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- Dr. Taylor, O.
FOREWORD

I am indeed very pleased to write the foreword to this maiden edition of the Standard Treatment Guidelines (STG) for the Nigerian health care system. I am aware that the process of its production began in 2005 involving contributions and recommendations of various experts and stakeholders in the health care sector.

The STG is an important tool for the attainment of comprehensive and effective health care delivery services thereby achieving the goals of the National Drug Policy, which inter alia are: the availability of safe, efficacious and affordable medicines to satisfy the healthcare needs of the majority of the population and ensure the rational use of drugs. The fulfillment of the above mentioned goals is part of the strategic thrust of the Health Sector Reform Programme aimed at the reduction of disease burden and the improvement of access to quality health services. It is expected that the STG will become a major reference document for all health workers both in the public and private sectors.

It is instructive to note that the development of the STG followed due process with wide consultations and meetings involving various stakeholders and interest groups. The document that has come out of this process is a reflection of the quality of the inputs that went into its development. In my opinion, this maiden edition of the STG has been produced and serialized in such a way as to assist health care providers especially doctors in the effective discharge of their duties as prescribers. It will also ensure discipline as only those medicines recommended will be prescribed for patients within a given health facility.

I commend all those who worked tirelessly towards the completion of this maiden edition STG. Special mention and gratitude must go to the World Health Organization (WHO) for sponsoring and providing sustained technical support to the committee. Without this support, this STG would not have seen the light of the day.

Finally, let me quickly add that this STG must be widely circulated and disseminated. Everything possible must be done to ensure that practitioners maximize the benefit of such a useful document. If it has worked in other parts of the world, it should also work in Nigeria. It must also be subjected to regular reviews in view of the dynamic nature of health care management.

Dr. Hassan Muhammed Lawal, CON
Supervising Minister of Health
PREFACE

This first edition of Standard Treatment Guidelines (STG) for the Nigerian health practitioner is coming relatively later than those of many other countries. It is indeed a welcome development.

The standard of medical practice and the wage bill of health services are usually remarkably improved by health personnel putting to use STG. This among other benefits can only lead to improved health of the community.

In Nigeria our health indices are among the worst in the world. Our country Nigeria does not lack the manpower or the necessary infrastructure to turn things around. What appears to be lacking is the organization of health services required to put both to optimal use. Efforts such as the actualization of our own national STG and the various health reforms currently in progress will definitely improve our situation.

It is therefore my pleasure and privilege to write the preface to this maiden edition of the STG. This is the outcome of a long journey that started several years ago. The previous chairmen of the National Formulary and Essential Drugs Review Committees made efforts to start the project but were unsuccessful due to lack of funds.

The current committee had the luck of being assisted by the country office of the World Health Organization (WHO) in not only this endeavor but in the preparation and printing of the last edition of the Nigerian Essential Medicines List. The desk officer, Dr Ogori Taylor showed great commitment to the project and the country owes a debt of gratitude to WHO.

In preparing this document every effort was made to ensure that the stakeholders own the project so that it is not seen as an imposition. Accordingly, the major contributions came from various practitioners and their associations as well as from many practitioners whose input were judged crucial to the success of the project. We also adopted the acceptable practices in the field that were in use by special health projects such as HIV/AIDS, Malaria, TB/Leprosy programmes etc. The academia was also involved. There were several fora where the contributions were discussed openly with the stakeholders and consensus arrived at.

It is my hope therefore that this document will be widely used by Nigerian health practitioners. I salute the contributors and those that helped in one way or the other. The committee of course accepts responsibility for any lapses but also hopes that these would be brought to our attention for correction in subsequent editions.

Professor Ibrahim Abdu-Aguye, MBBS; FMCP; SFIAM; FIICA; D. Sc (Hon) Chairman, National Formulary and Essential Drugs Review Committee.

SECTION A

Chapter 1: Alimentary Tract
Gastrointestinal Disorders ...................................1
Amoebiasis ..........................................................1
Bacillary Dysentery .................................................1
Cholera ................................................................2
Constipation ..........................................................2
Diarrhoea (acute) .....................................................3
Gastritis ................................................................4
Giardiasis ..............................................................4
Haemorrhoids ........................................................5
Pancreatitis ..........................................................5
Pepic Ulcer Disease ..................................................6
Upper Gastrointestinal Bleeding ...............................6
Hepatic And Biliary Disorders ................................7
Hepatitis ...............................................................7
Hepatic Encephalopathy .........................................8
Jaundice ................................................................9
Liver Cirrhosis ........................................................9
Haemostasis And Bleeding Disorders .....................16
Nutritional Disorders ..............................................10
Kwashiorkor And Marasmus ....................................10
Micronutrient Deficiencies .....................................10
Obesity ................................................................11

Chapter 2: Blood And Blood-forming Organs
Anaemias ............................................................13
Blood Transfusion ..................................................14
Haemostasis And Bleeding Disorders .....................16
Leukaemias ...........................................................16
Lymphomas ...........................................................19
Sickle Cell Disease ..................................................20

Chapter 3: Cardiovascular System
Angina Pectoris .....................................................23
Cardiac Arrhythmias ..............................................23
Congenital Heart Disease ......................................24
Deep Venous Thrombosis ......................................24
Heart Failure .......................................................25
Hyperlipaemia ......................................................26
Hypertension .......................................................26
Infective Endocarditis ............................................27
Myocardial Infarction ..............................................29
Myocarditis ..........................................................30
Paediatric Cardiac Disorders ................................30
Pericarditis ..........................................................30
Pulmonary Embolism ............................................31
Pulmonary Oedema ..............................................32
Rheumatic Fever ...................................................32
Rheumatic Heart Disease ......................................33

Chapter 4: Central Nervous System
Non-psychiatric Disorders ..................................34
Dizziness ............................................................34
Headaches ..........................................................35
Meningitis ...........................................................36
Migraine ............................................................37
Parkinsonism .......................................................38
Seizures/epilepsies ...............................................39
Stroke ................................................................41
Syncope .............................................................42
The Unconscious Patient ........................................42
Psychiatric Disorders .............................................43
Alcoholism (alcohol Dependence) .........................43
Anxiety Disorder ...................................................44
Bipolar Disorders ..................................................44
Delirium .............................................................45
Depression ..........................................................46
Insomnia .............................................................47
Panic Disorder .......................................................47
Schizophrenia .......................................................48

Chapter 5: Dental And Oral Disorders
Acute Necrotizing Ulcerative Gingivitis .................49
Acute Periapical Abscess .......................................49
Alveolar Osteitis ....................................................50
Cellulitis .............................................................50
Dental Caries ........................................................50
Gingivitis ............................................................51
Neoplasms Of The Oral Cavity ...............................51
Oral Thrush (candidiasis) .......................................51
Perioralitis ..........................................................52
Periodontitis ........................................................52
Pulpitis .............................................................53
Salivary Gland Diseases .......................................54
Temporo-mandibular Joint Disorders ....................54

Chapter 6: Dermatology
Bacterial Infections ...............................................56
Cellulitis .............................................................56
Furunculosis (boils) ...............................................56
Impetigo Contagiosa ...............................................57
Dermatitis And Eczema .........................................58
Atopic Dermatitis (atopic Eczema) .........................58
Contact Dermatitis ...............................................59
Exfoliative Dermatitis (erythroderma) .....................59
Parasitic Dermatoses .............................................60
Cutaneous Larva Migrans (creeping Eruption) .....60

TABLE OF CONTENTS
Chapter 1: Alimentary Tract

**AMOEBIASIS**

**Introduction**
A common parasitic infection of the gastrointestinal system caused by the protozoan *Entamoebahistolytica*. Acquired through faeco-oral transmission.

**Clinical features**
- It may present as:
  - *Amebic dysentery*
  - Persistent mucoid/bloody diarrhoea
  - Abdominal pain
  - Fever/chills
- *Amebic abscess*

This can occur in any of the following forms as a result of spread via the bloodstream:
- Liver abscess: swelling, pain in the right sub-costal area
- Intracranial space-occupying lesion
- Lungs: cough and blood stained sputum
- Amoeboma: swelling anywhere in the abdomen

Anal ulceration may occur by direct extension from the intestinal infection.

**Chronic Carriers**
- Symptom-free

**Differential diagnoses**
- *Bacillary dysentery*
- Any other cause of bloody diarrhoea
- Cancer of the liver
- Other causes of liver enlargement

**Complications**
- Rupture of abscess into the lungs, peritoneum
- Space-occupying lesion in the brain
- Right inguinal mass

**Investigations**
- Stool: microscopy for cysts and motile organisms (amoebic dysentery)
- Full Blood Count
- Chest radiograph (in amoebic liver abscess)
- Abdominal Ultrasound Scan

**Treatment objectives**
- Rehydrate adequately
- Eradicate the protozoa

**Drug treatment**
- *Amebic dysentery*
  - Correct dehydration (see section on rehydration)
  - Metronidazole
    - **Adult:** 800 mg 8 hourly for 5 days
    - **Child:** 30 mg/kg/day in 3 divided doses for 5 days
- *Amebic liver abscesses*
  - Metronidazole
    - **Adult:** 800 mg 8 hourly for 10 days
    - **Child:** 50 mg/kg/day in 3 divided doses for 7-10 days

**Non-drug treatment**
- Aspiration is indicated to prevent spontaneous rupture of abscesses.

Consult a surgeon.

**Asymptomatic cyst carriers**
- Treat cyst carrier if patient is a food handler:
  - Diloxanide furoate
    - **Adult:** 500 mg every 8 hours for 10 days
    - **Child:** 25 mg/kg: 20 mg/kg orally every 8 hours for 10 days

**Notable adverse drug reactions**
- Metronidazole is contraindicated in pregnancy.
- Avoid alcohol during treatment and at least 48 hours after treatment.

**Prevention**
- Provision of safe drinking water
- Sanitary disposal of faeces
- Regular examination of food handlers and appropriate treatment where necessary.

**BACILLARY DYSENTERY**

**Introduction**
An important cause of colonic diarrhoea in developing countries.

**Clinical features**
- mucoid bloody diarrhoea associated with severe central and lower abdominal pain
- Tenesmus
- Moderate-grade pyrexia
- Sometimes only a mild, self-limiting diarrhoea lasting 2-3 days
- Articular features occasionally

**Complications**
- Septicaemic spread with multi-system involvement occasionally.

**Differential diagnoses**
- *Amebic dysentery*
- Idiopathic enterocolitis (ulcerative)
- Campylobacter infection
- Colorectal cancer

**Complications**
- Septicaemia/bacteremia
- Severe rectal bleeding
- Intestinal perforation
- Reiter’s syndrome

**Investigations**
- Stool microscopy, culture and sensitivity
- Full Blood Count
- Urea, Electrolytes and Creatinine

**Treatment objectives**
- Rehydrate adequately and rapidly
- Eradicate the infective organism
- Prevent spread of the infection

**Drug treatment**
- Oral Rehydration Therapy (see rehydration under diarrhoea)
- Parenteral hydration therapy (see rehydration under diarrhoea)

**Notable adverse drug reactions**
- Ciprofloxacin may induce tendinitis especially in children.

**Precaution**
- Ciprofloxacin is not recommended for use in children less than 18 years.
- Antidiarrhoeal medicines are not advised.

**Oral Rehydration Therapy**
- *Amebic dysentery*
  - **Adult:** 2000 ml of ORS
  - **Child:** 100 ml of ORS

**Antibiotic therapy**
- **Ciprofloxacin:**
  - **Adult:** 500 mg orally every 6 hours for 5 days

**Chloroquine:**
- **Adult:** 200 mg orally once daily for 5 days
- **Child:** 12 - 18 years, 200 mg on first day, then 100 mg daily

**Erythromycin:**
- **Adult and child over 8 years:** 250 - 500 mg orally every 6 hours for 5 days or 500 mg - 1 g every 12 hours
- **Child up to 2 years:** 125 mg every 6 hours; 2 - 8 years: 250 mg every 6 hours

**Supportive measures**
- Monitor fluid intake and output (vomits, urine and stool)
- Provide access to safe drinking water
- Food hygiene
- Safe disposal of human waste
- Cholera vaccine

**CHOLERA**

**Introduction**
An acute severe diarrhoeal illness of worldwide importance; endemic in many developing countries.

**Clinical features**
- Caused by *Vibrio cholerae El Tor* (classical and *El Tor* species).
- Excessive secretion of fluid is mediated by the release of enterotoxin (released by the bacillus), which acts on the enteroctyes of the small intestine via cyclic AMP.
- Highly infectious; spread by faeco-oral route.

**Clinical features**
- Mild watery diarrhoea
- Severe life-threatening diarrhoea leading to hypovolaemic shock if untreated
- Occasionally, vomiting

**Complications**
- Hypovolaemic shock with multiple end organ failure leading to death
- Hypoglycaemia
- Paralytic ileus

**Investigations**
- Stool microscopy, culture and sensitivity
- Full Blood Count
- Urea, Electrolytes and Creatinine

**Treatment objectives**
- Rehydrate adequately and rapidly
- Eradicate the infective organism
- Prevent spread of the infection

**Drug treatment**
- Intravenous Ringer’s lactate/Darrow’s solutions
- Antibiotic therapy
- Tetracycline:
  - **Adult:** 500 mg orally every 6 hours for 5 days

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**Or:**
- Doxycycline:
  - **Adult:** 200 mg orally once daily for 5 days
  - **Child:** 12 - 18 years, 200 mg on first day, then 100 mg daily
- Severe infections, 200 mg orally daily
- Erythromycin:
  - **Adult and child over 8 years:** 250 - 500 mg orally every 6 hours for 5 days or 500 mg - 1 g every 12 hours
  - **Child up to 2 years:** 125 mg every 6 hours; 2 - 8 years: 250 mg every 6 hours
- Doses doubled in severe infection

**Sulfamethoxazole-trimethoprim (Co-trimoxazole)**
- **Adult:** 960 mg orally every 12 hours for 5 days
- **Child:** 6 weeks - 6 months 120 mg 12 hourly; 6 months - 2 years 240 mg; 6 - 12 years 480 mg; 12 - 18 years 960 mg orally every 12 hours for 5 days

**Supportive measures**
- Monitor fluid intake and output (vomits, urine and stool)
- Provide access to safe drinking water
- Food hygiene
- Safe disposal of human waste
- Cholera vaccine

**CONSTIPATION**

**Introduction**
A clinical condition characterized by infrequent bowel opening and/or passage of hard stools.

**Aetiology**
- Inadequate fibre in diet (simple constipation)
- Drugs e.g. antidepressants, narcotic analgesics, etc
- Diseases of the anus, rectum and colon e.g. fissures, haemorrhoids, cancer
- Functional: Irritable bowel syndrome
- Metabolic diseases e.g. hypothyroidism, hypercalcemia

**Clinical features**
- Stools are often hard
- Abdominal bloating
- Occasional flatulence
- Relevant associated history to determine aetiology

**Or:**
- Senna:
  - **Adult:** 500 mg orally daily
  - **Child:** 125 mg orally daily
- Lactulose:
  - **Adult:** 15 ml orally daily
  - **Child:** 5ml orally daily

**Investigations**
- Stool examination including microscopy
- Proctoscopy/sigmoidoscopy
GIARDIASIS

**Introduction**
A parasitic infection caused by *Giardia lamblia*. Worldwide in distribution but more common in developing countries. Spread by the faeco-oral route.

**Pathogenesis**
Invasion of the upper small intestine by the parasite evokes inflammation, leading to progressive villous atrophy.

**Clinical features**
Acute disease: watery diarrhoea with abdominal pain and dehydration
Chronic disease: diarrhoea, steatorrhoea and weight loss from malabsorption syndrome— with lactose intolerance, xylose malabsorption and vitamin B_{12} deficiency

**Complications**
- Acute gastritis: haemorrhage
- Chronic gastritis: peptic ulcer disease; gastric cancer

**Differential diagnoses**
- Peptic ulcer disease (acute gastritis)
- Celiac disease
- Other causes of gastrointestinal malabsorption such as amoebiasis, giardiasis

**Investigations**
- Stool examination including microscopy, culture and sensitivity
- Full Blood Count
- Urea, Electrolytes and Creatinine
- Serology (e.g. Widal test)
- Histology of gastric biopsy for definitive diagnosis

**Treatment**
- RANITIDINE 150 mg orally once daily as required
- OMEPRAZOLE 20 mg orally once daily as required
- THERAPEUTIC ENDOSCOPY
- Antibiotics: metronidazole 750 mg orally every 8 hours for 7 days
- Clarithromycin 500 mg orally twice daily for 7 days
- Doxycycline 100 mg orally twice daily for 7 days

**Prevention**
- Avoid risk factors (NSAIDs, alcohol, etc)

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- Clarithromycin 500 mg orally twice daily for 7 days
- Doxycycline 100 mg orally twice daily for 7 days

**Prevention**
- Avoid risk factors (NSAIDs, alcohol, etc)
Notable adverse drug reactions
Metallic taste and vomiting from metronidazole

Prevention
Good sanitary habits
Uncontaminated water and food supplies

HAEMORRHOIDS
Introduction
Enlarged or varicose veins of the tissues at the anus or rectal outlet.
Engorgement of the vascular complex or thrombus often leads to the symptoms of disease.
The pathophysiologic mechanisms are complex and vary with the subject.
May be external or internal.

Clinical features
Internal haemorrhoids: typically painless but present with bright red rectal bleeding
May become thrombosed and protrude into the anal canal
External haemorrhoids when thrombosed cause acute perineal pain with or without necrosis and bleeding
Fibrosed external haemorrhoids present as anal tags

Differential diagnoses
Colorectal cancer
Adenomatous polyps
Inflammatory bowel disease

Complications
Bleeding, necrosis, perineal sepsis, mucus discharge

Investigations
Analoscopy
Full blood count including blood film

Treatment objectives
Relieve pain
Prevent complications

Non-drug treatment
Increase fibre in foods
Increase fluid intake
Avoid foods that cause constipation
Stool softeners
Regular exercise

Drug treatment
Suppositories/ointments of preparations containing hydrocortisone acetate with or without lidocaine hydrochloride plus astringent(s)

Surgery
Elastic band ligation
Sclerosis, photoocoagulaton, cryosurgery, excisional haemorrhoidectomy

Caution
Each drug treatment course should not exceed 7 days

PANCREATITIS
Introduction
A state of inflammation of the pancreas, which can be acute or chronic.

Aetiology
Varied, but most important are:
Gallstones
Alcohol ingestion
Abdominal trauma
Infections
Idiopathic in as many as 20-30% cases
Occurrence is worldwide, but commoner in areas of the world where gallstones and alcohol ingestion are common.

Pathophysiology
Autolysis of pancreatic tissue by pancreatic enzymes as a result of “secretory block” in the pancreatic bed (often caused by stones).

Clinical features
Acute pancreatitis:
Epigastric pain: may radiate to the back in over 50% of cases
Nausea, vomiting, abdominal distension
Severe abdominal tenderness with features of hypovolaemia in severe cases

Differential diagnoses
Peptic ulcer disease
Cholecystitis

Investigations
Serum amylase: raised in 80% of acute cases
Serum lipase: if raised is more specific than serum amylase
Alanine aminotransferase: a rise above 3-fold suggests pancreatitis of gallstone origin
CT scan
Abdominal ultrasound: least useful in acute pancreatitis

Complications
Hypovolaemic shock
Acute renal and respiratory failure
Pneumomediastinum
Gastrointestinal bleeding
Electrolyte imbalance (hypo & hypercaemia)
Pancreatic pseudocysts

Treatment objectives
Relieve pain
Prevent complications

Non-drug treatment
Renal failure: haemodialysis
Respiratory failure: mechanical ventilation
Gallstones: Endoscopic Retrograde Cholangio Pancreatography (ERCP) with sphincterotomy
Pancreatic pseudocyst; surgery

Drug treatment
Analgesics
Treat specific complications

Supportive measures
Bed rest
Monitor vital signs; fluid intake/output

Investigations
Full Blood Count
Liver Function Tests
Urea, Electrolytes and Creatinine
Occult blood test
Stool microscopy
Endoscopy

Differential diagnoses
Gastritis
Duodenitis
Non-Ulcer Dyspepsia
Gastro-duodenal malignancy
Oesophagitis
Gall bladder diseases

Treatment objectives
Relieve pain
Promote healing of ulcers

Surgery
Gastric outlet obstruction

UPPER GASTROINTESTINAL BLEEDING
Introduction
Bleeding from the lower oesophagus, stomach or duodenum up to the level of ligament of Treitz.
Occurs worldwide and is responsible for significant mortality and morbidity.
Major causes include bleeding from:
- Peptic ulcer disease
- Oesophageal and gastric varices
- Mallory-Weiss tear
- NSAID-related mucosal bleeding
- Neoplasia
Bleeding is either from rupture of engorged varices or from disruption of the oesophageal or gastro-duodenal...
**Clinical features**
Depends on whether the bleeding is acute or chronic, mild or severe.

**Various presentations**
- Haematemesis
- Melaena
- Haematochezia
- Hypovolaemia
- Iron deficiency anaemia (with its associated symptoms)

**Differential diagnoses**
Black stools from ingestion of iron tablets
Haematemesis/melaena from previously swallowed blood (from the upper respiratory tract and oral cavity)

**Complications**
- Hypovolaemic shock
- Congestive heart failure from chronic severe anaemia

**Investigations**
Upper gastrointestinal endoscopy: picks up lesions in 90% of cases
- Upper gastrointestinal barium radiography: 80% detection rate
- Selective mesenteric arteriography
- Radio isotope scanning
- Stool- occult blood test
- Full Blood Count

**Treatment objectives**
- Restore and maintain haemodynamic status
- Control bleeding
- Prevent recurrence of bleeding

**Non-drug treatment**
- Carefully monitor vital signs (pulse, blood pressure, respiration and temperature) as frequently as necessitated by the patient's condition
- Insert a nasogastric tube to aspirate gastric contents and/or to introduce agents to constrict the blood vessels
- Blood transfusion: whole blood (acute bleeding) or packed cells (chronic) bleeding. Up to 5 - 6 pints of blood may be needed in severe cases
- Plasma expanders in the absence of blood

**Continuous Central Venous Pressure (CVP) monitoring**

**Drug treatment**

**Bleeding peptic ulcers/esions**
- Proton Pump Inhibitors
  - Omeprazole 40 mg orally once daily for 4 weeks
  - Omeprazole 20 mg orally once daily for 4 weeks

**Hepatitis A**

**Introduction**
Inflammation of the liver that can be caused by infectious agents, drugs and other toxins

**The most predominant and important presentation of liver disease worldwide**
- The suffixes acute, chronic, viral, autoimmune, alcoholic etc. define the agents causing hepatic injury or their duration as the case may be

**Hepatitis and Biliary Disorders**

**Hepatitis A**

**Aetiology**
- Varies, depending on geographical region:
  - Viruses, alcohol and drugs are the commonest aetiologic agents

**Important risk factors**
- Family history
- Alcohol ingestion
- Previous blood transfusion
- Contamination of food and water by sewage
- Drug ingestion
- Sexual contact

**Clinical features**
Acute hepatitis:
- Mild-to-moderate jaundice
- Vague upper quadrant discomfort
- With or without mild fever
- There may be enlargement of the liver below the costal margin with varying consistency (depending on the stage of the liver disease)

Chronic hepatitis:
- Re-occurrence of jaundice may suggest a chronic illness

**Differential diagnoses**
- Liver abscess
- Metabolic liver disease/disorder

**Complications**
- Fulminant hepatic failure
- Bleeding tendencies

**Investigations**
- Liver Function Tests
- Serologic markers of Hepatitis A, B, C, D and E
- Abdominal ultrasonography

**Treatment objectives**
- Provides supportive measures
- Prevent progression to chronic phase
- Non-drug treatment
  - High carbohydrate and low protein diet
  - Discontinuation of hepatotoxic medication

**Drug treatment**
**Hepatitis A**

**Acute**
- Self-limiting disease. No specific drug treatment

**Hepatitis B**

**Acute**
- Self-limiting to fulminant
  - Treatment is supportive

**Chronic**
- Interferon alfa -2b: 10 million units subcutaneously 3 times weekly for 4 months
- Lamivudine: 100 mg orally daily for 1 year

**Liver transplant**
- Chronic Hepatitis C:
  - Interferon alfa -2b
  - 3 million units subcutaneously 3 times weekly for 4 months

**Ribavirin**
- 400 mg orally twice daily for adults with body weight less than 65 kg; 400 mg in the morning and 600 mg in the evening for adults weighing 65-85 kg; 600 mg twice daily for adults weighing over 85 kg

**Hepatitis D**
- Interferon alfa -2b: 3 million units subcutaneously 3 times weekly for 4 months
- Lamivudine: 100 mg orally once daily for 4 months

**Hepatitis E**
- Largely supportive

**Notable adverse drug reactions**
- Interferon alfa 2b and Ribavirin haematoapoetic toxicity
- Flu-like illness
- Leucopenia
- Psychiatric-like symptoms
- Development of early resistance if therapy exceeds 1 year

**Prevention**
- Prevention of faecal contamination of food and water
- Screen blood and blood products for hepatotrophic viruses
- Immunization against hepatitis A, B
- Reduction of drug misuse/abuse
- Pre-exposure prophylaxis (as for NPI/EPI)
- Post-exposure prophylaxis

**Hepatitis C**

**Introduction**
A state of disturbed central nervous system function as a result of hepatic insufficiency
- Characterized by changes in personality, cognition, motor function, level of consciousness
- One-year survival rate is 40%
- Nitrogenous substances, particularly ammonia, reach the brain via portosystemic shunts leading to alteration of neurotransmission

**Predisposing factors**
- Reduced blood supply to the liver
- Infection of the liver
- Bleeding into the gut
- Electrolyte imbalance (hypokalaemia and hypomagnesaeimia)
- Poor bowel evacuation

**Clinical features**
- Cognitive abnormalities: may be mild and recognizable only with psychometric testing but may be severe with frank confusion, altered level of consciousness and coma
- Hyper-reflexia
- Feter hepaticus
- Insomnia
- Flapping tremor (asterixis)

**Differential diagnoses**
- Intracranial lesions (haemorrhage, tumour, abscess etc.)
Investigations
LFTs: determine levels and nature of bilirubin, liver enzymes (AST, ALT, Alkaline phosphatase)
Abdominal ultrasound scan: look out for canalicular dilatation and stones

Clinical features
Ascites and pedal oedema
Screening for aetiologic factors in chronic liver disease
e.g. viral markers for hepatotrophic viruses (e.g. Hepatitis B & C)

Prevention
Abstinence from alcohol
Reassurance and monitoring
Phototherapy in neonatal jaundice

LIVER CIRRHOSIS
Introduction
An advanced stage of chronic liver disease associated with permanent distortion of the liver architecture and replacement of some destroyed hepatocytes with fibrous tissue

Diagnosis
A common clinical state of varying aetiologies
Classified as haemolytic, hepatic or obstructive
Clinical jaundice occurs when the level of serum bilirubin exceeds 2.5 mg/dL
The bilirubin may be conjugated, unconjugated or mixed

HAEMOLYTIC JAUNDICE
Definition
Deficiencies of minerals (iron, iodine, zinc, calcium, phosphorus, magnesium, copper, potassium, sodium, chloride, fluoride etc); folic acid and vitamins

Jaundice
A common clinical state of varying aetiologies
Classified as haemolytic, hepatic or obstructive
Clinical jaundice occurs when the level of serum bilirubin exceeds 2.5 mg/dL
The bilirubin may be conjugated, unconjugated or mixed

Important causes
Diseases of the liver and the biliary tract
Conditions that cause excessive red cells haemolysis: infections, haemoglobinopathies

Clinical features
Discolouration of the sclera and other mucus membranes
With or without pruritus (especially with cholestatic jaundice)

Hepato-renal syndrome
Investigations
LFTs
PT, PTTK, Serum albumin
Liver biopsy
Ultrasonic examination of the liver

Screening for aetiologic factors in chronic liver disease
e.g. viral markers for hepatotrophic viruses (e.g. Hepatitis B & C)

Treatment objectives
Prevent further liver damage
Prevent deterioration of liver function
Symptomatic relief from anaemia, fatigue and oedema

NON-DRUG TREATMENT
Encourage high fibre and low salt diet
Enhance opening of bowel
Correction of anaemia
Reduce oedema and ascites

Drug treatment
Ascites and pedal oedema
Spironolactone tablets 25 - 100 mg orally 12 hourly
Furosemide 20 - 80 mg orally 12 hourly
Salt-poor albumin for intractable ascites

LIDDLE'S SYNDROME
Definition
A condition characterized by hypertension and hypokalaemia

Aetiology
Increased aldosterone production

Clinical features
High blood pressure
Low potassium levels

Complications
Cardiovascular disease
Renal damage

NUTRITIONAL DISORDERS
KWASHIORKOR AND MARASMS
Introduction
Adequate nutrition is the intake and utilization of energy-giving and body building foods and nutrients, to maintain well-being and productivity

Malnutrition includes generalized malnutrition that manifests as stunting, underweight, wasting (kwashiorkor and marasmus), obesity as well as deficiencies of micronutrients.

Kwashiorkor is protein-energy malnutrition
Marasmus is malnutrition resulting from inadequate calorie intake

Nutritional counselling
Adequate nutrient intake: may require assistance and special preparations e.g. nasogastric feeding, etc.
Periodic growth monitoring

Drug treatment
May be indicated where there are specific infections/infestations

Micronutrient deficiencies
Deficiencies of minerals (iron, iodine, zinc, calcium, phosphorus, magnesium, copper, potassium, sodium, chloride, fluoride etc); folic acid and vitamins

Aetiology
Defective dietary intake
Increased requirements
Increased loss (e.g. worm infestation)

Epidemiology
Global; high percentages in under-developed countries, especially sub-Saharan Africa

Clinical features
Iron: anaemia
Iodine: goitre
Zinc, copper: manifestations of enzyme and insulin deficiencies
Calcium: rickets, osteomalacia
Phosphorus and fluoride: teeth and bone abnormalities
ANAEMIAS

Introduction
Anaemia is a reduction in the haemoglobin concentration in the peripheral blood below the normal range expected for the age and sex of an individual.

The determination of haemoglobin concentration should always take the state of hydration and altitude of residence of the individual into consideration.

Investigations
- Blood, urine and stool tests
- Other investigations as appropriate

Treatment objectives
- Correct nutrient deficiencies
- Ensure adequate intake
- Prevent complications

Treatment
- Administration of specific nutrients (as concentrates in foods)
- Food supplementation
- Treat underlying diseases

Prevention
- Nutritional counselling
- Optimal breastfeeding and appropriate weaning practices
- Adequate intake of locally available, nutritious foods
- Personal/food/water hygiene
- Prophylactic therapies for malaria

OBESSION

Introduction
A major component of the metabolic syndrome. Being overweight or obese significantly increases the risk of morbidity and mortality from Type 2 diabetes and its co-morbidities.

Successful weight reduction has a positive impact on morbidity and mortality outcomes.

Obesity is a result largely of diet and lifestyle.

Measurements for evaluation
- Body mass index (BMI): calculation for overall obesity
- Waist circumference: determination of central fat distribution

BMI is calculated as follows

\[
BMI = \frac{weight \text{ in kg}}{height \text{ in m}^2}
\]

Classification of BMI
- Underweight: <18.5 kg/m²
- Normal weight: 18.5 - 24.9 kg/m²
- Overweight: 25 - 29.9 kg/m²
- Obesity (Class 1): 30 - 34.9 kg/m²
- Obesity (Class 2): 35 - 39.9 kg/m²
- Extreme obesity (Class 3): >40 kg/m²

BMI represents overall adiposity

The pattern of distribution of fat in the body (whether mostly peripheral or central) is assessed by the use of the waist/hip ratio (WHR)

\[
\text{WHR} = \frac{Waist \text{ circumference (in cm)}}{Hip \text{ circumference (in cm)}}
\]

Waist circumference: measure midway between the lower rib margin and the iliac crests

Hip circumference: the largest circumference of the hip

- Upper limits: 102 cm and 88 cm in men and women respectively
- Always bear in mind the possibility of an underlying cause: although these may not be common, specific therapy may be available
- Clinical presentation may therefore require specific investigations to exclude conditions such as
  - Hypothyroidism
  - Hypercortisolism
  - Male hypogonadism
  - Insulinoma
  - CNS disease that affects hypothalamic function

Complications
- Cardiovascular:
  - Coronary disease
  - Stroke
  - Congestive heart failure
- Pulmonary:
  - Obstructive sleep apnoea
  - 'Obesity hypoventilation syndrome'
- Endocrine:
  - Insulin resistance and type 2 diabetes mellitus
- Hepatobiliary:
  - Gall stones
- Reproductive:
  - Male hypogonadism
  - Menstrual abnormalities
  - Infertility
- Cancers:
  - In males, higher mortality from cancer of the colon, rectum and prostate
  - In females, higher mortality from cancer of the gall bladder, bile ducts, breasts, endometrium, cervix and ovaries
  - Bone, joint and cutaneous disease:
    - Osteoarthritis
    - Gout
  - Acanthosis nigricans
  - Increased risk of fungal and yeast infections
  - Venous stasis

Treatment objectives
- To educate patient and care givers
- Achieve an ideal body weight

Prevent complications

Management
Assess dietary intake, level of physical activity, BMI (total body fat) and waist circumference (abdominal fat) on presentation and at regular monitoring.

Assess efficacy of weight loss measures
Integrate weight control measures into the overall management of diabetes mellitus and co-morbidities if
- BMI is >25
- Waist circumference is more than 102 cm and 88 cm in men and women respectively
- Maintain records of goals, instructions and weight progress charts
- Surgical intervention may be required in extreme cases
Types of blood transfusion

Standard Treatment Guidelines for Nigeria 2008

**Platelet count**

**Erythrocyte sedimentation rate**

**Blood film examination for morphology of cells**

**Thick and thin films for malaria parasites**

**Urine analysis:**

- Colour, pH, clarity, specific gravity
- Microscopic examination of fresh urine specimen
- Protein
- Glucose
- Occult blood

**Stool:**

- Colour, consistency
- Examination for ova and parasites
- Occult blood

**Plasma:**

- Blood Urea Nitrogen (BUN)
- Total protein and albumin
- Bilirubin
- Creatinine (if BUN is abnormal)

**Others:**

- Coombs test for the presence of antibodies to red cells
- Ham's test (acidified serum test)
- Bone marrow aspiration and trephine biopsy
- Haemoglobin electrophoresis
- Sickle test (metabisulphite and solubility)
- Family studies

**Treatment objectives**

- Restore haemoglobin concentration to normal levels
- Prevent/treat complications

**Supportive measures**

- Bed rest in severe cases: initially necessary, especially when cardiovascular symptoms are prominent
- Treat cardiac failure by standard measures
- Balanced diet with adequate protein and vitamins
- Correct dietary deficiencies (e.g. iron, folic acid)

**Drug treatment**

- Haematinics e.g. iron, vitamin B₁₂, folic acid
- The specific haematonic indicated should be given alone
- Response to adequate treatment is important in confirming diagnosis
- Iron deficiency
- Oral iron therapy:
  - Ferrous sulfate 200 mg (containing 65 mg of iron) 1 tablet 2-3 times daily
  - Treat for 3-6 months to correct deficits in haemoglobin and body stores
- Parenteral therapy:
  - Not necessary unless there is intolerance to oral iron

**Indications for parenteral iron:**

- Anaemia diagnosed in late pregnancy
- Correction of anaemia just before an operative procedure
- Haemorrhage expected to continue unabated
- Iron dextran given as “total dose” infusion
- Dose in mL (of 50 mg/mL preparations) = [Patient's wt. in kg X (14 Hb in g/dL)] + 10

**Notable adverse drug reactions, caution**

**Oral iron preparations:**

- Nausea, epigastric pain, diarrhoea, constipation, skin eruptions
- Reduce dosage and frequency of administration to reduce these effects

**Parenteral iron:**

- Local reactions: phlebitis and lymphadenopathy
- Systemic reactions: may be early or late: headache, fever, vomiting; general aches and pains, backache, chest pain, dyspnoea, syncope; death from anaphylaxis
  - A test dose should be administered: 25 mg intramuscularly or by intravenous infusion over 5 to 10 minutes
  - Total-dose infusion should be avoided in patients with history of allergy

**Haemolytic anaemia**

- Response to therapy is satisfactory if administered dose is limited to the minimal daily requirement
- Treatment with vitamin B₁₂ (cobalamin) to replace body stores
  - Six 1000 micrograms intramuscular injections of hydroxocobalamin given at 3-7 day intervals
- Maintenance therapy: patients will need to take vitamin B₁₂ for life
  - 1000 micrograms hydroxocobalamin intramuscularly once every 3 months

**Notable adverse drug reactions, caution**

- Toxic reactions are very rare and are usually not due to cobalamin itself
- Pharmacologic doses of folie acid produce haemato logical response in vitamin B₁₂-deficient patients but worsen the neurological complications
- Large doses of vitamin B₁₂ also give haematological response in folate-deficient patients

**Prevention**

- Balanced diet
- Prompt treatment of all illnesses

**BLOOD TRANSFUSION**

**Introduction**

- Blood transfusion is the administration of blood for therapy
- It is potentially hazardous: blood should be given only if the dangers of not transfusing outweigh those of transfusion.

