CAASA

The Cuban Alumni Association of South Africa

Evidence-Based Internal Medicine Study & Conduct Guide for NMFC students

2nd Edition

2025

Foreword & Acknowledgements

Dear Reader,

It is with immense pride and dedication that I present this study guide tailored for South African medical students who have trained in Cuba and are now returning to complete their medical degree in South African universities.

This guide has been meticulously designed as a foundational resource to bridge the gap between the Cuban medical curriculum and the South African healthcare context, with a specific focus on Internal Medicine. The aim is to equip you with practical knowledge and clinical insights that will enhance your ability to manage common and complex medical conditions within the unique challenges of our healthcare system. Each section has been thoughtfully crafted to cover essential aspects of Internal Medicine, from diagnostic approaches to management protocols and evidence-based practices. This guide integrates key principles from the South African healthcare framework, ensuring relevance to our settings, including district hospitals, tertiary institutions, and primary care clinics.

While this guide provides a solid foundation, it is by no means exhaustive. Internal Medicine is a vast and ever-evolving field, and I strongly encourage further in-depth study and active engagement with senior clinicians to deepen your understanding. The content here is designed to complement your clinical rotation and academic learning, fostering confidence as you transition into the South African medical system. This study guide reflects our shared commitment to excellence in patient care and medical education. I trust that it will serve as a valuable tool in your journey to becoming a skilled and compassionate clinician in South Africa.

I would like to extend my gratitude to the many mentors, colleagues, and students who contributed to the creation of this guide. Their feedback and insights have been invaluable in shaping the content. I also acknowledge the contributions of the Cuban medical education system, which has instilled a robust foundation in primary healthcare and preventive medicine among its graduates.

Thank you for your dedication to advancing the field of medicine and for your commitment to improving the health and wellbeing of our communities.

Dr SD Ntshalintshali
MBChB (ISCM-VC Cuba, UKZN), FCP(SA), MMed Clin Immunol (UKZN), Rheum Cert Phys (SA), Rheum Cert (EULAR), MPhil Rheum (Stell), MRCP Rheum SCE (UK), PhD Medicine candidate (UKZN)
Associate Editor, African Journal of Rheumatology
Adjunct Lecturer of Rheumatology, University of KwaZulu Natal
Head of Rheumatology, Greys Hospital
KZN Department of Health
Pietermaritzburg
South Africa

Content:

Section 1: Clinical Practice.

Section 2: Clerking and Clinical Presentation.

Section 3: Principles of Resuscitation.

Section 4: Common Medical cases in South Africa

Section 5: Medical Emergencies

SECTION 1: CLINICAL PRACTICE

1.1 Professionalism

- South African medical staff and students adhere to the **Batho Pele Principles** and the **Patient's Rights Charter** in practicing their profession.
- Punctuality should be maintained at all times.
- All students should wear appropriate clothing that reflects professionalism.
- Name badges must be worn at all times for clear identification.
- Courtesy and common sense should be exhibited in all interactions with patients, relatives, and colleagues, ensuring respect and dignity.

1.2 Informed Consent

- Informed consent includes verbal and written consent for history taking, physical examinations, and any procedures.
- Consent must be obtained from a lucid patient aged 16 years or older for medical purposes, or 18 years and older for surgical purposes. For minors or mentally impaired individuals, consent should be obtained from a parent or guardian. In their absence, the senior medical authority should be consulted.
- Verbal consents should be documented in the patient's file (history and progress notes).
- Written consents must be completed on the appropriate form, signed by the patient and the person obtaining consent, and witnessed by two separate individuals.

1.3 Appropriate Documentation

Documentation is a cornerstone of safe and effective clinical practice. Adhere to the following guidelines:

- Write legibly using black ink to ensure readability.
- Include the patient's name, date of birth, and file number at the top of each page.
- Recognize that the patient's file is a legal document and hospital property.
- Ensure all entries are dated, timed, and signed in a way that identifies the writer.
- Use the provided admission proforma for all admissions.
- Maintain chronological order in notes; clearly indicate if writing in retrospect.
- Provide clear and concise progress notes for both inpatients and outpatients.
- Justify clinical decisions by clearly documenting reasoning.
- Correct errors by crossing out with a single line, writing the corrected entry, and signing and dating the change.
- Record details of discussions with patients or relatives, including key information and names of attendees.
- Document unexpected events and actions taken in response.
- Always remember that documentation reflects your clinical thought process for others to read and understand.

1.4 Appropriate Prescribing

Although students are prohibited from prescribing medication, they should familiarize themselves with proper prescribing practices:

- Complete patient details on each prescription sheet.
- Indicate any known allergies.
- Write drug names generically using block letters and black ink.
- Clearly specify the dose, frequency, route of administration (e.g., IM, IV, SC), and start/stop dates.
- Ensure each prescription is signed with the prescriber's name, rank, and registration number or by using a personalized stamp.
- Indicate date and signature when discontinuing medications.
- Follow the same rules for discharge medications (TTO) and outpatient prescriptions.

1.5 Talking with Patients and Relatives

Effective communication is critical in building trust and ensuring patient-centred care:

- Maintain confidentiality at all times, except in cases of legal obligations (e.g., patient's impaired consciousness, life-threatening diseases, patient consent, or court orders).
- Be honest and open during discussions.
- Admit if you do not know the answer to a question and seek advice from senior staff when uncertain.
- Document all details of interviews with patients or relatives in the case notes, including key information and those present during discussions.
- Engage relatives early to gather important medical and social information, particularly for critically ill or confused patients.

1.6 Patient Death

If a patient's death is suspected:

- Death must be confirmed by a doctor within 15 minutes of the event, including a review of brainstem reflexes.
- Relatives should be contacted by nursing staff within 30 minutes to discuss next steps (e.g., mortuary transfer or body collection).
- Offer bereavement counselling when indicated.
- Issue death certificates within 24 hours for natural deaths.
- Refer unnatural deaths for postmortem examination; defer issuing death certificates.
- Offer families postmortem options for unclear natural deaths or those that pose potential risks to others.
- Notify the medical registrar or senior medical officer for all unexpected deaths.
- Cancel all patient appointments and referrals following death confirmation.

1.7 Organ and Tissue Donation

Students may not participate directly in organ or tissue donation but should understand key principles:

- Organ donation typically occurs after confirmation of brainstem death or cardio-respiratory arrest in intensive care settings.

- Tissue donation is possible even after cardio-respiratory death due to lower metabolic requirements.
- All potential donors should be referred to the local transplant or tissue donation coordinator promptly.

1.8 Discharges and Discharge Summaries

- Once discharge is decided, initiate the process immediately.
- Discharge summaries serve as medico-legal documents and must be completed accurately.
- Summaries should include the main diagnosis, concurrent diagnoses, major procedures, discharge medications, dietary advice, and follow-up plans.

References

1. Department of Health, South Africa. Batho Pele Principles. Available at: www.gov.za.
2. Health Professions Council of South Africa. Guidelines for Good Practice in the Health Care Professions. Pretoria: HPCSA; 2023.
3. World Medical Association. Declaration of Helsinki: Ethical Principles for Medical Research Involving Human Subjects. JAMA. 2013;310(20):2191-4.
4. South African Medical Association. Patient’s Rights Charter. Cape Town: SAMA; 2022.
5. Medical Protection Society. Medical Records: Keeping Them Right. Johannesburg: MPS; 2023.
6. Organ Donor Foundation of South Africa. Guidelines for Organ and Tissue Donation. Cape Town: ODFSA; 2023.

SECTION 2: CLERKING AND CLINICAL PRESENTATION

1. Introduction

Clerking and clinical case presentation form the foundation of medical practice. They are essential skills for acquiring a patient's clinical history, conducting an examination, and communicating findings. Mastery of these skills is vital for medical students and clinicians, ensuring high-quality patient care and accurate diagnoses.

