

Medicine

Susmit

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# Contents

<b>Contents</b>	<b>1</b>	<b>6 Neurology</b>	<b>27</b>
<b>1 Respiratory medicine</b>	<b>2</b>	6.1 Raised ICP . . . . .	27
1.1 Tuberculosis . . . . .	2	6.2 Neurological emergencies . . . . .	28
<b>2 Cardiology</b>	<b>3</b>	6.3 Status epilepticus . . . . .	29
2.1 Presenting problems in CVS disease	3	6.4 All jerks root values . . . . .	29
2.2 ECG . . . . .	3	6.5 Subarachnoid haemorrhage . . . . .	30
2.3 Coronary Artery Disease . . . . .	6	6.6 Subacute combined degeneration	30
2.4 Arrhythmias . . . . .	6	6.7 Cauda equina and Conus	
2.5 Atrial fibrillation . . . . .	7	medullaris lesions . . . . .	31
2.6 Myocardial Infarction . . . . .	9	6.8 Neurogenic Bladder . . . . .	33
<b>3 Dermatology</b>	<b>10</b>	<b>7 Diabetes Mellitus</b>	<b>35</b>
3.1 Anatomy and physiology . . . . .	10	7.1 Mechanism of insulin secretion . .	35
3.2 Principles of management of skin		7.2 Incretin effect . . . . .	35
disease . . . . .	11	7.3 Diabetic ketoacidosis (DKA) . . .	36
3.3 Skin cancers . . . . .	12	7.4 Hypoglycaemia . . . . .	37
3.4 Fungal infections . . . . .	13	7.5 Insulin therapy . . . . .	39
3.5 Scabies . . . . .	13	7.6 Oral Hypoglycaemic Agents . . .	39
3.6 Acne . . . . .	13	7.7 Diabetic retinopathy . . . . .	41
3.7 Eczemas . . . . .	15	<b>8 Gastrointestinal diseases</b>	<b>42</b>
3.8 Psoriasis . . . . .	17	8.1 Weight loss . . . . .	42
3.9 Hypopigmentation . . . . .	20	<b>9 Haematology</b>	<b>43</b>
3.10 Hyperpigmentation . . . . .	20	9.1 Chronic myeloid leukaemia (CML)	43
3.11 Pseudorandom factoids . . . . .	21	<b>10 Nutritional diseases</b>	<b>45</b>
<b>4 Nephrology</b>	<b>22</b>	10.1 Vitamins . . . . .	45
4.1 UTI . . . . .	22		
<b>5 Rheumatology</b>	<b>24</b>		
5.1 Investigations of musculoskeletal			
disease . . . . .	24		
5.2 Seropositive vs Seronegative			
arthritis . . . . .	25		
5.3 Osteoarthritis . . . . .	25		
5.4 Spondyloarthropathies . . . . .	26		

# Chapter 1

## Respiratory medicine

### 1.1 Tuberculosis

#### Side effects of anti-TB drugs

- **Isoniazid:**
  - Hepatitis
  - Rash
  - B<sub>6</sub> deficiency → peripheral neuropathy (so pyridoxine supplement required during therapy)
- **Rifampicin:**
  - Hepatitis
  - Rash
- **Pyrazinamide:**
  - Hepatitis
  - Hyperuricaemia (rarely turns into gout)
- **Ethambutol:**
  - Retrobulbar neuritis (reversible)
  - Arthralgia
- **Streptomycin:**
  - Ototoxicity (8th nerve palsy)
  - Rash

# Chapter 2

## Cardiology

### 2.1 Presenting problems in CVS disease

#### Features of benign murmur

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

### 2.2 ECG

#### Anatomy of an ECG



## Abnormalities of components

### Pathological Q

- Depth  $> 2\text{mm}$
- Height  $> 1\text{mm}$
- Present in  $\geq 2$  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

## 2.3 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

## 2.4 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
- $\beta$ -blocker
- If haemodynamic state compromised, DC cardioversion
- Recurrent SVT  $\rightarrow$  catheter ablation

## 2.5 Atrial fibrillation

### Causes

- **Cardiac**
  - CAD (including acute MI)
  - Mitral stenosis (MS; rheumatic mitral valve disease)
  - Hypertension
  - Cardiomyopathy
- **Non-cardiac**
  - Thyrotoxicosis
  - Pulmonary embolism
  - Pneumonia
  - Alcoholism

### 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**
  - $\beta$ -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

## 2.6 Myocardial Infarction

### Management of acute MI



# Chapter 3

## Dermatology

### 3.1 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

## 3.2 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- $\alpha$  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 N<sub>2</sub>*
  - Causes cell membrane destruction  $\rightarrow$  death
- Laser
- PDT / photodynamic therapy

