Table of Contents

How to Use This Resource: Your Guide to GP Fellowship Success 1
The Role of the General Practitioner in Australia 2
CARDIOVASCULAR DISEASES 6
Cardiomyopathy 7
Coronary Artery Disease (CAD) & ischemic heart disease (IHD) 12
Dyslipidaemia 16
Hypertension (HTN) 19
Pericardial Disease 23
Endocarditis & Myocarditis 26
Valvular Heart Disease 31
Vascular Diseases 36
Thromboembolic disease (DVT and PE) 41
Peripheral Arterial Disease and Lymphedema 45
Syncope 51
Cardiovascular disease risk assessment 55
Heart Failure 59
Atrial Fibrillation & Atrial Flutter 63
STEMI and Carotid Stenosis 66
SKIN DISORDERS 71
Allergic and Immune-Mediated Skin Disorders 72
Infectious Disease Manifestations of the Skin 78
Miscellaneous inflammatory, pigmentary, and paraneoplastic disorders 85
Neoplasms of the Skin 91
ENDOCRINE AND METABOLIC HEALTH 97
Metabolic Syndrome and Diabetes Mellitus (DM) 98
Thyroid Disorders 102
Metabolic Bone Diseases: Osteoporosis, Paget's Disease, Hyperparathyroidism 107
Pituitary and Hypothalamic Disorders 111
Adrenal Gland Disorders 117
HAEMATOLOGICAL DISORDERS 123
Coagulation Disorders 124
Inherited and Acquired Disorders of Haemostasis 127

Antiphospholipid Syndrome (APS) 132
Platelet Disorders 136
Red Blood Cell Disorders 140
White Blood Cell Disorders 144
Leukemias 147
Lymphomas 150
Plasma Cell Disorders 153
Multisystem Hematology 156
GASTROINTESTINAL DISORDERS 159
Oral and Salivary Gland Disease 160
Oesophageal Disease 164
Gastric Disorders 168
Gastrointestinal Bleeding 172
Disorders of the Small Bowel 175
Malabsorption and Maldigestion 178
Acute Abdomen and Related Conditions 181
Colorectal Cancer (CRC) 184
Inflammatory Bowel Disease (IBD) in Primary Care 187
Common Abdominal Wall Hernias 189
Biliary Tract Disease 191
Interpreting Liver Function Tests and Managing Common Liver Diseases 194
Cirrhosis, Complications, and Related Liver/Biliary Diseases 196
Complications of Chronic Liver Disease and Related Hepatobiliary Conditions 199
Hepatocellular Carcinoma and Hereditary Haemochromatosis 201
Liver Disorders: Wilson's Disease, Benign Lesions, Infections & Transplantation Principles 203
Pancreatic Disorders: Inflammation, Cysts, and Neoplasms 205
MUSCULOSKELETAL DISORDERS 208
Common Orthopaedic Injuries: Assessment and Management Principles 209
Peripheral Nerve Injuries and Complex Regional Pain Syndrome 212
Septic Arthritis: Assessment and Management 214
Common Bone and Joint Infections and Inflammatory Conditions 217
Seronegative Spondyloarthropathy 220
Inflammatory Myopathies: Polymyositis and Dermatomyositis 222

Temporomandibular Disorders and Myofascial Pain Syndrome 224
Systemic Sclerosis (Scleroderma) 226
Systemic Autoimmune and Inflammatory Disorders 229
Selected Musculoskeletal and Peripheral Nerve Conditions 232
Selected Joint and Periarticular Conditions 235
Low Back Pain (LBP) 238
NEUROLOGICAL DISORDERS 240
Neurological Localization and Common Syndromes 241
Headache: Assessment and Management 244
Transient Ischemic Attack (TIA) and Stroke 247
Assessment and Initial Management of Coma 251
Aphasia: Assessment and Types in Primary Care 254
Seizures and Epilepsy: Assessment and Management in Primary Care 257
Vertigo: Assessment and Management of Common Causes 260
Central Nervous System Infections: Meningitis, Encephalitis, and Related Conditions 263
Disorders of the Neuromuscular Junction 266
Demyelinating disorders 269
Dementia (Major Neurocognitive Disorder) in Primary Care 272
Neurodegenerative Movement Disorders 275
Selected Neurodegenerative and Movement Disorders 278
Intracranial Neoplasms: Recognition and Referral in Primary Care 281
Neurocutaneous Syndromes and Related Genetic Disorders 284
Selected Neurological Syndromes: 286
EAR, NOSE AND THROAT DISORDERS 289
Otitis Media in Primary Care: AOM, OME, and CSOM 290
Otitis Externa (Including Malignant Otitis Externa) 293
Hearing Loss: Assessment and Common Causes 295
Infections of the Head, Neck, and Sinuses 298
Rhinitis in Primary Care: Allergic and Non-Allergic 300
Rhinitis in Primary Care: Allergic and Non-Allergic 302
Common ENT Conditions 304
Laryngeal Disorders 306
Benign and Malignant Laryngeal Lesions 308

WOMENS AND REPRODUCTIVE HEALTH 310
Obstetric Fundamentals: Terminology and Physiological Adaptations in Pregnancy.....311
Antenatal Care and Prenatal Screening/Diagnostic Testing..... 313
Teratogenic Exposures in Pregnancy 316
Congenital and Perinatal Infections 319
Early Pregnancy Complications: Miscarriage and Hyperemesis Gravidarum..... 321
Diabetes in Pregnancy..... 323
Hypertensive Disease in Pregnancy..... 325
Common Complications of Pregnancy: Bleeding, Growth Issues, and Multiples..... 327
Normal Labour, Delivery, and Initial Postpartum Care Principles 329
Intrapartum and Delivery Complications..... 331
The Puerperium..... 333
Lactation and Breastfeeding Management..... 336
Amenorrhea and Dysmenorrhea 338
Secondary amenorrhea..... 340
Abnormal Uterine Bleeding..... 342
Contraception and Emergency Contraception 344
Disorders of Adrenal & Ovarian Function: CAH and PCOS..... 347
Infertility 349
Menopause..... 351
Common Benign Gynaecological Conditions..... 354
Gynaecological Emergencies and Infections 357
Benign Breast Disorders 360
Breast Cancer..... 363
Gynaecologic Neoplasms..... 366
Urological Gynaecology and Pelvic Organ Prolapse..... 368
Female Sexual Interest/Arousal Disorder (FSIAD)..... 370
Cervical Screening in Australia..... 372
Mammography Screening for Breast Cancer..... 374
Preconception Screening and Counselling..... 376
Female Sexual Interest/Arousal Disorder (FSIAD) in General Practice..... 378
CHILD AND YOUTH HEALTH 380
Well- Child Care 381

Selected Genetic Disorders: Cystic Fibrosis and Turner Syndrome 384
Neonatal Assessment and Common Problems- I 386
Neonatal Assessment and Common Problems- II 389
Congenital Heart Disease 392
Autism Spectrum Disorder & Child Development 394
Paediatric Gastrointestinal Disease 396
Pediatric Urology 399
Inguinal Hernia 401
Paediatric Immunology 403
Kawasaki Disease 405
Pediatric Infectious Disease- I 408
Paediatric Infectious Diseases- II 412
Paediatric Neurology 415
Paediatric Haematology 418
Childhood Leukemia 420
Paediatric Oncology 422
Paediatric Musculoskeletal Disorders 424
Child Abuse 426
Perianal Dermatitis 429
Childhood Immunisation, School-Entry Health Checks & Immunisation Status 431
OPTHALMOLOGY 433
Common Eye Disorders 434
Refractive Errors 436
Age-Related Eye Disorders 439
Diabetic Retinopathy, Conjunctivitis and Acute Red Eye Presentations 442
RESPIRATORY DISORDERS 445
Spirometry in Australian General Practice 446
Obstructive Lung Disease 449
Chronic Obstructive Pulmonary Disease COPD 452
Obstructive Lung Diseases: Bronchiectasis 454
Restrictive lung diseases 456
Cryptogenic organizing pneumonia 459
Eosinophilic pulmonary syndromes 461

Acute Respiratory Failure & ARDS 463
Mechanical Ventilation 466
Coronaviruses and COVID-19 468
Pulmonary Thromboembolism 470
Solitary Pulmonary Nodules & Lung Cancer 472
Respiratory Tract Infections 475
Histoplasmosis, Coccidioidomycosis, and Blastomycosis 478
Mycobacterial Infections 480
Pneumocystis jirovecii pneumonia 483
Anthrax 485
Acute pharyngitis 487
Haemoptysis 490
Pleural Disease 493
Pneumothorax 496
Pulmonary sleep disorders 499
Obesity Hypoventilation Syndrome 502
MENTAL HEALTH 505
Childhood and Adolescent Disorders 506
Intellectual Developmental Disorder/Intellectual Disability 509
Schizophrenia 512
Dissociative Disorders 515
Generalized Anxiety Disorder 518
Obsessive-Compulsive Disorder (OCD) 521
Trauma-Related, Neurocognitive, and Delirium Disorders 524
Major Depressive Disorder 526
Persistent Depressive Disorder (Dysthymia) 529
Personality Disorders 532
Defense Mechanisms 535
Substance Use Disorders 537
Eating Disorders 540
Sexual Changes with Aging 543
Somatic Symptom, Trauma-Related Presentations, and Risk Assessment 546
Suicide Risk Assessment and Management 549

RENAL DISORDERS 552
Hypernatremia And Hyponatremia 553
Hypercalcemia & Hypocalcaemia 556
Hypomagnesemia 560
Acid-Base Disorders 563
Renal Tubular Acidosis (RTA) 566
Chronic Kidney Disease (CKD) 569
Glomerular disease 572
Nephrotic Syndrome 575
Polycystic Kidney Disease 578
Urinary Incontinence 580
Interstitial Cystitis 583
Common Urological Malignancies 585
Genitourinary Infections 588
Uncomplicated UTI / Lower UTI / Acute Simple Cystitis 591
Prostatitis 594
Sexually Transmitted Diseases 598
Urinary Stone Disease (Nephrolithiasis) 602
MENS HEALTH 605
Scrotal Pain and Swelling 606
Erectile Dysfunction 608
Benign Prostatic Hyperplasia 611
Depression and Anxiety, Substance Use and Reluctance to Seek Help 613
Testosterone Deficiency, Gynecomastia, Male Infertility, Preconception Counselling 616
Intimate Partner Violence, Aggression in Male Patients 622
Male Sexually Transmitted Infections 625
Sexual Identity & Health 628
Male Infertility & Preconception Care 631
Sexual and Reproductive Health 634
Gonococcal Infections in Primary Care 637
PREVENTION IN PRIMARY CARE 640
Smoking Cessation Advice 641
Obesity Screening in Adults 643

Geriatric Assessment in General Practice 644
Nutritional Deficiencies in the Elderly 648
Youth and Adolescent Health Assessment in Primary Care 651
Refugee and New Migrant Health 653
Health Screening for Aboriginal and Torres Strait Islander Peoples in General Practice 656
Responding to Domestic and Family Violence 659
Osteoporosis Screening in Older Adults 662
OCCUPATIONAL AND LIFESTYLE FACTORS 665
Erectile Dysfunction in Shift Workers 666
Mental Health in FIFO and High-Stress Occupations 669
Skin Cancer Screening and Prevention in Outdoor Workers 672
Lifestyle Modification Counselling 675
Assessing Fitness to Drive: Medical Conditions and GP Responsibilities 678
Occupational Health in General Practice: Common Conditions and Assessment Principles 681
Envenomation and Trauma from Hazardous Australian Fauna 684
Managing Terrestrial Australian Fauna Encounters 687
The General Practitioner's Role in the Australian Workers' Compensation System 690
AKT & KFP EXAM TECHNIQUES 693
KFP and AKT Exam Technique Advice 694

How to Use This Resource: Your Guide to GP Fellowship Success

This guide supports your Australian and NZ (RACGP) General Practice fellowship journey. Think of it as a concise, GP-focused UpToDate, providing high-yield information clearly for real-world practice.
Organized by clinical area, each chapter offers summaries of essential knowledge, diagnosis, and management relevant to ANZ and current guidelines.
We update this resource every few months. Email us with suggestions for additions, updates, or corrections.

What are Exam Pearls? Throughout chapters, find Exam Pearls – high-yield facts and crucial clinical insights frequently evaluated in exams. These key concepts are designed for easy learning and recall for exams and daily practice. Make notes or revise on the go.

Navigating This Resource:

- Start with chapters matching your learning needs. Follow sequentially or jump to specific topics.
- Read each chapter thoroughly, noting key concepts and recommendations. Content is concise yet comprehensive.
- Focus intently on "Exam Pearls" for exam preparation. Consider highlighting or creating flashcards.
- Integrate this resource with your clinical experiences to reinforce learning and apply evidence-based practices.
- Utilize this resource for exam revision. The structure and "Exam Pearls" are ideal for consolidating knowledge.

Remember, this guide supplements your formal training and supervision. Always refer to official guidelines and seek supervisor advice for patient management.
By using this resource effectively, you will build a strong knowledge base and clinical reasoning for your GP fellowship exams and providing quality patient care in Australian general practice.

The Role of the General Practitioner in Australia

Introduction

Australian GPs are central to the nation's primary healthcare. They provide comprehensive, person-centred, continuous care across all life stages. Operating within Medicare, GPs are the first patient contact, managing diverse conditions, coordinating care, advocating, and promoting health/preventing disease. The GP-patient relationship is key to effective primary care. Understanding this role's breadth is vital for effective practice and fellowship pathways.

1. Clinical Care and Diagnosis

GP's core function is direct clinical care, including:

Managing Undifferentiated Illness: Skilled in history, examination, and differential diagnoses for non-specific symptoms.

Diagnosing and Managing Acute Conditions: Common infections, minor injuries, chronic condition exacerbations, and identifying urgent cases.

Managing Chronic and Complex Conditions: Central to long-term management of diseases like diabetes, cardiovascular disease, asthma, COPD, arthritis, chronic kidney disease, and mental health. Includes monitoring, medication, lifestyle advice, and multidisciplinary care using Medicare's Chronic Disease Management (CDM) items. Managing multimorbidity is key.

Investigations and Prescribing: Ordering/interpreting tests (blood, pathology, imaging) for diagnosis and management. Prescribing safe, rational, cost-effective medications adhering to the Pharmaceutical Benefits Scheme (PBS).

Minor Procedures: Performing appropriate minor surgical and therapeutic procedures like skin lesion assessment/excision, biopsies, suturing, wound care, abscess drainage, joint injections, cryotherapy, and long-acting reversible contraception insertion/removal.

2. Continuity of Care

Continuity of care is ongoing, relationship-based care by a GP over time, a defining feature of Australian general practice.

Longitudinal Relationship: Building trust through repeated consultations allows deep understanding of patient history, circumstances, values, and beliefs.

Benefits: Linked to improved patient satisfaction, adherence, health outcomes (especially chronic disease), reduced hospitalisation, and lower costs.

Monitoring Health Trajectories: GPs monitor physical, mental, and social changes, enabling early detection and proactive interventions.

Care Transitions: GPs ensure smooth transitions, following up hospital discharge summaries/specialist letters, reconciling medications, and integrating specialist advice.

3. Whole-of-Life Care and Prevention

GPs provide care across the lifespan, from prenatal to end-of-life, with a strong focus on preventive health.

Preventive Activities:

Immunisations: Delivering vaccines under the National Immunisation Program (NIP), seasonal influenza, COVID-19, and travel vaccines. Maintaining accurate records (Australian Immunisation Register - AIR).

Collaboration: Working with ambulance services, retrieval teams (e.g., RFDS), and local hospitals during emergencies.

6. Special Roles in Rural and Remote Areas

Rural and remote GPs often have a broader scope than urban counterparts.

Expanded Clinical Scope: Frequently possess extra skills in emergency medicine, obstetrics (including deliveries), anaesthetics, minor surgery, and inpatient care.

Hospital Role: Often hold Visiting Medical Officer (VMO) or Senior Medical Officer (SMO) roles in local rural hospitals, providing inpatient care, emergency coverage, and procedures.

Aboriginal and Torres Strait Islander Health: Providing primary care to Indigenous communities, requiring cultural safety, understanding specific health priorities (e.g., chronic disease, rheumatic heart disease), Closing the Gap initiatives, and collaboration with Aboriginal Community Controlled Health Organisations (ACCHOs) and Aboriginal Health Workers/Practitioners. Utilising relevant MBS items like the 715 Health Assessment.

Challenges & Rewards: Navigating isolation and resource limits while enjoying community integration and diverse practice.

7. Administrative and Professional Duties

Beyond patient care, GPs have significant professional responsibilities.

Medical Records: Maintaining accurate, legible, contemporaneous, and comprehensive records is legal and ethical. Proficiency with practice software and understanding health informatics (including My Health Record) are essential.

Ethical and Legal Practice: Adhering to the Medical Board of Australia's code of conduct. Understanding informed consent, confidentiality, privacy, mandatory reporting, and professional boundaries.

Medicare Compliance: Understanding the Medicare Benefits Schedule (MBS), billing accurately, meeting item descriptors, and maintaining records. Understanding Practice Incentives Program (PIP) requirements.

Continuing Professional Development (CPD): Engaging in ongoing learning to maintain skills, fulfilling Medical Board and college (RACGP or ACRRM) requirements.

Teaching and Supervision: Many GPs teach medical students and supervise GP registrars.

Practice Management and Quality Improvement: Participating in meetings, audits, developing policies, and engaging in quality improvement activities (e.g., PIP QI data submission).

8. Alignment with RACGP/ACRRM Core Domains

GP roles align with RACGP and ACRRM core competency domains for training and assessment:

Communication and the Patient-Doctor Relationship: Empathy, listening, shared decision-making, clear information, cultural competence.

Applied Professional Knowledge and Skills: Integrating knowledge, procedural skills, and evidence-based practice.

Population Health and the Context of General Practice: Understanding epidemiology, prevention, screening, health promotion, social determinants, and the healthcare system.

Professional and Ethical Role: Demonstrating integrity, ethics, self-reflection, managing uncertainty, lifelong learning, fitness to practice.

Organisational and Legal Dimensions: Understanding practice management, Medicare, record-keeping, teamwork, quality improvement, and medico-legal responsibilities.

Demonstrating competence across these domains is fundamental to fellowship.

CARDIOVASCULAR DISEASES

Cardiomyopathy

Cardiomyopathies are diverse heart muscle diseases causing structural and functional abnormalities, distinct from IHD, hypertension, or valvular issues, though these can coexist. Crucial for GPs, they manifest as heart failure (HF), arrhythmias, or sudden cardiac death (SCD), especially in younger individuals.

Classification is based on morphology and physiology: Dilated (DCM), Hypertrophic (HCM), and Restrictive (RCM), plus Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC) and Takotsubo Cardiomyopathy. Pathology can be cardiac (primary/genetic/idiopathic) or systemic (secondary). GP role: recognition, initial investigation, timely referral, HF/comorbidity co-management, and family history for genetic implications.

Types of Cardiomyopathies

Dilated Cardiomyopathy (DCM)

Diagnosis Essentials

Presentation usually with Heart Failure (HF) symptoms/signs.

Echocardiogram confirms: Left ventricular (LV) dilation and impaired systolic function (LVEF ≤ 40%), excluding other causes like severe hypertension or valve disease.LV walls may be thin or normal.

Right ventricular (RV) dilation/dysfunction may worsen prognosis.

General Considerations

Most common cardiomyopathy, major HFrEF cause leading to transplant. Unlike IHD-related HFrEF, DCM's primary driver isn't ischaemia. Prognosis varies, often poor with symptom onset (historically ~50% 5-year mortality, improving with modern HFrEF therapies).

Aetiology

Often 'idiopathic', but underlying causes include:

Genetic/Familial: 20-50% of idiopathic DCM, often autosomal dominant. Check family history (HF, DCM, arrhythmias, SCD). Specialist-coordinated genetic testing.

Myocarditis (Viral/Inflammatory): Post-viral or autoimmune can cause DCM.

Toxic:

Alcohol Excess: Common, reversible with dose-dependent effect. Quantify intake.

Chemotherapy: E.g., Anthracyclines, Trastuzumab. Monitor during/after.

Illicit Drugs: Cocaine, amphetamines.

Tachycardia-Induced Cardiomyopathy: Persistent rapid heart rates cause reversible LV dysfunction. Rate/rhythm control is key.

Peripartum Cardiomyopathy: Late pregnancy or early postpartum, diagnosis of exclusion.

Metabolic/Endocrine: Thyroid disease, Acromegaly, Phaeochromocytoma (rare). Diabetes is a major risk factor/comorbidity.

Nutritional Deficiencies: Severe thiamine, selenium, carnitine (rare in Australia).

Infiltrative Diseases: Haemochromatosis, Sarcoidosis, Amyloidosis (can present as DCM later).

Clinical Findings

Symptoms: Insidious HF onset: dyspnoea on exertion, fatigue, orthopnoea, PND, peripheral oedema. Acute presentation possible.

Signs: Tachycardia, late hypotension, elevated JVP, displaced/diffuse apical impulse, S3 gallop, functional mitral/tricuspid regurgitation murmurs, pulmonary crackles, peripheral oedema, ascites.

Investigations (GP Role)

History: Symptoms, NYHA class, risk factors, 3-generation family history, alcohol/drug use, medications.

Examination: Assess for HF signs and potential causes.

ECG: Often abnormal, non-specific. May show sinus tachycardia, LBBB (worse prognosis), LVH pattern, AF, ventricular arrhythmias, poor R wave progression.

CXR: Cardiomegaly, pulmonary congestion, pleural effusions.

Bloods: BNP/NT-proBNP (elevated), FBC, UEC, LFTs, TFTs, HbA1c, Iron Studies. Consider Troponin (acute/suspected myocarditis), specific serology/autoimmune screen if indicated.