2. Objectives of Clerking

The purpose of clerking is to:

- Establish rapport with the patient.
- Obtain a detailed history of the presenting complaint, past medical history, family history, and social history.
- Conduct a thorough physical examination.
- Formulate a differential diagnosis and initial management plan.
- Communicate findings effectively to other healthcare professionals.

3. History Taking

3.1 Presenting Complaint

The presenting complaint (PC) is the primary reason the patient seeks medical attention. Document it in the patient’s own words, including its duration (e.g., "Chest pain for 3 days").

3.2 History of Presenting Complaint (HPC)

Explore the presenting complaint comprehensively:

- Onset: When did it start?
- Duration: How long has it lasted?
- Character: Nature of the symptom (e.g., sharp, dull).
- Radiation: Does it spread to other areas?
- Associated Symptoms: Are there accompanying symptoms?
- Exacerbating/Relieving Factors: What worsens or alleviates it?
- Severity: Use a pain scale if appropriate.

3.3 Systematic Review

Perform a system-by-system review to elicit any associated symptoms not mentioned initially.

3.4 Past Medical History (PMH)

Ask about:

- Chronic illnesses (e.g., diabetes, hypertension).
- Previous hospitalizations or surgeries.
- Allergies and adverse reactions to medications.

3.5 Family History (FH)

Inquire about:

- Hereditary conditions (e.g., diabetes, cardiovascular disease).
- History of similar illnesses in close relatives.

3.6 Social History (SH)

Explore:

- Lifestyle factors (e.g., smoking, alcohol consumption, drug use).
- Occupation and its relevance to the presenting complaint.
- Living conditions and support systems.

3.7 Drug History

Document:

- Current medications, including dosages.
- Over-the-counter or herbal remedies.
- Adherence to prescribed treatments.
- Usage of illicit drug and route.
- Usage of alcohol and quantify by units/week.
- Smoking and quantify using pack-years.

4. Physical Examination

4.1 General Inspection

Observe the patient for:

- Appearance: Well or ill-looking, distressed.
- Nutritional status.
- Evidence of cyanosis, oedema, clubbing, pallor, jaundice, or dehydration.

4.2 System-Specific Examination

Conduct a focused examination of the system involved in the presenting complaint. For example:

- **Cardiovascular System:** Inspect, palpate, auscultate, and percuss as needed.
- **Respiratory System:** Look for chest wall deformities, assess breathing patterns, palpate, percuss, and auscultate lung fields.
- **Abdomen:** Inspect, palpate, percuss, and auscultate for abnormalities.
- **Neurological System:** Cognitive function, assess for meningism, cranial nerves, motor function (observe for movements and trophism, assess tone, reflexes, and power), sensory function (touch, temperature, pain, vibration, proprioception and describe the dermatome where abnormal findings were present), autonomic function, and cerebellar function including gait.
- **Musculoskeletal System:** Look (wasting, swelling, etc), feel (warmth of joints, crepitus of joints) and move (passively first then actively, and note the difference). Look for skin changes, alopecia, oral and genital ulcers, swollen parotids, scratchy eyes and dry mouth.

4.3 Special Tests

Perform relevant special tests based on the clinical scenario (e.g., neurological reflexes, gait assessment).

5. Problem List and Differential Diagnosis

Develop a concise problem list and construct a differential diagnosis (a list of possible diagnoses according to history and examination). Use the history and examination findings to prioritize conditions. Usually a total of 3 differential diagnoses is acceptable.

6. Investigations

6.1 Basic Investigations

Request basic tests as appropriate:

- Blood tests (e.g., full blood count, renal function tests).
- Urinalysis.

6.2 System-Specific Investigations

Examples:

- **Cardiology:** ECG, echocardiogram, urine dipstick (infective endocarditis).
- **Respiratory:** Chest X-ray, pulmonary function tests.

7. Management Plan

Outline an initial management plan based on findings:

- Symptomatic treatment.
- Lifestyle modification.
- Specific interventions.
- Referrals if necessary.

8. Clinical Case Presentation

8.1 Structure of Presentation

A structured presentation ensures clarity:

1. **Case summary**: Brief demographic description, relevant comorbidities (relevant to the current presentation), and lastly the current clinical problem (not symptoms & signs, but the actual syndrome or diagnosis if known). At this stage mention any associations, complications or precipitants that might be found in that particular patient. Each system might have its own particularities.

    1.1. **Cardiac case example:** I had the pleasure to interview Mr Lethlape today, he is a 45 years old married father of 2 children from Soshanguve, and works as a plumber for the past 15 years. He suffered from rheumatic fever around the age of 15 (relevant history), and now presents with a problem of mitral stenosis (clinical problem) most likely rheumatic in nature (etiology). He is currently in heart failure, NYHA class IV, complicated with atrial fibrillation, pulmonary hypertension and infective endocarditis (complications). Mr Lethlape requires an urgent mitral valve replacement.

    1.2. **Respiratory case example**: Mr Ndlovu is a 65 years old single father of 6 children from Soweto. He worked in the goldmines for 25 years until his retirement last year. He has no other comorbidities of note except for his 30 pack-year history of smoking. He now presents with clinical features suggestive of chronic obstructive pulmonary disease with an MRC class III dyspnoea complicated by cor-pulmonale and a possibility of lung cancer his occupational history, presence of clubbing and significant weight loss.

    1.3. **Musculoskeletal case example**: Ms Swarts is a 24 years old scholar from Brits. She has a strong 1st degree family history of inflammatory arthritis. She presents with a problem of a symmetric polyarthritis predominantly affecting the small joints of hands and feet for the past 6 weeks most likely due to rheumatoid arthritis (RA) given her family history and clinical presentation. Her RA is associated with sicca symptoms and

is currently ACR functional class II with a severely active disease with a CDAI score of 22.

2. **Introduction:** Patient's demographic details and presenting complaint.
3. **History:** Briefly summarize the HPC, PMH, FH, SH, and drug history.
4. **Examination Findings:** Report relevant positive and the relevant negative findings.
5. **Problem List:** Concisely list clinical problems identified and present your differential diagnoses.
6. **Investigations:** Highlight key findings.
7. **Management Plan:** Present your proposed plan.

8.2 Presentation Tips

- Be concise but comprehensive.
- Highlight key points relevant to the diagnosis.
- Avoid unnecessary repetition.

9. Common Pitfalls in Clerking and Presentation

- Poor summary that is too long involving symptoms and signs without committing to a clinical problem or syndrome. Summaries should be true to their name, short, concise and straight to the point.
- Omitting important details in the history or examination.
- Failing to ask about drug allergies.
- Overloading the presentation with irrelevant details.
- Presenting findings in a disorganized manner.

10. Conclusion

Effective clerking and case presentation are vital for accurate diagnosis and management. They require practice, attention to detail, and a structured approach. Using resources like Talley and O'Connor's *Clinical Examination* helps refine these skills.

References

1. Talley NJ, O'Connor S. *Clinical Examination: A Systematic Guide to Physical Diagnosis*. 9th ed. Elsevier; 2020.
2. Epstein O, Perkin GD, Cookson J, de Bono DP. *Clinical Examination*. 4th ed. Mosby; 2008.

SECTION 3: PRINCIPLES OF RESUSCITATION

3.1 Primary and Secondary Survey:

The principles of resuscitation involve a systematic approach to assessing and stabilizing a critically ill patient. These principles are best conceptualized as a two-tiered process comprising the **primary survey** and the **secondary survey**.