## 3.3 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

## 3.4 Fungal infections

### Types

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

## 3.5 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

## 3.6 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



## 3.7 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



## 3.8 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

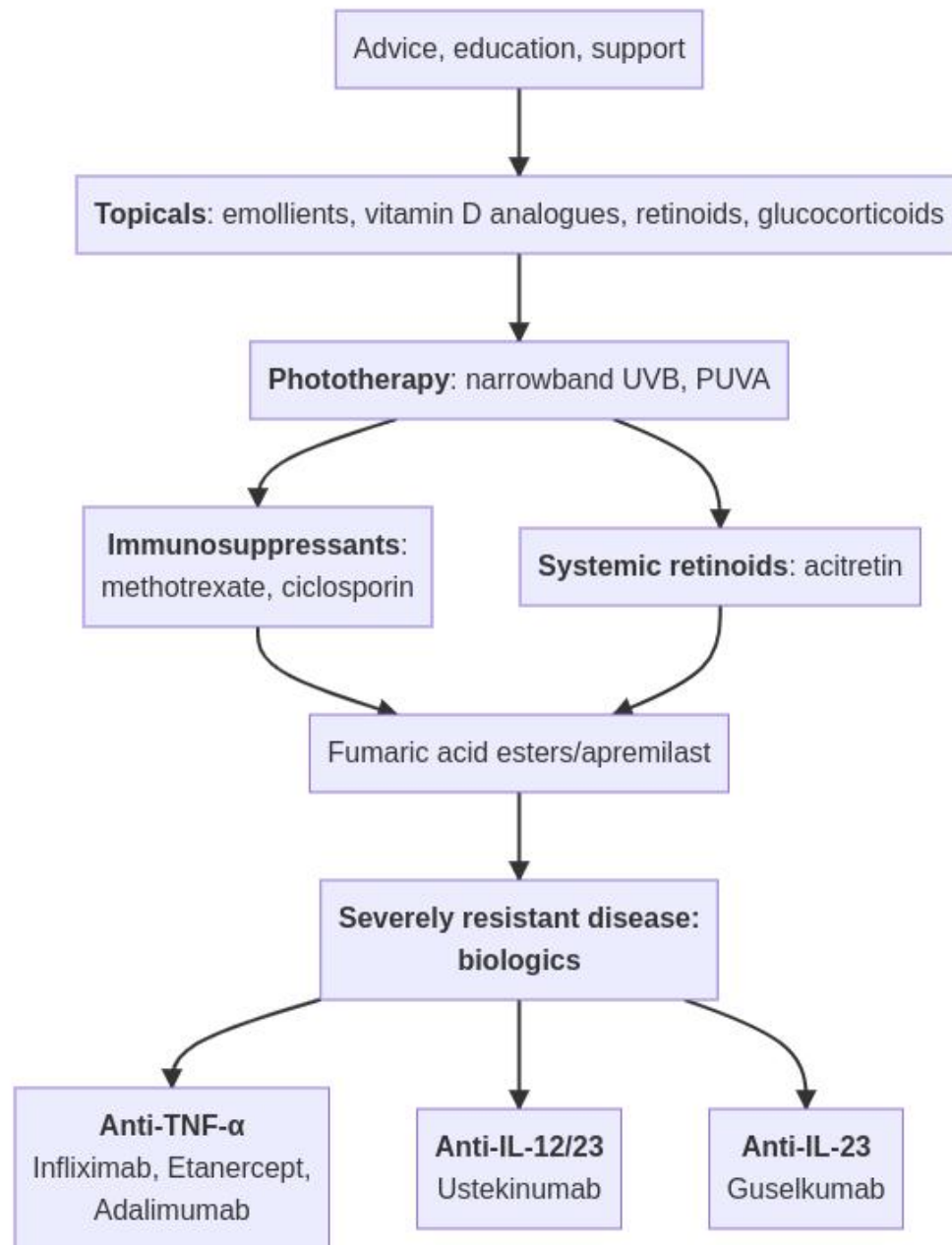
- **Sunlight**
- **Trauma**
- **Infection**
  - $\beta$ -haemolytic strep ↑ guttate psoriasis
  - HIV may initially present with severe psoriasis
- **Drugs**
  - Antimalarials
  - $\beta$ -blockers
  - Lithium
  - NSAIDs
- **Stress and anxiety**

### 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



## Complications

- Psoriatic arthropathy
- Exfoliative dermatitis
- Secondary infection
- Hyperuricaemia and gout

## 3.9 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

## 3.10 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

## 3.11 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 4

## Nephrology

### 4.1 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  $\geq 2\text{L/day}$
- 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 5

## Rheumatology

### 5.1 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
- <sup>99</sup>Tc radiolabelled bisphosphonate used

#### DEXA (Dual Emission X-ray Absorptiometry)

- Measure BMD (bone mineral density)
  - $< -2.5$  → osteoporosis
  - Between  $-2.5$  and  $-1$  → osteopenia
  - $> 2.5$  → 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)

## 5.2 Seropositive vs Seronegative arthritis

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

## 5.3 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

## 5.4 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 6

## Neurology

### 6.1 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
- **Depressed consciousness**
- **Papilloedema**
- **Bradycardia**
- **Hypertension**