Echocardiogram: Key diagnostic test. Confirms LV dilation, LVEF ≤ 40%, assesses RV/diastolic/valve function, pulmonary pressures. GP referral essential.

Specialist Investigations: Cardiac MRI, Coronary Angiography, Genetic testing, Endomyocardial biopsy (rare, specific cases).

Management in General Practice

Follows HFrEF guidelines, address specific cause.

Referral: Cardiology for confirmation, cause investigation, specialist advice, advanced therapies. GP-specialist co-management typical.

Lifestyle Modification: HF guidelines (SNAP).

Treat Reversible Causes: Alcohol abstinence, thyroid treatment, rate/rhythm control for tachy-cardiomyopathy.

Pharmacotherapy (HFrEF 'Quad Therapy'): Initiate, titrate, monitor per HF guidelines:

- ARNI/ACEi/ARB
- Beta-Blocker
- MRA
- SGLT2 Inhibitor

Diuretics: Loop diuretics for congestion.

Iron Deficiency: IV Iron if criteria met.

Anticoagulation: For concurrent AF or LV thrombus. Not routine for DCM/HFrEF in sinus rhythm.

Device Therapy Awareness: Refer if LVEF ≤ 35% despite ≥3 months optimal medical therapy (OMT) for ICD or if LBBB + QRS ≥ 130-150ms + symptoms for CRT.

Hypertrophic Cardiomyopathy (HCM)

Diagnosis Essentials

Genetic, unexplained Left Ventricular Hypertrophy (LVH), typically asymmetric.

Primary issue: impaired diastolic relaxation/filling.

May have Dynamic Left Ventricular Outflow Tract (LVOT) Obstruction (~70%).

Leading SCD cause in young, especially athletes.

Clinical Findings

Presentation: Variable, often asymptomatic. Symptoms: Dyspnoea on exertion, Angina, Palpitations, Presyncope/Syncope (exertional - Red Flag!), Fatigue. SCD may be first sign.

Signs: Harsh systolic ejection murmur (lower left sternal border/apex) varying with preload. May have S4 gallop, prominent apical impulse, bisferiens carotid pulse.

Diagnosis

Family History: Crucial – inquire about HCM, unexplained syncope, or SCD at young age.

ECG: Often abnormal (~90%).

Echocardiogram: Key test. Confirms unexplained LVH, assesses LVOT obstruction, SAM, diastolic dysfunction, LA size.

Specialist Investigations: Cardiac MRI, Holter, Exercise stress test, Genetic testing.

Management in General Practice

High Suspicion: Young athletes with syncope or relevant family history.

Referral: ALL suspected/confirmed HCM patients to Cardiology, ideally with HCM expertise.

Initial Advice (Pending Specialist Review): Avoid high-intensity sports, maintain hydration, avoid certain drugs.

Presentation: Acute chest pain/dyspnoea after clear stressor.

ECG: Often anterior ST elevation or deep T wave inversions. QT prolongation possible.

Biomarkers: Troponin elevated, often less than expected. BNP/NT-proBNP usually high.

Echocardiogram: Characteristic regional wall motion abnormality ('apical ballooning').

Coronary Angiography (+ /- Ventriculography): Essential to rule out obstructive CAD. Confirms typical systolic LV shape ('octopus pot').

Management

Acute Phase: Manage as suspected ACS initially. Supportive care: treat HF, manage arrhythmias. Anticoagulation if severe LV dysfunction/apical thrombus suspected.

Long-Term: Usually continue ACEi/ARB and/or Beta-blocker until LV function recovers. Follow-up echo needed. Address stressors. Low recurrence.

Selected Secondary Cardiomyopathies

1. Cardiac Amyloidosis

Aetiology: Infiltrative, amyloid fibril deposition.

Symptoms: Right HF, syncope, thromboembolism, extracardiac signs.

Diagnosis: Echo, ECG (low voltage), biopsy. Cardiac MRI/bone scan for ATTR.

Treatment: Diuretics, pacemaker, underlying amyloidosis treatment. Transplant if needed.

2. Cardiac Hemochromatosis

Aetiology: Intracellular iron accumulation (hereditary), restrictive progressing to dilated cardiomyopathy.

Symptoms: Diastolic dysfunction, arrhythmias, bronze skin, arthritis, diabetes, cirrhosis.

Diagnosis: Iron markers, liver tests, genetic testing, echo, cardiac MRI for iron quantification.

Treatment: Iron chelation, phlebotomy, associated disease treatment.

3. Cardiac Sarcoidosis

Aetiology: Inflammatory, noncaseating granulomas in myocardium, often systemic.

Symptoms: HF, arrhythmias, SCD, systemic.

Diagnosis: Imaging (PET-CT, MRI), biopsy, exclude other causes.

Treatment: Standard HF therapy, arrhythmia management, ICD for SCD prevention, immunosuppressants, regular cardiac monitoring.

4. Endocrine-Related Cardiomyopathies

Acromegalic Cardiomyopathy: Growth hormone excess. Treatment: GH control, optimal HF management.

Diabetic Cardiomyopathy: Structural/functional abnormalities. Treatment: Optimize HF and diabetes management.

5. Chemotherapy-Related Cardiomyopathy

Aetiology: Cardiotoxic agents.

Symptoms: HF signs with reduced LVEF.

Diagnosis: Reduced LVEF on echo, serial monitoring.

Treatment: Stop chemotherapy if severe, standard HF therapy, consider secondary prevention.

6. Other Cardiomyopathies

Myocarditis: Myocardial inflammation. Treatment: HF management, pacing, transplant in severe cases.

Peripartum Cardiomyopathy: Late pregnancy or early post-partum. Treatment: HF management, avoid teratogens, echo monitoring, avoid future pregnancies with persistent low EF.

Coronary Artery Disease (CAD) & ischemic heart disease (IHD)

Ischemic Heart Disease (IHD), predominantly from atherosclerotic coronary artery disease (CAD), is Australia's leading cause of death and morbidity, incurring significant healthcare costs. Atherosclerosis narrows coronary arteries, reducing blood flow (ischemia) and oxygen to the heart (myocardium). IHD ranges from asymptomatic to stable exertional symptoms (Chronic Coronary Syndromes [CCS]) and life-threatening Acute Coronary Syndromes (ACS) like unstable angina, NSTEMI, and STEMI. Australian GPs are vital in IHD management: primary prevention, CCS diagnosis and management, ACS recognition and initial care, and long-term secondary prevention/rehabilitation.

Pathophysiology of Atherosclerosis and Ischaemia

Atherosclerosis is a slow inflammatory process in medium/large arteries, initiated by endothelial dysfunction (often risk factor-driven). LDL-C infiltration triggers inflammation, macrophage (foam cell) formation, and smooth muscle proliferation, forming plaques.

- Stable Plaques: Thick fibrous cap, lipid core. Cause fixed stenosis, limiting blood flow during exertion, leading to predictable ischemia/CCS.
- Vulnerable Plaques: Thin cap, large lipid core, inflammation. Prone to rupture/erosion, exposing thrombogenic material, causing rapid thrombus formation and ACS.

Myocardial Response to Ischaemia

- Myocardial Stunning: Reversible contractile dysfunction post-reperfusion after prolonged ischemia. Function recovers over hours/days.
- Myocardial Hibernation: Chronic contractile dysfunction in viable myocardium due to chronic reduced blood flow. Function may improve with revascularisation.

Risk Factors for Coronary Artery Disease

Recognising and managing risk factors is key for IHD prevention.

- Non-Modifiable:
  - Age, Sex, Family History (premature CAD)
  - Ethnicity (higher rates in South Asian, Aboriginal and Torres Strait Islander peoples)
- Modifiable:
  - Smoking, Hypertension, Dyslipidaemia (high LDL-C, low HDL-C, high Triglycerides), Diabetes Mellitus, Obesity (central), Physical Inactivity, Unhealthy Diet, Psychosocial Factors (stress, depression, isolation), chronic kidney disease (CKD), Inflammatory Conditions
- Metabolic Syndrome: Central obesity, hypertension, insulin resistance/T2DM, high triglycerides, low HDL-C – significantly increases ASCVD risk.

Prevention of Ischaemic Heart Disease in General Practice

- **Primary Prevention:**
  - Lifestyle Modification (SNAP): Smoking cessation, Nutrition, Alcohol limitation, Physical activity.
  - Blood Pressure Control: Target <130/80 mmHg generally.
  - Lipid Management: Statin therapy based on risk and discussion. LDL-C reduction or targets (e.g., <2.0 mmol/L).
  - Diabetes Management: Optimise glycaemic, BP, and lipid control.
  - Aspirin: Not routinely recommended for primary prevention. Consider selectively in high-risk, low-bleeding risk individuals after discussion.
- **Secondary Prevention:**
  - Known IHD patients are High Risk. Aggressive risk factor control and mandatory medications:
    - Risk Factor Targets: BP <130/80 mmHg; LDL-C <1.8 mmol/L (consider <1.4 mmol/L if very high risk); HbA1c <7% (individualised).
    - Cardiac Rehabilitation: Strongly recommended post-MI/revascularisation.

elevation (concave up) and PR depression (though can sometimes mimic NSTEACS). Troponin can be mildly elevated.

Musculoskeletal Chest Wall Pain: Usually localized and reproducible on palpation. Pain may be sharp or aching and can be exacerbated by movement or breathing.

Gastroesophageal Reflux Disease (GERD) / Oesophageal Spasm: Can cause retrosternal chest pain that may be burning or tight. Often associated with meals or lying down and may be relieved by antacids.

Pulmonary Embolism (PE): Can present with sudden onset chest pain (often pleuritic), shortness of breath, tachycardia, and hypoxia. Risk factors for PE should be considered. ECG may show sinus tachycardia or other non-specific changes.

Aortic Dissection: Typically presents with sudden onset, severe, tearing or ripping chest pain that may radiate to the back. Often associated with hypertension or connective tissue disorders. May have unequal pulses or blood pressure in the arms.

Anxiety / Panic Attack: Can cause chest tightness, shortness of breath, palpitations, and dizziness. Often associated with psychological stress. Symptoms are usually shorter in duration and may be accompanied by hyperventilation. ECG is usually normal.

Pleurisy / Pneumonia: Often presents with pleuritic chest pain (sharp, worse with breathing or coughing), cough, fever, and shortness of breath. Auscultation may reveal abnormal breath sounds.

Conclusion

IHD management is crucial for Australian GPs, encompassing primary prevention based on absolute risk, CCS diagnosis and management, and ACS recognition/initial care. Understanding pathophysiology, risk factors, presentations, and evidence-based strategies for CCS/NSTEACS, guided by Australian guidelines and risk assessment, is vital for improving patient outcomes and GP fellowship success. Emphasis on lifestyle modification, optimal medical therapy, and timely referral are key.

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Murphy et al. (2018): Heart failure guidelines – NHF & CSANZ – *Heart, Lung & Circ*

Clinically Determined High Risk: Existing ASCVD, diabetes + age > 60 or microalbuminuria, moderate/severe CKD (eGFR < 45), confirmed FH, BP ≥ 180/110 mmHg, TC > 7.5 mmol/L, Aboriginal/Torres Strait Islander peoples > 74 years.
Excluding Secondary Causes: Consider poorly controlled diabetes (check HbA1c), hypothyroidism (check TSH), obesity/metabolic syndrome, excessive alcohol, CKD (check eGFR/ACR), nephrotic syndrome, cholestatic liver disease (check LFTs), and medications (review).
Management Strategies
Step 1: Lifestyle Modification (SNAP): Smoking cessation, Nutritional advice (reduce saturated/trans fats, increase fibre, consider plant sterols, limit refined carbs), Alcohol limitation (NHMRC guidelines), Physical activity (≥ 150 min moderate or ≥ 75 min vigorous aerobic weekly, plus muscle strengthening). Consider dietitian/exercise physiologist referral.
Step 2: Address Secondary Causes: Manage underlying conditions/medications.
Step 3: Pharmacotherapy (Guided by Risk & LDL-C):

- Initiation (Primary Prevention): High risk or clinically high risk: statin. Moderate risk: intensive lifestyle first, consider statin if poor response or LDL-C > 3.0 mmol/L (or > 2.5 mmol/L). Low risk: lifestyle focus, statin generally not recommended unless markedly elevated LDL-C (> 5.0 mmol/L).
- Statins: First line for LDL-C lowering. High-intensity (≥ 50% reduction), Moderate-intensity (30-49% reduction). Manage Statin-Associated Muscle Symptoms (SAMS) by validating, excluding other causes, checking CK (if severe symptoms), de-challenging, and re-challenging with lower dose or different statin.
- Ezetimibe: Add-on to statin if LDL-C target not met, or monotherapy if statin intolerant (check PBS criteria). Lowers LDL-C ~15-20%.
- PCSK9 Inhibitors: Specialist initiated for very high-risk patients not at LDL-C target despite max statin + ezetimibe (strict PBS). Potent LDL-C lowering.
- Fibrates: Primarily for severe hypertriglyceridemia (TG > 10 mmol/L) to reduce pancreatitis risk. Caution in CKD, avoid gemfibrozil + statin.
- Prescription Omega-3 Fatty Acids: Adjunct for very high TG.

Treatment Targets (NHFA): LDL-C: High risk/secondary prevention < 1.8 mmol/L (consider < 1.4 if very high risk), Moderate risk considers < 2.5 mmol/L. Non-HDL-C: Aim ~0.8 mmol/L higher than LDL-C target. TG: Initially < 10 mmol/L if severe, optimal likely < 1.7 mmol/L for ASCVD risk reduction.
Step 4: Monitoring and Follow-up: Check lipids 6-12 weeks after therapy change, then annually. Monitor adherence, side effects (especially SAMS). Routine LFT/CK monitoring not needed unless clinically indicated. Annually reassess all risk factors and reinforce lifestyle.
Management in Specific Populations: Earlier screening for Aboriginal/Torres Strait Islander peoples. Aggressive risk factor management for diabetes. Statins generally safe in CKD (adjust doses for some), caution with fibrates. Statins contraindicated in pregnancy/breastfeeding. Individualised approach for older adults (>75 years).

Differential Diagnoses for Dyslipidaemia:

- Normal physiological variation: Lipid levels can fluctuate within a normal range.
- Transient elevations due to acute illness: Infections or other acute conditions can temporarily affect lipid profiles.
- Medication-induced lipid changes: Many medications can alter lipid levels (e.g., diuretics, beta-blockers, corticosteroids, oral contraceptives).
- Post-prandial lipaemia: Although non-fasting screening is common, recent high-fat meals can elevate triglycerides.
- Errors in lipid testing or calculation: Laboratory or calculation errors can lead to inaccurate results.
- Lipoprotein(a) elevation: Elevated Lp(a) is an independent ASCVD risk factor but may not always present as standard dyslipidaemia.

Hypertension (HTN)

Hypertension (high BP) is a leading modifiable risk factor for CVD, stroke, CKD, and cognitive decline in Australia, with prevalence increasing with age. Often asymptomatic ("silent killer"), accurate measurement, timely diagnosis, and effective management by Australian GPs are crucial for mitigating cardiovascular risk. This outlines BP measurement, diagnostic criteria, assessment, and evidence-based management (lifestyle and pharmacological) relevant for fellowship exams.

Accurate Blood Pressure Measurement

Reliable BP measurement is key.

- Technique Essentials: Validated, calibrated device; correct cuff size (bladder encircling 80-100% mid-upper arm); rested patient (5 min seated, back supported, feet flat, arm at heart level, avoid caffeine/smoking/exercise 30 min prior, quiet and still); manual procedure (inflate 30 mmHg above radial pulse disappearance, deflate 2-3 mmHg/beat, record first and last Korotkoff sounds, average ≥2 readings 1-2 min apart); measure both arms initially, use higher reading arm thereafter if >10-15 mmHg difference.
- Methods:
  - Clinic BP: Manual sphygmomanometry, Automated Office Blood Pressure (AOBP - multiple readings after rest, often lower, may correlate better with out-of-office BP).
  - Out-of-Office BP (Preferred): Ambulatory Blood Pressure Monitoring (ABPM - gold standard, 24-hour readings, average BP, diurnal variation, strongest risk predictor; for diagnosis confirmation, white coat/masked HTN, nocturnal BP, resistant HTN). Home Blood Pressure Monitoring (HBPM - patient measures at home, validated device, standardised conditions over several days; empowers patients, improves adherence, useful for diagnosis/monitoring).
- White Coat Hypertension (WCH): Elevated clinic BP, normal out-of-office. Requires out-of-office monitoring.
- Masked Hypertension (MH): Normal clinic BP, elevated out-of-office. Similar risk to sustained HTN. Suspect with normal/high-normal clinic BP but target organ damage or high CVD risk. Requires out-of-office monitoring.

Diagnosis of Hypertension

Align with NHFA/HSRA guidelines. Single elevated reading usually insufficient.

- Diagnostic Thresholds (Australia):
  - Clinic BP: ≥ 140/90 mmHg (average of multiple readings over 2-3 visits).
  - 24-hour ABPM Average: ≥ 130/80 mmHg.
  - Daytime ABPM Average: ≥ 135/85 mmHg.
  - Nighttime ABPM Average: ≥ 120/70 mmHg.
  - HBPM Average: ≥ 135/85 mmHg (average over several days, excluding first day).
- Diagnostic Process: Initial elevated clinic reading (≥140/90 mmHg): repeat after 5 min rest. If still elevated, follow-up visits OR out-of-office monitoring (ABPM/HBPM). Confirmation: Multiple clinic visits (average ≥140/90 mmHg over 2-3 visits weeks apart). ABPM/HBPM (strongly recommended, especially for borderline readings or WCH/MH suspicion; diagnosis if averages exceed thresholds).
- Exceptions (Immediate diagnosis/management): Severely elevated clinic BP (≥ 180/110 mmHg) on first presentation. Hypertensive emergency (acute end-organ damage).

Assessment of the Hypertensive Patient

Thorough assessment post-diagnosis.

- History: Duration, previous readings/treatments, symptoms of target organ damage/secondary causes, lifestyle (SNAP), family history (HTN, premature CVD, CKD, diabetes, FH), medications (including OTC/complementary that can increase BP).

Examination: Accurate BP, weight/height/BMI/waist, fundoscopy (retinopathy), cardiovascular (apex beat, S4, murmurs, bruits, peripheral pulses), respiratory (heart failure signs), abdomen (enlarged kidneys, aortic aneurysm), neurological (focal deficits), other signs of secondary causes.

Investigations (ALL newly diagnosed): UEC (K +, creatinine/eGFR), Lipid Profile, HbA1c or fasting glucose, Urinalysis (protein/blood, ACR if proteinuria), 12-lead ECG (LVH, ischaemia, arrhythmias).

Further Investigations (if suspected): Urine ACR, Echocardiogram, Renal Ultrasound/Doppler/CT/MR Angiography, Plasma Aldosterone-to-Renin Ratio (ARR), Urinary free Cortisol/Dexamethasone Suppression Test, Plasma/24hr Urinary Metanephrines/Catecholamines, TSH, Sleep Study.

Classification and Causes

Primary (Essential) Hypertension (~95%): No single cause, gene-environment interactions. Risk factors: non-modifiable (age, sex, family history, ethnicity), modifiable (high salt, excessive alcohol, obesity, inactivity, poor diet, smoking, stress).

Secondary Hypertension (~5%): Identifiable underlying cause. Suspect: young onset (<30 years), abrupt onset/worsening, resistant HTN, specific clinical clues. Common causes: CKD (most common), renovascular disease, primary aldosteronism, OSA, drug-induced, thyroid disease, excessive alcohol. Rarer causes: pheochromocytoma, Cushing's, coarctation, hyperparathyroidism, genetic syndromes.

Cardiovascular Risk Assessment

Use the Australian Absolute CVD Risk Calculator (integrates BP with other risk factors) to estimate 5-year risk and guide management intensity. Certain conditions automatically confer high risk.

Management of Hypertension

Goals: Lower BP to target to reduce long-term risks.

Treatment Goals (NHFA/HSRA): General target < 140/90 mmHg (clinic). Lower targets (< 130/80 mmHg for established CVD, CKD/diabetes with albuminuria, high CVD risk if tolerated; consider < 120 mmHg systolic in selected high-risk non-diabetics with careful monitoring). Individualise targets, avoid overly aggressive lowering in frail elderly or postural hypotension risk. Aim for diastolic BP > 60-70 mmHg in CAD.

Lifestyle Modifications (Foundation): Salt restriction (< 4g salt/day), weight reduction (healthy BMI), healthy diet (DASH-like), regular physical activity (≥150 min moderate or ≥75 min vigorous aerobic weekly + resistance x2), limit alcohol (≤10 std drinks/week, ≤4/day), smoking cessation.

Pharmacological Therapy:

Initiation Thresholds: High/clinically high risk: ≥ 140/90 mmHg. Moderate risk: ≥ 140/90 mmHg despite 3-6 months lifestyle. Low risk: ≥ 160/100 mmHg.

First-Line Agents ('ACD'):

A (ACEi/ARB): Younger patients, diabetes, CKD, HF. Monitor K + /renal function.

C (CCB - Dihydropyridine preferred): Older patients, isolated systolic HTN. Peripheral oedema common.

D (Thiazide-Like Diuretic - Indapamide/Chlorthalidone preferred): Older patients, isolated systolic HTN, HF (volume). Monitor electrolytes.

Beta-blockers NOT typically first-line unless compelling indication.

Combination Therapy: Often required (≥2 agents). Consider low-dose combinations if BP significantly above target. Preferred: ACEi/ARB + CCB or TLD. Avoid ACEi + ARB. Beta-blockers can be added if needed.

