

Medicine

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Chapter 1

Cardiology

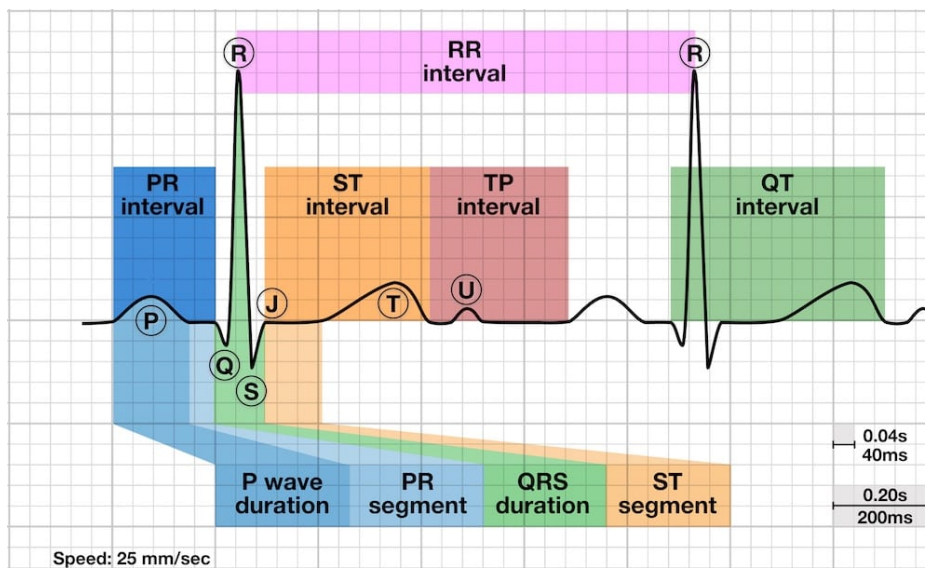
1.1 Presenting problems in CVS disease

Features of benign murmur

- Soft
- Midsystolic
- Heard at left sternal edge
- No radiation
- No other cardiac abnormalities

1.2 ECG

Anatomy of an ECG



Abnormalities of components

Pathological Q

- Depth $> 2\text{mm}$
- Height $> 1\text{mm}$
- Present in ≥ 2 leads
- Assocd with loss of R height ($Q > R/4$; normally $Q \leq R/4$)
- Indicates *transmural* myocardial necrosis

Segments vs intervals

- e.g. ST segment = end of S \rightarrow start of T
- PR interval = start of P \rightarrow start of R

ST segment elevation

- Normal: upto 1mm in limb leads, upto 2mm in chest leads
- Causes
 - **STEMI: convexity** upwards
 - **Acute periCARDitis:: conCAvity** upwards
- Indicates ongoing myocardial injury

Myocardial infarction

A somewhat interesting physiological explanation on how the changes arise

Sites of infarction based on lead

- Septal: V_1, V_2
- Anterior: V_3, V_4
- Lateral: I, aVL, V_5, V_6
- Extensive anterior: V_1-V_6
- Anterolateral: I, aVL, V_1-V_6

Reciprocal changes

- Acute STEMI in some surface of the heart \rightarrow ST elevation in corresponding leads, and ST depression in reciprocal leads

Site	Facing	Reciprocal
<i>Septal</i>	V_1, V_2	V_7, V_8, V_9
<i>Anterior</i>	V_3, V_4	None
<i>Lateral</i>	I, aVL, V_5, V_6	II, III, aVF
<i>Inferior</i>	II, III, aVF	I, aVL
<i>Posterior</i>	V_7, V_8, V_9	V_1, V_2

Basic pathophys of STEMI

- Occurs due to proximal **complete occlusion of major coronary artery**



A. Before the onset of infarction

↓

B. In acute phase, ST elevation

↓

C. Progressive loss of R and deepening Q

↓

D. Resolution of ST elevation; fully developed pathological Q; T inversion

↓

E. In old infarcts, T-wave inversion may or may not persist

- ST elevation resolves after a few days

NSTEMI

- **Partial occlusion of major or complete occlusion of minor** coronary artery
- *Subendocardial/partial-thickness MI* → **no pathological Q**
- **ST depression + T inversion** in chest leads

1.3 Coronary Artery Disease

- Diseases arising due to narrowing of the lumen of one or more coronary arteries and the resulting ischaemia/infarction of the myocardium or the conductive system.
- **Types:**
 - Stable angina: Fixed atheromatous stenosis
 - Unstable angina:
 - * dynamic obstruction
 - * due to plaque rupture/erosion with thrombosis
 - MI
 - Heart failure
 - Arrhythmia
 - Sudden cardiac death
 - * ventricular arrhythmia
 - * asystole
 - * massive MI

1.4 Arrhythmias**Classification according to ECG morphology**

- **Narrow complex:** QRS < 120ms (3 small sqs)
 - Sinus tachycardia
 - Atrial fibrillation (irregular narrow complex tachycardia)
 - Atrial flutter
 - AV Nodal Re-entry Tachycardia (AVNRT aka SVT)
- **Broad complex:** QRS > 120ms (3 small sqs)
 - Ventricular tachycardia
 - AV Re-entry Tachycardia (AVRT e.g. Wolff-Parkinson-White syndrome)
 - * Abnormal band of conductive tissue connecting atria and ventricles (accessory pathway)

Management of SVT

- Carotid sinus massage or
- Valsalva manoeuvre
- If the manoeuvre fails,

- Adenosine (3-12mg IV) or
- Rate-limiting CCB (Verapamil 5mg IV) or
- β -blocker
- If haemodynamic state compromised, DC cardioversion
- Recurrent SVT \rightarrow catheter ablation

1.5 Atrial fibrillation

Causes

- CAD (including acute MI)
- Mitral stenosis (MS; rheumatic mitral valve disease)
- Hypertension
- Thyrotoxicosis
- Cardiomyopathy
- Pulmonary embolism

