

GP Institute of Australia

FRACGP Fellowship Exam Guide

Revision Essential Series

An Essential Companion for the RACGP exams

FIRST EDITION: Fourth revision

- A G Matt

“The eyes cannot see what the mind does not know.”

— Unknown

ACKNOWLEDGEMENT OF COUNTRY

We acknowledge the Traditional Custodians of the lands on which we work and live and recognise their continuing connections to land, waters and culture. We pay our respects to Elders past and present and extend that respect to all Aboriginal and Torres Strait Islander peoples today.

FRACGP Fellowship Exam Guide: Rapid Revision

An Essential Companion for the RACGP exams

First Edition

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CARDIOVASCULAR MEDICINE

CARDIOMYOPATHY

KEY POINTS: Muscle disease presenting as failure, arrhythmia or sudden death. Four echo patterns — dilated, hypertrophic, restrictive, Takotsubo. Three-generation family history every case; exertional syncope never benign.

Feature	DCM	HCM	RCM	Takotsubo
LV cavity/EF	Dilated; LVEF ≤40%	Normal/small; EF normal-high	Normal; EF preserved early	Normal; EF ↓ transiently, recovers
Wall thickness	Normal/thin	↑↑ asymmetric septal	↑ from infiltrate	Normal
Bedside clue	S3, displaced apex	Murmur louder standing/Valsalva	Right-sided congestion	Coronary mimic post-stressor
Sudden death	Moderate	Highest — young, athletes	Moderate; heart block	Low
First move	Four-pillar therapy	Cardiology; exercise advice	Hunt infiltrate	Angiography

▲ RED FLAGS: Exertional syncope, or sudden death in relative under 50 — same-day cardiology; no competitive sport until cleared.

Dilated cardiomyopathy — causes worth chasing

Cause	Clue and action
Familial	20–50% of idiopathic DCM; autosomal dominant; three-generation pedigree
Alcohol	Quantify standard drinks; reversible with abstinence
Tachycardia-induced	Persistent AF or SVT; rate or rhythm control restores EF
Anthracycline, trastuzumab	Baseline plus serial echo; cardio-oncology before stopping cancer therapy
Peripartum	Late pregnancy to 5 months postpartum; diagnosis of exclusion
Infiltrative/endocrine	Ferritin, transferrin saturation, TSH, serum ACE; cardiac MRI

Four pillars — LVEF ≤40%, start all four, titrate after

Pillar	Agent and dose	Practice point
ARNI or ACEi	Sacubitril-valsartan 24/26 mg bd → 97/103 mg bd; or ramipril 2.5 → 10 mg daily	36 h washout from ACEi
Beta-blocker	Carvedilol 3.125 mg bd → 25 mg bd; bisoprolol 1.25 → 10 mg daily	Only when euvolaemic; halve, never stop, if flaring
MRA	Spironolactone 12.5–50 mg daily	Potassium, creatinine at 1 and 4 weeks
SGLT2 inhibitor	Dapagliflozin or empagliflozin 10 mg daily	No titration; works without diabetes
Adjuncts	Furosemide for congestion; IV iron if ferritin <100	Anticoagulate only for AF or LV thrombus

Hypertrophic cardiomyopathy — sudden death and sport

Issue	Detail
Suspect	Unexplained LVH; ECG abnormal ~90%; systolic murmur louder standing/Valsalva, softer squatting
Risk markers	Prior arrest or sustained VT, unexplained syncope, maximal wall ≥30 mm, apical aneurysm, non-sustained VT, family sudden death
Risk tool	Specialist 5-year sudden-death estimate drives ICD; reassess 1–2 yearly
Sport	2024 guidance softened — recreational exercise encouraged; competitive sport by shared decision at expert centre
Drugs	Non-vasodilating beta-blocker first; mavacamten (PBS 2024, specialist) if gradient ≥50 mmHg persists
Avoid	Nitrates, dihydropyridines, dehydration, diuretic excess when obstructive

Restrictive — think infiltrate

Step	Detail
Amyloid clue	Low ECG voltages despite thick walls; apical sparing on strain; carpal tunnel, macroglossia, periorbital purpura
Confirm	Serum and urine free light chains plus electrophoresis, then bone scintigraphy for ATTR
Treat	Tafamidis 61 mg daily (PBS 2024, ATTR, NYHA I–II); cautious diuresis — preload dependent
Others	Haemochromatosis (venesection), sarcoid (steroids, pacing), radiation, eosinophilic

Takotsubo

Element	Detail
Setting	Emotional or physical stressor; postmenopausal women
Tests	Anterior ST elevation or deep T inversion; troponin lower than territory predicts
Discriminator	Apical ballooning crossing coronary territories; unobstructed arteries

RESPIRATORY MEDICINE

ACUTE PHARYNGITIS

KEY POINTS: ~15–20% of GP consults; **80–90% viral**, self-limiting

Key task: identify **GAS** — only common pathogen where antibiotics prevent Acute Rheumatic Fever (ARF)

Balance stewardship vs ARF prevention — lower threshold in Aboriginal/Torres Strait Islander & Pacific Islander patients

Pathogen	Clue	Antibiotic
Viral (rhinovirus, adenovirus, influenza)	Coryza, cough, gradual onset; conjunctivitis (adenovirus)	No
GAS	Sudden onset, high fever, exudate, tender anterior nodes, no cough/coryza ; scarlet fever rash	Yes — Pen V 10d or BPG IM stat
EBV (mononucleosis)	Marked fatigue, prolonged (1–4wk), posterior/generalised nodes, splenomegaly ~50%; avoid amoxicillin	No — supportive
Groups C/G Strep	Indistinguishable from GAS clinically; no ARF risk	Symptomatic only
Fusobacterium necrophorum	Adolescents; RADT-negative; risk of Lemierre's syndrome	Yes — metronidazole or benzylpenicillin
Diphtheria (rare; unvaccinated/travel)	Grey membrane, bull neck, low fever	Urgent — antitoxin + penicillin; notify PHU

Feature	GAS	Viral
Onset	Sudden (hours)	Gradual
Cough/coryza	Absent — strongest negative predictor	Present
Fever	High, >38.5°C	Variable
Exudate	~50% have it	Also seen in EBV, adenovirus
Rash	Scarlet fever (sandpaper)	Rare

Modified Centor/McIsaac Score — every adult with sore throat	
Criterion	Points
Fever >38°C	+1
Absence of cough	+1
Tender anterior cervical nodes	+1
Tonsillar swelling/exudate	+1
Age 3–14y (+1) / ≥45y (–1)	±1
≤1: no testing, no antibiotics	—
2–3: RADT/culture, treat if positive	—
≥4 or high ARF-risk: RADT/culture ± empirical treatment pending result	—

Management

Situation	Drug	Dose/Duration	Note
Standard, no allergy	Phenoxymethylpenicillin (Pen V)	Child 12.5mg/kg (max 500mg) BD–QID; Adult 500mg BD ×10 days	Gold standard; complete full 10 days even if better by day 3–5
Adherence concern / high ARF risk	Benzathine Pen G IM	Child <20kg 450,000U; ≥20kg 900,000U; Adult 1.2M units — single dose	Preferred in ATSI/Pacific Islander high-risk communities
Penicillin allergy — mild rash	Cephalexin	Child 12.5–25mg/kg (max 500mg) BD; Adult 500mg BD ×10 days	~2% cross-reactivity
Penicillin allergy — IgE/anaphylaxis	Roxithromycin or Azithromycin	Roxithromycin 300mg OD ×10d; Azithromycin 500mg day 1, 250mg days 2–5	Macrolide resistance rising (~3–5%)

- **Start antibiotics within 9 days of onset** to prevent ARF — goal is ARF prevention, not faster symptom resolution
- **Suppurative:** peritonsillar abscess (trismus, uvula deviation, muffled voice) → urgent ENT/ED, I&D + IV antibiotics
- Retropharyngeal/parapharyngeal abscess (children <6y; neck stiffness, drooling) → airway emergency
- Lemierre's syndrome (Fusobacterium, IJV thrombophlebitis, septic emboli) → urgent admission, IV benzylpenicillin + metronidazole

- **Non-suppurative (post-GAS):** ARF 2–4wk after pharyngitis (Jones criteria); notifiable; prevented only by antibiotic treatment
- PSGN 1–3wk after pharyngitis (haematuria, hypertension, oedema) — antibiotics do NOT prevent it
- Sydenham's chorea — delayed weeks–months, usually self-limiting

RENAL MEDICINE

HYPERNATRAEMIA

KEY POINTS: $\text{Na}^+ > 145 \text{ mmol/L}$ = relative **free water deficit**; almost always reflects impaired thirst or reduced water access — thirst is a powerful defence mechanism. Rate of correction matters as much as direction — too fast in either sodium disorder causes fatal cerebral injury.

