

**Overuse and strain injuries**

**Symptoms and signs**
Typically acute onset, relevant predisposing trauma, single regional pain, non-progressive, only with one or a few movements. Localised tenderness, pain reproduced by resisted active movements and stress tests. No or mild signs of inflammation, no systemic upset.

**Risk factors** – age, obesity, hypermobility, occupational and recreational usage.

**Bursae** – hollow sacs lined with synovium that contain synovial fluid and help structs move smoothly in relation to each other.
**Adventitious bursa** – new bursa arising over stressed area.

**General management** – avoid predisposing factors, pain relief, exercise and rehabilitation.
Muscle injuries resolve over days but tendons and ligaments may take much longer.

**Lateral or medial epicondylitis**
**Lateral** = tennis, extensors, most common.
**Medial** = golfers, flexors.

**Symptoms and signs** - poorly localised distally radiating pain, which is worse with use. Tenderness along enthesis, exacerbated by resisted wrist flexion (golfer’s) or extension (tennis).
Management – NSAIDs, physio, elbow splint, injection of LA and steroid, surgical release.

Olecranon bursitis – fluctuant tender swelling over olecranon. Can be due to trauma, also infection, gout and RA.

De Quervain’s tenosynovitis
Tenosynovitis = inflammation of tendon sheaths. Mostly occurs around wrist. With RA get it with extensor tendons causing tendon rupture.

deQuervain’s tenosynovitis – abductor pollicis longus and extensor pollicis brevis. Often due to overuse, also gonococcal infection. Get pain around radial styloid and tendon sheath. Forced ulnar deviation causes pain (Finklestein’s test).
Management – wrist splints, injection of LA and long acting steroids, surgical decompression.

Shoulder conditions
Rotator cuff tear or strain:
Usually older people due to degeneration (commonly found on post-mortem), especially supraspinatus and posterior muscles. Get upper arm pain, tends to have acute or subacute onset, be non progressive and related to movement. Middle arc pain and pain on resisted active movement. Best imaged with MRI. Manage with physio or surgical repair of cuff.

Supraspinatus tendonitis:
Middle arc pain and pain on resisted active movement. Manage by LA and steroid injection to subacromial bursa.

Subacromial bursitis:
Middle arc pain, not aggravated by resisted active movements. Rarely communicates with subdeltoid bursa. Diffuse pain felt over deltoid and biceps. Manage by LA and steroid injection to subacromial bursa.

Impingement:
Osteophyte compresses subacromial structures, with narrowing of subacromial space. May see on xray. Manage by LA and steroid injection to subacromial bursa. Can surgically remove undersurface of acromion (a “subacromial decompression”).

Adhesive capsulitis (true frozen shoulder):
Especially middle aged females. More common in diabetics, can be triggered by rotator cuff lesion, local trauma or MI. Acute fibrosis causes stiffness, pain on all movements and loss of range of movement. Characteristically there is marked limitation of external rotation. May occur following immobilisation. Typically lasts for 18 months- 6 months where pain is main symptom, 6 months of stiffness and 6 months of recovery.
Treat with reassurance and observation if patient can cope with limitations. Intra-articular steroids may improve degree of pain, but little effect on range of movement.
MUA with physio follow-up has been main surgical treatment. Alternatively, arthroscopy with division of the coraco-humeral ligament is increasingly used.

Trochanteric bursitis and gluteal enthesopathy
Pain over greater trochanter. Worse with activity or lying on that side.
Often older obese women, can be secondary to abnormal gait.
With gluteal enthesopathy get pain on resisted active hip abduction.
Pre- or infra- patellar bursitis and anserine bursitis

**Pre-patella bursitis** – pain over ant patella and tender fluctuant swelling in front of patella, worse on kneeling.

**Infrapatellar bursitis** – ant inf knee pain, tender fluctuant swelling in front of (superficial) or behind (deep) patella tendon.

**Anserine bursitis** – pain over upper medial tibia.

Plantar fasciitis and subcalcaneal bursitis

Heel pain, especially on walking. Enthesopathy of plantar fascia attachment to calcaneus (see calcaneal spur on xray).

Can be associated with sero- arthritis.

**Management** – padded soles, physio, steroid injections.

Achilles tendinitis and enthesopathy

Pain, swelling and tenderness over tendon or tendon insertion. Pain exacerbated by passive dorsiflexion, resisted plantar flexion, squeezing tendon and standing on tiptoe.

Due to overuse or generalised inflammatory conditions.

**Management** – rest, NSAIDs, physio.

Carpal tunnel syndrome

**Pathology** - median nerve compressed in carpal tunnel, distal to wrist crease. This runs under transverse carpal ligament and over carpal bones.

Very common condition, esp in middle aged and older women.

**Aetiology** - mostly idiopathic, may be associated with DM, hypothyroidism, trauma (esp wrist fracture), RA, pregnancy.

**Clinical features** – pain and altered sensation in distribution of median n. (not palm as palmar branch comes off before tunnel) often worse in early morning and wakes patient. Thenar muscle wasting. Can provoke symptoms by Phalen’s (prolonged wrist flexion) and Tinel’s (tap proximal to transverse carpal ligament) tests.

**Investigations** – nerve conduction studies.

**Management** – surgery to divide transverse carpal ligament and decompress tunnel. Can do under local, good results. May try wrist splinting and steroid injection first.

Ulnar nerve problems

**Pathology** - compression in cubital tunnel around elbow. Ulnar nerve passes behind medial epicondyle and between heads of flexor carpi ulnaris. Can also be entrapped at wrist in canal of Guyon.

Mostly idiopathic but can be secondary to malunited fracture.

**Clinical features** - get pain at medial epicondyle radiating down to ulnar distribution in hand, worse with elbow flexed, and symptoms reproduced by pressure over condyle. Can get ulnar n motor weakness.

**Ulnar claw hand** – hyperextension of MCP and flexion of DIP and PIP, due to failure of intrinsic muscles. Initially passively correctable then becomes fixed.

**Management** - investigate with nerve conduction studies.

Can do surgery to decompress.
Ganglion
Pathology – derived from capsule of joints or tendon sheaths – small cysts filled with viscous fluid. Most common around wrist. Not derived from synovial tissue.
Clinical features – fluctuant transilluminating swellings. Can be painful or uncomfortable.

Dupytren’s contracture
Common disorder in palmar fascia – get fibrosis and contraction.

Aetiology - associated with liver disease, diabetes, anti-convulsants. Often family history. More common in men, equal sex ratio after menopause. Associated conditions can affect side of foot (Lederhose’s) or penis (Peyronie’s).

Clinical features - get palpable nodule in palm or finger, fibrous band causes retraction of fingers (typically MCP and PIP of little and ring) so fixed flexion (table-top test, can’t get hand flat). This can cause problems with function, catching fingers and appearance.


Erythema nodosum
Acute inflammatory skin lesion – tender nodules on anterior lower leg. Can cause acute arthropathy, treat with rest and NSAIDs.

Causes:
Infection – strep, TB, fungal.
Drugs – sulphonamides, OCP.
Conditions – sarcoidosis, lymphoma, Behcet’s, IBD.

Causes of clubbing
Respiratory – chronic suppurative lung disease, malignancy, fibrosing alveolitis.
Cardiac – endocarditis, cyanotic heart disease.
Abdominal – celiac, IBD, cirrhoses.
Idiopathic – can be familial.
Back and Neck Pain

**Differential diagnosis**
Frequently has no definable pathology.
Other conditions can cause referred pain to the back e.g. peptic ulcer, pancreas, kidney, aortic aneurysm, endometriosis.

**MSK back pain** – can be mechanical (inc root entrapment), inflammatory or destructive.

**Pancreato-biliary disease** - sudden onset, epigastric or hypochondrial pain radiating to back, diminishing after 72 hours, vomiting, jaundice, fever.

**Dissecting aortic aneurysm** - severe and central chest pain radiating through to the back, associated with hemiplegia, unequal arm pulses and BP, paraplegia and anuria.

**Back pain**
Significant health problem in developed countries – most common reason for disability and missed work. >70% prevalence.

>95% is simple mechanical back pain. Mostly neck and lumbar spine. 5% associate with nerve root pain. <2% has serious spinal pathology.

**Risk factors for becoming chronic** - previous episodes, widespread pain, long duration, radiating leg pain, restrictions of movement.

**MSK back pain history**
Mainly to identify serious or specific disorders. Age, occupation.

**Pain** – timing, history of injury, precipitating and relieving factors, diffuse or well localised, site, radiation, nature of pain.

**Worse with flexion** – disc prolapse, annular tear.

**Worse with extension and rotation** – facet joint dysfunction, spondylolysis. Exacerbated by walking or inactivity.

**Acute onset** – prolapsed disc, vertebral crush fracture.

**Other symptoms** – paraesthesia, motor disturbance, bladder and bowel symptoms, systemic symptoms.

**General enquiry** – esp for features of malignancy or infection.

**Red flags** – age <20 or >55, trauma, constant non-mechanical progressive pain (esp night pain), progressive weakness, thoracic pain, structural deformity, previous history (cancer, steroids, drug abuse, HIV, TB, immune suppression), signs of cauda equina syndrome (difficulty micturating, faecal incontinence, saddle anaesthesia, progressive motor and sensory disturbance), signs of systemic upset (night sweats, malaise, weight loss).

**Waddell's inappropriate signs of lumbar spine pain** – suggest non-organic pain. Ability to sit up on couch with legs straight but limited straight leg raise, superficial tenderness, tenderness on rotating trunk by rotating pelvis not lumbar spine, axial loading of spine by pressing on head.

**Psychological factors** – important for developing back pain and for it becoming chronic.

**Aetiology**

**Mechanical** – sudden onset, eased by rest, unilateral symptoms, no obvious root distribution, doesn’t radiate past buttock or thigh, worse with lifting, bending and coughing, previous episodes, general good health. Includes trauma, degenerative (spondylosis, spondylolysis, spondylolisthesis, spinal stenosis), facet joint arthritis (changes of OA) or dysfunction (synovial joint, post to spine, loaded in extension), disc prolapse or annular tear (disc is fibrocartilagenous joint, pain on forward flexion), nerve root compression.
Inflammatory – spondyloarthropathies, sacroiliitis. Spondylitis pain is usually axial and symmetrical.

Infective – discitis, osteomyelitis, paraspinal abscess.

Bone disorders – osteoporotic fracture, Paget’s disease, osteomalacia, hyperparathyroidism.

Neoplastic – metastases, multiple myeloma, primary tumour.

Referred pain – neck from shoulder, IHD, oesophagus, pleura, diaphragm, meningitis. Low back from hip, pancreatitis, Ca colon, renal calculus, endometriosis, AAA.

Distinguishing referred pain from radicular pain:
Referred pain – dull, poorly localised, bilateral, improved by rubbing.
Radicular pain – sharp, well localised, follows dermatome, may have paraesthesia.

Management
90% resolves within 6wks.
Aims – to rule out serious pathology and to prevent becoming chronic (about 10%).
Try to avoid intervention as patient may become dependent, educate patient to manage pain.
For vast majority – reassurance, education (e.g. correct lifting), analgesia, physiotherapy.

Investigation - if persists, neurological involvement, systemic disease or red flags.
Bloods - should always be normal with mechanical back pain. ESR good screening test – increased with infection and malignancy.
Xrays - only indicated if history of trauma or red flags. Need flexion and extension views if suspect instability. Over 60% of people over 50 have signs of spondylosis (vertebral sclerosis, osteophytes, OA of facet joints) but these may be incidental.
Can also do MRI, CT with intrathecal contrast or isotope scan if suspect mets.

Analgesia – TENS, heat and cold, paracetamol, NSAIDs, amitryptaline, epidural corticosteroids, steroid injection to facet joints.
Surgery – if nerve root or spinal cord compression or instability (do fusion).

Degeneration of vertebrae
Spondylosis = non-specific term, meaning progressive degeneration of spine, includes loss of disc space, osteophyte formation, OA of facet joints.
Spondylolysis – degeneration or defective development of pars interarticularis (neural arch) allowing spondylolthesis.
Spondylolisthesis – forward displacement of vertebrae relative to others. Asymptomatic or can get lower back pain, radiculopathy or spinal stenosis. Various types depending on aetiology (congenital, isthmic, degenerative, traumatic, pathological). Myer grading of severity, grade 1-5 depending on degree of slippage, grade 5=100% slippage (spondyloptosis). Can cause nerve root compression or lumbar stenosis.