Investigations

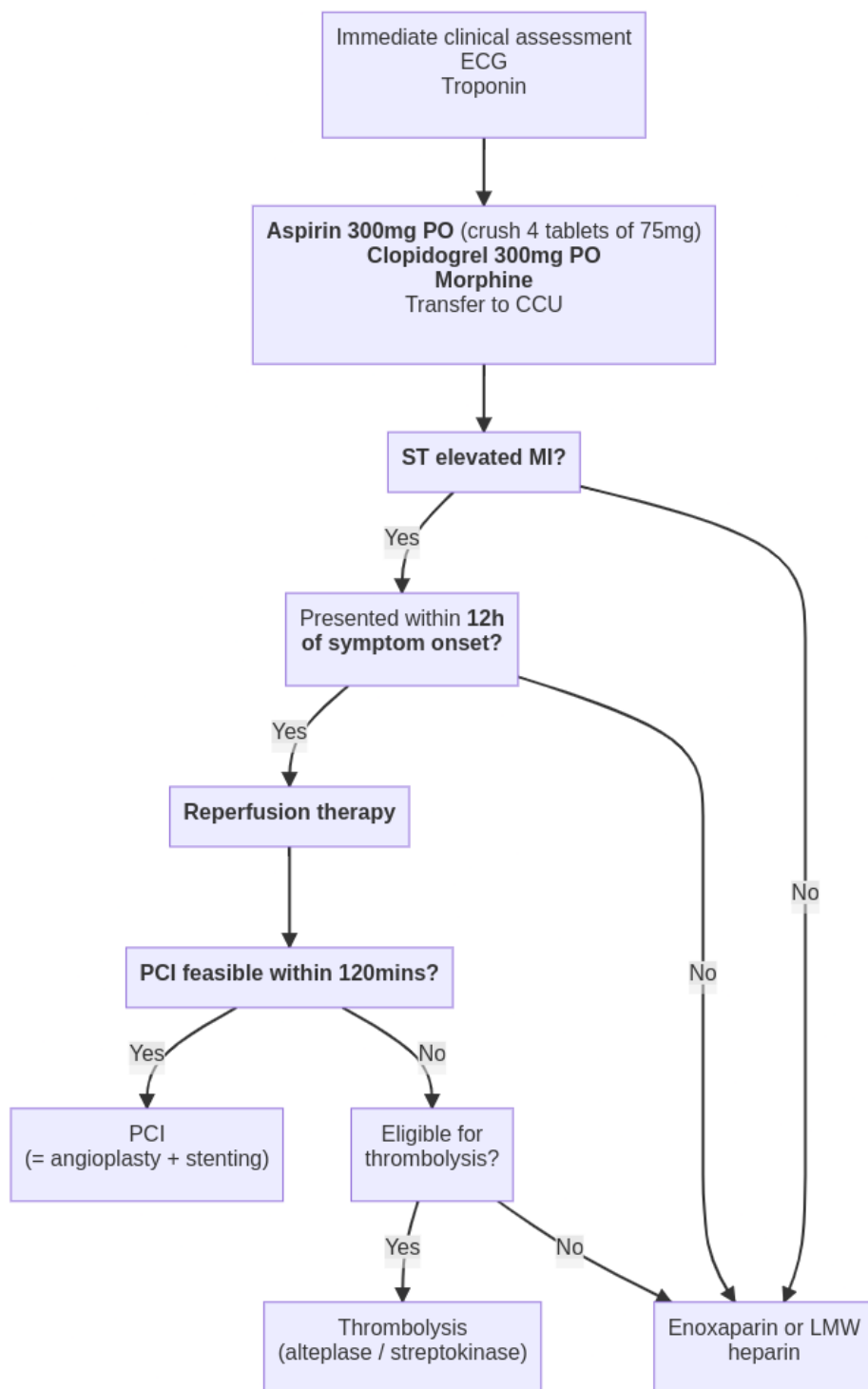
- ECG
- Echo: to see valvular condition
- Thyroid function test: to exclude thyrotoxicosis

Management of AF

- **Rhythm control:**
 - Pharmacological cardioversion
 - * Pt stable + no history of heart disease \rightarrow IV flecainide
 - * Structural / ischaemic heart disease \rightarrow IV amiodarone
 - DC cardioversion if drugs fail
- **Rate control**
 - β -blockers
 - Digoxin
 - Rate-limiting CCB: verapamil / diltiazem
- **Thromboprophylaxis:**
 - Oral Warfarin
 - Target INR: 2.0-3.0
 - Reduces risk of stroke by $\frac{2}{3}$
 - Start 4wks before cardioversion, continue till 3mo after successful cardioversion

1.6 Myocardial Infarction

Management of acute MI



Chapter 2

Dermatology

2.1 Anatomy and physiology

- Layers of skin:
 - Epidermis: further layered into (from out→in)
 - * corneum
 - * lucidum
 - * granulosum
 - * spinosum
 - * basale
 - Dermis: contains
 - * blood vessels
 - * nerves
 - * pilosebaceous units (hair follicle + sebaceous gland)
 - Subcutis: adipose

Epidermal appendages

- Hair follicles:
 - phases of growth
 - * anagen:
 - active growth
 - lasts years in scalp hairs
 - * catagen:
 - transitional
 - lasts days (in scalp)
 - * telogen:
 - resting
 - lasts months (in scalp)
- Sebaceous glands
 - usually *associated with a hair follicle*

- androgens $\rightarrow \uparrow$ sebum
- oestrogen $\rightarrow \downarrow$ sebum