Compelling Indications: HF (ACEi/ARB + Beta-blocker + MRA), post-MI (Beta-blocker + ACEi/ARB), CKD with albuminuria (ACEi/ARB), Diabetes (ACEi/ARB often preferred), AF (Beta-blocker/Non-DHP CCB), BPH (Alpha-blocker as add-on).

Resistant Hypertension: BP above target on ≥3 drugs (including diuretic). Confirm true resistance, intensify lifestyle, review meds, screen for secondary causes, optimise diuretic (consider MRA as 4th line). Consider referral.

Peripheral artery disease (claudication)
Sciatica

Red Flags

Sudden onset of unilateral leg swelling and pain, especially with risk factors.
Calf pain on palpation along the course of the deep veins.
Concurrent chest pain or shortness of breath (suggesting possible PE).
Phlegmasia alba dolens or phlegmasia cerulea dolens.
Palpable cord in the subcutaneous tissue (suggesting superficial thrombophlebitis, which can sometimes be associated with DVT).
Significant leg swelling or pain in a postpartum woman.
Leg swelling or pain in a patient with known malignancy.
Failure to improve with conservative management for suspected musculoskeletal injury.

Conclusion

Deep Vein Thrombosis is common and potentially serious in Australian primary care. Effective management relies on understanding Virchow's triad, high suspicion, clinical prediction rules (Wells' score), diagnostic algorithm (D-dimer, CUS), and anticoagulation (predominantly DOACs). Preventing hospital-acquired thrombosis is vital. GPs play a key role in managing long-term Post-Thrombotic Syndrome through education and compression therapy, as well as coordinating ongoing care and secondary prevention.

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Peripheral Arterial Disease and Lymphedema

This chapter addresses Peripheral Arterial Disease (PAD), caused by atherosclerosis reducing arterial blood flow, and Lymphedema, resulting from impaired lymphatic drainage causing chronic swelling. PAD signifies systemic atherosclerosis with high cardiovascular risk, while lymphedema primarily impacts quality of life and predisposes to infection. Understanding assessment, diagnosis, and management for both conditions in Australia is crucial for GPs.

Peripheral Arterial Disease (PAD)

Peripheral Arterial Disease (PAD) is atherosclerotic obstruction of arteries supplying limbs (lower more common). This causes oxygen supply/demand mismatch, especially during exertion, leading to claudication, and severe cases, critical limb ischaemia. PAD indicates systemic atherosclerosis, significantly increasing risk of MI, stroke, and cardiovascular death. Pathophysiology involves plaque formation in arterial intima, causing luminal narrowing (stenosis) or occlusion, similar to CAD.

Risk Factors for PAD are those for systemic atherosclerosis:

- Smoking: Strongest modifiable factor. Causes endothelial dysfunction, inflammation, plaque progression. Cessation paramount.
- Diabetes Mellitus: Increases PAD risk and severity, often affecting distal arteries. Associated with neuropathy, increasing ulcer risk.
- Age: Prevalence increases markedly > 65 years.
- Hypertension: Contributes to endothelial damage and atherosclerosis.
- Hyperlipidaemia: Elevated LDL-cholesterol promotes plaque formation.
- Chronic Kidney Disease (CKD): Associated with accelerated atherosclerosis and vascular calcification.
- Family History of PAD, CAD, or stroke.
- Male Sex (though prevalence increases in women post-menopause).
- Obesity & Physical Inactivity.

Clinical Presentation varies widely, from asymptomatic to limb-threatening ischaemia.

- Asymptomatic PAD: Many (~40-50%) are asymptomatic, diagnosed via screening (e.g., ABI) or incidentally. Still carries high systemic cardiovascular risk.
- Intermittent Claudication (IC): Classic symptom. Muscle pain/cramping/aching/fatigue (usually calf, but can be buttock, hip, thigh, or foot) induced by walking, occurs reproducibly after certain distance, relieved quickly by rest. Location suggests occlusion site.
- Chronic Limb-Threatening Ischemia (CLTI): Formerly Critical Limb Ischemia (CLI). Severe chronic PAD with high amputation/mortality risk. Defined by PAD plus ischaemic rest pain OR ischaemic tissue loss (non-healing ulcer/gangrene).
- Acute Limb Ischaemia (ALI): Vascular emergency with sudden cessation of limb perfusion, threatening viability. Causes: Embolism (usually cardiac origin), Thrombosis in situ, Graft/Stent Occlusion, Trauma, Aortic Dissection. Clinical Features ("6 Ps"): Pain, Pallor, Pulselessness, Poikilothermia, Paraesthesia, Paralysis.

Physical Examination Findings in Chronic PAD:

- Pulses: Diminished/absent peripheral pulses distal to stenosis. Compare sides. Listen for bruits over major arteries.
- Skin Changes: Coolness, pallor on elevation, dependent rubor, shiny/atrophic skin, hair loss, thickened nails.
- Capillary Refill: Delayed (> 2-3 seconds, severely prolonged in CLTI).
- Ulcers/Gangrene: Assess location, appearance, signs of infection.
- Muscle Atrophy.

Heart Failure

Heart Failure (HF) is a complex clinical syndrome, not a single disease, characterized by typical symptoms (e.g., breathlessness, ankle swelling, fatigue) and signs (e.g., elevated jugular venous pressure, pulmonary crackles, peripheral oedema) caused by a structural and/or functional cardiac abnormality. This results in reduced cardiac output and/or elevated intracardiac pressures at rest or during stress. HF significantly impacts quality of life and survival, and is a leading cause of hospitalisation in Australia, particularly among older adults. General Practitioners are central to the diagnosis, ongoing management, coordination of care, and optimization of outcomes for patients living with HF in the community.

Pathophysiology Simplified for General Practice

HF fundamentally arises from the heart's inability to pump blood effectively (systolic dysfunction) or fill adequately (diastolic dysfunction), or often a combination.

- Systolic Dysfunction (Impaired Pumping): Ventricle (usually left) contracts weakly, leading to reduced Ejection Fraction (HFrEF).
- Diastolic Dysfunction (Impaired Filling): Ventricle becomes stiff, impairing filling even with normal contractility, leading to increased filling pressures (HFpEF).
- Neurohormonal Activation: Body activates compensatory mechanisms (RAAS, SNS) in response to reduced cardiac output or increased wall stress. Chronic activation is detrimental, causing cardiac remodelling, fibrosis, worsening fluid retention, and increased myocardial oxygen demand. Guideline-directed medical therapies target blocking these maladaptive neurohormonal systems.
- Left-sided vs Right-sided HF: Left HF causes pulmonary congestion (dyspnoea, orthopnoea, PND, crackles). Right HF causes systemic congestion (peripheral oedema, elevated JVP, hepatomegaly, ascites). Right HF most commonly caused by Left HF.

Aetiology (Common Causes Encountered by GPs)

Identifying the underlying cause is important as it may influence management.

- Ischaemic Heart Disease (IHD): Most common cause in Australia, resulting from previous Myocardial Infarction (MI).
- Hypertension: Chronic pressure overload leads to LV hypertrophy, diastolic dysfunction, and eventually systolic dysfunction.
- Valvular Heart Disease: Aortic stenosis, mitral regurgitation, aortic regurgitation etc.
- Cardiomyopathies: Dilated Cardiomyopathy (DCM), Hypertrophic Cardiomyopathy (HCM), Restrictive Cardiomyopathy (e.g., Amyloidosis, Haemochromatosis).
- Arrhythmias: Especially chronic, poorly controlled Atrial Fibrillation (AF).
- Alcohol Excess.
- Diabetes Mellitus: Increases risk significantly.
- Chronic Kidney Disease (CKD).
- Less Common: Thyroid disease (hypo/hyper), viral myocarditis, congenital heart disease, COPD (cor pulmonale - primarily right HF), chemotherapy, sleep apnoea.

Classification Systems (Exam Relevance)

Two key **Classification**s used in practice and likely in exams:

- Based on Left Ventricular Ejection Fraction (LVEF): Crucial for guiding therapy. Determined by Echocardiography.
  - HFrEF (HF with Reduced Ejection Fraction): LVEF ≤ 40%. (Systolic HF)
  - HFmrEF (HF with Mildly Reduced Ejection Fraction): LVEF 41% – 49%.
  - HFpEF (HF with Preserved Ejection Fraction): LVEF ≥ 50%. (Diastolic HF)

Exam Pearls: HF Classification by LVEF

HFrEF: LVEF ≤ 40% (Reduced) -> Quad therapy (ARNI/ACEi/ARB + BB + MRA + SGLT2i).

Red Flags (Carotid Artery Stenosis):

Sudden onset of focal neurological deficit (weakness, numbness, speech difficulty).
Transient loss of vision in one eye (amaurosis fugax).
Carotid bruit associated with neurological symptoms.
History of TIA or stroke.
Presence of multiple risk factors for atherosclerosis.
Rapidly recurring transient neurological symptoms.
Any neurological deficit that does not completely resolve within one hour.
New onset of severe headache associated with neurological symptoms.

Conclusion

Both STEMI and symptomatic carotid artery stenosis are critical vascular events where prompt recognition and intervention significantly alter outcomes. STEMI management revolves around rapid reperfusion via PPCI or fibrinolysis. Carotid stenosis management focuses on stroke prevention via aggressive medical therapy for all, with revascularisation for selected symptomatic patients.

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Allergic and Immune-Mediated Skin Disorders

Allergic and immune-mediated skin disorders are common in Australian general practice. These conditions, from eczemas and urticaria to psoriasis and drug eruptions, cause considerable patient distress. Accurate diagnosis relies on pattern recognition, thorough history (including triggers and timing), and examination. Effective management involves addressing pathophysiology, providing symptomatic relief, educating patients on trigger avoidance and skincare, and knowing when to refer to specialists. This chapter covers assessment and management principles for key conditions relevant to GP exams and daily practice.

Types of Hypersensitivity Reactions

Understanding hypersensitivity mechanisms helps contextualise many immune-mediated skin diseases.

- Type I (Immediate/IgE-Mediated): IgE on mast cells/basophils bind antigen, triggering rapid histamine release. Examples: Urticaria (wheals), Angioedema, Anaphylaxis.
- Type II (Cytotoxic/Antibody-Mediated): IgG/IgM bind to antigens on cell surfaces, leading to complement activation or cytotoxicity. Examples: Pemphigus vulgaris, Bullous pemphigoid.
- Type III (Immune Complex-Mediated): Antigen-antibody complexes deposit in tissues, activating complement and neutrophils, causing inflammation. Example: Vasculitis.
- Type IV (Delayed-Type/Cell-Mediated): Sensitised T-lymphocytes react to antigen, releasing cytokines, causing inflammation (24-72 hours). Examples: Allergic Contact Dermatitis, some Drug Eruptions, aspects of Psoriasis.

Atopic Dermatitis (Eczema)

Definition & Aetiology

Atopic dermatitis (AD or eczema) is a common, chronic, relapsing inflammatory skin disease with intense pruritus and eczematous lesions, typically starting in infancy. Pathogenesis involves epidermal barrier dysfunction (e.g., Filaggrin mutations), immune dysregulation (skewed T-cell responses, IgE production), environmental factors (allergens, irritants, climate, infections), and genetic predisposition (atopic triad family history).

Clinical Features

Presentation varies with age: Infants (<2 years): Pruritic, erythematous, weeping/crusted papules/vesicles on face, scalp, extensor limbs. Children (2 years - puberty): Drier, papular lesions on flexures, lichenification. Adults: Localised, chronic with lichenification/dryness on flexures, hands, face, neck. Xerosis universal. Prone to secondary skin infections (Staph aureus, Herpes simplex - 'eczema herpeticum').

Diagnosis

Primarily clinical, based on history and examination using diagnostic criteria. Key features: pruritus, typical morphology/age-specific patterns, chronic/relapsing, personal/family atopy history. Allergy testing not required for diagnosis. Skin biopsy usually not needed.

Management

Stepwise approach: basics, managing flares, long-term prevention.

- Basic Measures (Essential for All): Emollients/Moisturisers (liberal, frequent), Trigger Avoidance (irritants, allergens where relevant).
- Managing Acute Flares: Topical Corticosteroids (TCS) first-line anti-inflammatory (potency based on severity/site/age). Topical Calcineurin Inhibitors (TCIs) second line for sensitive areas/maintenance (Authority Rx). Wet Dressings (severe, exudative flares, specialist guidance). Antihistamines (sedating for sleep). Treating Secondary Infection (oral/topical antibiotics, antiseptic washes, systemic antivirals for eczema herpeticum).

Postherpetic Neuralgia (PHN), Herpes Zoster Ophthalmicus (HZO - ophthalmic emergency!), Herpes Zoster Oticus (Ramsay Hunt Syndrome), disseminated zoster, motor neuropathy, meningitis/encephalitis (rare), secondary bacterial infection. Diagnosis: Usually clinical. PCR of vesicular fluid can confirm. Management: Antiviral Therapy (oral Valaciclovir, Famciclovir, or Aciclovir within 72 hours). Analgesia essential (simple to neuropathic agents). Lesion Care: Keep clean/dry, cover. Specific Complications: Urgent ophthalmology referral for HZO. Consider oral corticosteroids for Ramsay Hunt (specialist advice). Manage PHN with neuropathic agents. Prevention: Zoster vaccine (Shingrix preferred, funded on NIP for specific groups). Exclusion: Cover lesions, avoid contact with non-immune.

Other Human Herpesviruses (Briefly)

Epstein-Barr Virus (EBV / HHV-4): Infectious Mononucleosis ('Glandular Fever'). Triad: Fever, Pharyngitis, Lymphadenopathy. Fatigue prominent. Splenomegaly. Hepatomegaly/hepatitis. Ampicillin rash. Diagnosis: Monospot test, EBV serology. Management: Supportive. Cytomegalovirus (CMV / HHV-5): Usually asymptomatic in immunocompetent. Mono-like illness (heterophile negative). Major pathogen in immunocompromised. Congenital CMV important. Diagnosis: PCR, serology, biopsy. Rx: Ganciclovir, Valganciclovir (specialist). HHV-6 / HHV-7: Roseola Infantum ('Sixth Disease'). Abrupt high fever then maculopapular rash trunk outwards. Benign, self-limiting. HHV-8 (KSHV): Kaposi Sarcoma.

Human Papillomavirus (HPV)

Cause benign warts and associated with malignancy (cervical, anal, oropharyngeal cancers - high-risk types). Common Warts (Verruca Vulgaris), Plantar Warts (Verruca Plantaris), Plane Warts, Genital Warts (Condylomata Acuminata). Management of Cutaneous Warts: Often self-resolve. Treatment aims to destroy infected tissue: Topical (Salicylic acid, Podophyllotoxin [genital], Imiquimod [genital/peri-anal - Rx]). Destructive (Cryotherapy, Curettage + /- Cautery, Laser). Prevention: HPV Vaccine (Gardasil 9) on NIP.

Molluscum Contagiosum

Cause: Poxvirus infection. Common in young children, sexually active adults, immunocompromised. Transmission: Direct skin contact, fomites. Clinical Features: Small (2-5mm), pearly, umbilicated papules. Management: Often self-limiting, treatment if troublesome: Cryotherapy, Curettage, Topical podophyllotoxin/Imiquimod/Potassium hydroxide.

Common Viral Exanthems (Brief for differentiation)

Measles, Rubella, Erythema Infectiosum (Fifth Disease / 'Slapped Cheek'), Roseola Infantum (Sixth Disease), Hand, Foot, and Mouth Disease (HFMD).

Exam Pearls: Viral Skin Infections

HSV: Recurrent painful vesicles (oral/genital). Dx: PCR. Rx: Antivirals (Episodic/Suppressive). Whitlow=finger. Eczema Herpeticum=Emergency. VZV (Chickenpox): Itchy vesicles in CROPS (different stages). Supportive Rx mostly. Vaccine preventable. VZV (Zoster/Shingles): Unilateral dermatomal vesicles + PAIN. Rx: Antivirals <72h + Analgesia. Complications: PHN, HZO (Ophtho emergency!), Ramsay Hunt (Facial palsy). Vaccine preventable (Shingrix preferred). Warts (HPV): Common/Plantar/Genital. Rx: Salicylic acid, Cryotherapy. Vaccine preventable. Molluscum: Umbilicated papules. Poxvirus. Often self-limiting. Cryo/curettage if needed. Exanthems: Measles (3 C's + Koplik spots then rash head->down), Rubella (Rash head->down fast + nodes), Erythema Infectiosum (Slapped cheek then lacy rash), Roseola (Fever THEN rash trunk->out), HFMD (Mouth ulcers + Hand/Foot vesicles).

Contact dermatitis (irritant or allergic)
Steroid-induced rosacea-like eruption
Carcinoid syndrome (flushing)
Telangiectasia due to other causes (e.g., sun damage)

Red Flags (Rosacea):

Sudden onset of severe facial redness and swelling.
Presence of nodules or thickened skin (phymatous changes) developing rapidly.
Ocular symptoms such as severe eye pain, blurred vision, or photophobia (may indicate ocular rosacea with keratitis).
Lack of response to standard rosacea treatments.
Development of pustules or cysts that are atypical for rosacea.
Unilateral facial flushing or redness.
Association with other systemic symptoms.

Differential Diagnosis: (Pityriasis Rosea):

Secondary syphilis (consider if palms/soles involved, lymphadenopathy)
Tinea corporis (scrape for hyphae)
Guttate psoriasis
Drug eruption
Viral exanthem
Nummular eczema
Lichen planus (less likely to have herald patch)
Seborrhoeic dermatitis (less distinct oval plaques)

Red Flags (Pityriasis Rosea):

Atypical distribution of the rash (e.g., face, scalp, palms, soles).
Presence of systemic symptoms such as fever, malaise, or significant lymphadenopathy (consider secondary syphilis or drug eruption).
Rash that is intensely itchy or painful.
Lack of a herald patch.
Failure of the rash to resolve within the typical timeframe (12 weeks).
Suspicion of secondary syphilis based on risk factors or other clinical findings.
Mucosal involvement.

Differential Diagnosis: (Vitiligo):

Tinea versicolor (scrape for hyphae, may fluoresce under Wood's lamp)
Post-inflammatory hypopigmentation (history of prior inflammation)
Pityriasis alba (often slightly scaly, less sharply demarcated)
Chemical leukoderma (history of exposure to certain chemicals)
Albinism (generalized lack of pigment, often with eye involvement)
Idiopathic guttate hypomelanosis (small, scattered white spots on sun-exposed areas)
Leprosy (hypopigmented patches with sensory loss)
Sarcoidosis (hypopigmented macules)

Red Flags (Vitiligo):

Rapid onset or widespread depigmentation.
Associated symptoms such as hair loss (alopecia areata), eye problems, or thyroid dysfunction (consider associated autoimmune conditions).

Diameter greater than 6 mm.
Evolution or change in size, shape, colour, or elevation.
Elevated surface.
Firm texture.
Growing progressively over more than one month (especially for nodular melanoma).

Conclusion

Assessment/management of skin neoplasms are significant in Australian general practice due to high UV-induced skin cancer prevalence. GPs must recognise benign lesions, identify pre-malignant lesions (Actinic Keratosis), and diagnose main skin cancers (BCC, SCC, Melanoma) accurately/promptly. Requires strong clinical skills, dermoscopy/biopsy use, knowledge of management options, and patient education on sun protection/self-examination. Familiarity with other conditions enhances diagnostic capability.

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Treatment of choice. Start low dose in elderly/IHD. Take empty stomach, separate from Ca/Fe. Monitor TSH q4-8wks initially, aim normal range (lower half often best). Lifelong Rx usual. Subclinical Hypo: Treat if TSH>10. Consider Rx if TSH 5-10 + Symptoms / Pregnant / + TPOAb / Goitre / <70y. Monitor if untreated. Myxoedema Coma = EMERGENCY: Severe Hypo + Hypothermia + ↓LOC. ICU + IV T4 + IV Hydrocortisone.

Thyroiditis

Definition &

Thyroiditis = thyroid gland inflammation. Can cause pain and transient/permanent dysfunction. Often triphasic course: Thyrotoxic -> Hypothyroid -> Euthyroid. LOW Radioiodine Uptake during thyrotoxic phase. ATDs are INEFFECTIVE.

Types of Thyroiditis

Subacute (de Quervain's / Granulomatous) Thyroiditis (presumed post-viral, neck pain/tenderness, elevated ESR, self-limiting, Rx pain with NSAIDs/Prednisolone, Beta-blockers for Sx). Painless (Silent / Lymphocytic) Thyroiditis (autoimmune, non-tender goitre, triphasic TFTs, Rx Beta-blockers/Levothyroxine, higher risk permanent Hypo). Postpartum Thyroiditis (painless within 12 months postpartum, autoimmune, triphasic TFTs, supportive Rx, higher risk permanent Hypo). Drug-Induced Thyroiditis. Acute (Suppurative) Thyroiditis (rare bacterial infection, severe neck pain/fever, tender swelling, Rx IV antibiotics + /- surgical drainage).

Exam Pearls: Thyroiditis Patterns

Common Feature: Often Triphasic TFTs (Hyper -> Hypo -> Euthyroid). LOW Radioiodine Uptake during thyrotoxic phase (hormone release, not overproduction). ATDs are INEFFECTIVE. Subacute (de Quervain's): Post-Viral. PAINFUL/Tender Goitre. ↑↑ESR. Rx: NSAIDs or Prednisolone + Beta-blockers. Usually recovers fully. Painless (Silent/Lymphocytic) / Postpartum: Autoimmune (Anti-TPO + often). Non-tender goitre/normal gland. Rx: Beta-blockers (hyper phase), + /- Levothyroxine (hypo phase). Higher risk permanent Hypo. Postpartum = within 1yr of delivery.