- **Primary Survey**
  - The primary survey focuses on the acute assessment of vital signs and the immediate management of life-threatening conditions. It prioritizes rapid stabilization of airway (A), breathing (B), circulation (C), and disability (D) before proceeding further.
  - Immediate targeted examinations, investigations, and interventions are carried out at this stage. Key investigations may include arterial blood gases (ABG), serum glucose, electrolytes (e.g., potassium), haemoglobin, and clotting profiles.
  - Radiological evaluations such as a 12-lead ECG and chest X-ray (CXR) are essential. A computed tomography (CT) scan of the brain may also be indicated, particularly in unconscious or neurologically impaired patients.
  - Administer antibiotics early, but ensure appropriate cultures, including blood cultures, are collected beforehand.
- **Secondary Survey**
  - The secondary survey identifies the underlying cause of the patient's condition, evaluates compromised organ systems, and assesses for precipitating factors.
  - Utilize the AMPLE mnemonic:
    - Allergies
    - Medications
    - Past medical history
    - Last meal
    - Events leading to the presentation
  - The effectiveness of cardiopulmonary resuscitation (CPR), if undertaken, should also be evaluated during the secondary survey.

3.2 Cardiopulmonary Resuscitation (CPR)

- Effective CPR is the cornerstone of resuscitation and is now guided by the **CABD** approach (Circulation, Airway, Breathing, and Disability):
  - **C: Circulation**
    - Circulation is the most critical component of CPR. Begin with chest compressions if there is no detectable pulse or in cases of pulseless electrical activity (PEA).
    - Perform compressions at a rate of 100-120 compressions per minute, with a depth of approximately 5-6 cm. Use a compression-to-breath ratio of 30:2.
    - After every five cycles of CPR, reassess pulse, blood pressure (BP), and brainstem reflexes.
    - Failure to achieve hemodynamic stability after the first cycle warrants the use of vasopressor agents (e.g., adrenaline) while continuing CPR.

- **IV Access**
  - Establish intravenous access promptly. Large-bore (16G or larger) cannulas are preferred for volume resuscitation. Consider central venous access via the femoral vein if peripheral access proves challenging.
- **AB: Airway and Breathing**
  - Assess airway patency and breathing by engaging the patient verbally or by observing spontaneous respiratory efforts.
  - In patients presenting with chronic respiratory conditions, perform an ABG immediately to tailor oxygen therapy appropriately.
  - High-flow oxygen (15 L/min via a reservoir mask) is recommended initially, achieving FiO2 of up to 90%. Adjust oxygen delivery based on ABG results.
  - Observe respiratory patterns, including rate, depth, rhythm, and accessory muscle use. Identify signs of paradoxical movement or asymmetry.
- **D: Disability**
  - Document the patient's Glasgow Coma Scale (GCS), specifying eye, verbal, and motor responses.
  - Evaluate pupil size, symmetry, and reactivity to light.
  - Arrange for transfer to a high-care or intensive care unit (ICU) post-stabilization.

3.3 Respiratory Failure and Mechanical Ventilation

- **Definition**: Respiratory failure is the inability to maintain adequate gas exchange across the alveolar-capillary membrane. There are 4 identified types of respiratory failure, however the two most common forms are:
  - **Type 1 (Hypoxemic)**: Low arterial oxygen levels ($PaO_2 < 8$ kPa) despite supplemental oxygen.
  - **Type 2 (Hypercapnic)**: Elevated carbon dioxide levels ($PaCO_2 > 6$ kPa) with accompanying acidosis (pH < 7.30).
- **Mechanisms**
  - Hypoventilation, ventilation-perfusion mismatch, shunting, and diffusion impairment contribute to respiratory failure. The alveolar-arterial (A-a) gradient calculation can aid in identifying these mechanisms.
- **Indication for Mechanical Ventilation**
  - Initiate intermittent positive pressure ventilation (IPPV) in patients with worsening hypoxemia or hypercarbia unresponsive to non-invasive measures.
- **Ventilation Modes and Settings**
  - Common modes: Assist-Control Ventilation (ACV), Synchronized Intermittent Mandatory Ventilation (SIMV), and Pressure Support Ventilation (PSV).
  - Initial settings:
    - FiO_2: 100% (adjust based on ABG results)
    - Tidal volume: 6-8 mL/kg
    - Respiratory rate: 12-20 breaths/min
    - Positive end-expiratory pressure (PEEP): 5-15 cmH_2O

3.4 Shock

- **Definition**: Shock is characterized by inadequate oxygen delivery to tissues due to hypovolemia, cardiac dysfunction, or distributive factors.

- **Etiology**
  - Types include hypovolemic, septic, cardiogenic, obstructive, anaphylactic, and neurogenic shock.
- **Management Principles**
  - Correct hypoxemia with high-flow oxygen.
  - Secure intravenous access and correct hypovolemia using appropriate fluids (e.g., crystalloids for sepsis, colloids for hypovolemia, or blood products for hemorrhage).
  - Initiate empirical antibiotics for septic shock and consider vasopressors (e.g., norepinephrine, adrenaline) if fluid resuscitation is insufficient.
  - Address underlying causes promptly, such as surgical interventions for intra-abdominal sepsis or thrombolysis for pulmonary embolism.

3.5 Blood Transfusion and Blood Products

- Indications for blood transfusion must be judiciously assessed, considering the scarcity of blood products.
- Follow stringent protocols for cross-matching and transfusion.
- Monitor vital signs closely and manage transfusion-related reactions immediately.

3.6 Automated External Defibrillation (AED)

- Utilize an AED for patients in cardiac arrest due to ventricular fibrillation (VF) or pulseless ventricular tachycardia (VT). Follow device prompts for shock delivery.

3.7 Transfer to High-Care or ICU

- Ensure timely transfer of patients to high-care or ICU after stabilization in the emergency department. The goal is to transition within 6 hours of presentation or provide a clear rationale for delays.
- Discuss all ICU admissions with the on-call consultant, incorporating clinical and laboratory findings.

References

1. Talley NJ, O'Connor S. Clinical Examination: A Systematic Guide to Physical Diagnosis. 8th ed. Sydney: Elsevier; 2022.
2. American Heart Association. Advanced Cardiovascular Life Support (ACLS) Provider Manual. Dallas, TX: AHA; 2020.
3. Vincent JL, De Backer D. Circulatory Shock. N Engl J Med. 2013;369(18):1726-1734.
4. Singer M, Deutschman CS, Seymour CW, et al. The Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3). JAMA. 2016;315(8):801-810.
5. JRCALC. UK Ambulance Services Clinical Practice Guidelines. Bridgwater: Class Professional Publishing; 2021.

SECTION 4: COMMON MEDICAL CASES IN SOUTH AFRICA

4.1 RESPIRATORY SYSTEM

The respiratory system conditions are highly prevalent in South Africa, often exacerbated by smoking, environmental factors, and HIV/AIDS. Common conditions include:

- **Obstructive Lung Disease**: Asthma, chronic obstructive pulmonary disease (COPD).
- **Restrictive Lung Disease**: Pulmonary fibrosis, interstitial lung disease.
- **Bronchiectasis**: Often secondary to tuberculosis (TB) or recurrent infections.
- **Lung Carcinoma**: Linked to smoking and environmental carcinogens.
- **Pleural Effusion**: Can be secondary to TB, malignancy, or heart failure.

4.2 CARDIOVASCULAR SYSTEM

Cardiovascular diseases remain a significant contributor to morbidity and mortality in South Africa. HIV may exacerbate some of these conditions. Common cases include:

- **Cardiomyopathy**: Dilated, hypertrophic, or restrictive cardiomyopathy, including HIV-associated forms.
- **Valvulopathy**: Rheumatic heart disease, particularly mitral stenosis or regurgitation.
- **Endocardial Disease**: Infective endocarditis, often secondary to valvular heart disease or IV drug use.
- **Pericardial Disease**: Pericarditis, including TB-related pericarditis and pericardial effusion.
- **Congenital Heart Disease**: Cyanotic and acyanotic conditions such as Tetralogy of Fallot and atrial septal defects.
- **Coronary Artery Disease**: Ischemic heart disease, acute coronary syndrome.
- **Peripheral Vascular Disease**: Atherosclerosis leading to claudication or limb ischemia.

