

Medicine

Susmit

2022-07-06

Contents

Contents	1	5.4 Spondyloarthropathies	30
1 Respiratory medicine	2	6 Neurology	31
1.1 Tuberculosis	2	6.1 Raised ICP	31
2 Cardiology	3	6.2 Neurological emergencies	32
2.1 Presenting problems in CVS disease	3	6.3 Status epilepticus	33
2.2 ECG	3	6.4 All jerks root values	33
2.3 Coronary Artery Disease	6	6.5 Subarachnoid haemorrhage	34
2.4 Arrhythmias	6	6.6 Subacute combined degeneration	34
2.5 Atrial fibrillation	7	6.7 Cauda equina and Conus	
2.6 Myocardial Infarction	9	medullaris lesions	35
3 Dermatology	10	6.8 Neurogenic Bladder	37
3.1 Anatomy and physiology	10	7 Diabetes Mellitus	40
3.2 Principles of management of skin		7.1 Mechanism of insulin secretion . .	40
disease	11	7.2 Incretin effect	40
3.3 Skin cancers	12	7.3 Diabetic ketoacidosis (DKA) . . .	41
3.4 Fungal infections	13	7.4 Hypoglycaemia	42
3.5 Scabies	13	7.5 Insulin therapy	44
3.6 Acne	13	7.6 Oral Hypoglycaemic Agents . . .	44
3.7 Eczemas	15	7.7 Diabetic retinopathy	46
3.8 Psoriasis	17	8 Gastrointestinal diseases	47
3.9 Hypopigmentation	20	8.1 Weight loss	47
3.10 Hyperpigmentation	20	9 Haematology	48
3.11 Pseudorandom factoids	21	9.1 Chronic myeloid leukaemia (CML)	48
4 Nephrology	22	10 Nutritional diseases	50
4.1 Nephrotic syndrome	22	10.1 Vitamins	50
4.2 Acute glomerulonephritis	24		
4.3 UTI	26		
5 Rheumatology	28		
5.1 Investigations of musculoskeletal			
disease	28		
5.2 Seropositive vs Seronegative			
arthritis	29		
5.3 Osteoarthritis	29		

Chapter 1

Respiratory medicine

1.1 Tuberculosis

Side effects of anti-TB drugs

- **Isoniazid:**
 - Hepatitis
 - Rash
 - B₆ 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 ≥ 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
- β -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**
 - 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

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- 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

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 scabies 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