Management - mainly manage by posture advice and muscle strengthening. Surgical fusion if severe and recurrent pain or if neurological compromise.

Spinal stenosis:
Congenital narrowing with superimposed degenerative narrowing.
Mostly present with spinal claudication – pain related to exercise, tingling in legs, tends to work down legs. Relieved by stopping walking and flexion.
Manage conservatively with weight loss and NSAIDs. Some will need surgery.
Scoliosis
Most obvious on bending forward, get rib hump (Adam’s test). Almost always associated with rotation. Surgery if interferes with breathing, growth or movement.

Aetiology - usually idiopathic, esp in girls. Also congenital (wedge vertebrae, hemivertebrae, congenital bar, block vertebrae), trauma or neuromuscular (CP, spina bifida, muscular dystrophy) or secondary (e.g. to leg length discrepancy).

Neck pain
Aetiology
Mechanical – postural, disc prolapse, cervical spondylosis, whiplash, non-specific.
Inflammatory – infection, spondylitis, RA, polymyalgia rheumatica.
Metabolic – osteoporosis, osteomalacia, Paget’s disease.
Neoplasia – metastases, myeloma, intratehcal tumours.
Other – fibromyalgia, torticollis.
Referred pain – pharynx, cervical lymph nodes, thyroid, teeth, angina, aortic aneurysm, Pancoast tumour, diaphragm.

Most episodes do not have demonstrable spinal pathology.

Fibromyalgia

Symptoms and signs
Multiple tender trigger points (hyperalgesic) with a pain which is a diffuse, muscular, aching discomfort and does not respond to analgesics or physiotherapy (gets worse). Disproportionate disability and high levels of distress are typical.
Also stiffness, subjective swelling, fatigueability, disability, low mood, poor sleep, poor concentration and tiredness.
May only present with pain in one site initially.
Examination is usually normal, except for trigger points.

Often associated with other medically unexplained health problems e.g. chronic fatigue syndrome, IBS, pre-menstrual syndrome, tension headache, irritable bladder, allodynia, anxiety and depression.

Diagnosis
Criteria for diagnosis: hyperalgesia in all 4 limbs and 2 axial sites (11 out of 18 sites give wince-withdrawal response) with other symptoms of medically unexplained pain.
Hypermgesic tender sites: suboccipital muscle insertions, low cervical spine (interspinous ligaments), skin roll of trapezius, mid supraspinatus, pectoralis (lateral to 2nd costochondral junction), 2cm distal to lateral epicondyle of the elbow, low lumber spine (interspinous ligaments), gluteal upper outer quadrants of buttocks, posterior to greater trochanteric prominence, medial fat pad of the knee. Also test at control sites (e.g. on forehead) – if hyperalgesia at these sites as well likely to have severe psychological disturbance or be malingering.

Other causes of multiple regional pain – generalised hypermobility (risk of strains and sprains, may be primary or due to Marfan’s, Ehler-Danlos or acromegaly), endocrine disease (hyperparathyroidism, hypothyroidism, osteomalacia, hypercalcaemia), inflammatory polyarthritis, Parkinsonianism, polymyalgia rheumatica, polymyositis, SLE, paraneoplastic syndromes (esp leukaemia and lymphoma). Tietze syndrome is pain over costrochondral junctions, this is probably part of fibromyalgia.
Tests are necessary only to exclude serious pathology, all should be normal in fibromyalgia:

- **Radiology**
- **Inflammatory markers** – should not be raised.
- **Blood tests** – FBC, thyroid function, creatine kinase, RF and ANA, calcium and alk phos (hyper PTH and osteomalacia).
- **EMG and nerve conduction studies.**

**Epidemiology**
Prevalence 2%, much more common in females, increases with age.

**Risk factors** – psychological distress e.g. divorce, alcoholism, traumatic injury, low income, somatisation.

**Sleep and pain physiology**
Poorly understood aetiology.

- **Reduced restorative sleep** - reduced delta wave (REM) sleep is found in people with fibromyalgia. Deprivation of this in normal volunteers produces the symptoms of fibromyalgia.
- **Abnormal pain processing** – people with fibromyalgia have a reduced threshold to pain perception and tolerance and spinal cord pain amplification (e.g. dermatographism, allodynia). Increased substance P and decreased 5HT levels.

Pain, illness, anxiety and psychological distress may cause sleep disturbance. This creates a vicious circle of non-restorative sleep, fibromyalgia symptoms and reduced activity and poor fitness.

**Management and rehabilitation**
Acknowledge reality of symptoms and distress.
Education about nature of problem, pain control and improvement of sleep. Explain that pain does not reflect damage or disease. Explain pathology and that condition is common. No blame causation, explain effect of stressors. Involve family if possible.
Avoid repeat investigation.
Low dose amitryptaline may help.
Aerobic exercise helps wellbeing and sleep quality.

**Joint Pain**

**Presentation**

**Symptoms** – pain, stiffness, deformity, inflammation, loss of function.
**Signs** – localised tenderness, changed attitude, swelling, redness, heat, crepitations,
**Summary** - number of joints affected, distribution (upper or lower limb, small or large joints), symmetry and associated extra-articular features if present.

**Differential diagnosis**
Arthritis is classified as inflammatory or mechanical/degenerative (OA).
**Inflammatory** – mono, oligo (2-4 joints) or polyarthritis (symmetrical or assymetrical). Can be acute, chronic or palindromic (episodes e.g. gout). Suggested if – swelling and effusion (leaking blood vessels), redness (increased blood flow), heat, pain, loss of function (esp prolonged morning stiffness), systemic features of inflammation (weight loss, night sweats).
**Mechanical** - suggested if crepitus, deformity or instability.

Acute monoarthritis
**Red, hot, swollen joint** - septic until proven otherwise.
Also crystal arthritis, trauma, presentation of sero- or RA, haemarthrosis (e.g. haemophilia).
If already damaged or inflamed joint – septic arthritis, exacerbation of underlying disease, pesudogout, secondary avascular necrosis, cartilage problem, haemarthrosis.
Aspirate joint, infection screen, start on IV antibiotics.
Joint aspirate needs gram stain and microscopy and polarised light microscopy for crystals.

**Chronic monoarthritis**
OA, crystal arthritis, chronic infection e.g. TB.
Do synovial biopsy and culture if infection suspected.

**Acute or chronic oligoarthritis**
Consider septic arthritis with virulent organism (esp if immunocompromised or sequential).
Mostly OA.
If inflammatory – sero -, erythema nodosum, presentation of polyarthritis, gout.

**Inflammatory polyarthritis**
Can get bursitis, tendinits or synovitis. This then causes secondary joint damage so need early treatment to prevent this.
Look for extra-articular manifestations.

**Symmetrical** – nodal generalised OA, RA, psoriatic, connective tissue disease.
**Assymetrical** – seronegative, especially with dactylitis, enthesitis and predominantly large joint involvement.

**Juvenile chronic arthritis** – pauciarticular (ANA+, often chronic ant uveitis), poly articular onset (RF+), systemic onset.
**Various viral infections** – rapid onset polyarthritis, associated with fever and rash e.g. erythrovirus, hepatitis, mumps, rubella, infectious mononucleosis, HIV. Usually self-limiting by 6 weeks.

**Initial investigations** – FBC, ESR, LFT, RF, ANA and xrays.

**Non-musculoskeletal causes of joint disease**
Renal bone disease, Charcot joint, haemophilia (intra-articular bleed), haemoglobinopathies (vaso-occlusive episodes), haemochromatosis, thyroid disease (generalised aches, carpal tunnel with hypo, osteoporosis with hyper), acromegaly (increased movement).

**Orthopaedic surgery complications**
**General** - chest infections, venous thromboembolism (esp lower limb, if low risk early mobilisation, if medium or high then heparin and mechanical calf pump), cardiovascular events, pressure sores.

**Early local** - infection of joint (various techniques to reduce risk, treat any infections e.g. UTI before surgery), tight cast (ischaemia limb), compartement syndrome, nerve injury.

**Late local** - dislocation of hip replacement, infection, periprosthetic fracture, aseptic loosening (10-15 yrs after, revision is hard due to loss of bone).

**Joints**
**Fibrous joints** – minimal movement, e.g. skull sutures.
**Fibrocartilagenous joints** – limited movement, e.g. symphysis pubis, costochondral junctions, intervertebral discs.
**Synovial joints** – large range of movement, most limb joints, also TMJ and costovertebral. Smooth movement depends on articular cartilage (type II collagen, hyaline cartilage, less water and therefore less shock absorption with age) and synovial fluid (ultafiltrate into which synovial cells secrete hyaluronan and proteoglycans).
Synovial membrane – type A synoviocytes are phagocytic, type B secrete synovial fluid.
Hinge joints – collateral ligaments prevent movement at right angles.
Shoulder
Glenohumeral joint, sternoclavicular joint, acromioclavicular joint. Also scapular over thoracic cage but
this rarely causes problems (may get painless ‘clon’).
Have subacromial space between AC joint and GH joint, through which supraspinatus tendon passes
with a bursa.
GHJ has large range of movement but is unstable. Glenoid fossa is very shallow so easily to dislocate,
depenened by labrum. Ligaments and capsule are also very lax so stability depends on rotator cuff.

Causes of shoulder pain:
SCJ – can be affected by OA but usually asymptomatic, if symptomatic must consider septic arthritis.
Get localised pain over joint.
ACJ – target for OA. Stress by forced adduction and superior arc. Get localised pain over joint. Can do
LA and steroid injection to joint.
Gleno-humeral joint – target site for RA and sero- arthritis. Can also get OA. Tight pack position for
GH joint is hands behind head – if can do this GH arthritis is unlikely. Pain refers to upper arm,
insidious onset, fluctuates, affects all movements.
Rotator cuff tear, supraspinatus tendonitis, subacromial bursitis, impingement – all give middle
arc pain and refer to upper arm. Resisted active movement gives pain with rotator cuff tear and
supraspinatus tendinitis.
Bicipital tendonitis – usually well localised, aggravated by stressing biceps.
Bony pain
Referred pain - from neck (cervical spondylosis), heart (ischaemic pain), chest (pneumonia),
diaphragm.

Can identify cause of painful arc by series of LA injections to see effect on pain— subacromial bursa,
ACJ, GHJ.

Rotator cuff:
Supraspinatus – initial abduction.
Infraspinatus – external rotation.
Teres minor – external rotation.
Subscapularis – internal rotation.
All insert to greater tuberosity except subscapularis which inserts into lesser tuberosity.
In practice all work together and contract whenever shoulder is moved.

Limitation of abduction – capsular stiffness or joint arthritis (also affects other movements), rotator
cuff tear, impingement on cuff.

Abduction arc:
1st part – GH problem, esp if also external rotation.
Middle arc – subacromial space is maximally small, therefore painful middle arc if subacromial
impingement, subacromial bursitis, supraspinatus tendonitis (which would hurt with resisted active
movement).
Superior arc – ACJ problem.

Hip
Most of stability from ligaments and muscles.
Hip joint pain – feel at hip, or in groin, ant lower thigh or knee.

Other causes of pain in hip:
Trochanteric bursitis - pain over greater trochanter.
Ischigluteal bursitis – pain aggravated by sitting.
Adductor enthesopathy – pain and tenderness over adductor insertions.
Lumbar spine.
Knee

**Patellofemoral compartment** – ant knee pain, worse on going up and down hill (weightbearing on flexed knee, which puts patella against femur so stresses compartment), progressive pain when sitting with knee flexed.

**Medial and lateral femoro-tibial compartments.** Tibia has medial and lateral plateau and spines. **Superior tibiofemoral joint.**

2 menisci – fibrocartilaginous discs, lateral and medial, damaged in OA. Menisci are also found in wrist, TMJ and ACJ. Main function is to equalise pressure distribution.


**Collateral ligaments** – varus valgus stability. Medial is thick and part of capsule, lateral is thin and separate from capsule.

**Bursae** – pre-patellar, deep and superficial infra patellar, anserine.

Mostly a hinge joint, some rotation to lock in extension. Patella gives mechanical advantage to quads, lose 20% strength if no patella.