4.3 NEUROLOGICAL SYSTEM

Neurological conditions often present due to vascular, degenerative, or infectious causes, many linked to HIV or TB. Key conditions include:

- **Cerebrovascular Accident (Stroke)**: Ischemic and hemorrhagic.
- **Parkinson's Disease**: Neurodegenerative disorder affecting motor function.
- **Cerebellar Disease**: Tumors, infections, or degeneration.
- **Bulbar and Pseudobulbar Palsy**: Often linked to motor neuron disease or stroke.
- **Lateral Medullary Syndrome**: Stroke in the posterior inferior cerebellar artery.
- **Paraplegia**: Often secondary to spinal cord TB, transverse myelitis, metastasis (prostate or multiple myeloma or trauma).
- **Peripheral Neuropathy**: HIV-associated neuropathy, diabetes-related neuropathy.
- **Myasthenia Gravis**: Autoimmune disorder affecting neuromuscular junction transmission.

4.4 GASTROINTESTINAL SYSTEM

Gastrointestinal conditions include hepatobiliary diseases and inflammatory conditions.

- **Jaundice**: Can indicate liver disease, hemolysis, or biliary obstruction.
- **Ascites**: Often due to cirrhosis, malignancy, or TB peritonitis.
- **Chronic Liver Disease**: Alcoholic liver disease, hepatitis B or C, non-alcoholic steatohepatitis.
- **Splenomegaly**: Common in portal hypertension, infections, or hematologic conditions.
- **Portal Hypertension**: Secondary to cirrhosis or schistosomiasis.
- **Inflammatory Bowel Disease**: Crohn's disease and ulcerative colitis.

4.5 RENAL SYSTEM

Renal conditions are prevalent, particularly in patients with hypertension, diabetes, or HIV. Common cases include:

- **Nephrotic Syndrome**: Often secondary to glomerular diseases like membranous nephropathy.
- **Nephritic Syndrome**: Post-infectious glomerulonephritis, SLE, or IgA nephropathy.
- **Hematuria**: Can indicate infection, stones, or malignancy.
- **Polycystic Kidney Disease**: An inherited condition.
- **Chronic Kidney Disease**: Secondary to diabetes, hypertension, or HIV nephropathy.

4.6 ENDOCRINE SYSTEM

Endocrine disorders involve hormonal imbalances, often linked to systemic diseases or tumours. Common conditions include:

- **Pituitary Disease**: Pituitary adenomas, hypopituitarism.
- **Thyroid Disease**: Hypothyroidism, hyperthyroidism, and thyroid nodules.
- **Parathyroid Disease**: Hyperparathyroidism causing hypercalcemia.
- **Adrenal Disease**: Addison’s disease, Cushing’s syndrome, adrenal tumors.
- **Gonadal Disease**: Hypogonadism, polycystic ovary syndrome.
- **Short Stature**: Growth hormone deficiency, Turner syndrome, Down’s syndrome.
- **Tall Stature**: Acromegaly, gigantism, MEN 1 syndrome, Klinefelter’s syndrome.

4.7 HAEMATOLOGICAL SYSTEM

Haematological conditions often present due to anaemia or malignancies, many linked to HIV or TB. Common cases include:

- **Anemia**: Iron deficiency, pernicious anemia, hemolytic, anemia of chronic disease.
- **Thrombocytopenia**: Immune thrombocytopenia, drug-induced thrombocytopenia.
- **Pancytopenia**: Bone marrow failure syndromes, HIV-associated cytopenias.
- **Leukemia**: Acute or chronic forms of myeloid or lymphoid leukemia.
- **Lymphoma**: Hodgkin’s and non-Hodgkin’s lymphoma.
- **Thrombophilia**: Deep vein thrombosis or pulmonary embolism (Antiphospholipid, factor V Leiden disease, Anti-thrombin III deficiency, Protein C&S deficiency, Polycythemia rubra vera, Essential thrombocytosis).

4.8 MUSCULOSKELETAL SYSTEM

Rheumatological conditions are common, particularly autoimmune diseases and inflammatory conditions. Examples include:

- **Arthritis**: Rheumatoid arthritis, reactive arthritis, seronegative spondyloarthropathy, crystal arthropathies (gout, CPPD), juvenile idiopathic arthritis, osteoarthritis, septic arthritis.
- **Systemic Lupus Erythematosus (SLE)**: Autoimmune multisystem disorder.
- **Systemic Sclerosis**: Connective tissue disease with skin tightening and organ involvement.
- **Sarcoidosis**: Granulomatous disease affecting the lungs, skin, joints or eyes.
- **Vasculitis**: Polyarteritis nodosa, granulomatosis with polyangiitis, eosinophilic granulomatosis with polyangiitis, Takayasu arteritis.

Study Tips

- Always associate these conditions with HIV/AIDS, as it influences disease presentation and progression.
- Focus on differential diagnosis, management guidelines, and complications specific to the South African context.
- Use local guidelines, including those from the **South African Department of Health** and **SA Thoracic Society**.

SECTION 5: MEDICAL EMERGENCIES

5.1 Community-Acquired Pneumonia (CAP)

Definition: Acute lower respiratory tract infection accompanied by recently developed radiological signs.

Clinical Diagnosis:

- Symptoms: Dyspnea, pleuritic chest pain, cough (often initially dry), sputum (mucopurulent, rusty, or bloodstained), wheeze, fever, malaise, rigors, and myalgia.
- Signs: Tachypnea, tachycardia, cyanosis, dullness on percussion, crackles, bronchial breathing, and pleural rub.
- Assess sputum characteristics and presence of cyanosis to evaluate severity.

Causative Organisms:

- **Typical pathogens**:
  - *Streptococcus pneumoniae* (31%)
  - *Haemophilus influenzae* (7%)
  - Influenza virus (7%)
  - *Staphylococcus aureus* (2%)
  - Gram-negative organisms (e.g., *Klebsiella pneumoniae* 2%)
- **Atypical pathogens**:
  - *Mycoplasma pneumoniae* (10%)
  - *Chlamydia psittaci* (4%)
  - *Legionella pneumophila* (2%)
- In 35% of cases, the causative organism remains unidentified.

Assessment of Severity:

- Use **CURB-65** criteria:
  - Confusion
  - Urea > 7 mmol/L
  - Respiratory rate ≥ 30/min
  - BP < 90/60 mmHg
  - Age ≥ 65 years
- A score of 2 or more indicates a higher mortality risk and ICU need. Additional markers include co-morbidities, multilobar involvement, and atrial fibrillation.

Initial Investigations:

- ABG, CXR, FBC, U&E, blood cultures, and sputum microscopy, culture, and sensitivity (MCS).
- Atypical pneumonia: Legionella antigen in urine, throat swab for viral studies.

Initial Management:

- Oxygen therapy, IV fluids, antipyretics, and antibiotics.
- **Antibiotic Therapy**:
  - *Mild cases*: Amoxicillin 500 mg orally tds.
  - *Moderate cases (unable to tolerate oral meds)*: Ampicillin 1 g IV tds.
  - *Severe cases*: Ceftriaxone 1 g IV bd plus a macrolide (e.g., clarithromycin 500 mg bd).
  - For suspected *Staphylococcus aureus*: Add cloxacillin 1 g IV q6h.
  - If *Mycoplasma* or *Legionella* suspected: Add clarithromycin or rifampicin in severe cases.