Thyroid Nodules & Neoplasms

Thyroid Nodules

Discrete lesion in thyroid. Prevalence common. Vast majority (>95%) benign. Goal = exclude malignancy. Assessment: History (risk factors!), Examination, Thyroid Function Tests (TSH first), Thyroid Ultrasound (key imaging), Fine Needle Aspiration (FNA) Biopsy (gold standard). Management based on FNA result (Bethesda category) and clinical context. Requires specialist input.

Exam Pearls: Thyroid Nodules & Cancer

Nodules: Very common, >95% benign. Goal = exclude malignancy (~5-10%). Workup: 1. History (Risk factors!) + Exam. 2. TSH. 3. If TSH Normal/High -> Ultrasound (Assess risk features: Hypoechoic, Microcalcs, Taller>Wide, Irregular margin). 4. If TSH Low -> Radionuclide Scan (Hot=Benign usually). 5. FNA Biopsy based on US risk/size (>1-1.5cm generally). FNA Results (Bethesda I-VI): II=Benign (Monitor). V/VI=Malignant/Suspicious (Surgery). III/IV=Indeterminate (Repeat FNA/Molecular/Surgery). I=Non-diagnostic (Repeat FNA). Malignancy Risk Factors: Childhood Radiation Hx, Family Hx, Age<20/>70, Male, Rapid growth, Hoarse, Hard/Fixed nodule, LAD, "Cold" nodule.

Thyroid Cancer

Relatively uncommon, generally excellent prognosis for differentiated types. Differentiated Thyroid Cancer (DTC) (Papillary [most common], Follicular). Medullary Thyroid Cancer (MTC) (from C-cells, ↑Calcitonin). Anaplastic Thyroid Cancer (rare, aggressive). Management requires specialist MDT care.

Exam Pearls: Thyroid Nodules & Cancer

Thyroid Cancer Types: Papillary (~80%): Most common, Lymphatic spread, Excellent prognosis. Rx: Surgery + /- RAI + TSH suppression. Monitor Tg/Anti-Tg. Follicular (~15%): Haematogenous spread, V Good prognosis. Rx similar to Papillary. Medullary (~3-5%): From C-cells (↑Calcitonin). Check for MEN2 (RET gene)! Rx: Surgery (RAI ineffective). Monitor Calcitonin/CEA. Anaplastic (<2%): Elderly, Rapid growth, very poor prognosis. Palliative Rx.

Differential Diagnosis: (Elevated TSH):

Primary Hypothyroidism (Hashimoto's thyroiditis most common)
Subclinical Hypothyroidism
Recovery phase of non-thyroidal illness (sick euthyroid syndrome)
Medication-induced hypothyroidism (e.g., amiodarone, lithium)
Iodine deficiency (rare in Australia)
Congenital hypothyroidism (detected in newborns)
TSH-secreting pituitary adenoma (rare, usually with elevated T4/T3)
Resistance to thyroid hormone (rare genetic condition)

Red Flags (Elevated TSH):

TSH > 10 mU/L, especially if symptomatic.
Presence of goitre with elevated TSH.
Symptoms of hypothyroidism developing rapidly.
Elevated TSH in a pregnant woman.
Elevated TSH in a child or adolescent.
Coexistence of other autoimmune conditions.
Palpable thyroid nodule with elevated TSH.
Failure of TSH to normalize with appropriate levothyroxine treatment.

Differential Diagnosis: (Low TSH):

Primary Hyperthyroidism (Graves' disease most common)
Subclinical Hyperthyroidism
Transient thyrotoxicosis due to thyroiditis
Toxic multinodular goitre or toxic adenoma
Excessive levothyroxine replacement
Non-thyroidal illness (sick euthyroid syndrome, acute phase)
TSH-secreting pituitary adenoma (rare, usually with elevated T4/T3)
Factitious thyrotoxicosis (intentional ingestion of thyroid hormone)

Red Flags (Low TSH):

TSH < 0.1 mU/L.
Presence of thyroid nodule(s) with suppressed TSH.
Symptoms of hyperthyroidism (palpitations, tremor, weight loss).
Eye signs suggestive of Graves' ophthalmopathy.
Rapid onset of thyrotoxic symptoms.
Atrial fibrillation with low TSH.
Family history of Graves' disease or other hyperthyroid conditions.
Low TSH in a pregnant woman.

Conclusion

Red Flags (Polyuria and Polydipsia):

High urine output (>3 L/day) with low urine specific gravity.
Nocturia causing significant sleep disturbance.
Rapid onset of polyuria and polydipsia, especially after head trauma or pituitary surgery.
Dehydration despite increased fluid intake.
Hypernatraemia (elevated serum sodium levels).
New onset of seizures or altered mental status.

Differential Diagnosis: (Symptoms of Acromegaly):

Normal physiological changes with ageing
Arthritis (osteoarthritis)
Carpal tunnel syndrome due to other causes
Obstructive sleep apnoea due to obesity
Diabetes mellitus (unrelated to acromegaly)
Fluid retention due to heart failure or kidney disease
Connective tissue disorders
Paget's disease of bone

Red Flags (Acromegaly):

Noticeable increase in hand or foot size requiring larger shoes or rings.
Coarsening of facial features (enlarged jaw, nose, forehead).
Excessive sweating (hyperhidrosis).
New onset or worsening of snoring and daytime sleepiness (OSA).
Development of diabetes mellitus or worsening glycaemic control.
Persistent headaches or visual field changes.

Differential Diagnosis: (Symptoms of Hyperprolactinemia):

Pregnancy and breastfeeding (physiological)
Medication side effects (antipsychotics, metoclopramide)
Primary hypothyroidism
Chronic kidney disease
Stress
Polycystic ovary syndrome (PCOS)
Normal variation
Chest wall stimulation

Red Flags (Hyperprolactinemia):

Galactorrhoea in non-pregnant, non-breastfeeding women or in men.
Unexplained menstrual irregularities or amenorrhoea.
Erectile dysfunction or decreased libido in men.
Visual field defects or persistent headaches.
Very high prolactin levels on initial testing (>5000 mU/L).
Presence of a pituitary mass on imaging.

Differential Diagnosis: (Symptoms of Hyponatraemia - SIADH):

Dehydration (hypovolaemic hyponatraemia)
Heart failure (hypervolaemic hyponatraemia)
Cirrhosis (hypervolaemic hyponatraemia)

Coagulation Disorders

Haemostasis

Haemostasis is the physiological process stopping bleeding while maintaining normal blood flow. It involves blood vessels, platelets, coagulation factors, natural anticoagulants, and fibrinolysis. Disorders can cause excessive bleeding or inappropriate clotting. GPs frequently manage patients with haemostatic issues, prescribe related medications, and interpret coagulation tests. Understanding haemostatic principles is essential for safe primary care.

Normal Haemostasis

Haemostasis involves sequential steps: Vascular Phase (vasoconstriction), Platelet Phase (primary haemostasis - platelet plug formation via adhesion [vWF-GPIb], activation, aggregation [GPIIb/IIIa-Fibrinogen]), Coagulation Phase (secondary haemostasis - fibrin clot formation via Extrinsic [PT/INR], Intrinsic [aPTT], and Common pathways; Vitamin K dependency for factors II, VII, IX, X, C, S), Regulatory Mechanisms (natural anticoagulants like Antithrombin, Protein C/S, TFPI), and Fibrinolysis (clot breakdown via Plasminogen -> Plasmin -> Fibrin degradation -> D-dimers).

Exam Pearls: **Haemostasis**

Primary Haemostasis = Platelet Plug: Adhesion (vWF-GPIb) -> Activation -> Aggregation (GPIIb/IIIa-Fibrinogen). Defect -> Mucocutaneous bleeding (petechiae, epistaxis). Secondary Haemostasis = Fibrin Clot: Coagulation Cascade (Intrinsic [aPTT pathway], Extrinsic [PT/INR pathway], Common). Needs Factors + Calcium + Phospholipids. Vit K needed for II, VII, IX, X, C, S. Defect -> Deep tissue bleeding (joints, muscles). Fibrinolysis = Clot Breakdown: Plasminogen -> Plasmin (via tPA) -> Fibrin degradation -> D-dimers.

Laboratory Assessment of Haemostasis

Common tests for GPs: Full Blood Count (FBC) (Platelet Count), Prothrombin Time (PT) / International Normalised Ratio (INR), Activated Partial Thromboplastin Time (aPTT), Thrombin Clotting Time (TCT or TT) (Fibrinogen Level), Mixing Studies, D-dimer. Specialist Tests: Platelet Function Analyser (PFA-100), Platelet Aggregometry, Specific Factor Assays, vWF assays, Thrombophilia screen, Lupus Anticoagulant/Antiphospholipid antibodies.

Exam Pearls: **Coagulation Tests**

Platelet Count (FBC): Screens for thrombocytopenia/thrombocytosis. PT/INR: Extrinsic + Common pathways. Monitors Warfarin. ↑ in Warfarin, Vit K def, Liver dz. aPTT: Intrinsic + Common pathways. Monitors UFH. ↑ in UFH, Haemophilia A/B/C, vWD (if ↓FVIII), Liver dz, DIC, Lupus Anticoagulant (paradoxical thrombosis risk!). Mixing Study: Differentiates Factor Deficiency (Corrects) vs Inhibitor (Fails to Correct). D-dimer: Fibrinolysis marker. High Negative Predictive Value -> Used to Rule Out VTE in LOW pre-test probability patients. Low Specificity (many causes for elevation).

Clinical Approach to Bleeding Disorders

History: Crucial for differentiating causes (Type of Bleeding, Severity & Frequency, Onset, Triggers, Medications, Past Medical History, Family History). Examination: Look for signs of bleeding and underlying causes.

Medications Affecting Haemostasis

GPs frequently prescribe and manage these. Understanding mechanism, monitoring, and reversal is essential. Antiplatelet Agents (Aspirin, P2Y12 Inhibitors, Dipyridamole, GP IIb/IIIa Inhibitors). Anticoagulants (Heparins: UFH, LMWH, Vitamin K Antagonist: Warfarin, Direct Oral Anticoagulants (DOACs): Dabigatran, Rivaroxaban, Apixaban, Edoxaban). Thrombolytics (Fibrinolytics).

Exam Pearls: Drugs Affecting Haemostasis

Antiplatelets: Aspirin, Clopidogrel. Arterial thrombosis prevention. Increase bleeding risk. Heparins: UFH (IV, monitor aPTT, Protamine reversal, HIT risk!), LMWH (SC, no routine monitor, renal adjust, partial Protamine reversal, lower HIT risk). Potentiate Antithrombin. Warfarin: Oral Vit K antagonist (II, VII, IX, X, C, S). Monitor INR (target 2-3). Slow onset/offset, interactions. Reverse: Vit K (slow), PCC (rapid). BRIDGE with LMWH/UFH for VTE start. C/I Pregnancy. Preferred for Mech Valves, APS. DOACs: Dabigatran (DTI), Riva/Apixa/Edoxa (Xa Inhib). Oral, fixed dose, no routine monitoring. Preferred NVAF/VTE. Rapid onset/offset. Reversal: Idarucizumab (Dabi), Andexanet (Riva/Apixa) - limited access Aus. PCC?. Renal/Liver dose adjust/C/I. C/I Mech Valves, Mod-Sev MS, APS. Thrombolytics: tPA/TNK-tPA. Dissolve clot (STEMI, PE, Stroke). Risk ICH! Check Contraindications! Reverse: Tranexamic Acid.

Management of Anticoagulation Issues

Managing Supratherapeutic INR (Warfarin). Managing DOAC-Associated Bleeding.

Exam Pearls: Anticoagulant Reversal

Warfarin - Major Bleed: STOP Warfarin + IV Vit K (slowly!) + 4-Factor PCC (Prothrombinex-VF). Warfarin - INR >9, No Bleed: STOP Warfarin + Oral Vit K (2.5-5mg). Dabigatran - Major Bleed: STOP Dabi + Idarucizumab (Praxbind) + /- Supportive care. Apixaban/Rivaroxaban - Major Bleed: STOP DOAC + Andexanet Alfa (if available/indicated) OR consider PCC + /- Supportive care. Heparin (UFH): Protamine Sulfate. LMWH: Partial reversal with Protamine.

Differential Diagnosis: (Presenting with easy bruising or bleeding):

Thrombocytopenia (low platelet count)
Von Willebrand Disease (vWD)
Coagulation factor deficiencies (e.g., haemophilia)
Liver disease (impaired clotting factor synthesis)
Vitamin K deficiency
Medication side effects (e.g., anticoagulants, antiplatelets, NSAIDs)
Connective tissue disorders (e.g., Ehlers-Danlos syndrome)
Henoch-Schönlein purpura (vasculitis)

Red Flags (Presenting with easy bruising or bleeding):

Spontaneous bleeding (e.g., nosebleeds, gum bleeding) without obvious trauma.
Heavy or prolonged bleeding after minor cuts or procedures.
Petechiae (small red or purple spots) on the skin.
Unexplained large bruises or haematomas.
Heavy menstrual bleeding (menorrhagia).
Bleeding into joints (haemarthrosis).
Family history of bleeding disorders.
New onset of bleeding or bruising with no clear cause.

Differential Diagnosis: (Presenting with suspected thrombosis):

Deep vein thrombosis (DVT)

Clinical Features:

Triad: Fever + Headache + Altered Mental State (Confusion, Behaviour Δ, ↓LOC).

Seizures common. Focal Deficits common. (Temporal lobe signs -> think HSV!).

Diagnosis:

MRI Brain = Best imaging (Temporal changes suggest HSV?).

LP + CSF: Lymph pleocytosis, Normal Glucose usually. CSF PCR for HSV/VZV/Entero = CRITICAL!

EEG helpful (r/o NCSE).

Management:

Urgent Hospital!

Start EMPIRICAL IV ACICLOVIR (10mg/kg TDS IV) ASAP in ALL suspected encephalitis! Does not wait for results. Treat 14-21d if HSV.

Supportive Care. Immunotherapy if Autoimmune.

Notify!

Other Key CNS Infections

Cryptococcal Meningitis: Immunocompromised mainly (HIV CD4<100). Subacute HA/Altered LOC. Meningism often mild/absent. ↑ICP common. Dx: LP -> CSF CrAg (Best!), India Ink +. Rx: Antifungals (AmphoB etc) + Manage ↑ICP (Specialist).

Cerebral Toxoplasmosis: Immunocompromised (HIV CD4<100). HA/Fever/Seizure/Focal deficit. Dx: Imaging = Multiple Ring-Enhancing Lesions + Toxo IgG +. Rx: Pyrimethamine + Sulfadiazine + Folinic Acid (Specialist).

Brain Abscess: Focal pus. Source? (Sinus/Ear/Dental/Blood). HA/Fever/Focal signs/Seizures. Dx: Ring-Enhancing Lesion on CT/MRI + Contrast. Rx: Drainage + /- Prolonged IV Abx (Neurosurg/ID consult).

Differentials for Fever, Headache, Altered LOC, Neck Stiffness:

Subarachnoid Haemorrhage (SAH) (Sudden severe HA, neck stiffness, LOC change).

Severe Migraine / Complicated Migraine.

Stroke (esp. Haemorrhagic / Posterior Circulation).

Severe Systemic Infection / Sepsis with encephalopathy.

Metabolic Encephalopathy (DKA, Uraemia, Hepatic, Electrolyte disturbance).

Autoimmune disease with CNS involvement (Vasculitis, SLE Cerebritis).

Drug Overdose / Withdrawal / Toxicity (Serotonin Syndrome, NMS).

Brain Tumour (Primary/Metastatic causing HA/LOC change).

Red Flags / Urgent GP Actions for CNS Infections:

Suspected Bacterial Meningitis (Fever, HA, Neck Stiffness, Altered LOC) -> Urgent ED Transfer (000).

Petechial/Purpuric Rash + Fever/Illness -> Suspect Meningococcal -> Give STAT IV/IM Benzylpenicillin/Ceftriaxone PRE-TRANSFER + Urgent ED (000).

Suspected Encephalitis (Fever, HA, Altered LOC, Seizures, Focal signs) -> Urgent ED Transfer (000) (Needs empirical Aciclovir ASAP).

Signs Raised ICP (Papilloedema, Cushing's triad, ↓LOC).

New Seizure + Fever/HA.

Focal Deficits + Fever/HA.

Action: High suspicion key. Rapid ABCDE. Urgent Hospital Transfer (000). Pre-hospital Abx ONLY for suspected meningococcal disease + significant delay (local protocol). Do NOT perform LP in community. Provide collateral Hx. Notify PHU later for confirmed bacterial cases.

Disorders of the Neuromuscular Junction

NMJ: Synapse where motor neurons signal muscles via Acetylcholine (ACh). NMJ Disorders -> Muscle weakness/fatigue. Key Disorders:

- Myasthenia Gravis (MG): Autoimmune attack postsynaptic ACh Receptors (AChR) or MuSK.
- LEMS: Autoimmune attack presynaptic Calcium Channels (VGCC). Often paraneoplastic (SCLC!).
- Botulism: Toxin blocks presynaptic ACh release.

GP Role: Recognise patterns, Initial Ix, Urgent Referral (Neuro/ED for crisis/Botulism), Long-term shared care.

Myasthenia Gravis (MG)

Patho: Autoimmune vs AChR (~85%) or MuSK (~5-8%). Reduced functional AChRs -> Fatigable weakness. Thymus involved (Hyperplasia/Thymoma).

Features: FLUCTUATING, FATIGABLE WEAKNESS (worse with use / end of day).

- Ocular (>50% onset): Ptosis, Diplopia (Pupils NORMAL).
- Bulbar: Dysarthria, Dysphagia (worsen with use).
- Limb: Proximal > Distal. Neck weak ('dropped head').
- Normal Sensation & Reflexes.

Myasthenic Crisis: Life-threatening respiratory failure! Trigger (Infection common). -> Urgent ICU/HDU! Needs Airway support + PEX/IVIG.

Diagnosis (Neuro): Clinical (Fatigability tests, Ice Pack test). Serology: AChR-Ab -> MuSK-Ab if neg. RNS: Shows Decrement (>10%). SFEMG (most sensitive). CT Chest (Rule out Thymoma!).

Management (Neuro + GP shared care):

- Symptomatic: Pyridostigmine (AChE Inhibitor). Manage cholinergic SEs.
- Immunosuppression (IST): Most need Prednisolone + /- Azathioprine/Mycophenolate. Rituximab (esp MuSK +).
- Rapid IST (Crisis): PEX / IVIG.
- Thymectomy: If Thymoma OR young (<~65) AChR + Generalised MG.
- General: Avoid certain meds (provide list!), MedicAlert, Support groups (MG Assoc Aus).

Lambert-Eaton Myasthenic Syndrome (LEMS)

Patho: Autoimmune vs Presynaptic VGCC -> ↓ACh release.

Aetiology: Paraneoplastic (~50-60%) - esp. SMALL CELL LUNG CANCER (SCLC)! OR Autoimmune. MUST SCREEN FOR CANCER!

Features (Triad):

- Proximal Weakness (Legs>Arms) -> IMPROVES briefly with exertion.
- Autonomic: Dry Mouth! Constipation, Erectile Dysfunction.
- Areflexia -> INCREASES post-exercise.

Diagnosis (Neuro): Clinical + Anti-VGCC Abs + RNS shows INCREMENT (>60%) post-exercise. Urgent Malignancy Screen! (CT C/A/P + /- PET).

Management (Neuro + /- Onc): Treat Cancer! Symptomatic: Amifampridine (3,4-DAP - SAS access) + /- Pyridostigmine. IST for autoimmune LEMS.

Botulism

Patho: Botulinum Toxin (C. botulinum) -> Irreversibly blocks Presynaptic ACh Release -> Flaccid Paralysis.

Sources: Foodborne (improper canning!), Infant (<1y - AVOID HONEY!), Wound (IVDU/trauma).

Features: Acute DESCENDING, SYMMETRIC Flaccid Paralysis + Bulbar Palsy ("4 D's": Diplopia, Dysphagia, Dysarthria, Dry mouth + /- Dilated Pupils) + Autonomic Sx. Sensation INTACT. No Fever. RESPIRATORY FAILURE = Critical Threat! Infant: Constipation, floppy, poor feed.

Diagnosis: Clinical! Notify Public Health Unit (PHU) URGENTLY! Lab confirmation (toxin assay) takes days. EMG shows pre-synaptic block + /- Increment.

Management: EMERGENCY -> Hospital/ICU!

Supportive Care: Intensive Respiratory Monitoring -> Early Intubation/Ventilation!

Botulism Antitoxin ASAP! (Via PHU). Equine HBAT (Adults), Human BIG-IV (Infants <1yr).

Wound Botulism ONLY: Surgical Debridement + Antibiotics.

Recovery slow (weeks-months).

Differentials for Muscle Weakness / Paralysis:

Guillain-Barré Syndrome (GBS) (Ascending paralysis, areflexia, sensory sx).

Brainstem Stroke (Acute focal signs, asymmetry, UMN signs).

Myopathy (Inflammatory, Metabolic, Dystrophy - proximal weakness, ↑CK).

Spinal Cord Lesion (Sensory level, UMN/LMN signs, Bowel/Bladder).

Motor Neurone Disease (MND) (Progressive UMN + LMN signs, fasciculations).

Tick Paralysis (Australia - *Ixodes holocyclus* - Ascending paralysis - Find/Remove Tick!).

Periodic Paralysis (Episodic weakness, K + related).

Severe Electrolyte Disturbance (Hypokalaemia, Hypophosphataemia).

Red Flags / Urgent GP Actions for NMJ Disorders:

Acute Respiratory Distress / Difficulty Breathing -> Call 000 (Myasthenic Crisis / Botulism).

Acute Descending Paralysis + Bulbar signs ("4 D's") -> Suspect Botulism -> Urgent ED (000) + Notify PHU immediately.