## Management

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

## 6.2 Neurological emergencies

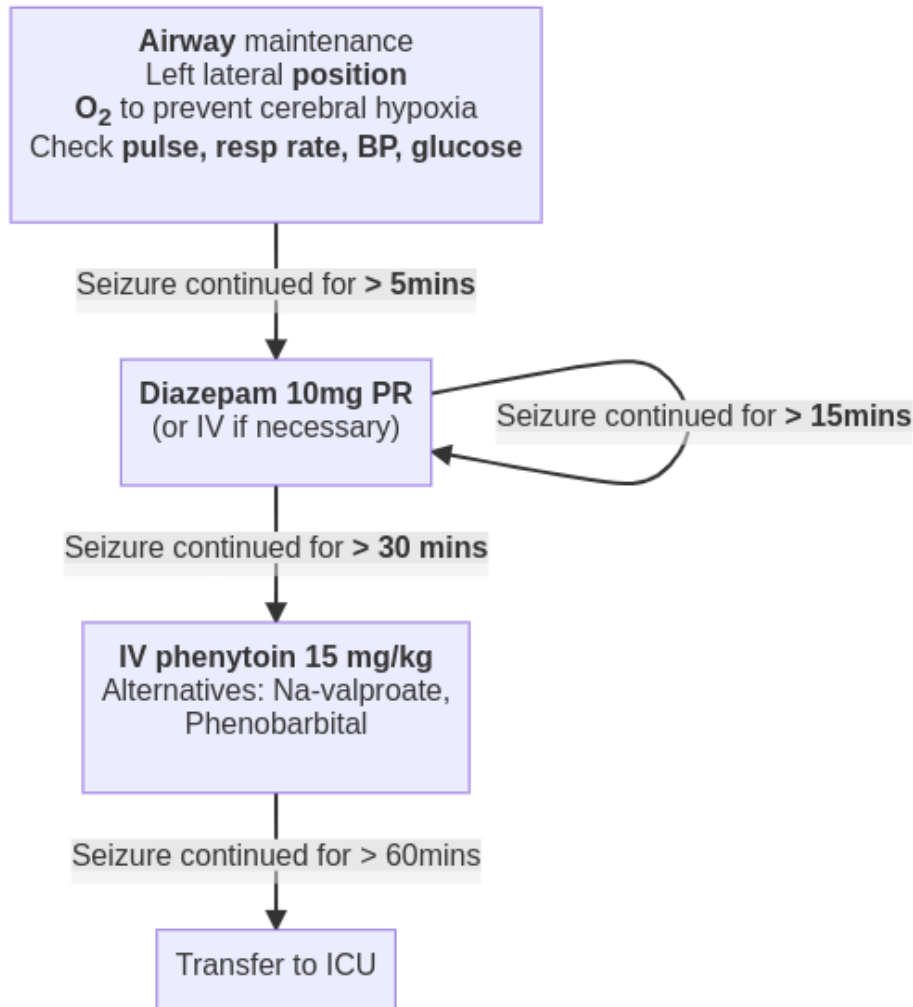
- **Status epilepticus**
- **Stroke** (if thrombo)
- **Subarachnoid haemorrhage**
- **Cord compression**
- **GBS**
- **Myasthenia gravis** (if bulbar and/or respiratory)

## 6.3 Status epilepticus

### Definition

- Continuous or recurrent **seizures** for  $\geq 30$  mins without gain of consciousness in between.
- Clinically we assume SE after 5mins of seizure activity.

### Management



## 6.4 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)

## 6.5 Subarachnoid haemorrhage

### Causes

- Ruptured berry aneurysm (85%)
- Arterio-venous malformations

### Features

- Sudden severe “thunderclap” headache (often occipital)
- Vomiting
- High BP
- Neck stiffness
- May be loss of consciousness
- Photophobia

### Investigations

- CT scan: hyperdense material in the subarachnoid space
- Lumbar puncture: blood, xanthochromia

### Management

- **Nimodipine** 30-60mg IV for 5-14d, followed by 360mg oral for 7d
  - *prevents delayed ischaemia*
- Insertion of **Pt coils** into aneurysm
- **Surgical clipping** of the neck of the aneurysm
- Surgical removal if AVM