**Indications** (must be clearly established)

- Transfusion of whole blood or red cell concentrates is important in the treatment of acute blood loss and of anaemia
- Red cells can be stored at 4°C for 5 weeks in media that are specially designed to maintain the physical and biochemical integrity of the erythrocytes and which maintain their viability after transfusion
- Citrate Phosphate Dextrose with Adenine (CPDA) is commonly used for collections of whole blood
- The use of whole blood as a therapeutic agent has been almost completely replaced by the use of blood fractions

**Types of blood transfusion**

- Autologous blood transfusion:
  - Transfusion of the patient's own blood to him/her
  - Safest blood for patients
- The three main types are:
  - Pre-deposit autologous transfusion
  - Immediate pre-operative phlebotomy with haemodilution
  - Intra-operative blood salvage

**Exchange transfusion**

- To remove deleterious material from the blood, for example, in severe jaundice resulting from haemolytic disease of the newborn
- Alternatives to red cell transfusion:
  - Perfluorochemicals such as Fluosol-DA
  - Polymerised haemoglobin solutions with good intravascular recovery

**Indications for blood transfusion**

- Symptomatic anaemias:
  - Recurrent haemorrhage
  - Haemolysis
  - Bone stem cell failure
  - Pure red cell aplasia
  - Severe anaemia of chronic disorders
  - Haematological malignancies (e.g. leukaemia, lymphoma)
  - Chemotherapy complicated by anaemia
  - In neonates:
  - Severe acute haemorrhage
  - Haemolytic disease of the newborn
  - Septicaemia
  - Prematurity
  - Bleeding disorders:
    - Congenital e.g. haemophilia
    - Acquired e.g. disseminated intravascular coagulopathy
  - Prevention or treatment of shock:
    - Clinical situations in which there is need to restore and/or maintain circulatory volume e.g. trauma, haemorrhage
    - To maintain the circulation (as in extracorporeal or cardiac by-pass shunts)

**Whole blood preparations**
Clinical features
- General symptoms of anaemia
- Bleeding
- Infections
- Anorexia
- Weight loss
- Lymphadenopathy (not common in AML except in the monocytic variant)
- Skin:
  - Macules, papules, vesicles
  - Pyoderma gangrenosum
  - Neutrophilic dermatitis
  - Leukaemic cutis
- Granulocytic sarcoma

Diagnostic features
- Septicaemia
- Miliary tuberculosis
- Malignant histiocytosis

Complications
- Worsening ill-health
- Infections
- Post-transfusion purpura

Investigations
- Full blood count with ESR, reticulocyte count
- Coomb's test
- Bone marrow examination
- Biochemical tests: serum electrolytes, urea, creatinine, uric acid
- Liver function tests
- Prothrombin time, partial thromboplastin time
- Human Leucocyte Antigen typing
- HIV I and II
- Cytochemical tests
- Acetate esterase
- Bone marrow cultures
- Cytogenetic studies
- Electron microscopy
- Immunological classification
- Terminal deoxynucleotidyl transferase demonstration in nuclei of B and T lymphocytes
- Abdominal ultrasound/CT scans

Drug treatment
- Furosemide 40 mg on administration of one unit of blood
- Hydrocortisone sodium succinate 100 mg injection
- Appropriate nutrition
- Adequate hydration

Notable adverse drug reactions, caution
- Furosemide: dehydratation and hypersensitivity
- Immediate: drowsiness, hypersensitivity
- Prevention:
  - Avoid/prevent accidents

ECMOSIS AND BLEEDING DISORDERS - refer for specialist care

LEUKAEMIAS

Introduction
- A heterogeneous group of diseases characterized by infiltration of the blood, bone marrow and other tissues by neoplastic cells of the haematopoietic system
- Two main types:
  - Myeloid leukaemia
  - Lymphoid leukaemia
- Each is further divided into acute and chronic
- Acute leukaemias are defined pathologically as blast cell leukaemias or malignancies of immature haematopoietic cells. The bone marrow shows > 30% blast cells:
  - Two main groups of acute leukaemias:
    - Acute myeloid leukaemia (AML)
    - Acute lymphoblastic leukaemia (ALL)
  - Childhood leukaemias: patients aged < 15 years
  - Adult leukaemias: patients aged ≥ 15 years
  - Leukaemias in adults aged > 60 years: an important group because:
    - Their responses to current treatment protocols both for ALL and AML are inferior
    - These patients are not usually considered for more radical treatment approaches such as autologous or allogeneic bone marrow transplantation
- 80% of adult cases: AML

Epidemiology/predisposing conditions
- Acute lymphoblastic leukaemia (ALL) and Acute myeloid leukaemia (AML)
- More common in industrialized than rural areas
- Environmental agents implicated in the induction of certain types of leukaemia:
  - Ionising radiation: X-rays and other ionizing rays
  - Chemical carcinogens
  - Benzene and other petroleum derivatives
  - Alkylating agents
  - Host susceptibility e.g. genetic disorders:
    - Bloom's syndrome
    - Fanconi's anaemia (AML)
    - Ataxia telangiectasia (ALL)
    - Down's syndrome
- Blast transformation in pre-existing myeloproliferative disorders:
  - Chronic myeloid leukaemia
  - Chronic lymphoid leukaemia
  - Chronic monocytic leukaemia

Treatment objectives
- Induce remission to achieve complete remission
- Maintain disease-free state

Non-drug treatment
- Appropriate nutrition
- Adequate hydration (at least 3 litres/24 hours)
- Erythrocyte transfusion as required
- Platelet concentrate transfusion as required
- Maintain electrolyte balance
**Drug treatment**

**Acute lymphoblastic leukaemia**
- Allopurinol 300 mg daily orally

**DVP Regime**
- Daunorubicin 30 mg/m² intravenously on days 8, 15, 22 and 29
- Vincristine 1.4 mg/m² to a maximum of 2 mg intravenously on days 8, 15, 22 and 29
- Prednisolone 60 mg orally once daily from day 1 to 28

**Maintenance**
- COAP for 14 days
- Prednisolone 40 mg/m² orally for 14 days
- Nervous system prophylaxis is not required
- Assess for remission after 3 courses

**Chronic Myeloid Leukaemia (CML)**
- Intrathecal treatment as for ALL if there is CNS disease of the monocytic type
- Allopurinol 300 mg daily orally
- Daunorubicin 30 mg/m² intravenously on days 8, 15, 18, 21, 24, 27, 30 and 33

**Prevention**
- COAP every 6 weeks for 2 years
- Intrathecal treatment as for ALL if there is CNS disease

**Chronic Lymphocytic Leukaemia**
- Standard Treatment Guidelines for Nigeria 2008

**Clinical features**
- Asymptomatic
- Abdominal swelling/pain
- Lethargy
- Shortness of breath on exertion
- Weight loss
- Unexplained haemorrhage at various sites e.g. gums, intestinal/urinary tracts
- Increased sweating
- Visual disturbances
- Gout
- Priapism
- Splenomegaly
- Anaemia
- Haemorrhage
- Fever
- Lymphadenopathy (rare in chronic phase)

**Complications**
- Blastic transformation
- Death

**Investigations**
- As above for acute leukaemia plus:
  - Determination of Philadelphia chromosome
  - Lactic dehydrogenase
  - Serum calcium

**Treatment objectives**
- Induce remission to achieve complete remission
- Maintain disease-free state
- Achieve absence of Philadelphia chromosome

**Non-drug treatment**
- Appropriate nutrition
- Adequate hydration
- Electrolyte balance

**Drug treatment**
- Hydroxyurea: 9 million units subcutaneously or intravenously twice weekly for 6-12 months
- Imatinib: 400 mg orally daily
- To be used strictly under specialist supervision

**Notable adverse drug reactions, caution**
- The above drugs (except the steroids) all cause profound myelosuppression
- Prophylactic nausea, vomiting, diarrhoea and abdominal discomfort
- Secondary malignancies
- Steroids: Cushing’s syndrome, hypertension, diabetes mellitus, immunosuppression, infections
- Vincristine: neurotoxicity
- Cyclophosphamide: alopecia, haemorrhagic cystitis
- Daunorubicin: myelosuppression, alopecia, cardiotoxicity

**Prevention**
- Avoid exposure to ionizing radiation
- Early detection and treatment

**Chronic Lymphocytic Leukaemia**
- Neoplastic proliferations of mature lymphocytes
- The diseases involve the blood bone marrow and other tissues

**Clinical features**
- Characterized by accumulation of small mature-look ing lymphocytes in the blood, marrow and lymphoid tissues
- B-cell disorders are more common
- B-cell CLL is more common in males than females
- Accounts for 60% of cases
- Rarely diagnosed below the age of 40 years

**Differential diagnoses**
- Low grade non-Hodgkin’s lymphomas with frequent blood and bone marrow involvement (leukaemia/lymphoma syndromes)
- Tuberculosis
- Viral infections
- Toxoplasmosis

**Complications**
- Richter transformation
- Progression of disease

**Investigations**
- Cell morphology:
  - Size
  - Nuclear: cytoplasmic (N:C) ratio
  - Regularity or irregularity of the nuclear outline
  - Characteristics of the cytoplasm (presence and length or absence of azurophil granules)
- Degree of nuclear chromatin condensation and its pattern
- Prominence, frequency and localization of the nucleolus

**Investigations**
- As for anaemia and other leukaemias

**Treatment objectives**
- Induce remission to achieve complete remission
- Maintain disease-free state

**Non-drug treatment**
- Appropriate nutrition
- Adequate hydration
- Maintenance of electrolyte balance
- Bone marrow transplant
Chapter 2: Blood and Blood-Forming Organs

Red cell and platelet concentrate transfusion as required

**Drug treatment**

**Chronic Lymphocytic Leukaemia**
- Allopurinol 100 mg orally every 8 hours
- Chlorambucil 5 mg/m² orally on days 1 to 3
- Prednisolone 75 mg orally on day 1; 50 mg orally on days 2 and 25 mg orally on day 3
- Repeat every 2 weeks

**Investigations**
- Full Blood Count (i.e. haemoglobin, haematocrit, leucocyte and differential counts; red cell indices, reticulocyte count)
- Erythrocyte sedimentation rate
- Coombs test
- Bone marrow aspiration and needle biopsy
- Serum Urea, Electrolytes
- Serum Uric acid
- Liver Function Tests: transaminases-ALT, AST, ALP, bilirubin; serum proteins
- HIV screening
- Immunoglobulins
- Chest X-ray

**Pathophysiology**
- Polymerization of the sickle haemoglobin may adhere to vascular endothelium, increasing the potential for decreased blood flow and vascular obstruction.
- Notable adverse drug reactions, caution: Many of the drugs are contraindicated in patients with hypersensitivity reactions to the respective medicines.
- Appropriate nutrition
- Adequate hydration
- **Supportive measures**
- Appropriate nutrition
- Adequate hydration
- Red cell and platelet concentrate transfusions as required

**Sickle cell-ß+ thalassaemia. Type I**
- Prednisolone 100 mg orally on days 1 - 8
- Prednisezone 40 mg orally on days 1 - 14
- Prednisezone 100 mg orally days 1 - 5
- Repeat every 3 weeks

**Supportive measures**
- Appropriate nutrition
- Adequate hydration

**Sickle cell crises**
- Vincristine 1.4 mg/m² (maximum 2 mg) on days 1 and 8
- Prednisolone 100 mg orally on days 1 - 8

**Hodgkin's lymphoma**
- MOPP
- Mechloretamine 6 mg/m² intravenously on days 1 and 8
- Vincristine 1.4 mg/m² (maximum 2 mg) intravenously on days 1 and 8
- Prednisolone 100 mg orally on days 1 - 8
- Procarbazine 100 mg orally on days 1 - 8
- CHOP
- Prednisezone 40 mg orally on days 1 - 14
- Prednisezone 40 mg orally on days 1 - 14

**Supportive measures**
- Appropriate nutrition
- Adequate hydration

**Notable adverse drug reactions, caution**
- All the drugs are contraindicated in patients with hypersensitivity reactions to the respective medicines.
- Profound nausea, vomiting, diarrhoea and abdominal discomfort
- Secondary malignancies
- Myelosuppression (except the steroids)
- Steroids (prednisezone) may cause Cushing's syndrome, hypertension, diabetes mellitus, suppression of immunity, infections
- Vincristine: neurotoxic
- Cyclophosphamide: alopecia and haemorrhagic cystitis
- Doxorubicin: cardiotoxic
- Prevention
- Avoid unnecessary exposure to irradiation and chemicals

**Lymphomas**

**Introduction**
- Solid neoplasms that originate in lymph nodes or other lymphatic tissues of the body
- A heterogeneous group of disorders
- Can arise at virtually any site
- More often occurs in regions with large concentrations of lymphoid tissues, e.g. lymph nodes, tonsils, spleen and bone marrow
- Two main groups:
  - Hodgkin's disease
  - Non-Hodgkin's lymphomas

**Hodgkin's disease**
- Characterized by Reed-Sternberg cells (large binucleate cells with vesicular nuclei) and prominent eosinophilic nucleoli
- Reed-Sternberg cells are occasionally found in other clinical conditions e.g. hyperplastic or inflammatory lesions of lymph nodes
- Non-Hodgkin's lymphomas: a heterogeneous collection of lymphoproliferative malignancies

**Clinical features**
- Vary widely from one patient to another:
  - Persistent anaemia/pallor
  - Growth retardation (variable)
  - Jaundice (variable)
  - Bone pains (recurrent)
  - Abdominal ultrasound scans
  - CT scans of chest and abdomen
  - Supplementary node biopsy

**Non-Hodgkin's lymphomas**
- CHOP (3 weekly):
  - Cyclophosphamide 750 mg/m² intravenously on day 1
  - Doxorubicin 50 mg/m² intravenously on day 1
  - Vincristine 1.4 mg/m² (maximum of 2 mg)
  - Prednisezone 100 mg orally on days 1 - 5
  - Prednisezone 100 mg orally on days 1 - 5

**CHOP (4 weekly):**
- Cyclophosphamide 750 mg/m² intravenously on days 1 and 8
- Doxorubicin 25 mg/m² intravenously on days 1 and 8

**Sickle cell-ß+ thalassaemia. Type III**
- Prednisezone 100 mg orally on days 1 - 8

**Sickle cell trait**
- Inheritance of one normal gene controlling formation of B Haemoglobin (HbB), and a sickle gene (HbS)
- Total haemoglobin A is more than haemoglobin S
- Normal haemoglobin F

**Sickle cell disease**
- Inheritance of two abnormal allelic genes controlling formation of B chains of haemoglobin, at least one of which is the sickle gene
- Polymerization of the sickle haemoglobin may lead to vaso-occlusion

**Pathophysiology**
- Red cells have reduced deformability and easily adhere to vascular endothelium, increasing the potential for decreased blood flow and vascular obstruction.
- Abnormalities in coagulation, leucocytes, vascular endothelium, and damage to the membranes of red cells contribute to sickling
- Haemolytic anaemia and vasoocclusion are the result of the various pathophysiological processes
- Organ damage is on-going and is often silent until far advanced
- The course of the disease is punctuated by episodes of pain

**Prevention**
- Avoid chemicals on body (e.g. benzene)
- Avoid ionizing radiation (X rays)
- Early detection and treatment

**Sickle cell-ß+ thalassaemia. Type II**
- Prednisezone 100 mg orally on days 1 - 8

**Sickle cell-ß+ thalassaemia. Type I**
- Prednisezone 100 mg orally on days 1 - 8

**Sickle cell-ß+ thalassaemia. Type II**
- Prednisezone 100 mg orally on days 1 - 8

**Sickle cell-ß+ thalassaemia. Type III**
- Prednisezone 100 mg orally on days 1 - 8

**Sickle cell-ß+ thalassaemia. Type I**
- Prednisezone 100 mg orally on days 1 - 8

**Sickle cell-ß+ thalassaemia. Type II**
- Prednisezone 100 mg orally on days 1 - 8

**Sickle cell-ß+ thalassaemia. Type III**
- Prednisezone 100 mg orally on days 1 - 8

**Sickle cell-ß+ thalassaemia. Type II**
- Prednisezone 100 mg orally on days 1 - 8

**Sickle cell-ß+ thalassaemia. Type I**
- Prednisezone 100 mg orally on days 1 - 8

**Sickle cell-ß+ thalassaemia. Type II**
- Prednisezone 100 mg orally on days 1 - 8

**Sickle cell-ß+ thalassaemia. Type III**
- Prednisezone 100 mg orally on days 1 - 8

**Sickle cell-ß+ thalassaemia. Type I**
- Prednisezone 100 mg orally on days 1 - 8

**Sickle cell-ß+ thalassaemia. Type II**
- Prednisezone 100 mg orally on days 1 - 8

**Sickle cell-ß+ thalassaemia. Type III**
- Prednisezone 100 mg orally on days 1 - 8

**Sickle cell-ß+ thalassaemia. Type I**
- Prednisezone 100 mg orally on days 1 - 8

**Sickle cell-ß+ thalassaemia. Type II**
- Prednisezone 100 mg orally on days 1 - 8

**Sickle cell-ß+ thalassaemia. Type III**
- Prednisezone 100 mg orally on days 1 - 8

**Sickle cell-ß+ thalassaemia. Type I**
- Prednisezone 100 mg orally on days 1 - 8

**Sickle cell-ß+ thalassaemia. Type II**
- Prednisezone 100 mg orally on days 1 - 8

**Sickle cell-ß+ thalassaemia. Type III**
- Prednisezone 100 mg orally on days 1 - 8
Supportive measures
- Antimalarials
  - Artemisinin-based combination therapy (see section on malaria)
- Adjunct treatment
- Investigations
- Notable adverse drug reactions, caution and contraindications

Membership of support group
- Regular health checks

Drug treatment
- Steady state (when patient is well with no complaints):
  - Proguanil
    - Adult: 200 mg orally daily
    - Child: under 1 year 25 mg daily; 1 - 4 years 50 mg; 5 - 8 years 100 mg; 9 - 14 years 150 mg orally daily
  - Folic acid 5 mg orally daily

Pain crises
- Mild pain
  - Paracetamol
    - Adult: 1 g, every 4 - 6 hours to a maximum of 4 g daily
    - Child: 1 - 5 years 120 - 250 mg; 6 - 12 years 250 - 500 mg; 12 - 18 years 500 mg every 4 - 6 hours (maximum 4 doses in 24 hours)
    - Aspirin (acetylsalicylic acid) 600 mg orally every 8 hours daily
    - Morphine 15 mg every 8 - 12 hours daily

- Emotional stress

Painful
- Tender, swollen bones
- Acute hepatopathy
- Acute chest syndrome
- Priapism

Painless
- Haematuria
- Cerebrovascular disease (accident) - in descending order of prevalence
  - Thrombotic stroke
  - Haemorrhage
  - Retinopathy (commonest in SC patients)

Anaemic crises
- Acute splenic (or hepatic) sequestration
- Hyper-haemolytic (e.g. precipitated by malaria)
- Megaloblastic (folic acid deficiency)
- Hypoplastic (due to infection or renal failure)
- Aplastic (e.g. due to envenomation of SC patients)

Differential diagnoses
- Connective tissue disorders e.g. rheumatoid arthritis
- Liver disease
- Other causes of failure to thrive

Complications
- Kidney:
  - Hyposthenuria (reduced ability to concentrate urine/conserve body fluids)
  - Haematuria
  - Albuminuria
  - Reduced kidney function
- Leg ulcers:
  - Occur around ankles
  - Heal slowly and tend to recur
- Bones and Joints
  - Osteomyelitis
  - Avascular necrosis
- These may cause:
  - Hip pain
  - Limping gait
  - Kyphoscoliosis when necrosis affects spinal vertebrae of bones
- Infections:
  - Salmonella osteomyelitis
  - Pneumococcal pneumonia
  - Pneumococcal meningitis (rare in adolescents and adults)
  - Tonsillitis and pharyngitis
- Brain and nerves:
  - Strokes, seizures (not common in adults)
  - Meningitis (not common in adults)
  - Cerebral haemorrhage
  - Mental neuropathy (rare)
- Cardiovascular/respiratory:
  - Heart failure

- Pulmonary hypertension
- Acute chest syndrome

Investigations
- Full Blood Count (haemoglobin, haematocrit, total leucocyte count and differential counts, platelet counts)
- Erythrocyte sedimentation rate
- Red cell indices (MCH, MCHC, MCV)
- Reticulocyte count
- Sickling tests: solubility test; metabisulphite test
- Haemoglobin electrophoresis
- - - Seizures
- - Using cellulose acetate paper at pH 8.4 (alkaline) or citrate agar gel at pH 5.6 (acidic)
  - Serum Electrolytes, Urea and Creatinine
  - Liver function tests (transaminases, bilirubin, serum albumin, alkaline phosphatase and prothrombin time)
- Urinalysis; microscopy, culture and sensitivity:
  - Sputum
  - Acid Fast Bacilli
  - Microscopy, culture and sensitivity
  - Stool:
    - Ova and parasites
    - Occult blood
  - Ultra sound scan:
    - Abdominal ultrasound scan
  - Kneenial Doppler ultrasonography
  - Chest radiograph

Treatment objectives
- Maintain (or restore) a steady state of health
- Prevent and treat complications
- Provide accurate diagnosis, relevant health education and genetic counselling to patients, relatives and heterozygotes
- Improve quality of life
- Provide a positive self-image in affected persons

Treatment strategies
- Counselling and health education
- Encouraging membership of support groups
- Providing infection prophylaxis (antimalarial; anti-pneumococcal, hepatitis B virus vaccines)
- Providing folate supplementation
- Avoiding pain-inducing conditions
- Providing prompt treatment of symptoms
- Advising on contraception
- Supervising pregnancy/Labour
- Providing regular health checks
- Limiting family size

Non-drug treatment
- Balanced diet
- Adequate fluid intake (at least 3 litres/24 hours)
- Avoidance of pain-inducing conditions
  - - Strenuous physical exertion or stress
  - - Mental frustration
  - - Sudden exposure to extremes of temperature
  - - Infections e.g. malaria

Standard Treatment Guidelines for Nigeria 2008
- Emotional stress

Adjunct treatment
- Blood transfusion (especially red cell transfusion)
- Anti-pneumococcal vaccine

Drug treatment
- Steady state (when patient is well with no complaints):
  - Proguanil
    - Adult: 200 mg orally daily
    - Child: under 1 year 25 mg daily; 1 - 4 years 50 mg; 5 - 8 years 100 mg; 9 - 14 years 150 mg orally daily
  - Folic acid 5 mg orally daily

Pain crises
- Mild pain
  - Paracetamol
    - Adult: 1 g, every 4 - 6 hours to a maximum of 4 g daily
    - Child: 1 - 5 years 120 - 250 mg; 6 - 12 years 250 - 500 mg; 12 - 18 years 500 mg every 4 - 6 hours (maximum 4 doses in 24 hours)
    - Aspirin (acetylsalicylic acid) 600 mg orally every 8 hours daily
    - Morphine 15 mg every 8 - 12 hours daily

- Not recommended for children under 16 years

Or:
- Ibuprofen 200 mg every 8 hours daily (or other non-steroidal anti-inflammatory drugs)
- Not recommended for children under 16 years

Moderate-to-severe painful crises

Parenteral therapy:
- Diclofenac sodium
  - Adult: 75 mg or 100 mg intramuscularly (as necessary)
  - Not recommended for children

Oral therapy:
- Paracetamol
  - Child: 1 - 5 years 20 mg/kg every 6 hours (maximum 90 mg/kg daily in divided doses) for 48 hours or longer if necessary and if adverse effects are ruled out

Then:
- 15 mg/kg every 6 hours (maintenance)
- 6 - 12 years: 20 mg/kg (maximum 1 g) 6 hourly (maximum 90 mg/kg daily in divided doses, not to exceed 4 g for 48 hours or longer if necessary and if adverse effects are ruled out

Then:
- 15 mg/kg every 6 hours (maximum 4 g daily)
  - 12 - 18 years: 500 mg - 1 g every 4 - 6 hours (maximum 4 doses in 24 hours)
  - Diclofenac potassium 50 mg every 12 hours daily

Or:
- Diclofenac sodium 100 mg once daily
Or:
- Morphine 15 mg every 8 - 12 hours daily

Antimalarials
- Artemisinin-based combination therapy (see section on malaria)

Supportive measures
- Counselling and health education
- Membership of support group
- Regular health checks

Notable adverse drug reactions, caution and contraindications
- Paracetamol should be used with caution in patients with hepatic impairment
- Opioid analgesics cause varying degrees of respiratory depression and hypotension
- They should be avoided when intracranial pressure is suspected to be raised

Prevention
- Advice on the risks involved in marriages between carriers, and between sicklers
- Anti-pneumococcal vaccine
Cardiac arrest: unstable angina

Arrhythmias

Clinical features

- Stable angina (chest discomfort on exertion and relieved by rest)
- Unstable angina (discomfort on exertion and at rest)

Myocardial infarction (chest pain or discomfort that lasts more than 30 minutes; may be associated with symptoms of cardiac failure, shock, arrhythmia)

**Differential diagnoses**

- Myalgia
- Pericarditis
- Aortic dissection
- Pleurisy

**Complications**

- Cardiac failure
- Myocardial infarction
- Arrhythmias
- Sudden death

**Investigations**

- Full Blood Count and differentials
- Urea, Electrolytes and Creatinine
- Fasting blood glucose
- Urinalysis; urine microscopy
- Electrocardiograph: resting, treadmill exercise
- Echocardiography (resting/exercise)
- Radio nuclide studies
- Cardiac enzymes (CK-MB)
- Coronary angiography

**Treatment objectives**

- Relieve discomfort
- Improve quality of life
- Prevent complications
- Relieve the obstruction
- Address the risk factors present

**Non-drug treatment**

- Dietary manipulation (low salt, low cholesterol diet)
- Exercise
- Stop smoking
- Reduce alcohol consumption

**Drug treatment**

- **ß blockers**
  - Atenolol 50 - 100 mg daily
  - Nitrates
  - Glyceryl trinitrate 0.3 - 1 mg sublingually, repeated as required
  - Or:
  - Isosorbide dinitrate 30 - 120 mg orally daily (up to 240 mg)
- **Calcium channel antagonists**
  - Verapamil 80 - 120 mg orally 8 hourly
- **Anti-platelets**
  - Aspirin (acetylsalicylic acid) 75 mg orally daily

**Treat as for acute myocardial infarction**

**Angioplasty (PTCA)**

**Sinus arrhythmias**

**Coronary artery bypass graft (CABG)**

**Anxiety**

**Treat/reduce risk factors**

**Other measures**

- **Aspirin**, thrombolytics: bleeding
- **Aortic dissection**: chest pain or discomfort that lasts more than 30 minutes; may be associated with symptoms of cardiac failure, shock, arrhythmia
- **Cardiac failure** (all anti-arrhythmics)
- **Blindness** (amiodarone)

**DEEP VEINS THROMBOSIS**

**Introduction**

Formation of blood clot(s) in the deep veins of the calf muscles or pelvis

- It has the potential of being dislodged to the lungs, causing pulmonary embolism
- Brought about by:
  - Hyper-coagulable states
  - Long periods of immobilization e.g. cardiac failure, following surgery, long-distance travel, etc
  - Malignancies
  - Genetic predisposition

**Clinical features**

- Could be asymptomatic
- Pain and swelling of the leg (calf muscles)

**Differential diagnoses**

- Deep vein thrombosis
- Cellulitis
- Infarctive crisis in sicklers
- Abscess (myositis)

**Complications**

- Pulmonary embolism

**Investigations**

- Full Blood Count and differentials
- Prothrombin time
- KCCT
- Doppler of the leg/pelvic vessels (veins)

**Differential diagnoses**

- Sinus arrhythmias
- Anxiety

**Complications**

- Cardiac failure
- Stroke
- Peripheral embolic phenomena

**Sudden death**

**Investigations**

- Electrocardiograph (resting, 24 hour Holter, 1 month Holter monitoring)
- Urea, Electrolytes and Creatinine
- Electrocardiography
- Electrophysiology

**Treatment objectives**

- Abolish the arrhythmias
- Treat complications
- Prevent further arrhythmias

**Non-drug treatment**

- Pacemaker insertion
- Ablation (electrophysiology)
- Cardioversion: acute arrhythmias

**Drug treatment**

- Depends on the type of arrhythmia
- Refer to a specialist for appropriate management

**Supportive measures**

- Patient education
- Efficient systems to facilitate patient recovery

**Notable adverse drug reactions**

- All anti-arrhythmics are pro-arrhythmics themselves
- Cardiac failure (all anti-arrhythmics)
- Blindness (amiodarone)

**Prevention**

- Prevention of conditions such as hypertension, rheumatic heart disease, diabetes mellitus, ischaemic heart disease, congenital heart diseases etc

**CONGENITAL HEART DISEASE**

**Introduction**

A heart defect that occurs during the formation of the heart in utero

Could be fatal (i.e. causes intrauterine death, or death at anytime afterwards)

- An important cause of perinatal morbidity/mortality

**Classified as**

- Cyanotic
- Ancytonic

**Clinical features**

- Will depend on the type of the defect:
  - Mild defects go unnoticed
  - Severe defects lead to failure to thrive
  - Heart murmurs

**Cardioversion: acute arrhythmias**

Relieve symptoms

**Pre-conception nutrition education**

**Counselling**

**GENETIC COUNSELLING**

**Investigations**

- Urea, Electrolytes and Creatinine
- Fasting blood glucose
- Urinalysis; urine microscopy
- Electrocardiograph (resting/exercise)
- Radio nuclide studies
- Cardiac enzymes (CK-MB)
- Coronary angiography

**Complications**

- Hyper-coagulable states
- Long periods of immobilization e.g. cardiac failure, following surgery, long-distance travel, etc
- Malignancies
- Genetic predisposition

**Clinical features**

- Could be asymptomatic
- Pain and swelling of the leg (calf muscles)

**Differential diagnoses**

- Cellulitis
- Infarctive crisis in sicklers
- Abscess (myositis)

**Complications**

- Pulmonary embolism

**Investigations**

- Full Blood Count and differentials
- Prothrombin time
- KCCT
- Doppler of the leg/pelvic vessels (veins)
**Chapter 3: Cardiovascular System**

- **Echocardiography**
- **Electrocardiography**
- **Venography (pelicv or calf veins)**

**Treatment objectives**
- Lyse the clot
- Prevent clot from being dislodged
- Relieve inflammation

**Non-drug treatment**
- Avoid stasis
- Achieve APTT of 1.5 to 2.5 of control: Heparin 5000 - 10,000 units by intravenous injection followed by subcutaneous injection of 15,000 units every 12 hours or intravenous infusion at 15 - 25 units/kg/hour, with close laboratory monitoring
- Warfarin 1 - 5 mg orally daily for 6 - 12 weeks

**Notable adverse drug reactions**
- Bleeding from heparin, warfarin
- Osteoporosis (heparin)

**Prevention**
- Low molecular weight heparin 5000 units subcutaneously every 12 hours
- Early mobilization

**HEART FAILURE**

**Introduction**
- A clinical state (syndrome) in which the heart is unable to generate enough cardiac output to meet up with the metabolic demands of the body
- The commonest cause in Nigeria is hypertension
- Other causes include dilated cardiomyopathy and rheumatic heart disease
- Cardiac failure can be classified as:
  - Left or right-sided
  - Congestive
  - Acute
  - Chronic
- Chronic cardiac failure is the commonest syndrome encountered in our setting

**Clinical features**
- Difficulty with breathing on exertion
- Paroxysmal nocturnal dyspnoea
- Orthopnoea
- Cough productive of frothy sputum
- Leg swelling
- Abdominal swelling
- The prominence of particular symptoms will depend on which side is affected

**Signs include:**
- Oedema
- Tachycardia (about 100 beats per minute)
- Raised jugular venous pressure
- Displaced apex
- S3 or S4 or both (With or without murmurs)

**Drug treatment**
- **Diuretics**
  - Furosemide 40 - 80 mg intravenously or orally
  - Spironolactone 25 - 100 mg once, every 8 - 12 hours daily
- **Vasodilators**
  - Angiotensin converting enzyme inhibitors (ACEIs)
  - Dopamine 2 - 5 microgram/kg/minute by intravenous infusion
- **Anticoagulants**

**Complications**
- Thrombo-embolic phenomena: stroke, pulmonary embolism

**Investigations**
- Full Blood Count with differentials
- Urea, Electrolytes and Creatinine
- Fasting blood glucose
- Urine micro-analysis
- Chest radiograph
- Electrocardiography
- Echocardiography

**Treatment objectives**
- Relieve symptoms
- Enhance quality of life
- Prevent complications
- Prolong life

**Non-drug treatment**
- Bed rest
- Low salt diet
- Exercise (within limits of tolerance)

**Hyperlipidaemia**

**Introduction**
- A clinical syndrome in which there are high lipid levels: cholesterol, or its fractions, or triglyceridaemia
- Can be primary (hereditary) or secondary - as a result of other diseases
- Incidence in Nigeria is thought to be low but recent studies show increasing incidence in association with diabetes mellitus and hypertension

**Clinical features**
- Patients present with complications of hypertension, ischaemic heart disease or the cause of secondary hyperlipidaemia

**Signs include:**
- Xanthomata, xanthelasmata, and corneal arcas

**Drug treatment**
- **Diuretics**
  - Furosemide 40 - 80 mg intravenously or orally
  - Spironolactone 25 - 100 mg once, every 8 - 12 hours daily
  - Potassium supplements
  - Angiotensin converting enzyme inhibitors (ACEIs)
  - Dopamine 2 - 5 microgram/kg/minute by intravenous infusion

**Vasodilators**
- **Anticoagulants**

**Hypertension**

**Introduction**
- A persistent elevation of the blood pressure above normal values (taken three times on at least two different occasions with intervals of at least 24 hours)

**Blood pressure ≥ 140/90 mmHg irrespective of age is regarded as hypertension**

**The commonest non-communicable disease in Nigeria**

**The commonest cause of cardiac failure and stroke**

**Hypertension may be:**
- Diastolic and systolic
- Diastolic alone
- Isolated systolic

**Clinical features**
- Largely is asymptomatic until complicated ("silent killer")
- Non-specific symptoms: headache, dizziness, palpitations etc
- Other symptoms and signs depending on the target organs affected e.g. cardiac or renal failure, stroke etc

**Standard Treatment Guidelines for Nigeria 2008**

**Treatment objectives**
- Warfarin: monitor INR 2 - 2.5
- Important in atrial fibrillation

**Supportive measures**
- Pacemakers for arrhythmias
- Ventricular assist devices

**Preventable adverse drug reactions**
- Digitalis: arrhythmias
- Potassium-sparing drugs: hyperkalaemia

**ACEIs:**
- Hypotension
- Hyperkalaemia

**Do not combine potassium supplements with potassium-sparing drugs**

**Prevent complications**
- Prolong life

**Differential diagnoses**
- Ischaemic heart disease
- Peripheral vascular disease
- Stroke

**Investigations**
- Urea, Electrolytes and Creatinine
- Fasting blood glucose
- Lipid profile
- Urine proteins

**Serum proteins (total and differential)**

**Treatment objectives**
- Lower lipid levels
- Prevent complications
- Treat complications

**Non-drug treatment**
- Stop smoking
- Reduce weight
- Exercise moderately and regularly

**Precautions**
- Dietary manipulation
- Early identification of individuals at risk

**HEART FAILURE**

**Introduction**
- A clinical state (syndrome) in which the heart is unable to generate enough cardiac output to meet up with the metabolic demands of the body

**The commonest cause in Nigeria is hypertension**

**Other causes include dilated cardiomyopathy and rheumatic heart disease**

**Cardiac failure can be classified as:**
- Left or right-sided
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**Signs include:**
- Oedema
- Tachycardia (about 100 beats per minute)
- Raised jugular venous pressure
- Displaced apex
- S3 or S4 or both (With or without murmurs)
**Chapter 3: Cardiovascular System**

**Differential diagnoses**
- White coat hypertension
- Anxiety/fright/stress

**Complications**
- Heart failure, ischaemic heart disease
- Brain:
  - Stroke (ischaemic, hemorrhagic)
  - Hypertensive retinopathy
- Kidney:
  - Renal failure
- Large arteries:
  - Aortic aneurysm

**Investigations**
- Full Blood Count
- Urinalysis; urine microscopy
- Urea, Electrolytes and Creatinine
- Uric acid
- Fasting blood glucose
- Lipid profile
- Chest radiograph
- Electrocardiography
- Echocardiography (not in all cases)
- Abdominal ultrasound
- Renal angiography (not in all cases)

**Treatment objectives**
- Educate patient about disease and need for treatment adherence
- Reduce blood pressure to acceptable levels
- Prevent complications (primary, secondary, tertiary)
- Rehabilitate

**Non-drug treatment** (lifestyle modification)
- Low salt diet
- Achieve/maintain ideal body weight (BMI 18.5 - 24.9 kg/m²)
- Stop smoking
- Regular moderate exercise
- Reduce polysaturated fatty acid intake

**Drug treatment**
- Diuretics:
  - Thiazides
  - Hydrochlorothiazide 2.5 - 10 mg orally daily
  - Or:
    - Hydrochlorothiazide 12.5 - 50 mg orally daily
  - Or:
    - Hydrochlorothiazide/amiloride 25/2.5 mg daily
  - Loop diuretics
  - Furosemide 40 - 80 mg orally daily
  - \(\beta\)-blockers:
    - Propranolol 40 - 80 mg orally every 8 - 12 hours
    - Atenolol 25 - 100 mg orally daily
  - Calcium channel antagonists:
    - Nifedipine retard 20 - 40 mg orally once or twice daily
- Or:
  - Amlodipine 2.5 - 10 mg orally once daily
  - Angiotensin converting enzyme inhibitors:
    - Captopril 6.25 - 50 mg orally once or every 8 - 12 hours
  - Or:
    - Lisinopril 2.5 - 20 mg orally once daily
  - Angiotensin receptor blockers:
    - Losartan 50 - 100 mg orally daily
  - Other vasodilators:
    - Hydralazine 25 - 100 mg orally once or every 12 hours
- Or:
  - Prazosin 0.5 - 1 mg orally daily
  - Centrally acting drugs:
    - Alpha methylldopa 250 - 500 mg orally twice, three or four times daily
    - Fixed combinations:
      - Reserpine plus dihydroergocristine plus clopamide
      - 0.25/0.5/5 mg one-two tablets orally daily
  - Or:
    - Lisinopril plus hydrochlorothiazide 20/12.5 mg daily

**Hypertensive emergencies**
- Treatment should be done by the experts
- Involves the administration of antihypertensives by the parenteral route (usually intravenous hydration or sodium nitroprusside)

**Supportive measures**
- Patient/care giver education

**Notable adverse drug reactions**
- caution and prevention

**Dental procedures**
- Under local or no anaesthesia, for those who have NO pasado carditis, and have NOT received more than a single dose of a penicillin in the last one month:
  - Amoxicillin
  - Adult: 3 g orally 1 hour before procedure
  - Child under 5 years: 750 mg orally 1 hour before procedure; 5 - 10 years: 1.5 g

For penicillin-allergic patients or patients who have received more than a single dose of a penicillin in the previous one month:
- Azithromycin
  - Adult: 500 mg orally one hour before procedure
  - Child under 5 years: 200 mg orally, 5 - 10 years: 300 mg

**INFECTIVE ENDOCARDITIS**
- The sub-acute form usually occurs on damaged valves (e.g. rheumatic heart disease, congenital heart disease), shunts, and atherosclerotic lesions
- Causative organisms include staphylococci, streptococci enterococci, haemophilus, actinobacillus, clostridium bacillus, eikenella, and kingella species (‘HACEK’ organisms)

**Clinical features**
- Acute:
  - High fever with rigors
  - Delirium
  - Shock
  - Development of new murmurs
  - Severe cardiac failure
  - Abscesses may form in many parts of the body (e.g. brain)
- Subacute:
  - Low-grade fever
  - Signs of carditis
  - Arthralgia
  - Finger clubbing
  - Splenomegaly
  - Osler’s nodules
  - Janeway lesions
  - Roth spots

**Differential diagnoses**
- Malignant neoplasia
- Rheumatic heart disease

**Complications**
- Heart failure, ischaemic heart disease
- Destruction of heart valves
- Systemic embolism (could be infective)

**Investigations**
- Full Blood Count and differentials; ESR
- Urinalysis; urine microscopy
- Blood cultures X 3 (the yield is higher at the time of pyrexia)
- Echocardiography

**Treatment objectives**
- Stop the infection
- Treat cardiac failure
- Prevent coagulation disorders

**Non-drug treatment**
- Bed rest
- Low salt diet

**Drug treatment**
- Initiate therapy with:
  - Benzylpenicillin 7.2 g daily by slow intravenous injection or intravenous infusion in 6 divided doses for 4 - 6 weeks
  - May be increased up to 14.4 g daily if necessary (e.g. in endocarditis)
- Plus:
  - Gentamicin 60 - 80 mg intravenously or intramuscularly every 8 hours for 2 weeks

**Following bacteriological confirmation institute appropriate antimicrobial therapy**
- Staphylococci:
  - Flucloxacillin
  - 250 mg - 2 g intravenously every 6 hours for 4 - 6 weeks
- Candida:
  - Systemic antifungals