New LEMS diagnosis -> Urgent comprehensive malignancy screen.

Rapidly worsening weakness / Swallowing difficulty in known MG -> Urgent ED/Neuro review (Crisis?).

Action: Recognise patterns. Refer suspected MG/LEMS to Neurology. Urgent ED referral (000) for respiratory compromise / suspected Botulism. Immediate PHU notification for suspected Botulism vital for antitoxin. Screen LEMS for cancer. Provide MG drug avoidance list. Advise re driving restrictions if applicable.

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Demyelinating disorders

Demyelination: Damage to nerve myelin sheath -> impaired conduction. Focus:
- Multiple Sclerosis (MS): Chronic, Immune-mediated CNS disease. Relapsing/Progressive.
- Guillain-Barré Syndrome (GBS): Acute, Immune-mediated PNS polyradiculoneuropathy. Rapid paralysis.

GP Role: Suspect diagnosis, Initial Ix, Timely Referral (Neuro; ED/ICU for GBS/Severe MS relapse), Long-term MS shared care, GBS rehab coordination.

Multiple Sclerosis (MS)

- Patho: CNS inflammation, demyelination, axonal damage (Brain, Spinal Cord, Optic Nerves). Disseminated in Time & Space. Cause: Genes + Environment (↓Vit D, EBV, Smoking?).
- Epidemiology (Aus): ~1:700 Aus (~33k). F>M (~3:1). Onset 20-40y.
- Subtypes: RRMS (~85% onset: Relapses/Remissions); SPMS (Progressive after RRMS); PPMS (~15%: Progressive from onset); CIS (First episode).
- Clinical Features (Variable! Depends on lesion location):
  - Sensory: Numbness, Tingling, Lhermitte's sign (shock on neck flexion).
  - Motor: Weakness (UMN), Spasticity, Gait/Balance issues.
  - Visual: Optic Neuritis (Unilat painful ↓vision, RAPD), INO (Gaze palsy).
  - Brainstem/Cerebellar: Vertigo, Ataxia, Dysarthria.
  - Spinal Cord: Weakness, Sensory level, Bowel/Bladder dysfunction.
  - FATIGUE (Very common). Cognitive issues ('Cog Fog'). Mood disturbance.
  - Uhthoff's phenomenon (Sx worse with heat).
- Diagnosis (Neuro - McDonald Criteria): Clinical + MRI Brain/Spine (KEY!) showing DIS & DIT (T2 lesions + /- Gad + lesions). CSF: Oligoclonal Bands (if Dx uncertain).
- Management (Neuro + GP Shared Care):
  - Acute Relapse: High dose Steroids (IV/Oral) if functionally significant.
  - Disease-Modifying Therapies (DMTs) - RRMS: Start EARLY! Aim: ↓Relapses/Disability. Many options (Injectables/Orals/Infusions). Neuro decision based on activity/safety/etc. (Ocrelizumab only DMT for PPMS).
  - Symptomatic Rx: Fatigue, Spasticity (Baclofen), Pain, Bladder issues etc. MDT input.
  - Lifestyle: Vit D, Exercise, Stop Smoking. Vaccinate (avoid live vax on many DMTs). Driving considerations (Austroads).

Guillain-Barré Syndrome (GBS)

- Patho: Acute immune attack on PNS nerves/roots. Often Post-Infectious (*Campylobacter* common). Demyelinating (AIDP) most common Aus; Axonal (AMAN/AMSAN) exists.
- Clinical Features:
  - Rapidly Progressive, ASCENDING, Symmetric Weakness. (Nadir <4 weeks).
  - Areflexia / Hyporeflexia (KEY!).
  - Sensory Sx: Often mild (Distal tingling).
  - Cranial Nerves: Bilateral Facial Palsy (~50%). Bulbar/Eye movements. (Miller Fisher = Oculomotor + Ataxia + Areflexia).
  - Autonomic Dysfunction (~70%): BP/HR lability -> Monitor closely!
  - Respiratory Failure (~25-30%): Diaphragm/Intercostal weakness -> Requires URGENT monitoring/support!

Diagnosis (Hospital/Neuro): Clinical pattern + CSF: ↑Protein + Normal WCC (Albuminocytologic dissociation - often after 1wk). NCS/EMG: Confirms (usually after 1-2wks). Exclude mimics (esp Spinal Cord!).

Management (Hospital Admission - HDU/ICU often):

Supportive Care (CRUCIAL):

Monitor Respiratory Function (FVC, NIF)! -> Low threshold for Intubation/Ventilation. Autonomic monitoring. DVT Prophylaxis. Pain control. Pressure/Eye/Bowel/Bladder care.

Immunomodulatory Therapy: If unable walk (start early!). IVIG OR Plasma Exchange (PEX). STEROIDS NOT EFFECTIVE!

Rehabilitation: Prolonged Physio/OT essential. NDIS support potential.

Prognosis: Most recover (~85% walk), often slow. Mortality ~3-7%. Residual deficits ~20%. Relapse ~5% (-> CIDP).

Differentials for MS/GBS Presentations:

(Consider based on specific presenting symptoms)

Stroke (esp. Brainstem/Lacunar for MS mimic; rarely GBS mimic).

Spinal Cord Lesion (Compression, Transverse Myelitis - mimics MS myelopathy or GBS).

CNS Infection (Meningitis/Encephalitis - mimics MS relapse).

Neuromyelitis Optica Spectrum Disorder (NMOSD) (Optic neuritis, myelitis - MS mimic, check Aquaporin-4 Abs).

Systemic Autoimmune Disease (Vasculitis, Sarcoidosis, SLE - can have CNS involvement).

Botulism / Tick Paralysis (mimics GBS - descending paralysis usually).

Myasthenic Crisis (mimics GBS bulbar/respiratory failure - fatigability key).

Functional Neurological Disorder.

Red Flags / Urgent GP Actions:

Suspected GBS (Rapidly progressive weakness + Areflexia) -> Urgent ED Referral (000) (Risk Resp Failure/Autonomic Instability).

Acute significant neurological deficit suggesting MS Relapse (e.g., Optic Neuritis, Transverse Myelitis) -> Urgent Neuro/Ophtho review + /- ED.

Difficulty Breathing / Swallowing in known/suspected GBS or MS -> Call 000.

Signs of Spinal Cord Compression (Acute limb weakness + sensory level + bladder/bowel retention) -> Urgent ED/Neurosurgery.

Fever + Neurological Signs -> Consider CNS Infection -> Urgent ED.

Action: Recognise patterns. Prioritise GBS -> Urgent Hospitalisation (000). Timely Neuro referral for suspected MS/CIS. Coordinate MS shared care (DMTs, Vaccinations, Comorbidities). Address MS driving fitness. Coordinate GBS post-discharge rehab. Provide support resources.

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Dementia (Major Neurocognitive Disorder) in Primary Care

Dementia: Significant cognitive decline INTERFERING with Independence (IADLs/ADLs). Chronic, progressive. Common (AD main cause). Affects ~500k Australians. GP Role: Recognise decline, Initial Assessment (Screen, Ix Reversibles), Diagnosis collaboration, MDT Care Coordination, ACAT referral, BPSD management, Carer Support, Safety (Driving!), Advance Care Planning (ACP).

Definition & Key Mimics

Dementia Criteria: Sig Cognitive Decline + Functional Impairment. Not Delirium/Depression alone.
MUST Exclude Delirium FIRST! (Acute onset, Fluctuating, Inattention).
Differentiate From:
- MND/MCI: Cognitive decline BUT Functionally Independent. High risk progression.
- Depression: Mood primary, Cog Sx improve with Rx. Screen mood (GDS/PHQ-9).
- Normal Ageing: No functional impact.

Clinical Assessment for Dementia

History (Patient + COLLATERAL INFORMANT ESSENTIAL!):
- Cognitive Domains: Memory, Language, Executive Fn, Visuospatial, Social Cog. Onset? Progression?
- FUNCTION: Ability perform IADLs (Finances, Meds, Driving, Shop) & ADLs (Self-care). Assistance needed?
- BPSD: Agitation, Aggression, Psychosis (Visual Hallucinations->DLB?), Wandering, Apathy, Sleep (RBD->DLB?), Anxiety/Depression. Triggers? Impact?
- SAFETY: Driving (accidents?), Wandering, Falls, Home Safety, Vulnerability.
- Meds (esp Anticholinergics!), ETOH, FHx.

Examination:
- General: Vitals, Sensory (Hearing/Vision check!).
- Neuro: Gait (NPH?), Parkinsonism (DLB?), Focal signs (VaD?).
- Cognitive Screen: MoCA / RUDAS preferred (Aus). Interpret score in context. Clock Draw.
- Mood Screen**:** GDS / PHQ-9.

Investigating Suspected Dementia

Screen for Reversible Causes (Essential!):
- Bloods: FBC, EUC, Ca, LFTs, Gluc/HbA1c, TSH, B12/Folate.
- Consider: Syphilis/HIV, Urinalysis (r/o UTI -> delirium).
- Medication Review & Deprescribe: Esp. anticholinergics, benzos, opioids.

Brain Imaging (Recommended):
- MRI Brain: Preferred (Atrophy patterns [AD/FTD], Vascular dz, Excludes mimics).
- CT Head: Alternative (R/o major lesions).

Specialist Ix: LP (rare), Neuropsych Assessment (detailed cognitive profile).

Major Dementia Subtypes

Alzheimer's Disease (AD): Most common (~60-70%). Episodic Memory Loss early/prominent. Gradual decline. Medial Temporal Atrophy (MRI).

Vascular Dementia (VaD): 2nd commonest (~15-20%). Cerebrovascular Dz. Stepwise or gradual decline. Executive Dysfunction often prominent. Rx: Stroke Prevention!

Dementia with Lewy Bodies (DLB): Core Features (≥2): Fluctuating Cognition, Visual Hallucinations, RBD, Parkinsonism. SEVERE ANTIPSYCHOTIC SENSITIVITY!

Frontotemporal Dementia (FTD): Younger onset common (<65). bvFTD: Personality/Behaviour change. PPA: Language decline. Frontal/Temporal atrophy.

Parkinson's Disease Dementia (PDD): Dementia >1yr AFTER motor PD onset.

Others: NPH (Gait + Incontinence + Dementia ->? Shunt), CJD (Rapid!), HIV, Alcohol.

Management of Dementia

Principles: Person-Centred Care. GP coordinates MDT. ACAT referral vital (Home Care Pkgs, Respite etc). Regular Review. Carer Support (Dementia Australia! Carer Gateway!). ACP early! Driving Safety (Austroads! Notify DLA if unfit).

Non-Pharmacological (MAINSTAY!): Cognitive Stimulation/Rehab, Exercise, Diet, Environmental Mods (Safety, Cues), Meaningful Activities.

Pharmacological (Cognitive Enhancers): Modest symptomatic benefit AD/PDD/DLB only. Specialist initiation/PBS criteria.

AChEIs (Donepezil etc): Mild-Mod AD. (+ PDD). Cholinergic SEs.

Memantine: Mod-Severe AD.

NOT for FTD/VaD.

BPSD Management:

Assess/Treat Triggers FIRST! (Pain! Infection! Constipation! Meds! Environment!). Rule out DELIRIUM.

Non-Pharm Strategies FIRST! (Carer support/DBMAS, Environment, Activities).

Pharm Rx = Last Resort (Severe sx/Risk harm). Specialist advice! Risperidone low dose short term (PBS criteria AD only). AVOID ANTIPSYCHOTICS in DLB! Avoid Benzos.

Differentials for Cognitive Decline / Dementia:

Delirium (Acute, Fluctuating, Inattention - Must exclude!).

Depression ('Pseudodementia').

Mild Cognitive Impairment (MCI) / Mild Neurocognitive Disorder (MND) (No functional impairment).

Medication Side Effects / Polypharmacy.

Sensory Impairment (Hearing/Vision loss).

Metabolic/Endocrine (Thyroid disease, B12 deficiency).

Structural Brain Lesion (Tumour, Subdural Haematoma).

Normal Pressure Hydrocephalus (NPH) (Gait/Incontinence/Cognition triad).

Red Flags / Urgent GP Actions for Dementia:

Acute onset / Rapidly progressive cognitive decline (weeks/months) -> Urgent Ix/Referral (R/o Delirium, CJD, Tumour).

Fluctuating level of consciousness/attention -> Suspect Delirium -> Find/Treat underlying cause urgently.

New focal neurological signs with cognitive decline -> R/o Stroke, Tumour, SDH -> Urgent Imaging/Referral.

Gait disturbance + Incontinence + Cognitive decline -> Consider NPH -> Referral.

Significant safety concerns (Driving, Wandering, Neglect, Carer stress) -> Prompt intervention (Family meeting, ACAT, ?Driving Notification).

Severe BPSD causing immediate risk -> Urgent Assessment + /- Psychogeriatric input / ED.

Action: Always exclude Delirium. Use Collateral Hx. Screen Cognition (MoCA/RUDAS) & Mood. Investigate Reversibles (Bloods, Meds). Arrange Imaging (MRI pref). Timely Specialist Referral.

Enhanced Physiological: Fine, fast postural. Triggers: Stress, Caffeine, Meds (Salbutamol!). Rx: Treat trigger.

Essential Tremor (ET): Most common path tremor. Bilateral Action tremor (Hands + /- Head/Voice). Improves with ETOH. Family Hx common. NO PD signs. Rx (if disabling): Propranolol OR Primidone.

Intention Tremor: Worsens approaching target -> Cerebellar Lesion.

Asterixis ('Flap'): NOT tremor. Lapses tone. Sign Metabolic Encephalopathy (Hepatic!).

Assessment: Hx, Exam (type, distribution, assoc signs?). Ix: TFTs etc. Refer Neuro if uncertain/PD/Cerebellar suspected.

Wilson Disease (Neuro Aspects)

Autosomal Recessive -> Copper overload -> Liver + Neuropsych Sx.

Neuro Sx (Young adults <50y): Parkinsonism, Dystonia, Dysarthria, Ataxia, Tremor.

Dx Keys: Kayser-Fleischer rings, ↓Caeruloplasmin, ↑Urine Copper.

GP Role: Consider if young + Liver/Movement/Psych disorder. Early Rx vital!

Differentials (Consider based on presentation):

MND/ALS (UMN + LMN signs).

Cervical Myelopathy/Radiculopathy (UMN + /-LMN mimic).

Restless Legs Syndrome (RLS).

Peripheral Neuropathy (mimics RLS sensory sx / MND weakness).

Parkinson's Disease (if parkinsonism).

Essential Tremor (if action tremor).

Drug-Induced Movement Disorder (Parkinsonism, Chorea, Akathisia, Tremor).

Functional Movement Disorder.

Red Flags / Urgent GP Actions:

MND/ALS: Progressive weakness + UMN & LMN signs. Dysphagia/Choking -> Urgent Speech Path! Respiratory Sx (Dyspnoea/Orthopnoea) -> Urgent Resp Ix/Referral (Assess NIV need!).

RLS: Severe refractory Sx, Suspected serious secondary cause. Augmentation on DAs.

Tremor: Assoc Neuro signs (Rigidity -> PD?; Ataxia -> Cerebellar?). Acute onset. Asterixis -> Urgent Ix for metabolic cause.

Wilson's: Any suspicion -> Urgent referral.

Action: Urgent Neurology referral for suspected MND. Check Ferritin for RLS. Differentiate Tremor type -> Rx ET / Refer PD/Cerebellar. Coordinate MND MDT care. Provide Support Org info (MND Aus). Address Driving Safety. Initiate ACP (esp MND). Urgent action for respiratory compromise (MND) or metabolic disturbance (Asterixis).

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Intracranial Neoplasms: Recognition and Referral in Primary Care

Brain Tumours: Primary (from CNS tissues) vs. Metastatic (from elsewhere - MORE COMMON in adults). Cause Sx via mass effect, oedema, ICP rise, focal damage. GP Role: Recognise Sx (often non-specific initially), ID Red Flags -> Urgent Ix/Referral (Neuro/Neurosurg/Neuro-Onc), Initial Symptom Mx (Seizure/ICP), Supportive/Palliative Care, MDT liaison.

General Principles

- Classification:
  - Primary: Gliomas (Astrocytoma/GBM, Oligo), Meningioma common adult. WHO Grade I-IV.
  - Metastatic: Lung > Breast > Melanoma > Renal > CRC commonest sources. Often multiple lesions.
- Risk Factors: Primary mostly unknown (Radiation exposure, Rare genetic syndromes [NF1/2 etc]). Prior Cancer -> Mets risk.
- Clinical Presentation (Variable - depends on Type/Location/Size):
  - Raised ICP Sx: Headache (worse morning/Valsalva, progressive), N/V, Altered LOC, Papilloedema! (Fundoscopy key), CN VI palsy.
  - Focal Deficits: Based on location (e.g., Frontal -> Personality Δ; Temporal -> Seizure/Memory; Parietal -> Sensory/Neglect; Occipital -> Vision; Cerebellum -> Ataxia).
  - Seizures: Common presentation (~30-50%). New adult onset -> Investigate!
- Red Flags:
  - New Seizure (Adult).
  - Progressive Focal Neurological Deficit.
  - Raised ICP Sx (HA worse morning/Valsalva, N/V, Papilloedema).
  - Unexplained Personality / Cognitive Change.
  - Papilloedema on fundoscopy.
  - Worsening / Different HA pattern (esp >50y).

Diagnosis & Initial Management

- Diagnosis (Specialist coord): Hx + Exam (incl Fundoscopy!).
  - Imaging: MRI Brain + Gadolinium = Investigation of Choice. CT Head (+ /- Contrast) useful urgently (r/o bleed/mass effect).
  - Biopsy / Histopathology = Definitive Dx: Determines Type/Grade + Molecular Markers (guides Rx/Px).
  - Other Ix: Staging Scans (if Mets suspected). EEG (seizures). LP usually CONTRAINDICATED if mass effect!
- Initial Management (GP/ED):
  - Urgent Referral / Admission: If Red Flags / Tumour suspected -> Urgent Neuro/Neurosurg discussion/transfer.
  - Manage Raised ICP / Oedema: Dexamethasone (e.g., 4-8mg BD + PPI). Start on specialist advice/imaging confirmation. Monitor SEs.
  - Seizure Management: Acute (Benzos if >5min). Start AED (Levetiracetam common 1st choice) if seizure occurred.
  - Driving Restrictions: APPLY! (Advise patient - Austroads guidelines). Specialist input needed.

Common Brain Tumour Types

Metastases: Most common adult brain tumour! Sources: Lung>Breast>Melanoma etc. Rx: Dex + RT (SRS pref if few; WBRT if many) + /- Surgery + /- Systemic Rx for primary.

Glioblastoma (GBM): Most common PRIMARY Malignant adult. Gr IV Astro. Poor prognosis. Rx: Surgery + RT + Temozolomide chemo.

Meningioma: Most common PRIMARY Benign adult. Dural based. Rx: Observe vs Surgery vs RT/SRS.

Oligodendroglioma: Often Seizures, Calcified. IDH mut + 1p/19q co-deletion = Better Px.

Pituitary Adenoma: Mass effect (Bitemporal Hemianopia!) + /- Hormone issues.

Vestibular Schwannoma: CPA tumour. Unilat Hearing Loss/Tinnitus. Bilateral = NF2.

Primary CNS Lymphoma: Rare. Immunocomp risk ↑. Rx: High Dose Methotrexate chemo.

Differentials (for Headache, Seizure, Focal Signs, Cognitive Change):

Stroke (Ischaemic / Haemorrhagic).

Brain Abscess / CNS Infection.

Subdural Haematoma (SDH) (esp chronic).

Vascular Malformation (AVM, Cavernoma).

Multiple Sclerosis (MS) / Inflammatory Lesion.

Epilepsy (Primary - after structural exclusion).

Severe Headache Disorder (Migraine etc - but check red flags!).

Metabolic Encephalopathy / Delirium.

Urgent GP Actions / Red Flags:

Action: Maintain high index of suspicion if Red Flags present. Urgent Neuroimaging (MRI + Gd preferred). Urgent ED referral / Neuro-Oncology discussion if tumour suspected/confirmed with concerning features. Start Dex (+ PPI) for oedema/ICP signs (after discussion). Start AED if seizure occurred. Address driving restrictions IMMEDIATELY. Provide patient/family support (Brain Tumour Alliance Australia - BTAA). Liaise with MDT.

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Neurocutaneous Syndromes and Related Genetic Disorders

Inherited disorders affecting neuroectoderm derivatives (Skin, CNS/PNS, Eyes) + /- other systems (tumours). Mostly Autosomal Dominant (AD). **GP Role:** Recognise features, Surveillance participation, Genetic counselling referral, Complication Mx, Coordinate MDT care, Link Support Orgs (CTF, TSA, VHL Alliance) & NDIS.

Neurofibromatoses & Schwannomatosis

Neurofibromatosis Type 1 (NF1): AD (NF1 gene). ~1:3000.

Dx Criteria (Need ≥2): ≥6 Café-au-lait macules (CALMs), ≥2 Neurofibromas (NFs) or 1 Plexiform NF, Axillary/Inguinal Freckling, Optic Pathway Glioma (OPG), ≥2 Lisch Nodules, Specific Bony Lesion, 1st degree relative with NF1.

Features: Learning difficulties common! Risk: Malignant Peripheral Nerve Sheath Tumour (MPNST from plexiform NF), OPG (<8-10y), HTN, Scoliosis.

Mx: MDT. Surveillance (Annual Ophth <8-10y, Skin, BP, Neuro, Dev). Selumetinib for specific plexiform NFs (PBS). Genetic counsel. Children's Tumour Foundation (CTF) Aus support.

Neurofibromatosis Type 2 (NF2): AD (NF2 gene). ~1:35k.