5.2 Acute Exacerbation of COPD (AECOPD)

Definition: A worsening of the patient's previously stable COPD condition.
Clinical Features:
- **Symptoms**: Increased breathlessness, sputum production (purulence or volume), wheeze, chest tightness, ankle edema.
- **Signs**: Tachypnea, accessory muscle use, wheezing, cyanosis, confusion, peripheral edema.

Common Precipitants:
- Lower respiratory tract infections, pneumothorax, continued smoking, or treatment non-adherence.

Initial Investigations:
- ABG, CXR (rule out pneumothorax), peak flow measurement, FBC, U&E, sputum culture, and ECG.

Initial Management:
- Oxygen therapy (Venturi mask at 24-28% or 2 L/min nasal prongs).
- Reassess ABG within 20 minutes of starting or adjusting oxygen.
- Bronchodilators: Nebulized salbutamol 5 mg and ipratropium bromide 500 mcg every 4-6 hours.
- Systemic corticosteroids: Prednisolone 30-40 mg orally or hydrocortisone IV.
- Antibiotics: As per CAP protocol.
- Consider **Non-Invasive Ventilation (NIV)** for hypercapnia with acidosis (pH < 7.35).

5.3 Acute Asthma Attack

Presentation:
- Breathlessness, wheezing, chest tightness, cough, and accessory muscle use.

Classification:
- **Severe Attack**: RR ≥ 25/min, HR ≥ 110 bpm, PEF < 50% of predicted, SpO2 < 92%.
- **Life-Threatening (Status Asthmaticus)**: Silent chest, feeble respiratory efforts, bradycardia, hypotension, confusion.

Investigations:
- ABG, CXR, and assessments similar to AECOPD.

Stepwise Management:
1. Salbutamol 5 mg nebulized in 8 L/min oxygen every 10-15 minutes.
2. Prednisolone 40 mg orally or hydrocortisone 200 mg IV.
3. Ipratropium bromide 500 mcg nebulized.
4. IV magnesium sulfate (2 g over 20 minutes) for persistent symptoms.
5. Consider aminophylline infusion or IV salbutamol in refractory cases.
6. Escalate to ICU and consider mechanical ventilation if no improvement.

5.4 Tension Pneumothorax

Definition: Life-threatening accumulation of air in the pleural space causing cardiorespiratory compromise.
Clinical Features:
- Associated with trauma, positive-pressure ventilation, rib fractures, or central line insertion.

- Signs: Hypotension, raised JVP, tracheal deviation, reduced air entry, hyper-resonance, and cyanosis.

Management:

- Immediate decompression with a 14G cannula in the 2nd intercostal space, mid-clavicular line.
- Insert intercostal drain and confirm placement with CXR.

5.5 Pulmonary Embolism (PE)

Definition: Pulmonary vascular obstruction caused by thromboembolism.

Risk Factors:

- Immobility, hormonal contraceptives, thrombophilia, malignancy, surgery.

Clinical Features:

- Symptoms: Sudden-onset dyspnea, pleuritic chest pain, hemoptysis, syncope.
- Signs: Tachypnea, tachycardia, loud P2, right heart strain.

Investigations:

- FBC, U&E, D-dimers, troponins, ECG (sinus tachycardia, S1Q3T3 pattern, RBBB).
- Imaging: CXR, V/Q scan (if CXR normal), or CT pulmonary angiography.

Management:

- Anticoagulation (e.g., low molecular weight heparin) for 6 months if stable.
- Thrombolysis in hemodynamically unstable patients with elevated troponins and right ventricular strain.
- Consider percutaneous thrombectomy for large proximal thrombi.

References

1. British Thoracic Society. *Guidelines for the Management of Community Acquired Pneumonia in Adults*. Thorax. 2009.
2. Global Initiative for Chronic Obstructive Lung Disease (GOLD). *Global Strategy for the Diagnosis, Management, and Prevention of COPD*. 2023.
3. Global Initiative for Asthma (GINA). *Global Strategy for Asthma Management and Prevention*. 2023.
4. Light RW. *Pleural Diseases*. 6th ed. Lippincott Williams & Wilkins; 2013.
5. Kearon C, Akl EA, Comerota AJ, et al. *Antithrombotic Therapy for VTE Disease*. Chest. 2012;141(2_suppl):e419S-e494S.

5.2 Hypertensive Crisis

Hypertensive Emergency:

- **Definition**: BP > 220/120 mmHg with evidence of target organ damage (e.g., CVA, encephalopathy, acute kidney injury [AKI], acute left ventricular failure [LVF], or acute onset blindness).
- **Management**:
  - Decrease systolic BP by 20% or diastolic BP to 100 mmHg over the first 12-24 hours.
  - **First-line Agent**: Labetalol IV, except in acute LVF with pulmonary edema, where nitroglycerine (Nitrocine) is preferred.
  - Commence oral antihypertensives within 24 hours.

Hypertensive Urgency:

- **Definition**: BP > 220/120 mmHg without target organ damage.
- **Clinical Features**: Headache, tinnitus, and scotomas.
- **Management**:
  - Oral antihypertensives to achieve BP < 140/90 mmHg gradually.

Eclampsia:

- **Definition**: Complication of pre-eclampsia with seizures, often associated with HELLP syndrome.
- **Management**:
  - Treat as hypertensive emergency.
  - Urgent referral to Obstetrics, as definitive management involves removal of the placenta.
  - Note: Seizures may occur up to 24 hours postpartum.

5.4 Acute Coronary Syndrome (ACS)

ST-Elevation Myocardial Infarction (STEMI):

- **Clinical Features**: Chest pain (typical/atypical), ST elevation or new LBBB on ECG, and elevated cardiac biomarkers.
- **Investigations**:
  - ECG, CXR, urine dipstick, blood tests (FBC, U&E, LFT, INR, troponins, TFT, lipogram, HIV ELISA, Hepatitis B, HbA1C).
  - Troponins should be repeated 6 hours after the first sample.
- **Management**:
  - Sublingual nitroglycerin (TNT), dual antiplatelet therapy (Clopidogrel and Aspirin).
  - **Thrombolysis**: Anti-TPA (Alteplase or Metelyse) or Streptokinase if PCI is unavailable.
  - Anticoagulation: Clexane (1 mg/kg bd if <75 years, 0.75 mg/kg bd if >75 years).
  - Atorvastatin 80 mg nocte.
  - Initiate ACE inhibitors on day 2 and beta-blockers after echocardiography.
  - Oxygen only if SpO2 < 94%.
  - Morphine is contraindicated.
- **Complications**:
  - Hypotension (RV infarct, reperfusion injury, cardiogenic shock).
  - Arrhythmias (urgent transfer to CCU required).

Non-ST Elevation Myocardial Infarction (NSTEMI):

- Clinical features include chest pain, ST depression, inverted T waves, or normal ECG with elevated biomarkers.
- **Management**: Similar to STEMI except for PCI/thrombolysis, which is reserved for STEMI.

Unstable Angina:

- Clinical features include chest pain, normal ECG, and negative biomarkers.
- **Management**: Nitrates, beta-blockers, and calcium channel blockers (diltiazem, verapamil).

5.5 Heart Failure

Definition: Acute or chronic decrease in cardiac output due to structural or intrinsic cardiac disease, often precipitated by acute events (e.g., MI, infection, arrhythmias).

Classification:

- **Functional**: NYHA I-IV.
- **Anatomical**: Left, right, or biventricular.
- **Output**: High or low output failure.

Clinical Presentation:

- **Symptoms**: Dyspnea, orthopnea, PND, cough with frothy sputum, edema.
- **Signs**: Pulsus alternans, Cheyne-Stokes breathing, basal crackles, elevated JVP.

Investigations:

- CXR, ECG, cardiac biomarkers, FBC, U&E, CRP, blood cultures, and urine dipstick.