**Notable adverse drug reactions**
- Penicillin: rashes, anaphylaxis
- Gentamicin: nephropathy

**Prevention**
- Prophylactic antibiotics for patients at risk who are undergoing:
  - Dental procedures
  - Under local or no anaesthesia, for those who have NOT had endocarditis, and have NOT received more than a single dose of a penicillin in the last one month:
    - Amoxicillin
  - Adult: 3 g orally 1 hour before procedure
  - Child under 5 years: 750 mg orally 1 hour before procedure; 5 - 10 years: 1.5 g
  - For penicillin-allergic patients or patients who have received more than a single dose of a penicillin in the previous one month:
    - Azithromycin
    - Adult: 500 mg orally one hour before procedure
    - Child under 5 years: 200 mg orally, 5 - 10 years: 300 mg
  - Patients who have had endocarditis:
    - - Amoxicillin plus gentamicin intravenously as for procedures under general anaesthesia (see below)
    - Dental procedures under general anaesthesia, and no special risk:
      - Amoxicillin
      - Adult: 1 g intravenously at induction of anaesthesia; 500 mg orally 6 hours later
      - Child under 5 years: a quarter of adult dose; 5 - 10 years: half adult dose
  - Special risk, e.g. previous infective endocarditis, or patients with prosthetic valves:
    - Amoxicillin plus gentamicin intravenously
    - Adult: 1 g amoxicillin plus 120 mg gentamicin at induction
      - Then oral amoxicillin 500 mg 6 hours after procedure
      - Child under 5 years: a quarter of adult dose of amoxicillin plus 2 mg/kg gentamicin intravenously at induction
        - 5 - 10 years: half adult dose for amoxicillin; 2 mg/kg gentamicin
Chapter 3: Cardiovascular System

Standard Treatment Guidelines for Nigeria 2008

**Treat the effect on the heart**
**Treat complications**
**Non-drug treatment**

**Drug treatment**
- Treat underlying cause(s)
- Anti arrhythmics (depends on the type of arrhythmias)
- Anticoagulant: warfarin
- Anti-cardiac failure: digoxin, diuretics, potassium supplements
- Steroids: prednisolone (not in all cases)
- Multivitamins
- Anti-oxidants: ascorbic acid (vitamin C), vitamin E

**Notable adverse drug reactions**
- Antiarrhythmics may be pro-arrhythmic

**Non-drug treatment**
- As for genitourinary tract manipulation

**MYOCARDIAL INFARCTION**

**Introduction**
- Occurs when an area of heart muscle is necrosed or permanently damaged because of an inadequate supply of oxygen (heart attack)

**Clinical features**
- Precordial pain: discomfort, heaviness, tightening lasting 30 minutes or more
- Shortness of breath
- Palpitations
- Cough productive of frothy sputum
- Signs of right or left-sided cardiac failure and shock

**Differential diagnoses**
- Pulmonary embolism
- Aortic dissection
- Pericarditis

**Complications**
- Cardiac failure
- Ventricular aneurysm
- Arrhythmias: heart block, ventricular tachycardia, ventricular fibrillation, atrial fibrillation
- Sudden death

**Investigations**
- Full Blood Count; ESR
- Urea, Electrolytes and Creatinine
- Urine acid
- Fasting blood glucose
- Lipid profile
- Enzyme assays: AST, CK-MB, and LDH
- Electrocardiograph monitoring throughout admission
- Coronary angiography (in case of secondary angioplasty)

**Treatment objectives**
- Relieve pain (discomfort)
- Relieve obstruction
- Treat complications
- Prevent future episodes

**Non-drug treatment**
- Bed rest

**Dietary control** (low cholesterol)
- Exercise (later)
- Weight reduction (later)
- Stop smoking

**Drug treatment**
- Aspirin (acetylsalicylic acid) 150 - 300 mg orally stat, then 75 - 150mg daily
- Morphine 10 mg by slow intravenous injection over 5 minutes (i.e. 2 mg/minute)
- Unfractionated heparin
- Adult: 5,000 - 10,000 units (75 units/kg) by intravenous injection as loading dose followed by continuous infusion of 15 - 25 units/kg/hour
- 15,000 units 12 hours by subcutaneous injection
- Small adult or child: lower loading dose, then 15 - 25/kg/hour by intravenous infusion, or 250 units/kg every 12 hours by subcutaneous injection
- Lowmolecular weightheparin
- Enoxaparin: 30 mg intravenous bolus (optional) then 1mg/kg subcutaneously every 12 hours for 7 - 8 days
- Thrombolytics
- Streptokinase
- Adult: 1,500,000 units by intravenous infusion over 60 minutes, then 250 units over 30 minutes according to condition (with monitoring)
- Child: 1 month - 12 years, initially 2,500 - 4,000 units/kg over 30 minutes followed by continuous infusion of 500-1,000 units/kg/hour for up to 3 days until reperfusion occurs
- 12 - 18 years: initially 250,000 units intravenously over 30 minutes, followed by intravenous infusion of 100,000 units/hour for up to 3 days until reperfusion occurs
- Recombinant plasminogen activator (use by specialist physician)
- Alteplase 15 mg intravenously over 1 - 2 minutes, followed by intravenous infusion of 50 mg over 30 minutes then 35 mg over 60 minutes
- B blockers
- Atenolol 50 - 100 mg orally daily
- Propranolol 180 - 240 mg orally in 2 - 4 divided doses daily
- Lisinopril 2.5 - 10 mg daily
- Maintenance anti-anginal therapy

**Non-drug therapy**
- Coronary artery bypass graft (CABG)
- Secondary or rescue PTCA

**Supportive measures**
- Treat arrhythmias
- Oxygen: 100% at 5L/minute

**MYOCARDITIS**

**Introduction**
- Inflammatory process affecting the myocardium
- A common disorder; usually occurs in association with endocarditis and pericarditis

**Possible causes:**
- Infections: viral, bacterial, protozoal
- Toxins e.g. scorpion sting
- Poisons e.g. alcohol
- Drugs e.g. chloroquine
- Allergy e.g. to penicillin
- Deficiencies e.g. thiamine
- Physical agents e.g. radiation

**Clinical features**
- Largely asymptomatic
- A few may present with palpitations; symptoms of cardiac failure

**Physical examination:**
- Arrhythmias
- Tachycardia
- Raised JVP
- Cardiomegaly
- S3 or S4 (with or without murmurs of regurgitation in the mitral/tricuspid areas)

**Differential diagnoses**
- Other forms of cardiac failure, e.g. peripartum cardiac failure

**Complications**
- Cardiac failure
- Arrhythmias
- Thrombus formation

**Investigations**
- Full Blood Count and differentials
- Urea, Electrolytes and Creatinine
- Electrocardiography
- Echocardiography
- Myocardial biopsy

**Treatment objectives**
- Eliminate/withdraw the offending agent(s)

**Investigations**
- Full Blood Count and differentials
- Urea, Electrolytes and Creatinine
- Electrocardiography
- Echocardiography
- Myocardial biopsy

**Treatment objectives**
- Eliminate/withdraw the offending agent(s)

**Notable adverse drug reactions, caution**
- Heparin or streptokinase: bleeding (risk of bleeding in recent stroke, diabetic retinopathy, brain tumours, peptic ulcer disease or surgery)
- Laboratory monitoring is essential: preferably daily, and dose adjusted accordingly
- Aspirin: dyspepsia
- β-blockers: bradycardia
- Should be avoided in patients presenting with this symptom

**Prevention**
- Treat hypertension, diabetes mellitus, and hyperlipidaemia
- Stop smoking
- Nutrition education

**PAEDIATRIC CARDIAC DISORDERS (Refer for Specialist Care)**

**PERICARDITIS**

**Introduction**
- An inflammation of the pericardium which may arise from viral, bacterial, fungal or protozoal infections
- Other causes: metabolic, malignancy, connective tissue disease, radiation, trauma etc
- May be acute or chronic

**Clinical features**
- Acute pericarditis:
  - Chest pain
  - Retrosternal
  - Sharp
  - Radiating to the left shoulder
  - Made worse by breathing or coughing
  - Relieved by the upright position
- Low grade fever
- Pericardial friction rub
- Chronic pericarditis:
  - Insidious onset
  - There may be:
    - Dyspnoea on exertion
    - Leg and abdominal swelling

**Differential diagnoses**
- Endomyocardial fibrosis
- Sarcoidosis
- Amyloidosis

**Complications**
- Pericardial tamponade
Constrictive pericarditis

**Investigations**
- Electrocardiography
- Full Blood Count and differentials
- Chest radiograph
- Echocardiography

**Treatment objectives**
- Relieve distress from pain and tamponade
- Relieve constriction
- Treat the effect on the heart
- Treat complications
- Eradicate the organism (if cause is infection)

**Non-drug treatment**
- Bed rest

**Drug treatment**
- NSAIDs
  - Indomethacin 50 mg orally every 8 hours or
  - Ibuprofen 400 - 800 mg orally every 12 hours
- Steroids
- Prednisolone 30 mg orally every 8 hours and tapered
- Anti-tubercular drugs or other antimicrobial agents (if mycobacterium or other microbes are causative)

**Supportive measures**
- Pericardiocentesis
- Pericardiectomy

**Notable adverse drug reactions**
- NSAIDs/steroids: dyspnea and upper GI bleeding
- Prevention
  - Avoid radiation
  - Prevent infection

**PULMONARY EMBOLISM**
(Also see in Respiratory system)

**Introduction**
- Blockage of the pulmonary artery or one of its branches by a blood clot, fat, air, or clumped tumour cells
- The most common form is thrombo-embolism; occurs when
  - A blood clot (generally a venous thrombus) becomes dislodged from its site of formation and embolizes to the arterial blood supply of one of the lungs
  - The calf veins (deep vein thrombosis) and right ventricle are sources of embolism
- Some predisposing factors:
  - Congestive cardiac failure
  - Trauma
  - Surgery
  - Prolonged immobilization
  - Malignancies
  - Stroke

**Clinical features**
- Depend on how massive the embolism is:
  - No symptoms
  - Moderate-to-severe cases:
    - Difficulty in breathing

**Differential diagnoses**
- Lobar pneumonia
- Myalgia
- Pleuritis (pleurisy)

**Complications**
- Right-sided cardiac failure
- Haemorrhagic pleural effusion

**Investigations**
- Full Blood Count and differentials
- Electrocardiograph
- Sinus tachycardia
- Notable atrial fibrillation/flutter
- S wave in lead 1, Q wave in lead 3 and an inverted T wave in lead 3
- QRS axis >90º, quite often
- Chest radiograph
- Blood gases (arterial)
- Ventilation/perfusion lung scanning
- Echocardiography
- Pulmonary artery angiogram

**Treatment objectives**
- Relieve discomfort
- Relieve the obstruction(s)
- Prevent complications
- Prevent further episodes

**Non-drug treatment**
- Bed rest
- Mobilization

**Drug treatment**
- Heparin
  - 5000 - 10,000 units intravenously stat, followed by 1000 - 2000 units per hour (APTT or INR 1.5 - 2.5 greater than normal)
- Enoxaparin
  - 1.5 mg/kg (150 units/kg) subcutaneously every 24 hours, usually for at least 5 days (and until adequate oral anticoagulation is established)
- Warfarin 1 - 5 mg (INR 1.5 - 2) for 6 - 12 weeks (as maintenance after initial parenteral anticoagulation)

**PULMONARY OEDEMA**

**Introduction**
- Occurs when there is congestion of the lungs with fluid, usually in a scenario of left-sided cardiac failure
- Results in stiffness of the lungs and flooding of the alveoli, with difficulty in breathing
- May also follow inflammatory processes
- May be acute or chronic

**Clinical features**
- Difficulty in breathing, with a sensation of drowning
- Cough productive of frothy (sometimes pink) sputum
- Central cyanosis
- Sweating, agitation etc
- Other symptoms of left-sided cardiac failure

**Examination**
- Wide-spread crepitations
- Rhonchi (in severe cases)
- Other signs of left-sided cardiac failure

**Differential diagnoses**
- Pulmonary embolism
- Pneumonia

**Complications**
- Hypoxaemia
- Coma

**Investigations**
- Blood gases
- Urea, Electrolytes and Creatinine
- Echocardiography
- Chest radiograph
- Electrocardiography

**Treatment objectives**
- Relieve oedema
- Relieve discomfort
- Treat underlying cause

**Non-drug treatment**
- Bed rest
- Sit on bed with legs hanging down

**Drug treatment**
- Oxygen 3 - 5L/min
- Morphine 10 mg stat
- Loop diuretics
- Furosemide 40 - 120 mg intravenously stat; maintenance with 40 - 500 mg daily in single or divided doses
- Aminophylline 250 mg intravenously slowly over 1 - 2 minutes, followed by intravenous infusion of 90 mg over 2 hours
- To be used by a specialist physician

**Notable adverse drug reactions**
- Heparin, warfarin or streptokinase: bleeding
- NSAIDs/steroids: dyspnea and upper GI bleeding

**Supportive measures**
- Nursing care (e.g. nurse in cardiac position)

**RHEUMATIC FEVER**

**Introduction**
- A result of abnormal reaction of antibodies developed against antigens of group Aß-haemolytic streptococci
- Infection is usually of the throat; occasionally the skin in a sensitized individual
- Antibodies damage the heart (endocardium, myocardium and pericardium)
- Commonest streptococcal strains in Africa are C and G

**Clinical features**
- Fever
- Arthralgia
- Abnormal movements of the hands (upper hands)
- Diagnosis: Duckett-Jones' diagnostic criteria

**Major:**
- Carditis
- Sydenham's chorea
- Erythema marginatum
- Subacute nodules
- Arthritis (migratory polyarthritis)

**Minor:**
- Fever
- Leucocytosis
- Arthralgia
- Raised ESR
- Raised ASO titre (> 200 IU)
- Previous history of rheumatic fever

**Diagnosis**
- 2 major criteria
- Or:
  - 1 major plus 2 (or more) minor criteria
Investigations
- Electrocardiography (resting/exercise)
- Lipid profile
- Echocardiography
- Chest radiography
- Coronary angiography

Treatment objectives
- Relieve symptoms
- Prevent recurrence of rheumatic attack
- Repair and replace affected valves

Penicillin: anaphylactic reaction
Salicylates; steroids: peptic ulceration
Cushingoid effects are increasingly likely with doses of prednisolone above 7.5 mg daily

Penicillin may cause hypersensitivity reaction / anaphylaxis
Penicillin may cause anaphylactic reaction
Salicylates; steroids: peptic ulceration
Cushingoid effects are increasingly likely with doses of prednisolone above 7.5 mg daily

Prevention
Good sanitation.
School surveys - identify carriers of streptococcus and treat

RHEUMATIC HEART DISEASE
Introduction
A complication of rheumatic fever
A common cause of cardiac failure in Nigeria

In Africa manifests later compared to Caucasians
The mitral valve is most affected, followed by the aortic, then the tricuspid
The lesions can occur in various combinations of stenosis and regurgitation

Clinical features
Shortness of breath on exertion
Paroxysmal nocturnal dyspnoea
Orthopnoea
Leg and abdominal swelling
Cough with production of frothy sputum
Pedal and sacral oedema
Small volume pulse which may be irregular

With or without tachycardia
With or without hypotension
Raised JVP
Displaced apex
Left ventricular hypertrophy
Right ventricular hypertrophy
Thrills
Palpable P2
Soft S1; loud P2
S3 or S4
Systolic/diastolic murmurs

Differential diagnoses
Constrictive pericarditis
Endomyocardial fibrosis
Dilated cardiomyopathy

Complications
Arrhythmias e.g. atrial fibrillation, heart block
Cardiac failure
Emolic phenomena
Endocarditis

Other measures:
- Valve replacement
- Valve repair
- Treat endocarditis

CHAPTER 4: CENTRAL NERVOUS SYSTEM

NON-PSYCHIATRIC DISORDERS

DIZZINESS
Introduction
Simply means ‘light-headedness’
Usually due to impaired supply of blood, oxygen and glucose to the brain
May suggest some form of unsteadiness, or could precede a fainting spell

Causes:
- Side effects of medications, notably anti-hypertensives and sedatives
- Anaemia
- Arrhythmias
- Fever
- Hypoglycaemia
- Brain stem lesions
- Alcohol overdose
- Excessive blood loss
- Prolonged standing
- Autonomic neuropathy (especially in diabetic patients)
- May be accompanied by vertigo (giddiness) in some individuals
- May culminate in loss of consciousness

Clinical features
- Light-headedness
- Feeling faint especially on attempting to stand or after squatting
- Weakness

Differential diagnoses
- Benign positional vertigo
- Labyrinthine disorders
- Hysteria
- Premonitory symptoms of epilepsy
- Migraine aura

Warning symptom of posterior circulation stroke
Falls with injury
Stroke

If due to intracranial tumour: raised intracranial pressure with coning
If due to other intracranial pathology: cranial nerve palsies

Investigations
- Full Blood Count and differentials
- Electrocardiography
- Echocardiography
- Random blood glucose
- X-ray sinuses
Complications

Investigations

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Septicaemia with meningism

Cranial nerve palsies

Secondary headaches

Medical or surgical management of identified causes

Antibiotics for infections like meningitis, sinusitis

Steroids for vasculitis

Aspirin and other NSAIDs: use with caution in patients with history of dyspepsia, and asthma

Tricyclic antidepressants: use with caution in patients with cardiac symptoms

Tricyclic antidepressants: anticholinergic effects eg urinary retention in the elderly

Prevention

Reduce stress levels

Prophylactic medications if attacks last more than 15 days a month, or are severely incapacitating (in the presence of other causes)

Early detection and correction of refractive errors, sinusitis, oto-rhino-laryngologic and dental problems.

Hormone secretion (SIADH)

Heat stroke

Syndrome of Inappropriate Anti-Diuretic Hormone secretion (SIADH)

Investigations

Lumbar puncture for CSF analysis

- To demonstrate presence of inflammatory cells (after exclusion of raised intracranial pressure by fundoscopy or CT scan)

Full Blood Count and differentials

Blood culture

Electrolytes, Urea and Creatinine

HIV screening

Treat complications (if any)

Migraine headache

Secondary headaches

Differential diagnoses

Meningitis

Hysteria

Reflexive error

Cervical spondylitis

Brain tumour

Haemorrhagic stroke

Complications

Depend on the cause and type

Some are benign with no sequelae

Coning (depending on cause)

Blindness (following temporal arteritis, unrelieved raised intracranial pressure)

Infections

Following generalized convulsions

Metabolic derangements

Alcohol hangover

Drugs

Irritation of sensory cranial nerves

Inflammation or diseases of structures/organisms in the head region: eyes, nose, sinuses, ears, cervical vertebrae

Atypical headache

Sleep disorders (hypoaxia)

Brain stem malformations

HIV infection

Clinical features

Depend on the underlying type/cause(s):

Tension type

Heaviness in the head

Crawling sensation

“Peppery sensation”

Tight-hand sensation

Poor sleep

Disturbed concentration

Cluster type

Recurrent, frequent, brief attacks of disturbing pain

in the head

Pain around the eyes and forehead

Redness of the eyes

Nasal stuffiness

Drooping of the eyelids

- Meningitis

Hysteria

Reflexive error

Cervical spondylitis

Brain tumour

Haemorrhagic stroke

- See below

Secondary headaches: presence of additional symptoms

Fever

Vomiting

Neck stiffness

Alteration in level of consciousness

Conulsions

Cranial nerve deficits

Limb weakness (hemiparesis, quadriparesis)

Papilloedema as evidence of raised intracranial pressure

Evidence of disease in other organs

Evidence of drug or alcohol abuse

Differential diagnoses

Meningitis

Hysteria

Reflexive error

Cervical spondylitis

Brain tumour

Haemorrhagic stroke

Complications

Depend on the cause and type

Some are benign with no sequelae

Coning (depending on cause)

Blindness (following temporal arteritis, unrelieved raised intracranial pressure)

Clinical features

Fever

Headache

Vomiting

Photophobia

Alteration in level of consciousness

Treatment objectives

Eliminate the precipitating factor or disease

Prevent recurrence

Non-drug treatment

Psychotherapy

Physiotherapy/biofeedback

Dexamethasone

Primary headaches

Simple analgesics and non-steroidal anti-inflammatory agents

Tricyclic antidepressants

- Amitriptyline 10 - 25 mg daily at night

- Lorazepam 1 - 2.5 mg at night. Use lower doses for the elderly patient

- 2 - 4 g daily by intravenous injection or by intravenous infusion over 2 - 4 minutes

- Penicillin V 2-4g by slow intravenous injection

Or:

Chloramphenicol 100 mg/kg intravenously every 4 hours

- May be useful for H. influenzae infection

Tuberculosis:

Standard anti-tuberculous drugs (including pyrazinamide and isoniazid for their good penetration of the blood-brain barrier)

Anti-ptyretics:

Aspirin (acetylsalicylic acid)
**Non-drug treatment**
- Manage in a quiet (and dark) room
- Psychotherapy
- Physiotherapy/biofeedback

**Notable adverse drug reactions, caution and contraindications**
- Aspirin and other NSAIDs: use with caution in patients with history of dyspepsia and in asthmatics
- Ergotamine: use should not exceed 4 - 6 mg per attack
- Caution in patients with vascular and renal disorders
- Not recommended for children
- Opiates: risk of addiction
- ß-blockers: slow down cardiovascular function; reduce sensitivity to hypoglycaemia in diabetics

**Ergotamine**
- Use should not exceed 4-6 mg per attack
- Caution in patients with vascular and renal disorders
- Not recommended for children

**Eliminate pain**
- Opiates: risk of addiction
- ß-blockers: slow down cardiovascular function; reduce sensitivity to hypoglycaemia in diabetics

**Introduction**
- Avoid precipitants
- These must be identified for effective prevention

**Prevention**
- Reduce stress levels as much as possible
- Immunize against communicable diseases
- Chemoprophylaxis (Rifampicin or ciprofloxacin)
- - Must be administered slowly intravenously to avoid respiratory depression
- - May cause aplastic anaemia
- - May cause chills and fever
- - Extravasation causes inflammation and thrombophlebitis
- - Contraindicated in congestive cardiac failure and pulmonary oedema

**Migraine**

**Introduction**
- Headache resulting from changes in the calibre of certain blood vessels in the brain with resulting physical, autonomic and emotional disturbance
- Can be very incapacitating
- Affects more females than males, usually between the ages of 15 and 50 years

**Clinical features**
- Vascular headaches
  - Common migraine (or migraine without aura)
  - Tending pain usually affecting one side of the head around the temples, associated nausea and vomiting
  - Dislike of light and noise
  - Attacks of pain preceded by seeing flashes of light
  - Disturbances in the field of vision (scotomas)
- Visual hallucinations
- Childhood periodic syndromes:
  - Abdominal pain and vomiting
  - Alternating hemiplegia
  - Benign positional vertigo
- Basilar artery migraine - predominantly brain stem symptoms
  - Dysarthria
  - Vertigo
  - Tinnitus
  - Decreased hearing
  - Diplopia
- Ataxia
  - May coexist with tension-type headache
  - May present without headache (migraine equivalent) usually seen in psychiatry
  - May present with complications: stroke-like manifestations

**Chemoprophylaxis**
- Treat contacts during meningococcal epidemics with either ciprofloxacin or rifampicin
- - Rifampicin
- - Mannitol 20% solution

**Adult:**
- 10 - 20 mg at a rate of 0.5 ml per 30 seconds, repeated if necessary after 30 - 60 minutes; may be followed by intravenous infusion to a maximum of 3 mg/kg over 24 hours
- Child:
  - neonate 0.5 - 1 mg/kg every 12 - 24 hours (every 24 hours in neonates born before 31 weeks gestation)
  - 1 month - 12 years: 0.5 - 1 mg/kg (maximum 4 mg/kg), repeated every 8 hours as necessary
  - 12 - 18 years: 20 - 40 mg, repeated every 8 hours as necessary; higher doses may be required in resistant cases
- Or:
  - Mannitol 20% solution
  - Adult: 50 - 200 g by intravenous infusion over 24 hours, preceded by a test dose of 200 mg/kg by slow intravenous injection
  - Child:
    - neonate 0.5 - 1 g/kg (2.5 - 5 ml/kg of 20% solution) repeated if necessary 1 - 2 times after 4 - 8 hours
    - 1 month - 18 years: 0.5 - 1.5 g/kg (2.5 - 7.5 ml/kg of 20% solution); repeat if necessary 1 - 2 times after 48 hours

**Vascular diseases**
- Abdominal pain and vomiting
- Alternating hemiplegia
- Basilar artery migraine - predominantly brain stem symptoms
- Dysarthria
- Vertigo
- Tinnitus
- Decreased hearing
- Diplopia
- Ataxia
  - May coexist with tension-type headache
  - May present without headache (migraine equivalent) usually seen in psychiatry
  - May present with complications: stroke-like manifestations

**Complications**
- Stroke
- Epilepsy
- Blindness

**Investigations**
- Neuro-imaging
  - CT scan
  - MRI
  - EEG

**Treatment objectives**
- Eliminate pain
- Prevent recurrence

**Non-drug treatment**
- Manage in a quiet (and dark) room
- Psychotherapy
- Physiotherapy/biofeedback

**Drug treatment**

**Acute attack**
- Aspirin (acetylsalicylic acid) tablets 300 - 900 mg every 4 - 6 hours when necessary maximum 4g daily
- Child and adolescent - not recommended (risk of reye’s syndrome)
- With an anti-emetic agent (e.g. metoclopramide), or other non-steroidal anti-inflammatory agents plus metoclopramide
- Ergotamine preparations (useful only during the aura phase)
  - Adult: 1 - 2 mg orally at first sign of attack; maximum 4 mg in 24 hours
  - Do not repeat at intervals of less than 4 days; maximum 8 mg in any one week
  - Not to be used more than twice in any one month
  - Child: not recommended

**Prophylaxis**
- Consider for patients who:
  - Suffer at least 2 attacks a month
  - Suffer an increasing frequency of headaches
  - Suffer significant disability inspite of suitable treatment for acute attacks
- Cannot take suitable treatment for acute attacks
- Available options are:
  - Propanolol
  - 40 mg orally every 8 - 12 hours
  - Tricyclic antidepressants, notably amitryptiline
  - 10 mg orally at night, increased to a maintenance dose of 50 - 75 mg at night
  - Sodium valproate
    - Initially 300 mg orally every 12 hours, increased if necessary to a maximum of 1,2 g daily in 2 divided doses
  - In refractory cases:
    - Cypéroptadine
    - An antihistamine with serotonin-antagonist and calcium channel-blocking properties
    - 4 mg orally; a further 4 mg if necessary; maintenance 4 mg every 4 - 6 hours
Postural instability with frequent falls
Gait changes: shuffling gait with flexed posturing
Parkinsonism may occur in association with other neurodegenerative diseases

Differential diagnoses
- Multi-infant dementia
- Alzheimer's disease
- Normal pressure hydrocephalus
- Brain tumour
- Benign essential tremor
- Depression
- Creutzfeldt-Jakob disease

Complications
- Recurrent falls with attendant complications e.g. subdural haematoma
- Dementia
- Depression

Investigations
- Diagnosis is essentially clinical
- Neuro-imaging: CT scan/MRI for exclusion of possible differentials

Drug treatment
- L-dopa/carbidopa (dose expressed as levodopa) 50 mg orally every 6 - 8 hours increased by 100 mg once or twice weekly depending on response
- Anti-cholinergic drugs for tremors
  - Trihexyphenidyl (benzhexol) 1 mg orally daily, increased gradually (usually 5 - 15 mg in 3 - 4 divided doses up to a maximum of 20 mg)
  - Dopamine receptor agonists
    - Bromocriptine 1 - 1.25 mg orally in the first week; 2 - 2.25 mg orally in the 2nd week; 2.5 mg twice daily in the 3rd week; 2.5 mg three times daily in the 4th week, increasing by 2.5 mg every 1 - 2 weeks according to response (usual range is 10 - 40 mg daily)
    - Ropinirole 1 - 3 mg orally once daily (in resistant cases)

Supportive measures
- Physiotherapy for postural adjustments
- Antidepressants
- Amitriptyline for pain (which could be quite incapacitating) especially with dopamine-replacement drugs

Notable adverse drug reactions, caution and contraindications
- Dopamine replacement drugs: dyskinesia, pain
- Advisable to start with small doses and gradually increase
- There is need for dosage and timing adjustments when side effects manifest
- Dop-a-agonists: postural hypotension; may cause vomiting

- Caution is advised to avoid falls
- Anticholinergic drugs: constipation; memory problems
- Contraindicated in the presence of glaucoma

Prevention
- Avoid identified causative agents where feasible
- Timely and appropriate treatment to prevent/reduce complications

SEIZURES/EPILEPSIES

Introduction
A seizure results from abnormal excessive electrical discharge of brain cells
- Epilepsy is a condition characterized by recurrent (≥ 2) seizures unprovoked by any immediate identifiable cause
- May be idiopathic or could follow:
  - Cerebral infections
  - Metabolic derangements (glucose, electrolytes, fluids)
  - Stroke
  - Tumours
  - Head trauma
  - Birth injury/asphyxia
  - Drug abuse/overdose/withdrawal
- Neuro-degeneration

Clinical features
- Classical attack with sudden loss of consciousness, convulsions (tonic and/or clonic)
- Abnormal sensation or perception
- Autonomic disturbances: epigastric discomfort, sphincteric incontinence
- Semi-purposive actions (automatisms)
- Aura
- Loss of postural tone (sudden falls without convulsions)
- Limb paralysis (Todd's paralysis) usually after attacks

Differential diagnoses
- Migraine headache
- Syncope
- Narcolepsy
- Panic attacks
- Catatonic schizophrenia
- Transient ischaemic attacks
- Hysteria
- Ménière's disease

Complications
- Status epilepticus
- Cardiac arrhythmias
- Renal failure from myoglobinuria
- Cerebral hypoxia/anoxia resulting in brain damage
- Sudden death

Investigations
- Electroencephalography
- Neuro-imaging: CT scan, MRI
- Random blood glucose
- Urea, Electrolytes and Creatinine

Treatment objectives
- Arrest convulsions/attacks
- Treat underlying cause if identified
- Improve quality of life

Drug treatment
- Parenteral drugs are recommended for acute attacks/status epilepticus
- Diazepam

Adult: 10 - 20 mg by slow intravenous injection; repeat if necessary in 30 - 60 minutes
Child: 200 - 300 micrograms/kg or 1 mg per year of age

Could be given per rectum as rectal solution in restless patients
- 500 micrograms/kg (up to a maximum of 30 mg) in adults and children over 10 kg
- Phenytin

Adult: initially 15 mg/kg by slow intravenous injection or infusion (with blood pressure and Electrocardiograph monitoring) at a rate not more than 50 mg/minute; then 100 mg every 6-8 hours
Child: neonate - 12 years: initial loading dose 20 mg/kg by slow intravenous injection, then 2 - 4 mg/kg orally every 12 hours, adjusted according to response (usual maximum dose 7.5 mg/kg every 12 hours)

1 month - 12 years: initially 1.5 - 2.5 mg/kg every 12 hours, adjusted according to response to 2.5 - 5 mg/kg every 12 hours (usual maximum dose 7.5 mg/kg every 12 hours or 300 mg daily)

12 - 18 years: initially 75 - 150 mg every 12 hours, adjusted according to response up to 150 - 200 mg every 12 hours (usual maximum 300 mg every 12 hours)

Sodium valproate

Adult: 600 mg daily in 2 divided doses
Child: neonate, initially 20 mg/kg orally or per rectum once daily; usual maintenance dose 10 mg/kg twice daily

1 month - 12 years: initially 5 - 7.5 mg/kg every 12 hours; maintenance 12.5 - 15 mg/kg every 12 hours

12 - 18 years: usually 300 mg every 12 hours, increased in steps of 200 mg at 3-day intervals; usual maintenance 500 mg - 1 g twice daily (maximum 1.25 g twice daily)

Partial seizures
- Carbamazepine

Adult: 100 - 200 mg orally 1-2 times daily
- Not recommended in pregnancy
Child: neonate - 12 years: initially 5 mg/kg orally at night or 2.5 mg/kg twice daily, increased as necessary by 2.5 - 5 mg/kg every 3 - 7 days; usual maintenance 5 mg/kg every 8 - 12 hours

12 - 18 years: initially 100 - 200 mg 1 - 2 times daily, increased slowly to usual maintenance of 400-600 mg every 8 - 12 hours

Absence attacks
- Ethosuximide

Adult: 500 mg daily initially; increase by 250 mg at intervals of 4 - 7 days to doses of 1 - 1.5 g daily (maximum dose 2 g daily)

Child over 6 years: same as adult dose
Up to 6 years: 250 mg daily; increase gradually to 20 mg/kg daily (maximum 1 g daily)

Non-drug treatment
- Psychotherapy
- Health education to patients, relatives and public
- Discourage harmful cultural practices e.g. burning, mutilation

Notable adverse drug reactions, caution and contraindications
- Administer 10 - 20 mL per rectum as an enema
- Child: neonate-0.4 mL/kg (maximum 0.5 mL) as a single dose; up to 3 months: 0.5 mL; 3 - 6 months: 1 mL; 6 - 12 months: 1.5 mL; 1 - 2 years 2 mL; 3 - 5 years 3 - 4 mL; 6 - 12 years 5 - 6 mL (administered as a single dose per rectum) per kg body weight
- Not recommended in pregnancy
- Cerebral decompression with mannitol 20% infusion or furosemide if indicated (see meningitis)

Maintenance therapy in day-to-day care

Generalized epilepsies
- Phenobarbital

Adult: 60 - 180 mg orally daily
Child: 5 - 8 mg orally daily

Phenytoin

Adult: 150 - 300 mg orally daily
Child: neonate- initial loading dose by slow intravenous injection then 2 - 4 mg/kg by mouth every 12 hours adjusted according to response (usual maximum 7.5 mg/kg every 12 hours)

1 month - 12 years: 1.5 - 2.5 mg/kg orally every 12 hours (usual maximum 7.5 mg/kg every 12 hours or 300 mg daily)

12 - 18 years: initially 75 - 150 mg every 12 hours, adjusted according to response up to 150 - 200 mg every 12 hours (usual maximum 300 mg every 12 hours)

Sodium valproate
Most antiepileptics: skin rashes, especially Stevens-Johnson syndrome; exfoliative dermatitis
Introduce drugs singly because of possible interaction between drugs
Doses must be gradually increased to avoid toxicity and other side effects
Do not use paraldehyde if it has a brownish colour or the odour of acetic acid
All antiepileptics must be withdrawn slowly so as not to precipitate status epilepticus

**Prevention**
Prompt treatment of fever in children to avoid febrile convulsions
Prevention of head injuries
Treat diseases of the brain early to avoid poor healing and death of brain cells
Immunization of children against communicable diseases
Address causative factors (see above)
Avoid driving and swimming unattended, and operation of machinery

**STROKE**

**Introduction**
A condition resulting from disruption of blood supply to brain cells with disability lasting more than 24 hours or resulting in death
Could result from:
- Occlusion (ischaemic)
- Rupture of blood vessels with bleeding into the brain substance or into the subarachnoid space (haemorrhagic)

**Clinical features**
Classical stroke:
- Sudden motor weakness, with/without speech, visual and sensory impairment
Subarachnoid haemorrhage:
- Severe headache, neck stiffness and positive Kernig's sign
Stroke-in-evolution:
- Gradual onset of deficit with progression
Mass lesion:
- Sudden rise in intracranial pressure
- Loss of consciousness, respiratory changes, pupillary changes
- Sudden death
- Lacunar syndrome:
- Complete deficits: speech defects with clumsy hand involvement
- Pure motor and/or pure sensory deficits
Dementia:
- Arises from small, recurrent strokes resulting in cognitive impairment and functional dependence

**Differential diagnoses**
Brain tumour
Subdural haematoma

**Brain abscess**
Meningitis/encephalitis
Cerebral malaria
Migraine headache
Multiple sclerosis
Metabolic derangements e.g. hypoglycaemia, hyperosmolar non-ketotic coma

**Complications**
Tentorial herniation with coning and death
Cardiac arrhythmias
Depression
Epilepsy
Dementia
Parkinsonism
Hyperglycaemia

**Investigations**
- Neuro-imaging with CT scan/MRI to determine stroke type and choice of management
- Lumbar puncture for CSF analysis in suspected subarachnoid haemorrhage
- Electrocardiography
- Echocardiography
- Carotid Doppler ultrasound study
- Cerebral angiography
- Full Blood Count with differentials
- Random blood glucose
- Urea, Electrolytes and Creatinine
- Chest radiograph
- HIV screening

**Treatment objectives**
- Restore cerebral circulation
- Limit disability
- Treat identified risk/predisposing factors
- Reduce raised intracranial pressure
- Treat complications (if any)

**Non-drug treatment**
- Attention to calories, fluid balance
- Physiotherapy for passive muscle exercises
- Nursing care (frequent turning and bladder care) to prevent decubitus ulcers and urinary tract infection
- Rehydration

**Drug treatment**
- Cerebral decompression if there is evidence of raised intracranial pressure
- Furosemide 40 mg every 8 hours by slow intravenous injection for 6 doses
- Treat underlying conditions such as diabetes mellitus, hypertension, and thrombosis

**Notable adverse drug reactions, caution**
- Rebound cerebral oedema when mannitol is discontinued
- Thrombolytic agents: bleeding tendencies
- Diazepam by the intravenous route must be administered slowly to avoid respiratory depression and laryngeal spasm

**Prevention**
- Treat/control known risk factors
- Hypertension
- Diabetes mellitus
- Cardiac diseases
- Hyperlipidaemia
- Obesity
- Smoking
- Excessive alcohol consumption
- Give low dose aspirin (acetylsalicylic acid) to patients at risk if tolerated

**SYNCOPE**

**Introduction**
Loss of consciousness and postural tone as a result of diminished cerebral blood flow
May be due to:
- Vaso-vagal attack
- Cardiac causes
- Prolonged standing
- Severe emotional disturbance
- The more severe form is associated with various heart diseases:
  - Arrhythmias (especially complete heart block)
  - Hypertrophic cardiomyopathy
  - 'Heart attack' (myocardial infarction)
  - Atrial myxoma
  - Aortic stenosis
  - Dissecting aneurysm
- Other causes:
  - Pulmonary embolism
  - Vertebral-basilar insufficiency
  - Subclavian steal syndrome
  - Carotid sinus pressure
  - Migraine headache

**Clinical features**
- Sudden loss of consciousness
- Cold extremities
- Bluish discoloration of extremities (cyanosis)
- Pulse irregularities (or pulselessness)
- Hypotension (or unrecordable blood pressure)
- Fainting induced by pressure on the neck
- Fainting induced by coughing, micturition

**Differential diagnoses**
Epilepsy
Myocardial infarction
Stroke
Aortic dissection
Hysteria

**Complications**
Cerebral hypoxia/anoxia resulting in brain damage
Stroke
Sudden death

**THE UNCONSCIOUS PATIENT**

**Introduction**
An unresponsive patient who may also have breathing and circulatory problems
May be neurological or may result from other systemic diseases
An easy way of finding the cause is to think in terms of the vowels
A: Apoplexy (stroke)
E: Epilepsy
I: Infections e.g. meningitis-encephalitis
O: Overdosing with drugs, alcohol intoxication, toxins
U: Uraemia and other metabolic disorders
Other causes include:
- Head injury
- Brain tumours (with complications)