Deformities:

**Genu varus** – from front, knees go out. Mostly from OA.

**Genu valgus** – from front, knees go in. Mostly inflammatory synovitis.

**Genu recurvatum** – from side, knees hyperextended. Mostly generalised hypermobility.

**Posterior tibial subluxation** – from side, tibia slips backwards on knee. Mostly from arthritis whilst growing or post cruciate rupture.

**Fixed flexion deformity** – from side.

Swelling:

Within joint – horseshoe shaped.

**Pre-patellar bursitis** – over patella. Distinguish swelling from hypertrophied synovium (boggy) from fluid (fluctuant).

**Locking** – can’t fully extend. Often due to split meniscus, typically by twisting on bent knee.

**Giving way** – dysfunction of extensor mechanism, tracks wrongly (usually muscle weakness so goes laterally).

Knee pain:

**Fat pads** – medial.

**MCL enthesopathy or injury** – medial above and below joint line.

**Meniscus** – well localised joint line tenderness and loss of full extension.

**Pain from joints** e.g. OA.

**Bursitis**

**ITB syndrome** – lateral

**Enthesopathy** - LCL enthesopathy, popliteus tendon enthesopathy.

**Referred** – front of thigh from hip, back of leg from spine.

**Posterior pain** – mainly due to complications e.g. popliteal cyst, posterior tibial subluxation.

Wrist and hand

**Carpal bones** (some lover’s try positions that they can’t handle):

Distal row (radial to ulnar) – trapezium, trapezoid, capitate, handle

Proximal row – scaphoid, lunate, triquetrum, pisiform.

**Ulnar nerve** – supplies all hand muscles except lateral 2 lumbricles, opponens pollicis, abducens pollicis and flexor pollicis brevis (thenar eminence, supplied by median nerve). Claw hand.

**Median nerve** – thumb to middle of ring finger on palmar side, back of thumb. Supplies thenar eminence.

**Radial nerve** – small area on back of hand base of thumb.
Fingers have collateral ligaments down sides. Tight with MCP flexed and PIP extended (splint in this position to prevent ligaments tightening). Palmar plate on flexor side.

**Thumb base** – common site for OA. Generalised reduction in mobility, well localised pain.

**Tendon rupture** – if complete rupture can’t extend finger at all. If partial rupture can’t extend a bit but not fully due to tendon slips.

**Ankle and foot**  
**Fibula** – lateral malleolus.  
**Tibial** – medial malleolus.

**Ligaments** – anterior talofibular ligament, posterior talofibular ligament, lateral calcaneofibular ligament. Inferior tibio-fibular. Medial collateral or deltoid ligament.

**Fibrofatty pads** - under MTPJ and tip of toe. If claw toes, pad isn’t under metatarsal head so get pain.

**Ankle joint** – plantar and dorsiflexion.  
**Subtalar joint** – eversion and inversion.  
**Mid foot** – eversion and inversion, some plantar and dorsiflexion.

**MTP** – targeted by gout and OA.  
**Big toe IP** – targeted by sero-arthritis.  
**All MTPs** – targeted by RA.  
OA of the ankle is usually secondary to trauma.

**Toe deformities:**  
**Hammer toe** – hyperextension of MTP and DIP, flexion of PIP.  
**Claw toe** – flexion of PIP and DIP.  
**Mallet toe** – flexion of DIP.  
**Hallux valgus** – valgus deviation of 1st MTP, varus deviation of 1st metarsal (often primary problem) and lateral deviation and rotation of phalanges. Metatarsal head is exposed medially, this prominence (which may be increased by an exostosis) results in formation of a bursa due to excess pressure (a bunion). If severe, also have pronated toe (can take very little weight on it), laterally subluxed sesamoids and tendons. Get pain over bunion and 1st MTP. Mostly managed with comfortable foot wear. Minority need surgery – many different types, mostly osteotomies, may give pain for several months.  
**Hallux rigidis** – OA of 1st MTP. Conservative management with analgesics. Surgery if severe, mainly fusion.
Elbow
3 compartments – humero-ulnar, humero-radial, proximal radio-ulnar.
Get cubital varus or valgus, usually secondary to a supracondylar fracture of the elbow.

Leg length discrepancy
Produces scoliosis.

Causes:
Fracture which heals with shortening.
Damage to growth plate – injury or sepsis.
Dislocation or dysplasia of hip joint.
Overgrowth – e.g. due to juvenile chronic arthritis.
Functional – scoliosis, foot drop, adduction contracture (so have to hitch up pelvis to maintain legs parallel).

Nerves
Femoral – knee extensors.
Obturator – hip adductors.
Sciatic – knee flexors, hamstrings.
Post tibial – ankle and toe plantarflexors.
Deep peroneal – ankle and toe dorsiflexors.
Superficial peroneal – ankle evertors.
Osteoarthritis

**Pathology**
Structural failure of synovial joints due to degeneration (age related) and dynamic processes (attempt at repair). Chronic non-inflammatory arthritis characterised by cartilage loss (need for shock absorption and smooth gliding surface). Get disruption of surface of cartilage (fibrillation and fissuring) and softening (due to increased water content), then loss of cartilage, loss of joint space, bone-bone apposition (subchondral bone exposed and eburnated). This causes subchondral sclerosis and cysts. Peri-articular bone response ossifies fibrocartilage and causes osteophytes. Also get bone remodelling, capsule thickening, synovial hyperplasia and secondary bursitis or enthesopathy.

Can affect any synovial joint, but typically affects DIPs, thumb CMC, hips, knees, big toe MTP, apophyseal joints of spine, ACJ and sterno-clavicular joint.

**Clinical features**

**Typical symptoms** - joint pain, short-lasting morning stiffness and joint instability leading to loss of function. Usually insidious onset with variable symptoms, worse with use, relieved by rest.

**Relative prevalence**: hand > knee (10%) > hip (4%).

**Examination** – antalgic gait, muscle weakness and wasting (esp gluteal, quads), joint line tenderness, varus deformity, flexion deformity, bony swelling, effusion, coarse crepitus, restricted movement (thickened capsule, osteophyte), minimal inflammation.

**Hand signs** – typically thumb CMC (squaring of thumb base), DIPs (Heberden’s nodes) and PIPs (Boucher’s nodes). Swelling is bony and usually non-tender.

**Knee** – all joints, esp medial tibial-femoral, causing varus deformity, and patello-femoral, often fixed flexion. More common in females. Often get associated CPPD deposition, which may cause inflammation. Need to xray standing to see joint space narrowing.

**Hip** – reduced movement, esp internal rotation and abduction. Often fixed flexion deformity, leg length discrepancy due to shortening. Mostly targets superior pole of joint. Usually get pain deep to ant groin with radiation to thick, buttock or knee. Gait is antalgic, Trendelenberg, fixed flexion (can cause circumduction) and shortened leg. Can get secondary trochanteric bursitis.

**Spine** – direct pain and pain due to osteophytes impinging on nerve roots.

**Risk factors**
Affects 25% over 65s.
Radiographically 80% have evidence of OA.
Hip, hand and generalised OA are more prevalent in Caucasians.
All joints except the hip are more common in women.

**Susceptibility** – genetic, age, hypermobility, heavy physical work or repetitive adverse loading of joints, trauma, obesity, diabetes, high bone mineral density, malalignment of joint.

**Classification**
Can classify by presence of nodes, no.of sites and presence of calcium crystal deposition.
Can be primary or secondary.

Secondary causes:

**Congenital or developmental conditions** – developmental dysplasia of the hip, slipped femoral epiphysis, Perthe’s disease, hypermobility.

**Metabolic** – e.g. calcium pyrophosphate deposition disease.

**Traumatic** – either involving joint or causing deformity or instability e.g. fracture, meniscal injury, joint instability e.g. ACL rupture, neuropathic joint.

**Inflammatory** – RA, septic arthritis, gout.
Primary generalised nodal – one variant of OA. Typically affects middle-aged women, predominantly hand IPJs with nodes, though relatively unimpaired hand function. Some patients may develop erosive OA and IPJ instability. Nodal OA may become generalised esp affecting knee. Strong genetic predisposition (1 in 3 chance if mother affected).

Young-onset OA – previous trauma, localised instability, prior joint disease (e.g. juvenile idiopathic arthritis), metabolic disease (acromegaly, haemochromtosis), avascular necrosis, neuropathic joint, endemic OA (environmental cartilage toxins e.g. Kashin-Beck disease in Russia).

Investigations
Not systemic or inflammatory so all blood tests, including inflammatory markers, should be normal. Mainly investigate by plain xray, rarely MRI, arthroscopy.

Correlation between radiographic evidence of OA and symptoms is variable – best at hip, poor at most small joints. Pain and disability often correlate more with muscle weakness and adverse psychosocial factors.

Radiological features
Joint space narrowing showing cartilage loss, typically focal.
Subchondral sclerosis – due to loss of shock absorption and increased stress on bone.
Osteophytes.
Cysts – bone comes into contact with synovial fluid due to loss of cartilage, probably due to increased pressure in joint.
Also attrition, deformity, soft tissue swelling and osteochondral bodies.

Management
Structural changes are permanent so try to relieve pain, improve function and prevent progression. Prognosis is good for hand OA, better for knee than hip.

Conservative
Patient education.
Avoid aggravating activities but remain active.
Physiotherapy, aerobic and strengthening exercises – moderate but long term improvements in pain and disability.
Reduce adverse mechanical factors – lose weight, shock absorbing footwear.
Occupational therapy - walking aids (use on opposite side to problem), braces and collars.
Heat and cold to induce muscle relaxation.

Pharmacological
No disease modifying drugs.
Simple analgesia – paracetamol.
Topical – NSAIDs, capsaicin cream.
Oral NSAIDs – only if simple analgesia fails, not always helpful as OA is not inflammatory.
Oral opioids.
Corticosteroid injections – e.g. dexamethasone. Either intra-articular or peri-articular. suppresses inflammation, helps relieve moderate and severe pain and can increase mobility, lasts for up to 2m. Especially knee and thumb base. Never inject if local or systemic infection. Can cause joint tissue atrophy, infection.
Hyaluronan injection – intra-articular injection, esp knee. Supplement natural synovial fluid, effective for some patients in relieving pain. As above contra-indicated if infection.

Glucosamine – take with food. Efficacy debated, may have modest symptom relief.
Surgery
Only for a small proportion of patients.

**Indications** – uncontrolled pain (especially when interferes with sleep or severely limits walking), progressive immobility, functional impairment.

Operations:
- **Washout and debridement** - remove osteophytes, some pain relief.
- **Total joint replacement** - 98% happy with hip replacement and 85% with knee at 2 years. Failure rate (mainly due to loosening) at 15 years is 15% for hip replacements and 10% for knee replacements.
- **Osteotomy** - realignment of joint.
- **Excision arthroplasty** - take on one surface of joint and allow fibrous tissue to form, mainly AC joint, MTPs and trapeziectomy, also hip if infected.
- **Arthrodesis** - fuse joint, mainly ankle and wrist.

**Gout**

**Pathology**
Prolonged hyperuricaemia causes crystal deposition in joint space and bone (tophi).

Urate is produced from:
- **Purine metabolism** (2/3) – hypoxanthine to xanthine to uric acid by xanthine oxidase).
- **Diet** (1/3) - so can affect level by diet manipulation).
  Lost by renal excretion (2/3) and intestinal uricolysis (1/3).

Hyperuricaemia can result from:
- **Reduced renal excretion** (90%) – genetic under-excretors, prolonged diuretic or cyclosporine use, dehyrdration, renal impairment, low dose aspirin, lead toxicity, lactic acidosis, hypothyroidism.
- **Overproduction** (10%) – alcohol, myeloproliferative disorders, Lesch-Nyan (HGPRT deficiency), G6PD, various specific or unknown (most common) enzyme defects.

Persistent hyperuricaemia leads to clumps of monosodium urate monohydrate crystals, which form crystals over many years (depends on various factors that promote or inhibit crystal nucleation). This is promoted by cartilage damage (e.g. OA), iron and hypothyroidism. Crystals are then found in joint space, between flares are covered with lipoproteins so don't produce inflammatory response.