Hallmark: Bilateral Vestibular Schwannomas (VS) -> Hearing loss, Tinnitus, Balance issues (Teens/Young Adult onset).

Other: Meningiomas, Ependymomas, other Schwannomas. Juvenile cataracts.

Dx: Bilateral VS is diagnostic (MRI).

Mx: MDT (Specialised centre). MRI surveillance (Brain/Spine). Audiology vital. VS Rx: Observe/Surgery/Radiosurgery.? Bevacizumab. Genetic counsel. CTF Aus support.

Schwannomatosis (SWN): AD/Sporadic (SMARCB1/LZTR1 genes). NOT NF2.

Features: Multiple non-vestibular schwannomas. Chronic PAIN is major issue. + /- Meningiomas.

Dx: Multiple schwannomas WITHOUT bilateral VS; No NF2 mutation.

Mx: MDT focus on PAIN management. Surgery for symptomatic tumours. Genetic counsel complex. CTF Aus support.

Other Major Syndromes

Tuberous Sclerosis Complex (TSC): AD (TSC1/2). mTOR pathway defect. Multi-system **Hamartomas**.

Brain: Tubers, SEN, SEGA -> Epilepsy (Infantile Spasms!), ID/ASD common.

Skin: Ash-leaf spots, Angiofibromas, Shagreen patch, Ungual fibromas.

Kidney: AMLs (Bleed risk!). Lungs: LAM (Women). Heart: Rhabdomyomas.

Dx: Clinical criteria or Genetic test.

Mx: MDT. Lifelong surveillance (MRI Brain/Abdo etc). mTOR inhibitors (Everolimus - PBS listed) for SEGA/AML/LAM. TSA Aus support.

Sturge-Weber Syndrome (SWS): Somatic GNAQ mutation (Not inherited).

Triad: Facial Port-Wine Stain (V1), Leptomeningeal Angioma, Glaucoma.

Complications: Seizures, Stroke-like episodes, Dev delay.

Dx: Clinical + MRI Brain + Ophtho assessment.

Mx: MDT. Seizure/Glaucoma control. Laser for PWS.? Aspirin.

Von Hippel-Lindau (VHL): AD (VHL gene). Tumour predisposition.

CNS Haemangioblastomas (Cerebellum/Retina), Renal Cell Ca, Phaeochromocytomas, PNETs, ELSTs (Ear).

Dx: Clinical criteria or Genetic test.

Mx: MDT. Lifelong surveillance (MRI Brain/Spine/Abdo, Ophth, Metanephrines). Surgery. Belzutifan (HIF-2α inhib - PBS listed). VHL Alliance Aus support.

Otitis Media in Primary Care: AOM, OME, and CSOM

OM Spectrum: Middle ear inflammation. Common (esp children).
- AOM: Acute infection + MEE + Inflammation signs.
- OME: 'Glue Ear'. MEE without acute infection -> Hearing loss.
- CSOM: Chronic infection + Discharge + TM Perf.

GP Role: Accurate Dx (Otoscopy + /- Tympanometry), Guideline-based Rx (Abx stewardship!), Monitor complications, Refer Audiology/ENT.

Acute Otitis Media (AOM)

Dx Criteria (Needs All 3):
- Acute Onset Sx (<48h pain/irritable).
- Middle Ear Effusion (MEE) on Otoscopy (Bulging TM best sign; OR ↓Mobility/Air-fluid/Otorrhoea).
- TM Inflammation (Red OR Pain).

Risk Factors: Young, Day care, Dummy, Smoke exposure, Viral URTI. ATSI children higher risk/complications.

Pathogens: *S. pneumoniae, H. influenzae* (non-typeable), *M. catarrhalis*.

Features: Otalgia (Pain) key! Fever, Irritability. Otorrhoea if perf.

Management (Aus Guidelines):
- ANALGESIA FIRST! (Paracetamol / Ibuprofen regularly).
- Antibiotics SELECTIVELY:
  - Immediate Abx IF: <6m old; 6m-2y + Bilateral/Severe Sx; ANY age + Systemically unwell / Perf + Otorrhoea / Immunocomp/High Risk / ATSI child.
  - Consider Watchful Wait / Delayed Script IF: ≥2y, Mild/Mod Unilateral, reliable f/u. Review if no better 24-48h.
- Abx Choice: Amoxicillin (High dose) 1st line (5-10d). If fails -> Amox-Clav. Refer TG.

Complications: Perf, OME, CSOM, Mastoiditis (Urgent!), Facial Palsy, Meningitis (Rare).

Otitis Media with Effusion (OME / 'Glue Ear')

MEE WITHOUT acute infection. Due to Eustachian tube dysfunction.

Features: Often Asymptomatic! Conductive Hearing Loss ->? Speech/Language Delay, Learning issues. NO pain/fever.

Diagnosis: Otoscopy (Dull/Retracted TM, ↓Mobility, Air-fluid) + Tympanometry (Type B = Flat trace) confirms MEE. Audiometry confirms hearing loss.

Management:
- Watchful Waiting ('Active Monitoring') for 3 MONTHS standard initial approach.
- Audiology if persists ≥3m OR developmental concerns.
- Refer ENT IF: Persists ≥3m + Sig Bilateral Hearing Loss/Speech Delay; OR persists >6-12m; OR Recurrent AOM; OR TM retraction.
- Medical Rx (Abx, Steroids etc) = NOT EFFECTIVE!
- Surgery (ENT): Grommets (+ /- Adenoidectomy) if persistent + significant impact.

Chronic Suppurative Otitis Media (CSOM)

Chronic Discharge (>6wks) + TM Perforation. Usually painless. Conductive hearing loss.

Pathogens: Often Pseudomonas, Staph aureus.

Types:
- Tubotympanic ('Safe'): Central perf. Mucosal dz.

Atticoantral ('Unsafe'): Attic/Marginal perf -> CHOLESTEATOMA risk! (Keratin sac -> erodes bone -> Serious Complications!). -> Urgent ENT referral if suspected!

Diagnosis: Otoscopy (Perf + /- Cholesteatoma). Discharge Swab (M/C/S). Audiometry. CT scan if cholesteatoma/complications suspected.

Management (ENT):

Regular Aural Toilet (Clean debris!). Microsuction ideal.

Topical Antibiotic + /- Steroid Drops: Ciprofloxacin + /- Dex/HC = 1st line Aus.

Water Precautions. Oral Abx usually ineffective (unless complication).

Surgery: Often needed (Myringoplasty, Tympanoplasty, Mastoidectomy [for cholesteatoma]).

Differentials for Ear Symptoms (Pain/Loss/Discharge):

Otitis Externa (Canal inflamed, tragal/pinna tenderness).

Foreign Body / Impacted Cerumen.

Referred Pain (Dental, TMJ, Tonsillitis, Neck).

Eustachian Tube Dysfunction (without effusion).

Barotrauma.

Ramsay Hunt Syndrome (Shingles -> vesicles, facial palsy, pain).

Traumatic TM Perforation.

Malignancy (Rare - persistent pain/blood).

Red Flags / Urgent GP Actions:

Signs of Mastoiditis (Postauricular swelling/redness/pain) -> Urgent ENT/ED.

Facial Nerve Palsy with OM -> Urgent ENT/ED.

Vertigo / Severe SNHL with OM -> Suspect Labyrinthitis / Intracranial -> Urgent ENT/ED.

Signs Intracranial Complication (Severe HA, Altered LOC, Fever, Neck stiff, Focal signs) -> Urgent ED (000).

Suspected Cholesteatoma (Attic/Marginal perf, foul discharge, white debris) -> Urgent ENT referral.

Aboriginal or Torres Strait Islander child with AOM -> Lower threshold Abx, closer monitoring OME/CSOM.

Failure AOM Sx improve 48-72h -> Review Dx/Rx.

Persistent OME >3m + Hearing Loss/Speech Delay -> Audiology + ENT referral.

Action: Careful Otoscopy key. Analgesia for AOM crucial. Judicious Abx use. Objective tests (Tymp/Audio) for OME. Refer CSOM to ENT. Act urgently on Red Flags for complications. Educate parents. Follow ATSI guidelines.

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Otitis Externa (Including Malignant Otitis Externa)

OE ("Swimmer's Ear"): Inflammation/Infection of External Auditory Canal (EAC) skin. Common. Differentiate from Otitis Media. Recognise infectious vs. eczematous cause. Beware Malignant Otitis Externa (MOE) (Elderly Diabetic/Immunocomp) = EMERGENCY! GP Role: Dx, Rx, ID Red Flags, Urgent referral (MOE!).

Acute Otitis Externa (AOE)

Patho: ↓EAC protection (↓Wax, ↑pH, Moisture, Trauma [Cotton buds!]) -> Overgrowth.

Causes: Bacterial (~90%): Pseudomonas (Swimmer's!), Staph aureus. Fungal (~10%): Aspergillus (Black spores), Candida (White). Eczema common trigger/co-factor.

Risk Factors: Water exposure, Trauma, Hearing aids, Eczema, Immunocompromise.

Features: Otalgia (severe). Pruritus. Fullness. Hearing loss. Otorrhoea. KEY SIGN: Pain on Tragus/Pinna Pull!

Exam: Otoscopy: EAC Red/Swollen + /- Debris. TM often normal/poorly seen.

Diagnosis: Clinical (Sx + Signs). Swab M/C/S + Fungi if severe/recurrent/refractory. Exclude AOM + Perf.

Management (Aus TG):

- Pain Relief: Paracetamol + /- NSAIDs.? Opioids if severe.
- Aural Toilet: Crucial! Clean debris (Dry mop/Gentle irrigation/Microsuction).
- Topical Drops (Mainstay 7-10d):
  - Mild OE: Acetic acid + /- Steroid (Vosol HC®).
  - Mod-Severe Bacterial: Ciprofloxacin + /- Steroid (Ciproxin HC® / Ciloxan D®) = 1st Line.
  - Fungal (Otomycosis): Clean well + Clotrimazole 1% solution.
  - Eczematous: Topical Steroid.
- Ear Wick: If canal swollen shut.
- Keep Ear DRY! (Water Precautions).

Oral Antibiotics: NOT Routine! Only if spread beyond EAC / Immunocomp / MOE suspected.

Follow-up: ~1 week (sooner if severe/wick). If no better -> Reassess.

Prevention: Avoid trauma, Dry ears post-swim, Plugs, Manage eczema.

Malignant (Necrotizing) Otitis Externa (MOE)

Invasive Pseudomonas -> Osteomyelitis of EAC/Temporal Bone/Skull Base. Aggressive!

Risk Factors: ELDERLY DIABETIC! Immunocompromised.

Features: SEVERE, deep Otalgia (worse night). Purulent Otorrhoea. Granulation Tissue on EAC floor (Key!). CN Palsy (esp CN VII - Facial Nerve!) = Ominous sign.

Diagnosis: High suspicion + CT/MRI (bone erosion) + Cultures (Pseudomonas). ESR/CRP HIGH. Biopsy r/o malignancy.

Management: URGENT Hospital Admission + ENT/ID Consult!

- Prolonged IV Anti-Pseudomonal Abx (e.g., Cipro/Ceftazidime 6-8 + weeks).
- + /- Surgical Debridement.
- Strict Glucose Control! Monitor ESR/CRP.

Differentials for Ear Pain / Discharge:

Acute Otitis Media (AOM) + /- Perforation.

Foreign Body / Impacted Cerumen.

Referred Pain (Dental, TMJ, Throat, Neck).

Furunculosis (Boil in canal).

Ramsay Hunt Syndrome (Shingles - vesicles, facial palsy).
Malignancy of EAC / Temporal Bone (rare - persistent pain/bleeding).
Temporomandibular Joint (TMJ) Dysfunction.
Cellulitis of Pinna / Periauricular Skin.

Red Flags / Urgent GP Actions:

Suspected Malignant Otitis Externa (MOE) (Elderly diabetic + Severe pain + Granulation + /- CN VII Palsy) -> Urgent ENT Referral/ED.
Cellulitis spreading beyond EAC.
Severe OE in Immunocompromised.
Failure AOE response to Rx after ~1 week -> Reassess,? Swab,? Refer.
Cranial Nerve Palsy with ear infection.
Vesicles in EAC / Pinna (Consider Ramsay Hunt).
Severe pain out of proportion to signs.
Action: Recognise typical AOE. Treat: Analgesia + Aural Toilet + Topical Drops. High suspicion MOE in at-risk pts -> Urgent Referral! Oral Abx only if specific indications. Advise water precautions. Swab if needed.

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Infections of the Head, Neck, and Sinuses

Common GP presentations. Focus on:
- Ludwig's Angina: LIFE-THREATENING deep neck infection -> Airway risk!
- Acute Lymphadenitis: Node inflammation (Infectious/Reactive).
- Rhinosinusitis: Viral (common cold) vs. Bacterial (ABRS).

GP Role: Recognise urgency (Ludwig's!), Differentiate node causes, Judicious Abx use (Sinusitis!), Timely referral.

Ludwig's Angina

What: Rapid Bilateral Cellulitis floor of mouth (Submandibular/lingual/mental). Often Dental Source. Polymicrobial.

EMERGENCY Why?: AIRWAY OBSTRUCTION! (Tongue elevation/oedema).

Features: Acute onset Bilateral "woody" neck/submandibular swelling, ↑Tongue, Drooling, Dysphagia, Muffled voice. LATE: Stridor/Dyspnoea!

Dx: Clinical! CT Neck confirms extent (urgent, ?post-airway secure).

Mx: IMMEDIATE HOSPITAL (ED)!
- SECURE AIRWAY (PRIORITY!) - Anaesthetics/ENT/MaxFac.
- High-Dose IV ANTIBIOTICS (Broad: Benzpen + Metro + Gent / PipTaz etc).
- Surgical Drainage often needed.

Acute Lymphadenitis

What: Inflamed Lymph Node(s). Usually secondary to local infection.

Causes: Bacterial (Staph aureus/Strep pyogenes), Viral (URTI, EBV!), Specific (Cat Scratch, TB). DDx: Malignancy (persistent/hard/fixed node?), Autoimmune.

Features (Bacterial): Acute Tender, Enlarged node(s), often Unilateral Cervical. + /- Red/Warm. Fluctuance = Abscess. Look for source!

Dx: Clinical + /- Ultrasound (confirm node vs abscess). Biopsy if suspicious/persistent.

Rx (Bacterial): Treat source + Antibiotics (Flucloxacillin / Cephalexin) 7-10d. Review 48-72h. Drainage if Abscess.

Sinusitis (Rhinosinusitis)

Inflammation nose + sinuses. Acute (ARS) <12w; Chronic (CRS) >12w (-> ENT).

Aetiology (ARS): VIRAL (>90-98%!) - Self-limiting. Acute Bacterial (ABRS) rare (~0.5-2%).

Features: Nasal Blockage, Discharge (clear->purulent), Facial Pain/Pressure, ↓Smell.

Differentiating Viral vs Bacterial (ABRS criteria for Abx):
- Severe Onset: High Fever (≥39) + Purulent D/C OR Pain ≥3-4 days start.
- 'Double Sickening': Worsens after initial improvement (day 5-7 +).
- Persistent Sx: ≥10 days with NO improvement.

Dx: Clinical. Imaging NOT routine for ARS (only if complications/refractory).

Red Flags / Complications (RARE -> URGENT Referral/ED!):
- Orbital: Periorbital swelling/redness, Proptosis, ↓Vision, Ophthalmoplegia.
- Intracranial: Severe HA, Altered LOC, Focal Neuro signs, Meningism.
- Bony: Frontal Swelling (Pott's Puffy Tumour).

Management (ARS):
- Symptomatic Relief (Mainstay): Analgesia, Intranasal Saline Irrigation, Intranasal Corticosteroids (INCS). Topical Decongestants (<5 days!).

Laryngeal Disorders

Larynx (Voice box) issues common GP presentations (Hoarseness/Dysphonia, Chronic Cough). Focus:
- Laryngitis: Acute (Usually Viral/Self-limiting) vs Chronic (>3wks).
- Laryngopharyngeal Reflux (LPR): Gastric reflux -> Larynx/Pharynx irritation. Often NO heartburn.

GP Role: Dx common causes, Symptomatic Mx (Acute Laryngitis), ID Red Flags (Chronic Hoarseness -> Exclude Cancer!), Initiate LPR trial, Refer ENT/Speech Path.

Laryngitis

Acute Laryngitis (< 3 weeks):
- Cause: VIRAL (most common, post-URTI); Vocal Strain; Irritants. Bacterial rare (Epiglottitis = EMERGENCY!).
- Features: Hoarseness (key!), + /- URTI sx. Stridor = RED FLAG! -> Urgent ED!
- Dx: Clinical. Laryngoscopy usually NOT needed.
- Mx: SUPPORTIVE! Relative Voice Rest (AVOID whisper!), Hydration, Avoid Irritants (Smoke!). NO ANTIBIOTICS!

Chronic Laryngitis (> 3 weeks):
- REQUIRES ENT REFERRAL for LARYNGOSCOPY -> Exclude MALIGNANCY! (Esp Smoker/Drinker >40-50y).
- Causes: Reflux (LPR/GERD), Smoking, Chronic Vocal Misuse (->Nodules/Polyps), Laryngeal Cancer (SCC), Vocal Cord Palsy, Neuro dz.
- Mx: Treat underlying cause!

Laryngopharyngeal Reflux (LPR)

Patho: Gastric contents reflux past UES -> Irritates Larynx/Pharynx. Heartburn often ABSENT!

Features: Chronic/Vague Sx: Hoarseness, Chronic Cough, Globus (lump sensation), Throat Clearing. Also: Sore throat, Mild dysphagia.

Diagnosis: Primarily Clinical + /- Suggestive Laryngoscopy (ENT). Empirical Trial High Dose BD PPI (e.g., Pantoprazole 40mg BD) for ≥ 8-12 WEEKS = Key diagnostic/therapeutic step.

Management:
- Lifestyle/Diet Changes (Essential!): Weight loss, elevate bed head, avoid triggers (Fatty/Spicy/Acidic food, Caffeine, Alcohol), Avoid late meals. Stop smoking!
- Acid Suppression: BD PPI Trial (2-3 months) -> Taper if responds. + /- Alginates (Gaviscon®).
- Referral: ENT (Uncertain Dx, Red Flags, No PPI response), Gastro (GERD Sx?), Speech Path (Voice therapy).

Differentials for Hoarseness / Chronic Cough / Throat Symptoms:

Laryngitis (Acute Viral / Chronic).
Laryngopharyngeal Reflux (LPR) / GERD.
Benign Vocal Cord Lesions (Nodules, Polyps - Vocal misuse).
Laryngeal Malignancy (SCC) - MUST EXCLUDE!
Vocal Cord Palsy (Recurrent Laryngeal Nerve injury).
Neurological Disorder (Parkinson's, MND, Spasmodic Dysphonia).
Smoking / Chronic Irritant effect.
Asthma / Post-Nasal Drip (Common causes chronic cough).

Benign and Malignant Laryngeal Lesions

Larynx (Voice box) lesions commonly cause Hoarseness (Dysphonia). Range from Benign (Nodules, Polyps - vocal misuse/irritation) to RRP (HPV) to Malignancy (SCC). Persistent Hoarseness (>3wks) = RED FLAG -> Exclude Cancer! GP Role: Assess voice changes, ID risk factors, manage simple acute laryngitis, Timely ENT Referral (esp Chronic Hoarseness) for Laryngoscopy, Liaise MDT.

Benign Vocal Cord Lesions

(Often due to Phonotrauma / Chronic Irritation)

Nodules ('Singer's'): Bilateral, Mid-cord. Cause: Chronic Vocal Abuse. Sx: Hoarseness, Vocal fatigue. Rx: VOICE THERAPY (Mainstay!).

Polyps: Usually Unilateral, Mid-cord. Cause: Acute/Chronic Vocal Trauma/Irritation (Smoking!). Sx: Hoarseness (breathy/rough). Rx: Voice Therapy trial + /- SURGERY often needed.

Cysts: Unilateral, Submucosal. Sx: Hoarseness. Rx: Usually SURGERY.

Reinke's Oedema: Bilateral Diffuse Swelling. Cause: SMOKING! Sx: Deep, husky voice. Rx: STOP SMOKING! + /- Voice Therapy/Surgery later.

Contact Ulcer/Granuloma: POSTERIOR Larynx (Arytenoids). Cause: Reflux (LPR!) / Vocal Misuse / Intubation. Sx: Throat pain/Globus, Mild hoarseness. Rx: Aggressive PPI + Voice Therapy.

Recurrent Respiratory Papillomatosis (RRP)

Cause: HPV 6 & 11. Benign warty growths in Larynx. Recurrent.

Onset: Juvenile (<5y) OR Adult (20-40y). HPV Vaccine (Gardasil9) PREVENTS JoRRP!

Sx: Progressive Hoarseness = #1. + /- Stridor / Airway obstruction (esp kids!).

Dx: Laryngoscopy (ENT). Biopsy confirms.

Rx: NO CURE. Repeated Surgical Debulking = Mainstay.

Laryngeal Malignancy (SCC)

Type: Squamous Cell Carcinoma (SCC) >90%.

Risk Factors: SMOKING!! + ALCOHOL!! Age >50. M>F.

Clinical: PERSISTENT HOARSENESS > 3 WEEKS = CARDINAL SYMPTOM! -> URGENT ENT REFERRAL!

Other Sx: Persistent Sore Throat / Referred Otalgia, Dysphagia, Neck Lump (Nodes!), Stridor (Late!).

Dx: Laryngoscopy + Biopsy. Staging (CT/PET).

Management (MDT):

Early Glottic: Laser Surgery (TLM) OR Radiotherapy (RT). (Voice preserving).