Category	Mechanism/Examples
Poor intake	Elderly, infants, cognitive impairment, immobility, altered consciousness
Extra-renal loss	Fever, sweating, burns, tachypnoea — especially with poor intake
Central DI	Deficient ADH — TBI, pituitary tumour, craniopharyngioma, post-surgery, idiopathic
Nephrogenic DI	ADH resistance — lithium (commonest drug cause), CKD, hypercalcaemia, hypokalaemia, congenital
Osmotic diuresis	Hyperglycaemia, mannitol, high-protein enteral feeds, post-obstructive urea diuresis
GI loss	Osmotic diarrhoea (lactulose, sorbitol), unreplaced vomiting
Na^+ gain (uncommon)	Hypertonic saline/bicarbonate, salt poisoning, mild primary hyperaldosteronism

- Severity correlates with degree/rapidity of rise: mild (Na 145–155) — thirst, lethargy; moderate (155–165) — confusion, ataxia; severe (> 165) — seizures, coma, bridging vein rupture
- Volume status: most patients are hypovolaemic; euvoalaemia suggests DI; hypervolaemia suggests sodium gain

Test	Interpretation
Serum osmolality	Expected $> 295 \text{ mOsm/kg}$ in hypernatraemia — confirms the picture
$\text{Uosm} > 600$	Appropriate concentration — extra-renal loss ($\text{UNa} < 20$) or Na gain ($\text{UNa} > 100$)
$\text{Uosm} < 300$	Diabetes Insipidus despite hypernatraemia — water deprivation/DDAVP challenge required
$\text{Uosm} 300\text{--}600$	Partial DI, osmotic diuresis, or transition state

Correction — the critical step

Timing	Rate
Acute ($< 48 \text{ h}$)	$\sim 1 \text{ mmol/L/hr}$ for first few hours; near-normal within 24 h
Chronic ($> 48 \text{ h}$) / unknown	Max 10 mmol/L/24 h ($\sim 0.4 \text{ hr}$); replace free water deficit over 48–72 h

- Free Water Deficit (L)** = $0.6 (\text{♂})$ or $0.5 (\text{♀}) \times \text{weight (kg)} \times [(\text{Na}/140) - 1]$; add ongoing losses
- Step 1: if hypovolaemic/shocked — resuscitate with isotonic 0.9% NaCl FIRST, before addressing free water deficit
- Treat cause: DDAVP for central DI; cease lithium; manage hyperglycaemia; ensure water access for impaired patients
- Monitor: serum Na every 2–4 h, fluid balance, neurological status — adjust infusion rate to target

Extreme sodium values: $\text{Na} < 120$ or $> 160 \text{ mmol/L}$ warrants urgent assessment regardless of symptoms — both extremes carry a substantial risk of neurological injury and mandate hospital-level monitoring during correction.

- Common drug causes summary: hyponatraemia via SIADH — SSRIs, carbamazepine, valproate, TCAs, NSAIDs, cyclophosphamide; hypernatraemia via nephrogenic DI — lithium (commonest), also demeclocycline, foscarnet

△ RED FLAGS: $\text{Na} > 160 \text{ mmol/L}$, seizures/coma, or polyuria with hypernatraemia (suspect DI) → hospital urgently. Post-correction neurological deterioration → consider overcorrection (ODS) or inadequate correction.

★ EXAM TIP: Central DI responds to DDAVP; nephrogenic DI (lithium, CKD, hypercalcaemia, hypokalaemia) does not — a key exam discriminator.

✓ CLINICAL PEARL: Serum sodium reflects water balance relative to sodium, not total body sodium — it is the primary determinant of plasma osmolality and therefore of cell volume. Both hyper- and hyponatraemia cause neurological symptoms through osmotic water shifts into or out of brain cells.

- GP recognition: check EUC in any patient with neurological change, falls, confusion, or risk factors (elderly, diuretics, SSRIs, lithium, cardiac/liver disease)
- Thirst and water access: active carer attention to water intake is essential in elderly and cognitively impaired patients to prevent hypernatraemia
- Further DI workup if suspected: MRI pituitary/brain, ADH levels, formal water deprivation/DDAVP challenge under specialist supervision
- Urine sodium helps distinguish renal from extra-renal water loss when Uosm alone is ambiguous — interpret alongside volume status and Uosm together, not in isolation

Fluid choice comparison	
Fluid	Role
Oral/NGT water	Preferred route when tolerated — plain water or dextrose-equivalent
IV 5% dextrose (D5W)	Most common IV option — dextrose metabolised, leaving free water
IV 0.45% NaCl	Contains some sodium — less free water per litre than D5W, useful when partial sodium replacement is also needed

Severity grading by clinical features

Severity	Na (mmol/L)	Features
Mild	145–155	Thirst, lethargy, irritability, weakness
Moderate	155–165	Confusion, agitation, ataxia, muscle twitching
Severe	> 165	Seizures, coma, intracranial haemorrhage (bridging vein rupture)

- Diabetes insipidus confirmation: water deprivation test (fluid withheld, Uosm measured hourly) followed by DDAVP

CHILD AND YOUTH HEALTH

WELL-CHILD CARE AND ANTICIPATORY GUIDANCE

KEY POINTS: Every visit — plot growth, review milestones, check the Immunisation Register and give overdue doses, screen parental mood, document in the Blue Book.
Highest-yield messages: safe sleep, iron-rich solids with early allergen introduction, red reflex at every infant visit.

Blue Book schedule	
Visit	Core content
Birth	Vitamin K, hepatitis B dose, examination, red reflex, femoral pulses, hips
1–4 weeks	Bloodspot and hearing results, feeding, weight regain, jaundice, maternal mood
6–8 weeks	Full examination, hips, red reflex, 1st immunisations, Edinburgh Postnatal Depression Scale
4–18 months	Immunisations, growth, milestones; solids and iron from 6 months; dental visit by 12 months
2–4 years	BMI, language and autism surveillance, screen time; 4-year vision and hearing screen

Safe sleep and injury prevention	
Domain	Advice
Safe sleep	Supine every sleep; face uncovered; firm flat cot (AS/NZS 2172); own cot in parents' room 6–12 months; smoke-free; breastfeed; supervised tummy time
Sudden infant death	Peaks 2–4 months, 90% before 6 months. Never bed-share on a sofa, or with smoking, alcohol or sedatives
Car restraint	Rearward-facing to at least 6 months; harnessed seat to 4 years; booster to about 145 cm; rear seat, never front row under 4 years
Home	Hot water 50°C; four-sided pool fencing plus arms-reach supervision; poisons locked, ring 13 11 26; anchor furniture; no whole nuts or uncut grapes before 3–4 years

Feeding and nutrition	
Element	Guidance
Solids	Around 6 months, never before 4 months; iron-rich first; lumps by 9–10 months
Allergens	Peanut, egg, cow's milk, wheat, nuts, fish, sesame, soy in the first year, ideally by 12 months, then at least twice weekly. Delay does not prevent allergy
Avoid under 12 months	Honey, cow's milk as main drink, whole nuts, juice
Cow's milk	Full-cream from 12 months, under 500 mL/day — excess causes iron deficiency
Iron deficiency	Elemental iron 3–6 mg/kg/day, continued 3 months after haemoglobin normalises

Feeding and nutrition	
Element	Guidance
Teeth	Brush from first tooth; low-fluoride paste 18 months–6 years; dentist by 12 months

Screening and surveillance	
Domain	Action threshold
Red reflex	Absent, white or asymmetric → ophthalmology same or next day (cataract, retinoblastoma)
Squint	Any constant squint, or intermittent beyond 3–4 months → ophthalmology
Hearing	Confirm newborn screen; effusion over 3 months with language concern → audiology
Development	PEDS or ASQ-TRAK; refer any loss of skills, no words by 18 months, no two-word phrases by 24 months
Growth	WHO charts, corrected age to 2 years if preterm; concern if two major centiles crossed downward
Lead	Screen on risk; venous $\geq 5 \mu\text{g/dL}$ (0.24 $\mu\text{mol/L}$) → remove source, optimise iron, notify public health unit
Screen time	None under 2 years except video calls; 1 hour at 2–5 years; 2 hours at 5–17 years

▲ RED FLAGS: Button battery — time-critical; oesophageal necrosis within 2 hours. Nil by mouth, ring 13 11 26, urgent X-ray and endoscopic removal.
Bruising in a non-mobile infant, or injury not fitting the history — abuse; mandatory report.
Developmental regression at any age, or faltering growth crossing two centiles — refer.