## 6.6 Subacute combined degeneration

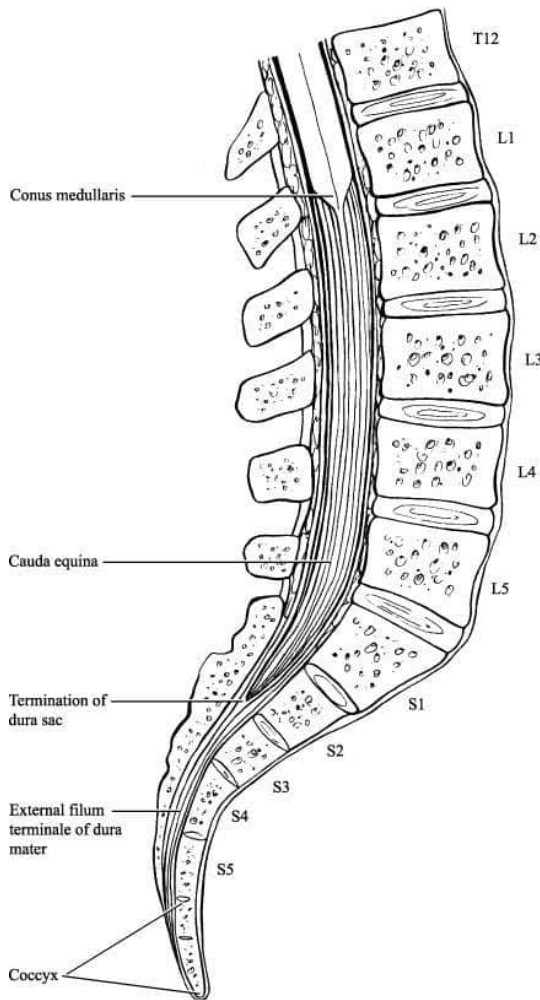
### Features

- **Peripheral neuropathy**: due to **demyelination**
- Signs of **dorsal column lesion**: **position and vibration sense lost**
- Signs of **pyramidal lesion**:
  - **plantar extensor**
  - **knee jerk brisk**
  - **ankle jerk absent**: as peripheral neuropathy affects longer nerves first, the afferent pathway for ankle jerk is damaged by the PN while the same for knee jerk is not. Combined with the corticospinal tract lesion, this makes knee jerks brisk and ankle jerks absent.
- **Optic atrophy**: death of retinal ganglion cell axons

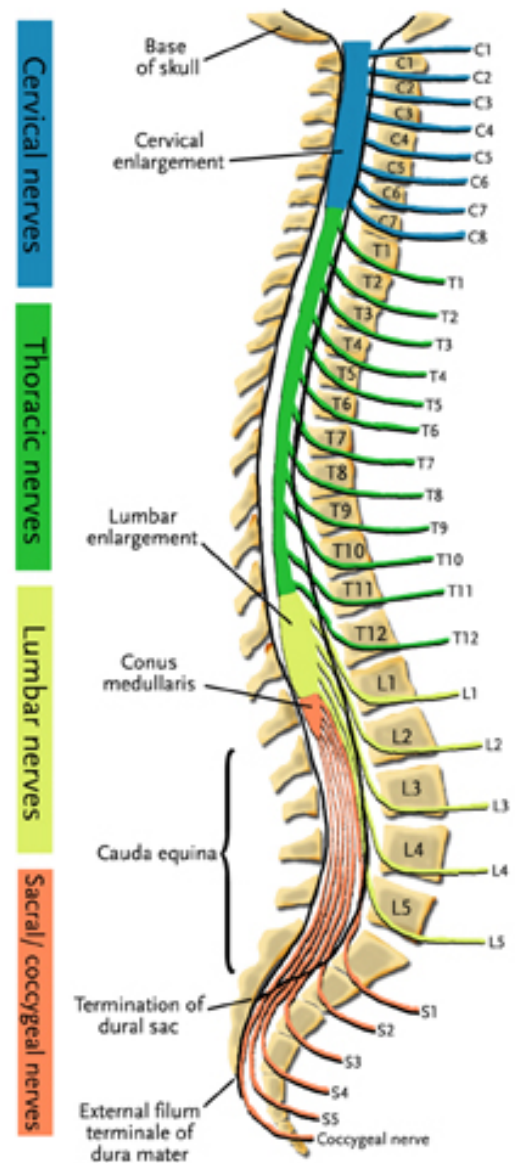


## 6.7 Cauda equina and Conus medullaris lesions

### Anatomy



(a) The Conus and the Cauda



(b) Vertebrae with corresponding spinal cord segments

- During development, the spine grows faster than the spinal cord, which is why the spinal nerves exit the spinal column at increasingly oblique angles.
- The spinal cord ends as the tapered conus medullaris at around L1.

## Relevant physiology

- S2-4:
  - Parasympathetic fibres for bladder sphincter (activation causes emptying)
  - Somatic fibres for pudendal nerves (activation causes relaxation of urethral and anal sphincters → emptying).
- Conus is surrounded by spinal nerve roots bundled up together around it, so injury / compression in this region leads to a combination of UMN and LMN lesion features.
- Lesion in the cauda region leads only to LMN lesion features as there's no cord there.