Advanced: Chemoradiotherapy (CRT) OR Laryngectomy (Total = Permanent Stoma / Loss voice).

Differentials for Hoarseness / Throat Symptoms:

Acute Laryngitis (Viral/Strain).

Laryngopharyngeal Reflux (LPR) / GERD.

Benign Vocal Cord Lesions (Nodule, Polyp, Cyst, Granuloma).

Laryngeal Malignancy (SCC) - MUST EXCLUDE!

Vocal Cord Palsy (Nerve injury - Thyroid surgery?, Tumour?).

Neurological Disorder (Parkinson's, MND, Spasmodic Dysphonia).

Smoking effect / Chronic Irritant exposure.

Hypothyroidism.

Red Flags / Urgent GP Actions:

HOARSENESS > 3 WEEKS (esp >40-50y, smoker/drinker) -> Urgent ENT referral for Laryngoscopy.
STRIDOR -> Urgent ED Transfer / Call 000 (Airway compromise!).
Rapidly progressive symptoms.
Associated Haemoptysis.
Associated significant Dysphagia / Odynophagia.
Palpable Neck Mass.
Significant unexplained Weight Loss.
Referred Otalgia with normal ear exam + Laryngeal Sx.
Action: Take Hx (Voice use, Smoking, Alcohol, Reflux). REFER Hoarseness >3wks URGENTLY. Act immediately on STRIDOR. Initiate LPR trial if suspected. Provide voice care advice. Facilitate smoking/alcohol cessation.

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Obstetric Fundamentals: Terminology and Physiological Adaptations in Pregnancy

Antenatal care needs understanding of terms & profound physiological adaptations. **GP Role:** Confirm/Date pregnancy, Antenatal care (Shared), Interpret Ix (Pregnancy norms!), Manage Sx, ID pathology, Educate/Support.

Obstetric Terminology & Dating

Gravidity (G): Total pregnancies. Parity (P): Births ≥ 20 weeks. (G_ P_ notation).

Dating: First Trimester Ultrasound (CRL ~7-13 + 6wks) = MOST ACCURATE / Recommended Aus. Naegele's Rule (LMP + 9m + 7d) less reliable. FH @ Umbilicus ≈ 20wks.

Diagnosis of Pregnancy

Sx: Amenorrhoea, N/V, Fatigue (Suggestive).

Tests: Urine hCG (~missed period). Serum β-hCG (Sensitive; Monitor early viability [Doubles q48-72h IUP] /? Ectopic).

Ultrasound: Confirms IUP (vs Ectopic!), Dates (CRL), Viability (Heartbeat ~6wks).

Physiological Changes in Pregnancy

(Essential to differentiate normal from pathology)

Cardiovascular: ↑CO, ↓SVR, ↓BP (nadir mid-preg). Supine Hypotension (>20wks). Oedema common. Flow murmur common.

Haematological: ↑Plasma Vol >> ↑RBC Mass -> Physiological Anaemia (Hb <110/105/110). ↑Iron Needs! (Screen/Supplement). HYPERCOAGULABLE -> ↑VTE Risk (esp Postpartum!). ↑WCC normal.

Respiratory: ↑Tidal Vol -> Compensated Resp Alkalosis (↓PaCO2, ↓HCO3). Dyspnoea common. Nasal congestion.

Renal: ↑RBF/GFR -> ↓Creatinine/Urea (Use preg ranges!). Hydronephrosis. ↑UTI risk. Glucosuria common.

Gastrointestinal: Progesterone -> ↓LES tone (GERD common), ↓Motility (Constipation, N/V 1st tri). ↑Gallstone risk.

Musculoskeletal: Lordosis -> Back pain. Ligament Laxity -> Pelvic Girdle Pain.

Skin: Hyperpigmentation, Striae common.

Endocrine: ↑Hormones (hCG, E, P, hPL, Prolactin). hPL -> Insulin Resistance/GDM risk! Thyroid: ↑TBG -> ↑Total T4/T3 BUT Normal Free T4/T3 (use preg ranges). TSH low-normal 1st tri. ↑Iodine Need! (Supplement).

Differentials for Common Pregnancy Symptoms:

(Consider pathology vs. physiological change)

Hyperemesis Gravidarum (vs normal NVP).

Pre-eclampsia (vs physiological oedema / Chronic HTN).

Urinary Tract Infection (UTI) (vs normal urinary frequency).

Iron Deficiency Anaemia (vs physiological anaemia).

Pulmonary Embolism (PE) / Deep Vein Thrombosis (DVT) (vs physiological dyspnoea/leg oedema).

Gestational Diabetes Mellitus (GDM) (vs normal glucosuria / increased thirst).

Asthma Exacerbation (vs physiological dyspnoea).

Hypothyroidism (vs normal fatigue / requires pregnancy-specific TFT ranges).

Red Flags / Urgent GP Actions in Pregnancy:

Vaginal Bleeding + /- Pain -> Urgent assessment (R/o Ectopic/Miscarriage/APH).
Reduced Fetal Movements -> Urgent CTG/US assessment.
Symptoms/Signs of Pre-eclampsia (Severe HA, Visual Δ, Epigastric pain, HTN, Proteinuria) -> Urgent assessment/admission.
Symptoms of VTE (Unilat leg swelling/pain; Acute dyspnoea/chest pain) -> Urgent assessment/admission.
Symptoms of Preterm Labour (<37 weeks).
Suspected Ruptured Membranes.
Severe Nausea/Vomiting (Hyperemesis) -> Assess hydration + /- Admission.
Fever / Signs of Infection (UTI etc) -> Investigate/Treat promptly.
Action: Confirm IUP + Accurate dating (early US). Use pregnancy reference ranges for Ix. Screen/manage Anaemia (Iron!). Counsel re VTE risk. Screen GDM/Pre-eclampsia/Infections per guidelines. Provide lifestyle/supplement advice (Iodine/Folate). Promptly investigate/refer Red Flag symptoms.

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Antenatal Care and Prenatal Screening/Diagnostic Testing

Antenatal Care: Health supervision during pregnancy -> Optimise maternal/fetal outcomes. GP Role: Vital in shared care - Initial consult, Confirm/Date pregnancy, Risk assessment, Coordinate Screening (NHMRC guidelines), Manage common issues, Monitor, Lifestyle advice, Referrals.

Terminology & Dating

G/P: Gravidity (G) = Total pregnancies; Parity (P) = Births ≥ 20 weeks.
Dating: 1st Trimester Ultrasound (CRL ~7-13 + 6wks) = MOST ACCURATE / Recommended Aus.

Diagnosis of Pregnancy

Sx: Amenorrhoea, N/V etc (Suggestive).
Confirm: Urine hCG; Serum β-hCG; Ultrasound (Confirms IUP!, Dates, Viability).

Routine Antenatal Ix & Schedule (Aus NHMRC)

Booking (~8-12w):
- Bloods: FBC, Blood Group/Abs, Infections (Rubella, Syph, HIV, HBV, HCV). ?Hb Electrophoresis/Ferritin/Vit D/TSH.
- Urine: MSU M/C/S (Screen ASB!).
- Other: Offer CST/STI screen. Dating USS. Supplements: Folate + Iodine!

1st Tri Screen (~10-14w): Aneuploidy: Discuss cFTS vs NIPT. High risk -> Counsel + /- CVS/Amnio.
2nd Tri (~18-20w): Morphology Ultrasound.
Mid-Preg (~24-28w): GDM Screen (75g OGTT). Repeat FBC,? Blood Group Abs.
3rd Tri:
- Anti-D (@ 28w & 34w): ALL RhD Neg women (if Ab neg).
- GBS Screen (35-37w): Low Vag/Rectal Swab.

Nutrition, Exercise, Substances, Weight

Weight Gain: Based on pre-preg BMI (IOM).
Nutrition: Balanced diet. Routine Supplements: Folate + Iodine. Screen/Treat Iron def. Ensure Vit D ok. Food Safety crucial (Avoid Listeria risks, Raw meat/eggs, High Mercury fish).
Substances: NO Alcohol! Stop Smoking (NRT safer). Avoid Illicit Drugs. Limit Caffeine (<200mg/d).
Exercise: ~150min moderate activity/wk encouraged (unless C/I). Avoid high risk activities/supine >20wks.

Group B Streptococcus (GBS) Prophylaxis

Screen: Swab @ 35-37wks.
Offer Intrapartum Antibiotic Prophylaxis (IAP) IF: GBS + screen OR GBS Bacteriuria OR Prev GBS baby OR Unknown status + Risk factor (Preterm / ROM ≥18h / Fever).
Rx: IV Benzylpenicillin (1st line). Aim ≥4h cover pre-delivery.
No IAP IF: Planned CS pre-labour/intact membranes OR GBS Neg screen.

Prenatal Diagnostic Testing

Invasive tests (CVS ~11-14w; Amnio ≥15w) for definitive Dx post high-risk screen. Small miscarriage risk. Needs Genetic Counselling.

Differentials for Common Pregnancy Red Flags:

(Consider based on specific presentation)

Miscarriage / Ectopic Pregnancy (for PV bleeding/pain).
Placental Abruption / Placenta Praevia (APH causes).
Pre-eclampsia / HELLP Syndrome.
Gestational Diabetes (GDM) (implications/complications).
Urinary Tract Infection (UTI) / Pyelonephritis.
Thromboembolism (DVT/PE).
Fetal Growth Restriction (FGR) / Fetal Compromise.
Preterm Labour.

Red Flags / Urgent GP Actions in Antenatal Care:

Vaginal Bleeding + /- Pain -> Urgent Assessment (R/o Ectopic/Miscarriage/APH).
Reduced Fetal Movements -> Urgent assessment (CTG/US).
Symptoms/Signs of Pre-eclampsia (Severe HA, Visual Δ, Epigastric pain, HTN, Proteinuria) -> Urgent assessment/admission.
Symptoms suggestive of VTE (Unilat leg swelling/pain; Acute dyspnoea/chest pain) -> Urgent assessment/admission.
Symptoms of Preterm Labour (<37 weeks) -> Urgent assessment/admission.
Suspected Ruptured Membranes.
Severe Nausea/Vomiting (Hyperemesis) -> Assess hydration + /- Admission.
Fever / Signs of significant infection.
Action: Ensure accurate dating (early US!). Follow NHMRC screening. Provide key supplement/lifestyle advice. Use pregnancy reference ranges. Monitor BP/Urine/Weight/FH. Act urgently on Red Flags. Counsel re screening options. Administer Anti-D. Screen/Treat GBS per protocol. Timely specialist referrals vital.

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Teratogenic Exposures in Pregnancy

Teratology: Study of causes of birth defects (~3% baseline risk Aus). **Teratogens:** Agents increasing risk (Meds, Alcohol, Drugs, Infections, Radiation, Maternal conditions). **GP Role:** ID exposures, Pre-conception/Antenatal counselling, Med review, use reliable Aus resources, Minimise harm (esp organogenesis).

Principles of Teratology

Timing Critical:
- <2w post-conception: All-or-none.
- Wks 3-8 post-conception (5-10 LMP): ORGANOGENESIS = MAX RISK structural defects.
- Wk 9 (Fetal): Growth/Functional effects.

Dose-Response relationship. Genetic susceptibility varies.

Assessing Risk & Counselling (Aus)

Key Info Resources:
- TGA Pregnancy Categories (A, B1-3, C, D, X): Guide ONLY! Know limitations.
- MotherSafe (NSW - Nat access): Specialist Advice! Excellent resource.
- Therapeutic Guidelines (TG), AMH, RANZCOG guidelines.

Counselling:
- Pre-conception = IDEAL: Review meds/conditions, Optimise/Switch meds, Folate/Iodine, Lifestyle (NO Alcohol!).
- During Preg: Detail exposure (Timing!). Balanced risk info. Support decisions. Document!

Teratogenic Exposures

AEDs (Specialist Co-Mx!):
- Valproate = HIGHEST RISK (NTD/Neurodev!) -> AVOID! (Specialist Rx only).
- Carbamazepine/Phenytoin/Topiramate: Specific risks.
- Safer Options: Lamotrigine, Levetiracetam (preferred).
- Mgmt: Pre-conception plan! Aim Monotherapy lowest dose. High Dose Folic Acid (5mg) ESSENTIAL!

Anticoagulants:
- Warfarin: CONTRAINDICATED! Switch!
- Heparin/LMWH: SAFE.
- DOACs: Avoid. Switch LMWH.

Cardiovascular Drugs:
- ACEi / ARBs: CONTRAINDICATED! (esp 2nd/3rd tri). Stop!
- Safer HTN Rx: Methyldopa, Labetalol, Nifedipine.
- Statins: Stop.

Psychotropics (Perinatal Psych input recommended!):
- Lithium: Small ↑Ebstein's risk. Monitor.
- SSRIs: Low risk usually. Neonatal Adaptation Syndrome possible.
- Atyp Antipsychotics: Lower risk malformations vs others. Monitor maternal/neonatal SEs.
- Benzos: Avoid regular use.

Retinoids:
- Systemic Isotretinoin: MAJOR TERATOGEN (Cat X) - ABSOLUTELY CONTRAINDICATED! Stringent prevention needed.

Antibiotics (Refer TG!):

Safe: Penicillins, Cephalosporins, Macrolides (most).
Caution/Avoid: Trimethoprim (1st tri?), Sulfa/Nitrofurantoin (near term).
Contraindicated/Avoid: Tetracyclines, Aminoglycosides, Fluoroquinolones.

Immunosuppressants (Specialist Mx!): Methotrexate/Mycophenolate = CONTRAINDICATED pre-conception! AZA/HCQ often continued.

Non-Pharm Teratogens:
Alcohol: NO SAFE LEVEL! -> FASD risk. Advise Abstinence!
Smoking: Major adverse effects! STOP! (NRT safer).
Illicit Drugs: Variable risks. Advise cessation/referral.
Radiation: Diagnostic X-ray/CT = NEGLIGIBLE RISK. MRI/US safe.
Maternal Infections (TORCH etc): Vax pre-preg! Hygiene!
Maternal Conditions: Poorly controlled Diabetes/PKU/Thyroid etc increased risk. Optimise pre-conception!
Hyperthermia (>38.9°C early):? Small ↑NTD risk. Treat fever (Paracetamol).

Differentials for Congenital Abnormality / Adverse Outcome:

(Why avoiding teratogens is only part of the picture)
Chromosomal Abnormalities (e.g., Trisomies).
Single Gene Disorders (e.g., CF, Thalassaemia).
Multifactorial Inheritance (Gene + Environment - e.g., most NTDs, Clefts).
Maternal Illness (Unrelated to teratogen - e.g., GDM effects).
Intrauterine Infection (TORCH).
Substance Abuse Effects (overlaps teratogens).
Unknown Cause (~50% birth defects).
Uterine Constraint / Vascular Disruption.

Red Flags / Urgent GP Actions Re Teratogens:

Patient on HIGH-RISK Teratogen (Cat D/X) planning pregnancy / unexpectedly pregnant -> Urgent Specialist review to discuss/switch meds ASAP. Do NOT stop essential meds (e.g., AEDs) abruptly -> Liaise urgently.

Significant exposure to known teratogen during critical period (Wks 5-10 LMP) -> Counsel (MotherSafe!) + Refer Genetic Counselling/MFM.

Patient with chronic condition (Epilepsy, DM, Psych, Autoimmune) planning pregnancy -> Refer for Pre-conception counselling to optimise meds/control.

Previous child with birth defect / Family Hx -> Genetic Counselling referral.

Patient using alcohol/illicit drugs during pregnancy -> Counsel + Offer referral Drug/Alcohol services.

Action: Proactively review meds pre-conception/1st visit. Use reliable Aus resources (TGA, MotherSafe). Provide balanced risk counselling. Emphasise Folate/Iodine. Strongly advise NO ALCOHOL/SMOKING. Facilitate timely specialist input. Document discussions.

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Congenital and Perinatal Infections

Infections (Congenital/Intrapartum/Postpartum) -> Adverse Outcomes (Miscarriage, Defects, Disability, Sepsis). TORCH + pathogens key. **GP Role:** Prevention (Vax, Counselling), Antenatal Screening, Hygiene/Food safety advice, recognise signs -> Investigate/Refer (Obstetrics/Paeds/ID/Public Health).

TORCH Infections & Other Key Pathogens

Toxoplasmosis: Source: Raw meat/Cat faeces. Risk: Primary infection preg -> Congenital Triad (Chorioretinitis, Hydrocephalus, Intracranial Calcs). Mgmt: Prevention (Hygiene!). Routine screening NOT done Aus.

Other:

Syphilis: Transplacental -> Congenital Syphilis. Screening @ Booking Standard Aus! Rx: Penicillin for Mom prevents! Notifiable.

VZV (Chickenpox): <20wk infection -> Congenital Varicella Syndrome. Peripartum rash -> Neonatal Varicella (Severe!). Mgmt: Vax PRE-preg! VZIG if susceptible exposed preg. Aciclovir Rx maternal illness.

Parvovirus B19: Risk Fetal Anaemia/Hydrops if infected <20wks. Dx: Maternal serology if exposed. Rx: Monitor US + /- Intrauterine transfusion.

Listeria: Foodborne -> Miscarriage/Stillbirth/Neonatal Sepsis. Prevention: Food Safety Advice!

Zika: Mosquito/Sexual -> Congenital Zika Syndrome (Microcephaly!). Prevention: Travel advice!

Rubella: CRS Triad: Deafness + Cataracts + Cardiac defects. Risk highest <12wks. Prevention: MMR Vax Pre-preg/Postpartum! Check immunity booking. Rare Aus (Vax). Notifiable.

CMV: Commonest congenital infection! Via body fluids. SNHL = Major sequela! (Often delayed). Dx: Neonatal Urine/Saliva PCR <3wks. Prevention: Hygiene! No routine screening Aus. Rx: Ganciclovir (Symptomatic neonates only).

HSV: Intrapartum transmission risk (esp Primary genital HSV near term) -> Neonatal HSV (Serious!). Mgmt: Suppressive Aciclovir from 36wks if recurrent GH Hx. C-Section if active lesions @ labour. Urgent IV Aciclovir if Neonatal HSV.

HIV: MTCT risk <1% with: Maternal ART + Neonatal PEP + Avoid Breastfeeding (Aus). Screening @ Booking Standard Aus**!** Specialist co-Mx.

Differentials for Neonate with TORCH-like signs:

(e.g., IUGR, HSM, Jaundice, Rash, Microcephaly, Seizures)

Bacterial Sepsis (GBS, E. coli etc).

Congenital Heart Disease.

Metabolic Disorder (Inborn Error of Metabolism).

Chromosomal Abnormality / Genetic Syndrome.

Haemolytic Disease of the Newborn (ABO/Rh).

Hypoxic-Ischaemic Encephalopathy (HIE).

Neonatal Abstinence Syndrome (NAS) / Drug Withdrawal.

Neonatal Liver Disease (e.g., Biliary Atresia).

Red Flags / Urgent GP Actions:

Maternal exposure to significant infection (Rubella, Varicella, Toxo etc) -> Check immunity/Serology + /- Specialist advice/Prophylaxis.

Maternal fever / systemic illness during pregnancy -> Investigate cause, specialist input if concerned.

Positive Syphilis/HIV/Hep B/Hep C screen -> Urgent referral to Specialist service.

Early Pregnancy Complications: Miscarriage and Hyperemesis Gravidarum

Common GP issues:

- Miscarriage: Spontaneous loss <20wks (or <400g). Common (~15-20%).
- Hyperemesis Gravidarum (HG): Severe NVP -> Dehydration/Wt Loss.

GP Role: Diagnose, Differentiate types, Counsel Mx options, Psychosocial support, ID severity -> Urgent Referral (EPAS/ED), Follow-up.

Spontaneous Abortion (Miscarriage)

- Cause: Fetal Chromosomal Abnormalities (~50-60%) = Most common! Maternal age >35 risk factor.
- Types: Threatened (Bleed, Os Closed, Viable); Inevitable (Bleed + Pain, Os Open); Incomplete (RPOC); Complete (Empty uterus); Missed (Non-viable IUP, Os Closed); Septic (Infection -> EMERGENCY!).
- Dx: Hx + Exam (Speculum: Os Open/Closed?) + TV Ultrasound (KEY! Location/Viability/RPOC) + /- Serial βhCG. Blood Group/RhD.
- Mx (<12-14wks): Discuss options (Expectant vs Medical [Misoprostol] vs Surgical [MVA/D&C]). Refer Early Pregnancy Assessment Service (EPAS) ideal.
- Post-Miscarriage:
  - Anti-D Ig (625 IU IM): ALL RhD Negative women (if ≥6-8wks / Surgery / Heavy bleed). Within 72h.
  - Psychosocial Support: Acknowledge loss. Refer SANDS / Red Nose.
  - Recurrent (≥3): Specialist referral/workup.

Hyperemesis Gravidarum (HG)

- Severe NVP -> Wt Loss >5% + Dehydration + Ketonuria.
- Complications: Malnutrition, Lytes↓ (K!), Wernicke Encephalopathy (THIAMINE Def!) -> Neuro EMERGENCY!
- Dx: Clinical + Objective signs + Exclude other N/V causes (UTI, Gastro, Molar etc). Ix: Urine Ketones!, UEC (K +!), TSH. USS Essential (r/o Molar/Multiples).
- Mx (Stepwise): Hospital if severe!
  - Lifestyle/Diet: Small frequent low-fat meals.
  - Antiemetics (Safe in Preg):
    - 1st Line: Pyridoxine (B6) + /- Doxylamine (Restavit®).
    - 2nd Line: Metoclopramide OR Prochlorperazine (Stemetil®).
    - 3rd Line: Ondansetron (Counsel small cleft risk 1st tri?).
  - Hospital Rx: IV Fluids + Electrolytes. CRITICAL: Give THIAMINE (IV/IM) BEFORE Dextrose! IV Antiemetics.? Nutrition support. Steroids rare (specialist).
  - Psych support.