NEONATAL ASSESSMENT AND COMMON PROBLEMS: PART I

KEY POINTS: APGAR documents, it does not direct — resuscitate on breathing, tone and heart rate; ventilation is what matters.
Full examination at **24–72 hours**: red reflex, femoral pulses, palpated palate, hips, anal patency.
Jaundice in the **first 24 hours** is pathological; **conjugated** jaundice always is — pale stools mean biliary atresia, and the Kasai window closes at **60 days**.

Newborn resuscitation	
Step	Action
First 60 s	Dry, warm (plastic wrap undried if <32 weeks), sniffing position, stimulate; oximeter right hand
Ventilate	Apnoea, gasping or heart rate <100 → 40–60/min, air 21% at term, 21–30% preterm; reassess after 30 s
Compressions	Heart rate <60 after effective ventilation → two-thumb 3:1 (90:30), oxygen 100%

MUSCULOSKELETAL MEDICINE

ORTHOPAEDIC TRAUMA — ASSESSMENT PRINCIPLES

KEY POINTS: Mechanism-focused history + look/feel/move/neurovascular exam underpins all MSK trauma assessment.
Neurovascular compromise and compartment syndrome override every other finding — act immediately.

History	
Domain	Detail
Mechanism	Energy, direction of force
Pain	SOCRATES framework
Function	Weight-bearing, ROM, grip
Associated Sx	Neurovascular — numbness, tingling, pallor
PMHx	Bone health, bleeding disorders, immunosuppression
Medications	Anticoagulants, steroids
Tetanus status	Especially open wounds
Occupation/ activities	Functional demands, sport, return-to-work/play planning

Examination	
Step	Assess
Look	Swelling, bruising, deformity, skin integrity — open fracture
Feel	Point tenderness, temperature, crepitus, compartment tension
Move	Active/passive ROM, joint stability (special tests), motor function
Neurovascular	Distal pulses, cap refill, sensation

Modality	Use
X-ray	1st line fracture/dislocation — min 2 views
Ultrasound	Soft tissue, tendon/ligament, effusion
CT	Complex fracture, detailed bone anatomy
MRI	Ligament/cartilage/occult fracture

Initial management	
Element	Detail
RICE	Rest, ice, compression, elevation
Analgesia	Paracetamol → NSAID → opioid PRN, stepwise
Immobilise	Sling/splint/backslab as indicated
Open fracture	Sterile dressing, do NOT probe wound, NV check, tetanus prophylaxis, urgent ED
NV compromise	Urgent ED/orthopaedic referral

⚠ RED FLAGS: 6 Ps of compartment syndrome — pain out of proportion (early, worse on passive stretch), pallor, pulselessness (late), paraesthesia, paralysis, pressure. Urgent surgical decompression.
Open fracture; dislocation of a major joint (hip/shoulder/knee); inability to weight-bear (Ottawa rules); significant deformity/gross instability; suspected spinal injury with neurological symptoms.

- **Differentials for MSK trauma** — sprain (ligament), strain (muscle/tendon), fracture, dislocation, tendinopathy, bursitis, osteoarthritis, nerve entrapment (e.g. carpal tunnel syndrome).

Grading soft-tissue injury severity	
Grade	Sprain/strain severity
I	Mild — minimal fibre disruption, stable, mild swelling
II	Moderate — partial tear, some instability, moderate swelling/bruising
III	Severe — complete tear/rupture, marked instability, may need surgical repair

Analgesia is stepwise (paracetamol → NSAID → opioid PRN) but should never delay reduction of a dislocation or obscure a deteriorating neurovascular exam — reassess after each analgesic step, not just before it.

✓ CLINICAL PEARL: Pain out of proportion to injury, worsened by passive stretch, is the critical **EARLY** sign of compartment syndrome — never dismiss as 'just pain'. Scaphoid, paediatric supracondylar and Jones fractures each carry disproportionate risk relative to apparent severity.

Safe analgesia choice matters in trauma: avoid NSAIDs in suspected fracture with active bleeding risk or renal impairment, use paracetamol as the base agent for most patients, and reserve opioids for severe pain with a clear plan for short-course use and safety-netting about driving/sedation.

Document a clear neurovascular baseline **BEFORE** any reduction or splinting, and repeat it immediately after — a deteriorating neurovascular exam after intervention (e.g. after a splint is applied) is easy to miss if no baseline was recorded, and changes management urgently.

Pathological fractures — a fracture from a mechanism too minor to normally break bone — should prompt consideration of an underlying lesion (metastasis, myeloma, primary bone tumour, osteoporosis, Paget's disease); request appropriate imaging and bloods rather than treating it as a routine traumatic fracture alone.

Osteoporotic fragility fractures (wrist, hip, vertebral) are sentinel events — any minimal-trauma fracture in a patient over 50 should trigger bone density assessment and fracture risk evaluation, since the risk of a subsequent fracture is substantially higher in the following one to two years without intervention.

UPPER LIMB FRACTURES & DISLOCATIONS

PSYCHIATRY

MAJOR DEPRESSIVE DISORDER

Core diagnosis MDE — ≥5 of 9 SIG E CAPS symptoms, ≥2 weeks, most of most days; ≥1 must be depressed mood or anhedonia; causes functional impairment; not due to substance/medical cause; **never** manic/hypomanic (else bipolar — screen every patient before prescribing).

SIG E CAPS — 9 DSM-5 Symptoms

Domain	Feature
Sleep	insomnia or hypersomnia
Interest	anhedonia — loss of pleasure in almost all activities
Guilt	worthlessness or excessive/inappropriate guilt
Energy	fatigue nearly every day
Concentration	impaired concentration or indecisiveness
Appetite/weight	±5% body weight change in a month, or appetite change
Psychomotor	agitation or retardation, observable by others
Suicidality	recurrent thoughts of death, ideation, plan or attempt

Severity — PHQ-9

Severity	Criteria
Subthreshold	<5 symptoms; PHQ-9 0–4
Mild	5 symptoms; PHQ-9 5–9
Moderate	5–7 symptoms; PHQ-9 10–19
Severe	most of 9 symptoms; PHQ-9 ≥20

Key Specifiers

Specifier	Distinguishing feature	Management note
Melancholic	pervasive anhedonia; early-morning waking; mood worse AM; psychomotor change; profound guilt	better response to medication/ECT than CBT alone
Atypical	mood <i>reactivity</i> (brightens to positive events); hypersomnia; hyperphagia; leaden paralysis	responds well to SSRIs and psychotherapy

Antidepressant Prescribing

Agent	Class	Starting → therapeutic dose	Key notes
Escitalopram	SSRI	5mg (2.5mg elderly) → 10–20mg OD	best tolerated, fewest interactions; first choice for most
Sertraline	SSRI	25mg → 50–200mg OD	most prescribed in Australia; safest in pregnancy; low cost
Fluoxetine	SSRI	10–20mg → 20–60mg OD	long half-life, lowest discontinuation risk; CYP2D6 inhibitor
Paroxetine	SSRI	10mg → 20–60mg OD	most anticholinergic; highest discontinuation risk; avoid in pregnancy
Venlafaxine XR	SNRI	37.5mg → 75–225mg OD	good for pain/anxiety; check BP with dose rises
Duloxetine	SNRI	30mg → 60–120mg OD	good for neuropathic/chronic pain; take with food