- Sweat glands
 - innervated by *sympathetic cholinergic* fibres

2.2 Principles of management of skin disease

Topical treatments

- Ointments vs Creams
 - Ointments preferred to creams for dry skin (e.g. chronic eczema) as
 - * more hydrating
 - 80% oil + 20% water in ointments (vs 50-50 for creams) \rightarrow prevent water loss from skin by oil layer
 - * less preservatives \rightarrow less risk of allergy
- Emollients
 - Moisturise, lubricate, protect skin
 - *Vehicles without active drug*
- Glucocorticoids

Phototherapy

- UVB
- Psoralen UVA
 - Psoralen:
 - * natural photosensitiser from plant source
 - * cross-link DNA strands on excitation with UVA
 - Cumulative exposure to PUVA $\rightarrow \uparrow$ risk of SCC, so reserved for UVB resistance
- Uses
 - Psoriasis
 - Atopic eczema
 - Vitiligo
 - Chronic urticaria

Systemics

- Antihistamines
- Retinoids
 - *Anti-inflammatory*
 - Promote *differentiation of skin cells*

- **Teratogenic**
 - * must be prescribed with robust contraception
 - * females must have negative pregnancy test before, during, and after therapy
- **Immunosuppressants**
 - Glucocorticoids e.g. prednisolone
 - Methotrexate
 - Azathioprine

Biologics

- Biological *inhibitors of proinflammatory cytokines*
- **TNF- α inhibitors**
 - Infliximab
 - Etanercept
- **Interleukin inhibitors**
 - Ustekinumab: IL-12, 23
 - Guselkumab: IL-23
 - Secukinumab: IL-17
- *Rituximab*:
 - Binds to CD20 \rightarrow cause ADCC of B cells
 - As terminally differentiated plasma cells don't have CD20 they're safe
 - Use: pemphigus vulgaris

Non-surgical therapy

- **Cryo**
 - *Liquid N₂*
 - Causes cell membrane destruction \rightarrow death
- Laser
- PDT / photodynamic therapy

2.3 Skin cancers

Classification

- Non-melanoma skin cancer (NMSC): most common
 - SCC
 - BCC
- Melanoma
 - Less common
 - More metastatic risk \rightarrow cause of most skin cancer deaths

2.4 Fungal infections

Types

- Superficial
 - Dermatophytes: aka **ringworm** / **tinea**sis
 - * *Trichophyton*
 - * *Epidermophyton*
 - * *Microsporum*
 - Yeast
- Deep: less common
 - Chromomycosis
 - Sporotrichosis

2.5 Scabies

Agent

Caused by the mite *Sarcoptes scabiei hominis*

Diagnosis

- Identify the skin burrow
- Visualize the mite by dermatoscope / extracting with a needle

Treatment

- Affected + all asymptomatic family members / physical contacts
- Topical permethrin / malathion
 - 2 applications
 - 1 wk apart
 - Whole body, except head
- Oral Ivermectin:
 - Single dose
 - For poor adherence, immunosuppression or heavy infestation

2.6 Acne

- *Chronic inflammation of pilosebaceous units*

Pathogenesis

Key components are:

- ↑ Sebum production
- Colonisation of pilosebaceous ducts by *Propionibacterium acnes*
- Occlusion of pilosebaceous ducts

Features

- Hallmark: **comedone**
- Greasiness of skin

Management

- **Mild disease**
 - Topical Benzoyl peroxide
 - Topical Retinoids
 - Topical antibiotics
 - * Erythromycin
 - * Clindamycin
- **Moderate disease:** topical *plus*
 - Systemic tetracycline
 - Oestrogen containing OCP
 - Isotretinoin: if inadequate response to topical+systemic therapy for 6 months
- **Severe disease**
 - Isotretinoin 0.5-1 mg/kg for 4 months:
 - * Reduce sebum secretion and follicle colonisation
 - * Teratogen
 - * Pregnancy must be avoided during treatment *and* within 2 mo of drug cessation
 - Systemic glucocorticoid (with isotretinoin)
 - If unable to use isotretinoin
 - * UVB phototherapy
 - * PDT

2.7 Eczemas

- Seborrhoeic dermatitis is associated with *Malassezia* yeasts

Features

Most types have the following clinical features:

Acute

- Ill-defined erythema, oedema
- Papules, vesicles, bullae
- Exudation
- Scaling

Chronic

- Above features
- Lichenification
 - Skin thickening with pronounced skin markings, 2° to chronic scratching
 - Fissures
 - Dyspigmentation

Management of eczema



2.8 Psoriasis

- Chronic inflammatory hyperproliferative skin disease
- **Characteristics**
 - **Well-defined erythematous scaly plaques**
 - Affecting **extensor surfaces, scalp, nails**

Histological features

- Keratinocyte hyperproliferation + abnormal differentiation → nucleated stratum corneum cells (transit time from basale to corneum reduced to 5 from 28 → keratinocytes reach the surface while immature)
- Inflammation with Th-1 and Th-17 infiltration
- Tortuosity of dermal capillaries and release of VEGF