## Features of Cauda Equina syndrome vs Conus Medullaris syndrome

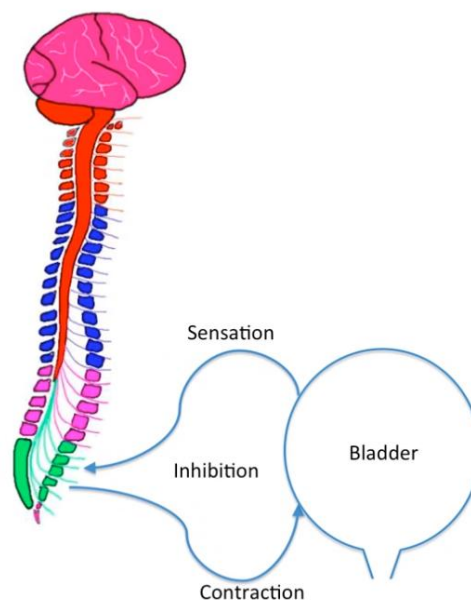
Features	Cauda Equina Syndrome	Conus Medullaris
Vertebral level	L2-sacrum	L1-L2
Spinal level	Injury to the lumbosacral nerve roots	Injury of the sacral cord segment (conus and epiconus) and roots
Severity of symptoms and signs	Usually severe	Usually not severe
Symmetry of symptoms and signs	Usually asymmetric	Usually symmetric
Pain	Prominent, asymmetric, and radicular	Usually bilateral and in the perineal area
Motor	Weakness to flaccid paralysis	Normal motor function to mild or moderate weakness
Sensory	Saddle anesthesia, may be asymmetric	Symmetric saddle distribution, sensory loss of pin prick, and temperature sensations (Tactile sensation is spared.)
Reflexes	Areflexic lower extremities; bulbocavernosus reflex is absent in low CE (sacral) lesions	Areflexic lower extremities  (If the epiconus is involved, patellar reflex may be absent, whereas bulbocavernosus reflex may be spared.)

Reflexes	Areflexic lower extremities; bulbocavernosus reflex is absent in low CE (sacral) lesions	Areflexic lower extremities  (If the epiconus is involved, patellar reflex may be absent, whereas bulbocavernosus reflex may be spared.)
Sphincter and sexual function	Usually late and of lesser magnitude;  lower sacral roots involvement can cause bladder, bowel, and sexual dysfunction	Early and severe bowel, bladder, and sexual dysfunction that results in a reflexic bowel and bladder with impaired erection in males
EMG	Multiple root level involvement; sphincters may also be involved	Mostly normal lower extremity with external anal sphincter involvement
Outcome	May be favorable compared with conus medullaris syndrome	The outcome may be less favorable than in patients with CES

## 6.8 Neurogenic Bladder

### Physiological control of micturition: the Micturition Reflex

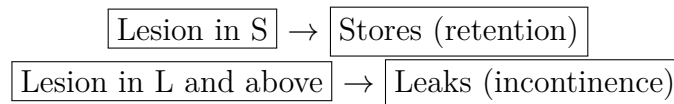
#### Bladder Reflex Cycle of Urination



## “Components”

- **Detrusor muscle**
  - Muscles of the bladder wall
  - Innervated by
    - \* **Parasympathetic:**
      - cholinergic M<sub>3</sub> receptors
      - pelvic nerve from **S2-4**
      - causes **contraction** → **urination**
    - \* **Sympathetic:**
      - $\beta_3$  receptors
      - hypogastric nerve from **T10-L2**
      - causes **relaxation** → **storage** / retention
- **Internal urethral sphincter**
  - Innervated by **only sympathetic**
    - \* hypogastric nerve from **T10-L2**
    - \* causes storage / retention
- **External urethral sphincter**
  - **Voluntary control**
    - \* UMN from pontine micturition centre
    - \* LMN from S2-4 (pudendal nerve)
    - \* causes retention