Key Specifiers

Specifier	Distinguishing feature	Management note
Psychotic	mood-congruent delusions (guilt, nihilism, somatic) or hallucinations	antidepressant + antipsychotic (not antidepressant alone); ECT often preferred
Peripartum	onset in pregnancy or ≤4 weeks postpartum (extended to 12m clinically)	EPDS ≥13 = probable depression; sertraline/paroxetine safest in breastfeeding
Seasonal	full episodes autumn/winter, remission spring/summer, ≥2 years	light therapy (10,000 lux, 30 min AM) first-line

Differentials to Exclude

Diagnosis	Discriminator	Action
Bipolar disorder	lifetime manic/hypomanic episode, even subtle or unrecalled	screen with MDQ before any antidepressant; if positive, do not start SSRI alone
Hypothyroidism	cold intolerance, weight gain, bradycardia, slow reflexes	check TSH/free T4 in all new presentations; treat cause first
Substance-induced	symptoms track with use/withdrawal, alcohol most common	UDS; reassess after 4–6 weeks abstinence before diagnosing MDD
Adjustment disorder	identifiable stressor, <5 symptoms or <2 weeks duration	supportive therapy; resolves within 6 months of stressor ending
Normal grief	waves of sadness, self-esteem intact, function largely preserved	support only, unless full MDE criteria also met (grief with MDE)

ENDOCRINOLOGY

METABOLIC SYNDROME AND PREDIABETES

KEY POINTS: Metabolic syndrome is a risk marker, not a disease — treat each component and the absolute cardiovascular risk. Prediabetes is not inevitable progression: intensive lifestyle change roughly halves it. Never prescribe a drug 'for metabolic syndrome'.

IDF criteria — central obesity required PLUS two or more	
Component	Threshold
Waist circumference	Men ≥94 cm, women ≥80 cm (Europid); ethnicity-specific cut-offs
Triglycerides	≥1.7 mmol/L or treated
HDL cholesterol	Men <1.0, women <1.3 mmol/L, or treated
Blood pressure	≥130/85 mmHg or treated
Fasting glucose	≥5.6 mmol/L or known type 2 diabetes

Prediabetes thresholds		
Category	Test	Value
Impaired fasting glucose	Fasting venous plasma glucose	6.1–6.9 mmol/L
Impaired glucose tolerance	2-hour 75-g oral glucose tolerance test	7.8–11.0 mmol/L
High-risk HbA1c	Laboratory HbA1c	6.0–6.4% (42–47 mmol/mol)
Diabetes	Any threshold exceeded	Fasting ≥7.0; 2-hour ≥11.1; HbA1c ≥6.5%

- **Progression** — roughly 5–10% per year to type 2 diabetes; impaired fasting glucose *plus* impaired glucose tolerance is highest risk
- **Metabolic syndrome** — about 5-fold diabetes risk, 2–3-fold cardiovascular risk
- **Look for** — fatty liver, polycystic ovary syndrome, obstructive sleep apnoea
- **Reversion** — many return to normoglycaemia; the label communicates risk, not destiny
- **Already at risk** — a person with prediabetes already carries excess cardiovascular risk, so treat blood pressure and lipids now rather than waiting for the diabetes label
- **Complications** — retinopathy and neuropathy occasionally appear before diagnostic thresholds are crossed

Who to screen and how often	
Group	Interval
Adults >40	AUSDRISK 3-yearly; score ≥12 → fasting glucose or HbA1c
Aboriginal and Torres Strait Islander people	From age 18: HbA1c or fasting glucose 3-yearly
Previous gestational diabetes	Oral glucose tolerance test at 6–12 weeks postpartum, then 1–3 yearly for life

Who to screen and how often	
Group	Interval
High risk	Polycystic ovary syndrome, cardiovascular disease, obesity, hypertension, dyslipidaemia, antipsychotics or glucocorticoids, family history — test directly, yearly
Known prediabetes	HbA1c or fasting glucose at least annually
Symptoms	Polyuria, polydipsia, weight loss, blurred vision, recurrent infection — diagnostic testing now, not a risk score

Baseline workup	
Measure	Detail
Anthropometry	Waist at the midpoint between lowest rib and iliac crest; body mass index; blood pressure on two occasions
Bloods	Fasting lipids, fasting glucose or HbA1c, liver function, urate, eGFR and urine albumin-to-creatinine ratio
Risk calculation	Australian absolute cardiovascular disease risk calculator; recalculate after any change
Comorbidity	Sleep apnoea screening questionnaire, menstrual history, liver imaging if transaminases raised

- **Lifestyle first** — 5–10% weight loss, 150 min/week moderate activity plus twice-weekly resistance work, Mediterranean or low-glycaemic-index pattern; cuts progression about 58%, outperforming metformin
- **Refer** — accredited practising dietitian and exercise physiologist under a chronic disease management plan; structured group programs beat advice alone
- **Blood pressure** — target below 130/80 mmHg
- **Lipids** — statin decided by absolute cardiovascular risk, not by the syndrome label
- **Smoking, alcohol** — cessation and moderation
- **Sleep** — treat obstructive sleep apnoea; short sleep worsens insulin resistance
- **Review** — repeat weight, waist and glycaemia at 3 months to prove the plan is working
- **Metformin in prediabetes** — not TGA-registered and not PBS-subsidised for this indication; off-label, private cost
- **Consider only if** — age <60 with body mass index ≥35, previous gestational diabetes, or HbA1c climbing despite genuine lifestyle change
- **Dose** — 500 mg daily with food, titrate to 1 g twice daily; never a substitute for lifestyle change
- **Weight agents** — no PBS subsidy at the prediabetes stage; private script only
- **Trap** — treating a number rather than a person: statin and antihypertensive decisions follow absolute risk, not the count of syndrome components
- **Trap** — an HbA1c of 6.0–6.4% mandates lifestyle intervention and annual review, not a diabetes diagnosis and not a script
- **Trap** — a normal fasting glucose does not exclude impaired glucose tolerance; do the oral glucose tolerance test when previous gestational diabetes or polycystic ovary syndrome makes post-load hyperglycaemia likely
- **Trap** — vague advice fails; specify kilograms, minutes per week and a review date

NEUROLOGY

NEUROLOGICAL LOCALISATION & COMMON SYNDROMES

KEY POINTS: Localising the lesion (cortex vs brainstem vs cord vs peripheral nerve) directs targeted imaging and reveals emergencies at the bedside.

Three bedside rules carry the most exam weight: the CN III pupil rule, UMN vs LMN facial palsy, and cauda equina/conus red flags.

Examination sequence	What to check
Mental state/ LOC	Orientation, attention, GCS
Cranial nerves	Visual fields, pupils, eye movements, facial symmetry, palate/tongue
Motor	Tone, power (MRC 0–5), bulk, fasciculations
Sensory	Light touch, pinprick, proprioception, vibration
Coordination	Finger-nose, heel-shin, dysdiadochokinesia
Reflexes	Biceps/triceps/supinator, knee, ankle, plantar
Gait	Normal, ataxic, spastic, shuffling/magnetic, waddling

Lobe	Key function
Frontal	Motor cortex, executive function, personality, Broca's speech area (dominant hemisphere)
Parietal	Sensory cortex, spatial awareness, language (part Wernicke's)
Temporal	Hearing, memory (hippocampus), emotion (amygdala), Wernicke's speech area
Occipital	Vision

Lobe	Key function
Basal ganglia	Motor control — Parkinson's disease (dopamine deficiency), Huntington's disease
Limbic system	Emotion processing, memory formation (amygdala, hippocampus)

RAISED ICP & HERNIATION — NEUROSURGICAL EMERGENCY

Sign category	Features
Raised ICP	Headache worse am/bending, N&V, ↓consciousness, papilloedema
Cushing's triad (late)	↑BP, ↓HR, irregular respirations
Uncal herniation (early key sign)	CN III compression → ipsilateral fixed dilated pupil + ptosis + 'down and out' eye

⚠ RED FLAGS: Ipsilateral fixed dilated pupil = urgent referral. Do not wait for further neurological deterioration.