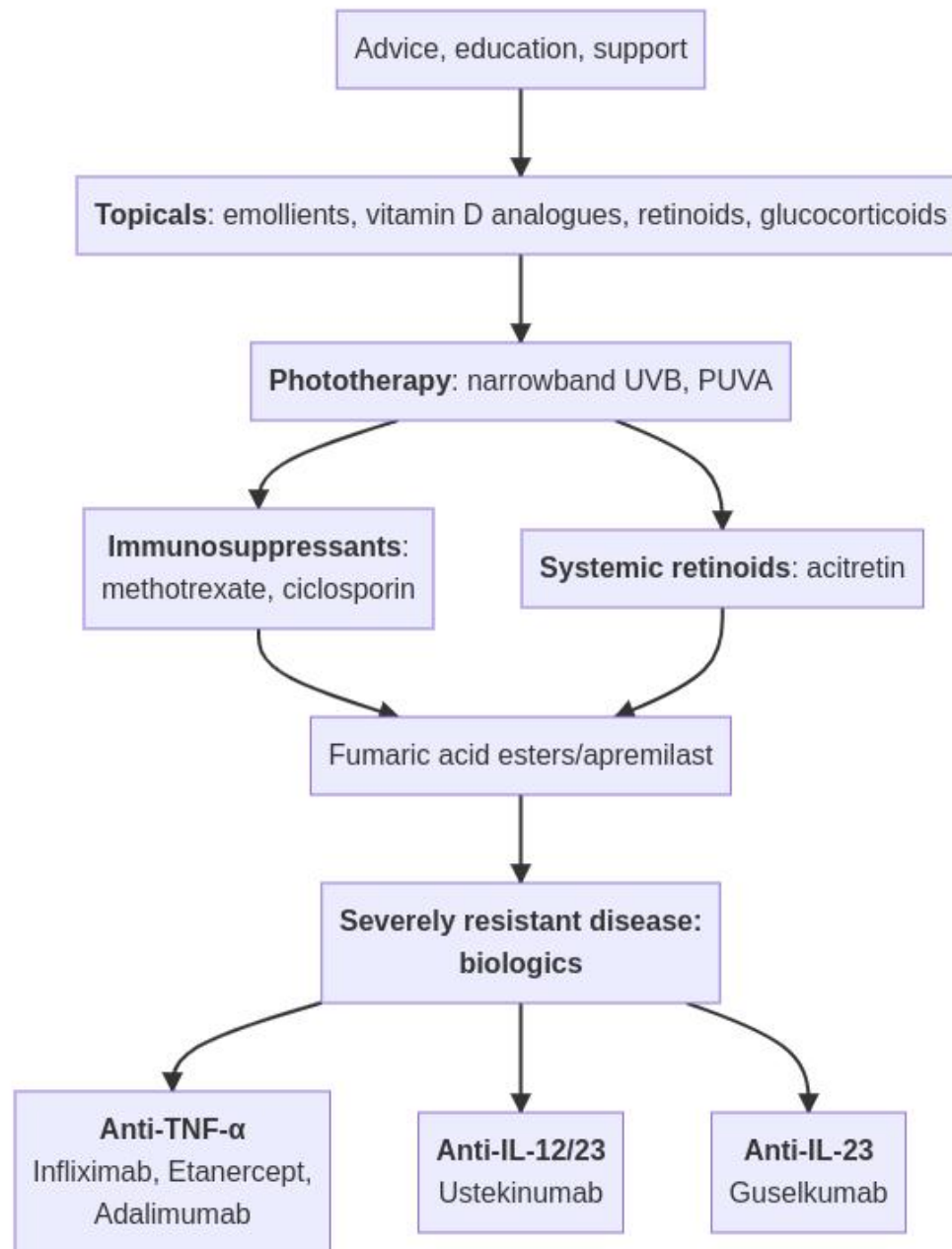
- **Sunlight**
- **Trauma**
- **Infection**
 - β -haemolytic strep ↑ guttate psoriasis
 - HIV may initially present with severe psoriasis
- **Drugs**
 - Antimalarials
 - β -blockers
 - Lithium
 - NSAIDs
- **Stress and anxiety**

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



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 Nephrotic syndrome

Features

- Massive proteinuria (> 3.5 g/day (medicine) or $1 \text{ g/m}^2/\text{day}$ (paediatrics))
 - Generalised oedema (pitting)
 - Ascites, pleural **effusion**, pericardial effusion
- Features of reduced circulatory volume
 - Scanty urination (colour normal)
 - Pulse: weak
 - BP: low
 - Capillary refill: prolonged ($> 3\text{s}$)
- Hyperlipidaemia & lipiduria
 - due to \uparrow lipoprotein production by liver
- **Recurrent infections**
 - loss of immunoglobulins with urine

Histopathology

- Histological types include
 - **Minimal change disease:**
 - * no pathology visible with light microscope, visible pathology only under electron microscope.
 - * most common type in **children** (2-8y)
 - **Membranous nephropathy:** most common type in **adults**