**Clinical features**
Varying levels of impaired consciousness:
- Comatose: no response to stimulus, however painful
- Stuporose: a state deeper than sleep; vigorous stimulation required to stimulate response
- Other features:
  - Cessation of respiration or abnormal ventilatory patterns: Cheyne-Stokes, ataxic, apneustic, gasping etc
- Unresponsiveness or variable response to painful stimuli

**Investigations**
- Electrocardiography
- Echocardiography
- Neuro-imaging: CT scan, MRI, carotid Doppler
- Random blood sugar
- Urea, Electrolytes and Creatinine
- Chest radiograph
- HIV screening
Chapter 4: Central Nervous System

Non-drug treatment
Psychosocial interventions
Cognitive behavioural therapy
Marital and family therapy
Group therapy

Drug treatment
Only occasionally required, and following careful assessment

Note
- Detoxification is required for severe withdrawal syndrome or delirium tremens
- This will involve the administration of a long-acting benzodiazepine and thiamine supplements over 7 - 10 days

Supportive measures
Rehabilitation to
- Sustain abstinence
- Acquire an alcohol-free lifestyle
- Prevent relapse

Prevention
Health education (including school health education, peer group education and self help group e.g. alcoholic anonymous)
Government regulation of alcohol use

AHXNODY ORDER
Introduction
Generalized anxiety disorder (GAD) is characterized by exaggerated worry and tension, even when there is little or no cause for anxiety

A chronic disorder affecting about 2 - 3% of the population

Clinical features
- Pre-occupations: often of diverse nature
- Poor concentration
- Muscle aches and headaches
- Irritability
- Sweating
- Fatigue
- Insomnia
- Shortness of breath

Differential diagnoses
Medical causes of suggestive symptoms and signs (e.g. hyperthyroidism)

Complications
- Chronicity
- Co-morbid depression
- Medical morbidity (e.g. hypertension)

Investigations
- To exclude medical/physical cause(s)

Treatment objectives
Achieve remission of symptoms
- Prevent relapse

Non-drug treatment
Cognitive-behavioural therapy

Drug treatment
Diazepam 10 - 20 mg orally daily
Or:
Imipramine 50 - 150 mg orally daily
Or:
Fluoxetine 20 - 60 mg orally daily

Supportive measures
- Relaxation techniques
- Exercise
- Psychotherapy

Notable adverse drug reaction, caution
The risk of dependence (and withdrawal syndromes) limits the utility of benzodiazepines for treatments of long duration

Prevention
- Avoid of undue and extreme stress
- Avoid psycho-active substances

BIPOLAR DISORDERS
Introduction
A type of mood disorder in which there is (typically) alternation of a depressive phase and a manic or hypomanic phase

Experienced by about 1% of the adult population at some point in their lifetime
About equal incidence between males and females
May be precipitated by psychosocial stress; strong genetic vulnerability often present

Clinical features
- Depressive phase:
  - Low mood
  - Impaired appetite and sleep
  - Ideas of worthlessness or hopelessness
  - Suicidal ideation
- Other depressive symptoms and signs
  - Manic or hypomanic phase:
    - Elation
    - Euphoria
    - Irritability
    - Expansive mood
    - Disturbed sleep
    - Grandiosity
    - Disinhibition

Differential diagnoses
- Schizo-affective disorder
- Schizophrenia
- Organic mood/affective disorder (including effects of drug abuse)

Complications
Social and personal consequences of inappropriate behaviour (e.g. unplanned pregnancy, sexually-transmitted infections, etc)
Suicide
Increased risk of morbidity (reduce life expectancy) (e.g. trauma and accidents)
Increased mortality

Investigations
Investigations as indicated to rule out organic/medical causes

Full Blood Count and renal function tests (to determine suitability of mood stabilizers)

Treatment objectives
Reduce risk to self and others
Normalize mood
Return to full functional status
Prevent recurrence

Drug treatment
Cognitive-behavioural therapy as sole treatment in mild cases, and adjunct in all others
Electroconvulsive therapy (ECT)

- An effective and essentially safe treatment for severe and acute presentations
- A course of 8-12 treatments are usually needed

Drug treatment

Treat underlying causes
Lithium
- 1”line drug following established diagnosis
Adult: initially 1 - 1.5 g daily
Prophylaxis: initially 300 - 400 mg daily
Child: not recommended
- Measure serum lithium concentration regularly (every three months on established regimens)
- Adjust dosage to achieve serum levels of 0.6 - 1.2 mEq/L

Sodium valproate
Adult: 750 mg - 2 g orally/day
Child: neonate, initially 20 mg/kg orally once daily; usual maintenance dose 10 mg/kg every 12 hours daily
1 month - 12 years: initially 5 - 7.5 mg/kg every 12 hours, usual maintenance dose 12.5 - 15 mg/kg every 12 hours (up to 30 mg/kg twice daily)
12 - 18 years: initially 300 mg every 12 hours, increased in steps of 200 mg daily at 3-day intervals; usual maintenance dose 0.5 - 1 g twice daily (maximum 1.5 g daily)

Carbamazepine
Adult: 600 - 1,800 mg orally daily
Child: 1 month - 12 years: initially 5 mg/kg orally at night or 2.5 mg/kg twice daily, increased as necessary by 2.5 - 5 mg/kg every 3 - 7 days
- Maintenance dose 5 mg/kg 2 - 3 times daily, increased slowly to usual maintenance of 400 - 600mg 2 - 3 times daily

Antidepressants
- TCAs or SSRIs may be indicated in depressive phase
Antipsychotics
- Haloperidol 1.5 to 3 mg orally 2 - 3 times daily (may be indicated in acute manic phase)
Child 2 - 12 years: initially 12.5 - 25 micrograms/kg orally twice daily, adjusted according to response to maximum 10 mg daily
12 - 18 years: initially 0.5 - 3 mg daily, adjusted according to response to lowest effective maintenance dose (as low as 5 - 10 mg daily)

Supportive measures
Pschotherapy and social intervention for patient and relatives/caregivers

Notable adverse drug reactions
- More likely with doses above recommended upper limits
- Lithium
- Gastrointestinal disturbances
- Tremors
- Confusion
- Myoclonic twitches
- Carbamazepine: hypersensitivity reactions

Transient memory impairment is common following ECT

Prevention
No primary preventive measures are clearly delineated

Adherence to therapy with mood stabilizers until discontinuation is considered prudent (this is individually determined)

DELIRIUM

Introduction
A transient disorder of brain function
Manifests as a global cognitive impairment and behavioural disturbance

More common at the extremes of life though it can occur at any age
Incidence up to 15% has been reported among general medical inpatients; up to 40% among acutely ill geriatric patients

Post-reduction and mis-diagnosis are common

The most common causes are:
- Trauma
- Infections
- Metabolic derangements
- Side effects of drugs

Clinical features
- Disturbance of consciousness
- Disorientation
- Memory deficits
- Language disturbances
- Perceptual disturbances
- Rapid fluctuations
- Disruption of sleep-wake cycle
- Psychomotor hyperactivity
- Mood alterations

Differential diagnoses
- Dementia
- Acute (idiopathic) psychotic disorders

Complications
- Increased mortality

Investigations

- Electrocardiogram
- Full Blood Count and differentials
- Thyroid function test
- Renal function tests
- Glucose level

Prevention

- Giving reassurance to patient and relatives/caregivers
- Early treatment of infective and metabolic conditions
- Care with the use of drugs (especially anticholinergic medications) in the elderly

DEPRESSION

Introduction
A disorder of mood and affect in which the predominant emotion is sadness/unhappiness
Can occur alone (unipolar depression) or as part of an alternation disorder in which elevation of mood also occurs (bipolar disorder)

Varies in severity from mild to severe Life events, especially those involving loss, are often (but not always) the triggers

Strong genetic is vulnerability sometimes present

Occurs in about 2 - 5% of the population at any given time and in about 10 - 25% in their lifetime
Women are generally at an elevated risk

Clinical features
Sadness, unhappiness, feeling low
Loss of interest in usual activities
Reduced energy
Disturbance of sleep and appetite
Impaired concentration
Ideas of worthlessness, guilt, or failure
Morbid or suicidal rumination or ideation
Somatic complaints of various types

Differential diagnoses
Normal grief reaction
Mood disorders
Anxiety disorders
Substance use disorders
Post-traumatic stress disorder

Complications

- Increased mortality
- Usually transient but may be associated with increased morbidity (e.g. from falls) and mortality
- Suicide
- Recurrence (in 50% or more)

Investigations

- Full Blood Count and differentials
- Thyroid function test
- Indicative infection screen

Prevention

- Normalizing mood
- Suicide prevention
- Return to active life
- Prevent recurrence
- Non-drug treatment
- Cognitive-behavioural treatment
- Inter-personal psychotherapy

Drug treatment

- Tricyclic antidepressants (TCAs)
- Amitriptyline in increasing doses up to 150 mg orally/day
- Fluoxetine 20 - 80 mg orally/day

Supportive measures

- Supportive psychotherapy for patients and family/caregivers

Notable adverse drug reactions, caution
Tricyclic antidepressants:
- Dryness of the mouth
- Urinary retention
- Constipation
- Blurring of vision
- Selective Serotonin Reuptake Inhibitors (SSRIs)
- Sleep disturbance
- Sexual dysfunction
- Serotonin syndrome
- Cardiac toxicity, especially in overdose with TCAs and SSRIs

Increased suicidal ideation in adolescents
Non-drug treatment
Psycho-social interventions as indicated (including social and occupational therapy)
Psycho-education for patient and relatives / caregivers
Supportive psychotherapy
ECT (especially for catatonic forms)

Drug treatment
Chlorpromazine

Adult: initially 25 mg orally every 8 hours (or 75 mg at night), adjusted according to response to usual maintenance dose of 75 - 300 mg daily
- Elderly: a third to half adult doses
By deep intramuscular injection: 25 - 50 mg every 6 - 8 hours
Child: 1 - 5 years: 500 micrograms/kg orally every 6 - 8 hours (maximum 40 mg daily), 6 - 12 years: a third to half adult dose (maximum 75 mg daily)
Haloperidol
- Adult: initially 1.5 - 3 mg every 8 - 12 hours or 3 - 5 mg every 8 - 12 hours in severely affected or resistant patients
- In resistant schizophrenia, up to 30 mg daily may be needed, adjusted according to response to the lowest effective maintenance dose (as low as 5 - 10 mg daily) Elderly, initially half adult dose
Child: initially 25 - 50 micrograms/kg daily in 2 divided doses (maximum 10 mg)
Fluphenazine
- Adult: initially 2 - 10 mg every 8 - 12 hours, adjusted according to response to 20 mg daily
- Doses above 20 mg daily (10 mg in elderly) only with special precaution
Or: 25 - 100 mg intramuscularly fortnightly to monthly
Child: not recommended

Supportive measures
Sleep hygiene
Behavioral modifications to enhance relaxation
Avoid habits and lifestyles that promote insomnia
Improve environmental/sleeping conditions

Drug treatment
Supportive psychotherapy
Social and occupational therapy
Cognitive therapy (as adjunct in the treatment of persisting psychotic experience)
Rehabilitation

Notable adverse drug reactions
Extrapyramidal and Parkinsonian symptoms (may require anticholinergic medication)
Tardive dyskinesia
Weight gain
Aggranulocytosis (monitor blood counts in patients on clozapine)

Prevention
No clear/specific scope for primary prevention at present
Secondary and tertiary:
- Early and effective treatment
- Rehabilitation to reduce disability

Non-drug treatment
Psycho-social interventions as indicated (including social and occupational therapy)
Psycho-education for patient and relatives / caregivers
Supportive psychotherapy
ECT (especially for catatonic forms)
**ACUTE NECROTIZING ULCERATIVE GINGIVITIS**

**Definition**
A polymicrobial, endogenous infection

**Aetiology**
Fusiform and spirochaete bacteria

**Epidemiology**
In developing countries, seen almost exclusively in children

**Related to poverty and malnutrition**

**In industrialized countries, most common in young adults with neglected mouths; smoking and stress have been associated**

**Clinical features**
Crater ulcers straiting at the tips of the interdental papillae

Ulcers spread along gingival margins

Gingival soreness and bleeding

Foul breath

Metallic taste

Increased salivation

Cervical lymphadenopathy and fever in advanced cases

**Differential diagnoses**

- Primary herpetic gingivo-stomatitis
- HIV-associated acute ulcerative gingivitis

**Gingival ulceration in acute leukocoria or aplastic anaemia**

**Investigations**
Smears from ulcers show predominantly spirochaetes and gram negative fusiform bacteria

**Treatment objectives**
- Treat infection
- Restore oral health

**Non-drug treatment**
- Oral hygiene (debridement) is essential
- Metronidazole

**Drug treatment**

**Metronidazole**
Adult: 200 mg orally 8 hourly for 3 days
Child: 1 - 3 years: 50 mg orally every 8 hours for 3 days; 3 - 7 years: 100 mg every 12 hours; 7 - 10 years: half adult dose

**Supportive therapy**
Ascorbic acid

**Adults** not less than 250 mg orally daily (in divided doses)

Child: 1 month - 4 years: 125 - 250 mg in 1 - 2 divided doses

- 4 - 12 years: 250 - 500 mg daily in 1 - 2 divided doses; 12 - 18 years 500 - 1 g daily in 1 - 2 divided doses

- Ferrous sulfate

Adult: 200 mg orally three times daily taken before food

Child 6 - 12 years: half adult dose

**Follow-up treatment**
Rehabilitation of the mouth

Once the acute phase has subsided, oral hygiene should be brought to as high a standard as possible to lessen the risk of recurrence

**Sequestrectomy**

**Notable adverse drug reactions, caution**

- Metronidazole: nausea, vomiting, unpleasant taste; disulfiram-like effect with alcohol.

**ACUTE PERIAPICAL ABSCESS**

**Definition**
A localized collection of pus in the periapical region of a tooth

**Aetiology**
May develop either directly from acute periapical periodontitis or more usually from a chronic periapical granuloma

**Clinical features**
Painful swelling at the root of tooth

Sinus (may be present)

Tooth is tender to biting or percussion

**Tooth mobility**

**Differential diagnoses**

- Inflammatory radicular cyst
- Osteomyelitis
- Periodontal abscess

**Investigations**
Radiographs (periapical)

**Treatment objectives**
- Remove source of infection e.g. fish-bone, other foreign objects
- Drain abscess using local anaesthesia
- Treat residual infection

**Non-drug treatment**
Extraction (or endodontic treatment) i.e. root canal therapy

**Drug treatment**
Amoxicillin
Adult: 250 mg orally every 8 hours for 5 to 7 days

Child: up to 10 years 125 mg every 8 hours, doubled in severe infections

- Metronidazole

Adult: 200 mg orally every 8 hours for 3 days

Child: 1 - 3 years: 50 mg orally 8 hourly for 3 days; 3 - 7 years: 100 mg every 12 hours; 7 - 10 years: half adult dose

**ALVEOLAR OSTEITIS**

**Introduction**
The most frequent painful complication of extractions

Caused by destruction of the clot that normally fills the socket

**Pathogenesis**
Rapid spread is most likely related to release of large amounts of streptokinase and hyaluronadase which are produced by most strains of streptococci

**DENTAL CARIES**

**Definition**
A progressive bacterial damage to teeth exposed to the saliva

**Classification**
- Enamel caries
- Dentine caries
- Root surface caries

**Aetiology**
Develops over time in the presence of certain interacting variables

**Investigations**
Culture (blood and swab) and sensitivity testing

**Non-drug treatment**
- Early treatment of carious teeth

**Drug treatment**
- Aggressive antibiotic treatment
- Intravenous co-amoxiclav (given over 3 to 4 minutes) in combination with intramuscular gentamicin for 5 days
- Injection co-amoxiclavulanate

Adult: 1,000/200 mg intravenously every 8 hours

Child: neonate and premature infants, 25 mg/kg every 12 hours; infants up to 3 months, 25 mg/kg every 8 hours, 3 months to 12 years, 25 mg/kg every 8 hours increased to 25 mg/kg every 6 hours in more severe infections

- Injection gentamicin:
  - Adult: 3 - 5 mg/kg daily in divided doses every 8 hours
  - Child: up to 2 weeks: 3 mg/kg every 12 hours; 2 weeks - 12 years: 2 mg/kg every 8 hours

**Precaution**
Gentamicin may cause significant ototoxic and nephrotoxic effects

**Prevention**
Early treatment of carious teeth

The fascial space infections may involve sublingual, submandibular and/or parapharyngeal spaces

Ludwig's angina is bilateral cellulitis of the sublingual and submandibular spaces

**Clinical features**
Diffuse, tense, painful swelling of the involved soft tissues

- Malaise
- Elevated temperature

Ludwig's angina causes airway obstruction which can quickly result in asphyxiation

**Complications**
Suppuration and abscess formation may occur later if treatment is neglected or delayed

**Respiratory difficulty: cellulitis affecting mandibular teeth**

**Investigations**
Culture (blood and swab) and sensitivity testing

**Non-drug treatment**
- Drainage of the swelling to reduce pressure (oral drain may also be placed)
- Secure the airway by tracheostomy if necessary

**Drug treatment**
- Aggressive antibiotic treatment
- Intravenous co-amoxiclav (given over 3 to 4 minutes) in combination with intramuscular gentamicin for 5 days
- Injection co-amoxiclavulanate

Adult: 1,000/200 mg intravenously every 8 hours

Child: neonate and premature infants, 25 mg/kg every 12 hours; infants up to 3 months, 25 mg/kg every 8 hours, 3 months to 12 years, 25 mg/kg every 8 hours increased to 25 mg/kg every 6 hours in more severe infections

- Injection gentamicin:
  - Adult: 3 - 5 mg/kg daily in divided doses every 8 hours
  - Child: up to 2 weeks: 3 mg/kg every 12 hours; 2 weeks - 12 years: 2 mg/kg every 8 hours

**Precaution**
Gentamicin may cause significant ototoxic and nephrotoxic effects

**Prevention**
Early treatment of carious teeth

**DENTAL CARIES**

**Definition**
A progressive bacterial damage to teeth exposed to the saliva

**Classification**
- Enamel caries
- Dentine caries
- Root surface caries

**Aetiology**
Develops over time in the presence of certain interacting variables
Predisposing factors

Investigations

Clinical features

- Denture wearing
- Reduced salivation (e.g. drug induced)
- Antibiotic therapy (especially broad spectrum)
- Poorly controlled diabetes mellitus
- Steroid therapy (chronic)
- Salivary gland damage (e.g. post radiation)
- Malnutrition
- HIV infection
- Leukaemia
- Iron, vitamin B, folic acid deficiency

PERICORONITIS

Agranulocytosis

Introduction

An inflammatory condition of the gum/flap around a partially erupted tooth

Common around the lower last molars or wisdom teeth

Upper canine may also be affected

Classification

Acute

Chronic

Acute-on-chronic

Aetiology

Food impaction and plaque accumulation under gum flap

Trauma to gum flap from opposing tooth

Ulcerative gingivitis

Reduced resistance

Aerobes in plaque

Clinical features

Soreness and tenderness around partially-erupted tooth

Swelling

Enlargement of regional lymph nodes

Fever

Abscess formation

Investigations

Radiographs

- To establish the position of the affected tooth and its relationship to the second molar

- May show impacted third molar

Non-drug treatment

When mouth opening is possible: careful irrigation under the gum flap to clear debris, using warm saline mouthwash

- To be done frequently until stagnation area is removed

Drug treatment

- Amoxicillin

- Hexetidine mouthwashes to alternate with warm saline mouthwashes

- 2% three times daily for 1-2 weeks

- Continue for 48 hours after lesions have resolved

Non-drug treatment

- Carbohydrate diet

- Viridans streptococci bacteria

- Susceptible tooth surface

Pathogenesis

- Enamel caries progresses in the following stages:

- Early (sub-microscopic) lesion

- Phase of non-bacterial enamel crystal destruction

- Cavity formation

- Bacterial invasion of enamel

Clinical features

- Pulpitis

- If not treated can cause apical periodontitis and dentineocellular abscess

Investigations

- Periapical radiographs

- Bitewing radiographs

- Electric pulp tests

- Thermal test

Non-drug treatment

- Amalgam filling, Glass Ionomer Cement (GIC) composite and Atraumatic Restorative Technique (ART) for enamel caries

- Amalgam filling, GIC for dentine caries

- Root Canal Therapy, pulp capping pulpotomy, pulpectomy for pulp involvement

Drug treatment

- Analgesics pre-operatively

- Paracetamol 1 g 4-6 hourly orally to a maximum of 4 g daily

- Metronidazole: nausea, vomiting and metallic taste

- Metronidazole is contraindicated in pregnancy

- Avoid alcohol during treatment with metronidazole, and for at least 48 hours after

Prevention

- Oral health education

- Scaling and polishing every six months

NEOPLASMS OF THE ORAL CAVITY refer to specialist care

ORAL THRUSH (Candidiasis)

Introduction

A clinical infection of mucous membranes due to the fungus species Candida albicans is the most frequently isolated strain

Classification

- Acute oral candidosis

- Chronic oral candidosis

- Denture association candidosis/denture stomatitis

Pathogenesis/aetiology

- Imunosupression results in the Candida albicans (a normal oral commensal) becoming virulent

- It invades and proliferates in superficial epithelium

- Results in a thick plaque which is oedematous and not easily rubbed off

- Some patients may require systemic antimicrobial medicines

- Fluconazole

- 50 mg orally daily for 7-14 days

- Chronic: 6 mg/kg on the first day, then 3 mg/kg daily

- For neonates up to 2 weeks old: administer every 72 hours; 2-4 weeks old: administer every 48 hours

PERIODONTITIS

Introduction

An inflammatory condition of the periodontium:

- periodontal ligament, cementum, alveolar bone, gingivae

Classification

- Acute periodontitis

- Chronic periodontitis

- Juvenile periodontitis

- Other sub-classifications
Acute periodontitis
Relatively uncommon
Of short duration; may be due to trauma, abscess or ulceration
Characterized by pain
- May be associated with bleeding, fever, swelling and redness of the mucosa, unpleasant taste in the mouth

Chronic periodontitis
A sequel of chronic gingivitis
Symptoms are the same as in the acute type, but with less pain and longer history

Clinical features
Inflammation
- Destruction of the periodontal membrane fibres
- Resorption of the alveolar bone
- Migration of the epithelial attachment along root towards the apex
- Pocket formation around the tooth

Juvenile periodontitis
An uncommon disease characterized by periodontal destruction, often in the absence of overt gingival inflammation

Epidemiology
Prevalence 1:1000; male = female
Onset at puberty or earlier

Clinical features
- Affects the first permanent molar and incisors
- Actinobacillus, Actinomyces comitans has been isolated from the affected sites
- Results in drifting and loss of the first permanent molar and incisors

Investigation
- Radiology may reveal marked bone loss interdentally, infra-radically and apically

Complications
- Tooth loss
- Malocclusion
- Temporo-Mandibular Joint (TMJ) dysfunction syndrome

Non-drug treatment
- Control of plaque bacteria by use of antiseptic solution
- Establishing a healthy gingival and periodontal attachment
- Oral hygiene instruction and motivation
- Regular scaling and polishing
- Root planing
- Splinting of mobile tooth
- Periodontal surgery
- Bone regenerative techniques e.g. using Polytetrafluoroethylene (PTFE) membranes, Bio-Oss, Bio-membrane

Drug treatment
- Metronidazole
  - Adult: 200 mg orally every 8 hours for 5 days
  - Child 1 - 3 years: 50 mg orally every 8 hours; 3 - 7 years: 100 mg every 12 hours; 7 - 10 years: 100 mg every 8 hours; 10 - 18 years: 200 mg every 8 hours
- Plus:
  - Tetracycline 250 mg orally daily for up to 21 days
  - Child under 12 years: metronidazole and amoxicillin (or erythromycin for those sensitive to penicillin)

Precaution
- Tetracyclines should not be given to children under 12 years

PULPITIS
Introduction
Inflammation of the dental pulp

The single most important disease process affecting the dental pulp
Accounts for virtually all pulpal disease of any clinical significance

Clinical features
- Pain which is difficult to localize
- May radiate to the adjacent jaw and occasionally to the face, ear or neck
- May be triggered by:
  - Cold or hot stimulants
  - A recumbent position
  - Occasionally by mastication when food particles get into a carious cavity

Import: Important to determine whether pulpitis is reversible or irreversible

Reversible pulpitis:
- The pulp can recover with removal of stimulus
- Pain lasts for only a few moments after removal of the initiating stimulus

Irreversible pulpitis:
- The pulp cannot recover even after removal of stimulus
- Characterized by pain which lingers for at least one minute after removal of stimulus
- May be spontaneous

Complications
- The sequelae of untreated pulpitis (in the order in which they occur) are:
  - Reversible pulpitis
  - Irreversible pulpitis
  - Pulpal necrosis
  - Apical periodontitis
  - Periapical abscess
  - Cellulitis

Investigations
- Of primary importance is the use of a pulp tester to test the vitality of the pulp
- The following can be used:
  - Electric pulp tester
  - Cold or hot water bath
  - Ethyl chloride spray
  - Hot gutta percha sticks
  - Ice sticks

Treatment objectives
- To exclude the pulp from the stimulus (or stimuli) in reversible pulpitis
- To remove the pulp in irreversible pulpitis

Non-drug treatment
- Reversible:
  - Indirect pulp capping
  - Direct pulp capping
  - Conventional filling using amalgam, composite or GIC
  - Desensitization with strontium chloride
  - Root canal therapy
  - Extraction

Drug treatment
- Paracetamol
  - Adult: 500 mg - 1 g orally every 4 - 6 hours (to a maximum of 4 g) for 5 - 7 days
  - Child over 50 kg: same as adult dosing
  - 6 - 12 years: 250 - 500 mg; 1 - 5 years: 125 - 250 mg; 3 months - 1 year: 125 - 250 mg for 5 - 7 days
  - NSAIDs may be required in some patients

Notable adverse drug reactions
- Aspirin is contraindicated in children less than 16 years as it may precipitate Reye's syndrome

Prevention
- Prevent dental caries (the most important cause of pulpitis)
  - Seek prompt dental attention

SALIVARY GLAND DISEASES
Introduction
A wide spectrum of disorders

Diseases due to obstruction
- Salivary calculi
- Parotid papilla and duct strictures
- Salivary fistulae
- Mucoceles and cysts
- Ranula

Sialadenitis
Diseases which result from inflammation of the salivary glands
- Mumps
- Suppurative parotitis
- Chronic sialadenitis

Xerostomia
Dry mouth

It can be caused by the following:
- Sjogren's syndrome
- Irradiation
- Dehydration
- Psychogenic
- Drugs

TEMPORO-MANDIBULAR JOINT DISORDERS
Introduction
These disorders can be grouped under the following conditions:
- Temporo-Mandibular Joint (TMJ) pain-dysfunction syndrome
- Osteoarthritis
- Rheumatoid arthritis

Sjogren's syndrome
- Presents with dryness of the eyes and mouth (primary type)
- In the secondary type, dryness occurs in association with rheumatoid arthritis or other connective tissue disease

Neoplasms of the salivary gland
The next most common neoplasms of the mouth after squamous cell carcinomas
- Above 70% develop in the parotid gland
- Over three-quarters are benign
- Women are slightly more frequently affected

Classification
The modified WHO classification (1972) includes:
- Epithelial tumours
  - Adenomas
  - Pleomorphic adenoma ('mixed tumour')
  - Monomorphic adenomas

- Warthin's tumour, oxyphilic adenoma
- Carcinomas:
  - Mucoepidermoid carcinoma
  - Acinic cell carcinoma
  - Adenocarcinoma
  - Epidermoid carcinoma
  - Undifferentiated carcinoma
- Malignant mixed tumour
- Non-epithelial tumours
  - Lymphomas
  - Sarcomas

Clinical features
- Benign tumours are generally asymptomatic
- May be associated with bleeding, fever, swelling and redness of the mucosa, unpleasant taste in the mouth
- May be spontaneous
- May be triggered by:
  - Cold or hot stimulants
  - A recumbent position
  - Occasionally by mastication when food particles get into a carious cavity

Investigations
- Sialography
- Radiology may reveal marked bone loss interdentally, infra-radically and apically
TMJ pain dysfunction syndrome

- The most common problem in or around the TMJ
  - Clinical features
    - Equal frequency between genders, but five times as many females seek treatment
    - Patients are usually between 15 and 40 years
  - Unilateral or bilateral dull pain within the TMJ and/or surrounding muscles, sometimes on waking or during eating or speech
  - TMJ may lock in the open or closed positions, occasionally
  - TMJ sounds such as clicking, crunching or grating are often described
  - Associated headache is usually located in the temporal region
  - Pain is cyclical and usually resolves, but may recur
  - May be associated with psychological stress
  - Differential diagnoses
    - Migraine
    - Psychologic depression

- Treatment objectives
  - Most symptoms are self-limiting and do not require treatment
  - Treatment should be conservative and reversible

- Non-drug treatment
  - Educate patient about the condition, emphasizing its frequency and self-limiting nature
  - Soft diet
  - Apply moist heat to painful muscles
  - Physiotherapy

- Drug treatment
  - Analgesics as appropriate
  - Anxiolytics
  - Diazepam 5 mg orally 1 hour before sleep, then 2 mg every 12 hours, for up to 10 days (maximum)

- Supportive measures
  - Occlusal splints

- Osteoarthrits
  - Rare
  - Increasing incidence after 50 years
  - Joint crepitus denotes degenerative joint disease
  - May be accompanied by pre-auricular pain, but not involving the masticatory muscles
  - Radiographs (e.g. panoramic, trans-pharyngeal, trans-cranial, oblique, lateral, open and closed) show degenerative joint disease

- Rheumatoid arthritis
  - A disease of unknown aetiology
  - Autoimmune mechanisms and immune complex formation have been implicated

- Usually begins in early adult life and affects females more frequently
  - Patients rarely complain of pain from TMJ but clinical examination shows TMJ involvement in 50% of cases
  - Limitation of mouth opening; softness, crepitus, referred pain, and tenderness on biting
  - Severe disability is unusual

- Trauma
  - Clinical features include:
    - Condylose fracture or trauma arthritis
    - Pain and trismus of traumatic arthritis resolve after one week
  - Micro-trauma from parafunction may result in chronic symptoms
  - Dislocation is usually a result of trauma and is rare; very rarely it occurs after yawning

- Developmental defects
  - Aplasia of the condyle is extremely rare and may be unilateral or bilateral
  - Hypoplasia of the condyle may be congenital or acquired
  - Cause of congenital hypoplasia is not known; either one or both condyles may be involved
  - Acquired hypoplasia may be secondary to trauma, infection or radiation
  - Hyperplasia of the mandibular condyle is rare and self-limiting. Cause is unknown. It is generally unilateral with resultant facial asymmetry, deviation of mandible to the opposite side and malocclusion

- Ankylosis
  - Follows trauma, infection or other inflammatory condition

- Infection
  - Follows penetrating trauma to joint or spread from middle ear

- Neoplasia
  - Primary neoplasms arising from the structures of the TMJ are extremely rare
  - Benign tumours such as chondromas and osteomas are more frequent than sarcomas arising from bone or synovial tissues
  - Others are secondary carcinomas

- CHAPTER 6: DERMATOLOGY

BACTERIAL INFECTIONS

CELLULITIS

- Introduction
  - A suppurative bacterial infection of the skin and soft tissue, often with involvement of underlying structures: fascia, muscles and tendons
  - Most often due to β-haemolytic streptococci or Staphylococcus aureus
  - Usually (but not always) follows some discernible wound
  - Often a complication of immunosuppression like diabetes and HIV/AIDS

- Clinical features
  - Areas of oedema; rapidly spreading
  - Erythema (rapidly becomes intense and spreads)
  - Tenderness and warmth
  - Often accompanied by fever, lymphangitis, regional lymphadenitis

- Systemic signs of toxicity
  - Area becomes infiltrated and pits on pressure
  - Sometimes the central part becomes nodular and surrounded by a vesicle that ruptures and discharges pus and necrotic material

- Differential diagnoses
  - Erysipelas
  - Deep vein thrombosis

- Complications
  - Unusual in immunocompetent adults; children and compromised adults are at higher risk immunocompetent adults; children and compromised adults are at higher risk
  - Septicaemia
  - Gangrene
  - Metastatic abscesses
  - Recurrent cellulitis may predispose to chronic lymphoedema

- Investigations
  - Blood culture
  - Full Blood Count with differentials
  - Fasting blood glucose
  - HIV screening
  - Wound swab for microscopy, culture and sensitivity
  - Urinalysis

- Treatment objectives
  - Eradicate infection
  - Treat underlying immunosuppression
  - Prevent complications

- Drug treatment
  - Ampicillin/cloxacillin
  - Adult: 500 mg - 1 g orally every 6 hours for 5 - 7 days
  - Child under 5 years: a quarter adult dose; 5 - 10 years: half adult dose
  - Or:
    - Cloxacillin
    - Adult: 500 mg orally every 6 hours for 5 - 7 days

- Prevention
  - Treat any wound promptly

- FURUNCULOSIS (Boils)

- Introduction
  - Infection of a hair follicle by staphylococcal organisms, that leads to an inflammatory nodule, with separate heads
  - A carbuncle is merely two or more confluent furuncles, with separate heads
  - Recalcitrant cases may occur with a background of immunosuppression
    - Alcoholism:
    - Malnutrition:
    - Blood dyscrasias:
    - Disorders of neutrophil function:
    - Diabetes:
    - AIDS
  - May occur in patients with atopic dermatitis
  - May be iatrogenic

- Clinical features
  - Can be found on all body sites where hairs are present
  - Starts with a small, yellow creamy pustule that rapidly evolves into a red nodule, often with a central yellow plug
  - As the lesion expands, it becomes:
    - Painful and tense
    - Associated with local oedema, lymphangitis, regional lymphadenopathy and fever
  - Eventually, the central part of the nodule becomes soft
Chapter 6: Dermatology

and drains spontaneously

Healing occurs after about 1 - 2 weeks with scar formation

**Differential diagnoses**

- Folliculitis
- Cutaneous myiasis
- Acne inversa in the axilla or groin

**Complications**

- Cellulitis
- Septicaemia
- Carvenous sinus thrombosis when the lesions are on the head and neck

**Investigations**

- Wound swab for bacteriology and sensitivity
- Full Blood Count with differentials
- Fasting blood glucose
- HIV screening
- Urinalysis

**Treatment objectives**

- Treat infection
- Correct predisposing factors
- Prevent complications

**Drug treatment**

**Topical antibiotics**

- Gentamicin 0.3% cream
- Resistance may set in with prolonged use

**Systemic antibiotics**

- Levofloxacin
- Usually unnecessary except for head and neck lesions, or when the boil is accompanied by fever, chills, regional lymphadenopathy, or a feeling of being unwell

- Co-trimoxazole

**Adult**

- 960 mg orally every 12 hours for 5 - 10 days

**Child**

- 6 weeks - 5 months: 120 mg; 6 months - 5 years: 240 mg; 6 - 12 years: 480 mg taken orally every 12 hours for 5 - 10 days

- Erythromycin

**Adult and child over 8 years**

- 250 - 500 mg orally every 6 hours - 1 g 12 hourly for 5-10 days

**Child**

- Up to 2 years: 125 mg orally every 6 hours; 2 - 8 years: 250 mg every 6 hours for 5 - 10 days

**Surgical treatment**

- A small puncture wound often gives less of a scar than allowing spontaneous rupture; it also reduces the pain

**Should be under antibiotic cover to prevent septicaemia**

**IMPETIGO CONTAGIOSA**

**Introduction**

A superficial, highly contagious, bullous skin disorder caused by coagulase positive staphylococci and occasionally β-haemolytic streptococci

**Clinical features**

- Children are more commonly affected
- Initial lesions are superficial vesicles, or bullae found around orifices: eyes, nose and ears

**Supportive measures**

- Desbride crusted lesions: Dislodging antibacterial agent
- Avoid auto-inoculation e.g. with fingers, shaving brushes, handkerchiefs, or pillow cases

**Standard Treatment Guidelines for Nigeria 2008**

- **Strict personal hygiene**
- **Treat underlying skin disease(s)**

**Notable adverse drug reactions**

- Sulphonamide and co-trimoxazole: fixed drug eruption

**DERMATITIS AND ECZEMA**

**ATOPIC DERMATITIS (Atopic eczema)**

**Introduction**

- Inflammation of the superficial dermis and epidermis, leading to disruption of the skin
- Dermatitis and eczema are used interchangeably

**Clinical features**

- Atopic dermatitis looks different at different ages and in people of different races

**Essential features are:**

- Pruritic, exudative, or lichenified eruptions on face, neck, upper trunk, wrists and hands, and in the antecubital and popliteal folds
- Personal or family history (in about 70% of cases) of allergic manifestations e.g. asthma, hay fever, allergic rhinoconjunctivitis, or eczema

**Complications**

- Chronic unbearable, unrelenting itch
- Erythroderma without infections
- Social setting in which other modalities are impossible

**Smallpox vaccination is absolutely contraindicated**

**Guidelines for the use of potent topical steroids in infants**

- Do not use on the face, axillae, diaper area or flexures
- Do not dispense more than 50 g per week

**Adjuvance measures**

- Exclusive breastfeeding; milk substitute if need be
- Attention to cleanliness especially in the diaper region
- Avoid excessive bathing, vigorous rubbing, or chafing
- Avoid unduly heavy, tight, or soiled clothing
- Treat local infections
- Pat (rather than rub) skin dry after bath and immediately lubricate skin with petroleum jelly or emuslising ointment
- Showers should be warm to cool, not hot
- Tub soaking is good, if followed by adequate lubrication
- Avoid wool; its fibers are irritating
- Emotional stress leads to increased scratching
- In patients and parents of affected children, other psychologiac techniques may be useful
- Secondary skin infection with bacteria such as Staphylococcus aureus may worsen the dermatitis and itching
- Patients must consciously be shielded from anyone with varicella or herpes simplex
- Keep finger nails trimmed short

**Tuberculin test**

- Adults: initially up to 10 - 20 mg orally daily
- Preferably taken as a single dose in the morning after breakfast
- In severe disease: up to 60 mg orally daily, as a short course for 5-10 days

- Ar: Triamcinolone acetonide 40 mg by deep intramuscular injection, into gluteal muscle

**Criteria for systemic steroid therapy**

- Failed maximal therapy; little improvement after environmental changes

**Phases**

- Acute: Several days - 1 week
- Subacute: 1 - 4 weeks
- Chronic: More than 4 weeks

**Acute Phase**

- 50 - 100 mg thrice daily for 3 days
- 25 - 50 mg thrice daily for 3 days

**Subacute Phase**

- 25 - 50 mg daily for 3 days
- 12.5 - 25 mg daily for 3 days

**Chronic Phase**

- 12.5 - 25 mg daily for 3 days
- 6.25 - 12.5 mg daily for 3 days

**Suppress inflammation**

- Reduce itching
- Prevent complications

**Drug treatment**

- Topical:
  - Hydrocortisone 1% or betamethasone valerate 0.1%
  - Apply twice a day until the skin improves then decrease to once a day or less frequently as needed

- Systemic therapy:
  - Steroids (only to control acute exacerbations)
  - Prednisolone
  - Adol: initially up to 10 - 20 mg orally daily
  - Preferably taken as a single dose in the morning after breakfast
  - In severe disease: up to 60 mg orally daily, as a short course for 5-10 days

**Eosinophilia and increased serum IgE levels may be present but are nonspecific**

**Blinded food challenges:** for diagnosing food allergy

**Impetigo Contagiosa**

**Introduction**

A superficial, highly contagious, bullous skin disorder caused by coagulase positive staphylococci and occasionally β-haemolytic streptococci

**Clinical features**

- Debride crusted lesions with soap and water or desloughing antibacterial agents
- Dry weepy lesions with astringent such as potassium permanganate, sodium chloride 0.9% solution, hydrogen peroxide

**Drug treatment**

- Erythromycin

**Adult and child over 8 years**

- 250 - 500 mg orally every 6 hours or 500 mg - 1 g every 12 hours for 5 - 10 days

**Child**

- Up to 2 years: 125 mg orally every 6 hours; 2 - 8 years: 250 mg every 6 hours for 5 - 10 days

**Or:**

- Co-trimoxazole

**Adult**

- 960 mg orally every 12 hours for 5 - 10 days

**Child**

- 6 weeks - 5 months: 120 mg; 6 months - 5 years: 240 mg; 6 - 12 years: 480 mg taken orally every 12 hours for 5 - 10 days

**Wound swab for bacteriology and sensitivity**

**Treatment objectives**

- Treat infection
- Treat underlying pruritic dermatoses
- Prevent complications

**Non-drug treatment**

- Allow spontaneous rupture; it also reduces the pain

**Other**

- Chelation therapy
- Antihistamines

**Tubersculosis**

- Skin test
- Tuberculin test

**Footnote**

- Certain patients may require higher doses

**Chloramphenicol**

- Erythromycin
- Erythromycin

**Ampicillin**

- Erythromycin

**Clindamycin**

- Erythromycin

**Trimethoprim-sulfamethoxazole**

- Erythromycin

**Gentamicin**

- Erythromycin

**Eosinophilia and increased serum IgE levels may be present but are nonspecific**

**Blinded food challenges:** for diagnosing food allergy
Chapter 6: Dermatology

CONTACT DERMATITIS

Introduction
An acute or chronic dermatitis that results from direct skin contact with chemicals or allergens.