Exacerbating factors

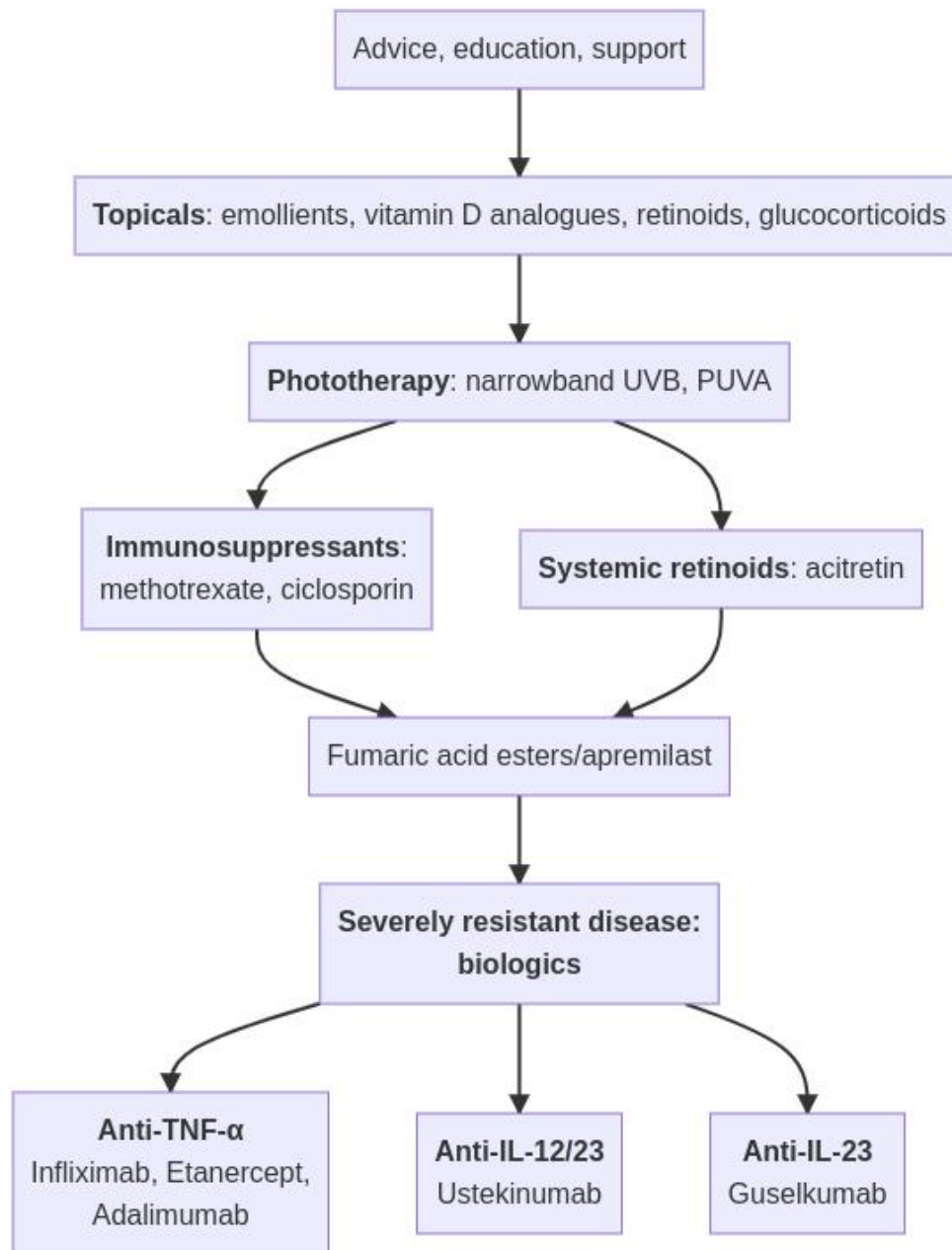
- **Sunlight**
- **Trauma**
- **Infection**
 - β -haemolytic strep ↑ guttate psoriasis
 - HIV may initially present with severe psoriasis
- **Drugs**
 - Antimalarials
 - β -blockers
 - Lithium
 - NSAIDs
- **Stress and anxiety**

Clinical types

- **Plaque psoriasis:**
 - most common
 - well-demarcated erythematous plaques
 - silver-white scales in untreated
 - * bleed on scraping (due to dilated vessels underneath) → **Auspitz sign**
 - **Sites**
 - * extensor surfaces
 - elbows
 - knees
 - lower back
 - * scalp
 - * nails

- **Guttate** psoriasis:
 - follows *Strep* throat
 - common in children/adolescent
 - UVB highly effective
 - may herald the onset of plaque psoriasis in adulthood
- **Erythrodermic** psoriasis: generalised → medical emergency
- **Pustular** psoriasis

Management of psoriasis



2.9 Hypopigmentation

Causes

- Vitiligo
- Albinism
- Pityriasis alba
- Pityriasis versicolor

Vitiligo

- **Acquired**
- Cell-mediated **autoimmune destruction of melanocytes**
- Loss of melanocytes → hypopigmented patches

Albinism

- **Autosomal recessive**
- **Reduced melanin production by normal number of melanocytes**
- ↑↑ risk of sunburn, skin cancer

2.10 Hyperpigmentation

Causes

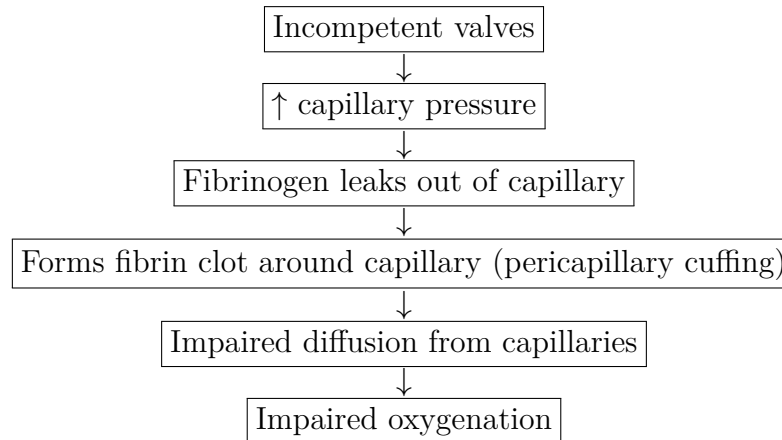
- **Endocrine**
 - Melasma/chloasma:
 - * in pregnancy / some OCP users
 - * discrete patches of facial pigmentation
 - Addison's disease
 - Cushing's syndrome
 - Nelson's syndrome
 - * hyper-ACTH 2° to bilateral adrenalectomy for Cushing's
 - * due to loss of -ve feedback from plasma cortisol
 - CKD
- **Drugs**
 - Amiodarone
 - Anti-cancers:
 - * Bleomycin: Hodgkin's
 - * Busulfan: CML
 - Chloroquine
 - Psoralens

2.11 Pseudorandom factoids

SPF (sun protection factor)

- $\frac{\text{UV dose for producing erythema with sunscreen}}{\text{UV dose for producing erythema without sunscreen}}$

Mechanism of venous ulceration



Chapter 3

Nephrology

3.1 UTI

Definition

Presence of $> 10^5$ organisms/mL in a mid-stream sample of urine.