Pathogenesis

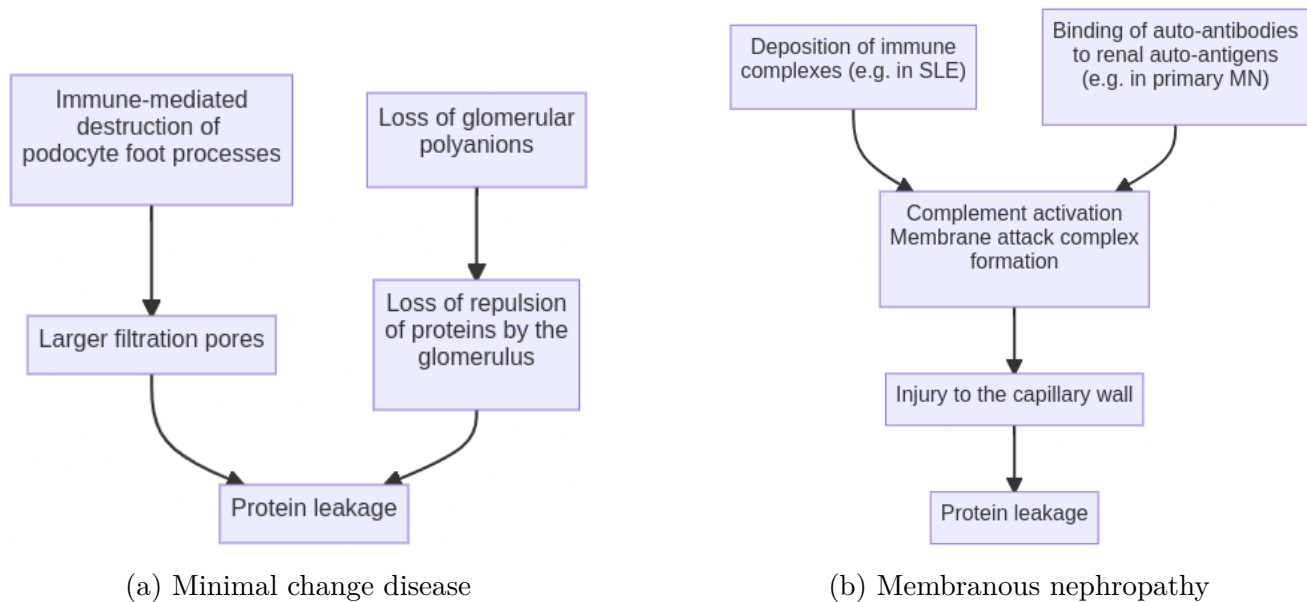


Figure 4.1: Pathogenesis of nephrotic syndrome

Minimal change disease

- Effacement of podocyte foot processes

Membranous nephropathy

- MAC-mediated glomerular capillary injury
- Aetiology:
 - Primary: HLA-DQA1
 - Secondary:
 - * Drugs e.g. NSAIDs, penicillamine (treatment for Wilson's)
 - * Hep-B
 - * Malignancy
 - * SLE

Investigations

- Urine RME
 - Albuminuria
 - Granular & hyaline casts
 - Pus cells if associated with UTI
- Diagnostic
 - 24h total protein: $> 1\text{g}/\text{m}^2/\text{day}$

- PCR (Protein-Creatinine ratio) > 2
- Serum albumin < 25 g/L
- Serum cholesterol > 220 mg/dL
- Renal biopsy: if nonresponsive to corticosteroid therapy

Treatment

- **Specific:**
 - Prednisolone 1 mg/kg/day for 6wks
 - After 6wks start tapering (alternate-day therapy)
- **Supportive:**
 - Diet:
 - * normal with adequate protein
 - * salt restriction
 - Oedema: if severe,
 - * restrict salt and fluid intake
 - * diuretics
 - Hypovolaemia: infuse albumin
 - Infections: treat & prevent

4.2 Acute glomerulonephritis

Pathogenesis

- Occurs following sore throat or skin infection by **group-A β -haemolytic streptococcus** (nephritogenic strains: 12, 49)
- **Antibodies against streptococcal M protein cross-react** with glomerular antigens because of molecular mimicry
- Complement activation, inflammatory cell infiltration. Net effects:
 - Oliguria
 - Haematuria
 - Hypervolaemia, hypertension, hyperkalaemia
 - Azotaemia
 - Acidosis
- **Complications** (due mostly to acute hypertension)
 - Acute LVF
 - Hypertensive encephalopathy
 - Acute kidney injury

Features

- Age: 5-12y; history of strep throat/skin infection a few wks prior

- Scanty, smoky urine
- Puffy face
- Hypertension
- Features of complications
 - **ALVF**:
 - * cough
 - * respiratory distress
 - * orthopnoea
 - * gallop rhythm
 - * bilateral basal crepitations
 - **Hypertensive encephalopathy**:
 - * headache, blurred vision, convulsion, delirium, papilloedema
 - **AKI**: anuria

Investigations

- **Evidence of nephritis**
 - Urine RME:
 - * RBC, RBC casts
 - * Mild proteinuria
 - * Leucocytes
 - Serum C3: ↓
 - Serum C4: normal
- **Evidence of prior strep infection**
 - ASO titre: ↑
 - Anti-DNAse B: ↑
- **Evidence of complications**
 - Serum electrolytes: may show hyperkalaemia and acidosis
 - Serum creatinine
 - X-ray chest

Treatment

- **Rest**
- **Diet**: restrict fluid, salt, protein.
- **Diuretics**: furosemide
- **Antibiotics**: phenoxymethyl penicillin PO to prevent spread of remaining strep within the body
- **Antihypertensive**