Management:

- **Acute**: Fluid restriction, salt restriction, diuretics (furosemide), spironolactone, ACE inhibitors (enalapril), beta-blockers (carvedilol), and inotropes (dobutamine).
- **Chronic**: Risk factor management, medical therapy, and in advanced cases, mechanical support or transplantation.

5.6 Pulmonary Edema

Definition: Acute accumulation of fluid in alveoli due to cardiogenic or non-cardiogenic causes.

Causes:

- **Cardiogenic**: Acute MI, arrhythmias, valvular disease.
- **Non-Cardiogenic**: Trauma, aspiration, infections, massive transfusions, oxygen toxicity, PE.

Clinical Presentation:

- Respiratory distress, haemoptysis, and frothy sputum.

Management:

- Oxygen supplementation, nitrates, diuretics, and addressing the underlying cause.

5.7 Arrhythmias

Definition: Any deviation from sinus rhythm.

- **Classification**:
  - Tachyarrhythmias (narrow or broad complex).
  - Bradyarrhythmias (narrow or broad complex).

Tachyarrhythmias:

- **Narrow Complex Regular (SVT)**:

        - Attempt Valsalva maneuvers (<40 years).
        - Adenosine 6 mg IV stat (repeat with 12 mg if no response).
        - Consider synchronized cardioversion (50 J).
    - **Narrow Complex Irregular (AF)**:
        - Amiodarone infusion or electrical defibrillation.
        - Anticoagulation is mandatory.
    - **Broad Complex Regular (VT)**:
        - Electrical defibrillation (200-360 J) if hypotensive.
        - Amiodarone infusion if BP stable.
    - **Broad Complex Irregular (VF)**:
        - High-energy defibrillation (200 J), followed by antiarrhythmic infusion.

Bradyarrhythmia:
- **Narrow Complex**: Exclude drug effects (beta-blockers, calcium channel blockers).
- **Broad Complex**: Ensure pacemaker availability, consider temporary pacing for AV block.

5.8 Pericardial Tamponade

Definition: Acute diastolic failure caused by fluid/blood accumulation in the pericardial space.
Clinical Features:
- Beck's Triad: Hypotension, elevated JVP, muffled heart sounds.

Investigations:
- CXR, ECG, echocardiography, blood tests (FBC, U&E, LFT, CRP, ESR).

Management:
- Urgent pericardiocentesis: Insert an 18G cannula 1 cm below the xiphoid process and aspirate.

References

1. American Heart Association. Advanced Cardiovascular Life Support (ACLS) Provider Manual. Dallas, TX: AHA; 2020.
2. European Society of Cardiology. *Guidelines for the Management of Acute Coronary Syndromes*. Eur Heart J. 2021;42(14):1289-1367.
3. McMurray JJV, et al. *Heart Failure: ESC Guidelines for Diagnosis and Treatment*. Eur Heart J. 2021;42(36):3599-3726.
4. Pericardial Disease. In: Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine. 12th ed. Elsevier; 2021.

5.9 Altered Mental State

Delirium:
- **Definition**: Acute onset cognitive dysfunction caused by metabolic, infectious, or traumatic factors.
- **Management**:
  - Identify and treat the underlying cause.
  - Calm agitated patients with antipsychotics such as haloperidol (1-2 mg IV or IM). Avoid benzodiazepines unless managing withdrawal syndromes, as they can exacerbate symptoms.

Dementia:
- **Definition**: Chronic cognitive dysfunction, reversible (e.g., folate deficiency, tertiary syphilis) or irreversible (e.g., Alzheimer's, Huntington's disease, Parkinson's plus syndromes).
- **Management**:
  - Not an acute emergency unless associated with psychosis.
  - Assess using the Montreal Cognitive Assessment (MoCA).
  - Refer for long-term management (neurology or home-based care).

Acute Psychosis:
- **Definition**: Disorganized speech and behavior, hallucinations, and delusions.
- **Management**:
  - Initial verbal de-escalation.
  - Administer oral antipsychotics (chlorpromazine 50-100 mg or haloperidol 5-10 mg).
  - If uncooperative, offer chemical restraint (lorazepam 2-4 mg IM/IV).
  - If agitation persists, consider mechanical restraint in a secure facility.
  - Investigations: FBC, U&E, CMP, TFT, RPR, CXR, CT brain, lumbar puncture (LP).
  - For meningitis features: Start empiric ceftriaxone (2 g BD) until LP results are available.

5.10 Acute Stroke

Definition: Sudden onset hemiparesis or hemiplegia caused by ischemia, hemorrhage, or transient ischemic attack (TIA).

Causes:
- Ischemia, hemorrhage, TIA, reversible ischemic neurological deficit (RIND), Todd's paresis.

Clinical Features:
- Altered mental state, cranial nerve involvement, hemiparesis, hemiplegia, dysphasia, meningism, arrhythmias, carotid bruits.

Investigations:
- Blood tests: FBC, U&E, INR, APTT, LFT, CRP, ESR. For young strokes, include ANCA, antithrombin III, Factor V Leiden, antiphospholipid antibodies, ANA.
- Imaging: CXR, ECG, echocardiography, CT brain.

Management:
- **Hemorrhagic Stroke**: For GCS ≥4, discuss with neurosurgery for craniotomy.
- **Ischemic Stroke**: Consider thrombolysis (alteplase) if presenting within 3 hours of symptom onset and CT brain is normal.

5.11 Status Epilepticus

Definition: Continuous or recurrent seizures lasting >15-30 minutes with impaired consciousness between seizures.

Causes:

- Non-adherence to anticonvulsants, meningitis, head injury, electrolyte abnormalities, space-occupying lesions.

Investigations:

- Bloods: U&E, CMP, FBC, CRP, ESR, INR, RPR, HIV ELISA.
- Imaging: CXR, CT brain (immediate), LP (if no contraindications), EEG.

Management:

1. Abort seizures: Diazepam 10 mg IV/PR stat.
2. Load anticonvulsants: Phenytoin 15-20 mg/kg IV. Repeat if seizures persist.
3. If seizures continue: Valproate 25 mg/kg IV or phenobarbital 20 mg/kg IV over 20 minutes.
4. Persistent seizures: Sedate with propofol, intubate, and transfer to ICU.

5.12 Meningitis & Encephalitis

Definitions:

- **Meningitis**: Inflammation of the meninges due to bacteria, viruses, fungi, or TB.
- **Encephalitis**: Brain inflammation secondary to meningitis, primary viral infection, or prion disease.

Clinical Features:

- Headache, fever, photophobia, lethargy.
- Signs: Kernig's sign, Brudzinski's sign, cranial nerve involvement, altered consciousness (encephalitis).

Investigations:

- Blood: FBC, CRP, U&E, ESR, INR, HIV.
- Imaging: CT brain if focal signs or raised ICP suspected; LP for definitive diagnosis.

Management:

- Empiric antibiotics: Ceftriaxone 2 g IV BD.
- For viral encephalitis: Acyclovir 800 mg IV QID for 10-14 days.
- Supportive therapy: Antipyretics, analgesia, DVT prophylaxis (e.g., enoxaparin 40 mg SC daily).

5.13 Subarachnoid Hemorrhage (SAH)

Definition: Bleeding into the subarachnoid space, often due to aneurysm rupture.

Causes:

- Aneurysms (saccular, mycotic), AV malformations.

Clinical Features:

- Sudden severe headache, loss of consciousness, cranial nerve palsies.

Investigations:

- Blood: FBC, U&E, coagulation profile.
- Imaging: Non-contrast CT brain, LP (xanthochromia if CT inconclusive).

Management:

- Urgent referral to neurosurgery for aneurysm repair.
- Start nimodipine 60 mg PO QID to prevent vasospasm.
- DVT prophylaxis: Pneumatic compression stockings.