Differentials:

(For Early Pregnancy Bleeding / Severe NVP)

Ectopic Pregnancy (MUST EXCLUDE!)

Miscarriage (Threatened / Inevitable / Incomplete / Complete / Missed).

Molar Pregnancy.

Cervical/Vaginal cause bleeding (Polyp, Ectropion, Infection).

Urinary Tract Infection (UTI) (can cause N/V).

Gastroenteritis.

Hyperemesis Gravidarum (vs Normal NVP).
Other medical causes N/V (Hepatitis, Pancreatitis, DKA etc).

Red Flags / Urgent GP Actions:

Early Preg Bleeding:
- Haemodynamic instability / Peritonism -> Suspect Ruptured Ectopic -> Urgent ED (000).
- Significant pain (esp unilateral) -> High Ectopic suspicion -> Urgent TVUS + /- hCG / ED.
- Heavy bleeding -> Urgent assessment/ED.
- Fever/Offensive discharge/Tenderness -> Suspect Septic Miscarriage -> Urgent ED.

Severe NVP / HG:
- Signs severe dehydration/Wt loss >5-10%.
- Neurological signs (Confusion, Ataxia, Eye signs) -> Suspect Wernicke's -> Urgent ED + THIAMINE STAT!
- Inability to tolerate oral intake despite Rx.
- Electrolyte disturbance.

Action: Always exclude Ectopic! TVUS key. Assess stability. Refer EPAS/ED urgently if Ectopic/Heavy Bleed/Sepsis/Severe HG. Provide miscarriage support. Give Anti-D if Rh Neg. Give Thiamine BEFORE Dextrose in HG with IV fluids. Stepwise antiemetics for HG.

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Diabetes in Pregnancy

Diabetes poses risks mother/foetus. Includes:
- Pregestational DM: T1DM/T2DM existing before pregnancy.
- Gestational DM (GDM): Glucose intolerance onset during pregnancy.

Prevalence increasing Aus (GDM ~15-20%). Optimal glycaemic control vital! GP Role: ID risk, Screen (Aus guidelines), Diagnose GDM, Initial Mx/Shared Care, Pre-conception Care (Pre-existing DM crucial!), Postpartum follow-up (T2DM risk!).

Pregestational Diabetes (T1DM / T2DM)

Risks (Poor control): Congenital Malformations! Miscarriage, Stillbirth, Macrosomia, Preterm, Pre-eclampsia, Neonatal Hypoglycaemia/RDS.

PRE-CONCEPTION CARE (ESSENTIAL! MDT): Aim HbA1c <6.5-7.0% PRE-conception. HIGH Dose Folate (5mg). Review meds (Stop ACEi/ARB/Statin!). Assess complications.

Mx During Preg (Specialist MDT): Tight Glycaemic Control (Targets: F<5.3, 2hr<7.0). Frequent SMBG/CGM. Insulin needs ↑↑↑. Close Fetal monitoring. Delivery ~38-39wks often.

Postpartum: Insulin needs ↓↓ Dramatically!

Gestational Diabetes Mellitus (GDM)

Glucose intolerance onset during pregnancy. Usually resolves postpartum.

Risk Factors: Age>40, BMI>25, Ethnicity (Asian, ATSI etc), Family Hx, Prev GDM/Macrosomia, PCOS.

Screening (Aus ADIPS): Early if High Risk. ALL women -> 75g OGTT @ 24-28 weeks.

Diagnosis (ADIPS Criteria - Need ≥1): Fasting ≥ 5.1 OR 1-hr ≥ 10.0 OR 2-hr ≥ 8.5 mmol/L.

Management (MDT - GP often co-manages):
- Lifestyle: Diet (Dietitian!) + Exercise.
- SMBG: 4x daily usually (Use Targets).
- Pharmacotherapy (If targets unmet): Metformin (common 1st line drug Aus) OR Insulin.

Monitoring: Fetal Growth Scans (esp if on Meds). Deliver ~38-39wks if on Meds.

Postpartum (CRUCIAL GP Role): STOP GDM meds! Screen baby BGLs. Screen Mother with 75g OGTT @ 6-12 weeks postpartum. HIGH future T2DM risk (>50%) -> Counsel Lifestyle + Regular lifelong T2DM screening (q1-3y).

Differentials:

(For Hyperglycaemia in pregnancy / GDM risk factors/complications)

Undiagnosed Pregestational DM (T1/T2).

Transient Hyperglycaemia (Stress, Infection, Steroids).

Impaired Glucose Tolerance (IGT) (on postpartum screen).

Polycystic Ovary Syndrome (PCOS) (risk factor).

Medication-induced hyperglycaemia (esp Corticosteroids).

Normal Pregnancy physiological changes mimicking symptoms.

Other causes Macrosomia (Non-diabetic).

Non-diabetic Renal Glycosuria.

Red Flags / Urgent GP Actions:

Newly diagnosed DM (not GDM) during pregnancy -> Urgent Endocrine/Specialist referral.

Known T1DM/T2DM planning pregnancy -> Urgent referral for Pre-conception care. Ensure 5mg Folate.

Poor glycaemic control during pregnancy -> Escalate care / Refer specialist.

Symptoms/Signs of DKA -> Urgent ED.

Hypertensive Disease in Pregnancy

HDP common (~10% Aus preg), major maternal/perinatal risk. Includes:

- Chronic HTN: Pre-existing / <20wks / persists >12wks postpartum.
- Gestational HTN (GHTN): New HTN ≥140/90 after 20wks, no other PET features.
- Preeclampsia (PET): New HTN ≥140/90 after 20wks + ≥1 New Organ Dysfunction (Renal [Proteinuria/↑Cr], Haem [↓Plt/Haemolysis], Liver [↑LFTs/Pain], Neuro [HA/Visual/Seizure], FGR).
- Eclampsia: PET + Seizures (Emergency!).
- HELLP: Haemolysis, Elevated Liver enzymes, Low Platelets (Emergency!).

GP Role: ID risk, Screen, Manage Chronic HTN (shared care), Start Aspirin prevention, ID PET Warning Signs -> Urgent action, Postpartum F/U (CVD risk!).

Risk Factors & PET Prevention

- High Risk (Needs Aspirin): Prev PET, CKD, Autoimmune, DM, Chronic HTN.
- Moderate Risk (Aspirin if ≥2): Nulliparity, Age≥40, BMI≥35, Fam Hx, Multiples.
- Prevention: Low Dose Aspirin (100-150mg nocte) from 12wks (ideally <16wks) to ~36wks if High Risk OR ≥2 Mod Risk factors.

Management

- Goal: Prevent severe maternal HTN/complications, safe delivery. DELIVERY = Cure PET/Eclampsia. MDT approach.
- Chronic HTN: Pre-conception: STOP ACEi/ARB! Aim BP <140/90. High Dose Folate (5mg). Start Aspirin. During Preg: Target BP <140/90. Safe Meds: Labetalol, Nifedipine (SR), Methyldopa. Monitor for Superimposed PET.
- GHTN: Close surveillance (BP, Urine PCR, PET Sx). Start Meds if BP persists ≥140/90. Deliver ~Term.
- Preeclampsia (PET): Specialist Obstetric Care! Often Admission.
  - PET without Severe Features: Monitor closely. Antihypertensives. Deliver ~37 weeks.
  - PET with Severe Features: Hospital! Stabilise + Expedite Delivery.
    - Urgent BP Control (if ≥160/110): Aim SBP 130-150/DBP 80-100. IV Labetalol / Oral Nifedipine / IV Hydralazine.
    - Seizure Prophylaxis/Rx: IV Magnesium Sulfate (MgSO4) = Drug of choice! MONITOR TOXICITY (Loss reflexes, ↓RR). Antidote=Calcium Gluconate.
    - Delivery: ≥37wks -> Deliver. 34-37wks -> Deliver post stabilisation. <34wks -> Corticosteroids + Deliver if unstable / ~34wks.
- HELLP / Eclampsia: Emergencies! Stabilise (BP control, MgSO4) -> EXPEDITE DELIVERY.

Postpartum Management & Long-Term Implications

- Postpartum: Monitor BP closely (can peak Day 3-6). Continue/adjust meds. GHTN/PET usually resolves <6-12wks.
- Long-Term: HDP ↑↑ Lifetime CVD Risk! (Chronic HTN, IHD, Stroke).
- GP Role: Counsel re long-term risk. Annual CVD risk screen (BP, Lipids, Gluc/HbA1c). Lifestyle advice!

Differentials:

(For HTN in pregnancy / PET-like symptoms)

- Chronic Hypertension (Essential/Secondary).
- Gestational Hypertension.
- Preeclampsia / HELLP / Eclampsia.
- White Coat Hypertension.
- Renal Disease (Glomerulonephritis/CKD).

Migraine (for Headache/Visual Sx).
Gallbladder Disease / Gastritis / Hepatitis (for RUQ/Epigastric Pain).
Anxiety.

Red Flags / Urgent GP Actions:

New onset BP ≥ 140/90 after 20 weeks -> Assess for PET features + Increase surveillance.
BP ≥ 160/110 mmHg -> Urgent assessment/admission for BP control.
Symptoms/Signs of Severe PET (Severe HA, Visual Δ, RUQ/Epigastric pain, rapid swelling) -> Urgent ED / Obstetric assessment.
Suspected HELLP / Eclampsia -> Urgent ED (000).
Known Chronic HTN pre-pregnancy -> Ensure safe meds + Aspirin prophylaxis started.
Action: Screen BP/Urine protein regularly. Identify women needing Aspirin (start 12-<16wks). Recognise PET warning signs -> Urgent referral/admission. Ensure safe antihypertensives. Emphasise Postpartum BP check & Long-term CVD risk counselling. Facilitate pre-conception care.

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Common Complications of Pregnancy: Bleeding, Growth Issues, and Multiples

Early/Late pregnancy complications pose risks. Includes: Early Bleeding (Ectopic/GTD), APH (>20w - Abruption/Praevia/Vasa Praevia/Accreta), Fetal Growth (FGR/Macrosomia), Fluid (Poly), Multiples. GP Role: Recognise Sx, Initial Ix, Initial Mx/Support, Timely Referral (EPAS/Obstetrics/MFM/ED), Ongoing care coordination.

Early Pregnancy Bleeding & Related Issues (<20w)

Ectopic Pregnancy: Implantation outside uterus (Tube!). Life-threatening if ruptures! Sx: Pain (unilat.) + /- Bleed + /- Amenorrhoea. Shoulder tip pain=Rupture! Dx: Serial βhCG + TVUS (Empty uterus + hCG >1500 OR Adnexal mass). Mx: Urgent EPAS/Gynae/ED! Ruptured -> EMERGENCY SURGERY! Unruptured -> Methotrexate OR Surgery. Anti-D if Rh Neg!

Gestational Trophoblastic Disease (GTD - Molar Preg): Abnormal placental proliferation. Sx: 1st Tri Bleed, Uterus Large, hCG VERY HIGH, Severe HG. Dx: USS ('Snowstorm'). Mx: Urgent Gynae! Suction Curettage -> Serial hCG follow-up (GTD Registry!) + Contraception (~6m). Anti-D if Rh Neg.

Antepartum Haemorrhage (APH) (>20w)

Urgent Assessment Needed! ABCs! IV access. Estimate loss. Abdo exam (Tone?). FHR/CTG. Speculum. !!NO VE until Praevia Excluded by USS!! Ix: FBC, G&H/X-match, Coags. USS key.

Placental Abruption: Premature separation. PAINFUL bleed + Tender/Tense Uterus + /- Fetal Distress. Clinical Dx (USS poor). Rx: Obstetric Emergency! Resuscitate -> Urgent Delivery.

Placenta Praevia: Placenta covers/near os. PAINLESS Bright Red Bleed. Uterus Soft. Dx: USS. Rx: Conservative vs Planned C-Section (~37-38wks). Risk Accreta!

Vasa Praevia: Fetal vessels over os. Risk Rupture @ ROM -> Fetal Exsanguination! Sx: Bleed at ROM + Fetal Bradycardia. Rx: Planned C/S (~34-36wks). Emergency C/S if acute!

Placenta Accreta Spectrum (PAS): Abnormal invasion into uterine wall. Risk: Prior C-Section! -> Massive PPH risk. Dx: Antenatal US/MRI. Rx: Planned Caesarean Hysterectomy @ Tertiary Centre (~34-36w).

Disorders of Fetal Growth & Amniotic Fluid *(Part letter updated to F)*

Multiple Gestation: ↑Risks. Chronicity (Early US!) = Key! MC twins high risk (TTTS etc) -> Specialist MFM care/Surveillance.

Fetal Growth Restriction (FGR): EFW<10th + Pathology? Causes: Placental Insufficiency vs Fetal factors. Monitor: Growth USS + Umbilical Artery Dopplers! -> MFM care -> Timing delivery critical.

Fetal Macrosomia: EFW>90-95th/>4.5kg. Cause: Maternal DM! Risk: Shoulder Dystocia! Rx: Optimise GDM. Consider Elective C/S if very large.

Polyhydramnios (AFI≥24/SDP≥8): Causes: Maternal DM, Fetal Anomalies (GI Atresia), Idiopathic. Risk: Preterm labour, Cord prolapse, PPH. Rx: Treat cause? Amnioreduction if severe sx.

Differentials (Consider based on presentation):

Ectopic Pregnancy.
Miscarriage (Threatened / Inevitable / Incomplete etc).
Placental Abruption.
Placenta Praevia.

Vasa Praevia.
Fetal Growth Restriction (Pathological) vs. Constitutionally Small Baby.
Local Genital Tract Bleeding (Cervical/Vaginal).
Labour ('Show').

Red Flags / Urgent GP Actions:

Early Preg Bleeding + Haemodynamic instability / Peritonism / Shoulder tip pain -> Suspect Ruptured Ectopic -> Call 000.
Any APH (>20wks) -> Urgent Obstetric assessment (usually hospital).
APH + Pain/Tender Uterus -> Suspect Abruption -> Urgent ED/Obstetrics.
APH + Fetal Compromise -> Urgent Obstetric assessment/ED.
Painless APH -> Suspect Praevia -> Confirm placental location with USS before VE.
Bleeding with ROM -> Suspect Vasa Praevia -> Obstetric Emergency!
Fundal height discrepancy -> Ultrasound assessment.
Reduced fetal movements -> Urgent assessment (CTG/US).
Action: Exclude Ectopic in early bleeding/pain! NO VE in APH until Praevia excluded! Facilitate urgent referral/transfer for emergencies. Arrange timely USS. Coordinate care (EPAS/Obstetrics/MFM). Administer Anti-D if Rh Neg & indicated. Provide support.

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Gynaecological Emergencies and Infections

Prompt recognition and management are crucial for PID, ovarian torsion, paediatric vaginal discharge, and TSS. GPs play a vital role in diagnosis, initial management, and urgent referrals.

Pelvic Inflammatory Disease (PID)

Upper genital tract infection (endometritis, salpingitis + /- TOA).

Aetiology: Ascending infection, often STIs (Chlamydia, Gonorrhoea, Mycoplasma genitalium) + endogenous bacteria.

Risk Factors: Young age, multiple partners, new partner, inconsistent condoms, prior STI/PID, uterine instrumentation, BV, douching.

Clinical: Bilateral lower abdominal/pelvic pain + cervical motion, adnexal, or uterine tenderness. + /- Discharge, bleeding, fever. Can be subtle.

Diagnosis: Clinical suspicion. Exclude ectopic pregnancy (βhCG). Swabs for NAAT (GC/CT/M.gen). Pelvic USS if uncertain diagnosis, severe symptoms,? TOA.

Management: Empirical outpatient Rx (mild-mod): Ceftriaxone 500mg IM STAT + Doxycycline 100mg PO BD 14d + Metronidazole 400mg PO BD 14d. Review 48-72h. Admit if severe, pregnant,? TOA, unable to tolerate oral meds, failed outpatient Rx. Treat all partners within 6 months. Follow-up, consider test of cure.

Complications: Infertility, ectopic pregnancy, chronic pelvic pain.

Ovarian Torsion

Twisting of ovary/tube, compromising blood supply. Gynaecological emergency.

Risk Factors: Ovarian mass/cyst (>5cm), pregnancy, ovulation induction.

Clinical: Sudden severe unilateral pelvic pain + nausea/vomiting. Adnexal tenderness + /- mass.

Diagnosis: High suspicion. Exclude ectopic (βhCG). Pelvic USS Doppler: Enlarged oedematous ovary + /- reduced/absent flow (flow present does not rule out).

Management: URGENT Gynae referral. Laparoscopy for detorsion (if viable) + /- cystectomy/oophoropexy or salpingo-oophorectomy (if necrotic). Time critical (<6-8 hours).

Paediatric Vulvovaginitis / Vaginal Discharge

Common Causes: Non-specific (hygiene, irritants, threadworms), foreign body (foul/bloody discharge), Group A Strep.

Red Flags: STIs (consider sexual abuse - mandatory reporting), urogenital tumours (rare).

Assessment: Careful history (hygiene, irritants, threadworm symptoms, CSA risk). Gentle external inspection. Urine dipstick. Vulval swab M/C/S + /- STI NAAT if concern. Sticky tape test for threadworms.

Management: Hygiene advice, treat threadworms/GAS if indicated. Foreign body removal. Refer persistent/recurrent/uncertain cases, suspicion of CSA/rare pathology.

Toxic Shock Syndrome (TSS)

Rare, life-threatening toxin-mediated illness (Staph aureus or Strep pyogenes).

Risk Factors: Tampon use (menstrual TSS), surgical/wound infections (non-menstrual TSS).

Clinical: Rapid onset high fever, hypotension, diffuse sunburn-like rash followed by desquamation (palms/soles). Multisystem involvement (GI, muscular, mucous membranes, renal, hepatic, haematologic, CNS).

- *Example:* Avoid repeating information within the stem, as seen in a case of a female patient with a history suggestive of endometriosis; this will never score marks.
- *Example:* In the case of a chronic haematological condition, identify additional information in the patient's history to identify possible diagnoses, not just repeat the given information.
- *Example:* When asked for history to establish the diagnosis, as seen in a case of an elderly female patient with suspected cardiac failure, responding with the patient's age or foot swelling if already mentioned in the scenario will not gain marks.

Respond Directly to the Question Asked:

Ensure your answers directly address the specific question posed by the examiner. Read the question carefully multiple times to understand exactly what is being asked and within what parameters (e.g., psychological vs. physical triggers, non-pharmacological management, specific findings vs. diagnoses). Avoid providing information not requested or misinterpreting the question.

- *Example:* When asked for psychological triggers for chronic pain, avoid providing physical triggers. Similarly, give non-pharmacological answers when requested, not pharmacological ones.
- *Example:* When the question is assessing non-pharmacological management actions, avoid providing pharmacological management actions, as seen in a case of a woman with a complex past medical history presenting for routine prescriptions.
- *Example:* Ensure you answer the question as written, as seen in a case of an adult male requesting a second opinion for rhinosinusitis, and in a case of an elderly female patient complaining of urge incontinence. For the rhinosinusitis case, this included demonstrating responsibility for arranging onward clinical care and managing conduct directly, rather than just identifying an organic cause.
- *Example:* When asked for symptoms rather than diagnoses, as seen in a case of a male patient presenting with symptoms suggestive of chronic rhinosinusitis, provide symptoms.
- *Example:* When asked for the immediate investigations required, select investigations appropriate at the initial presentation, not those appropriate but required later.
- *Example:* Ensure you answer the question given and not provide examination findings or descriptors of lesions when asked about management, as seen in a case of dermatological and rheumatological issues.
- *Example:* When a question asks for lifestyle factors contributing to uncontrolled hypertension, providing pharmacological causes will not score.
- *Example:* Ensure you answer the question exactly as asked, as seen in a case of an elderly male patient with multiple symptoms asked for diagnoses other than hearing loss, and in a case of an elderly patient with lower thoracic midline pain asked for non-pharmacological treatment. A significant number of errors resulted from misreading these questions.
- *Example:* When asked for the specific abnormal findings on the ECG, provide the specific abnormalities shown, including the leads, as seen in a case of a young adult male presenting with acute onset of anterior chest pain, not just diagnoses.
- *Example:* When asked for specific examination findings for one condition, as seen in a case of an elderly man with dementia attending for driving licence medical asked for examination findings, avoid listing generic examinations or findings for unrelated conditions like Parkinson's disease.
- *Example:* When asked for a diagnosis rather than results or investigations, as seen in a case of a middle-aged diabetic patient who presents for routine diabetic review, provide the diagnosis.

- *Example:* Ensure you read the question correctly, as seen in a case of a female patient with a six-week-old baby asked for management/advice on feeding.

Contextualise for General Practice: Frame your management and approach within the context of a general practice environment, considering what is appropriate and feasible in this setting (e.g., immediate management, urgency of referral, scope of practice). Demonstrate insight into the urgency of referrals where appropriate.

  - *Example:* In a case of suspected deep venous thrombosis in an elderly female patient in a residential facility, appreciate that initiating GP-based treatments with anticoagulants is not appropriate and urgent input from secondary care is required.
  - *Example:* Avoid providing answers that are emergency-department-focused, such as attempting to obtain intravenous access, which is not appropriate in a general practice environment, when managing a rapidly deteriorating child.
  - *Example:* In a case of a 2-year-old boy with a persistent cough possibly due to an inhaled foreign body, demonstrate insight into the urgency of the referral; a simple answer of "refer" would not score marks.
  - *Example:* When identifying skin lesions, avoid generic terms such as 'reassure' or 'excise' with no details if the question is asking for specific management in a general practice context.