Features

- **LUTI:** cystitis/urethritis
 - Frequency
 - Urgency
 - Dysuria (burning urethral pain during micturition)
 - Haematuria
 - Strangury (intense desire to pass more urine after voiding, due to spasm of inflamed bladder wall)
- **UUTI:** acute pyelonephritis
 - Fever with chills and rigor
 - Vomiting
 - Loin pain
 - Renal angle tenderness

Commonly involved pathogens

- *E. coli*: 75%
- *Proteus*
- *Pseudomonas*
- *Streptococci*
- *Staph. epidermidis*

Investigations

- Dipstick test for nitrites, leucocyte esterase, and glucose

- Most urinary pathogens (e.g. *E. coli*, *Proteus* etc) reduce nitrate to nitrite
- UTI → Neutrophils in urine → leucocyte esterase
- Microscopy for WBC and organisms
- Urine culture

Treatment

Cystitis

- **1st choice**
 - **Trimethoprim** (200mg bds 3 days)
 - **Nitrofurantoin** (50mg qds 3 days)
- **Pregnancy**
 - Nitrofurantoin (50mg qds 7 days)
 - Cefalexin (250mg qds 7 days)
- Avoid trimethoprim during pregnancy, and nitrofurantoin at term

Pyelonephritis

- **1st choice**
 - **Cefalexin** (1g qds 14 days)
 - **Ciprofloxacin** (500mg bds 7 days)
- Hospitalise if no response within 24h

Epididymo-orchitis

- *1st choice*: Ciprofloxacin

Acute prostatitis

- *1st choice*: Trimethoprim

Prophylactic measures in women with recurrent UTI

- Fluid intake *ge* 2L/day
- Regular complete bladder evacuation
- Emptying the bladder before and after intercourse
- Good personal hygiene
- Continuous prophylactic trimethoprim (100mg) and nitrofurantoin (50 mg) at night

Chapter 4

Rheumatology

4.1 Investigations of musculoskeletal disease

Joint fluid aspiration

- Normal:
 - Amount small
 - Viscosity high
 - Colourless / pale yellow
- Inflammation:
 - Amount raised
 - Viscosity lowered (due to enzymatic degradation of hyaluronan & aggrecan)
 - Turbid (due to neutrophils)
- Crystal-induced arthropathies
 - Crystals seen by polarised light microscopy
 - Urate crystals → long, needle shaped, -ve birefringence
 - Ca pyrophosphate crystals → small, rhomboid, +ve birefringence ### Bone scintigraphy
- Dx of metastatic bone disease and Paget's
- ⁹⁹Tc radiolabelled bisphosphonate used

DEXA (Dual Emission X-ray Absorptiometry)

- Measure BMD (bone mineral density)
 - $< -2.5 \rightarrow$ osteoporosis
 - Between -2.5 and -1 \rightarrow osteopenia
 - $> 2.5 \rightarrow$ high bone mass (most common cause osteoarthritis)

Immunology

- RF

- Antibody to Fc fragment of human Ig
- 70% sensitive for RA (if nodules & extra-articular manifestations then 100% sensitive); specificity poor
- **RF +ve diseases**
 - * Rheumatoid arthritis
 - * Sjogren's syndrome
 - * SLE
 - * Old age (> 65)
- **ACPA**
 - Antibody to peptides in which arginine has been converted to citrulline by peptidylarginine deiminase, an enzyme abundant in inflamed synovium.
 - 70% sensitive, >95% specific for RA
- **ANA** (antinuclear antibodies)
 - 100% sensitive for SLE but poor specificity
 - **ANA +ve diseases**
 - * SLE
 - * Sjogren's
 - * Systemic sclerosis
 - * Rheumatoid arthritis
- **Complement C3**
 - Active SLE \rightarrow \downarrow C3 (due to consumption of C3 by immune complexes)

4.2 Seropositive vs Seronegative arthritis

- Seropositive: RF+ inflammatory arthritis
 - Rheumatoid arthritis
 - SLE
- Seronegative: RF- inflammatory arthritis
 - Ankylosing spondylitis
 - Reactive arthritis
 - Psoriatic arthropathy

4.3 Osteoarthritis

- Characterised by
 - degeneration of articular cartilage
 - subchondral osteosclerosis
 - osteophyte formation at joint margin
 - enlargement of affected joint
- Sites

- hips
- knees
- PIPs
- DIPs
- cervical and lumbar spine
- Investigations:
 - X-ray of affected joint: findings described above in characteristics
 - MRI spine if spine OA + suspected root compression / spinal stenosis
- Treatment
 - Conservative:
 - * Wt loss
 - * Exercise
 - * NSAIDs
 - * Intraarticular glucocorticoids
 - Surgical: if refractory
 - * Total joint replacement
 - * Osteotomy

4.4 Spondyloarthropathies

- Asymmetrical oligoarthritis associated with HLA-B27 and typically involving the spine
 - Ankylosing spondylitis
 - Reactive arthritis
 - Psoriatic arthropathy
 - Axial spondyloarthritis
 - Entropathic spondyloarthritis (arthritis associated with IBD)
- Common features:
 - Asymmetric oligoarthritis
 - Sacroilitis
 - Enthesitis (inflammation where tendon attaches to bone)