4.3 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 \rightarrow Neutrophils in urine \rightarrow 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
- ⁹⁹Tc 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

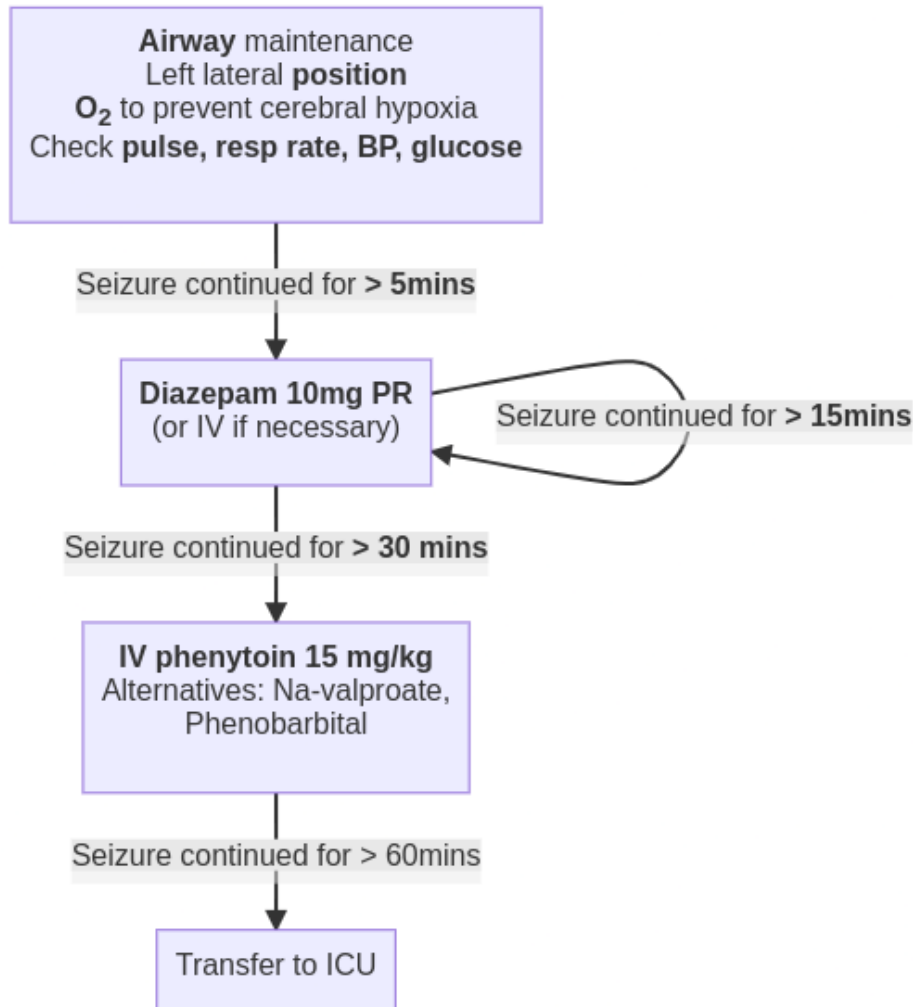
- **Status epilepticus**
- **Stroke** (if thrombo)
- **Subarachnoid haemorrhage**
- **Cord compression**
- **GBS**
- **Myasthenia gravis** (if bulbar and/or respiratory)

6.3 Status epilepticus

Definition

- Continuous or recurrent **seizures** for ≥ 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