**Acute flare:**
If serum urate level falls (illness, surgery, alcohol, drugs which lower serum urate e.g. allopurinol, aspirin) or there is trauma to the joint, some of the crystals in the joint space solubilise and are shed which disrupts covering, exposing naked crystal. This produces an inflammatory response and acute gout (usually 2/3 days after illness).

**Epidemiology**
Gout is the most common arthritis in men. Affects middle-older men and older females (very rare in females before menopause).
Prevalence 1%.

**Primary gout** – middle aged men, urate underexcretors (thought to be part of metabolic syndrome), esp if overweight, heavy beer drinkers, mainly affects feet initially.

**Secondary gout** – people on diuretics or with renal failure, older people, women, gout in OA joints (esp nodal generalised OA), affects hands and feet, can present with tophi, saturnine gout (lead poisoning).
Clinical features

Acute attack - red, hot, swollen joint. Very painful and tender (can’t wear sock), comes on very acutely (<12h), resolves over 2wks. Can get desquamation of skin as resolves. Typically target big toe (75% of gout sufferers, podagra), also ankle, feet, knees, elbow, DIPs. In older women affects fingers damaged by OA. Can also affect bursae esp olecranon bursitis.

Natural history - 90% of attacks are recurrent. Typically get attacks then periods free of symptoms. If untreated attacks become more frequent and start to affect other joints, causing joint damage. Chronic symptoms may develop after about 10 years.

Chronic tophaceous gout – get deposits of uric acid (tophi) around joints (esp hand and foot) and also in ear helix. Swellings are heterogenous with some soft and some hard areas. May appear white or discharge crystal. Cause joint damage by pressure necrosis and bone destruction – intra and extra capsular punched out lesions on xray. Crystals can be deposited in kidneys – urate nephropathy and may get uric acid stones causing renal colic.

Can have crystals in joint and be asymptomatic.

Diagnosis

Urate level – normal is <0.42mmol/l (this is solubility point in physiological conditions, same as 7mg/dl), higher in men than women. Or can use above 2SDs so 0.4 in men, 0.35 in women.
Uric acid is a negative acute phase protein so level during attack is unhelpful. Many people have hyperuricaemia but do not develop gout.

Joint aspiration - mainly to rule out sepsis in hot swollen joint, however can then do compensated polarised light microscopy of synovial fluid. Urate – needle shaped, large, numerous, brightly negatively birefringent (blue when perpendicular and yellow when parallel). Also see elevated cell count, mostly neutrophils.

Xray – punched out lesions with retained bone density if deposition in bone. Assess joint damage, may see changes of OA.

Management

Acute attack

NSAIDS – often indomethacin (nephrotoxic and crosses BBB)
Colchicine – stops neutrophils, may cause diarrhoea.
Joint aspiration and intra-articular or oral steroids.
Ice packs.

Lifestyle

Decrease alcohol, esp real ales and beer.
Decrease purine intake – esp red meat, shellfish.
Lose weight.
Try to get of diuretics, esp thiazides.
Check lipid profile, glucose, BP – risk of CVD.
Check renal function.

Allopurinol

Mechanism – blocks xanthine oxidase so don’t formation of urate from purines.
Starting allopurinol – can precipitate an acute attack as causes urate level to change. Some say start after acute attack has resolved, others say straight away to avoid another attack on starting. Start at a low does and build up to get serum urate down to about 0.25 mmol. Many use “anti-inflammatory cover” for the first week on starting allopurinol. Taken long term it lowers urate level, reducing or preventing further attacks. Usually lifelong treatment.
**Indications** – recurrent attacks (or single attack if patient wants), tophi (worry that they may have urate nephropathy), joint destruction, urate nephropathy, nephrolithiasis, before chemotherapy to prevent urate nephropathy (get purine breakdown and high urate load which form conglomerates in kidney). Very rarely causes life-threatening vasculitis.

Must not give with azothioprine as allopurinol blocks metabolism of azothioprine so it builds up causing neutropenia.

**Other rarely used hypouricaemic drugs** – probenecid, high dose aspirin, sulphinpyrazone. These are uricosurics (increase uric acid excretion, so only work if good kidney function, not useful if overproducer, risk of uric acid stones) and need several doses per day.

**Calcium pyrophosphate dehydrate crystal deposition**

Presentations:
- **Asymptomatic chondrocalcinosis** - significance is uncertain, mostly in knee.
- **Pseudogout** - acute arthritis. Usually affects single joint, less acute onset and longer episodes, elderly people. Primary target is knee, also wrist. Calcium levels also fall with acute phase response so predispose to attack after illness.
- **Chronic destructive arthropathy** - like OA but targets wrists and ankles and inflammatory component, also find crystals in OA – uncertain significance.

Common in elderly.

**Associations** (look for these in young people) – hyperPTH, haemochromatosis, hypophosphataemia, hypomagnesaemia, familial hypocalciuric hypercalcaemia.

**Compensated polarised light microscopy** – false negatives are more common. Crystals – variable shape, weakly positively birefringent.

**Xray** – chondrocalcinosis, changes of OA.

**Management** - immediate management is as an acute gout attack, there is no long term treatment. Management of chronic arthropathy is the same as for OA.

**Calcium phosphate crystals**

Basic calcium phosphate crystals e.g. apatite. This is the main mineral in bone and teeth. However, can get abnormal deposition in peri-articular tissues (esp tendons), in hyaline cartilage (with OA) and in subcut tissue and muscles (in CTDs). Abnormal deposition may be due to hyperparathyroidism, renal dialysis, vit D intoxication.

**Presentation:**
- **Acute arthritis.**
- **Destructive arthropathy** - found in joints with OA, rarely associated with rapidly destructive arthropathy.
- **Acute calcific periarthritis** - e.g. deposition in supraspinatus tendon, mostly asymptomatic but rarely causes severe acute inflammation, can also cause bursitis.

**Calcium oxalate crystals**

Acute and subacute arthritis.
**Rheumatoid Arthritis**

**Clinical features**
Chronic autoimmune symmetrical deforming inflammatory progressive polyarthritis. Affects all joints often with systemic or extra-articular features.
Range from mild self-limiting disease to chronic destructive disease.

Types of disease:
- **Chronic persistent** - typical form, follows relapsing and remitting course over many years with severe joint damage. +ve/-ve for IgM rheumatoid factor.
- **Palindromic** - monarticular attacks lasting 24-48hrs, move from 1 region to another, may progress to typical RA.
- **Transient** - self limiting lasting < 12 months and leaving no permanent joint. -ve IgM rheumatoid factor.
- **Remitting** - active for several years but then remits, leaving minimal damage.

Pain and stiffness usually starts in the small joints of the hands and feet or wrists and then affects larger joints. Also affects tenosynovium and bursae. Deformities and non-articular features develop as the disease progresses.
May be insidious onset (possible worse prognosis) or acute onset (more common in elderly). When examining try to determine if active or inactive (no inflammation but persisting deformity).

**Diagnosis** – more than four of morning stiffness, arthritis of 3 or more joints, arthritis of hand joints, symmetrical arthritis, rheumatoid nodules, rheumatoid factor, radiological changes, duration of 6 weeks or more.

Pattern of joint involvement:
All joints typically come on within 6m. Joint swelling is typically Boggy (synovium).
- **Hands and wrists** - usually severely affected, ulnar drift and palmar subluxation of the MCPs, fixed flexion (boutonniere deformality) or fixed hyperextension (swan neck deformity) of PIP, Z deformity of thumb, subluxation or fusion of the wrist, prominent ulnar head (piano key), dorsal subluxation of ulnar head. DIPS usually spared. May also see muscle wasting (esp dorsal interossei), bulging synovium, vasculitic lesions, nodules, extensor tendon rupture.
- **Shoulders**- initially like rotator cuff tendonitis then stiffening and tears.
- **Elbows** - swelling and painful fixed flexion deformity.
- **Feet** – painful swelling of MTP joints and hammer toe deformity, forward migration of fibrofatty pad leads to pressure on metatarsal heads and ulcers, subluxation of metatarsal heads and cock-up toes, valgus position of ankle, calcaneovalgus, hallux valgus, callousities, flat feet, soft tissue swelling.
- **Knees** - massive synovitis and knee effusions, erosion of cartilage and bone in later disease with secondary OA. Popliteal cysts.
- **Hips** - rarely affected in early RA, later, secondary OA develops and hip replacement is usually necessary.
- **Cervical spine** - bone destruction, ligament damage and atlantoaxial or upper cervical instability. Subluxation or local synovial swelling may damage spinal cord giving neurological signs.

**Rheumatoid nodules** – only sero+ patients, associated with more severe disease, 25% patients, mainly subcutaneous e.g. elbow, also other bony prominences e.g. Achilles and knee. Can also get similar granulomas in pleura, lung, pericardium and sclera. Consist of palisaded macophages and lymphocytes. Can get infected.
Extra-articular features:

**Systemic** - anorexia, weight loss, fatigue, susceptibility to infection.

**Musculoskeletal** – muscle wasting, tenosynovitis, bursitis, osteoporosis.

**Haematological** – anaemia of chronic disease (also due to NSAIDs), eosinophilia, thrombocytosis, splenomegaly, Felty’s syndrome (=splenomegaly, leucopenia, lymphadenopathy, vasculitis, anaemia in RA), lymphoma.

**Ocular** – episcleritis, scleritis (more serious, risk of secondary glaucoma, treat with NSAIDS or steroids), keratoconjunctivitis sicca (Sjogren’s), scleromalacia.

**Connective tissue and vasculitis** – digital arteritis, ulcers, pyoderma gangrenosum, mononeuritis multiplex, visceral arteritis Raynaud’s, Sjogren’s (25%).

**Cardiovascular** – pericarditis (mostly asymptomatic but can cause effusions and constrictive pericarditis), myocarditis, endocarditis, conduction defects.

**Pulmonary** – nodules, pleural effusions, fibrosing alveolitis, bronchiolitis, Caplan’s syndrome (pulmonary fibrosis and nodules). With breathlessness consider pulmonary fibrosis, methotrexate pneumonitis or infection.

**Neurological** – cervical cord compression (subluxation of C spine), compression neuropathies (e.g. due to hypertrophied synovium or joint subluxation), peripheral neuropathy.

**Complications** - ruptured tendons (esp extensor tendons of hand), carpal tunnel (synovitis of wrist flexors), joint infection, atlantoaxial subluxation (in flexion, risk of cord compression, due to erosion of transverse ligament around post of odontoid peg), amyloidosis (rare complication - extracellular deposits of insoluble protein, esp in kidney causing proteinuria).

**Epidemiology**
Prevalence 1.5%. Most common inflammatory arthritis in females.
F:M 3:1, more female predominance at younger ages.
Affects all races, highest in Pima Indians, lowest in black Africans and Chinese.
Bimodal peak – 30-40 and 60s.
60% rheumatoid factor positive.
Smoking is a risk factor.

**Aetiology**
Autoimmune disease.
Some genetic predisposition. HLA DR4 increases risk of disease and of severe erosive disease.

**Pathology**
Mainly synovitis. Get increased blood flow, synoviocytes proliferate creating more synovial fluid (swelling), neutrophils release proteolytic enzymes and damage articular cartilage (irreversible, inflammatory mediators e.g. TNF alpha and IL1, stimulate production of metalloproteinases and degrade cartilage). Inflammatory granulation tissue in rheumatoid disease = pannus. Get thickening of synovial membrane and inflammatory infiltrate.
Synovitis causes marginal erosions - erodes bone in bare areas (bone at end of cartilage in direct contact with synovium). No new bone response so non-proliferative and atrophic marginal erosions.

**Radiographic changes**
**Non-proliferative marginal erosion.**
**Osteopenia** – due to lack of use.
**Diffuse joint space narrowing** – with late disease, cytokines destroy cartilage.
**Deformity and fusion** (wrist and hindfoot).
**Dot-dash pattern** – early erosions.
**Effusions** – soft tissue swelling.
Management

Investigations
Initial investigations to aid diagnosis – FBC, ESR, CRP, rheumatoid factor, Xray.
Monitoring disease damage – x-rays.
Monitoring drugs – urinalysis, biochemistry, haematology.

Conservative
Splints or fusion to allow pain-free stability – esp wrist.
Physiotherapy to protect joints.
Occupational therapy.
Chirpody.