Vascular territory	Features
ACA	Contralateral leg > arm weakness/sensory loss
MCA	Contralateral arm/face > leg weakness/sensory loss + aphasia (dominant hemisphere) or neglect (non-dominant)
PCA	Contralateral homonymous hemianopia ± sensory loss
Lacunar (deep structures)	Basal ganglia, thalamus, internal capsule, pons → pure motor or pure sensory syndrome

CLASSIC BRAINSTEM STROKE SYNDROMES

Syndrome	Site/vessel	Features
Weber	Midbrain — PCA branches	Ipsilateral CN III palsy + contralateral hemiparesis
Wallenberg (lateral medullary)	Medulla — PICA	Ipsilateral facial pain/temp loss, ataxia, Horner's + contralateral body pain/temp loss; dysphagia/hoarseness
Locked-in	Ventral pons — basilar artery	Quadriplegia + anarthria; consciousness and vertical eye movements/blinking preserved

✓ **CLINICAL PEARL:** NPH triad in the elderly — Wet (urinary incontinence), Wobbly (magnetic/shuffling gait apraxia), Wacky (cognitive decline) — is potentially reversible with CSF shunting; refer to neurosurgery.

★ **EXAM TIP:** CN VI palsy is a 'false localising sign' of raised ICP — the long intracranial course of the nerve makes it vulnerable to pressure anywhere along its path, not just at the lesion site.

Cranial nerve lesion	Features
IV (trochlear)	Vertical diplopia (worse reading/going downstairs), head tilt AWAY from lesion
VI (abducens)	Horizontal diplopia (worse looking towards lesion), failed abduction — consider raised ICP (false localising sign)
X (vagus)	Uvula deviates AWAY from lesion side, hoarseness
XII (hypoglossal)	Tongue deviates TOWARDS lesion on protrusion

Cranial nerve lesion	Features
III (oculomotor)	Down-and-out eye, ptosis — pupil status is critical (below)

GASTROENTEROLOGY

ORAL & SALIVARY GLAND DISEASE

KEY POINTS: Colour rule: white patch that won't scrape off = **leukoplakia** (biopsy); fiery red patch = **erythroplakia** (biopsy, ~50–90% SCC risk).

Any oral lesion or ulcer >2–3 weeks without clear benign cause = biopsy or urgent referral.

Common benign oral lesions

Condition	Features	Management
Aphthous ulcers	Painful recurrent shallow ulcers, yellow base/red halo; if severe/atypical check FBC, iron, B12, folate, coeliac screen	Topical corticosteroid (triamcinolone in orabase); chlorhexidine mouthwash
Oral candidiasis	Risk: dentures, ICS, antibiotics, xerostomia, immunosuppression; chronic hyperplastic type needs biopsy	Topical nystatin/miconazole 1st line; oral fluconazole if refractory
Oral HSV	Primary gingivostomatitis vs recurrent cold sore	Oral aciclovir if started <72h
Mucocele/ranula	Bluish dome swelling, lip/floor of mouth	Often self-resolves; excise if persistent

Oral potentially malignant disorders (biopsy + surveillance all)

OPMD	Features	SCC risk
Leukoplakia	Non-scrapable white patch; worse if non-homogenous/floor of mouth/tongue	1–20%

Oral potentially malignant disorders (biopsy + surveillance all)

OPMD	Features	SCC risk
Erythroplakia	Fiery red; almost always dysplasia/SCC on biopsy	50–90%
Oral submucous fibrosis	Areca/betel nut ± tobacco; progressive trismus — STOP areca nut	2–8%
Erosive lichen planus	Red/ulcerated OLP (reticular white-lace form is low risk)	1–2%
Actinic cheilitis	Lower lip UV damage — sun protection essential; precursor to lip SCC	Significant

Salivary condition	Features	Management
Acute suppurative sialadenitis	Bacterial (S. aureus); painful unilateral swelling ± pus from duct	Flucloxacillin/cephalexin ± metronidazole, hydration, sialagogues
Mumps (viral parotitis)	Bilateral painful swelling; orchitis risk	Supportive — notifiable ; MMR prevents
Sialolithiasis	Submandibular >> parotid; mealtime pain/swelling; US first	Conservative; sialoendoscopy/surgery if persistent
Sjögren's syndrome	Dry eyes+ mouth ± parotid swelling; anti-Ro/La+; ↑caries and lymphoma risk	Artificial saliva/tears, dental care, rheumatology

Salivary tumour	Key features	Management
Pleomorphic adenoma (benign)	Commonest salivary tumour; slow, firm, mobile parotid mass	Parotidectomy
Warthin's tumour (benign)	Parotid only; older male smokers; cystic ± bilateral	Excision
Mucoepidermoid carcinoma	Commonest malignant salivary tumour (esp. parotid)	Surgery ± RT, MDT
Adenoid cystic carcinoma	Commonest in submandibular/minor glands; perineural spread → CN VII palsy, late recurrence	Surgery ± RT, MDT

Malignant-risk gradient by gland depth: parotid (~80% of tumours, ~80% benign) > submandibular (~50% malignant) > minor glands (>50% malignant).

Differentials by presentation

Presentation	Key differentials
Oral ulcers	RAS, HSV, trauma, OLP, malignancy, IBD/Behçet's
White patches	Candidiasis, leukoplakia, OLP, frictional keratosis, oral hairy leukoplakia
Salivary swelling	Infection, sialolithiasis, tumour, Sjögren's, sialadenitis, lymphoma

Oropharyngeal SCC — risk profile & work-up

Subtype	Risk factors	Diagnosis/staging
Oral cavity SCC (~90% of oral cancers)	Tobacco + alcohol (synergistic), areca nut, OPMDs	Biopsy suspicious lesion; CT/PET-CT ± panendoscopy
Oropharyngeal SCC	HPV-16 (tonsil/tongue base) — younger patients, better prognosis than HPV-negative	p16 (HPV) testing; neck lump often first sign

PREVENTION AND PREVENTIVE CARE

SMOKING CESSATION AND VAPING

KEY POINTS: Tobacco = Australia's leading preventable cause of death (~21,000/yr); CVD is largest attributable burden.
Brief GP advice + pharmacotherapy + behavioural support = highest quit rates. Ask every patient about ALL nicotine products (cigarettes, vapes, shisha, heated tobacco, pouches) — not just cigarettes.

Step	Action
Ask	Systematically identify and document smoking AND vaping status for every patient — make it a routine vital sign; time to first use, amount, duration, previous quit attempts
Advise	Clear, strong, personalised, non-judgmental advice to quit — link quitting to the patient's own presenting issue: 'the best thing you can do for your [blood pressure/pregnancy/breathing] is to stop smoking'
Assess	Dependence — <5 min to first cigarette or ≥31/day = high dependence (Heaviness of Smoking Index); Stages of Change (precontemplation → maintenance); vaping: time to first vape, near-continuous use, withdrawal
Assist	Quit date 2–4 weeks (specific date, not 'soon'); 4 D's (Delay/Distract/Deep breath/Drink water); combination NRT or varenicline; Quitline 13 78 48 callback (triples quit rate vs brief advice alone)
Arrange	Follow-up around quit date (days 3–7 hardest for withdrawal), then 1/3/12 months; celebrate success, monitor relapse warning signs, adjust pharmacotherapy, reframe relapse as learning not failure