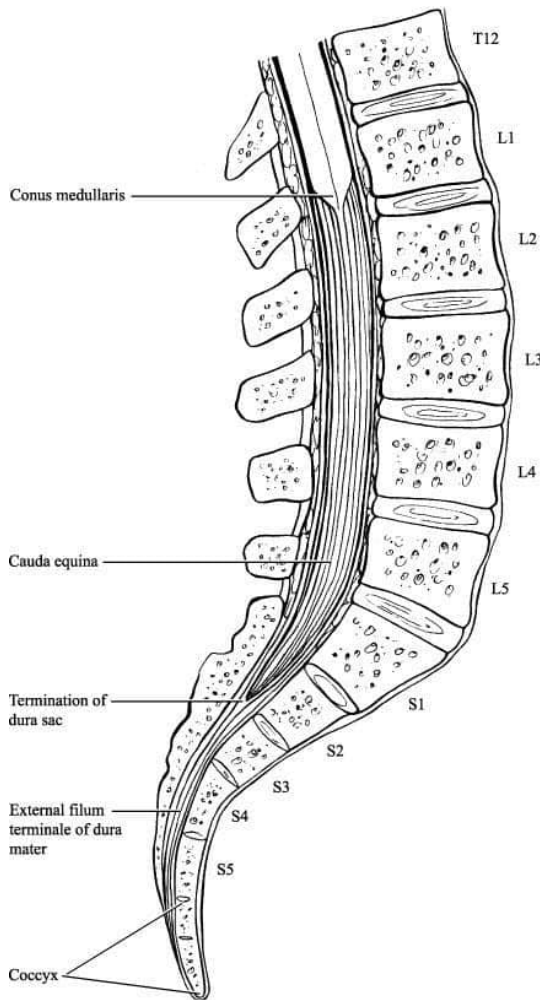
6.6 Subacute combined degeneration

Features

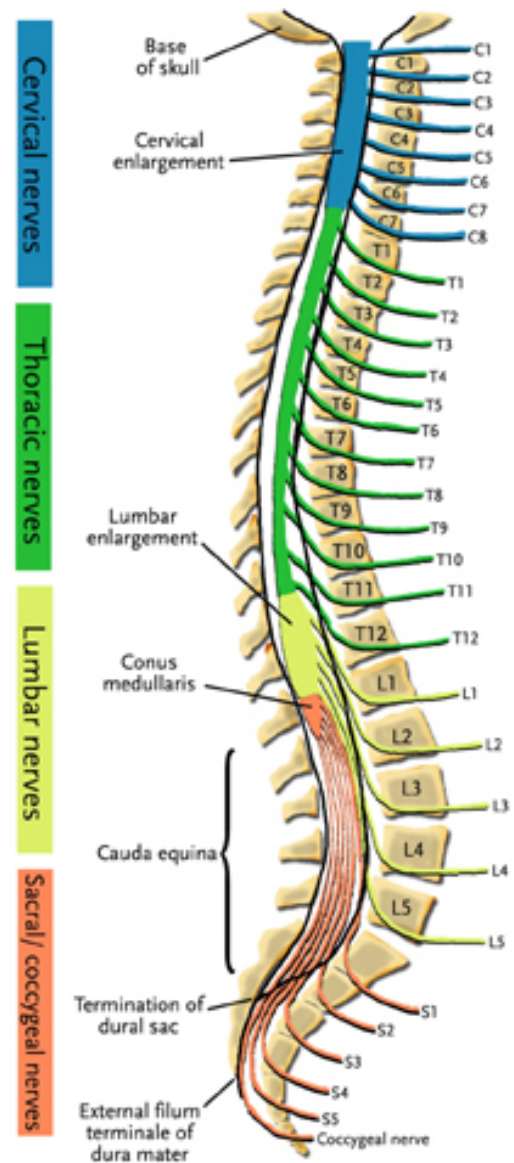
- **Peripheral neuropathy**: due to **demyelination**
- Signs of **dorsal column lesion**: **position and vibration sense lost**
- Signs of **pyramidal lesion**:
 - **plantar extensor**
 - **knee jerk brisk**
 - **ankle jerk absent**: as peripheral neuropathy affects longer nerves first, the afferent 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

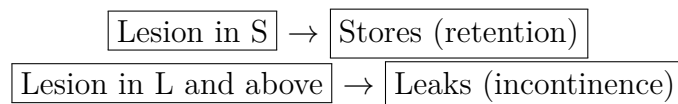
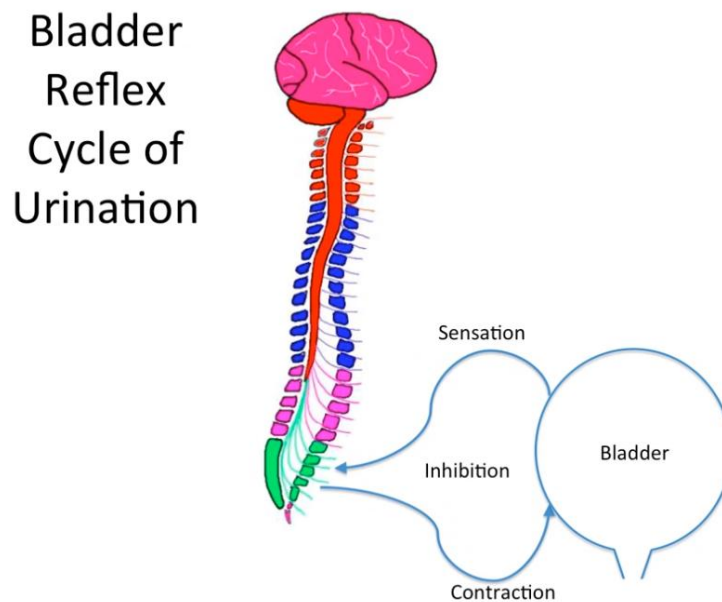
Physiological control of micturition: the Micturition Reflex