These agents could be:
- Chemicals
- Animal or plant products
- Physical agents like heat, cold, ultraviolet rays or ionizing radiation

Contact dermatitis is classified as:
- Irritant dermatitis
- Acute irritant dermatitis
- Cumulative insult dermatitis
- Allergic contact dermatitis
- Phototoxic dermatitis
- Photo-allergic dermatitis

Clinical Features
- Acute phase
  - Tiny vesicles, weepy and crusted lesions
  - Resolving or chronic contact dermatitis
  - Scaling, erythema, and possibly thickened (lichenified) skin
  - Itching, burning, and stinging may be severe
  - Contact dermatitis is recognized by the distribution and configuration of the lesion which usually corresponds to the contactant e.g.
    - Face: cosmetics
    - Photodermatitis: airborne allergens e.g. dust, fumes, sprays
    - Neck: nickel necklace, perfume, and collars of garments

DIFFERENTIAL DIAGNOSES

- Atopic dermatitis
- Seborrhoeic dermatitis
- Psoriasis
- Dermatophyte infection
- Lichen planus
- Face: lupos erythematous, pellagra, rosacea

Complications
- Impetiginization
- Secondary dissemination

Investigations
- Patch test
- Occupational site assessment

Treatment Objectives
- Cure the dermatitis
- Identify cause(s) and avoid further contact

Drug Treatment
- As for atopic dermatitis
- Counselling (after identifying the cause)
- Allergen replacement

EXFOLIATIVE DERMATITIS (Erythroderma)

Introduction
Refers to the involvement of all or most of the skin surface by a scaly erythematous dermatitis

Usually a secondary or reactive process to an underlying cutaneous or systemic disease

Some causes:
- Contact dermatitis
- Atopic eczema
- Seborrhoeic dermatitis
- Drug eruptions
- Lichenplanus and lichenoid eruptions
- Crusted scabies
- Pediculosis corporis
- Pityriasis rosea
- Psoriasis
- Pemphigus foliaceus
- Lymphomas and leukaemia
- Ichthyosis vulgaris
- Pityriasis rubra pilaris

Clinical Features
- May be acute or chronic
- The irritating process is followed by a patchy erythema which spreads rapidly within 24 hours
- Pyrexia, malaise and shivering
- Scaling
- Irritation and tightness
- Skin feels cold
- The peripheral skin is inflamed and oedematous, resulting in eczoptrion, with consequent epiphora

EXFOLIATIVE DERMATITIS (Erythroderma)

Investigation
- Full Blood count and differentials; ESR
- Urea and Electrolytes
- Histopathology
- Blood culture

Treatment Objectives
- Restore the skin to normal
- Treat underlying disease
- Prevent or treat complications

Drug Treatment
- Systemic steroids in high doses
  - Prednisolone 40 - 60 mg orally per day
- Treat impetiginization and septicaemia as appropriate

Adjuvant therapy
- Adequate hydration
- Emollients for skin (see Atopic eczema)
- Keep warm
- Adequate nursing care
- Appropriate nutrition and haematics

Prevention
- Avoid over-treatment of skin diseases and polypharmacy, generally
- Do not abuse the skin with "medicated" soaps and herbal concoctions
- Get appropriate management of skin disease(s) from qualified personnel

PARASITIC DERMATOSES

CUTANEOUS LARVA MIGRANS (Creeping eruption)

Introduction
An infection of the skin by various nematode larvae which migrate, but never reach internal organs or complete their life cycles.

Migration leads to twisting, winding linear skin lesions produced by the burrowing of larvae.

Victims are usually:
- People who go barefoot at the beaches
- Children playing in sandboxes and crawling on the bare ground
- Carpenter and plumbers working under homes
- Gardeners
- The most common causes are cat and dog hookworm
  - Anclylostoma braziliense
  - Anclylostoma caninum
  - Necator americans
- Gnathostoma spinigerum
- Strongyloides stercoralis

Clinical Features
- Shortly after entering the skin:
  - The larvae elicit intense pruritus
  - Tiny papules and even papulovesicles develop
- As the larvae begin to migrate:
  - Intermittent stinging pain occurs
  - Thin red, tortuous and minimally elevated lines are formed in the skin
  - Rate of migration varies with the species
  - Pruritus and excoriation promote secondary bacterial infections
- Intestinal infections with Strongyloides stercoralis may be associated with perianal larva migrans syndrome called 'larva currens' because of the rapidity of larval migration (up to 10 cm/hr)
- Larva currens is an autoinfection caused by penetration of the perianal skin by Strongyloides stercoralis

Diagnosis
- Ring worm

Complications
- Secondary bacterial infection
- Fatal Strongyloides stercoralis hyperinfection in immunocompromised patients

Investigation
- None useful to management

Treatment Objectives
- Eradicate the larvae
- Eradicating gut Strongyloides
- Tremp impetiginization
- Prevent re-infection

Drug treatment
- Ivermectin

Adult: 150 microgram/kg orally as a single dose
Child over 5 years old: 200 micrograms/kg orally daily
Osteomyelitis
Arthritis
Tetanus

Investigations
- Radiograph of the affected area
- Radiograph of the affected area

Treatment objectives
- Resolve local inflammation to permit easier removal of the bone
- Extract the bone
- Prevent and treat complications

Drug treatment
- Metronidazole
  - Adult: 500 mg orally every 8 hours for 7 days
  - Child: 7.5 mg/kg orally every 8 hours
- Mebendazole
  - Adult: 400 - 800 mg orally daily for 6 days
  - Child over 1 year: usually 100 mg orally twice daily for 3 days
- Ivermectin
  - Adult: 200 micrograms/kg orally as a single dose
  - Child: consult specialist companies

Pathophysiology
In the stomach, the larvae penetrate into the mesentery, where they mature sexually in 10 weeks. The female worm then passes through the cutaneous surface to deposit its larva, causing specific skin manifestations. When the parasite comes in contact with water, the larva rapidly discharges its larvae, which are ingested by the cyclops.

Clinical features
- The worm approaches the surface it may feel as a cordlike thickening
- It forms an indurated cutaneous papule
- Several hours before the head appears at the skin surface there is an induration of the skin around the site of the head of the larva
- Local erythema
- Burning sensation
- Pruritus
- Tenderness

Differential diagnoses
- Sickle cell ulcer
- Stasis ulcer
- Cellulitis
- Erysipelas
- Progressive lymphoedema

ONCHOCERCIASIS (River blindness)
Introduction
A common chronic filarial disease in tropical regions which frequently cause pruritus and blindness.

Causes:
days before treatment with ivermectin

**Surgical**

- Excise individual nodules (nodulectomy)

**Notable adverse drug reactions, caution and contraindications**

- No food or alcohol should be taken for at least 2 hours before or after dosage
- Pregnant women should not receive ivermectin until after delivery
- Breastfeeding mothers should not be treated until the infant is at least 1 week old

**Prevention**

- Use biodegradable insecticides to kill flies
- Netting and repellents remain crucial.
- Provide access to safe and portable water

In hyperendemic areas, treat the whole population twice yearly with ivermectin

**PEDICULOSIS (Lice)**

**Introduction**

- Diseases due to blood sucking lice
- Can be divided into three conditions:
  - Pediculosis capitis (head lice):
    - Caused by Pediculus humanus var. capitis
  - Pediculosis corporis (body lice):
    - Caused by *P. humanus var. corporis*
  - Pediculosis pubis (pubic lice):
    - Caused by *P. pubis*

**Clinical features**

- The arthropods are transmitted from human to human via:
  - Direct contact
  - Sharing of combs, brushes, towels (*P. capitis*)
  - Sharing underwear
  - Sexual intercourse or any intimate personal contact (*P. pubis*)

**Oral Pediculosis capitis:**

- Generally the only complaint is pruritus:
- Nits can easily be seen at the base of the hairs; careful inspection may reveal the adult louse
- Secondary impetiginization is common because of the itching
- Cervical nodes may become enlarged

**Pediculosis corporis:**

- Pruritus may be the only symptom in some patients
- Chronic scratching may result in characteristic hemorrhagic puncta and linear excoriations
- Patient eventually develops intensely pruritic papules

**and nodules, numerous excoriations, secondary infections and even lymphadenopathy**

- The combination of excoriations, hyperpigmentation, healed scars and secondary impetiginization is quite typical and known as “vagabond’s skin”
- Overcrowding and poor personal hygiene promote infestation
- Refugees, destitutes and vagrants are particularly vulnerable

**Pediculosis pubis:**

- Most often found in the pubic and axillary hairs
- Occasionally may be found on abdominal or trunk hairs
- On rare occasions may be seen on the scalp, eyebrows and even eyelashes
- Pruritus is also a symptom
- Classic clinical finding is the maculae cerulae
- Indistinct blue-grey or slate-coloured macules ranging in size from several millimeters to several centimeters
- They result from the bite of the louse causing small intracutaneous haemorrhages
- The colour is due to blood whose haemoglobin has been altered by the saliva

**Differential diagnoses**

- *P. capitis:* Seborrhoeic dermatitis, Pityriasis amiantacea, Peripilar keratin, Hair casts, Piedra
- *P. corporis:* Scabies, Atopic dermatitis, All pruritic dermatoses
- *P. pubis:* Scabies, Candidiasis, In the axillae trichomycosis axillaris

**Complications**

- Secondary bacterial infections
- The body louse serves as a vector for diseases:
  - Epidemic typhus (*Rickettsia prowazekii*)
  - Trench fever (*Bartonella quintana*)
  - Relapsing fever (*Borrelia recurrentis*)

**Investigations**

- *P. capitis* and pubis:
  - Examine louse or the nits on epilated hair strands (especially from behind the ears) under the microscope
- *P. corporis*:
  - Examine the seams of clothing for nits and lice

**Treatment objectives**

- Eradicate the lice
- Prevent re-infection
- Treat complications

**Drug treatment**

- *P. capitis*:
  - 1% permethrin cream rinse

## SCABIES

**Introduction**

- An intensely pruritic infestation caused by human mite *Sarcoptes scabiei*

**Complications**

- Conducted by close contact and rarely via fomites
- Occurs commonly in children and inmates of overcrowded institutions such as prisons and boarding houses
- Infection of households is common
- Secondary impetiginization is also another possible method of spread among adults
- Sharing a bed or using the same underwear will also suffice to contact the disease

**Clinical features**

- Severe pruritus worse at night is characteristic
- The typical lesion is the burrow
- It is hardly seen because of the marked excoriation and secondary infection on the skin

**Drug treatment**

- The phallus (especially in adults)
- General immune status and experience with *S. scabiei* play a role
- In a normal host, the initial infection is asymptomatic for about 3 - 6 weeks during which time the individual is capable of transmitting the disease
- All family or living unit members must therefore be treated, not just the itching ones

**CRUSTED SCABIES (Norwegian scabies)**

- An uncommon variant of scabies
- Patient fails to mount a resistance and the mites proliferate dramatically
- May be found among HIV/AIDS patients, institutionalized inmates like prisoners, refugees, and psychiatric patients

**Differential diagnoses**

- Infantile acropustulosis
- Atopic dermatitis
- Papular acral dermatitis of childhood
- Dermatitis herpetiformis

**Complications**

- Secondary bacterial infection leading to acute glomerulonephritis

**Investigations**

- Burrow scraping on a glass slide for microscopy
- Video dermatoscopy

**Treatment objectives**

- Treat the infestation
- Treat secondary bacterial infection
- Relieve pruritus

**Drug treatment**

- Scabicides:
  - Permethrin 5% cream
  - Benzyl benzoate 25% in emulsion

- *Adult:* apply over the whole body and wash off after 8-12 hours
- *Child:* supervision required with application and rinsing

- *Adult:* apply over the whole body; repeat without bathing next day and wash off 24 hours later
- *Child:* apply over all the body daily for 7-10 days

- *Eradicate the lice*

- *Treat secondary bacterial infection*

- *Relieve pruritus*
Systemic corticosteroids

Prednisolone

**Adult:** 20 - 40 mg orally daily for several weeks with reduction of dosage or switch to alternate-day therapy as soon as improvement is seen

**Child:** not recommended for children for this indication

Or:

**Triamcinolone acetonide** 40 mg intramuscularly once or twice (at a 6-week interval)

- If good results not achieved within two weeks increase rapidly to maximum 5 mg/kg daily

Notable adverse drug reactions

See Psoriasis

**Prevention**

Avoid precipitating drugs

PITYRIASIS ROSEA

**Introduction**

A common, mild, inflammatory exanthem

Tends to be seasonal

Chapter 6: Dermatology

- More common during the fall, winter and spring in temperate countries
- In Nigeria more common during the early part of the rainy season (though cases are seen throughout the year)

Clinical features

- LP has been found in children, young and middle-aged adults
- The skin lesions are flat-topped polygonal papules with a characteristic colour
  - Violaceous in fair skinned people but slate-grey on black skin
- Itching is mild-to-severe
- The lesions are distributed mainly on:
  - Flexor surfaces of the wrist
  - Lumbar area
  - The penis, tongue, buccal and vaginal mucous membranes

Drug treatment

**Topical corticosteroids:**

- Betamethasone dipropionate 0.1% cream
  - Apply 1 - 2 times daily
- Triamcinolone acetonide 0.1% cream and ointment
  - Apply 1 - 2 times daily

- For isolated or hyperkeratotic lesions apply corticosteroids under occlusion or use intralesional triamcinolone (see Psoriasis)

Scalp lesions:

- Forseps: Clobetasol propionate 0.05% lotion
  - Apply thinly 1 - 2 times daily for up to 4 weeks

Mouth lesions:

- Triamcinolone acetonide 0.1% in adhesive base
  - Apply a thin layer 2 - 4 times daily for a maximum of 5 days; do not rub in
- Tretinoin 0.025% cream

**Adult and child:** apply thinly 1 - 2 times daily

Complications

- Large number of adolescents and in young adults, but it has been described all age groups
- Rarely, there is an observable prodrome of pharyngitis, malaise and mild headache
- Complications may appear as a delayed reaction to a viral infection (most likely Human Herpes Virus 7)

Clinical features

- Diverse and numerous: hyperkeratosis, papules, plaques, purpuric, follicular, lichenoid, and psoriasiform
- A variant, inverse pityriasis rosea also occurs
  - Believed to be commoner in blacks
  - Affects the face, neck, distal extremities and the flexures
- Use of ampicillin early in the course of the eruption causes an explosive exacerbation of eruptions which become more inflammatory and urticarial
- Lesions may become impetiginized
- The disease persists for about 6 weeks but may last for 3 - 4 months
- Healing may occur with postinflammatory hyper/hypopigmentation
- Recurrences are uncommon (about 1%) but the lesions are usually mild and localized

**Differential diagnoses**

- Secondary syphilis
- Exanthematous or pityriasis rosea-like drug eruptions
- Lichen planus
- Parapsoriasis
- Pityriasis lichenoides chronica

**Complications**

- None

Investigations

- Non-specific
- VDRL
- If secondary syphilis is suspected (e.g. lesions on palms and soles with/without lymphadenopathy)

**Treatment objectives**

- To relieve symptoms (if any)
- Reassure patients about the harmless, self-limiting nature of the eruption

**Drug treatment**

- **Topical:**
  - Urea cream
  - Useful as a hydrating agent: apply twice daily
- **Systemic:**
  - Oral antihistamine
  - If pruritus is bothersome (see Urticaria)

**Systemic corticosteroids:**

- If complicated by ampicillin exanthematous eruption
  - Triamcinolone acetonide 40 mg intramuscularly as a single dose
- **Antibiotics:**
  - If lesions are impetiginized
  - Erythromycin 500 mg orally every 6 hours for 14 days

Notable adverse drug reactions

**Psoriasis**

**Introduction**
A chronic inflammatory skin disease which is characterized by:
- Increased epidermal proliferation
- Epidermal thickening
- Erythematous lesions with silvery white scales
- Affects people of all ages in all countries
- The disease runs a chronic and highly variable course (waxes and wanes)
- New lesions may replace older, regressing ones
- Unstable lesions may evolve into psoriatic erythroderma or generalized pustular psoriasis
- HIV/AIDS can lead to the onset or worsening of psoriasis

**Differential diagnoses**
- Guttate psoriasis:
- Pityriasis lichenoides et varioliformis acuta
- Pityriasis rosea
- Secondary syphilis (psoriasiform syphilis)
- Scalp, face, chest lesions:
- Seborrhoeic dermatitis
- Lupus erythematosus
- Chronic truncal psoriasis:
- Nummular dermatitis
- Lichen planus
- Small plaque parapsoriasis
- Tinea corporis
- Pityriasis rubra pilaris
- Intertriginous areas:
- Candidiasis
- Interttrigo
- Hailey-Hailey disease
- Nail:
- Tinea unguium
- Lichen planus
- Trachyonychia

**Complications**
- Erythroderma
- Arthritis mutilans

**Investigations**
- Histopathology

**Treatment objectives**
- To retard epidermal proliferation
- Reduce inflammation
- Prevent complications

**Drug treatment**
- Choice of treatment depends on the site, severity and duration of the disease, previous treatment, and the age of the patient
- Topical treatment:
  - Corticosteroid ointment
  - Hydrocortisone for the face and flexures
  - Betamethasone or clobetasol for the scalp, hands and feet
  - Application is followed by an occlusive dressing of a polyethylene film, which may remain in place for 12 - 24 hours to augment effectiveness
  - Dithranol ointment 0.1% - 2% (for moderately severe psoriasis)
  - Initiate under medical supervision
  - Start with 0.1%; carefully apply to lesions only, leave in contact for 30 minutes, then wash off thoroughly
  - Repeat application daily, gradually increasing strength to 2% and contact time to 60 minutes at weekly intervals
  - Wash hands thoroughly after use
  - Avoid contact with eyes and healthy skin
  - Coal tar solution (for chronic psoriasis)
    - Use either alone or in combination with exposure to ultraviolet light
    - Apply 1 - 4 times daily, preferably starting with a lower strength preparation
  - Coal tar bath
    - Use 100 mL in bath of tepid water and soak for 10 - 20 minutes
    - Use once daily, to once every 3 days for at least 10 - 20 minutes, and for at least 10 baths
  - Often alternated with ultraviolet (UVB) rays, allowing at least 24 hours between exposure and treatment with coal tar
  - Urea 10% cream or ointment (for dry scaling and itching skin)
    - Apply twice daily, preferably to damp skin
    - Apply once daily, preferably to damp skin
  - Fluocinolone acetonide 0.01% in oil
    - Suitable for childhood psoriasis
    - Combination therapy with calcipotriol and high-potency (Class I) steroids may provide:
      - Enhanced rates of clearance of plaque psoriasis,
      - Decreased rates of adverse effects, and steroid-sparing, allowing a shift to a less potent topical steroid or less frequent use of a Class I steroid
  - Salicylic acid 3 - 5% in cold cream or hydrophilic ointment (for thick scaling)
    - To be administered under expert supervision in both adults and children

**Standard Treatment Guidelines for Nigeria 2008**
- Fluocinolone acetonide 0.01% in oil
  - Apply and leave under a shower cap at night and shampoo in the morning
  - After shampooing and while the hair is still wet, massage thoroughly into the scalp skin
  - Attempting to remove scales by excessive brushing, scrubbing, or combing may result in sufficient trauma to worsen psoriasis (Koebner’s effect)
- Ultraviolet light (UVL)
  - For psoriasis involving more than 30% of the body surface
    - 290 - 320 nm ultraviolet B (UVB) three times weekly for 18 - 24 treatments
  - Lubricating the skin surface with mineral oil or petroleum jelly before UVL produces uniform penetration by reducing the reflection of light from the disrupted skin surface
- PUVA (psoralen plus ultraviolet A)
  - For patients who have not responded to standard UVB treatment
- Severe psoriasis unresponsive to outpatient UVL, may be treated in a day care centre with the Goeckerman regimen
- Systemic therapy:
  - Antibiotics to eliminate streptococcal pharyngitis
    - Adult: Initially 25 - 30 mg orally daily for 2 - 4 weeks; adjusted according to response. Usual range 25 - 50 mg daily (maximum 75 mg)
  - For psoriler, erythrodermic and plaque types, and psoriatic arthritis
    - Child: severe extensive psoriasis resistant to other forms of therapy, palmo-planar pustular psoriasis
      - 1 month - 12 years: 500 micrograms/kg orally once daily with food or milk; occasionally up to 1 mg/kg/day
      - To be administered under expert supervision in both adults and children
    - Methotrexate
      - Adult: 20 mg orally once weekly
      - Child: not licensed for this indication

**Investigations**
- Corticosteroid injections of triamcinolone are frequently used
  - Triamcinolone acetonide suspension 10 mg/mL may be diluted with sterile saline to make a concentration of 2.5 - 5 mg/mL
  - For nail lesions inject triamcinolone in the region of the matrix and the lateral nail fold
  - Scalp:
    - Soften scales with salicylic acid 3% in mineral/olive oil, massage in and leave on overnight
    - Then shampoo with a tar shampoo, and remove scales mechanically with a comb and brush
    - Scalp repeat daily until the scales are gone
    - If 3% is not very effective, use 6% salicylic acid
  - Or:
  - Methotrexate
  - Tinea corporis
  - Candidiasis
  - Intertrigo
  - Hailey-Hailey disease
  - Trachyonychia
  - Pityriasis rosea
  - Secondary syphilis (psoriasiform syphilis)
  - Nummular dermatitis
  - Lichen planus
  - Small plaque parapsoriasis
  - Tinea corporis
  - Pityriasis rubra pilaris
  - Intertriginous areas:
    - Candidiasis
    - Interttrigo
    - Hailey-Hailey disease
  - Nail:
    - Tinea unguium
    - Lichen planus
    - Trachyonychia
  - When scratched, scales fall off as tiny flakes that resemble scrapings from a candle (Candle sign)
  - If the scales are removed (exposing the dermal papillae) punctate bleeding from the enlarged capillaries occur (Auszpit sign)
  - Eruptive lesions may be intensely or mildly pruritic, or may be asymptomatic
  - All lesions begin as small scaly macules but may take divergent paths as they spread centrifugally

**Patterns seen may be:**
- Guttate
- Follicular
- Numular
- Geographic
- Erythodermic
- Annular
- Gyrate or serpiginous

**Favoured sites are:**
- Knees and elbows
- Scalp
- Palms and soles
- Nails
phototherapy or acitretin) should be instituted as the cyclosporine dose is reduced
- TNF inhibitors (Etaluzimab)
  - Indicated for moderate-to-severe chronic plaque psoriasis unresponsive to, or intolerant of other systemic therapy or photochemotherapy
  - Initially 700 micrograms/kg by subcutaneous injection then 1 mg/kg weekly
  - Discontinue if inadequate response after 12 weeks
  - Not recommended for children and adolescents

**Adjutant therapy**
- Diet: fish oils rich in Q-3 polyunsaturated fatty acids
- Patient education
- Emotional support

**Notable adverse drug reactions, caution and contraindications**
- Coal tar:
  - Contraindicated in inflamed, broken or infected skin
  - May cause irritation, photosensitivity reactions
- Hypersensitivity
- Skin, hair, fabrics and bathtubs discoloured brown and smell
- Dithranol:
  - Irritant: avoid contact with eyes and healthy skin
  - Contraindicated in hypersensitivity; avoid use on face, acute eruptions, and excessively inflamed areas
- Very expensive
- Dihydroxychloroquine: if excessive erythema occurs or lesions spread
- Conjunctivitis following contact with eyes
- Staining of skin, hair, and fabrics brown
- Vitamin D3 (calcipotriol): May irritate the skin (stinging)
- Very expensive
- Urea:
  - Avoid application to face or broken skin; avoid contact with eyes
  - May cause transient stinging and local irritation
- Steroids:
  - When extensive areas are treated or when there is erythrodeme psoriasis, sufficient may be absorbed to cause adrenal suppression
  - May induce tachyphylaxis
  - Rebond often occurs after stopping treatment, resulting in a more unstable form of psoriasis
- Intralosional injection may cause reversible atrophy at the injection site
- Salicylic acid:
  - Widespread application may lead to salicylate toxicity
  - Ultraviolet light:
    - Burning of skin may cause Koebner's phenomenon and an exacerbation
    - Increased risk of skin cancer particularly in persons with fair complexion and albinos. Examine one or more protected areas.
    - Use protective glasses to prevent cataracts
    - Causes premature ageing of the skin

**Should be administered only by experienced dermatologists**
- Methotrexate:
  - May cause blood disorders (bone marrow suppression), liver damage, pulmonary toxicity, GIT disturbances
  - If stomatitis and diarrhoea occur, stop treatment
  - Renal failure, skin reactions, alopecia, osteoporosis, arthralgia, myalgia, ocular irritation, may also occur
  - May precipitate diabetes
  - Monitor before and throughout treatment: blood counts and hepatic and renal function tests
  - Contraception during and for at least 6 months after treatment for both males and females
  - Contraindicated in pregnancy and breast feeding.
- Folic acid may be given to reduce toxicity
- Cyclosporine:
  - Nephrotoxic: monitor kidney function
  - Other side effects: hypertrichosis, hyperuricaemia, thrombocytopenia, malagnancies and lymphoproliferative disorders
  - (similar to other immunosuppressive therapies)
- Acetazolamide: See Acne- isotretinoin
- Tacrolimus:
  - See Adjuvant therapy
  - Contraindicated in pregnancy and breast feeding.
- Folic acid may be given to reduce toxicity
- Cyclosporine:
  - Nephrotoxic: monitor kidney function
  - Other side effects: hypertrichosis, hyperuricaemia, thrombocytopenia, hepatic and renal impairment.
  - Monitor platelet count during initial therapy, then every 3 months
- Contraindicated in immunodeficiency, severe infection, active tuberculosis; history of malignancy; pregnancy and breastfeeding
- May cause influenza-like symptoms, leucocytosis, arthralgia, paradoxical exacerbation of psoriasis or development of variant forms including psoriatic arthritis (discontinue treatment)

**Expensive**
- Prevention:
  - Avoid exacerbating factors e.g. abrasions, scratches, harsh fibre bathing sponges, and the drugs listed above
  - Prevent streptococcal sore throat and treat promptly when it occurs

**SUPERFICIAL FUNGAL INFECTIONS**

**DERMATOPHYTE INFECTIONS (Tinea)**

**Introduction**
- Superficial fungal infection that affects keratinized tissues
- Fungi that usually cause only superficial infections on the skin are called dermatophyte- classified in three periodically
  - Microsporum, Trichophyton and Epidermophyton
  - Can be acquired from humans, animals, soil or vegetable matter

**Common in tropical climate (which is hot and humid)**
- Infection could be spread by fomites
- May cause influenza-like symptoms, leucocytosis, arthralgia, paradoxical exacerbation of psoriasis or development of variant forms including psoriatic arthritis (discontinue treatment)

**Treatment objectives**
- To clear lesions and prevent recurrence

**Drug treatment**

**Topical**
- Ketocanazole
  - 2% cream apply twice daily
- Miconazole
  - 2% cream apply twice daily

**Systemic**
- Fluconazole
  - Adult: 50 mg orally daily for 2-4 weeks; up to 6 weeks in tinea pedis
  - Child: 1 month - 18 years 3 mg/kg (maximum 50 mg) daily for 2-4 weeks; up to 6 weeks in tinea pedis

**Notable adverse drug reactions**
- Fluconazole: numerous drug interactions
- Hepatotoxicity during long-term daily therapy
- Prevention:
  - Do not share combs, hair brushes, school caps, shoes, socks or underwear
  - Keep the feet dry; avoid tight-fitting covered shoes
  - Aerate the feet as often as possible
  - Use good antiseptic powder on the feet after bathing e.g. Antifungal powder

**PITYRIASIS VERSICOLOR (Tinea versicolor)**

**Introduction**
- Superficial yeast infection of the skin caused by Malassezia furfur species (normal commensals on the skin)
- Common in warm humid climates
- Predisposing factors:
  - Occlusion of the skin with pomades and greases

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**VARICELLA (Chickenpox)**

**Introduction**
Varicella Zoster virus is Human Herpes Virus 3.

Transmission is by direct contact with the lesions and by the respiratory route.

Initial replication occurs in the nasopharynx and conjunctiva.

After the primary infection, the virus remains dormant in nervous tissue.

- Reactivation later in life is typically manifested as Herpes zoster.

**Clinical features**
- Incubation period is 10 - 21 days.
- Vesicular eruptions consist of delicate “teardrop” vesicles on an erythematous base.

The eruption starts with faint macules that develop rapidly into vesicles within 24 hours.

- Successive fresh crops of vesicles appear for a few days, mainly on the trunk, face, and oral mucosa.

- New lesions usually stop appearing by the fifth day; the majority is crusted by the sixth day.

- Most disappear in less than 20 days without a scar, except larger and secondarily infected lesions.
- Low grade fever.
- Malaise.
- Headaches.
- The severity of the disease is age-dependent.

- Adults have more severe disease and a greater risk of visceral disease.

**Differential diagnoses**
- Variola minor.
- Disseminated zoster in immunosuppressed patients.
- Widespread papular urticaria.
- Coxackie and ECHO viruses eruption.

**Complications**
- Secondary bacterial infection.
- Pneumonia.
- Cerebellar ataxia and encephalitis.
- Reye’s syndrome.

**Investigations**
- Tzanck smear.
- Direct fluorescent antibody (DFA) staining.
- Polymerase Chain Reaction (PCR).

**Treatment objectives**
- Relieve itching and treat secondary bacterial infection.
- Reduce severity and scarring.

**Drug treatment**
- Aciclovir.
- Adult: 10 mg/kg intravenously three times daily for 7 days in immunocompromised patients.
- Child: see Herpes zoster.
- Antihistamine for pruritus.
- Co-trimoxazole or erythromycin for secondary infection.

**Notable adverse drug reactions, caution**
Aciclovir.

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**Chapter 6: Dermatology**
**Plane warts**  
More common in children and young adults.  
Usually appear in groups as smooth, yellow-brown, small, flat papules; most frequently on the face.

**Genital warts**  
Occur most often on warm, moist surfaces of the body  
In men, usual sites are the end and shaft of the penis, and below the foreskin (if uncircumcised)  
- In women, lesions occur on the vulva, vaginal wall, cervix, and skin surrounding the vaginal area  
- May develop in the perianal region or rectum  
- Especially in homosexual men, and in women who engage in anal sex  
- Usually appear 1 - 6 months after infection as soft erythematous papules, which may be greyish if hyperkeratotic  
New lesions develop rapidly and all coalesce, producing a cauliflower-like picture

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**VIRAL WARTS (Verrucae)**

**Introduction**
Infections caused by human papilloma viruses (HPV); include more than 80 types  
- Transferred between humans, or from animals to humans  
- Cause cutaneous tumours which tend to regress spontaneously but may rarely progress into cutaneous malignancies

**Clinical features**
Infection may be clinical, subclinical, or latent  
Clinical lesions are visible by gross inspection  
Subclinical lesions may be seen only by aided examination (e.g. the use of acetic acid soaking)
- Latent infection:  
  - HPV virus or viral genome is present in apparently normal skin  
  - Thought to be common, especially in genital warts, and explains in part the failure of destructive methods to eradicate warts
- Incubation period is highly variable; from weeks to years
  - Auto-inoculation is the rule  
  - Lesions may also occur on scratches (Koebner phenomenon)

Lesions are classified according to their positions and shape:

**Common warts**
- Warts growing with rough surface; round or irregular, greyish or brown  
  Generally appear on areas that are frequently injured, such as the fingers, around the nails (periangual warts); knees, face and scalp
  - Plantar warts  
    Develop on the soles of the feet, where they are usually flattened by the pressure of walking  
    - A reactive callus forms around lesions  
      Multiple warts may coalesce, resembling a tile or mosaic floor (mosaic warts)

May be extremely tender  
Unlike corns and calluses, plantar warts tend to bleed from tiny spots, like pinpoints when pared down with a blade

**Filiform warts**
- Long, thin, small growths that usually crop up on the eyelids, face, neck, or lips  
  People who chronically use corticosteroids as cosmetic bleaching creams are prone to multiple filiform warts

**Complications**
Squamous cell carcinoma of the perianal skin  
Cervical carcinoma from anogenital warts  
Obstructive laryngeal papillomatosis in babies infected through maternal birth canal

**Investigations**
Histopathology if in doubt

**Management**
Treating depends on their location, type, and severity, as well as duration of lesions

**Treatment objectives**
Eradicate the skin lesions  
Prevent complications

**Non-drug treatment**
Liquid nitrogen freeze  
Electro-desication  
Laser surgery

**Drug treatment**
Salicylic acid with lactic acid plaster  
- Apply carefully to wart; rub wart surface gently with file or pumice stone once weekly  
- May need to treat for as long as 3 months
  - Podophyllum resin  
    - Apply weekly under supervision e.g. in genitourinary clinic  
    - Imiquimod 5% cream  
      - Apply thinly once daily on 3 alternate days per week until lesions resolve (maximum 16 weeks)

**Notable adverse drug reactions, caution and contraindications**
Salicylic acid plaster  
- Avoid broken skin  
- Not suitable for anogenital region or large areas
  - Podophyllum  
    - Avoid normal skin and open wounds  
    - Keep away from face  
    - Should not stay on treated skin for more than 6 hours before washing

**Prevention**
Women with genital HPV infection should have routine cervical cytologic screening  
- Pappanicolaou (PAP) smear to detect cervical dysplasia

**MISCELLANEOUS DISORDERS**

**ACNE VULGARIS (Pimples)**

**Introduction**
One of the most common skin diseases  
A disorder of the pilosebaceous follicles  
Typically first appears during puberty when androgenic stimulation triggers excessive production of sebum  
Many factors interact to produce acne in a given patient  
- Genetics  
- Sebum production  
- Hormones  
- Bacteria  
- Properties of the sebaceous follicle  
- Immunologic  
- Over-production of stratum corneum cells (hyperkeratosis) obstructs the hair follicles at the follicular mouth producing open comedones, or blackheads  
- Just beneath the follicular opening in the neck of the sebaceous follicle it causes microcomedones (closed comedones, or whiteheads)

There is an overgrowth of gram-positive bacteria in the obstructed follicle; Propionibacterium acnes or Staphylococcus epidermidis; distally Pityrosporum ovale

Rupture of the comedonal contents into the dermis induces a foreign body reaction and inflammation

**Clinical features**
- Almost every individual has some degree of acne during puberty, with spontaneous resolution occurring in early adult life
- Occasionally, the disease persists into the fourth decade, or even remains a life-long problem
- Favoured sites are the face, upper back and upper chest and shoulders
- There may be mild soreness, pain, or itching
- May present differently in different age groups
- Pre-teens often present with comedones as their first lesions
- Teenage acne is invariably inflammatory and the lesions include firm red papules, pustules, abscesses, indurated nodules, cysts and rarely interconnecting draining sinus tracts
- Inflammatory acne can be classified as mild, moderate, or severe
- Mild acne:  
  - Few-to-several inflammatory papules and pustules, but no nodules  
  - Moderate acne:  
    - Several-to-many papules, pustules, and a few to several nodules
  - Severe acne (acne conglobata):  
    - Numerous fistulated comedones; extensive inflammatory papules; pustules; many cysts, abscesses, nodules, and draining sinuses
    - The lesions may be generalized, involving even the buttocks
    - Excoration of acne papules and microcomedones are common, and scarring may result
    - Usually, multiple shallow erosions or crusts are found

**Differential diagnoses**
Acne rosacea  
Dermatosis papulosa nigra  
Steatocystoma multiplex  
Syringoma  
Trichoepithelioma  
Warts  
Angiofibromas of tuberous sclerosis  
Condyloma lata  
Tuberculosis verrucosa cutis  
Palmoplantar keratoderma  
Epidermodysplasia verruciformis  
Squamous cell carcinoma  
Seborrhoeic keratosis  
Hypertrophic lichen planus  
Pemphigus vegetans  
Squamous cell carcinoma of the perianal skin  
Cervical carcinoma from anogenital warts  
Obstructive laryngeal papillomatosis in babies infected through maternal birth canal

**Complications**
Psychosocial problems from cosmetic disfigurement  
Post-inflammatory pigmented changes  
Pitted scars  
Keloïds
Acne fulminans (acute febrile ulcerative acne congolabata with polyarthritus and leukemoid reaction)

**Investigations**
- Usually, none required
- In the presence of unusual acne, hirsutism, premature pubarche, or androgenic alopecia (especially when associated with obesity and/or menstrual irregularities):
  - Screen for hyperandrogenism
  - Blood levels of free testosterone, dehydroepiandrosterone, and androstenedione
- If raised, test response of the hormones and cortisol to dexamethasone suppression

**Treatment objectives**
- Reduce severity of acne
- Prevent complications

**Drug treatment**
- **Comedonal acne**
  - Topical treatment only:
    - Tretinoin cream
      - Adult: 0.025% or 0.05% or 0.1% cream or gel applied nightly
      - Child: apply thinly 1 - 2 times daily
      - Or:
        - Benzoyl peroxide
          - Adult: 2.5% or 5% water-based or alcohol-based gels, applied twice daily
          - Child: apply up to 3 times daily
          - Tretinoin may be used at night and benzoyl peroxide or topical antibiotics in the morning because they have different modes of action and are complementary
      - Azelaic acid 20% cream
        - Adult and child: apply twice daily; initially once daily for sensitive skin
      - Salicylic acid solution 2%
        - Adult and child: apply up to 3 times daily
      - Tretinoin may be used at night and benzoyl peroxide or topical antibiotics in the morning because they have different modes of action and are complementary
      - It may take 8 - 12 weeks before observable improvement occurs
    - Clindamycin or erythromycin gel or solution twice daily
    - Adjuvant measures
      - Non-irritating cleansing agents to reduce facial sheen and bacterial flora
      - Emotional support

**Contraindications**
- Oil-based cosmetics, hair styling mousse, face creams and hair sprays
- Medicines that may induce acne

**PRURITUS**
- Commonly known as itching
- The most common unpleasant experience involving the skin; provokes a desire to scratch
- May be elicited by many normally occurring stimuli e.g.
  - Light touch
  - Temperature change
  - Emotional stress
  - Chemical, mechanical, thermal and electrical stimuli
- Mediated by the release of chemical substances e.g.
  - Histamine, kinins, and proteases
  - Prostaglandin E lowers the threshold for histamine-induced pruritus, while enkephalins, pentapeptides which bind to opiates receptors in the brain modulate pain and itching centrally

**Clinical features**
- At a low level, may merely be annoying
  - May actually torture the patient, interfere with sleep and lead to less than optimal performance
  - There are great variations from person to person
  - In the same person there may be variation in reactions to the same stimuli
  - In the elderly, senile pruritus due to dry skin may be particularly bothersome
  - Psychologic trauma, stress, absence of distractions, anxiety, and fear may all enhance itching
- Tends to be most severe at the time of undressing for bed
  - There are also regional variations
    - The ear canals, eyelids, nostrils, and perianal and genital areas are especially susceptible to pruritus
    - May be localized or generalized
  - May or may not be associated with skin lesions
  - Excoriations are typically linear and occur where the patient can reach with his hands
    - The middle of the back is typically spared except when the patient has used a back scratcher and the scratch is usually erythematous, with many tiny erosions scattered along it
    - Fresh marks are usually weepy or bloody; older ones

**Chapter 6: Dermatology**

**Adult and child over 12 years:** Antibiotic-resistant acne
- **Minocycline**
  - Adult and child over 12 years: 50 - 100 mg orally every 12 hours
  - Or:
    - Erythromycin
      - Adult and child over 12 years: 500 mg - 1 g every 12 hours
      - Or:
        - Clarithromycin 250 - 500 mg orally every 12 hours
          - In patients who do not tolerate any of the tetracyclines or who fail to improve
        - If there is marked improvement, taper the dose by 250 mg for tetracycline every 6 - 8 weeks while treating with topicals to arrive at the lowest systemic dose needed to maintain clearing

**Antibiotic-resistant acne**
- Oral contraceptives containing a non-androgenic progestin
  - Co-cyprindiol:
    - A mixture of cyproterone acetate and ethinylestradiol
      - 2000 parts to 35 parts
      - 1 tablet orally daily for 21 days starting on day 1 of menstrual cycle and repeated after a 7-day interval, usually for several months
      - For women with severe acne refractory to prolonged antibiotic therapy
    - Spironolactone may be added as an antiandrogen
      - Adult: 50 - 200 mg orally daily

**Moderate inflammatory acne**
- **Tetracycline**
  - Adult and child over 12 years: 500 mg orally every 12 hours
  - Or:
    - Clindamycin or erythromycin gel or solution twice daily
    - Adjuvant measures
      - Non-irritating cleansing agents to reduce facial sheen and bacterial flora

**Severe acne**
- Start with systemic antibiotics as above
  - Oral isotretinoin (13-cis retinoic acid)
    - Adult: 0.5 - 1 mg/kg/day for 20 weeks for a cumulative dose of at least 120 mg/kg
  - Child 12 - 18 years: 500 micrograms/kg once daily, increased if necessary to 1 mg/kg in 1 - 2 divided doses
    - Occasionally, acne does not respond or promptly recurs after therapy, but may clear after a second course
    - At least a 4-month rest period from the drug is recommended before a second treatment course is considered

**Adjuvant measures**
- Use non-irritating cleansing agents to reduce facial sheen and bacterial flora
- Emotional support

**Therapy**
- Check cholesterol and triglyceride levels every 2 - 4 weeks while on therapy
- Dapsone at such high doses is likely to cause methaemoglobinemia
- Where leprosy is still endemic (e.g. Nigeria), reserve treatment for leprosy

**Prevention**
- Avoid
  - Oil-based cosmetics, hair styling mousse, face creams and hair sprays
  - Medicines that may induce acne
**Crusted**
- Lesions may become impetiginized
- In addition to excoriations, some patients may have smooth, shiny fingernails (the polished nails of chronic pruritus)
- Biliary obstruction
- Diabetes mellitus
- Uraemia
- Lymphoma
- Hyperthyroidism
- Adverse reaction to medicines e.g. Histamine liberators, opioids
- Occult scabies
- Pediculosis
- Onchodermatitis
- Dermatitis herpetiformis
- Atopic eczema in remission
- HIV/AIDS
- Systemic mastocytosis

**Polycythaemia vera** is a notable cause of pruritus; usually induced by temperature changes
Some patients complain of pruritus provoked by bath or immediately post-bath

Factors include:
- Aquagenic pruritus
- Temperature-dependent pruritus due to cold/heat
- Cholinergic pruritus (when the core temperature is increased and there is sweating)
- Allergy to bath sponge or soap
- Mechanical scrubbing of the skin with coarse sponge causing degranulation of mast cells
- A forceful jet of water from the shower may trigger pruritus in some cases.