First-line pharmacotherapy

Option	Key dosing / points
Combination NRT	Patch (16h or 24h; 21 mg ≥20/day or high dependence, 14 mg for 10–20/day, 7 mg <10/day or tapering) + PRN short-acting (gum 'chew and park', lozenge, inhalator, mouth spray) for breakthrough craving. Most effective NRT strategy; PBS-subsidised; can pre-load before quit date; continue 8–12 weeks then individualise
Varenicline	0.5 mg daily d1–3 → 0.5 mg BD d4–7 → 1 mg BD d8+ (full dose). Most effective single agent — reduces withdrawal AND the rewarding effect of smoking. Quit date 1–2 wks after starting (or flexible quit date weeks 1–5). Standard course 12 weeks, extend to 24 wks if high relapse risk. Nausea (take with food, full glass of water); vivid dreams (move evening dose earlier). Reduce dose eGFR<30. EAGLES trial: no excess neuropsychiatric risk incl. stable mental illness. PBS Authority — max 2×12-week courses per 3 years
Bupropion SR	150 mg daily×3d then 150 mg BD; start 1–2 wks pre-quit, continue 7–9 weeks. Absolute CI: seizure disorder, current/past eating disorder, MAOI <14d. Inhibits CYP2D6 — check interactions with SSRIs, TCAs, antipsychotics, beta-blockers, opioids before prescribing
Cytisinicline	Nicotinic partial agonist, now available in Australia; lower cost, shorter titration, similar efficacy to varenicline; emerging cost-effective alternative

Setting a personalised quit plan

- Negotiate a specific quit date together rather than imposing one — patient-chosen dates within the 2–4 week window improve commitment
- Identify the patient's personal 'why' (a grandchild, a health event, cost) and revisit it explicitly at follow-up — motivation drawn from the patient's own values outweighs generic health warnings
- Pre-plan for the first weekend and any upcoming high-risk social event (celebration, work stress) rather than leaving the quit date to coincide with one by accident

Combination NRT — practical technique

Element	Detail
Patches	16h or 24h; apply to hairless skin, rotate sites to reduce irritation; 16h patch (remove at bedtime) suits vivid dreams/morning cravings; 24h suits night-waking cravers
Short-acting forms	Gum: chew 5–6 times until tingling then 'park' between cheek and gum, repeat, avoid acidic drinks 15 min before/during. Lozenge: dissolve slowly, don't chew. Inhalator: fastest-acting NRT for urgent cravings. Mouth spray: 1–2 sprays (1–2 mg) into mouth not throat
Pre-loading	Patch may be started before the quit date to reduce pre-quit smoking and lower withdrawal severity on quit day
Safety	Very safe; caution only in the immediate post-MI period or with serious arrhythmias — the risks of continued smoking almost always outweigh NRT risk, including in pregnancy

MEN'S HEALTH

MALE INFERTILITY & PRECONCEPTION

Snapshot Infertility = no conception after **12 months** (or **6 months** if female ≥ 35) unprotected regular intercourse
Male factor sole cause **20–30%**; contributes in up to **50%**
Assess **both partners concurrently** — never investigate one alone

Aetiology framework		
Category	Hormone pattern	Key causes
Pre-testicular (~1–2%)	Low T; low/inappropriately normal LH+FSH	Anabolic steroids/exogenous testosterone (most important reversible cause); Kallmann; prolactinoma; opioids; obesity
Testicular (~30–40%)	Low T (if severe); high LH+FSH	Idiopathic; varicocele (~30–40% of infertile men, most common correctable); Klinefelter (47XXY); cryptorchidism; mumps orchitis; chemo/radiation
Post-testicular (~10–20%)	Normal T, LH, FSH, testicular volume	CBAVD (CFTR mutation); epididymal obstruction (post-STI); vasectomy; retrograde ejaculation
Idiopathic (~30–40%)	Variable, often normal	No cause found

Hypogonadotropic vs hypergonadotropic hypogonadism — key causes		
Type	LH/FSH	Common causes
Hypogonadotropic (pre-testicular)	Low or inappropriately normal	Kallmann syndrome (anosmia + GnRH deficiency); prolactinoma/pituitary tumour or infiltration; exogenous testosterone/anabolic steroids (most common reversible cause); opioid use; severe obesity; haemochromatosis
Hypergonadotropic (testicular)	High (compensatory)	Klinefelter syndrome (47XXY); bilateral cryptorchidism; mumps orchitis; testicular torsion; chemotherapy/radiotherapy; idiopathic primary testicular failure

Hypogonadotropic causes are often reversible with hCG $\pm$ FSH; hypergonadotropic causes reflect established testicular damage — management shifts to sperm retrieval/ART rather than hormonal restoration.

Examination Red Flags

- **Testicular mass** — cancer until proven otherwise; urgent scrotal USS + AFP/ hCG/LDH **before** any surgery
- **Right-sided varicocele** — image renal tract to exclude RCC compressing gonadal vein (left-sided 85–90% normal)
- **Testes <15 mL** on orchidometer = abnormal; soft = spermatogenic failure, firm = obstructive/normal

Semen analysis — WHO 6th edition (2021) lower reference limits	
Parameter	Lower limit
Volume	≥ 1.4 mL
Concentration	≥ 16 million/mL
Total count	≥ 39 million/ejaculate
Progressive motility	$\geq 30\%$
Total motility	$\geq 42\%$
Morphology (strict Kruger)	$\geq 4\%$ normal forms
Vitality	$\geq 54\%$ live

2–3 samples, 1–4 weeks apart; 2–7 days abstinence; entire sample to lab within 60 min at body temperature.

- **Azoospermia** — confirm on centrifuged pellet; obstructive (normal FSH/volume, CBAVD) vs non-obstructive (raised FSH, primary failure) — TESE viable only if focal spermatogenesis present
- **Severe oligospermia (<5 million/mL) or azoospermia** — karyotype (exclude Klinefelter) + Y-microdeletion (AZFa/b = no retrieval; AZFc = TESE may succeed)
- **Low volume (<1.4 mL)** — post-ejaculate urine for retrograde ejaculation
- **CBAVD** — both partners CFTR-tested before ICSI/TESE; genetic counselling mandatory

Work-up once semen analysis abnormal	
Test	When / what it shows
Hormonal profile (AM total T, LH, FSH $\pm$ prolactin)	Raised FSH + low count = primary testicular failure; low FSH + low count = pre-testicular (hypogonadotropic)
Karyotype	Azoospermia or count <5 million/mL — excludes Klinefelter 47XXY
Y-chromosome microdeletion	AZFa/AZFb deletion = no sperm retrievable; AZFc = TESE may succeed
CFTR mutation analysis	CBAVD identified — both partners tested before ICSI/TESE
Scrotal Doppler USS	Confirms varicocele grade, testicular volume, excludes mass

Management

- **Anabolic steroid/exogenous testosterone use** — cease; recovery 6–24 months; hCG $\pm$ FSH (not TRT, which suppresses LH/FSH) accelerates recovery
- **Lifestyle** — smoking cessation; alcohol ≤ 10 drinks/week (NHMRC); weight loss 5–10% if obese; avoid scrotal heat; folate + antioxidants (e.g. Menevit) from 3 months pre-conception
- **Varicocele repair** — grade II–III + abnormal semen analysis + correctable/normal female partner; improves parameters in ~60–70%

DERMATOLOGY

ATOPIC DERMATITIS

KEY POINTS: Clinical diagnosis: itch plus eczematous morphology, chronic relapsing course, personal or family atopy. No test confirms it. Daily emollient plus adequate-potency corticosteroid to flares is the whole answer; undertreatment, not steroid toxicity, is the usual reason for failure.