“Components”

- **Detrusor muscle**
 - Muscles of the bladder wall
 - Innervated by
 - * **Parasympathetic:**
 - cholinergic M₃ receptors
 - pelvic nerve from **S2-4**
 - causes **contraction** → **urination**
 - * **Sympathetic:**
 - β_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**.

The details

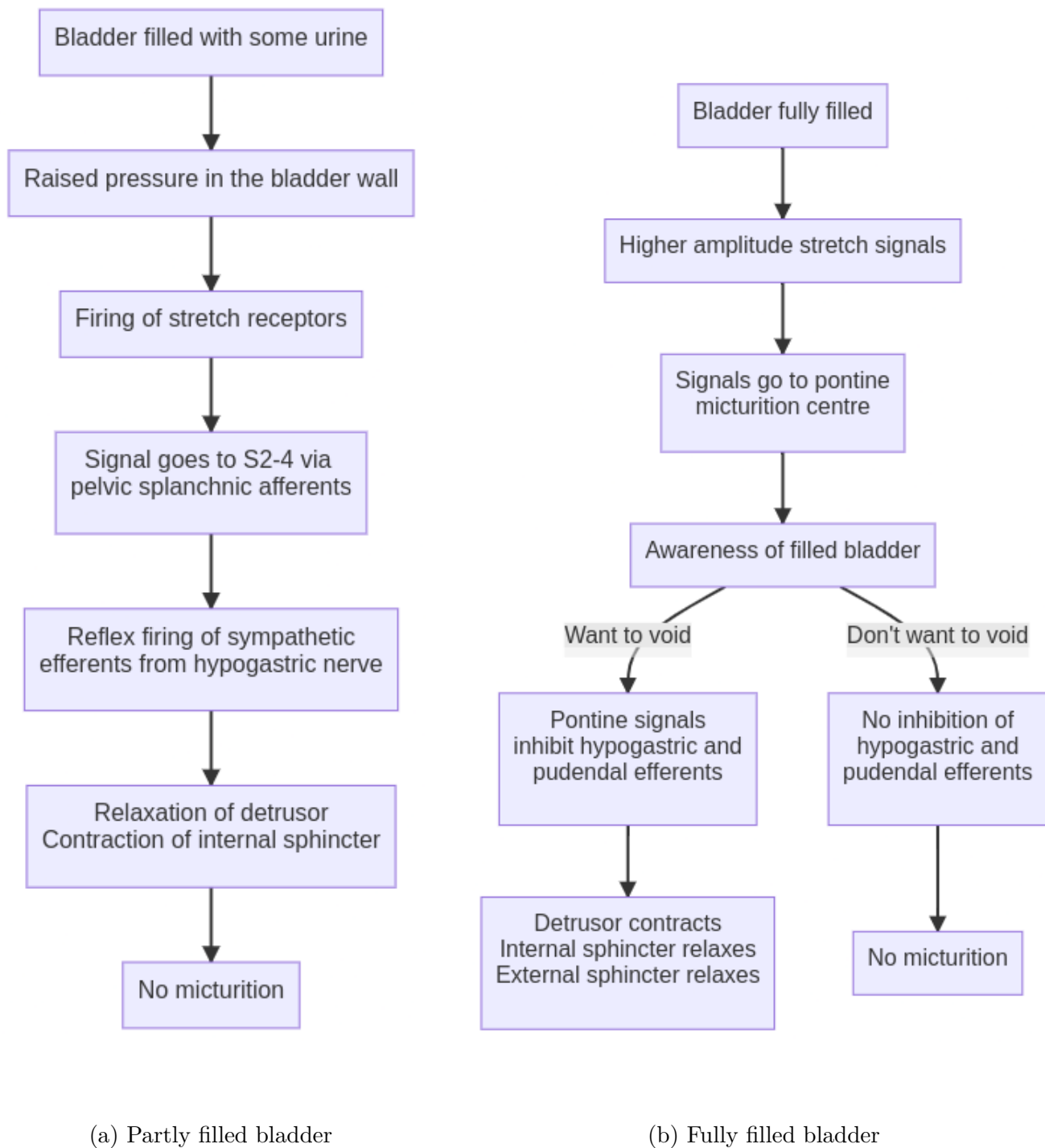


Figure 6.2: Neural control of micturition

Chapter 7

Diabetes Mellitus