## The big picture (highest yield for clinical interpretation)

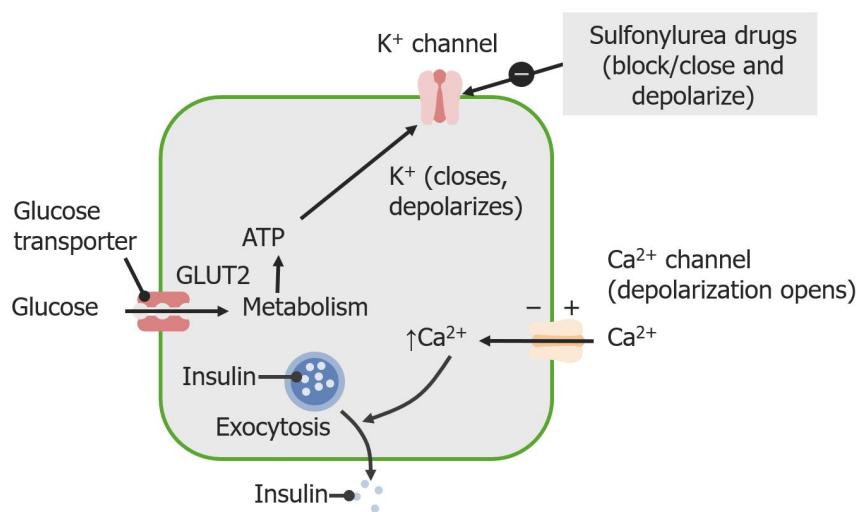


- The reflex spontaneously tries to void the bladder upon being filled even by small amounts. The only thing holding it back is the pontine micturition centre which always inhibits the circuit, unless we voluntarily signal it not to. We can't signal the bladder to empty itself. We can only signal the micturition reflex to not hold back.
- The reflex circuit is composed of afferents and efferents from S2-4. So any **lesion above S2-4** will leave the circuit intact, while damaging the telephone lines from the pontine centre. The end result is that we lose the inhibition of the reflex (which fires every few mins after storing some tens of millilitres of urine), and without anything holding it back, there's urinary **incontinence**.
- On the other hand, any **lesion at S2-4** will damage the circuit elements themselves. That means that the reflex will be lost. This means we no longer have access to the bladder emptying circuit. So there will be urinary **retention**.