Distribution by age

Age	Pattern
Infant under 2 y	Face, scalp, extensor limbs; weeping, crusted; nappy area spared

Corticosteroid potency matched to site

Potency	Australian example	Site	Use
Mild	Hydrocortisone 1% (Sigmacort, DermAid)	Face, eyelids, flexures, infants	bd until clear
Moderate	Methylprednisolone aceponate 0.1% (Advantan); triamcinolone 0.02% (Aristocort)	Trunk, limbs; face if mild fails	Advantan once daily
Potent	Mometasone 0.1% (Elocon, Novasone); betamethasone dipropionate 0.05% (Diprosone, Eleuphrat)	Thick or lichenified limb and trunk plaques	Daily, review at 2 weeks
Very potent	Betamethasone dipropionate 0.05% optimised vehicle (Diprosone OV)	Palms, soles, scalp only	Short bursts; clobetasol rarely stocked here

- **1 fingertip unit** — 0.5 g, covers two adult palms, about 2% body surface
- **Adult whole body** — roughly 40 units, 20 g per application; face and neck 2.5, one arm 3, one leg 6, trunk front 7, back 7
- **Prescribe enough** — 100 g of a potent product for a widespread adult flare; a 30 g tube lasts a child days
- **Emollient** — 250 to 500 g weekly adult, 250 g child, about ten times the steroid quantity; soap substitute, never soap

Flare adjuncts and infection

Measure	Detail
Wet dressings	Steroid, then damp tubular bandage under a dry layer, 4 h or overnight, 3 to 5 nights, for severe excoriated flares
Weeping or crusted	Dilute bleach bath 12 mL of 4% bleach per 10 L, 10 min, twice weekly; or potassium permanganate soaks
When to swab	Only for failure to respond, pustules, recurrent infection or suspected resistance; Staph aureus colonises nearly all
Sedating antihistamine	Night use for sleep only; no daytime antipruritic effect

⚠ RED FLAGS: Clustered punched-out vesicles or erosions with fever and pain on eczema is eczema herpeticum: valaciclovir 500 mg bd (child aciclovir five times daily), urgent ophthalmology if periocular, admit if systemic.

- **Pimecrolimus 1% (Elidel)** — PBS Streamlined, from 6 months; steroid-sparing on face, eyelids, flexures; burns for the first few days
- **Dupilumab (Dupixent)** — PBS Authority, 12 years and over only, severe disease with EASI 20 or more or severe hand or

Distribution by age

Age	Pattern
Child 2 y to puberty	Flexures (antecubital, popliteal), wrists, ankles; drier, lichenified
Adult	Flexures, hands, eyelids, neck; lichenification, chronic hand eczema

- **Essential criterion** — pruritus
- **Plus 3 of** — flexural involvement; onset under 2 y; history of asthma or hay fever (first-degree relative if under 4 y); generalised xerosis; visible flexural dermatitis
- **Not needed** — IgE, biopsy; food allergy testing only for immediate reactions or failure despite optimal topicals

face involvement, after 4 weeks of optimal topical therapy; initiated by dermatologist, immunologist or paediatrician

- **Avoid** — oral prednisolone for routine flares; rebound follows

✓ **CLINICAL PEARL:** Steroid phobia drives most treatment failure: name it, and explain that atrophy follows months of potent steroid on thin skin, not a two-week course, while untreated inflammation itself thins and scars.

CONTACT DERMATITIS

KEY POINTS: Distribution is the diagnosis: sharp margins, geometric or linear shapes, and odd sites. Irritant accounts for most cases; allergic needs prior sensitisation and patch testing to prove it. Identifying and removing the culprit is the only curative step.

Irritant versus allergic

Feature	Irritant	Allergic
Onset	Minutes to hours; cumulative	24 to 72 h after re-exposure
Who	Anyone given enough dose	Only the sensitised minority
Border	Confined strictly to contact area	Spreads beyond contact, may generalise
Symptom	Burning, stinging, pain over itch	Itch dominant, vesicles, weeping
Hands	Web spaces, dorsum, wet work	Fingertips, under rings, glove line
Course	Improves within days of removal	Persists 2 to 4 weeks after removal

HAEMATOLOGY

ANAEMIA — ASSESSMENT & CLASSIFICATION

KEY POINTS: Anaemia = ↓Hb/RBC mass — a **sign**, not a diagnosis; causes fatigue, dyspnoea, ↓quality of life
 Thresholds — men <130g/L; non-pregnant women <120g/L; pregnant <110g/L (check local reference range)
 MCV + reticulocyte count give the initial diagnostic framework; blood film is essential in every work-up

MCV	Category	Typical causes
<80 fL	Microcytic	IDA, thalassaemia, ACD (sometimes), sideroblastic anaemia
80–100 fL	Normocytic	ACD, CKD, hypothyroid, early deficiency, marrow failure, acute blood loss
>100 fL	Macrocytic	B12/folate deficiency (megaloblastic), alcohol, liver disease, hypothyroid, MDS

Reticulocytes	Interpretation	Causes
Low/normal	Underproduction	Deficiency states, ACD, CKD, marrow failure
High	↑Production — loss/destruction	Acute bleed (after 3–5 days), haemolysis

Key history & examination

Domain	Key points
Anaemia symptoms	Fatigue, dyspnoea, palpitations, headache, poor concentration
Bleeding	GI (melaena/PR bleed), menorrhagia, haematuria
Diet	Iron/B12/folate intake — vegan, elderly, alcohol; pica (IDA)
Malabsorption	Coeliac/IBD symptoms, bariatric surgery
Haemolysis	Jaundice, dark urine, gallstones, family history
Chronic disease	CKD, liver disease, RA, IBD, cancer
Marrow failure	Bruising, infections
Neurological	Peripheral neuropathy, balance/cognitive issues (query B12)
Medications	NSAIDs, anticoagulants, metformin, PPIs, chemotherapy
FHx/ethnicity	Thalassaemia, haemoglobinopathies

Condition	Signs
General	Pallor, jaundice, lymphadenopathy, tachycardia, postural drop
IDA	Koilonychia, angular cheilitis, glossitis
B12 deficiency	Glossitis; ↓vibration/proprioception, Romberg+
Haemolysis	Jaundice, splenomegaly
Marrow failure	Petechiae, bone tenderness, organomegaly

Initial investigations

Test	Role
FBC	Confirm Hb; MCV, MCH, MCHC, RDW; check WCC/platelets
Blood film	Crucial — review morphology, see below
Reticulocytes	Assess marrow response
Iron studies	Ferritin (stores; <15–30 = IDA — acute phase reactant); serum iron, TIBC, saturation
B12 + folate	Test concurrently
Consider	LFTs, UEC/eGFR, TSH, CRP/ESR, haemolytic screen (LDH, bilirubin, haptoglobin, DAT/Coombs)

★ **EXAM TIP:** Blood film checklist — size/colour (hypochromia, polychromasia); shape (target cells, spherocytes, **schistocytes** = **urgent**, sickle cells, teardrop/pencil cells, bite cells); inclusions (basophilic stippling, Howell-Jolly bodies); WBC/platelets (hypersegmented neutrophils = B12/folate deficiency; blasts = leukaemia)

GP role & differential diagnoses to keep in mind

- **GP role** — detect (opportunistic + symptomatic FBC), investigate systematically (MCV + reticulocytes first), manage common causes (IDA, ACD, B12/folate), refer complex/red-flag cases
- **Common differentials to actively work through** — IDA; ACD; thalassaemia trait; B12 deficiency (esp. pernicious anaemia); folate deficiency; haemolytic anaemia (AIHA, G6PD); anaemia of CKD; MDS/bone marrow failure — each has a distinct iron-study/reticulocyte/film signature (see following topics)

⚠ **RED FLAGS:** Hb <70–80g/L or rapid drop; haemodynamic instability (chest pain, SOB at rest, pre-syncope); active/significant bleeding
 Pancytopenia; blasts or schistocytes on film — leukaemia/MAHA emergency
 IDA + weight loss/bowel change — suspect malignancy; neurological symptoms + macrocytosis — urgent B12 treatment
 Failure to respond to appropriate therapy (e.g. oral iron for IDA); unexplained splenomegaly/lymphadenopathy

Transfusion & pregnancy considerations

Setting	Practical point
Transfusion threshold	Restrictive strategy generally preferred — trigger Hb ~70g/L in stable patients (~80g/L if cardiac disease); guided by symptoms, not number alone
Pregnancy	Iron studies + FBC at booking and 28 weeks; oral iron first-line for IDA; IV iron if intolerant or diagnosed after 34 weeks; anaemia thresholds lower than non-pregnant (trimester-specific)
Elderly	Anaemia is never 'normal ageing' — always investigate; multifactorial causes common (nutritional + CKD + chronic disease)

OPHTHALMOLOGY

SYSTEMATIC APPROACH TO THE RED EYE

KEY POINTS: A structured 3-5 minute assessment separates benign red eye from sight-threatening emergency. Always measure visual acuity (VA) FIRST. Ciliary flush (redness worst at the limbus) = intraocular inflammation until proven otherwise.