**Differential diagnoses**
All the above causes of pruritus

**Complications**
- Sleep disturbance
- Less than optimal performance at home, work or school
- Emotional disturbance
- Suicidal ideation

**Investigations**
As suggested by meticulous history and physical examination

**Treatment objectives**
- Identify and treat cause(s)
- Improve quality of life
- Prevent complications

**Drug treatment**
- Hydroxyzine hydrochloride
  - Initially 25 mg at night, increased if necessary to 25 mg - 3 times daily
  - Child: 6 months - 6 years: initially 5 - 15 mg daily,
  - increased if necessary to 50 mg daily in divided doses
- Over 6 years: initially 15 - 25 mg daily, increased if necessary to 50 - 100 mg daily in divided doses

**Aquagenic pruritus, mastocytosis, and pruritus of neurofibromatosis**
- Sodium cromoglycate
  - Adult: 200 mg orally taken before bath and immediately after meals
  - Dose may be increased after 2 - 3 weeks to a maximum of 40 mg/kg daily, reduced according to response
  - Or: Ketotifen
  - Adult: 2 mg orally taken before bath (with food)

**Colestyramine**
- 4-8 g orally daily in water (or other suitable liquid)
- Adult: 1 g orally once daily mixed with water; 1 - 6 years: 2 g once daily; 6 - 12 years: 4 g once daily; 12 - 18 years: 4 - 8 g daily, adjusted according to response in all age groups

**Urticaria and Angioedema**

**Introduction**
- An eruption of evanescent wheals or hives which can result from many different stimuli on an immunologic or non-immunologic basis

The most common immunologic mechanism is hypersensitivity mediated by IgE
- Another mechanism involves activation of the complement cascade.

**Child: not recommended because of associated burning**
- Oral: 10% hydrocortisone cream 1%
- Adult and child: dilute with aqueous cream in first 1 week of use if sting occurs
- Or: Emulsifying ointment BP
- Adult and child: can be used as soap substitute; rub on skin before rinsing off completely
- Or: Dexamethasone hydrochloride
  - Adult: apply thinly 3 - 4 times daily (coverage should be less than 10% body surface area)

**Adverse drug reactions, caution and contraindications**

**Ketotifen**
- Adult: 1 mg orally twice daily

**Sodium cromoglycate**
- Occasional nausea, rashes, and joint pain

**Ketotifen**
- Standard Treatment Guidelines for Nigeria 2008

**Clinical features**
- May be acute or chronic:
  - Acute urticaria is of sudden onset and lasts less than 6 weeks
  - Chronic urticaria persists for more than 6 weeks with either:
    - Daily emergence of new wheals (chronic continuous) or
    - Occasional hive-free periods (chronic recurrent)
  - The typical urticarial reaction is similar to the triple response of Lewis
    - Initial erythema
    - Next oedema (the hive)
    - Finally an erythematous ring surrounding the hive

**URTICARIA AND ANGIOEDEMA**

**Prevention**
- Use a cleansing bar (instead of soap) for baths
- Pat rather than rub skin dry after bath and immediately lubricate skin with petroleum jelly or emulsifying ointment

**Diagnosis**

**Chapter 6: Dermatology**
Angioedema is the involvement of deeper vessels
- Characterized by painless, deep, subcutaneous swelling
- Often involves periorbital, circumoral and facial regions; palms, soles and the genitalia
- May target the gastrointestinal and respiratory tracts, causing abdominal pain, coronya, asthma and respiratory problems
- Respiratory tract involvement may cause airway obstruction
- Anaphylaxis and hypotension may also occur

**Differential diagnoses**
- Cryate erythema
- Urticarial vasculitis
- Mastocytosis
- Pityriasis rosea (early lesions)

Bullous lesions:
- Pemphigus
- Pemphigoid
- Erythema multiforme

Fixed drug eruption
- Angioedema: “Calabar swelling”

Cellulitis
- Idiopathic scrotal oedema of children
- Melkerson-Rosenthal syndrome

Cold urticaria:
- Cryoglobulinemia
- Immune complex diseases
- Systemic lupus erythematosus and other collagen vascular diseases
- Macroglobulinemia
- Mycoplasma infections (cold hemagglutinins)
- Syphilis
- Familial cold urticaria
- Acquired cold urticaria

**Complications**
- Emotional distress in chronic cases

**Investigations**
- Suggested by meticulous history and physical examination

**Treatment objectives**
- To alleviate symptoms
- Eliminate and treat cause

**Drug treatment**

**Chlorphenamine maleate**
- Adult: 4 mg orally every 4 - 6 hours (maximum 24 mg daily)
- Child: under 1 year, not recommended
- 1 - 2 years: 1 mg every 12 hours 2 - 5 years: 1 mg every 4 - 6 hours (maximum 6 mg daily); 6 - 12 years: 2 mg every 4 - 6 hours (maximum 12 mg daily)
- If less sedation is required (e.g. day time)

**Adult and Child over 6 years: 10 mg orally daily or 5 mg every 12 hours**

**Chlorphenamine**

**Sodium cromoglycate**

**Antihistamines**
- **Adult:** 5 mg orally every 8 hours
- **Child under 12 years and elderly:** not recommended

**VITILIGO**

**Introduction**
- A disease characterized by acquired loss of melanocytes, leading to areas of depigmentation
- Sometimes associated with uveitis and other autoimmune phenomena
- Many autoantibodies can be demonstrated in vitiligo patients; those against melanocytes may rarely be demonstrable
- There is also a neural hypothesis
- Vitiligious patches often follow a dermatome
- A neurochemical mediator responsible for destroying the melanocytes has therefore been suggested
- There is also an occupational vitiligo
- Due to chemically induced depigmentation
- Seen among workers who are in contact with para-phenolic compounds or hydroquinones (but this is considered a different disorder)

**Clinical features**
- All ages are affected
- The dermatomal type is more common in the paediatric age
- The completely depigmented patches have distinct borders
- A few patients may have inflammatory vitiligo with raised erythematous borders
- These may have hypopigmented skin between the depigmented and normal skin (trichrome vitiligo)
- The distribution may be: Generalized (autoimmune type) Segmental (dermatomal type)
- The hair on the patches eventually turn white (acquired poliosis)
- The generalized type may be symmetrically distributed in the extremities
- Generalized vitiligo continues to spread while new lesions develop for years
- Spontaneous repigmentation may occur
- Favoured sites are: Extensor surfaces of the extremities Face and peri-orificial surfaces (around the mouth, eyes, nipples, umbilicus, penis, vulva, and anus)
- Focal vitiligo may affect one non-dermatomal site e.g. lips, vulva or penis
- Universal vitiligo applies to cases where the entire body surface is depigmented
- Generalized vitiligo may be associated with: Hyperthyroidism Hypothyroidism Pernicious anaemia Diabetes mellitus Addison’s disease
- Local loss of pigment may occur around a naevus and melanoma, the so-called halo phenomenon
- Vitiligo-like leucoderma occurs in about 1% of melanoma patients

**Disfiguration**
- Usually a good prognostic sign since it suggests an effective immune reaction against the tumour cells
- Segmental vitiligo affects only one part of the body
- It spreads rapidly in that area and then stabilizes
- It is not associated ith autoimmune diseases
- Favoured sites are the trigeminal area or an intercostal nerve distribution (zosteriform pattern)
- Just as with albinism, the interplay between the melanocytes of the eyes, ears, and skin is apparent
- The prototype is Vogt-Koyanagi-Harada syndrome: Vitiligo of the face, eyelashes, and scalp hair in association with:
  - Uveitis
  - Dysacousis
  - Alopecia areata
- Chemical vitiligo affects sites of contact with the chemicals
- When the chemicals are inhaled or a substantial quantity is absorbed through the skin, the distribution of the white patches may simulate the generalized autoimmune type

**Differential diagnoses**
- Post-burns depigmentation
- Tertiary stage of pinta
- Morphoea
- Lichen sclerosis
- Pityriasis alba
- Tinea versicolor
- Pseudobaldism
- Hypomelanosis of Ito

**Complications**
- Emotional problems due to cosmetic disfigurement

**Investigations**
- Exclude other autoimmune diseases if clinically suggestive
- See also notes on caution below

**Treatment objectives**
- Re-pigmentation
- Improve cosmetic appearance
- Emotional support

**Topical**
- Corticosteroids
- Hydrocortisone 1% or betamethasone valerate

**Adult: 0.1% apply once or every 12 hours (for focal or limited lesions)**
- **Child:** apply 1 - 2 times daily
- Psoralsens
- 8-methoxypsoralen (MOP) 0.05% - 0.1% in combination with ultraviolet-A radiation (PUVA) for focal or limited lesions

**Adult and child:** apply twice weekly
- Tacrolimus
- 0.1% ointment twice daily for 24 weeks
- Topical depigmentation
- Monobenzyl ether of hydroquinone
- 20%, apply twice daily for 3 - 6 months (if more then...
CHAPTER 7: EAR, NOSE AND THROAT

ACUTE OTITIS MEDIA

Introduction
Acute inflammation of the middle ear due to pyogenic organisms usually secondary to upper respiratory infection spreading from nasopharynx.

Common in infants and young children; more frequent during winter and rainy periods.

Usual organisms are streptococcus pneumococcus and staphylococcus.

Clinical features
Main symptoms:
- Earache
- Fever
- Deafness
- Ear discharge
- Malaise
- In babies, irritability

Complications
- Pneumonia
- Intracranial suppuration
- Meningitis
- Brain abscess
- Lateral sinus thrombosis
- Facial nerve paralysis
- Referral otalgia

Investigations
Ear swab for culture and sensitivity.
- Myringotomy: for persistent mucopurulent collection in middle ear with bulging eardrum.
- X-ray of mastoids: shows sclerosis, hypopneumatization, and bony erosion.
- Barotrauma.

Treatment objectives
Systemic decongestant.
- Prednisolone tablets 0.5 - 1.0 mg orally 2 - 5 times daily.
- Analgesics.
- Decongestants.

Non-drug treatment
- Myringotomy for persistent perforation.
- Asthma medication: good general health and clean airy environment.

Differential diagnoses
- Acute mastoiditis
- Facial nerve paralysis
- Labyrinthitis

Clinical features
- Chronic and recurrent ear discharge
- Pain is uncommon
- Discharge in the simple type is mucoid but thick and foul-smelling in the serious variety

Differential diagnoses
- Nasal obstruction and mouth-breathing
- Snoring at night
- Progressive deafness due to secretory otitis media

Drug treatment
- Antibiotics: Amoxicillin.
- Analgesics.
- Decongestants.

Prevention
unknown.

CHRONIC OTITIS MEDIA

Introduction
A chronic inflammatory condition of the middle ear mucosa with recurrent ear discharge.

Usual causes: acute otitis media with recurrent ear discharge.

Clinical features
Main complaints: recurrent ear discharge and increasing deafness.

Complications
- Pain is uncommon
- Discharge in the simple type is mucoid but thick and foul-smelling in the serious variety

Differential diagnoses
- Chronic infection of adenoid tissue.
- Nasal obstruction and mouth-breathing
- Progressive deafness due to secretory otitis media

Drug treatment
- Antibiotics.
- Decongestants.
- Prednisolone tablets.

Prevention
unknown.

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- Nasal obstruction and mouth-breathing
- Progressive deafness due to secretory otitis media

Drug treatment
- Antibiotics.
- Decongestants.
- Prednisolone tablets.

Prevention
unknown.
**Chapter 7: Ear, Nose and Throat**

**Drug treatment**

- **Antibiotic**
  - Co-amoxiclav
  - **Adul**: 500/125 mg orally every 8 hours for acute exacerbations up to 14 days
  - **Child**: 6 - 12 years: 250 mg orally every 12 hours; under 6 years: 125 mg every 12 hours

**Supportive measures**

- **Protection**
  - From water with Vaseline/cotton wool while bathing

**Caution**

- Topical treatment with ototoxic antibiotics is contraindicated in the presence of a perforation

**EPISTAXIS**

**Introduction**

- A condition of bleeding from the nose
- A clinical presentation rather than a disease entity on its own
- Bleeding is most often from ruptured vessels in the anterior nasal septum, sometimes from the posterior nose especially in the elderly
- Can arise from a wide variety of causes

**Clinical features**

- Trauma
- Inflammation of nose or sinuses
- Acute e.g. acute rhinitis/sinusitis
- Chronic e.g. tuberculosis, leprosy
- Neoplasms

**Manifestation of systemic diseases**

- Bleeding diatheses
- Blood dyscrasias
- Hypertension

**Clinical features**

- Bleeding from nose; often spontaneous but may follow obvious trauma or injury
- Varying amounts of blood, from few drops to torrential life-threatening haemorrhage
- Often intermittent; most bleeds stop spontaneously

**Differential diagnoses**

- Various pathological conditions, both local and systemic present with nasal bleeding

**Complications**

- Haemorrhagic shock
- Fatality

**Investigations**

- Full Blood Count, including platelet count
- Bleeding and clotting time
- Partial thromboplastin time
- Urea and Electrolytes and Creatinine
- X-ray sinuses
- CT scan

**Treatment objectives**

- To arrest bleeding in actively bleeding cases
- Replace significant blood losses and treat shock

**Identify and treat aetiological factors**

**Non-drug treatment**

- Pressure and compression of the nose between fingers to arrest bleeding
- Cotton wool pack soaked in epinephrine 1:1000 may be placed on bleeding area before compression to induce vasoconstriction
- Nasal packing with lubricated ribbon gauze
- Arrest of posterior bleed with rubber tampon or improvised Foley’s catheter balloon
- Cauterization of bleeding point or dilated vessels in anterior nasal septum
- Dilatation of cauter (electrical) or chemical cautery with silver nitrate stick

**Drug treatment**

- Treat underlying actiology
- Sedation if necessary
- Diazepam 5 mg orally twice daily for 1 - 2 days
- Antibiotics if infection is present
- Amoxicillin

**Adult**: 500 mg orally every 8 hours for 5 - 7 days

**Child**: 250 - 500 mg orally for 5 - 7 days

**Other drugs depending on identified causative factors**

- Intra-nasal infusion, crystalloids and blood as necessary
- Bed rest

**Preventive**

- Avoid/treat predisposing conditions

**FOREIGN BODIES IN THE AIRWAYS**

**Introduction**

- Children (most commonly) may aspirate pieces of play objects or food items accidentally into the airway
- May present as serious emergencies with imminent asphyxia
- The object if arrested at laryngeal level causes acute upper respiratory obstruction
- Sharp objects such as fish bone or pins may be impacted on the vocal cord and the resulting oedema causes progressive obstruction
- Small objects such as seeds may traverse the larynx and become arrested in the trachea or bronchus lower down
- Vegetables such as peanuts often cause severe reaction in the lungs with pneumonitis

**Clinical features**

- Difficulty in breathing with stridor occurs immediately or progressively
- Initial dyspnoea and cough may subside if the object passes down. Symptoms gradually return later
- Severe cases: stridor and severe cyanosis with imminent asphyxia requiring immediate intervention to prevent a fatal outcome
- Two-way stridor often occurs with tracheal foreign bodies

**Clinical features**

- In the lower airways objects may remain for long periods, with unexplained chest symptoms

**Differential diagnoses**

- Acute laryngitis
- Acute laryngeal oedema
- Bronchopneumonia
- Pulmonary tuberculosis

**Complications**

- Life-threatening asphyxia
- Lung collapse and atelectasis

**Investigations**

- Radiograph of neck and chest

**Treatment objectives**

- To maintain the airway and adequate respiratory function
- Remove the foreign object as expeditiously as possible

**Non-drug treatment**

- Immediate removal under anaesthesia by direct laryngoscopy or bronchoscopy as appropriate
- Tracheostomy where necessary to maintain airway

**Drug treatment**

- Antibiotic prophylaxis if necessary (for 3 days)
- Amoxicillin

**Child**: 6 - 12 years: 250 mg orally every 12 hours; under 6 years: 125 mg orally every 12 hours

- Steroid
- Hydrocortisone (for pneumonitis)
  - Child: 1 month - 1 year: initially 25 mg by intravenous or intramuscular injection every 8 hours
  - 1 - 6 years: initially 50 mg every 8 hours; 6 - 12 years: initially 100 mg every 8 hours; 12 - 18 years: initially 100 - 500 mg 3 times daily, adjusted in all age groups according to response

**Supportive measures**

- Oxygen
- Steam inhalation/nebulizer

**Prevention**

- Vigilant supervision of young children

**FOREIGN BODIES IN THE NOSE AND RHINOLITHS**

**Introduction**

- Children often insert various objects into the nostrils while playing: pieces of plastic toys, rolled paper, foam, seeds, some metal objects, etc
- The objects may remain undetected for long periods, particularly organic items, until they become infected
- Typically result in foul smelling unilateral nasal discharge
- Some inorganic objects may (after long periods) become coated by hard calcific deposits and become known as rhinoliths

**Clinical features**

- Often no indication or symptom
- May be accidentally noticed by parent
- Later, complaints of foul purulent unilateral nasal discharge of unknown origin

**Differential diagnoses**

- Acute or chronic rhinitis
- Sinusitis
- Nasal growth/polyp

**Complication**

- Secondary infection: rhinosinusitis

**Investigation**

- Radiograph of nose: for metallic or radio-opaque objects

**Treatment objectives**

- Remove object expeditiously without damage to ear structures or causing undue pain to patient

**Non-drug treatment**

- Removal by ear syringing
- Removal with appropriate hook, or alligator forceps
- Examination and removal under anaesthesia if difficult in the clinic

**Prevention**

- Vigilant supervision of young children

**MASTOIDITIS**

**Introduction**

- Develops as a complication of acute suppurative otitis media, mostly in children
- Follows acute otitis media (untreated or inadequately treated), or due to particularly virulent organisms
- Infection spreads from the tympanum posteriorly into the mastoid antrum and aircells
- Colliquative necrosis of the air cells and suppuration in the mastoid bone follows

**Potential complications**

- Haemorrhagic shock
- Fatality

**Investigations**

- Full Blood Count, including platelet count
- Bleeding and clotting time: partial thromboplastin time
- Urea and Electrolytes and Creatinine
- X-ray sinuses
- CT scan

**Treatment objectives**

- To arrest bleeding in actively bleeding cases
- Replace significant blood losses and treat shock

**Identify and treat aetiological factors**

**Non-drug treatment**

- Pressure and compression of the nose between fingers to arrest bleeding
- Cotton wool pack soaked in epinephrine 1:1000 may be placed on bleeding area before compression to induce vasoconstriction
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- Cauterization of bleeding point or dilated vessels in anterior nasal septum
- Dilatation of cauter (electrical) or chemical cautery with silver nitrate stick

**Drug treatment**

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- Sedation if necessary
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- Antibiotics if infection is present
- Amoxicillin

**Adult**: 500 mg orally every 8 hours for 5 - 7 days

**Child**: 250 - 500 mg orally for 5 - 7 days

**Other drugs depending on identified causative factors**

- Intra-nasal infusion, crystalloids and blood as necessary
- Bed rest

**Preventive**

- Avoid/treat predisposing conditions

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**Introduction**

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**Investigations**

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**Treatment objectives**

- To maintain the airway and adequate respiratory function
- Remove the foreign object as expeditiously as possible

**Non-drug treatment**

- Immediate removal under anaesthesia by direct laryngoscopy or bronchoscopy as appropriate
- Tracheostomy where necessary to maintain airway

**Drug treatment**

- Antibiotic prophylaxis if necessary (for 3 days)
- Amoxicillin

**Child**: 6 - 12 years: 250 mg orally every 12 hours; under 6 years: 125 mg orally every 12 hours

- Steroid
- Hydrocortisone (for pneumonitis)
  - Child: 1 month - 1 year: initially 25 mg by intravenous or intramuscular injection every 8 hours
  - 1 - 6 years: initially 50 mg every 8 hours; 6 - 12 years: initially 100 mg every 8 hours; 12 - 18 years: initially 100 - 500 mg 3 times daily, adjusted in all age groups according to response

**Supportive measures**

- Oxygen
- Steam inhalation/nebulizer

**Prevention**

- Vigilant supervision of young children
A subperiosteal abscess forms behind the ear in a child with a discharging ear

**Clinical features**
- Fever
- Pain behind the ear
- Mucopurulent ear discharge
- Progressive inflammatory swelling over the mastoid region

**Complications**
- Suppurating post-aural lymphadenitis from otitis externa

**Diagnosis**
- Externtate the infected air cells and drain the mastoid

**Drug treatment**
- Large doses of parenteral antibiotics
  - Amoxicillin
  - Neomycin/hydrocortisone ear drops

**Non-drug treatment**
- Cortical mastoidectomy to open the mastoid

**Pharyngitis**
- Chronic rhinitis from other causes
- Vasomotor rhinitis
- Chronic sinusitis

**Skin tests for allergens:** intradermal or prick tests

**Skin tests for sensitization:** for esophagitis

- Serological tests: radio-immunoassay for IgE antibodies

**Treatment objectives**
- Control and eradicate infection
- Prevent more serious complications

**Treatment**
- Control or suppress the allergic symptoms
- Prevent allergic reactions

**Supportive measures**
- Pain and itching
- Ear discharge
- Sensation of blockage due to accumulated debris in canal

**Prevention**
- Avoid trauma to ear canal (especially scratching)
- Keep ears dry

**PERITONSILLAR ABSCES (Quinsy)**

**Introduction**
- The main common local complication of acute tonsillitis
- A virulent streptococcal infection; may spread beyond the tonsillar capsule into the peri-tonsillar space, causing, first cellulitis, and later suppuration in the space

**Clinical features**
- Often referred pain to ipsilateral ear
- Difficulty in opening mouth for examination; mouth full of saliva
- Affected tonsil displaced downwards and medially, with swelling above and lateral to it, all inflamed and oedematous

**Prevention**
- Follows an attack of acute tonsillitis
- Increasing pain, fever and dysphagia
- Trismus- spread of oedema and infection to pterygoid muscles
- Often referred pain to ipsilateral ear
- Difficulty in opening mouth for examination; mouth full of saliva

**Differential diagnoses**

**Urinalysis for glycosuria**

**Blood glucose estimation in cases of recurrent furunculosis to exclude diabetes mellitus**

**Treatment objectives**
- Control infection / inflammation
- Relieve discomfort

**Non-drug treatment**
- Careful ear toilet to clear out debris
- Daily dressing with antiseptic gauze packed with topical steroid

**Antibiotics**
- Aminocillin
- Neomycin/hydrocortisone ear drops

**Supportive measures**
- Prevent more serious complications

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**Differential diagnoses**
- Parapharyngeal abscess
- Retropharyngeal abscess
Tonsillar tumors
Complications
Septicaemia
Parapharyngeal suppuration/abscess
Investigations
Throat swab
Full Blood Count with differentials
Treatment objectives
Rapid control of infection
Relief of pain and discomfort
Non-drug treatment
Throat swab, preferably under local anaesthetic when suppuration is definite

Drug treatment
Antibiotics
- Amoxicillin
  Adult: 500 mg - 1 g intravenously every 6 hours for 7 days
  Child: 50 - 100 mg/kg orally every 8 hours
  Analgesics
  - Paracetamol
  - Aspirin (Acetylsalicylic acid)
  Adult: 300 - 900 mg orally every 4 - 6 hours
  Note: recommended in children (risk of Reye's syndrome)

Supportive measures
- Intralesional infusion
  Bed rest
Notable adverse drug reactions
Aspirin may cause gastrointestinal irritation

Prevention
Elective tonsillectomy is advised after an episode of quinsy to prevent further (more severe) attacks

PHARYNGITIS (Sore Throat)
Introduction
A common cause of persistent sore throat in young and middle-aged adults, usually unaccompanied by other symptoms
- Often secondary to chronic nasal conditions with nasal obstruction e.g.
  - Vasomotor rhinitis
  - Nasal polyps
  - Septal deviation
- Obstruction causes mouth breathing with dryness of the throat
Other causes:
Secondary inflammation from postnasal discharge of sinuses

SINUSITIS
Introduction
Inflammation of the mucosal lining of the paranasal sinuses
- May be acute or chronic and affect one or more of the sinuses
- Most commonly the maxillary sinus or antrum (in very young children the ethmoidal sinuses)
  - Acute sinusitis is often sequel to acute rhinitis
  - Common organisms are streptococcus, pneumococcus, and haemophilus
  - Chronic sinusitis is more insidious
  - May be associated with chronic rhinitis and allergy but other factors such as air pollution, smoking, dental sepsis and poor general health may be contributory
  - Bacteriology is mixed: sometimes Gram negative and fungal organisms

Clinical features
Rhinorrhea
Nasal obstruction
Fever with pain over affected sinus in acute cases
Less dramatic symptoms in chronic sinusitis
- Intermittent nasal obstruction and discharge over a long period
- Little pain

Diagnosis
Diagnostic procedures
- X-ray of paranasal sinuses
- m-scan in complicated cases
- Aspiration: microbiology and culture
- Nasal swab for microscopy, culture and sensitivity

Treatment
Control symptoms by identifying and treating primary cause

Clinical features
- Subdural abscess
  - Meningitis
  - Cerebral abscess
  - Dural vein thrombophlebitis
  - Osteomyelitis of frontal or maxillary bones
  - Chronic pyrhythmoneitis and bronchitis

Investigations
- X-ray of sinuses: four-view
- Antrum roof puncture/lavage: specimen for culture
- CT scan in complicated cases
- Trephining of frontal sinus
- Incision and drainage, preferably under local anaesthetic
- Intranasal antrostomy
- Caldwell-Luc operation
- Fronto-ethmoidectomy

Pharyngitis
Introduction
An inflammatory condition of the palatine tonsils, most common in children
In half or more cases infection is by beta-haemolytic streptococcus, in others viral
Typically an acute infection
Chronic tonsillitis presents usually as recurrent acute infection
Essentially a disease of children but also occurs in young adults

Clinical features
Fever
Sore throat
Dysphagia

Tonsillitis
Introduction
An inflammatory condition of the palatine tonsils, most common in children
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Essentially a disease of children but also occurs in young adults

Clinical features
Fever
Sore throat
Dyspagia

Differential diagnoses
Adult:
- Septicaemia
- Parapharyngeal suppuration/abscess

Investigations
- Psuedoephedrine tablets

Supportive measures
- Paracetamol
- Aspirin (Acetylsalicylic acid)
- Supportive measures
  - Bed rest
  - Antipyretics
  - Analgesics

Prevention
Avoid airway irritants, smoking, and alcohol
Protect against air pollution
Maintain good general health and nutrition

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Treatment objectives
Allergic rhinitis
- Non-drug treatment
  - Supportive measures
    - Paracetamol
    - Antipyretics
  - Drug treatment
    - Antihistamines
    - Decongestants
    - Cetirizine
Child:
- Cetirizine

Investigations
- X-ray of sinuses: four-view
- Antrum roof puncture/lavage: specimen for culture
- CT scan in complicated cases
- Trephining of frontal sinus
- Incision and drainage, preferably under local anaesthetic
- Intranasal antrostomy
- Caldwell-Luc operation
- Fronto-ethmoidectomy

Non-drug treatment
- Non-drug treatment
  - Supportive measures
    - Bed rest
    - Antipyretics
    - Analgesics

Prevention
Avoid airway irritants, smoking, and alcohol
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Clinical features
Fever
Sore throat
Dysphagia
TRACHEOSTOMY

Introduction
A surgical procedure in which an opening is created into the trachea from the outside, commonly to bypass an upper respiratory obstruction.
- May also be done to provide easier access for care of the chest in some seriously ill patients.
- Also for respiratory support and artificial ventilation in patients with respiratory insufficiency or paralysis.

Most cases are done to bypass upper airway obstruction:
- Acute infections of the larynx
- Trauma
- Foreign body aspiration
- Acute laryngeal oedema
- Vocal cord paralysis
- Tumours

Some cases are done as part of, or to facilitate major head and neck surgery.
An appropriate-sized tracheostomy tube, portex or metal, is inserted to maintain the opening.

Clinical features
Acute presentation with clinical features of airway obstruction, stridor and incipient asphyxia following trauma.

Acute inflammatory conditions of the larynx, which would require the operation as an emergency.
Progressive lesions: may require less urgent intervention in anticipation of likely obstruction.
Cases with medical indications requiring respiratory support are usually done on a more elective basis.

Complications
- Haemorrhage
- Infection: wound and chest
- Damage to nerves and large vessels in the neck

Treatment objectives
To secure the airway.

Postoperative care of tracheostomy preferably in an intensive care unit, with suction, humidification, stoma care as appropriate.

Drug treatment
Broad spectrum antibiotic cover.

WAX IN THE EAR

Introduction
Wax (or cerumen) is a normal product of the human external ear.
- A dark brownish mixture of the secretions of the ceruminous and sebaceous glands in the outer third of the external auditory canal.
- Small quantities are produced continuously and function to lubricate the canal.
- Quantities produced and the consistencies vary.
- May be excessive in some people, causing deafness, ear ache, secondary infection and even vertigo.

Sensation of blockage and some degree of deafness are the most common complaints.
- Sometimes, pain and irritation
- Ear discharge in some cases
- Quantity seen varies
- May be soft or hard
- May be impacted in the deep meatus

Differential diagnoses
- Foreign bodies
- Otitis externa

Complications
- Superimposed infection: otitis externa
- Hearing impairment

Treatment objectives
- Evacuate the wax and clear the ear
- Removal with probe and cotton wool: for soft wax
- Ear syringing: for hard wax, often after preliminary softening with oily drops
- Occasionally, removal under anaesthesia if syringing is unsuccessful.

Drug treatment
- Ear drops to soften and loosen wax
- Warm olive oil
- Or: Chlorobutanol 5% paradichlorobenzene 2%, arachis (peanut) oil 57.3%

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Chapter 7: Ear, Nose and Throat

Chapter 8: Endocrine System

Diabetes Mellitus

Introduction
A group of metabolic diseases characterized by chronic hyperglycaemia.
- Results from defects in insulin secretion, insulin action or both.
- It is associated with acute as well as long-term complications affecting the eyes, kidneys, feet, nerves, brain, heart and blood vessels.
- Its classification has been revised by the WHO and is based on aetiology:
  - Type 1:
    - Results from destruction (usually autoimmune) of the pancreatic β cells.
    - Insulin is required for survival.
  - Type 2:
    - Characterized by insulin resistance and/or abnormal insulin secretion (either may predominate).
    - Both are usually present.
    - It is the most common type of diabetes.
  - Other specific types of diabetes- less common, and include:
    - Genetic disorders
    - Infections
    - Diseases of the exocrine pancreas
    - Endocrinopathies
    - Drugs

Gestational diabetes: appears for the first time in pregnancy.

Clinical features
Type 1 diabetes:
- Patients present at a young age (usually teens or twenties);
- Earlier presentation may also occur
- Rapid onset of severe symptoms: weight loss, thirst and polyuria
- Blood glucose levels are high and ketones are often present in the urine.
- If treatment is delayed, ketoacidosis (DKA) and death may follow.
- The response to insulin therapy is dramatic and gratifying.
- Misclassification of patients as “Type 1” is relatively common.
  - Insulin-treatment is not the same as insulin-dependence.