7.1 Mechanism of insulin secretion

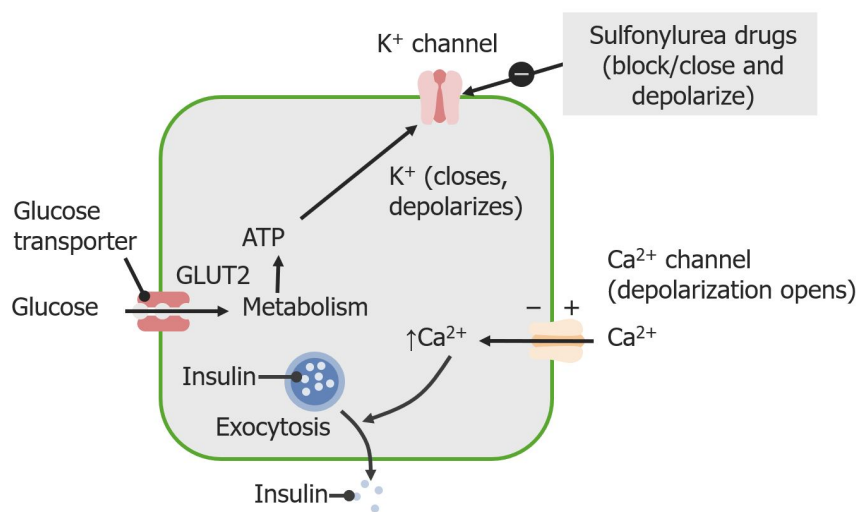


Figure 7.1: Mechanism of insulin secretion

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).

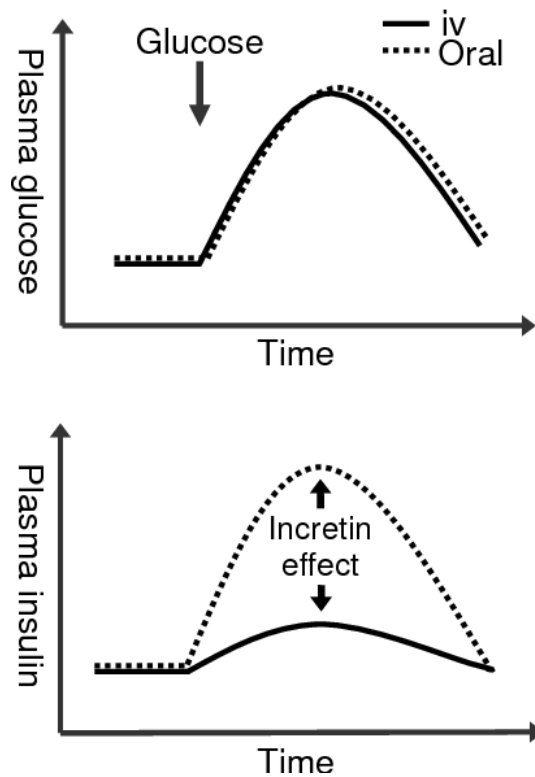


Figure 7.2: The incretin effect

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, O₂ sat, urine output
 - Every 2h: Venous HCO₃⁻ and K⁺
 - Every 4h: Serum electrolytes
- If K⁺ is low, 40mmol/L KCl with normal saline