Reactive arthritis

- “Reactive” to certain infections e.g. *Chlamydia*, *Campylobacter*, *Salmonella*, *Shigella*.
- **Reiter’s syndrome:**
 - Triad of *can’t see, can’t pee, can’t bend the knee*
 - * **Conjunctivitis**
 - * **Urethritis**
 - * **Reactive arthritis**
 - Due to *Chlamydia*

Chapter 5

Neurology

5.1 Raised ICP

- Normal ICP = **5-15 mmHg**

Causes

- **ICSOL**
 - Intracranial haemorrhage
 - Tumours e.g. glioma
 - Brain abscess
- **Hydrocephalus:** blockade of CSF circulation
 - Obstructive / non-communicating
 - Communicating
- **Cerebral oedema** e.g. meningoencephilitis
- **Venous sinus obstruction** e.g. cerebral venous thrombosis

Features

- **Headache**
- **Vomiting**
- **Diplopia / blurred vision:** Due to *6th nerve palsy*
 - 6th nerve palsy due to
 - * stretching of the long, slender nerve
 - * compression against petrous temporal bone
- **Papilloedema**
- **Bradycardia**
- **Hypertension**
- **Depressed consciousness**

Management

- According to cause:
 - Mass lesion → surgical decompression
 - Hydrocephalus → *ventriculoperitoneal shunt* operation
 - Oedema → glucocorticoids
- Supportive:
 - Head elevation
 - Fluid balance
 - BP control
 - Diuretics: mannitol

5.2 Neurological emergencies

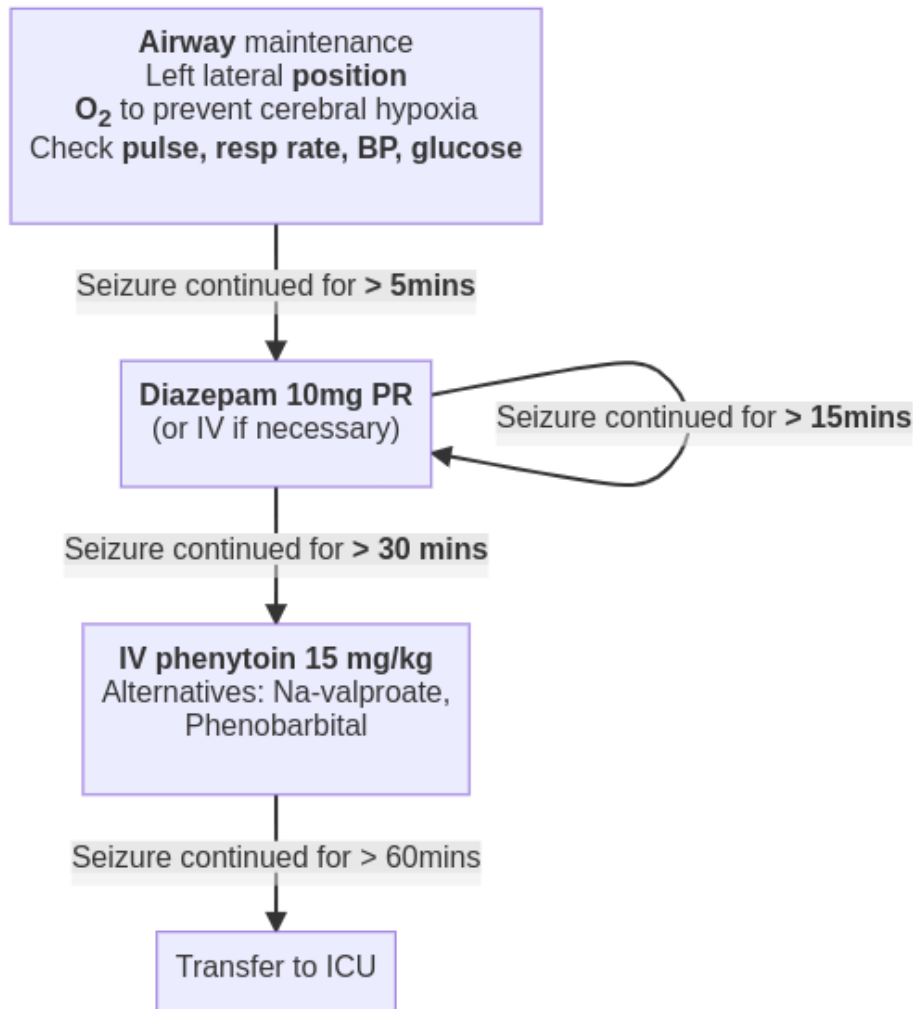
- **Status epilepticus**
- **Stroke** (if thrombo)
- **Subarachnoid haemorrhage**
- **Cord compression**
- **GBS**
- **Myasthenia gravis** (if bulbar and/or respiratory)

5.3 Status epilepticus

Definition

Continuous or recurrent **seizures** for ≥ 30 mins without **gain of consciousness** in between.

Management



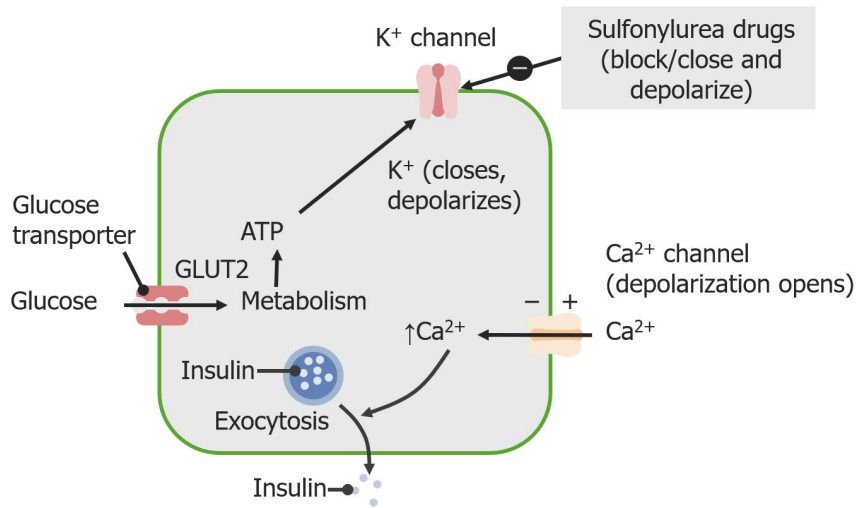
5.4 All jerks root values

- Biceps: C5
- Supinator: C6
- Triceps: C7
- Finger (aka Hoffmann test): C8
- Knee: L3, L4
- Ankle: S1, S2
- Plantar: S1 (technically not a jerk since it's a superficial reflex)