Type 2 diabetes:
- Most patients present with the classical symptoms including polyuria, polydipsia and polyphagia.
- Some patients present with sepsis, diabetic coma (hyperosmolar non-ketotic states).
- A minority is asymptomatic and therefore identified at screening.
- The patients usually do not seek medical attention early because of the insidious nature of the disease.
- Many present at diagnosis with features of diabetic.
Diagnosis

Straightforward in the majority of cases
May pose a problem for those with a minor degree of hyperglycaemia, and in asymptomatic subjects
- In these circumstances, two abnormal blood glucose results on separate occasions are needed to make the diagnosis
- If the results of point blood glucose testing are equivocal, an oral glucose tolerance test should be performed
- If diagnosis remains in doubt maintain surveillance

Goals of dietary management of Type 2 diabetes mellitus

The diagnosis of diabetes must be confirmed biochemically prior to initiation of any therapy

Symptoms of hyperglycaemia

Plus:
- Random venous plasma glucose ≥11.1 mmol/L or fasting venous plasma glucose ≥ 7.0 mmol/L

- Confirms the diagnosis of diabetes
- In asymptomatic subjects, a single abnormal blood glucose result is inadequate to make a diagnosis of diabetes
- Abnormal values must be confirmed at the earliest possible date using any of the following:
  - Two separate fasting or random blood samples
  - A 75 g oral glucose tolerance test

Values for the Diagnosis of Categories of Hyperglycaemia

<table>
<thead>
<tr>
<th>Glucose Tolerance State</th>
<th>Venous plasma (mmol/L)</th>
<th>Venous plasma (mg/dL)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Diabetes mellitus</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fasting</td>
<td>≥ 7</td>
<td>≥ 126</td>
</tr>
<tr>
<td>2 hour post-75 g glucose load</td>
<td>≥ 11.1</td>
<td>≥ 200</td>
</tr>
<tr>
<td><strong>Impaired glucose tolerance</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fasting</td>
<td>&lt; 7.0</td>
<td>&lt; 110</td>
</tr>
<tr>
<td>AND</td>
<td>≥ 7.8 and &lt; 11.1</td>
<td>≥ 140 and &lt; 200</td>
</tr>
<tr>
<td>2 hour post-75 g glucose load</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Impaired fasting glycaemia</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fasting</td>
<td>≥ 6.1 and &lt; 7.0</td>
<td>≥ 5.6 and &lt; 6.1</td>
</tr>
</tbody>
</table>

Unless there is unequivocal hyperglycaemia with acute metabolic decompensation or obvious symptoms, the diagnosis of diabetes should always be confirmed by repeating the test on another day

Chapter 8: Endocrine System

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Management

Goals:
- Early diagnosis
- Prevent and/or reduce short and long term morbidities
- Prevent premature mortality
- Improve quality of life and productivity of affected persons
- Promote self care practices and empowerment of people with diabetes
- Reduce the personal, family and societal burden of diabetes

Achievement of these goals is dependent on:

- Successful establishment of diabetes health care team, and infrastructure to support it, including provision of education for health care professionals and for people living with diabetes
- Treatment of co-morbidities
- Prevention and treatment of macrovascular and microvascular complications

Non-drug treatment

Education

The provision of knowledge and skills to people with diabetes mellitus
- To empower them to render self-care in their management

Principles of Diabetes Education

Should be locally applicable, simple and effective
- All members of the diabetes care team should be trained to provide the education
  - It must empower people with diabetes as well as their families
  - Provide them with adequate knowledge of diabetes and its sequelae
  - Create the right attitudes and provide resources to provide appropriate self care
  - The effectiveness of the programme must be evaluated and modified as necessary

What people with diabetes need to know

Diabetes is serious but can be controlled
- Complications can be prevented
- That the cornerstones of therapy are education, diet and exercise
- Their metabolic and blood pressure targets
- How to look after their feet and thus prevent ulcers and amputations
- How to avoid other long term complications
- That regular medical check ups are essential
- When to seek medical help

Diet

- One of the cornerstones of diabetes management
- Based on the principle of healthy eating in the context of social, cultural and psychological influences on food choices
- Dietary modification (and increasing level of physical activity) should be the first step in the management of newly diagnosed persons with Type 2 diabetes
- Should be maintained throughout the course of diabetes management

Goals of dietary management of Type 2 diabetes mellitus

- To achieve an ideal body weight
- An appropriate diet should be prescribed along with an exercise regimen
- Caloric restrictions should be moderate and yet provide a balanced nutrition
- Eat at least three meals a day. Binge eating should be avoided
- A snack between meals can be healthy for certain groups of people
- The diet should be individualized, based on traditional eating patterns, be palatable and affordable
- Animal fat, salt, and so-called diabetic foods should be avoided
- Pure (simple sugars) in foods and drinks should be avoided
- Eating plans should be high in carbohydrates and fibre, vegetables and fruits should be encouraged
- Dietary instructions should be written out, even if the person is illiterate: someone at home should be available to interpret to him/her
- Food quantities should be measured in volumes using available household items (e.g. cups), or be countable (e.g. number of fruits or slices of yam or bread)
- Weighing scales are generally unaffordable and/or difficult to understand
- Appetite suppressants generally yield poor and/or unsustainable weight reductions and are expensive

Physical activity

- One of the essentials in the prevention and management of Type 2 diabetes mellitus
- Regular physical activity:
  - Improves metabolic control
  - Increases insulin sensitivity
  - Improves cardiovascular health
  - Helps weight loss
  - Gives a sense of well-being
- Two main types of physical activity:
  - Aerobic or endurance exercise (e.g. walking, running)
  - Anaerobic or resistance exercise (e.g. lifting weights)
- Both types of activity may be prescribed to persons with type 2 diabetes mellitus; the aerobic form is usually preferred

General principles and recommendations

Diet

- Cardiovascular, renal, neurological and foot assessments
- Evaluation should be done before a formal exercise programme is commenced
- The presence of chronic complications excludes certain forms of exercises
- Prescribed physical activity programmes should be
Sulphonylureas and biguanides are the agents most widely available.

- Stocking these agents would meet the diabetes care needs of most diabetes facilities.

The choice of OGLAs should be informed by:
- Lifestyle
- Degree of control
- Access to medicines
- Economic status
- Mutual agreement between the doctor and the person with diabetes

Monotherapy with any of the drugs should be the initial choice:
- Use of stepped-care approach is recommended.
- If overweight (BMI > 25 kg/m²) or if insulin resistance is the major abnormality
- If metformin is the first choice
- If metformin is contraindicated thiazolidinediones may be used

Avoid metformin and long-acting sulphonylureas in elderly patients.

- Instead, use short-acting sulphonylureas and/or glinides or glitazones

Combination therapy using OGLAs with different mechanisms of action is indicated if monotherapy with one of the agents has failed.

The rapid acting secretagogues (glinides) and the alpha glucosidase inhibitors make for flexibility in the glycaemic management of Type 2 diabetes mellitus but are relatively very expensive.

When oral combination therapy fails, insulin should be added to the treatment regimen or should replace the OGLAs.

**Time Course of Action of Insulin Preparations**

<table>
<thead>
<tr>
<th>Insulin Preparation</th>
<th>Onset of Action</th>
<th>Peak Action</th>
<th>Duration of Action</th>
<th>Injections per day</th>
</tr>
</thead>
<tbody>
<tr>
<td>Very rapid acting (insulin analogues)</td>
<td>10 min</td>
<td>1 h</td>
<td>3 h</td>
<td>Immediately before meals</td>
</tr>
<tr>
<td>Short-acting</td>
<td>30 min</td>
<td>2 - 5 h</td>
<td>5 - 8 h</td>
<td>30 min before meals</td>
</tr>
<tr>
<td>Intermediate-acting (NPH or lente)</td>
<td>1 - 3 h</td>
<td>6 - 12 h</td>
<td>16 - 24 h</td>
<td>Once or twice daily</td>
</tr>
<tr>
<td>Biphasic mixtures (30/70; premixed)</td>
<td>30 min</td>
<td>2 - 12 h</td>
<td>16 - 24 h</td>
<td>Once or twice daily</td>
</tr>
</tbody>
</table>

**Drug treatment**

Oral hypoglycaemic agents:

- For Type 2 diabetes mellitus
  - Indicated:
    - When individualized targets are not met by the combination of dietary modifications and physical activity/exercise

**Important notes on Oral Glucose Lowering Agents (OGLAs)**
Monitoring glycaemic control
Clinical and laboratory methods are employed
HbA1c tests are desirable standard tests but are unavailable in most of the primary and secondary health facilities in Africa
Fasting plasma glucose performed in the laboratory in place of HbA1c is the best alternative
- Its average for repeated measurements gives a reliable indication of the control
Glycosuria is a poor means of assessment of control
Self Blood Glucose Monitoring (SBGM) should be encouraged
Results of self urine testing or blood glucose tests should be recorded in a logbook
Clinic protocols should set out in some detail, the parameters to be monitored at the initial visits, at regular follow-up visits, and at annual reviews
At the initiation of insulin therapy, appropriate advice on SBGM and diet should be given

Treatment of co-morbidities
Examples are obesity, hypertension and dyslipidaemias
- See relevant chapters

Diabetic foot problems
Introduction
People with diabetes are at increased risk of foot ulcers and amputations which are major causes of morbidity and disability
Both foot ulcers and amputations can be prevented by education, anticipation, early recognition and prompt management
The most common predisposing factors for ulcers and amputations are:
- Poor foot hygiene
- Peripheral vascular disease
- Deformities and abnormal biomechanics
- Unsuitable or no footwear
- Corrosion of management

Regular inspection and examination of the foot at risk Identify the at-risk foot
Education of health workers, people with diabetes and their families
Appropriate footwear
Early treatment of non- ulcerative and ulcerative foot problems

Diabetes in pregnancy
Introduction
Gestational diabetes mellitus (GDM) is any degree of glucose intolerance first recognised in pregnancy
If inadequately managed, GDM is associated with increased risk of perinatal morbidity and mortality
Diagnosis and prompt institution of therapy reduce the risks of poor outcomes
Screening for GDM

When:
- Between 24 and 28 weeks of gestation
  Who: Women with
  - high risk for GDM
  - BMI ≥ 25 kg/m²
  - Previous history of GDM
  - Glycosuria
  - Previous large baby (> 4 kg)
  - Poor obstetric history
  - Family history of diabetes
  - Known IGT / IFG

Management
Combined health care team- obstetrician, diabetologist, diabetes educator, and paediatrician/neonatologist
Initial therapy is dietary modification
- Spread carbohydrate over 3 small to moderate sized meals and 2 - 3 snacks/day
- Consider an evening snack to prevent starvation ketosis
- Energy intake should provide for desirable weight gain during pregnancy
- For obese women a 30 - 33% calorie restriction is advised

Diabetic ketoacidosis
Introduction
Severe uncontrolled diabetes requiring emergency treatment with insulin and intravenous fluids
Blood ketones (acetoacetate and 3-hydroxybutyrate) concentration > 5 mmol/L
Carries a high mortality in Africa
- Through late presentation, delayed diagnosis and inadequate treatment
- Presents at any age although there is a well defined peak at puberty
Causes include:
- Infection
- Management errors
- New cases of diabetes (treatment not commenced)
- No obvious cause in about 40% of cases

Indications for immediate hospital admission
- Repeated vomiting or inability to take adequate oral fluids
- Hyperventilation
- Any disturbance of consciousness
- Persistent ketonuria
- Presence of infections

Initial treatment plan for Diabetic Ketoacidosis in adults
- Fluids and electrolytes
  - One litre per hour for 3 hours; thereafter according to need
  - Sodium chloride 0.9% injection
  - Hypotonic (half-normal) saline: 75 mmol/L if plasma sodium exceeds 150 mmol/L
  - Glucose 5% when blood glucose level falls below 14 mmol/L
  - Potassium (K+) replacement
  - To be added into each litre of fluid
  - Initially, 5-10 units/hour; by continuous intravenous infusion
- Correct hypotension (should respond to adequate fluid replacement)

Diabetic non-ketotic hyperosmolar state
Introduction
Characterized by the insidious development of:
- Marked hyperglycaemia (usually > 30 mmol/L)
- Dehydration
- Pre-renal uraemia
- Significant hyperketonaemia does not develop

Two-thirds of cases occur in previously undiagnosed cases of diabetes
- Usually affects middle-aged or elderly patients and carries a mortality of over 30%
- Precipitating factors include:
  - Infections
  - Diuretic treatment
  - Drinking glucose-rich beverages

Treatment
- Rehydration
  - Insulin therapy
  - Electrolyte replacement
  - In a manner similar to that used for diabetic ketoacidosis

Hypoglycaemia
Introduction
Affects over 70% of patients on insulin therapy
Common causes of hypoglycaemia in persons with diabetes mellitus
- Engaging in more exercise than usual
- Delay or omission of a snack or main meal
- Administration of too much insulin
- Eating insufficient carbohydrate
- Overindulgence in alcohol
- Overdosing with sulphonylureas

In the presence of low blood glucose (< 2 mmol/L)
- Characteristic symptoms and signs include:
  - Light headedness
  - Headaches
  - Tremulousness
  - Palpitations
  - Sweating
  - Feeling of hunger
  - Tachycardia
  - Hypertension (usually systolic)
  - Stroke-like presentations
  - Coma

Acute management
- Oral glucose if patient is conscious
- If patient is unconscious:
Investigations

- Specific:
  - Serum T<sub>3</sub>, T<sub>4</sub>, and TSH levels
  - Measurement of T<sub>3</sub> and T<sub>4</sub> levels

- Non-specific:
  - Liver function tests
  - Standard treatment guidelines for Nigeria 2008

Treatment objectives

- Achieve normal metabolic rates
- Obtain normal serum T<sub>3</sub>, T<sub>4</sub>, and TSH levels
- Prevent complications

Aetiology

1. Grave’s disease (80% of patients)
2. Multinodular goitre
3. Autoimmune functioning solitary thyroid nodule
4. Thyroiditis (sub-acute or postpartum)

- Iodine induced- drugs such as:
  - Carbimazole
  - Propylthiouracil
- Non-iodine induced:
  - Drugs such as:
    - Amiodarone
    - Radiographic contrast media
    - Iodine prophylaxis programmes

Hyperosmolar non-ketotic state

- Mild hypercalcaemia
- Fasting blood glucose
- Glycosuria may be present

Dehydration

- Tachypnoea;
- Increased thirst
- Fatigue and apathy

Hypothroidism (Thyrotoxicosis)

**Introduction**

A clinical syndrome which results from exposure of the body to excess levels of the thyroid hormones, Thyroxine (T<sub>4</sub>), and Tri-iodothyronine (T<sub>3</sub>).

**Consequences**

- Increased thyroid activity
- Hypothyroidism symptoms

**Differential diagnosis**

- Simple goitre
- Malignant tumours of the thyroid

**Complications**

- Hyperthyroid crisis (thyroid storm)
- Compression of the trachea

**Prevention of diabetes**

- Generalized obesity, central obesity and physical inactivity are the major modifiable risk factors, and should be avoided/corrected.
HYPOTHYROIDISM (Myxoedema)

Introduction

Refers to subnormal amounts of thyroid hormones in the circulation, and the clinical features associated with this

Aetiology

- May be primary or secondary
- Primary hypothyroidism more common
- Probably an autoimmune disease; may occur as a sequel to Hashimoto's thyroiditis
- Post therapeutic hypothyroidism (medical or surgical)

Secondary hypothyroidism:

- Occurs when there is failure of the hypothalamic-pituitary axis due to
- Deficient secretion of TRH from the hypothalamus
- Lack of secretion of TSH from the pituitary

Clinical features

Generally in striking contrast to those of hyperthyroidism; may be quite subtle, with an insidious onset

In adults:
- Dull facial expression, slow speech and poor memory
- Puffiness of the hands, feet and face
- Lethargy and fatigue
- Thinning, dryness and loss of hair
- Hypoesthesia
- Bradycardia
- Reduced systolic and increased diastolic blood pressure

Weight gain
- Decreased reflexes
- Constipation
- Menstrual abnormalities

In infants:
- Mental and physical retardation
- If not corrected, cretinism

Complications

Endogenous depression
- Reactive depression

Contraindications

- Myxoedema coma
- Cretinism in the young

Investigations

- Total serum T and T levels
- TSH stimulation test
- TRH test

Treatment objectives

- Establish cause
- Establish the severity of hypothyroidism
- Restore normal body functions
- Prevent complications

Drug treatment

- Replacement therapy
- Levothyroxine sodium (thyroxine sodium)

Adult: initially 20 - 100 micrograms (50 micrograms for those over 50 years) orally daily, preferably before breakfast
- Adjusted in steps of 50 micrograms every 3 - 4 weeks until metabolism normalizes (usually 100 - 200 micrograms daily)

Child 1 month - 2 years:
- Initially 15 micrograms/kg orally once daily, adjusted in steps of 25 micrograms daily every 2 - 4 weeks until metabolism normalizes
- 2 - 12 years: initially 5 - 10 micrograms/kg once daily, gradually increased in 50 micrograms every 2 - 4 weeks until metabolism normalizes
- 12 - 18 years: initially 50 - 100 micrograms once daily, gradually increased in 50 micrograms every 3 - 4 weeks until metabolism normalizes (usual dose 100 - 200 micrograms daily)

Or:
- Lithothyronine sodium (1-triiodothyronine sodium)

Adult: initially 10 - 20 micrograms orally daily, gradually increased to 60 micrograms daily in 2 - 3 divided doses
- Small initial doses in the elderly

In hypothyroid coma:
- 5 - 20 micrograms by slow intravenous injection, repeated every 12 hours (as often as every 4 hours if necessary) Alternatively:
- 50 micrograms by slow intravenous injection initially then 25 micrograms every 8 hours, reducing to 25 micrograms daily

Child 12 - 18 years:
- 10 - 20 micrograms orally daily, gradually increased to 60 micrograms daily in 2 - 3 divided doses

In hypothyroid coma:
- 1 month - 12 years: 2 - 10 micrograms by slow intravenous injection every 8 hours (up to every 4 hours if necessary);
- Reduce to 1 - 5 micrograms in patients with cardiovascular disease
- 12 - 18 years: 5 - 20 micrograms, repeated every 12 hours (up to every 4 hours if necessary)
- Reduce to 10 - 20 micrograms in patients with cardiovascular disease

Supportive measures

- Treat anaemia, constipation and other complications as appropriate
- Immediate mechanical ventilation in myxoedema coma

Notable adverse drug reactions, caution and contraindications

Carbamazepine and propranolol
- May cause severe bone marrow suppression (including pancytopenia and agranulocytosis)
- They are contraindicated in breastfeeding mothers

Chapter 8: Endocrine System

Standard Treatment Guidelines for Nigeria 2008

CHAPTER 9: EYE DISORDERS

ACUTE ANTERIOR UVEITIS (Iritis)

Introduction

Inflammation of the iris (with or without the ciliary body)

- Usually occurs without any associated systemic inflammation
- Tends to recur

Clinical features

- Eye pain
- Photophobia due to ciliary spasm
- Exudation into anterior chamber
- Flare and cells

Differential diagnoses

- Infective conjunctivitis
- Acute iritis
- Acute glaucoma

Complications

- Secondary glaucoma
- Cataracts

Investigations

- Chest radiograph to exclude sarcoidosis and tuberculosis
- Spinal X-ray (especially lumbrosacral segment) to exclude ankylosing spondylitis

Treatment

- Corticosteroid drops for treatment of inflammation:
  - Betamethasone sodium phosphate 0.1%
  - Atropine sulfate 0.5% or 1%

- Establish cause
- Establish the severity of hypothyroidism
- Restore normal body functions
- Prevent complications

Caution

- Avoid atropine drops if there is risk of acute glaucoma
- Monitor serum levels of hormones to ensure that patients are not exposed to cardiac risks

Prevention

- Iodinated salt to prevent iodine deficiency

ACUTE KERATITIS

Introduction

- Infection or inflammation of the cornea
- Could be secondary to trauma
- Sometimes associated with infective conjunctivitis
- Could occur de novo

Clinical features

- Irritation, pain
- Red eye (conjunctival congestion)
- Eye discharge: watery; purulent if bacterial
- Photophobia
- Visual impairment, depending on the site and size of the ulcer and if interstitial
Chapter 9: Eye Disorders

Hypopion, if associated with uveitis (no hypopion if viral)
Ulceration of cornea, which stains with fluorescent; no ulcer in interstitial keratitis

Aetiology
Exogenous
- Marginal ulcers secondary to bacterial conjunctivitis (S. aureus)
- Central ulcers (Pneumococcus, Herpes simplex, fungi)
Keratomalacia (Vitamin A deficiency)
Exposure (7th cranial nerve palsy or dysthyroid eye disease)
Endogenous
- Interstitial keratitis of congenital syphilis
- Interstitial keratitis of Herpes zoster

Differential diagnoses
Infective conjunctivitis
Acute iritis
Acute glaucoma

Complications
Corneal perforation

Investigations
Corneal scraping for microscopy, culture and sensitivity

Drug treatment
Antibiotic drops (if bacterial)
- Chloramphenicol eye drops 0.5%
- Apply 1 drop at least every 2 hours, and then reduce frequency as infection is controlled and continue for 48 hours after healing
Atropine drops
- 1 drop up to 4 times daily
Antivirals (if dendritic ulcer)
- Idoxuridine 5% in dimethylsulfoxide
Adult and child over 12 years: apply to lesions 4 times daily for 4 days, starting at first sign of attack
Child under 12 years: not recommended

Topical steroids
- Only for interstitial keratitis where there is no active ulcer

Non-drug measures
Lateral tarsorrhaphy for exposure keratopathy

Caution and contraindications to treatment
Never use topical steroids in the presence of an active ulcer

Prevention
Avoid initial infection or trauma promptly to avoid progression to keratitis

ALLERGIC CONJUNCTIVITIS

Introduction
Could occur on its own or in association with generalized atopy (asthma, eczema, spring catarrh)

Clinical features
Irriting of the eyes with gittiness

EYE INJURIES

Introduction
Injuries to the eye could be caused by blunt or sharp objects or chemicals

Aetiology
Blunt injuries e.g. a fist or a ball hitting the eye
Sharp injuries e.g. glass, metal, broom stick, etc

Blunt injury
Evelids: peri-orbital haematoma and oedema
Conjunctivitis: subconjunctival haemorrhage and chemosis
Cornea: abrasion or oedema
Anterior chamber: hyphaema from tears of the iris or ciliary body
Iris: traumatic mydriasis
Traumatic uveitis
Angle recession
Lens: dislocation into anterior or posterior chambers; cataract
Vitreous haemorrhage
Retina: peripheral tear leading to retinal detachment; oedema with haemorrhage (Commotio Retinae)
Choroid: tear with haemorrhage
Rupture of the eyeball, usually posteriorly (rare)
Vitamin C (ascorbic acid)

Complications
Posterior synechiae, hypopyon, endophthalmitis
Refractive changes due to corneal scarring
Corneal opacity/scarring
Irreversible blindness (optic nerve avulsion)
- Symblepharon: adhesions between bulbar and tarsal conjunctiva
- Trachoma: other forms of conjunctivitis
- Keratoconus
- Corneal plaques
Differential diagnoses
Skin sensitivitiy test to detect allergen

Drug treatment
Antiinflammatory preparations
- Antazoline sulfate 0.5%, xylometazoline hydrochloride 0.05%
Adult and child over 5 years: apply 2 - 3 times daily
- Sodium camoglycate eye drops
Adult and child: apply four times daily
- Diclofenac sodium 0.1% eye drops
Adult and child: apply once daily
Phlyctenular conjunctivitis:
- Treat for tuberculosis using standard regimen

Caution
Xylometazoline is a sympathomimetic; use with caution in patients susceptible to angle closure glaucoma
Systemic absorption of antazoline and xylometazoline may result in interactions with other drugs

Prevention
Avoid allergen(s) as much as possible in cases where it/they have been identified

Management
Blunt injuries
- Treat individual injury
- Sharp injuries
- Suture lacerations
- Remove foreign bodies with magnet if possible, or by virex decrease
- Parenteral antibiotics, if infected
- Evisceration (removal of the contents of the eyeball) if ruptured globe, or if infection not settling on antibiotics

FOREIGN BODIES IN THE EYE

Introduction
Foreign bodies are usually in the form of small particles of metal, vegetable matter or insects which embed on the surface of the eye
- Occasionally a high velocity material, usually a metal could be propelled into the eye

Clinical features
- May be embedded on the tarsal or bulbar conjunctiva, the cornea or inside the eye
- Intraocular foreign body (IOFB)
- IOFBs may be in the anterior chamber, iris, lens or vitreous; on the retina or even behind the eyeball after doubly perforating the eye

Differential diagnoses
Corneal abrasion
Endothelitis
Complications
- Perforation of the eye
- Endothelitis
- Retinal toxicity from a metallic IOFB

Investigation
Radiograph of the orbit with a localizing ring

Management
Removal of subtarsal, conjunctival or corneal foreign body under magnification e.g. slit lamp microscope

Caution
Ultrasound should be avoided in an eye with a perforating wound

Prevention
Appropriate protective goggles for sports, welding, game hunting etc

Chapter 9: Eye Disorders

Clinical features
Blunt injury
Evelids: peri-orbital haematoma and oedema
Conjunctivitis: subconjunctival haemorrhage and chemosis
Cornea: abrasion or oedema
Anterior chamber: hyphaema from tears of the iris or ciliary body
Iris: traumatic mydriasis
Traumatic uveitis
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Treatment Guidelines for Nigeria 2008

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Vitreous haemorrhage
Retina: peripheral tear leading to retinal detachment; oedema with haemorrhage (Commotio Retinae)
Choroid: tear with haemorrhage
Rupture of the eyeball, usually posteriorly (rare)
Optic nerve: avulsion
Blow out fracture of the orbital wall

Sharp Injury
Lacerations of eyelids, conjunctivae, cornea, sclerae, or corneo-sclera
Uveal prolapse with or without lens extrusion
Intraocular foreign body
Endothelitis
Chemical burns
Acids coagulate surface proteins
Alkalii penetrate into the anterior chamber causing uveitis
- Symblepharon: adhesions between bulbar and tarsal conjunctiva

Differential diagnoses
Skin sensitivity test to detect allergen

Drug treatment
Antiinflammatory preparations
- Antazoline sulfate 0.5%, xylometazoline hydrochloride 0.05%
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- Sodium camoglycate eye drops
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Management
Blunt injuries
- Treat individual injury
- Sharp injuries
- Suture lacerations
- Remove foreign bodies with magnet if possible, or by virex decrease
- Parenteral antibiotics, if infected
- Evisceration (removal of the contents of the eyeball) if ruptured globe, or if infection not settling on antibiotics

Chemical burns
Copious rinsing of eyeball and fornices with sodium chloride 0.9% or clean water at site
In hospital, copious rinsing again, to dilute offending agent
Remove particles from eye e.g. lime or cement
Antibiotic ointment
Rodding of fornice with ointment to prevent symblepharon
Topical steroids for uveitis once cornea is re-epithelized
Vitamin C (ascorbic acid)

Caution and contraindications
Avoid the use of topical steroids in active corneal ulceration
Avoid the use of harmful traditional eye medications; may cause more complications

Prevention
Wearing of appropriate protective eye goggles for sports, welding and when working with chemicals

FOREIGN BODIES IN THE EYE

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Foreign bodies are usually in the form of small particles of metal, vegetable matter or insects which embed on the surface of the eye
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Clinical features
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Complications
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- Retinal toxicity from a metallic IOFB

Investigation
Radiograph of the orbit with a localizing ring

Management
Removal of subtarsal, conjunctival or corneal foreign body under magnification e.g. slit lamp microscope

Caution
Ultrasound should be avoided in an eye with a perforating wound

Prevention
Appropriate protective goggles for sports, welding, game hunting etc
INFECTIVE CONJUNCTIVITIS

Introduction
The commonest cause of a red eye is infective conjunctivitis which could be caused by bacteria or viruses.

Clinical features
Red eye (generalized)
Eye discharge: purulent or catarrhal, worse on waking from sleep
Eye discomfort: grittiness
Photophobia: mild
Swollen eyelids in ophthalmia neonatorum

Aetiology
Staphylococcus aureus
Haemophillus influenzae
Neisseria gonorrhoea
- Oozes out when the eyelids are opened
- The conjunctivae are less inflamed in chlamydial infection
- May or may not be present

Complications
Internal stye (chalazion)
Conjunctival swab for microscopy, culture and sensitivity

Drugs treatment
Antibiotic eyedrops or ointments
- Chloramphenicol 0.5%
- Gentamicin sulfate 0.3% applied as stated above
- Ofloxacin 0.3% solution applied as stated above
- A systemic cephalosporin e.g. ceftriaxone

Management
Topical antibiotics
- Gentamicin 0.3% eye drops

PREVENTION
Avoid prolonged use of steroids
No real preventive measures available

OPHTHALMIA NEONATORUM

Introduction
Infection in both eyes of a newborn baby in the first one month of life, without obstruction of the nasolacrimal ducts

Clinical features
Swollen eyelids:
- Corneal affectation which could lead to perforation
- The conjunctivae are less inflamed in chlamydial infection
- Oozes out when the eyelids are opened
- May or may not be present

Aetiology
Bacterial:
- Especially Neisseria gonorrhoea: starts within 3 days after birth
- Chlamydia (usually starts 1 week after birth)
Chemicals:
- Others

Differential diagnosis
Lid oedema following prolonged difficult labour

Complications
Corneal perforation
Endophthalmitis

Investigation
Conjunctival swab for microscopy, culture and sensitivity

Non-drug measures
- Chloramphenicol 0.5%
days

Adult and child over 2 years: apply every 4 hours for no more than 5 days

Ophthalmia Neonatorum
- Gentamicin sulfate 0.3% applied as stated above
- Ofloxacin 0.3% solution applied as stated above
Plus:
- A systemic cephalosporin e.g. ceftriaxone

Adult: 1 g every 12 hours intravenously for 7 days
Child: by intravenous infusion over 60 minutes

Neonates: 20 - 50 mg/kg once daily, by deep intramuscular injection, intravenous injection over 2 - 4 minutes, or by intravenous infusion 1 month - 2 years: 250 - 500 mg orally every 6 hours (or 500 mg - 1 g every 12 hours)
1 month - 2 years: 125 mg orally every 6 hours; dose doubled in severe infections
2 years: 250 mg 6 hourly; 8 - 18 years: 250 - 500 mg 6 hourly; dose doubled in severe infections

Complications
Corneal affectation which could lead to perforation

SCLERITIS/EPISCLERITIS

Introduction
Inflammation of the sclera and episclera
Usually self-limiting but relapses may occur

Clinical features
Dull, deep-seated pain in the eye

Differential diagnoses
Pterygium
Phlyctenular conjunctivitis
Trauma to the eye

Complications
Thinning of the sclera
Anterior staphyloma
Scleral perforation

Care
Topical steroids or NSAIDs for the duration of symptoms

TREATMENT GUIDELINES FOR NIGERIA 2008
Apply 1 drop at least every 2 hours, and then reduce frequency as infection is controlled
Or:
- Ciprofloxacin 10 mg/kg per dose intramuscularly 12 hourly for 2 days
- Gentamicin 0.3% eye drops
Apply twice daily. (not to be used for more than 10 days)
Or:
- Tetracycline 1% eye ointment
Apply 3 times daily for one week or more, depending on the severity of the condition

STYE ( Hordeolum)

Introduction
- Localized conjunctival congestion

Drug treatment
- Amoxillin 250 - 500 mg orally every 8 hours for 5 - 7 days

Caution
- Discourage the use of traditional eye medication
Prevention
Clean eyelids regularly and thoroughly
For recurrent styes, use baby shampoo to clean the eyelashes regularly

THE RED EYE

Causes
Infective conjunctivitis including ophthalmia neonatorum
Allergic conjunctivitis
Keratitis
Chapter 9: Eye Disorders

TRACHOMA

Introduction
- Caused by Chlamydia trachomatis, an organism midway between a bacterium and virus.
- The organism is found in the conjunctival as well as corneal epithelium and is responsible for two different conditions:
  - Trachoma (a severe disease)
  - Inclusion conjunctivitis (milder)
- Trachoma is commonly associated with poverty and unhygienic living conditions

Clinical features
- Acute phase:
  - Irritable red eye
  - Mucopurulent discharge
  - Eyelid oedema, pain, photophobia in severe cases
- Chronic phase:
  - Follicles on tarsal conjunctiva
  - Papillae
  - Superficial punctate keratitis
  - Pannus formation on superior cornea
- End stage:
  - Eyelid scarring with trichiasis, entropion
  - Conjunctival scarring
  - Limbal scarring with Herbert's pits
  - Corneal scarring

Differential diagnoses
- Other forms of infective conjunctivitis (especially viral)
- Allergic/vernal conjunctivitis
- Corneal scarring from other diseases

Complications
- Trichiasis
- Entropion
- Corneal scarring

Investigations
- Conjunctival scraping for microscopy
- Giemsa staining for trachoma inclusion bodies
- ImmunoFluorescence or Eliza test

Drug treatment
- Topical:
  - Tetracycline ointment applied 4 times a day for 6 weeks
- Systemic:
  - Erythromycin, tetracycline (not recommended for young children) or the newer antibiotics e.g. azithromycin as appropriate
  - Azithromycin

Adult: 500 mg orally once daily for 3 days
Child over 6 months: 10 mg/kg (maximum 500 mg) orally once daily for 3 days; over 6 months (body weight 15 - 25 kg) 200 mg once daily for 3 days; body weight 26 - 35 kg: 300 mg once daily for 3 days; body weight 36 - 45 kg: 400 mg once daily for 3 days

Surgical treatment
- Indicated for the treatment of trichiasis, entropion, corneal scarring
- Corneal graft, but entropion must be corrected first

Caution and contraindications
- Systemic tetracycline is contraindicated in young children
- Prompt surgery for trichiasis and entropion to prevent blindness from corneal scarring

XEROPHTHALMIA

Introduction
- The spectrum of eye diseases under Vitamin A deficiency
- Ranges from night blindness to conjunctival xerosis, to Bitot's spots, corneal xerosis and finally keratomalacia

Clinical features
- Night blindness
- Dryness of the conjunctiva and cornea (xerosis)
- Tearing
- Bitot's spots
- Corneal degeneration (keratomalacia)

Differential diagnosis
- Measles keratoconjunctivitis

Complications
- Corneal perforation
- Corneal scarring
- Blindness

Investigations
- Conjunctival impression cytology (where available)
- Serum Vitamin A levels

Non-drug treatment
- Nutrition education

Drug treatment
- Vitamin A capsules 200,000 IU orally daily for two days, then one capsule after one week
- Topical antibiotics and antivirals where applicable
- Padding the eye (for active corneal ulceration)

Caution
- Avoid the use of harmful traditional eye medication

Prevention
- Distribution of massive dose capsules of vitamin A to affected communities
- Nutrition and health education
- Fortification of foods with vitamin A

Chapter 10: Genito-Urinary System

Nephrology

ACUTE RENAL FAILURE

Introduction
- A syndrome characterized by rapid decline in glomerular filtration rate with retention of nitrogenous waste products, disturbance of extracellular fluid volume, electrolytes and acid-base homeostasis

Classification/aetiology
- Pre-renal Acute Renal Failure
  - Hypovolaemia (e.g. from haemorrhage, severe diarrhoea and vomiting etc)
  - Low cardiac output (e.g myocardiatis)
  - Renal hypoperfusion (e.g. from use of angiotensin converting enzyme inhibitors)
  - Systemic vasodilatation (e.g. sepsis)
  - Hyperviscosity syndromes (e.g polycythaemia)

Intrinsic renal failure
- Renovascular obstruction (e.g. renal vein thrombosis)
- Glomerular disease e.g. glomerulonephritis
- Interstitial nephritis (e.g. infections, allergic, from antimicrobials like rifampicin)
- Intratubular deposition and obstruction (e.g. uric acid, oxalate stones)

Post renal Acute Renal Failure
- Ureteric obstruction (from calculi, blood clots etc)
- Bladder neck obstruction from prostate hypertrophy
- Urethral obstruction (e.g. from strictures, congenital urethral valves)

Clinical features
- Thirst, dizziness, hypotension, tachycardia in pre-renal ARF
- Oliguria (not invariable)
- Oedema, hypertension
- Flank pain, hesitancy, nocturia, in post-renal ARF

Complications
- Volume overload
- Hyperkalaemia
- Metabolic acidosis
- Uraemic encephalopathy
- Hypertension

Diuretic management
- Acute-on-chronic renal failure
- Chronic renal failure

Investigations
- Urine microscopy: casts (granular, hyaline)
- Urinalysis: proteinuria, haematuria
- Serum Electrolytes, Urea and Creatinine
- Full Blood Count with differentials
- Abdominal ultrasound scan

Treatment objectives
- Correct primary haemodynamic abnormality
- Correct biochemical abnormalities
- Prevent further renal damage

Non-drug treatment
- Fluid challenge (where indicated)
- Low potassium, low salt, low protein diet
- Avoid or discontinue nephrotoxic drugs

Drug treatment
- Antihypertensive drugs (see treatment of hypertension)
- Loop diuretics
- Furosemide:
  - Initially 250 mg by intravenous infusion over 1 hour at a rate not exceeding 4 mg/minute
  - Give another 500 mg if urine output is satisfactory
  - Effective dose can be repeated every 24 hours
  - If no response, dialysis is probably required

Supportive therapy
- Regular intermittent haemodialysis
- Peritoneal dialysis

Prevention
- Close attention to cardiovascular function and intravascular volume in high risk patients, especially those with pre-existing renal insufficiency
- Avoid hypovolaemia (especially in patients on nephrotoxic drugs)
- Adequate hydration and sodium loading in patients to be exposed to radiocounter dye investigations (for example)

CHRONIC KIDNEY DISEASE

Also chronic renal failure

Introduction
- A progressive and persistent deterioration in kidney structure and function ultimately resulting in accumulation of nitrogenous waste products and disruption of acid-base homeostasis.
- Also associated with derangement in the kidney's osmoregulatory, metabolic and endocrine function

Aetiology
- Hypertension
- Diabetes mellitus
- Chronic glomerulonephritis
- Systemic lupus erythematosus
- Chronic pyelonephritis
- Genetic e.g. adult polycystic kidney disease, Alport's syndrome

Clinical features
- Nocturia
- Oliguria
- Bleeding tendencies
- Anaemia
- Hypertension (not invariable)
Chapter 10: Genito-Urinary System

Membranous glomerulopathy
Mebrano-proliferative glomerulonephritis
Mesangio-proliferative glomerulonephritis

**Clinical features**
- Generalized body swelling
- Passage of frothy urine

**Complications**
- Peripheral arterial or venous thrombosis
- Acceleration of atherosclerosis
- Protein malnutrition
- Vitamin D deficiency
- Increased susceptibility to infections
- Iron-resistant microcytic hypochromic anaemia

**Differential diagnoses**
- Other causes of body swelling
  - Congestive heart failure
  - Decompensated chronic liver disease
  - Protein losing enteropathy

**Investigations**
- Blood:
  - Serum proteins
  - Serum lipids
- Urine:
  - Urinalysis
  - Urine microscopy, culture and sensitivity

**Treatment objectives**
Slow down rate of decline of GFR
Manage hypertension
Control hypertension
Provide renal replacement therapy (if in end stage)

**Non-drug treatment**
- Diet: low salt, low protein, low potassium
- Avoid nephrotoxic agents

**Drug treatment**
- Anti-hypertensive agents (see treatment of hypertension)
- Diuretics (furosemide at doses appropriate for clinical condition)
- Vitamin D and calcium supplements
- Erythropoietin
  - Initially 50 units/kg 3 times weekly; adjusted according to response in steps of 25 units/kg 3 times weekly at intervals of at least 4 weeks
  - Total 75 - 300 units/kg weekly, as a single dose or in divided doses
- Iron supplements
- Ferrous sulphate

**Child:**
- 1 month - 1 year: 120 mg 3 - 4 times daily with feeds; 1 - 6 years: 300 mg; 6 - 12 years: 600 mg; 12 - 18 years: 1.25 g; all 3 - 4 times daily prior to, or with meals and adjusted as necessary
- Aluminium hydroxide: 300 - 600 mg orally 3 times a day with meals

**Supportive measures**
- Haemodialysis
- Peritoneal dialysis

**Definitive treatment is renal transplantation**

**Notable adverse drug reactions, caution and contraindications**
- See furosemide
- Potential for adverse drug reactions with drugs eliminated primarily by the kidneys e.g. aminoglycoside antibiotics, NSAIDs, metformin, etc
- Calcium-containing phosphate-binding agents are preferred in children but are contraindicated in hypercalcaemia or hypercalciuria

**Prevention**
Appropriate management of known causes of chronic renal failure e.g. hypertension and diabetes mellitus

**Aetiology**
Idiopathic in a significant proportion of cases

**Histologic types**
- Minimal change disease
- Focal segmental glomerulosclerosis

**SEXUALLY TRANSMITTED INFECTIONS**

**BACTERIAL VAGINOSIS**
- A clinical syndrome resulting from replacement of the normal hydrogen peroxide-producing Lactobacillus sp. in the vagina by high concentrations of anaerobic bacteria, such as Gardnerella vaginalis
- Mycoplasma hominis
- Mobiluncus curtisi

**Clinical features**
- Cause of the microbial alteration is not fully understood
- The associated malodour is due to the release of amines produced by anaerobic bacteria that decarboxylate lysine to caverdine, and arginine to putrescine
- Predispensing factors are the use of antiseptic/antibiotic vaginal preparations or vaginal douching

**Clinical features**
- Malodorous and increased white vaginal discharge that is homogenous, low in viscosity, and uniformly coats the vaginal walls
- The fishy-smelling discharge is particularly noticeable after sexual intercourse; usually no pruritus or inflamed vulvae