Surgery
25% will need joint replacement.
Can also do synovectomy (pain relief, slow progression and prevent tendon rupture in hands). In later stages may to osteotomies, arthrodesis or arthroplasties e.g. forefoot arthroplasty (excision of metatarsal heads).
May need multiple operations. Surgery is difficult due to osteopenia, poor healing, thin skin and deformity.

Drugs
Start with NSAID, if this fails to suppress disease then use disease modifying anti-rheumatic drug (DMARD). Tend to use these early now to prevent irreversible joint destruction.
All slow acting – don’t have immediate effect on inflammation, may take up to 6m to have beneficial effect. Affect progression of disease, may also help extra-articular features e.g. vasculitis.

Indications – persistent synovitis, severe extra-articular disease, steroid sparing (e.g. in polymyalgia rheumatica), inflammatory myositis.
All drugs are similar in efficacy – choice depends patient preference and co-morbidities. Usually start on sulphasalazine, then methotrexate (best toxicity to efficacy ratios). If first drug ineffective after 6m or not tolerated try another, can use in combination.
Also use low dose immunosuppressants e.g. methotrexate, cyclosporine, cyclophosphamide. With these need to be aware of infection – have influenza and pneumococcal vaccine, increased risk of neoplasia (esp solid tumours and lymphoma). Can also use corticosteroids.
All drugs are potentially toxic and need blood monitoring (except hydroxychloroquine).

Sulphasalzine – used for RA and peripheral sero -. Need to monitor FBC and LFTs. Side effects include rashes, GI, hepatitis, rarely blood dyscrasias, staining of urine and contact lenses.
Leflunomide – RA, need to monitor FBC, LFT and BP. Side effects include GI, alopecia, myelosupression, teratogenic, interference with reproduction.
Penicillamine – RA, now rarely used, need to monitor FBC and urine. Side effects – rash, proteinuria, thrombocytopenia.
Hydroxychlorquine – RA and mild lupus, antimalarial, usually better tolerated than others but may be less efficacious. Doesn’t need blood count monitoring but need to check visual acuity regularly. Side effects – retinopathy, GI disturbance, rashes, rarely convulsions, ototoxicity, blood disorders. Very toxic in overdose.
Gold – RA, now rarely initiated, give as IM injection or more rarely orally. Start with weekly injections until response then monthly. Discontinue if no response after 1g. Need to check FBC and urinalysis before each injection. Side effects – blood dyscrasias, nephrotoxicity, rash, mouth ulcers, GI upset (oral therapy), rarely pneumonitis, colitis, neuritis, cholestasis.
Methotrexate – RA, sero-, lupus, CTD, vasculitis. Need to monitor FBC and LFTs. Side effects – GI upset, hepatotoxicity (esp with alcohol), acute pneumonitis (need to stop immediately and give high dose IV corticosteroids), alopecia. Inhibits dihydrofolate reductase and cell division. Need to prescribe with folic acid to reduce risk of adverse effects.

Azathioprine – RA, sero-, lupus, CTD, vasculitis. Need to monitor FBC and LFTs. Side effects – GI upset, hepatitis, myelosupression. Increased toxicity if given with allopurinol as inhibits metabolism.

Cyclophosphamide – vasculitis, lupus and myositis. Need to monitor urine (for blood) and FBC. Side effects – GI upset, haemorrhagic cystitis, myelosuppression, azoospermia, anovulation. Alkylating agent, which binds DNA, RNA and protines.

Cyclosporin – RA, psoriasis and lupus. Need to monitor FBC, LFT, creatinine and BP. Side effects – GI upset, nephrotoxicity, hypertension.

Anti TNF therapy (e.g. infliximab- monoclonal antibody) - only after trying 2 other DMARDs and with active disease (scoring system based on swollen joints, tender joints and ESR). More effective if prescribe with methotrexate. More effective than standard DMARDs but expensive. Side effects – serious infection e.g. TB reactivation, possible malignancy due to immunosuppression.

Management of flares – rest, anti-inflammatory therapy, passive exercises. Mostly can be managed out of hospital but rarely need admitting for intensive treatment.

Burnt out disease – analgesics, OT, physiotherapy.

Prognosis
Increased mortality – 5yr survival is only 50% if severe disease.
About 80% become moderately to severely disabled.
Functional status at 1yr correlates with outcome.

Poor prognostic factors – higher baseline disability, female, involvement of MTPs, RF+, long duration.

**Sero-negative Spondarthritides**

Diseases
Pathology - chronic inflammatory arthritides. Cause inflammation, fibrosis, calcification and ossification (proliferative erosions, osteophytes, syndesmophytes, spurs, can lead to joint fusion).
Main target is enthesis (enthesopathies – so can affect cartilaginous and synovial joints), also capsulitis, synovitis and osteitis.
B27 related - >99% in reactive arthritis and ank spond, strong association with others, prevalence in general population is 10%.

Clinical features - typically come on sequentially, often associated with dactylitis (small joint synovitis and periarticular swelling in a digit) and anterior uveitis. Inflammatory mostly assymetrical arthritis which targets large joints in axial skeleton (sacroiliitis and sponylitis) and lower limb (except psoriatic). May also get other inflammation e.g. Achilles tendonitis, planar fascitis, bursitis.

Extra-articular features – iritis, conjunctivitis, skin lesions, mouth ulcers, aortic root fibrosis (aortic incompetence and conduction defects), erythema nodosum.

Prevalence – ankylosing spondylitis 0.2%, psoriatic arthritis 0.1%.

Management – mostly physio, NSAIDs (esp indomethacin), sulphasalazine. Oral steroids rarely used.

Reactive arthritis
Pathology - acute inflammatory asymmetrical oligoarthritis. Aseptic arthritis in response to infection at distant site, typically UG, GI and throat. Up to 3 weeks after bacteria exposure. Esp campylobacter, salmonella, shigella, chlamydia. Can also be HIV, very severe. Can do serological tests for organism. Mostly young men.
Clinical features - typically lower limb. Get oral ulcers. First attack is usually self-limiting but about half develop recurrent attacks though permanent joint damage is rare. Sacroileitis is often asymmetrical and syndesmophytes are coarse and asymmetrical. Often get large fluffy calcaneal spurs.

Reiter's disease – urethritis, conjunctivitis, arthritis. Keratoderma blenorrhagica (rash on soles of feet like pustular psoriasis), circinate balanitis (painless erosions).

Management – supportive and symptomatic for first attack. May need treatment with DMARDs if severe arthritis or intractable keratoderma blennorragica.

Gonorrhoea - causes tenosynovitis, arthralgia and occasionally monoarthritis (but direct infection not reactive arthritis).

Ankylosing spondylitis
Pathology - chronic inflammatory oligoarthritis and spondylitis, predominantly spine and sacro-iliac joints causing stiffening and fusion. Associated with IBD but don’t come on together and not helped by colectomy. M:F 3:1, typically young adults.

Clinical features - stiff and painful back coming on insidiously, usually in 20s. Initially usually stiffness of lumbar spine, then progresses through spine and may restrict chest expansion. Late disease causes stooped posture due to loss of lumbar lordosis and thoracic and cervical kyphosis. May affect other joints – about 10% start with a peripheral joint.

Associations – aortic incompetence, pulmonary fibrosis (apical not basal as in RA), amyloidosis, prostatitis.

Xray changes - usually start with sacroileitis (irregularity, loss of cortical margins, widening of joint space) then sacroiliac joint sclerosis, vertebral squaring, spotty ligamentous calcification, syndesmophytes (bridge vertebral bodies, fine, symmetrical and marginal) and then bamboo spine (syndesmophytes, calcification of ant longitudinal ligament, facet joint fusion). Risk of secondary cord compression.

Management – exercise programme is important to maintain spinal mobility. Can use sulphasalazine. Anti TNF therapy may work. May do spinal osteotomies to correct posture.

Psoriatic arthritis
Different subtypes, can precede skin disease (20%). 5% of patients with psoriasis get arthritis, increased risk if nail involvement.

Types – asymmetrical oligoarthritis (often dactylitis, upper and lower limbs), distal arthritis involving DIPs (mainly men), symmetrical RA like, arthritis mutilans (loss of joint and bone, get telescoped fingers), spondyloarthritis.

Psoriatic nail dystrophy – nail pitting, onycholysis (separation of nail), hyperkeratosis.

Management – sulphasalazine, methotrexate, rarely azathioprine (little effect on axial disease). Acitretin can help arthritis as well as skin lesionss. Also try to maintain mobility to prevent fusion.

Enteropathic arthritis
Flares up as disease does, cured by curing IBD. 10% of IBD patients. Typically large joint lower limb.
Arthritis mutilans
With RA and psoriatic arthritis. Lose a lot of tissue causing telescoping and instability of fingers (opera glass fingers).

Radiographic features

Sacroileitis – erosions cause joint space widening and new bone formation causes joint space narrowing. Fusing of joint.

Infection of Locomotor Tissues

Pathology
Risk factors
Increased exposure to infection – direct trauma, blood bourne infection (infective endocarditis, septicemia), IV drug abusers. 
Increased susceptibility to infection – elderly, chronic inflammation of joints (rheumatoid arthritis 10x risk, osteoarthritis), immunosupressed (AIDS, drugs esp steroids), high alcohol intake, artificial joints (20x risk), diabetes, sickle cell disease (salmonella osteomyelitis).

Common infecting organisms
Mostly staph aureus, also strep, gonococcus (young adult, rash, tenosynovitis, low grade fever, arthralgia), pseudomonas, gram- bacilli (older people), TB (chronic), staph epidermidis (prosthetic joints).
If elderly or immunocompromised can be gram-, fungi.

Mechanism
Spread - mostly haematogenous spread (esp in children), rarely direct contamination (e.g. open fracture, joint aspiration, surgery, through skin e.g. in RA if macerated skin between toes).
Septic arthritis - causes joint damage due to direct effects of organisms e.g. toxins and inflammatory response. Get progressive destruction of joint.
Osteomyelitis - causes immune response, swelling, increased intraosseous pressure, microvascular occlusion and bone necrosis (sequestrum = separated shard of dead bone, harbours infection).
Chronic osteomyelitis - get sequestrum, involcrum (new suubperiosteal bone formation if cortex perforated) and local bone loss with persistent drainage or sinuses.

Common locations for infection
Septic arthritis – 50% knee, 20% hip.
Septic bursitis – elbow and knee.
Acute osteomyelitis – tibia, femur, fibula, humerus, ulna, radius. Typically juxta-epiphyseal areas of long bone.
Chronic TB osteomyelitis – hip, knee, vertebrae.
Ostitis – short flat bones of feet and hands.

Hand sepsis – paronychia (horseshoe around nail bed), pulp space infection, septic arthritis, tendon sheath infection.
Clinical features
Local pain and inflammation.
Septic arthritis – hot red joint, held in loosepack position, rest pain and stress pain on movement.
Osteomyelitis – bony pain.
Pseudoparalysis.
Associated cellulitis.
Systemic effects e.g. fever (only 1/3), sweats, malaise.
Bone sepsis generally presents less dramatically in elderly patients or those with RA.

Complications – joint destruction, arthritis, AVN.

TB – usually presents as gradual onset chronic mild inflammatory monoarthritis, get caseating necrosis.
If affects spine get kyphosis and can lead to paraplegia. Soft tissue calcification, bone destruction and joint narrowing on xray, investigate with Mantoux test, MRI, biopsy and culture.

Management
Immediate
Still has high mortality, serious risk of joint destruction.
Clues are fever and raised WCC but not always present. CRP is nearly always raised.

If suspect sepsis:
Admit.
Joint aspirate - gram stain and culture (positive in about 90% cases, less with gonococcal so need genital tract swab) and crystals.
BLOODS - blood cultures, ESR, CRP and WCC.
XRAYS - unhelpful in early diagnosis but take as baseline. Xray features – erosion of cartilage and bone, osteopenia, periostitis, patchy sclerosis (osteonecrosis). Can do bone scan (hot spot of infection, cold spot of dead bone), white cell scans and MRI if unclear diagnosis.
Empirical antibiotics – IV flucloxacillin, add cefuroxime if immunosupressed. 2wks IV antibiotics, 4wks oral.
Analgesia.
Interventions - serial aspiration and possibly washout to decompress joint and remove fluid. Surgery to decompress bone or remove dead bone.
Active early rehab – can get severe fibrosis and stiffness.