Chapter 6

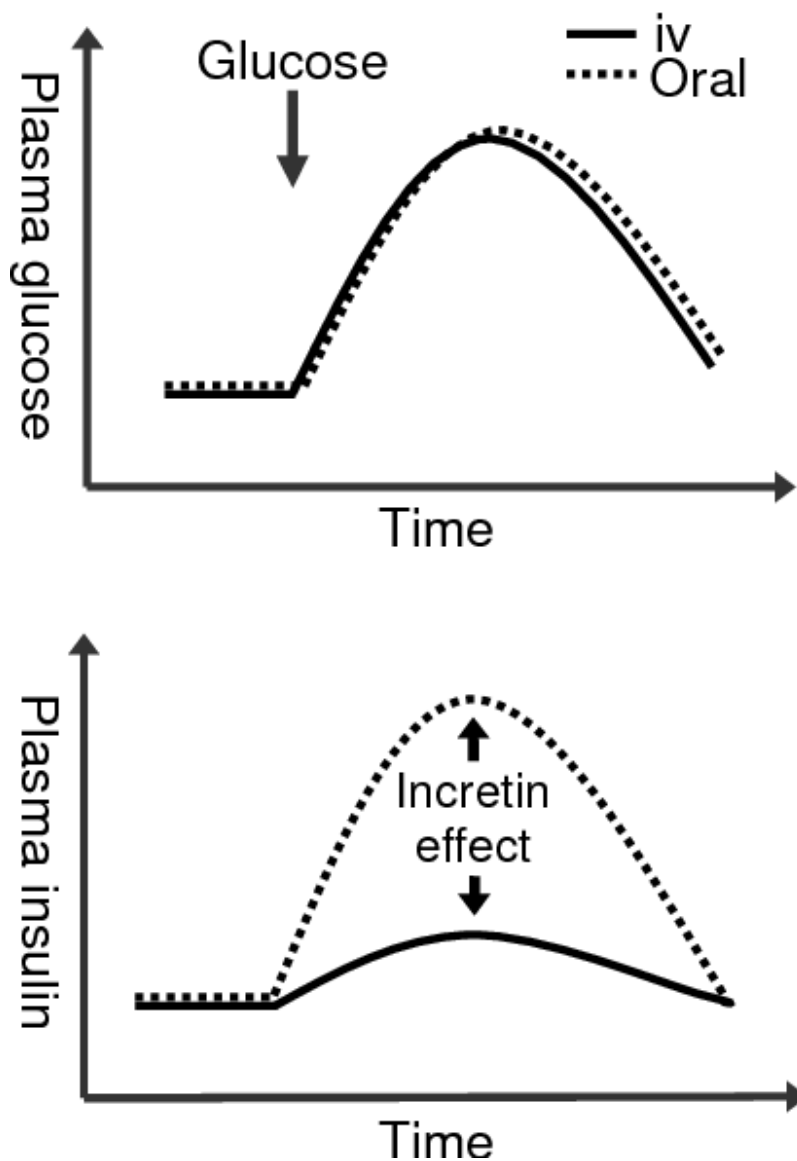
Diabetes Mellitus

6.1 Mechanism of insulin secretion



6.2 Incretin effect

For the same glucose load applied orally and IV, the oral load stimulates more insulin secretion (because oral load → release of gut peptides GLP-1 and GIP → ↑ insulin secretion).



6.3 Diabetic ketoacidosis (DKA)

- Medical emergency
- Cause of death
 - Children: cerebral oedema
 - Adults:
 - * **Hypokalaemia**
 - * ARDS
 - * Comorbidities: acute MI, sepsis, pneumonia
- Cardinal biochemical features
 - Hyperglycaemia → osmotic diuresis → dehydration, dyselectrolytaemia
 - Hyperketonaemia:
 - * Insulin deficiency + elevated catecholamines → unrestrained lipolysis to make FFA → hepatic ketogenesis

- Metabolic acidosis ### Clinical features
- **Symptoms**
 - Polyuria, thirst
 - Weakness
 - Nausea, vomiting
 - Abdominal pain
 - Blurred vision
- **Signs**
 - Dehydration
 - Hypotension
 - Tachycardia
 - Air hunger / Kussmaul breathing (deep and sighing breathing)
 - Acetone breath
 - Delirium, drowsiness, coma

Management

- **Establish IV access**
- **Volume replacement: 0.9% NaCl**
 - If systolic BP \geq 90mmHg: 1L over 1h
 - Else: $\frac{1}{2}$ L over 15mins \rightarrow reassess. If BP still $<$ 90mmHg, repeat.
- **Insulin therapy: IV 0.1 U/kg/h**
 - Corrects hyperglycaemia & acidosis
- **Monitor**
 - Every 1h:
 - * capillary blood glucose and ketone
 - * vitals: pulse, BP, resp rate, O₂ sat, urine output
 - Every 2h: Venous HCO₃⁻ and K⁺
 - Every 4h: Serum electrolytes
- If K⁺ is low, 40mmol/L KCl with normal saline