# Chapter 7

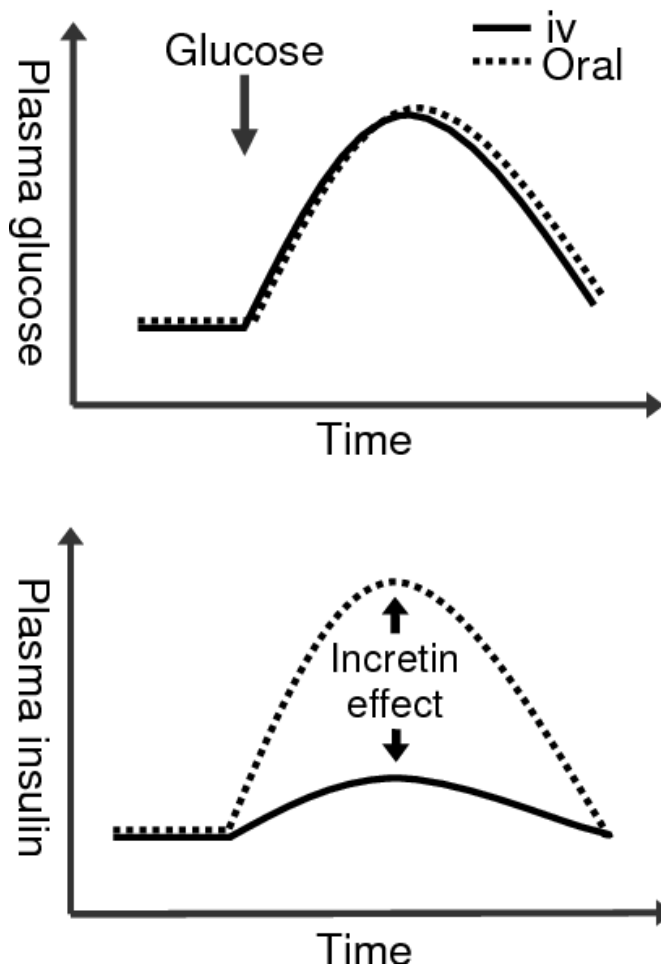
## Diabetes Mellitus

### 7.1 Mechanism of insulin secretion



### 7.2 Incretin effect

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



### 7.3 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

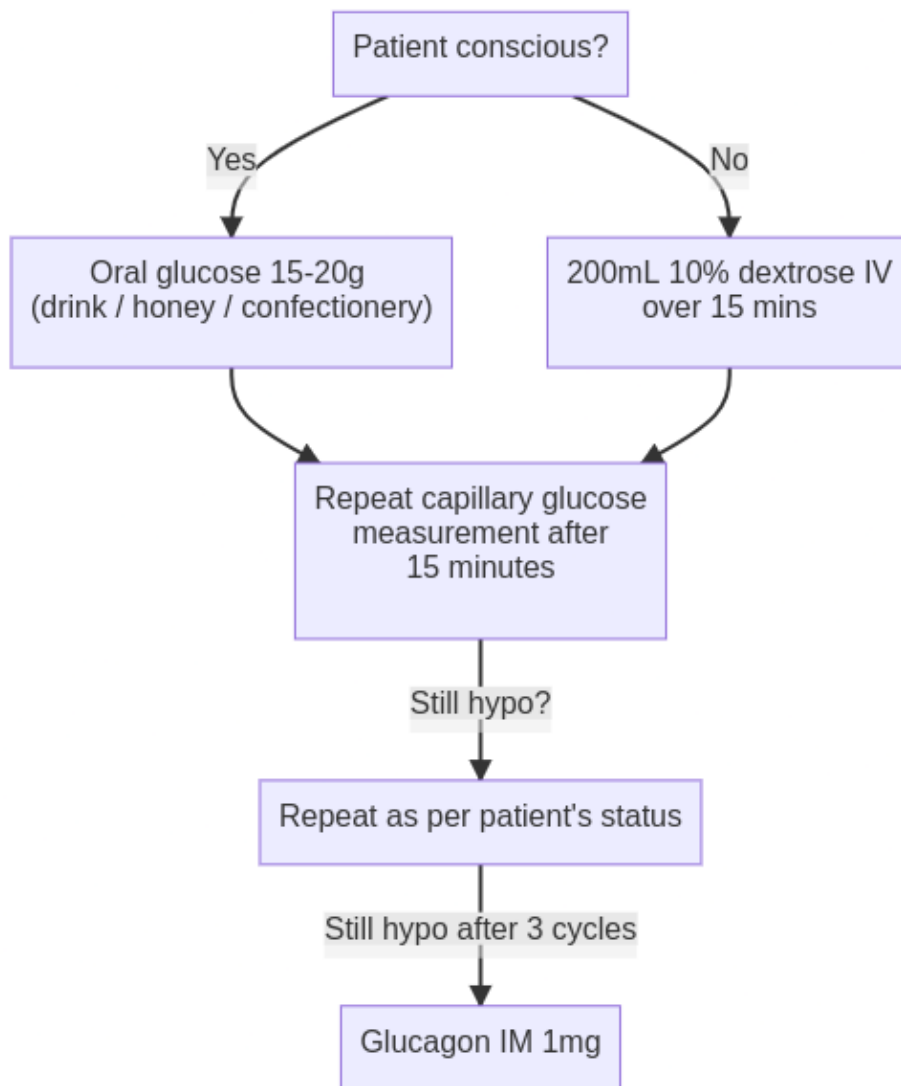
- **Establish IV access**
- **Volume replacement: 0.9% NaCl**
  - If systolic BP  $\geq$  90mmHg: 1L over 1h
  - Else:  $\frac{1}{2}$ L over 15mins  $\rightarrow$  reassess. If BP still  $<$  90mmHg, repeat.
- **Insulin therapy: IV 0.1 U/kg/h**
  - Corrects hyperglycaemia & acidosis
- **Monitor**
  - Every 1h:
    - \* capillary blood glucose and ketone
    - \* vitals: pulse, BP, resp rate, O<sub>2</sub> sat, urine output
  - Every 2h: Venous HCO<sub>3</sub><sup>-</sup> and K<sup>+</sup>
  - Every 4h: Serum electrolytes
- If K<sup>+</sup> is low, 40mmol/L KCl with normal saline

## 7.4 Hypoglycaemia

### Features

- **Autonomic**
  - Sweating
  - Trembling
  - Palpitations
- **Neuroglycopenic**
  - Delirium
  - Drowsiness
  - Speech difficulty
  - Incoordination

## Management



- 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



## 7.5 Insulin therapy

### Indications

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

### Preparations

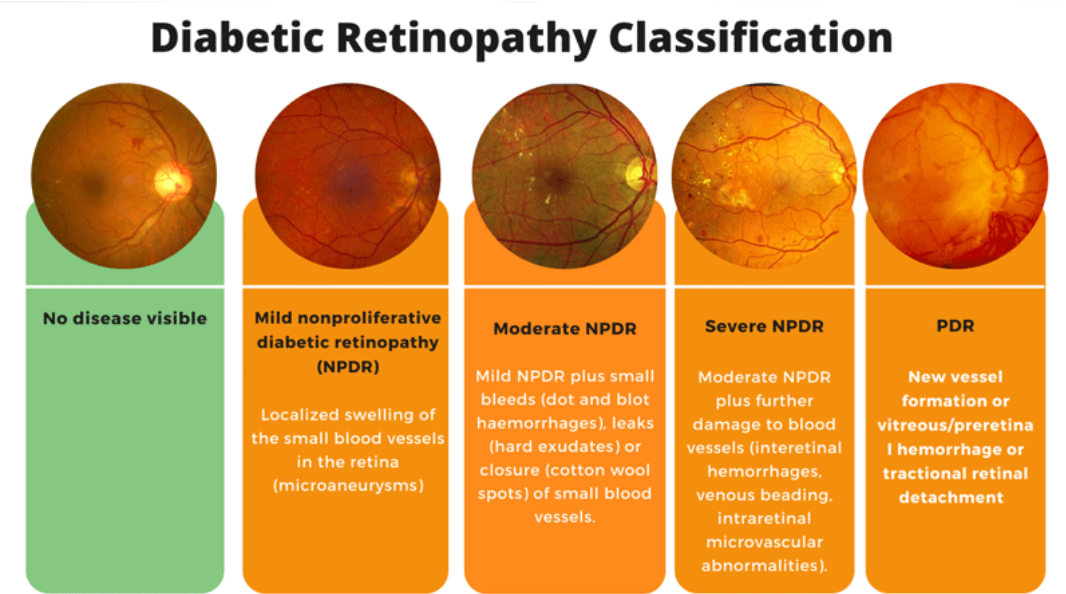
- **Rapid-acting** (rapid=LAG-less)
  - Lispro
  - Aspart
  - Glulisine
- **Short-acting:** soluble/regular insulin
- **Intermediate-acting:** Isophane (I for I)
- **Long-acting**
  - Glargine (gLARGE-in)
  - Detemir Route of administration: **subcutaneous**