Chronic osteomyelitis – needs resection of bone, reconstruct by distraction osteogenesis with external fixation device.
Manage TB with appropriate TB chemotherapy and possible surgical debridement.
Bone Diseases

Investigations in bone disease
Alk phos – raised with mets, osteomalacia, Paget’s, fractures, multiple myeloma, hyperPTH. Also depends on liver, can measure bone specific. Produced by osteoblasts, needed for calcification, raised if bone formation.
Acid phos – raised in metastatic prostate cancer.
Calcium – raised with hyperPTH, multiple myeloma, mets, vit D intoxication. Low with hypoPTH, poss slightly low in osteomalacia.
Phosphate – raised with mets and multiple myeloma. Low with hyperPTH and osteomalacia.

Osteoporosis
Pathology
Low bone mass and micro-architectural deterioration of bone tissue lead to bone fragility and increased fracture risk. Affects cancellous bone more than cortical bone.
Defined by bone mineral density – T score compares with young healthy females, Z score with an age matched control, value is no. of SDs below. Osteoporosis is defined by T score > -2.5 indicates osteoporosis, -1 - -2.5 indicated osteopaenia. Z score compares with someone of same age.
DEXA scanning – to measure bone density. Use to diagnose and monitor treatment. Indications – low trauma fracture, clinical features (height loss, kyphosis), osteopenia, corticosteroid therapy, family history, early menopause, diseases associated with osteoporosis.

Various types – type 1=postmenopausal, type 2=age associated, secondary, idiopathic.

Clinical features
Generally asymptomatic until pathological fractures:
Distal radius fractures, esp Colles.
Fracture of neck of femur – 25% of fractures have osteoporosis. Can try to prevent with hip protectors but poor compliance.

Can also prevent with progressive vertebral deformity (collapse of several vertebrae causing kyphosis – Dowager’s hump). With a vertebral fracture need to consider mets – examine breast and chest.
Diagnosis - pathological fracture, radiological evidence of osteopenia, DEXA.

Investigations – all bone biochemical markers are usually normal. Need to check calcium in all patients as may be secondary to hyperPTH.

Risk factors
Depends on peak bone mass attained (generally at age 25) and rate of bone loss. Prevalence is increasing.

Age – bone mass dramatically falls in women after the menopause if no HRT (5% per year).
Sex – most common in women. 1 in 3 women get an osteoporotic fracture at some point. In men can identify secondary cause in 50% cases.
Genetic risk – low bone mass, family history, various genes e.g. oestrogen and vit D receptors.
Oestrogen deficiency – early menopause, amenorrhoea, low BMI.
Drugs – esp corticosteroids (related to dose and duration, cause decreased calcium and secondary hyperPTH, also direct inhibition of osteoblasts), also GnRH agonists, aromatase inhibitors, anticonvulsants, heparin.
Vit D or calcium deficiency.
Poor peak bone mass – low physical activity and loading of skeleton, poor nutrition, hormones. Alcohol and smoking. Previous fracture or immobility. Low BMI

Diseases which can cause secondary osteoporosis – acromegaly, Cushing’s syndrome, hyperparathyroidism (increased bone turnover), hyperthyroidism (increased bone turnover), hypogonadism, diabetes, chronic liver or renal failure, anorexia nervosa, malabsorption (due to calcium deficiency and secondary hyperparathyroidism), inflammatory disease e.g. RA (increase bone resorption via inflammatory cytokines), immobility (disuse osteoporosis), myeloma, conditions with altered blood flow (e.g. algodystrophy).

Factors which increase bone mass (stimulate osteoblasts, inhibit osteoclasts) – calcitonin, oestrogen, testosterone, mechanical loading.

Factors which decrease bone mass (stimulate osteoclasts, inhibit osteoblasts) – IL1, TNF alpha, thyroid hormones, glucocorticoids. Low testosterone or oestrogen causes uncoupling of resorption and bone formation.

Management
Reduce risk – HRT, impact exercise, good calcium intake (1g per day), stop smoking, avoid excessive alcohol, vit D, prevent falls, prophylactic bisphosphonates if on high dose or prolonged corticosteroids.

Drugs are indicated if osteoporotic on BMD scan, esp if previous fracture:

Calcium and vitamin D – generally as an adjunct to other treatments, or in elderly.

Bisphosphonates – IV or oral, inhibit osteoclasts so prevent bone resorption, therefore increase density (usually by 5-8%) and decrease risk of fracture (by about 50%). Side effects – oesophageal ulceration and reflux.

HRT or oestrogen analogues e.g. raloxifene – generally don’t use HRT just for osteoporosis as side effects and safer treatments exist. Raloxifene is better but only reduces vertebral fractures.

Calcitonin – osteoclast inhibitor, unclear if effective for non-vertebral fractures.

Parathyroid hormone – bone forming, very effective, increased BMD by 10%. Expensive so only patients with severe osteoporosis or who have not responded to other treatments.

Monitor effectiveness of treatment by DEXA scan, takes 18m to respond. Can also use biochemical markers of bone turnover, respond more quickly. Usually stay on treatment longterm.

Osteomalacia
Inadequate mineralisation of osteoid (bone matrix), causing bone pain, bone fragility and fractures. Normally due to a defect in vitamin D availability or metabolism. Same as rickets in children (in this case also get deformity), but occurs after fusion of the epiphyses. Rare in West except in renal disease, Asian women and malabsorption. Subclinical disease is more common e.g. people with a poor diet and limited sun exposure.

Clinical features
Vague symptoms of bone or muscle pain, weakness and lethargy. Rarely pathological stress fractures and deformity, waddling gait due to marked proximal myopathy, tetany and other hypocalcaemic signs. Insidious onset.

Ricket’s – genu varum, rickety rosary (bossing of costochondral junctions), Harrison’s sulcus, thickening of epiphyseal plate.

Pathology
Decreased vit D causes defective calcium absorption thus secondary hyperPTH. This leads to bone resorption (stimulates both osteoclasts and osteoblasts) in attempt to normalise serum calcium.
This fails with ongoing Vit D deficiency so get progressive demineralisation of bone, which weakens bone. Get Looser’s zones – areas of unmineralised bone, risk of developing fracture.

**Aetiology**

**Inadequate dietary intake of vit D** – poor diet (found in oily fish), secondary to malabsorption.

**Inadequate exposure to sunlight** – esp Asian women.

**Renal disease** – fail to hydroxylate vit D.

**Inherited or acquired disorders of vit D metabolism** – rare, includes vit D resistant rickets (type 1 - can’t hydroxylate vit D, type 2 – problems with receptor).

**Aluminium intoxication** – inhibits mineralisation.

**Drugs** – anticonvulsants (affect vit D metabolism and toxic to osteoblasts), bisphosphonates (inhibit mineralisation).

**Hypophosphataemic rickets** – normal vit D levels but renal phosphate wasting. Usually due to inherited gene defects (some recognised e.g. X linked HR and autosomal dominant HR) but may be also due to tumour secretions.

**Investigation**

Renal function

Biochemistry in vit D deficient rickets: calcium and phosphate – normal or low, alk phos – high, PTH – high, vit D low (raised in type 2 vit D resistant rickets). Renal osteodystrophy is same except raised phosphate and normal vit D.

Hypophosphataemic rickets – very low phosphate, phosphaturia, raised alk phos, normal vit D and low-normal calcium.

Xray, DEXA, bone biopsy (definitive diagnosis – increased thickness of osteoid seams).

**Treatment**

Monitor serum calcium and phosphate.

Vit D and calcium supplements.

If due to renal failure or type 1 vit D resistant rickets need to give active metabolites.

Hypophosphatamic rickets – give phosphate and vit D.

Corrective osteotomies if deformity.

**Paget’s disease**

**Pathology**

Abnormal remodelling of bone and accelerated bone turnover (increased activity of osteoclasts and osteoblasts) - high rates of bone resorption (osteolytic phase) and disorganised immature new bone formation (woven bone). Increased vascularity of bone, decreased strength of bone.

Most commonly axial skeleton – femur, pelvis, tibia, lumbar spine, scapula, skull.

Affects older people – maybe up to 10% over 85s. More common in Caucasians, esp in North. Proportion have AD inheritance.

**Clinical features**

**Bone pain** – typical features of bony pain but not well localised – spreads to affect whole bone.

**Thickening of bone** – nerve root pain, deafness, cranial nerve compression.

**Pathological fractures and pseudofractures** – healing can be difficult.

**Deformity** - tibial bowing (sabre tibia, varus and procurvatum), skull bossing.

**Hypercalcaemia** – esp with immobilisation.

**Osteosarcoma** – rare complication, esp if had for many years.

Some present incidentally by Xray or biochemical testing.

**Complications** – fractures, cord compression, high output cardiac failure (increased vascularity of bone), hypercalcaemia, haemorrhage from bones.
Investigation

**Xray** – shows single disordered bone with generalised sclerotic changes, mosaic pattern.

**Isotope bone scanning** – increased uptake.

**Biochemical** – raised alk phos, increased urinary hydroxyproline (shows increased bone turnover, not very sensitive or specific), normal calcium and phosphate, normal or raised PTH, normal vit D.

Treatment

Mainly to control bone pain – analgesia, bisphosponates, salmon calcitonin.

Monitoring of bone activity and alk phos level.

Surgical correction – need fixation of bone as poor healing.

**Osteonecrosis**

**Pathology**

Due to loss of blood supply to the bones. Get necrosis of bone and bone marrow, then granulation tissue. If articular stress exceeds structural integrity get collapse of joint surface.

Causes:

**Interruption of extraosseous blood supply** – trauma, vasculitis.

**Interruption of intraosseous sinusoidal circulation** – nitrogen bubbles, sickle cell disease, thrombi, fat emboli.

**Extravascular compression of sinusoidal circulation** – nitrogen bubbles, Gaucher’s cells, malignant cells, high dose corticosteroids, alcoholism.

**High dose steroids** - most common cause due to fat redistribution and extravascular compression of vessel by fat in bone marrow.

**Bisphosponate therapy** – can cause osteonecrosis of jaw, especially if dental surgical procedure.

Typically affects femoral head, femoral epiphysis (Legg Calve Perthe’s disease), femoral condyle, tibial plateau, humeral head, talus, scaphoid (Prieser’s disease), lunate.

**Clinical features**

**Pain** - at rest and on movement, night pain due to increased osseous pressure.

Initially well localised typical bony pain with subacute onset, then subchondral collapse, interference with joint and joint pain and arthritis.

**Joint dysfunction** – limited movement, stiffness, muscle spasms.

Insidious onset.

**Investigation**

MRI shows early changes, later subchondral lucency on Xrays (crescent sign – represents subchondral fracture).

**Management**

**Prevention.**

Surgical intervention e.g. to stimulate revascularisation.

Arthroplasty.

**DISH**

Diffuse idiopathic skeletal hyperostosis.

Condition of older people, especially men, obese people and diabetics, with non-inflammatory new bone formation, especially at entheses and in spine. Usually asymptomatic, can get pain if ossifying enthesopathy e.g. calcaneal spur.
Musculoskeletal Malignancy

Bone metastases and multiple myeloma

Bone metastases

Commonest sites - vertebral body, pelvis, ribs, upper ends of femur and humerus, skull, scapulae.

Common sources of metastases – from carcinomas, breast, prostate, thyroid, bronchus, bowel, kidney.

Presentation – bone pain, swelling if in a superficial bone (can be hard or soft), pathological fracture. May also have hypercalcaemia (bone destruction) or anaemia, neutropenia, thrombocytopenia (bone marrow infiltration), spinal cord compression.

Multiple myeloma

Pathology - neoplasm of plasma cell. Get clonal proliferation of malignant plasma cells in bone marrow. These stimulate osteoclasts so get lytic lesions. More common in males.

Commonest sites – skull, clavicle, vertebrae and ribs.

Clinical features - most present with bone pain. 25% have no clinical or imaging signs of bone disease at presentation. Symptoms due to complications e.g. anaemia, immunosuppression, renal failure, hyperviscosity, heptato-splenomegaly, bleeding or hypercalcaemia. Mainly older people.