7.4 Hypoglycaemia

Features

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

Management

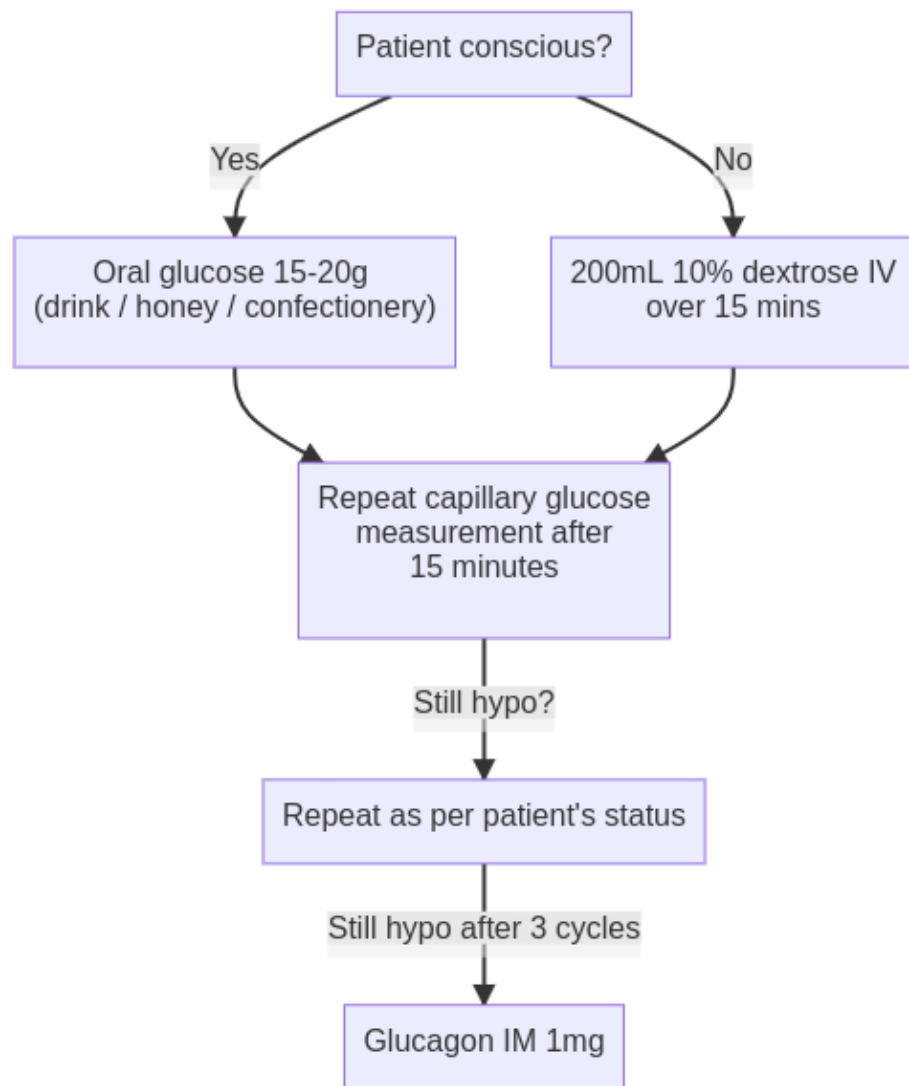


Figure 7.3: Managing hypoglycaemia

- 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 up to 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**

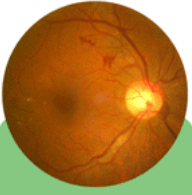

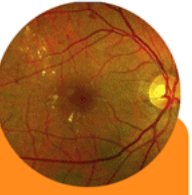
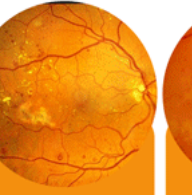
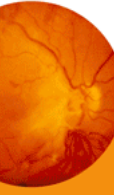
7.6 Oral Hypoglycaemic Agents

- **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 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)
- **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

Diabetic Retinopathy Classification

				
No disease visible	Mild nonproliferative diabetic retinopathy (NPDR) Localized swelling of the small blood vessels in the retina (microaneurysms)	Moderate NPDR Mild NPDR plus small bleeds (dot and blot haemorrhages), leaks (hard exudates) or closure (cotton wool spots) of small blood vessels.	Severe NPDR Moderate NPDR plus further damage to blood vessels (interretinal hemorrhages, venous beading, intraretinal microvascular abnormalities).	PDR New vessel formation or vitreous/preretinal hemorrhage or tractional retinal detachment

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

B₁ (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 α -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

B₁₂ deficiency

Functions of B₁₂

- 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-B₁₂ IM
- If combined folate and B₁₂ deficiency, only folate should not be given without B₁₂, as B₁₂ gets used up in folate recycling, deteriorating the B₁₂ deficiency symptoms. Always give both together.