History Question	Significance
Visual acuity change?	ANY reduction = red flag → ophthalmology; measure VA in every red eye
Pain vs discomfort?	True aching/throbbing pain = uveitis, keratitis, glaucoma, scleritis. Gritty discomfort = conjunctivitis, dry eye, foreign body
Photophobia?	Significant = uveitis, keratitis, corneal ulcer, AACG
Discharge type?	Watery = viral/allergic; purulent = bacterial; stringy mucoid = allergic; none = uveitis/episcleritis/scleritis/dry eye
Itch?	Hallmark of allergic conjunctivitis; minimal in viral/bacterial
Laterality?	Unilateral favours bacterial, HSK, uveitis, keratitis, glaucoma. Bilateral favours allergic or viral
Contact lens wear?	Higher infection risk (Acanthamoeba, Pseudomonas); cease lenses immediately; low threshold for urgent referral
Trauma/chemical exposure?	Urgent assessment and referral
Systemic disease?	Ankylosing spondylitis, reactive arthritis, IBD → uveitis. Zoster dermatome → HZO. Atopy → allergic conjunctivitis

Examination sequence

- 1. Visual acuity** — Snellen chart, pinhole if uncorrected; measure FIRST, every red eye
- 2. Pattern of redness** — diffuse conjunctival injection = conjunctivitis; ciliary flush (worst at limbus, fades peripherally) = uveitis/keratitis/AACG; sectoral/nodular = episcleritis/scleritis; flat homogeneous = subconjunctival haemorrhage
- 3. Discharge** — watery (viral/allergic); purulent (bacterial); mucoid/stringy (allergic); none (uveitis/dry eye)
- 4. Cornea** — inspect under good light for opacity/infiltrate/ulcer; fluorescein stain: linear = abrasion, dendritic = HSK, punctate = dry eye/UV keratitis
- 5. Pupil** — irregular = posterior synechiae (uveitis); fixed mid-dilated non-reactive = AACG; always compare with fellow eye
- 6. Eyelid and adnexa** — tender medial canthal swelling = dacryocystitis; evert upper lid for follicles (viral) vs papillae (allergic)
- 7. Pre-auricular node** — tender = viral conjunctivitis (adenovirus); absent in bacterial and allergic

Condition	Discharge/Itch	Pain/VA	Key Feature
Allergic conjunctivitis	Watery, INTENSE itch	Minimal pain, normal VA	Bilateral, chemosis, atopy
Viral conjunctivitis	Watery, mild itch	Gritty only, normal VA	Follicles, pre-auricular node, starts unilateral
Bacterial conjunctivitis	Purulent, mild itch	Gritty only, normal VA	Lids stuck on waking, no node
Corneal abrasion/FB	None, mild	Severe FB sensation, photophobia	Trauma history, fluorescein positive
HSV keratitis	Watery, none	Pain+photophobia, reduced VA if central	Dendritic ulcer, unilateral
Anterior uveitis	None	Aching pain+photophobia, VA may reduce	Ciliary flush, small/irregular pupil
AACG	None	SEVERE pain, N&V, halos, reduced VA	Fixed mid-dilated pupil, rock-hard globe

GP eye examination — essential skills

Test	Technique	When
Visual acuity	Snellen at 6m (LogMAR/picture chart in children); each eye separately, with spectacles or pinhole	Every patient with a visual complaint — always before referring
Red reflex	Direct ophthalmoscope, dim room, ~50cm; normal = bright orange-red equally in both eyes	All neonates/infants/children; any adult with suspected cataract
Pupil assessment	Direct and consensual light reflex; swinging flashlight for RAPD; shape and equality	Every eye complaint; screen for neurological disease
Fundoscopy	Direct ophthalmoscope; assess disc, macula, vessels; dilate with tropicamide 1% if needed	Suspected glaucoma, diabetic retinopathy, papilloedema, macular disease

OCCUPATIONAL HEALTH

OCCUPATIONAL HISTORY — CORNERSTONE OF ASSESSMENT

KEY POINTS: Take an occupational history at first encounter with every working-age patient and update it whenever a presentation could be work-related — ask about work before attributing symptoms to non-occupational causes.

Key question — 'Do symptoms get better on weekends/leave and worse back at work?' A positive temporal relationship is the single most useful clue to an occupational cause — document the answer explicitly.

Domain	What to elicit
Job/tasks	Current and all past job titles/employers — some diseases have long latency (mesothelioma up to 40+ yrs after asbestos). Specific tasks, hours, shift pattern
Physical	Noise (machinery, jackhammers, concerts — NIHL); vibration (hand-arm — white finger; whole-body — spine); ionising radiation (healthcare/nuclear); heat/cold extremes (heat illness, Raynaud's)
Chemical	Dusts (silica, coal, asbestos, hard metal, organic); solvents (toluene, benzene, chlorinated hydrocarbons — hepato-/neurotoxic, carcinogenic); metals (lead, mercury, arsenic, chromium); pesticides; isocyanates (occupational asthma)
Biological	Zoonoses (Q fever — abattoir/farming/vet; leptospirosis — water/soil; brucellosis); bloodborne viruses (healthcare, needlestick); mould/organic dust (Farmer's lung, ODS)
Ergonomic	Repetitive movement, sustained posture, manual handling (lifting/pushing/pulling), workstation design, keyboard/mouse use
Psychosocial	Work demands, control over pace/priorities, supervisor/colleague support, bullying, harassment, job insecurity, violence risk — leading cause of work-related mental-health conditions
PPE	What is provided vs what is actually used; correct/consistent use; training; fit-testing for respiratory protection
Workplace	Ventilation/air quality, engineering controls (dust extraction, enclosed cabs), health and safety protocols, employer surveillance programs

Domain	What to elicit
Non-occupational	Hobbies (woodwork, car painting — solvents; shooting — noise; gardening — zoonoses); smoking (synergistic with many occupational lung diseases); home environment

Investigation	Use
Spirometry	Screening/monitoring for occupational lung disease; compare to baseline and predicted values
Audiometry	Pure-tone testing in noise-exposed workers; NIHL shows characteristic 4 kHz notch — distinguish from presbycusis (bilateral high-frequency slope)
HRCT chest	Superior to CXR for early pneumoconiosis, interstitial disease, pleural plaques, mesothelioma
Allergen testing	Skin prick/specific IgE (RAST) for suspected occupational allergy; dermatologist patch testing for allergic contact dermatitis
Biological monitoring	Blood lead, urinary heavy metals, cholinesterase activity (organophosphate exposure) — usually via occupational physician or public health

- **Specialist referral —** occupational physician (complex disease, legal/compensation questions); respiratory physician (occupational asthma, pneumoconiosis); dermatologist (ACD, patch testing); audiologist (NIHL)

▲ RED FLAGS: A lifetime occupational history is mandatory, not just current employment — asbestos-related disease, mesothelioma and some occupational cancers present 20–40+ years after exposure ends.

HIGH-RISK OCCUPATIONS I — TRADES, HEALTH, AND LAND-BASED WORK

KEY POINTS: Occupation-specific risk profiles let the GP target screening and counselling — ask job title and industry at every new-patient registration and update it whenever it changes.

Occupation	Key risks	Critical GP pearl
Miners — coal/silica/ asbestos	Coal workers' pneumoconiosis (CWP), silicosis, asbestosis, mesothelioma, lung cancer, NIHL, MSDs	Coal → CWP (progressive massive fibrosis in severe disease). Silica → silicosis + markedly increased TB risk — screen. Asbestos → asbestosis, benign pleural plaques, mesothelioma, lung cancer (synergistic with smoking — cessation priority). HRCT more sensitive than CXR; latency 20–40+ yrs
Construction workers	MSDs (most prevalent occupational condition), NIHL, skin cancer, silicosis (cutting/drilling concrete/stone), asbestos (pre-2004 buildings), cement dermatitis	Manage MSDs with a biopsychosocial approach and screen for yellow flags. Hearing protection + audiometry. Aggressive sun safety. Engineered-stone silicosis is an emerging epidemic — ask specifically about benchtop fabrication. Wet cement causes both irritant and chromate-allergic dermatitis