Both may present with secondary symptoms e.g. weight loss, pain, fatigue.

Differential diagnosis

Symptoms and radiological features may also be produced by Paget’s disease, osteoporosis, osteosarcoma or osteoma, osteomyelitis, hyperparathyroidism.

Investigation

Myeloma - Bence-Jones protein in urine (monoclonal light chains), serum electrophoresis (for Ig secreted by plasma cells – single monoclonal band), FBC (Hb and WBC normal or decreased with MM), ESR greatly increased, serum calcium and alk phos, beta-2-microglobin (level is prognostic). Lytic lesions on radiology (need skeletal survey) with generalised osteoporosis and punched out lesions (pepper pot skull), typically endostial scalloping, cold on bone isotope scan (decreased uptake, can get false negatives) Diagnosis with bone marrow aspirate.

Metastases – PSA (prostate metastasis), serum calcium (raised), alk phos (raised), acid phos (raised with prostate mets), albumin, ESR (raised). Full skeletal survey, isotope bone scan (increased uptake), MRI, CT - metastases may show as lytic (poor margin definition, pathological fractures, most mets) or sclerotic (increased density, prostate and some breast) deposits.

Management

Supportive management of lesion:

Analgesia and nerve blocks for bone pain.
Radiotherapy if single lesion – helps pain.
Bisphosphonates – inhibit resorption, treat hypercalcaemia (along with increasing fluid intake).
Prevent pathological fractures by internal fixation, surgery to spine if unstable or neurological compromise.

Management of complications:

Treat anaemia, infection or renal failure.
If spinal cord compression – steroid therapy, analgesia, radiotherapy, surgical decompression, chemotherapy, hormone therapy.

Corrective management:

Radio, chemo or hormone therapy.
Bone marrow transplant for multiple myeloma.
Treat primary if metastasis.
**Primary tumours of bone**

**Benign tumours** – osteochondroma (esp around knee), osteoid osteoma, endochondroma (esp in hand), fibrous dysplasia (ground glass appearance on xray).

**Malignant tumours:**
Relatively rare, mostly in children.
Sarcomas – many different types, osteosarcoma, Ewing’s sarcoma, chondrosarcoma.
Definitive diagnosis by bone biopsy.

**Osteosarcoma** – especially around knee and proximal humerus, get lung mets so do CXR. Elevated periosteum gives Codmann’s triangle, also get sunray spicules and onion skinning on xray.

**Treatment** – surgery and multidrug chemotherapy.

**Multisystem Connective Tissue Diseases**

**Clinical features**
Autoimmune diseases, most common in young women, mainly targeting connective tissue and blood vessels. Most are inflammatory (except systemic sclerosis). Multifactorial aetiology – genetic, immunological (esp autoantibodies) and environmental factors.

**Causes of multisystem disease** – connective tissues diseases, vasculitis, infection, malignancy. MSCTD can overlap, in reality often have features of several diseases – mixed connective tissue disease.

Often start non-specifically and diagnosed late.

**Factors suggesting CTD** – symptoms affecting more than 1 system (especially if inflammatory), Raynaud’s, joint and skin symptoms, dry eyes and mouth, photosensitivity, alopecia, vasculitis, psychiatric history (esp lupus), miscarriages, thrombosis, pleurisy, pericarditis, anaemia, raised inflammatory markers, tiredness, unexplained fever.

**Concerning features** – systemically unwell, end organ damage (e.g. abnormal U&Es), vasculitis.

**Investigation**

**BP and urinalysis** – high rate of renal disease, glomerulonephritis with SLE and vasculitis.

**Bloods** – U&Es and LFTs (internal organ involvement), FBC (often mild normochromic normocytic anaemia, thrombocytopenia and lymphopenia), raised inflammatory markers (not in systemic sclerosis or anti-phospholipid syndrome), CPK (if muscle involvement), low complement (active vasculitis, SLE), cryoglobulins (SLE, infection esp Hep C, malignancy). CXR – small pleural effusion.

**ANA** – positive in virtually all cases of SLE but not diagnostic. Also positive with other CTDs (esp systemic sclerosis), infections, drug induced, healthy individuals. More significant if high titre and IgG.

**Other antibodies** – double-stranded DNA (more specific for lupus but false –s), Ro/La (Sjogren’s syndrome, SLE), centromere (systemic sclerosis esp limited cutaneous), Scl 70/topoisomerase 1 (systemic sclerosis esp diffuse), anticardiolipin (phospholipid syndrome), ANCA (vasculitides). Muscle histology and EMG.

**Schirmer tear test** - for Sjogren’s, measures flow of tears using absorbent paper strip.
Management
Mainly relies on corticosteroids and immunosuppressive drugs.

Systemic Lupus Erythematosis (SLE)
Pathology
Typically joints, skin, Raynauds, kidney, brain, pleurisy, pericarditis.
Excessive B lymphocyte activity gives hypergammaglobulinaemia and thus immune complex formation. These are deposited in microvasculature and can trigger complement activation and inflammatory response. Get immune complex deposition at dermal-epidermal junction – can be seen by immunofluorescence (lupus band test).
Many different autoantibodies produced.
Can be induced by drugs e.g. hydralazine, minocycline procainamide – this is usually less severe and settle on withdrawing drug.
More susceptible to infection – decreased complement, abnormal immune function, steroid treatment.
Worry about bacterial meningitis.

9:1 F:M. More common in Afro-Carribean and Asians.

Clinical features
Most common presentation is Raynaud’s with arthralgia.

General – tiredness (at increased risk of hypothyroidism so consider this), fever, poor concentration.
Skin and mucous membranes – photosensitive butterfly (malar, sparing nasolabial folds) rash, alopecia, painful mouth ulcers, discoid lupus rash (scarring and pigment change), livido reticularis, photosensitivity, Sjogren’s, Raynaud’s (1/3), vasculitis.
Musculoskeletal – arthritis (rarely erosive but can be deforming – Jaccoud’s arthritis), tenosynovitis, arthralgia, avascular necrosis.
Cardiorespiratory – pericardial effusion, pleural effusion, interstitial lung disease, pleuritis, pericarditis, Libman-Sacks endocarditis (non-infective vegetations).
Renal – ankle swelling, proteinuria, glomerularnephritis. Renal biopsies – mesangial changes, proliferative glomerulonephritis, inflammation, necrosis, sclerosis and fibrosis.
Haematological – anaemia, leucopenia, thrombocytopenia due to antibody mediated destruction of blood cells. These also fall due to drug treatments – to differentiate look for raised ESR and anti ds DNA.
Neuropsychiatric – seizures, psychosis, chorea.

Discoid lupus – condition confined to skin, chronic cutaneous lesions in areas exposed to sun. Initially erythematous then thick scale. Also scarring alopecia.
Associated with antiphospholipid syndrome.

Flares:
May be triggered by sunlight, oestrogen containing contraceptives, infection and stress.
Markers of active disease – raised ESR, high anti-ds DNA, reduced complement.
Life threatening flares – renal and cerebral vasculitis.

Diagnosis – recognition of symptoms and identification of autoantibodies, very unlikely if ANA negative.

Management
Avoid precipitating factors – strong sun block, avoid certain drugs, aggressive treatment of infection (can cause flares), control of BP.
Monitor urinalysis and blood pressure.
**Drugs** – NSAIDS if arthritis, mild SLE - hydroxychloroquine, moderate SLE – corticosteroids and immunosuppressants, severe SLE (severe nephritis and cerebral disease) - steroids and pulse cyclophosphamide (most effective but toxic). Sometimes use other immunosuppressive drugs for severe disease.

5yr survival is 90%, early mortality due to organ failure (mostly renal) or sepsis, late mortality due to CV disease.

**Antiphospholipid syndrome**
Can be primary or secondary to connective tissue diseases, esp lupus.
Get thromboses (arterial and venous) and embolism, miscarriages and thrombocytopenia. Also get migraines.
Blood tests – anticardiolipin antibodies and lupus anticoagulant.
Skin – livido reticularis.

**Management** – anticoagulation, low dose aspirin for all patients, lifelong warfarin following thrombosis. Treat hypertension, hyperlipidaemia and diabetes. Avoid OCP and smoking.

**Systemic sclerosis and CREST**

**Pathology**
Excessive production of collagen and microvascular occlusion causing fibrosis and ischaemia of skin and organs (may involve GI tract, heart, kidneys and lungs). 90% also have Raynaud’s.
Rarely inflammatory, therefore steroids are not useful.
3x as common in women.

**Classification**
Limited cutaneous systemic sclerosis, just affects extremities and face. Same as CREST (Calcinosis, Raynaud’s, oEsophageal involvement, Sclerodactyl, Telangiectasia).
Diffuse cutaneous disease.

**Clinical features**

**Early signs** – non-pitting oedema and puffy fingers with thick tight skin in hand.
**Late** – sclerodactyly, teleangiectasia, tight skin and limited movement, calcinosis.

**Skin and mucous membranes** – scleroderma (coup de sabre is localised scleroderma), digital pitting (lose finger pulp), digital ulceration (esp over DIP and PIP), teleangiectasia, calcinosis. Skin thickening causes decreased movement and contractures.

**Characteristic facies** – loss of skin folds, pinched nose, small mouth.

**GI** – dysphagia and reflux (oesophageal dysmotility), small bowel dysmotility (causes small bowel bacterial overgrowth so malabsorption), colon dysmotility (pseudoobstruction, constipation).

**Cardiorespiratory** – pericarditis, myocardial fibrosis, pulmonary hypertension (mostly limited form), interstitial fibrosis (mostly diffuse form). Pulmonary complications are main cause of death.

**Renal** – accelerated hypertension (scleroderma renal crisis).

**Musculoskeletal** – flexion contractures, arthritis, myopathy.

**Management**
No disease modifying treatment.
Monitor BP and renal function.
Can treat certain features – control hypertension (esp by ACE inhibitors), cyclical antibiotics if small bowel overgrowth.
Prognosis depends on internal organ involvement. 5yr survival is 70%. Poor prognosis associated with older age, diffuse skin disease, proteinuria, high ESR, pulmonary hypertension.
Sjogren’s syndrome
Dryness of mucous membranes esp eyes (keratoconjunctivitis sicca) and mouth (xerostomia). Get lymphocyte infiltration, autoimmune destruction and exocrine failure of lacrimal and salivary glands. This can lead to conjunctivitis, dental caries, parotid swelling. Can also get vaginal dryness and decreased GI secretions.
Usually secondary to autoimmune diseases e.g. RA, SLE, chronic active hepatitis, PBC.
Primary Sjogren’s also involves systemic features, arthritis and Raynaud’s.
9:1 F:M.

Management – artificial tears and saliva substitutes. Need to monitor for development of lymphoid malignancy (40x increased lifetime risk).

Idiopathic inflammatory myopathies
Injury and necrosis of myocytes with regeneration and inflammation. Get proximal muscle weakness and pain. Often systemic features e.g. fever, weight loss, fatigue.
In late disease muscle may replaced by fibrosis and fat.
Onset either in child hood or in middle-aged to elderly associated with malignancy (esp dermatomyositis), esp lung, oesophagus, breast, colon and ovary.

Investigations – raised CK, EMG (confirm myopathy and exclude neuropathy), muscle biopsy (look for necrosis, inflammation and regeneration).

Polymyositis - symmetrical proximal muscle weakness due to muscle inflammation. Associated with malignancy in about 15% cases. Leads to dysphagia, dysphonia and respiratory weakness. Signs – macular rash on back and shoulders, fever, Raynaud’s, polyarthralgia, myocardiitis.

Dermatomyositis – proximal muscle weakness like polymyositis but get skin lesions. Facial heliotrope rash, periorbital oedema, Gottren’s lesions (erythematous papules over knuckles), rashes in shawl distribution, periungal telangiectasis, calcinosis, vasculitis.

Inclusion body myositis – older patients, more distal weakness, especially wrist and quads. CK less raised, may get myopathic and neuropathic changes on EMG.

Differential diagnosis – other myopathies e.g. MND, MG, MD, glycogen storage disease, endocrine causes e.g. Cushing’s, infection myositis. Also need to consider if drug-induced due to corticosteroids.

Management – physio, prednisolone, immunosuppression. Less successful in inclusion body as only some have inflammatory component.
In older people need to check for underlying malignancy.