## 7.6 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  $\beta$ -cells → ↑ insulin secretion
  - **Side effects profile**
    - \* Wt gain
    - \* Hypoglycaemia
- **$\alpha$ -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- $\gamma$  agonist → enhance action of insulin
  - **Side effects profile**
    - \* Non-hypoglycaemic
    - \* Wt gain (increase fat cells)
- **SGLT-2 inhibitors:** empagliflozin, dapagliflozin
  - **MoA:** inhibit reabsorption of glucose in renal tubules → 25% of filtered glucose excreted
  - Resulting glycosuria can lead to genital fungal infections
  - Empagliflozin → 35% reduced mortality in heart failure

# 7.7 Diabetic retinopathy



# Chapter 8

## Gastrointestinal diseases

### 8.1 Weight loss

#### Causes

- **Endocrine**
  - DM (more in type I)
  - Thyrotoxicosis
  - Addison's
- **GI**
  - Any cause of dysphagia e.g.
    - \* Stroke
    - \* MS
    - \* Ca oesophagus
    - \* Achalasia cardia
    - \* Plummer-Vinson syndrome (oesophageal webs+IDA)
  - Malabsorption syndrome
    - \* IBD
    - \* Chronic pancreatitis (due to enzyme insufficiency)
    - \* Coeliac disease
- **Malignancies**
- **Chronic infection**
  - TB
  - AIDS
- **Psychological**
  - Depression
  - Anorexia nervosa
  - Bulimia nervosa
  - Alcoholism

# Chapter 9

## Haematology

### 9.1 Chronic myeloid leukaemia (CML)

Defining characteristic: **Philadelphia chromosome**

- Shortened **chr22** by *reciprocal translocation* with **chr9**
- Results in **BCR-ABL fusion gene**
- BCR-ABL codes for a **tyrosine kinase** which influences cell proliferation and survival

#### Features

- Wt loss
- Lethargy
- Abdominal discomfort
- Splenomegaly
- Hepatomegaly

#### Phases

- Chronic
- Accelerated
- Blastic crisis

#### Investigations

- **CBC:** anaemia, leucocytosis
- **PBF:**
  - Full range of granulocytic precursors, from *myeloblasts* to *mature neutrophils*.
  - Predominant: neutrophils and myelocytes.
  - Myeloblasts < 10%.
- **Bone marrow examination:**
  - Hypercellular marrow
  - ↑ M/E ratio

- ↓ erythropoiesis
- ↑ leucopoiesis
- **Chromosome analysis** to detect Ph chromosome

## Management

### Chronic phase

- **1st line: Tyrosine kinase inhibitors (TKIs):**
  - **Imatinib**
  - **Dasatinib**
  - **Nilotinib**
  - normalise blood count within a month, complete cytogenetic response (disappearance of Ph chr) within 6 months in 90% patients. Resample bone marrow at 6mo to confirm. Thereafter monitor 3-monthly by RT-PCR for BCR-ABL mRNA transcripts.
- **Allogeneic HSC transplant:** if TKI fails
- **Hydroxycarbamide**
- **Interferon:** in pregnancy

# Chapter 10

## Nutritional diseases

### 10.1 Vitamins

#### B<sub>1</sub> (thiamin) deficiency

##### Functions of thiamin

- Cofactor in different pathways of aerobic metabolism of glucose
  - decarboxylation of pyruvate to acetyl-coA (so bridge between glycolysis and Krebs)
  - decarboxylation of  $\alpha$ -ketoglutarate to succinate in Krebs

##### Features

- For its pivotal role in aerobic glucose meta, the largest blow is dealt to the tissues most dependent on glucose - the **brain** and the **heart**.
- **Beri-beri**
  - **Dry:** Neurological beri-beri
    - \* Peripheral neuropathy
    - \* Wrist/foot drop
    - \* **Korsakoff's psychosis**
    - \* **Wernicke's encephalopathy**
  - **Wet:** Cardiac beri-beri (wet as it causes generalised oedema)
    - \* Biventricular failure
    - \* Generalised oedema
    - \* Pulmonary oedema

##### Treatment

- Wet and Wernicke's should be treated by IV vit-B and C mixture
- Korsakoff's: irreversible, nonresponsive to thiamin

## **B<sub>12</sub> deficiency**

### **Functions of B<sub>12</sub>**

- Recycles folate → essential for **cell division** (especially in RBC)
- **Myelination**

### **Features**

- **Megaloblastic anaemia**
- **Glossitis**
- **Neurologic features**
  - Peripheral neuropathy
  - Autonomic neuropathy
  - Optic atrophy
  - Subacute combined degeneration of spinal cord

### **Treatment**

- Vit-B<sub>12</sub> IM
- If combined folate and B<sub>12</sub> deficiency, only folate should not be given without B<sub>12</sub>, as B<sub>12</sub> gets used up in folate recycling, deteriorating the B<sub>12</sub> deficiency symptoms. Always give both together.