5.14 Acute Lower Limb Paralysis

Definition: Rapid onset weakness in lower limbs, with or without sensory, bladder, bowel, or autonomic involvement.

Causes:

- **Flaccid Paralysis**: Guillain-Barré Syndrome (GBS), myopathy, botulism, transverse myelitis, poliomyelitis.
- **Spastic Paralysis**: Multiple sclerosis, transverse myelitis, spinal metastases, Pott's disease.

Investigations:

- Blood: CK, CMP, U&E, FBC, ESR, SACE, HIV ELISA, HTLV-1 serology.
- Imaging: Spine X-ray, CT brain, MRI spine (urgent if spinal shock suspected).
- Others: EMG, nerve conduction studies (NCS).

Management:

- Insert urinary catheter and initiate supportive care.
- Physiotherapy and occupational therapy.
- If GBS suspected: Administer IV immunoglobulin (0.4 g/kg/day for 5 days).

References

1. Adams RD, Victor M, Ropper AH. *Principles of Neurology*. 10th ed. McGraw-Hill; 2014.
2. Fisher M. *Stroke Prevention and Treatment*. Wiley-Blackwell; 2020.
3. Gilden DH. *Infections of the Nervous System*. New Engl J Med. 2005;352(25):2553-2562.
4. UK National Institute for Health and Care Excellence (NICE). *Meningitis (Bacterial) and Meningococcal Septicaemia in Children and Young People: Recognition, Diagnosis, and Management*. NICE Guidelines; 2021.
5. World Federation of Neurology. *Neurological Emergencies: Guidelines for Practice*. 2022.

5.15 Upper Gastrointestinal Bleed (UGIB)

Definition: Intraluminal bleeding from the gastrointestinal (GI) tract extending from the mouth to the ligament of Treitz.

Causes:

- Esophageal and gastric erosions (e.g., Boerhaave's syndrome, Mallory-Weiss tear).
- Bleeding varices.
- Peptic ulcers (e.g., due to NSAIDs, alcohol, *Helicobacter pylori*).
- Gastritis.

Clinical Features:

- Hematemesis, melena, lethargy, pallor, palpitations, and syncopal episodes.

Investigations:

- Blood tests: FBC, U&E, LFT, INR, amylase, lipase, CRP.
- Imaging: Erect CXR and AXR to rule out perforation or obstruction.

Management:

- Apply **CABD principles**.
- Insert two large-bore cannulas or a central line.
- Maintain mean arterial pressure (MAP) at 65 mmHg.
- Avoid nasogastric tubes (NGT).
- Transfuse if haemoglobin < 8 g/dL.
- Arrange for urgent gastroscopy (G-scope).
- If no G-scope is available, consider Sengstaken-Blakemore tube for variceal bleeds if experienced.
- Start propranolol or octreotide for varices to reduce splanchnic pressures.

5.16 Liver Failure

Definition: Inability of the liver to perform its metabolic (synthetic and detoxification) functions.

Causes:

- Chronic hepatic diseases (e.g., alcohol, non-alcoholic steatohepatitis, hemochromatosis, Wilson's disease).
- Drugs (e.g., paracetamol, isoniazid, rifampicin).
- Fulminant viral hepatitis.
- Toxins (e.g., mushrooms, herbal remedies).

Clinical Features:

- Signs of cirrhosis, asterixis, encephalopathy, jaundice, hypoglycaemia.

Investigations:

- Blood tests: FBC, VBG, LFT, INR, U&E, hepatitis screen, AFP, paracetamol levels, ammonium levels.
- Imaging: Abdominal ultrasound, CXR.

Management:

- Insert NGT and IV cannula.
- Start IV dextrose 5% (1L every 8 hours).
- Monitor glucose levels every 2-4 hours.
- Administer lactulose 15-30 mL PO tds.
- Investigate and address the underlying cause.

5.17 Acute Diarrhea and Dysentery

Definition: Passage of ≥3 loose stools in 24 hours. Presence of blood defines dysentery. May cause dehydration or renal impairment.

Causes:

- Viral (e.g., rotavirus, Norwalk virus).
- Bacterial (e.g., *Escherichia coli*, *Salmonella*, *Shigella*).
- Dysentery: *Shigella*, *Entamoeba histolytica*, *E. coli*.
- Antibiotic-associated (e.g., *Clostridioides difficile*).

Clinical Features:

- Loose stools, decreased skin turgor, dry mucosa, pallor, hypovolemic shock in severe cases.

Investigations:

- Stool MCS.
- Blood tests: FBC, U&E, LFT, INR, stool *C. difficile* toxin.
- Imaging: AXR.

Management:

- Insert an 18G IV line.
- Start IV fluids for dehydration or hypovolemic shock.
- Viral diarrhoea: Supportive care with hydration and electrolyte management.
- Bacterial diarrhoea: Start metronidazole PO; for dysentery, add ciprofloxacin or nalidixic acid.

5.18 Acute Pancreatitis

Definition: Inflammation of the pancreas leading to systemic release of pancreatic enzymes.

Causes:

- Alcohol intoxication, choledocholithiasis, hypertriglyceridemia, viral pancreatitis.

Clinical Features:

- Severe epigastric pain, vomiting, fever, dehydration, steatorrhea.

Investigations:

- Blood tests: FBC, U&E, LFT, amylase, lipase, CMP, lipid profile, HbA1C.
- Imaging: CXR (rule out ARDS), AXR (rule out ileus), abdominal ultrasound (rule out pseudocysts).

Management:

- Insert two wide-bore cannulas.
- Administer IV fluids (1L every 4-6 hours for 24 hours).
- Insert a urinary catheter and monitor output.
- Refer for surgical management and investigate underlying etiology.

5.19 Acute Abdomen

Definition: Sudden onset abdominal pain due to intra-abdominal organ pathology potentially requiring surgical intervention.

Causes:

- Bowel obstruction, perforation, peritonitis, mesenteric ischemia, pancreatitis, appendicitis.

Clinical Features:

- Abdominal distension, rebound tenderness, guarding, shoulder tip pain.

Investigations:

- Blood tests: FBC, U&E, LFT, INR, amylase, lipase.
- Imaging: CXR (air under diaphragm), AXR (dilated bowel loops, air-fluid levels), abdominal ultrasound.

Management:

- Keep the patient nil per mouth.
- Insert two large-bore IV cannulas.
- Insert a urinary catheter and monitor output.
- Refer for urgent surgical management.

References

1. Feldman M, Friedman LS, Brandt LJ. *Sleisenger and Fordtran's Gastrointestinal and Liver Disease*. 11th ed. Philadelphia: Elsevier; 2020.
2. Kumar PJ, Clark ML. *Kumar and Clark's Clinical Medicine*. 10th ed. Elsevier; 2021.
3. Williams NS, O'Connell PR, McCaskie A. *Bailey and Love's Short Practice of Surgery*. 27th ed. CRC Press; 2018.
4. WGO. *World Gastroenterology Organisation Global Guidelines*. 2022.

5.20 Acute Kidney Injury (AKI) and Dialysis

Definition: AKI is defined as an abrupt decline in renal function, characterized by a rise in serum creatinine by ≥0.3 mg/dL within 48 hours or ≥50% from baseline, or a urine output <0.5 mL/kg/hour for ≥6 hours (AKIN/RIFLE criteria).

Causes:

- **Pre-renal:** Dehydration, hypovolemia from diarrhea or vomiting, hemorrhage.
- **Renal:** Acute tubular necrosis (ischemic or nephrotoxic), glomerulonephritis, interstitial nephritis.
- **Post-renal:** Obstruction due to nephrolithiasis, prostate enlargement, or malignancies.