Polymyalgia rheumatica
Stiffness and pain in shoulder and hip muscles, worst in morning. Shouldn’t get muscle wasting and passive ROM should be preserved. Raised ESR and possible systemic features e.g. weight loss, fatigue, night sweats.
Typically affects elderly, rare in under 60s. Mostly rapid onset of symptoms. Association with giant cell arteritis – about 15% of people develop this.
Dramatic response to prednisolone, gradually reduce dose to minimum level possible. If steroids can’t be withdrawn after 2yrs try steroid sparing agents e.g. methotrexate.
**Systemic vasculitis**

**Pathology**
Immune mediated inflammation of various vessels. Characteristically see inflammatory infiltrate of wall, often with fibrinoid necrosis. Can lead to stenosis, occlusion or aneurysms. Main problem is infarction and haemorrhage of internal organs.

Classified depending on size of vessel. Range from benign and self limiting to life-threatening. All rare except HSP and temporal arteritis. Mostly more common in women.

**Clinical features**
Due to local tissue ischaemia (from vessel inflammation and narrowing) and systemic effects due to inflammation.
Skin – splinter haemorrhages, nail fold infarcts, purpura.

**Investigations**
Tissue biopsy (skin, nasal septum, muscle), urinalysis (prognosis mainly depends on renal involvement), visceral angiography. ANCA – c-ANCA with Wegener’s, p-ANCA with microscopic polyangitis, Churg-Strauss, polyarteritis nodosa.

**Management**
Steroids (mostly high dose at first), may need addition of immunosuppressant drugs or cyclophosphamide. Start treatment as early as possible to prevent irreversible damage.

**Large vessels:**
- **Takayasu’s** – including aorta and it’s branches. Get headaches, dizziness, visual disturbance, limb claudication, decreased pulses, arterial bruits. Typically young women. May need vascular surgery.
- **Giant cell/temporal arteritis** – affects branches of temporal and ophthalmic arteries. Typically thickened tender temporal artery. Get headache and scalp tenderness, jaw claudication, visual disturbance. Can lead to blindness (ischaemic optic neuritis due to arteritis of post ciliary artery) or stroke, medical emergency, needs high dose steroids. Ideally get temporal artery biopsy, may get false negatives due to skip lesions.

**Medium vessels:**
- **Behcet’s** – mouth and genital ulceration, erythema nodosum, arthritis, retinal vasculitis, hyperreactivity to minor trauma (pathergy). Especially in Japan and Mediterranean.
- **Kawasaki** – children, coronary arteries.

**Small vessels:**
- **Wegener’s** – main problem is respiratory and renal complications. URT infections (sinusitis, otitis media), epistaxis, scleritis, arthralgia, fever and weight loss, pulmonary nodules and haemorrhage, proptosis, glomerulonephritis. Granulomas can cause destruction and compression of structures.
- **Churg-Strauss** – vasculitis with asthma and eosinophilia. Also get pneumonia, heart failure, peripheral neuropathies, mesenteric vasculitis. SLE and RA related.
- **Microscopic polyangitis** – rapidly progressive glomerulonephritis and often pulmonary alveolar damage. Can get cutaneous involvement and neuropathy.
- **Other small vessel vasculitis** – mainly affects skin with purpuric rash. Due to HSP (nephritis, arthritis and purpura), drug hypersensitivity, malignany, CTDs, cryoglobulinaemias, infection. Good prognosis.
**Inherited conditions**

**Marfan’s** – skeletal disproportion (arm span greater than height), arachnodactyly (long, thin fingers), sternal depression, generalised hypermobility, lens dislocation, high arched palate. Complications include mitral valve prolapse, aortic incompetence and aortic dissection. Due to mutations in fibrillin gene.

**Ehler-Danlos** – generalised hypermobility, skin laxity, scoliosis, visceral vascular catastrophes. Mutations in several genes.

**Chronic Locomotor Conditions**

**Childhood locomotor disease**

**Normal variants** – flat feet, toe walkers, in-toeing (femoral anteversion, tibial torsion, metatarsus adductus), genu varum (toddlers), genu valgum (young children). Reassuring if symmetrical.

**Osgood-Schlatter disease**
Common, benign self-limiting knee pain due to periostitis of proximal tibial tuberosity, with tender swelling over insertion of patella tendon. Usually traumatic origin in active teenagers.

**Developmental dysplasia of the hip**
Same as congenital dislocation. Strong familial tendency, more common in females and breech babies. Need early diagnosis (neonate by Barlow and Ortolani manouvres, leg length discrepancy, US) so can treat, otherwise get early OA. Mostly present by screening, otherwise with limp, leg length discrepancy, asymmetric posterior skin creases, loss of abduction. Management – as neonate usually reducible manipulatively, maintain with splint and Pavlik harness. If later diagnosis need open reduction and osteotomies, usually worse outcome.

**Irritable hip**
Common in children up to 5. Transient synovitis following minor viral illness. Get hip pain and limp in a well child, no pain at rest. Must rule out septic arthritis if in doubt. Manage by rest and analgesia, self-limiting.

**Perthes’ disease**
Avascular necrosis of femoral head, prior to skeletal maturity. Especially boys aged 4-11. Get pain, insidious onset limp and decreased range of movement, lose abduction if subluxation. May see deformity on xray e.g. fragmentation of epiphysis, subluxation. 15% are bilateral. Management – controversial, can do surgery to hold hip in acetabulum, otherwise bracing and physio. Better prognosis in younger children and only part of head.

**Slipped upper femoral epiphysis**
Progressive translation of femoral head on femoral neck through epiphysis, during growth spurt. Typically boys aged 9-14, especially if hypogonadal or obese. Get hip or knee pain and limp. Request frog lateral xray. Do surgery to pin epiphysis, usually bilaterally as often both sides affected. Complications – avascular necrosis and premature fusion of epiphysis.
Club foot (talipes)
Congenital talipes equinovarus – shortening of Achille’s, ankle flexion, hind foot varus.
Congenital talipes calcaneovalgus – usually corrects easily.
Manage by strapping if correctable or surgery.

Pes planus
Flat feet – can be mobile (arch reforms on tiptoes) or rigid (older children, 2 bones fuse).
Mostly painless and need no treatment.

Pes cavus
High arched foot, often with claw toes.
Gives forefoot pain due to high pressures over metatarsal heads.
Can be idiopathic or due to neurological disorders giving muscle imbalance.
Management – shoes, surgery (osteotomy).

Cerebral palsy
Main problems – muscle spasticity and weakness, poor motor control, secondary bony deformities.
Tend to walk with scissor gait (tight hip adductors) and tiptoe walking (tight heel cord).
Management – physio, stretching, splints, antispasmodics.
Surgery to try to restore muscle balance – lengthen, divide or transfer tendons e.g. adductor tenotomies. Also surgery for treatment of secondary bone and joint deformities e.g. neuromuscular scoliosis.

Charcot joint
Accelerated joint damage due to neuropathy.
May be due to loss of pain sensation so injure joint. Also neuropathy affects SNS so vasodilate and increase blood flow to cartilage and bone causing hypervascularity and joint damage.
Can get rapid joint and bone destruction so joint becomes disorganised. Initially painful then flail painless joint. See gross destruction on xray.

Mainly palliative treatment with emphasis on joint protection.

Causes:
Diabetes – mostly fore, mid and hindfoot.
Syphilis – knee and spine.
Syringomyelia – shoulder, elbow, wrist.

Algodystrophy
Also known as complex regional pain and reflex sympathetic dystrophy.
Combination of severe neurogenic pain (typically burning and hyperaesthesia), vasomotor instability (autonomic disturbance – limb may go pink, purple, white - cold intolerance), swelling and stiffness.
May also have trophic changes and often local osteoporosis.
Usually an exaggerated response to injury esp in hand and foot, following nerve injuries or fractures.
Try to prevent by early mobilisation.
Rehabilitation

= the combined and co-ordinated use of medical, social, educational and vocational measures for training or retraining the individual to the highest possible level of functional ability. Aim to minimise impact of impairments on patient, restore and sustain optimal autonomy and participation and avoid additional obstacles. Simultaneous interventions at levels of pathology and impairment, activity and participation.

In terms of history, need detail in social history – accommodation, transport, family, employment, education, hobbies, sports, roles. May need supplementation from relatives and other staff. Also need information about functioning especially washing, toileting, feeding, transferring, dressing.

Performance – how person is actually capable of performing activities.
Capacity – what they could be capable of with optimum environment and rehabilitation.

International Classification of functioning, disability and health

Impairment – deviation from normal structure of function e.g. amputation.
Activity and handicap – inability to perform an activity e.g. can’t walk.
Participation restriction – the effect on life e.g. can’t go to the shop.

These need to be considered separately as the same impairment affects different people in different ways and the same handicap will have different effects on people’s lives.

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Pathology and impairment – the cause e.g. a stroke, and what they cannot do e.g. dysphasia, hemiparesis. Can still rehabilitate even if this is unknown e.g. rare neurodegenerative diseases.
Activity – execution of task or action e.g. walking, feeding. This includes basic ADL.
Participation – involvement in life situation, how activities relate to the person’s lifestyle e.g. walking to a football match, eating in a restaurant. This includes roles, interests, hobbies, employment and domestic life. Divide into learning, general tasks, communication, mobility, self-care, domestic, interpersonal interaction and relationships.

Environment – affects all aspects. Includes social, financial and attitudes as well as physical environment. Barriers and facilitators.
Risks and prevention – chance of pathology recurring or complications e.g. to prevent another stroke or contractures. Typically include falls, injury, depression, pressure sores, DVT, isolation and vulnerability, contractures, epilepsy, constipation, chest infection.

At each level consider goals that are important to patient and methods to achieve these goals.

Management of specific problems
Can target pathology and impairment or activity and participation.

Mobility – manage spasticity and prevent contractures, walking aids to carry part of load or help stability (e.g. sticks, crutches, frames), orthoses (e.g. for foot drop), shoes or insoles, hip replacement etc.
Speech – SALT, communication aids.
Diplopia – patched eye glasses, corrective surgery.
Difficulty reading – glasses, audiobooks, large print books, magnifiers.
Ataxia – special cups etc, lycra splints to fix distal parts of limbs, anti-epileptic medication (valproate, clonazepam, gabapentin).
Pain – need to treat this early. Treating pain increases speed of rehabilitation.
**Falls**  
Risk of fall – intrinsic factors e.g. neuromuscular function, vision and hearing, balance; extrinsic factors e.g. environmental hazards, drugs (diuretics, antihypertensives), urinary incontinence.  
Risk of fracture – force of impact, strength of bone.

**Pressure sores**  
Area of localised tissue death of previously healthy tissue, may involve underlying structures.  
**Typical areas** – heel, lateral malleolus, knee, ischium, greater trochanter, sacrum, elbow, scapula, occiput.

**Causes** – immobility (direct unrelieved pressure), friction or shearing (spasms, sliding in chair, dragging whilst transferring), malnutrition, inadequate seating.  
**Risk factors** – immobility or sensory impairment (e.g. post-op, pain, comatose, diabetic, spinal cord injury), concurrent disease (anaemia, diabetes, ischemia, malignancy), poor nutrition, poor oxygenation and blood supply, moisture, extremes of weight, incontinence.  
Score risk with Waterlow score.  
Stage 1 – superficial, redness that doesn’t subside when pressure relieved, epidermis intact.  
Stage 2 – damage extends into dermis.  
Stage 3 – full thickness of skin, into subcut tissue. Poor blood supply so difficult to heal.  
Stage 4 – extends into other structures.

**Prevention** – remove sources of irritation, frequent position changes, education, pressure reducing bed or chair, adequate nutrition, treat infections, encourage mobility, ensure not sitting on creases or catheter etc.  
**Early recognition** - frequently inspect skin.  
Keep weight off sores whilst healing.

**Complications** – septicaemia, anaemia, amyloidosis, osteomyelitis, fistulae, death. Significant morbidity.

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**Important Note**  
These notes were written by Liz Tatman, as a fourth year medical student in 2006. They are presented in good faith and every effort has been taken to ensure their accuracy. Nevertheless, medical practice changes over time and it is always important to check the information with your clinical teachers and with other reliable sources.  
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