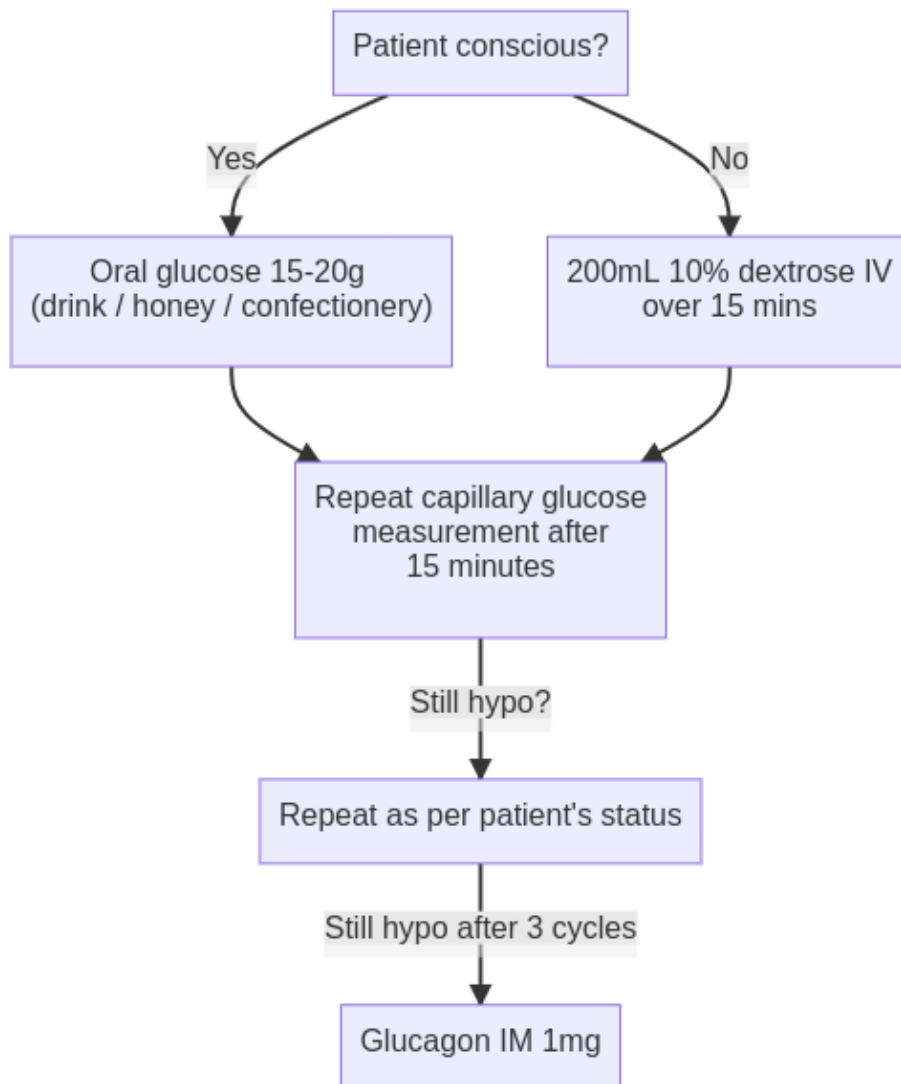
6.4 Hypoglycaemia

Features

- **Autonomic**
 - Sweating
 - Trembling
 - Palpitations
- **Neuroglycopenic**

- Delirium
- Drowsiness
- Speech difficulty
- Incoordination

Management



6.5 Insulin therapy

- Indications
 - Type I DM
 - Type II DM not controlled by OHA
 - DIP / GDM
 - DKA
 - Hyperkalaemia

Preparations

- **Rapid-acting** (rapid=LAG-less)
 - Lispro
 - Aspart
 - Glulisine
- **Short-acting:** soluble/regular insulin
- **Intermediate-acting:** Isophane (I for I)
- **Long-acting**
 - Glargine (gLARGE-in)
 - Detemir Route of administration: **subcutaneous**

6.6 Oral Hypoglycaemic Agents

- **Biguanides:** Metformin
 - Insulin *sensitiser*
 - **Mechanism of action**
 - * ↓ hepatic glucose production (gluconeogenesis and glycogenolysis)
 - * ↑ gut glucose uptake & utilisation
 - * weak inhibitor of mitochondrial respiration → ↑ AMP, ↓ ATP → ↑ glucose uptake utilisation etc.
 - **Side effects profile**
 - * Weight neutral
 - * Non-hypoglycaemic
 - * *Lactic acidosis*
- **Sulphonylureas:** Glibenclamide, Gliclazide, Glimepiride
 - Insulin *secretagogue*
 - **Mechanism of action:** Block K^+ channel in β -cells → ↑ insulin secretion
 - **Side effects profile**
 - * Wt gain
 - * Hypoglycaemia
- **α -glucosidase inhibitors:** Acarbose
 - **Mechanism of action:** delay absorption of carbs
 - **Side effects profile**
 - * Non-hypoglycaemic
 - * Flatulence
 - * Bloating
 - * Diarrhoea
- **Incretin-based therapies:**
 - **DPP-4 inhibitors:** Gliptins

- * **MoA**
 - DPP-4: breaks down GLP-1 & GIP → inhibit incretin effect
- **GLP-1 receptor agonists:** Exenatide, liraglutide
- **Thiazolidinediones:** Pioglitazone
 - **Mechanism of action**
 - * PPAR- γ agonist → enhance action of insulin
 - **Side effects profile**
 - * Non-hypoglycaemic
 - * Wt gain (increase fat cells)
- Oral fast-acting carbohydrate (10-15g) e.g. glucose drink / confectionery / honey to buccal mucosa
- Repeat capillary glucose measurement 10-15mins later
 - If still hypo, repeat upto 3 cycles
 - Still hypo after 3 cycles → glucagon 1mg IM