Clinical Features:

- Symptoms: Fatigue, nausea, and decreased urine output.
- Signs: Hypertension, fluid overload (peripheral edema, pulmonary crackles), and uremic features (pruritus, encephalopathy).

Investigations:

- Blood: FBC, UEC, LFT, coagulation profile.
- Imaging: Renal ultrasound (to exclude obstruction).
- Urine: Dipstick, microscopy, culture, and sensitivity.

Acute Management:

- Insert urinary catheter for monitoring output.
- Resuscitate with IV fluids (balanced crystalloids preferred).
- Treat underlying causes (e.g., antibiotics for infection).
- Dialysis indications: Refractory acidosis, hyperkalemia, fluid overload, uremia. Peritoneal dialysis or hemodialysis may be used based on clinical scenario and resources.
-

References

1. Kellum JA, Lameire N, Aspelin P, et al. KDIGO clinical practice guideline for acute kidney injury. Kidney Int Suppl. 2012;2(1):1-138. doi:10.1038/kisup.2012.1
2. Ronco C, Reis T, Husain-Syed F. Management of acute kidney injury in critically ill patients: An update. Nephrol Dial Transplant. 2021;36(3):452-460. doi:10.1093/ndt/gfaa259
3. Bellomo R, Kellum JA, Ronco C. Acute kidney injury. Lancet. 2012;380(9843):756-766. doi:10.1016/S0140-6736(11)61454-2

5.21 Diabetic Ketoacidosis (DKA)

Definition: DKA results from insulin deficiency, leading to hyperglycemia (>13.9 mmol/L), metabolic acidosis (pH <7.3), and ketonemia.

Clinical Features:

- Symptoms: Polyuria, polydipsia, abdominal pain, nausea, and vomiting.
- Signs: Tachycardia, hypotension, Kussmaul breathing, and fruity-smelling breath.

Management:

- **Fluid Therapy:** Begin with 0.9% saline (1 L/hour for the first hour). Adjust fluid type and rate based on hemodynamic status and corrected sodium levels.
- **Insulin Therapy:** Continuous IV infusion of regular insulin (0.1 U/kg/hour). Monitor glucose and potassium levels hourly.
- **Potassium Replacement:** Add potassium to fluids if levels <5.5 mmol/L. Avoid potassium supplementation in anuric patients.
- **Bicarbonate Therapy:** Reserved for pH <6.9 to prevent severe acidemia complications.

5.22 Addisonian Crisis

Definition: A life-threatening manifestation of adrenal insufficiency resulting in severe hypotension and electrolyte imbalance due to inadequate cortisol production.

Causes:

- Primary adrenal insufficiency (e.g., autoimmune adrenalitis, infections like tuberculosis).
- Secondary adrenal insufficiency (e.g., sudden withdrawal of long-term glucocorticoids, pituitary failure).
- Precipitating factors: Infections, trauma, or surgery in patients with known adrenal insufficiency.

Clinical Features:

- Symptoms: Lethargy, abdominal pain, nausea, vomiting, and confusion.
- Signs: Hypotension, hyperpigmentation (in primary cases), and hypoglycemia.

Investigations:

- Blood: FBC, UEC (elevated potassium, low sodium), cortisol, ACTH levels, and glucose.
- Imaging: Abdominal ultrasound and/or CT to evaluate adrenal glands.

Management:

- Immediate IV fluids (0.9% saline or 5% dextrose if hypoglycemic).
- Administer hydrocortisone 100 mg IV bolus, followed by 50 mg IV every 6 hours.
- Monitor electrolytes and glucose levels closely.

5.23 Thyroid Storm

Definition: An acute, life-threatening exacerbation of hyperthyroidism characterized by multi-organ dysfunction.

Clinical Features:

- Symptoms: Fever, palpitations, dyspnea, and gastrointestinal upset.
- Signs: Tachycardia, arrhythmias, tremors, and altered mental status.

Investigations:

- Blood: TFTs (elevated T3/T4, suppressed TSH), FBC, LFTs, and ECG for arrhythmias.

Management:

- Beta-blockers (e.g., propranolol) to control tachycardia.
- High-dose carbimazole or propylthiouracil (blocks thyroid hormone synthesis).
- Hydrocortisone 100 mg IV every 6 hours to inhibit T4-to-T3 conversion.

- Supportive therapy: Cooling blankets for hyperthermia and IV fluids.

5.24 Tumor Lysis Syndrome

Definition: A metabolic complication characterized by hyperuricemia, hyperkalemia, hyperphosphatemia, and hypocalcemia following rapid tumor cell lysis.

Causes:

- Commonly occurs after chemotherapy for hematological malignancies like leukemia and lymphoma.

Clinical Features:

- Symptoms: Fatigue, nausea, and muscle cramps.
- Signs: Cardiac arrhythmias and acute kidney injury.

Investigations:

- Blood: UEC, CMP, uric acid levels.
- ECG for hyperkalemia-induced changes.

Management:

- Aggressive IV hydration with 0.9% saline.
- Allopurinol 300 mg orally or rasburicase for hyperuricemia.
- Correct hyperkalemia and hypocalcemia cautiously.

5.25 Organophosphate Poisoning

Definition: Toxic exposure to organophosphate compounds, often used in pesticides, causing cholinergic overstimulation.

Clinical Features:

- SLUDGE syndrome: Salivation, Lacrimation, Urination, Diarrhea, Gastrointestinal upset, and Emesis.
- Severe cases: Respiratory failure and seizures.

Management:

- Atropine IV until drying of secretions.
- Pralidoxime 2 g IV over 15-30 minutes (to regenerate acetylcholinesterase).
- Supportive therapy with oxygen and mechanical ventilation if needed.

References

1. Kitabchi AE, Umpierrez GE, Miles JM, Fisher JN. Hyperglycemic crises in adult patients with diabetes. Diabetes Care. 2009;32(7):1335-1343. doi:10.2337/dc09-9032
2. Umpierrez GE, Korytkowski M. Diabetic emergencies—DKA, HHS, and EDKA. J Clin Endocrinol Metab. 2016;101(5):1233-1244. doi:10.1210/jc.2015-4073
3. Bornstein SR, Allolio B, Arlt W, et al. Diagnosis and treatment of primary adrenal insufficiency: An Endocrine Society clinical practice guideline. J Clin Endocrinol Metab. 2016;101(2):364-389. doi:10.1210/jc.2015-1710
4. Erturk E, Jafar N, Suri V. Management of adrenal crisis in emergency settings. Emerg Med Clin North Am. 2020;38(2):211-223. doi:10.1016/j.emc.2020.01.003
5. Bahn RS, Burch HB, Cooper DS, et al. Hyperthyroidism and other causes of thyrotoxicosis: management guidelines of the American Thyroid Association and American Association of Clinical Endocrinologists. Thyroid. 2011;21(6):593-646. doi:10.1089/thy.2010.0417
6. Ross DS. Thyroid storm. Endocrinol Metab Clin North Am. 2021;50(3):641-652. doi:10.1016/j.ecl.2021.05.008
7. Cairo MS, Bishop M. Tumour lysis syndrome: new therapeutic strategies and classification. Br J Haematol. 2004;127(1):3-11. doi:10.1111/j.1365-2141.2004.05094.x

8. Howard SC, Jones DP, Pui CH. The tumor lysis syndrome. N Engl J Med. 2011;364(19):1844-1854. doi:10.1056/NEJMra0904569
9. Eddleston M, Buckley NA, Eyer P, Dawson AH. Management of acute organophosphorus pesticide poisoning. Lancet. 2008;371(9612):597-607. doi:10.1016/S0140-6736(07)61202-1
10. Peter JV, Moran JL, Graham PL. Advances in the management of organophosphate poisoning. Crit Care. 2014;18(4):218. doi:10.1186/cc13910