**Differential diagnoses**
- Other causes of vaginal discharge: see Gonorrhoea

**Complications**
- Acute salpingitis
- Premature rupture of membranes
- Preterm delivery and low birth weight

**Investigations**
- Homogeneous milky discharge with pH > 4.5 (pH > 6.0 highly suggestive)
- Fishy odour from the biogenic amines; altered by addition of 10% KOH (Sniff test)
- Clue cells on a wet mount
- Clue cells are normal vaginal epithelial cells studded with bacteria, giving the cells a granular appearance

**Treatment objective**
To eliminate the organisms

**Drug therapy**
- Recommended regimen:
  - Metronidazole 400 mg orally, every 12 hours for 7 days
- Alternative regimen:
  - Metronidazole 2 g orally, as a single dose
- If treatment is imperative in the first trimester of pregnancy:
  - Give metronidazole 2 g orally as a single dose

**Recommended regimen for pregnant women**
- Metronidazole 200 orally, every 8 hours for 7 days, after the first trimester
- 2 g orally, as a single dose
- If treatment is imperative in the first trimester of pregnancy:
  - Give metronidazole 2 g orally as a single dose

**Notable adverse drug reactions, caution and contraindications**
- Metronidazole:
  - Causes a disulfiram-like reaction with alcohol
  - Avoid high doses in pregnancy and breast feeding
  - May cause nausea, vomiting, unpleasant taste, furred
Chapter 10: Genito-Urinary System

Standard Treatment Guidelines for Nigeria 2008

- Patients should therefore be followed up weekly until there is clear evidence of improvement

**Notable adverse drug reactions, caution and contraindications**
- Ciprofloxacin and ceftriaxone (see gonorrhoea)
- Erythromycin and azithromycin (see chlamydia)

**Prevention**
- Counselling, Compliance, Condom use and Contact treatment

**CHLAMYDIAL INFECTION**
(Other than Lymphogranuloma venereum)

**Introduction**
- The chlamydiae occupy a special place between bacteria and viruses
- They are a large group of obligate intracellular organisms
- Chlamydia trachomatis has a number of serovars and causes many different human infections
- Eye: trachoma; inclusion conjunctivitis
- Genital tract: lymphogranuloma venereum, non-gonococcal urethritis, cervicitis, salpingitis
- Respiratory tract: pneumonia
- C. trachomatis immunotypes D - K are isolated in about 50% of cases of non-gonococcal urethritis and cervicitis by appropriate techniques

**Clinical features**
- Infections are asymptomatic, but when an incubation period is determined, it is usually about 10 - 20 days
- If inclusions recur after therapy has been completed, erythromycin treatment should be reinstituted for 2 weeks
- It is important to treat the mother and her sexual partner

**Notable adverse drug reactions, caution and contraindications**
- Doxycycline and tetracycline
- Caution in patients with hepatic impairment, systemic lupus erythematosus and myasthenia gravis
- Antacids, aluminium, calcium, iron, magnesium and zinc salts and milk decrease the absorption of tetracyclines
- Deposition of tetracyclines in growing bones and teeth (by binding to calcium) causes staining and occasionally dental hypoplasia
- Should not be given to children under 12 years, or to pregnant or breast-feeding women
- With the exception of doxycycline and minocycline, tetracyclines may exacerbate renal failure and should not be given to patients with kidney disease
- May cause nausea, vomiting and diarrhea; hypersensitivity reactions. Headache and visual disturbances may indicate benign intracranial hypertension
- Candidal superinfection with prolonged therapy
- Erythromycin estolate is contraindicated during pregnancy because of drug-related hepato-toxicity; only erythromycin base or erythromycin ethylsuccinate should be used.
Littre abscess involving periurethral glands
Paraurethral abscesses
Proximal urethral involvement with frequency and terminal haematuria

Cowper's gland abscess involving the bulbourethral glands, producing a swelling behind the base of the scrotum that can produce a proximal or Cowper's stricture
Prostatitis
Proctitis
Urethral stricture leading to hydroureters and hydronephrosis

Chronic epididymo-orchitis leading to sterility
Contaminated fingers or other fomites can also lead to infection of the eyes- gonococcal conjunctivitis
- Haematogenous spread leading to meningitis, arthritis etc

Different diagnoses
Urethral discharge:
Spermatorrhoea/prostatorrhoea (sexual arousal)
- Trichomonas vaginalis and Candida albicans can also give rise to urethral discharge and balanitis

Ascending infections:
Escherichia coli, a common cause in the infantile male homosexuals
- Oralc 인fections may be transmitted non-sexually following genitourinary infections, surgery and instrumentation (including catheterization)
Scrotal swelling (epididymo-orchitis):
In older males, where there may have been no risk of STIs, other general infections may be responsible, e.g. Mycoplasma spp. or Chlamydia trachomatis
- Occasionally N. Gonorrhoeae reaches the bloodstream causing sepsis

Gonorrhoea in males
Clinical features
Present as foul-smelling urethral discharge of pus with dysuria 2 - 6 days after exposure
Some patients have a scanty discharge that cannot be distinguished from non-gonococcal urethritis
- Often asymptomatic during the day but there may be a drop of discharge in the morning
Urethral orifice is usually inflamed; there may be balanitis because of the irritation from the discharge and secondary infection
- About half of infected males are asymptomatic
- Ascending infection is common and may lead to inflammation of the epididymis (epididymitis)
Cystitis usually manifests by acute onset of unilateral testicular pain and swelling, often with tenderness of the epididymis and vas deferens
- Occasionally there is erythema and oedema of the overlying skin
- The adjacent testis is often also inflamed (orchitis), giving rise to epididymo-orchitis

Complications
Local complications (now uncommon):
Inflammation of the cervix and cervical canal (cervicitis)
is the commonest presentation in women
Urethritis: the urethra becomes the most common site in women who have had hysterectomy

The most frequent complaint is discharge, often accompanied with burning on urination
Over 50% of infected women are asymptomatic
Omphalophlegm gonorrhea from genital sex (fellatio) may present as sore throat

Complications
Local:
Infections of Skene’s periurethral glands and Bartholin's gland glands; a Bartholin's gland abscess may cause pain on sitting or walking

Vulvitis
Ascending infection to the endometrium, fallopian tubes, ovaries and peritoneum (pelvic inflammatory disease)
Ectopic pregnancy
Infertility
Perihepatic abscess (Fitz-Hugh-Curtis syndrome)

Risk of disseminated gonococcal infection during pregnancy and menstruation
- Premature rupture of membranes
- Premature labour
- Chorioamnionitis
- Septic abortion
- Ophthalmia neonatorum
- Oropharyngeal gonorrhoea

Differential diagnoses
Other causes of vaginal discharge:
Ascending infection is common and may lead to

Infections of the cervix and cervical canal (cervicitis)
- Premature rupture of membranes
- Premature labour
- Chorioamnionitis
- Septic abortion
- Ophthalmia neonatorum
- Oropharyngeal gonorrhoea

Differential diagnoses
Other causes of vulvar discharge:
- Premenstrually
- At the time of ovulation
- In pregnancy
- Use of contraceptive pills or an intrauterine device

Infective causes:
- Candidiasis
- Trichomoniasis
- Bacterial vaginosis
- Chlamydia
- Cervical herpes genitalis
- Cervical warts
- Syphilitic chancre
- Toxic shock syndrome (Staphylococcus aureus)
- β-haemolytic streptococcal infection, Mycoplasma infection
- Non-infective causes:
- Cervical ectropion
- Cervical polyp(s)
- Neoplasia e.g. cancer of the cervix
- Retained products (tampon, post-abortion, post-natal)
- Trauma
- Semen (post-coital)
- Contact irritants and sensitizers e.g. from douches or feminine hygiene sprays
- Bullous diseases of the mucous membranes

Investigations
Endocervical swab (through a vaginal speculum) for microscopy, culture and sensitivity

Gonorrhoea in children
Clinical features
Sexual abuse is a common cause of gonorrhoea in young girls
- Usually asymptomatic in young girls
- Pruritus and dysuria are common complaints
- Other infections caused by T. vaginalis, and C. albicans

Intestinal bacteria or pin worms due to inadequate cleaning after defaecation

Ophthalmia neonatorum
Gonococcal conjunctivitis in the neonate can be acquired perinatally
- Pubertal conjunctivitis; the lids swell; eyes are red and tender
- If not treated promptly, the cornea may be eroded and perforated, leading to secondary glaucoma, conophthalmus and blindness
- About 30% of babies infected will also show oropharyngeal gonorrhoea

Differential diagnoses
The silver nitrate prophylaxis can produce a chemical conjunctivitis, usually appearing 6 - 8 hours after treatment and resolving over 24 hours
- E. coli, staphlococci, streptococci, and Pseudomonas spp. can also cause conjunctivitis in the neonate

Treatment objectives
Eliminate the organism in the patient and sexual partner(s)
- Prevent re-infection
- Prevent complications
- Counsel and screen for possible co-infection with HIV so that appropriate management can be instituted

Drug therapy
Recommended regimen:
Ciprofloxacin 500 mg orally, as a single dose
Or:
Ceftriaxone 125 mg by intramuscular injection, as a single dose

Neonatal gonococcal conjunctivitis
Recommended regimen:
Ceftriaxone 50 mg/kg by intramuscular injection, as a single dose, to a maximum of 125 mg
Or:
Spectinomycin 25 mg/kg by intramuscular injection as
Secondary stage

- About 3 - 6 weeks post-contact a uni-or bilateral massive inguinal lymphadenopathy (bubo) appears
- The glands elongate along the Poupart’s ligament to the ligament, so that the depression formed by the ligament which separates these two groups of glands gives the “sign of the groove”
- Pain in the gland is usual, and as the glands are matted, the glands eventually become fluctuant, break down and discharge
- Inguinal lymphadenopathy occurs in only 20 - 30% of women with LGV
- There is primary involvement of the rectum, vagina, cervix, or posterior urethra, which drain to the deep iliac nodes
- Incubation period ranges from 10 - 40 days
- The early lesion is a papule or nodule which soon becomes ulcerated and has an offensive discharge
- Progressive indolent, serpiginous ulceration of the groins, pubis, genitals and anus may form. Pain on walking may be excruciating
- Persistent sinusess and hypertrophic depigmented scars are fairly characteristic
- Regional lymph nodes are not enlarged but with cicatisation, the lymph channels may be blocked causing pseudoelephantiasis of the genitalia
- Both the fibrotic scarring and elephantiasis-like lesion could cause obstructed labour
- Subcutaneous extension and abscesses may occur and form a pseudo-bubo in the inguinal region
- Healing is unlikely without treatment; the locally destructive lesion may eventually involve the groins, pubis and anus
- A squamous cell carcinoma may arise from chronic lesions.

Differential diagnoses

- Syphilis
- Chancroid
- Lymphogranuloma venereum
- Lupus vulgaris
- Deep mycosis
- Amebic ulcer
- Pyoderma gangrenosum
- Squamous cell and basal cell carcinoma

Complications

- Obstructed labour
- Squamous cell carcinoma

Investigations

- Direct microscopy

Treatment objectives

- Same as for gonococcal infection

Drug therapy

Recommended regimen:

- Azithromycin
  - 1 g orally on first day, then 500 mg orally, once a day
  - Doxycycline
  - 100 mg orally every 12 hours
  - Therapy should be continued until the lesions have completely epithelialized

Alternative regimen:

- Erythromycin
  - 500 mg orally every 6 hours
  - Tetracycline 500 mg orally every 6 hours

Men who are at risk of HIV infection should receive additional antibiotic treatment (as those with clinical neonatal conjunctivitis)

PREVENTION

- Counselling, Compliance, Condom use and Contact treatment

LYMPHOGRANULOMA VENEREUM

(CLINICAL BOBO; LYMPHOGRANULOMA INGUINALE; LYMPHOPATHIA VENERA; DURAND-NICOLAS-FAVRE DISEASE)

Introduction

A chronic disease caused by Chlamydia trachomatis (serotypes L1, L2, L3), an obligate intracellular microorganism

- Most common in Asia, Africa, and South America
- In Europe and North America, it is most prevalent among homosexuals, immigrants from endemic areas and people returning from endemic areas, such as soldiers, seamen, and vacationers

Clinical features

- A chronic granulomatous, locally destructive disease that is characterized by progressive, indolent, serpiginous ulceration of the groins, pubes, genitals and anus
- May be classified into primary, secondary, and late stages

Primary stage

- After an incubation period of 7 - 15 days, a papule or small non-indurated painless ulcer appears
- Usually goes unnoticed
- Extra-genital lesions (rectal, oral) have also been described
- Women probably act as asymptomatic carriers
- Patients are very rarely seen at the primary stage

Secondary stage

- About 3 - 6 weeks post-contact a uni-or bilateral massive inguinal lymphadenopathy (bubo) appears
- The glands elongate along the Poupart’s ligament to the ligament, so that the depression formed by the ligament which separates these two groups of glands gives the “sign of the groove”
- Pain in the gland is usual, and as the glands are matted, the glands eventually become fluctuant, break down and discharge
- Inguinal lymphadenopathy occurs in only 20 - 30% of women with LGV
- There is primary involvement of the rectum, vagina, cervix, or posterior urethra, which drain to the deep iliac nodes
- Incubation period ranges from 10 - 40 days
- The early lesion is a papule or nodule which soon becomes ulcerated and has an offensive discharge
- Progressive indolent, serpiginous ulceration of the groins, pubis, genitals and anus may form. Pain on walking may be excruciating
- Persistent sinusess and hypertrophic depigmented scars are fairly characteristic
- Regional lymph nodes are not enlarged but with cicatisation, the lymph channels may be blocked causing pseudoelephantiasis of the genitalia
- Both the fibrotic scarring and elephantiasis-like lesion could cause obstructed labour
- Subcutaneous extension and abscesses may occur and form a pseudo-bubo in the inguinal region
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- A squamous cell carcinoma may arise from chronic lesions.

Differential diagnoses

- Syphilis
- Chancroid
- Lymphogranuloma venereum
- Lupus vulgaris
- Deep mycosis
- Amebic ulcer
- Pyoderma gangrenosum
- Squamous cell and basal cell carcinoma

Complications

- Obstructed labour
- Squamous cell carcinoma

Investigations

- Direct microscopy

Treatment objectives

- Same as for gonococcal infection

Drug therapy

Recommended regimen:

- Azithromycin
  - 1 g orally on first day, then 500 mg orally, once a day
  - Or: Doxycycline
  - 100 mg orally every 12 hours
  - Therapy should be continued until the lesions have completely epithelialized

Alternative regimen:

- Erythromycin
  - 500 mg orally every 6 hours
  - Or: Tetracycline 500 mg orally every 6 hours

Men who are at risk of HIV infection should receive additional antibiotic treatment (as those with clinical neonatal conjunctivitis)
Vaginal discharge: a white-yellow frothy discharge is characteristic
Counsel and screen for possible co-infection with HIV
Burning sensation
Dysuria
Dyspareunia
The liabia are often swollen
The cervix may have punctuated haemorrhages producing a strawberry-like surface when viewed with a colposcope
Some men may have dysuria or a minimal urethral discharge
Other causes of vaginal discharge or urethral discharge:
- Squamous cell or basal cell carcinoma
- Fixed drug eruption
- Behcet's disease
- Chancroid
- Herpes
- Herpes zoster
- Lymphogranuloma venereum
- Granuloma inguinale
- Trichomoniasis
- Syphilis
- Acute salpingitis
- Chancres may also be located on the lips or tongue;
- Other causes of genital ulcers:
- Atypical lesions may be seen for various reasons e.g.
- Early syphilis: primary, secondary and early latent stages
- Primary syphilis is characterized by an ulcer or chancre at the site of infection or inoculation
- Manifestations of secondary syphilis include a skin rash, condyloma lata, mucocutaneous lesions and generalized lymphadenopathy
- Secondary syphilis: skin rash, condyloma lata, mucocutaneous lesions and generalized lymphadenopathy
- Late syphilis: late latent syphilis, gummatous, neurological and cardiovascular syphilis
- This section is only on primary syphilis
- Clinical features
- After an incubation period of 2 - 4 weeks (full range 90 days) the first lesion of syphilis may appear at the site of exposure, most commonly, the genitals
- Chancres may also be located on the lips or tongue; anorectal chancres frequently seen in male homosexuals
- Begins as a small, dusky-red macule which soon develops into a papule
- The surface of the papule erodes to form an ulcer which is typically round and painless with a clean surface and exudes a scanty yellow serous discharge teeming with spirochaetes
- Lesion is indurated and feels firm or hard on palpation; surrounding skin is oedematous
- Regional inguinial (or generalized) lymphadenopathy follows
- The glans are painless, moderately enlarged (not bobbles), discrete and never suppurate
- Atypical lesions may be seen for various reasons e.g.
- Early syphilis: primary, secondary and early latent stages
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- Early syphilis: primary, secondary and early latent stages
- Primary syphilis: an ulcer or chancre at the site of infection or inoculation

**Drug treatment**

Recommended regimen:
- Doxycycline
  - 100 mg orally, every 12 hours for 14 days
  Or:
  - Erythromycin
  - 500 mg orally every 6 hours for 14 days
  Alternative regimen:
  - Tetracycline 500 mg orally, every 6 hours for 14 days

**Adjuvant measures**

Aspirate fluctuant lymph nodes through healthy skin incision and drainage or excision of nodes may delay healing and is not recommended
Some patients with advanced disease may require treatment for longer than 14 days, and sequelae such as strictures and/or fistula may require surgery

**Notable adverse drug reactions, caution and contraindications**

See Chlamydia

**Prevention**

Counselling, Compliance, Condom use and Contact treatment

**SYPHILIS**

- Introduction
  - Infection caused by the spirochaete Treponema pallidum
  - Occurs worldwide
  - Can be classified as:
    - Congenital (transmitted from mother to child in utero)
    - Acquired (through sex or blood transfusion)
  - Acquired syphilis may be early or late
  - Primary syphilis is characterized by an ulcer or chancre at the site of infection or inoculation
  - Manifestations of secondary syphilis include a skin rash, condyloma lata, mucocutaneous lesions and generalized lymphadenopathy
  - Early syphilis: primary, secondary and early latent stages
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        - Early syphilis: primary, secondary and early latent stages
        - Primary syphilis: an ulcer or chancre at the site of infection or inoculation

**Investigations**

Dark field examination and direct fluorescent antibody tests of lesion exudates or tissue
VDRL; RPR

**Treatment objectives**

- Eliminate the organism in the patient and sexual partner(s)
- Prevent re-infection

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**TRICHOMEONIASIS**

**Introduction**

- Caused by the flagellated protozoan, Trichomonas vaginalis
- An extremely common infection, almost always transmitted via sexual contact
- Women are far more frequently affected and more likely to have symptoms
- Men are more likely to be asymptomatic and serve as carriers

**Clinical features**

Vaginal discharge: a white-yellow frothy discharge is characteristic
Burning sensation
Dysuria
Dyspareunia
The liabia are often swollen
The cervix may have punctuated haemorrhages producing a strawberry-like surface when viewed with a colposcope
Some men may have dysuria or a minimal urethral discharge and balanoposthitis
Co-infection with N. gonorrhoeae is common

**Differential diagnoses**

Other causes of vaginal discharge or urethral discharge:
- Gonorrhea
- Acute salpingitis
- Adverse pregnancy outcomes, particularly premature rupture of membranes, pre-term delivery and low birth weight

**Investigations**

Microscopy and culture of vaginal discharge

**Treatment objectives**

Eliminate the organism in the patient and sexual partner(s)
- Prevent re-infection
- Prevent complications
- Counsel and screen for possible co-infection with HIV

**Drug treatment**

Recommended regimen:
- Tinidazole
  - 2 g orally in a single dose
  Or:
  - Metronidazole
    - 400 mg or 500 mg orally every 12 hours for 7 days
  Or:
  - Tinidazole
    - 500 mg orally every 12 hours for 5 days

**Note**

Other 5-nitroimidazoles are also effective, both in single and in multiple dose regimens
Clinical examination:
- Vulval erythema (redness) or excoriations from scratching
- Vulval oedema
- Erosions and crusting on the adjacent intertriginous skin

Although treatment of sexual partners is not recommended, it may be considered for women who have recurrent infections
- A minority of male partners may have balanitis, which is characterized by erythema of the glans penis or inflammation of the glans penis and foreskin (balanoposthitis)

Differential diagnoses
- Other causes of vaginal discharge: see Gonorrhoea in women

Complications
- Emotional problems because of the recurrent nature of the infection, and dyspareunia
- Very serious emotional problems in a non-sexually active person wrongly “accused” by parents, spouse or health care providers

Treatment objectives
- Cure the infection
- Prevent recurrence

Drug therapy

Recommended regimen for balanoposthitis
- 5 mg/kg orally, every 8 hours for 5 days
- Avoid high doses in pregnancy and breast feeding
- May cause nausea, vomiting, unpleasant taste, furred tongue, and gastro-intestinal disturbances
- Generally not recommended for use in the first trimester of pregnancy

Prevention
- Counselling, Compliance, Condom use and Contact treatment

VULVO-VAGINAL CANDIDIASIS

Introduction
- Inflammation of the vagina and vulva, usually evolving from vaginal discharge and secondary external irritation

Candida albicans is the commonest cause of candidal vulvo-vaginitis; Candida glabrata has also been identified

Candidal vaginitis is most common in:
- Pregnancy
- Patients with diabetes mellitus
- Those on long-term antibiotic therapy or oral contraceptives
- Conditions associated with immunosuppression
- Corticosteroid use

Usually not acquired through sexual intercourse
- Because of the close proximity between the anus and female genitalia, re-infections may occur from the gastrointestinal tract

Clinical features
- Up to 20% of women with the infection may be asymptomatic
- If symptoms occur, they usually consist of vulval itching, soreness and a non-offensive vaginal discharge which may be curdy
Chapter 10: Genito-Urinary System

Standard Treatment Guidelines for Nigeria 2008

Androgen replacement in those with androgen deficiency:
- Testosterone enanthate
  - 250 mg intramuscularly every 2-4 weeks
Or:
- Oral methyl testosterone or fluoxymesterone
  - 120 - 160 mg daily for 2 - 3 weeks; maintenance 40 - 120 mg daily
Intra-corporal administration of:
  - Prostaglandin E
    - 5 - 15 microgram
  - Sildenafil citrate
    - 25 - 100 mg one hour before intercourse

Notable adverse drug reactions, caution and contraindications
- Androgens:
  - Not to be given to patients with prostate carcinoma
  - Phosphodiesterase inhibitors:
    - Altered vision, headache, dizziness and nasal congestion
  - Contraindicated in patients taking nitrates
  - Should be used with caution in patients with ischaemic heart disease

MALE INFERTILITY

Introduction
Failure to achieve conception after one year of regular, unprotected sexual intercourse in a couple trying to achieve pregnancy

Primary:
- When the man has never impregnated a woman
Secondary:
- When the man had impregnated a woman in the past

Male factor is responsible for about 50% of infertile unions

Clinical features
- Vital points in the history:
  - Duration of infertility
  - Ability to have erection, penetration and ejaculation
  - Family history of infertility
  - History of systemic disease e.g. diabetes mellitus, hypertension, chronic liver disease and tuberculosis
  - History of sexually transmitted infections and urinary tract infections
  - History of genital trauma
  - History of surgery: herniorrhaphy, orchidopexy, urethral surgeries, etc

Examination:
- Gynaecomastia
- Penile epispadias, hypospadias, penile deformities
- Scrotum: absence of testis, small sized testis, varicoceles, hard and irregular epididymis

Investigations
- Semen analysis x 3

POSTERIOR URETHRAL VALVES

Introduction
Congenital mucosal folds situated in the prostatic/membranous urethra, causing urine outflow obstruction

- Occurs in males
- The most common mechanical cause of renal deterioration in children

Clinical features
- Obstructive urinary symptoms
- Urethral retention
- Failure to thrive
- Distended bladder with palpable kidneys

Differential diagnoses
- Anterior urethral valves
- Congenital bladder neck hypertrophy
- Congenital urethral stricture
- Meatal stenosis
- Posterior urethral polyp

Complications
- Recurrent upper tract infections
- Septicaemia
- Bladder dysfunction
- Bladder stones
- Hydroureter/hydronephrosis
- Progressive renal impairment
- Failure to thrive

Investigations
- Urinalysis
- Urine microscopy, culture and sensitivity
- Full Blood Count
- Serum Urea, Electrolytes and Creatinine
- Abdominal ultrasound
Supportive measures
- Adequate hydration
- Pain relief

Prevention
- Avoid causative drugs

PROSTATITIS
Introduction
- An inflammation of the prostate or pain in the prostate, similar to that caused by an inflammation.
- Classified into:
  - Acute bacterial prostatitis
  - Chronic bacterial prostatitis
  - Chronic non-bacterial prostatitis

Risk factors:
- Ductile reflux
- Urinary tract infection
- Indwelling urethral catheterization
- Penetrating anal sex
- Sexually transmitted infections

Acute bacterial prostatitis
- Reckless from direct spread of ascending urethral infection or reflux of infected urine into the prostatic ducts
- E. coli is the main causative organism. Others are klebsiella, pseudomonas, Streptococcus faecalis and Staph aureus

Chronic bacterial prostatitis
- Caused by E. coli, Klebsiella, Mycoplasma and Chlamydia

Non-bacterial prostatitis
- An inflammation of indeterminate cause

Acute prostatitis
- Systemic features
  - Fever
  - Chills
  - Malaise
  - Nausea
- Local features
  - Dysuria
  - Frequency
  - Haematuria
  - Urethral discharge

Rectal examination:
- Hot bogy, swollen and very tender prostate

Chronic prostatitis
- Voiding symptoms: dysuria, frequency, urgency, haematuria
- Poor stream
- Urethral discharge
- Low back pain

Management
- To eradicate causative organisms
- Control pain

Drug treatment
- Antibiotics (based on local sensitivity)
  - Ciprofloxacin 500 mg orally every 12 hours for 28 days
  - Cotrimoxazole 960 mg orally every 12 hours for 28 days
- Non-steroidal e.g. diclofenac, ibuprofen etc
- Steroids e.g. prednisolone, dexamethasone
- alpha blockers e.g. prazosin, doxazosin
- Hormonal therapy e.g. finasteride, cyproterone

Prostatic abscess
- Surgical removal (for treatment of other conditions)

Non-drug treatment
- Prostatic massage (chronic prostatitis only)
- Physiotherapy
- Sitz baths

SCROTAL MASSES
The empty scrotum
Introduction
- A clinical situation in which the testis is absent from the scrotum
- May be bilateral or unilateral

Causes include:
- Undescended testis
- Ectopic testis
- Retractile testis

Absence (vanishing) testis

Clinical features
- Pain in one testicle: of sudden onset, severe in intensity, and radiates to the lower abdomen
- Nausea and vomiting
- Swollen, high lying testis with reddening of the scrotal skin

Tenderness. Pain can be increased by lifting the testicle up
- Absence of cremasteric reflex
- Abnormal lie of the testis on the opposite side
Chapter 10: Genito-Urinary System

Dribbling
Examination of the external genitalia may reveal:
- Urethral indurations
- Periurethral or perineal abscesses
- Urinary fistula

Differential diagnoses
- Benign prostatic hypertrophy
- Bladder calculi
- Bladder neck stenosis

Complications
- Urinary tract infections
- Urethral/bladder calculi
- Urinary retention
- Fournier's gangrene
- Perineal urinary fistulae
- Progressive renal failure

Investigations
- Urinalysis
- Urine microscopy, culture and sensitivity
- Urethroscopy
- Urethrogram
- Uroflowmetry
- Abdominal ultrasound
- Serum Urea, Electrolytes and Creatinine
- Full Blood Count

Treatment objectives
- To restore urethral patency

Differential diagnoses
- Acute pyelonephritis
- Renal and ureteric stones:
- Sudden onset loin pain radiating to the groin
- Haematuria
- Nausea and vomiting
- Stones in the bladder:
- Frequency
- Urgency
- Difficulty in passing urine
- Stones in the urethra:
- Urethral fistulae

Investigations
- Urinalysis
- Urine microscopy, culture and sensitivity
- Urethroscopy
- Urethrogram
- Uroflowmetry
- Abdominal ultrasound
- Serum Urea, Electrolytes and Creatinine
- Full Blood Count

Prevention
- Relieve symptoms
- Remove stones
- Prevent recurrence

Non-drug treatment
- Potassium citrate
- Thiazide diuretics

Notable adverse drug reactions, caution
- Nausea, epigastric pain, pruritus, headache, dizziness

Prevention
- Provision of and access to pipe-borne water
- Improvement in socio-economic conditions
- Mass chemotherapy in endemic areas
- Eradicating the intermediate hosts (water snails)

URINARY SCHISTOSOMIASIS
Introduction
A common parasitic infection of the urinary tract caused by a body fluke, Schistosoma haematobium

Clinical features
- Dysuria
- Frequency
- Urgency
- Poor stream
- Straining
- Hesitancy

Complications
- Acute voiding symptoms
- Chronic infection

Clinical features
- Acute symptoms
- Chronic alteration

Investigations
- Urinalysis
- Urine microscopy, culture and sensitivity
- Urine culture
- Serum calcium, phosphate and albumin
- Intravenous urography (IVU)
- Ultrasonography
- Computerized tomography (non-contrast enhanced)

Treatment objectives
- Relieve symptoms
- Remove stones
- Prevent recurrence

Non-drug treatment
- Potassium citrate
- Thiazide diuretics

Notable adverse drug reactions, caution
- Nausea, epigastric pain, pruritus, headache, dizziness

Prevention
- Provision of and access to pipe-borne water
- Improvement in socio-economic conditions
- Mass chemotherapy in endemic areas
- Eradicating the intermediate hosts (water snails)

URINARY TRACT CALCULI
Introduction
Occurrence of stone(s) in the kidney, ureter, bladder or urethra

Clinical features
- Occurrence of stone(s) in the kidney, ureter, bladder or urethra
- Incidence in Nigeria is 7 - 34 per 100,000
- Stones are different with respect to their composition
- Oxalate stones, phosphate stones, uric acid stones and cystine stones
- Factors promoting stone formation:
Chapter 11: Infectious Diseases/Infestations

FEVERS: MANAGEMENT APPROACH

Introduction
A leading cause for seeking medical care

In health, temperature is controlled within limits (in adults at a mean of 36.8 °C) with diurnal variations of about 0.5 °C

‘Fever’ is elevation of body temperature that exceeds the normal daily variation and occurs in conjunction with an increase in hypothalamic set point

In children younger than 5 years of age:
- Arectal temperature greater than 38 °C
- Oral temperature above 37.8 °C
- Axillary temperature above 37.2 °C

Important points in the history are:
- Chronology of symptoms
- Occupational history
- Travel history
- Geographic region
- Family history

Physical examination:
- Vital signs (axillary temperatures are unreliable)
- Skin, lymph nodes, eyes, nail beds, CNS, chest, abdomen, cardiovascular, musculo-skeletal and nervous systems
- Rectal examination is imperative
- The penis, prostate, scrotum and testes (for men)
- Pelvic examination (for women)

Investigations
The number of investigations will depend on the clinical circumstances. On occasions, patients may need to be extensively investigated

General:
- Full Blood Count
- Differential white blood cell count
- Urinalysis with examination of the urinary sediment
- Examination of any abnormal fluid collection

Microbiology:
- Smears and culture of specimens from the throat, urethra, anus, cervix, and vagina (as indicated)
- Sputum smears; culture
- Blood culture
- Urine microscopy, culture and sensitivity
- Cerebrospinal fluid examination

Abnormal fluid collection: specimens for microscopy, culture and sensitivity testing

Cerebrospinal fluid examination

FOOD POISONING

Introduction
A spectrum of disorders arising from:
- Infections acquired by eating contaminated food
- Clinical problems that result from eating food contaminated with toxins
- Clinical sequelae from inherently poisonous animals, plants or mushrooms
- Clinical forms:
  - Staphylococcal food poisoning:

Other investigations as may be indicated in the clinical circumstances

Complications
- Heat stroke in adults
- Febrile convulsions in children
- Complications associated with underlying cause(s) of fever

Treatment objectives
- To lower the temperature
- To treat underlying causes

Non-drug treatment
- Tepid sponging
- Liberal oral sips of water (if clinical state is not a contraindication)

Drug treatment
- Paracetamol:
  - Adult: 500 mg - 1 g orally every 4 - 6 hours; maximum 4 g daily
  - Child: 3 months - 1 year: 60 - 125 mg; 1 - 5 years: 120 - 250 mg; 6 - 12 years: 250 - 500 mg: repeated every 4 - 6 hours if necessary to a maximum of 4 doses in 24 hours
- Infants under 3 months should not be given paracetamol unless advised by a doctor
- Aspirin: (acetylsalicylic acid)
  - Adult: 300 - 900 mg orally (with or without food) very 4 - 6 hours if necessary; maximum 4 g daily
  - Treat the identified (or suspected) cause of fever
- Child: under 16 years, not recommended because of the risk of Reye's syndrome

Notable adverse drug reactions, caution
- Paracetamol:
  - Liver damage (and less frequently, renal damage) following over dosage
- Aspirin:
  - Gastrointestinal discomfort, nausea
  - Ulceration with occult bleeding
  - Hearing disturbances such as tinnitus (rarely deafness)
  - Use with caution in the following clinical conditions:
    - Asthma
    - Allergic disease
    - Impaired renal or hepatic function
    - Pregnancy
    - Breastfeeding
    - Elderly
- Dehydration

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- Food is contaminated by S. aureus when prepared unhygienically by individuals who are carriers
- Subsequent growth of S. aureus in the food and enterotoxin production occurs if the food is not cooked at temperatures sufficient to kill the bacteria, or is not refrigerated
- Food-borne botulism
- Non-typhoidal Salmonellosis
- Shigellosis
- E. coli food poisoning
- Campylobacter food poisoning
- Listeria monocytogenes food poisoning
- Yersinia enterocolitica food poisoning
- Norwalk virus food poisoning
- Hepatitis A virus food poisoning
- Giardiasis
- Helminthic parasitic food poisoning

Clinical features
- Staphylococcal food poisoning:
  - Nausea
  - Diarrhoea 2 - 6 hours after eating food contaminated by enterotoxin
- Food-borne botulism:
  - Incubation period is 18 - 36 hours, but depending on toxin dose, can extend from a few hours to several days
- Symmetric descending paralysis
- Dysarthria/dysphagia
- Nausea, vomiting and abdominal pain may precede or follow the onset of paralysis
- Non-typhoidal Salmonellosis:
  - Diarrhoea
  - Nausea
  - Vomiting
  - Abdominal cramps
  - Fever
  - Headache
  - Myalgia
  - Shigellosis:
  - Fever
  - Self-limiting watery diarrhoea
  - Bloody diarrhoea
  - Dysentry
  - Frequent passage, 10 - 30 times/day of small volume stools containing blood, mucus and pus
  - Abdominal cramps
  - Tenesmus
  - Campylobacter food poisoning:
    - A prodrome with fever, headache, myalgia and/or malaise
    - 12 - 48 hours later:
      - Diarrhoea and abdominal pain
      - E. coli food poisoning
      - Oral rehydration therapy accompanied by cramps
      - L. monocytogenes food poisoning:
        - Common source of outbreaks of acute gastritis

Not a major cause of sporadic diarrhoea
- Norwalk virus food poisoning:
  - Abrupt onset of nausea and abdominal cramps followed by vomiting and/or diarrhoea
- Hepatitis A virus food poisoning:
  - May cause large outbreaks of diarrhoea and vomiting from contaminated food, water, milk and shellfish
- Intrafamily and intranstitutional spread common

Differential diagnoses
Other causes of acute onset diarrhoea, nausea, abdominal cramps and vomiting with or without systemic manifestations

Complications
- Fluid and electrolyte derangements
- Others
- By no means limited to the stated organisms

Shigellosis:
- Dehydration
- Rectal prolapse
- Protein-losing enteropathy
- Malnutrition
- Haemolytic-uraemic syndrome
- Toxic megacolon
- Perforation
- Campylobacter food poisoning:
  - Bacteremia
  - Cholecystitis
  - Pancreatitis
  - Cystitis
  - Meningitis
  - Endocarditis
  - Arthritis
  - Peritonitis
  - Cellulitis
  - Septic abortion

Treatment objectives
- Restore fluid and electrolyte balance
- Neutralize toxin
- Eradicate microbe

Non-drug measures
- Gastric lavage in food-borne botulism

Drug treatment
- Appropriate fluid and electrolyte replacement
- Trivalent (types A, B, and E) equine anti-toxin should be administered as soon as possible after specimens are obtained for laboratory analysis for food-borne botulism
- Emetics in food-borne botulism
- Administer appropriate medicines

Shigellosis
- Oral Rehydration Therapy Plus:
  - Adult: Amoxicillin 50 - 100 mg/day orally every 8
HELMINTHIASIS

Introduction
Parasitic worm infestations can arise from different groups:

- **Nematodes (round worms)**
- **Ascaris**
- **Enterobius (pinworm)**
- **Trichuris (whipworm)**
- **Cestodes (flat worms/tapeworms)**

**Round worm infestations are associated with rural living and poor hygiene**

- Prevalent among school children and young adults
- Acquired through soil and fecal-oral contamination
- Flat worms and tape worms are acquired by eating under-cooked contaminated meat or fish
- Bladder worms (S. haematobium) are acquired by wading through streams and ponds contaminated with the vector snails

Clinical features
- **Ascaris**
  - Lung phase: Irritating, non-productive cough
  - Burning substernal discomfort, aggravated by coughing or deep inspiration
  - Dyspnoea
  - Blood-tined sputum
- Intestinal phase:
  - Usually no symptoms
  - Pain
  - Features of small bowel obstruction
  - Features of perforation
  - Intussusception
  - Volvulus
- Biliary tree occlusion: biliary colic, cholecystitis, cholangitis, pancreatitis, intrahepatic abscesses

Effects of migration of an adult worm up the oesophagus:
- Coughing
- Oral expulsion of the worm

**Hookworm**
- Most are asymptomatic
- Maculo-papular dermatitis
- Mild transient pneumonitis
- Epigastric pain, often with post-prandial accentuation
- Diarrhoea
- Weakness
- Shortness of breath
- Skin depigmentation

Perianal pruritus, worse at night owing to the nocturnal migration of the female worms
- Skin excoriation and bacterial superinfection

**Hookworm**
- Adult: 1 g intravenously slowly
- Child: 50 mg/kg/d 1 intravenously for 5 days
- E.coli food poisoning
  - Fluid and electrolyte replacement
  - +: Gentamicin
  - -: Amoxicillin

Prevention
- Appropriate environmental and personal hygiene
  - Use of sanitaire latrines or toilets
  - Decontamination of water supplies
  - - Use of sanitary latrines or toilets
  - - Hand washing with soap and water
  - - Decontamination of water supplies
  - - Use of sanitary latrines or toilets
  - Identify and treat chronic carriers among food handlers
  - Hygienic preparation and storage of food
  - Ensure that food is cooked at temperatures sufficient to kill bacteria
- Refrigerate food whenever possible
- Encourage exclusive breastfeeding
- Encourage measures to reduce the burden of malnutrition (with its attendant predisposition to severe infections)
- Administer a pentavalent vaccine (A, B, C, D, and E) for persons at high of botulism
- Report new cases to public health authorities

Standard Treatment Guidelines for Nigeria 2008

<table>
<thead>
<tr>
<th>Drug Treatment</th>
<th>Adult and child: 100 mg orally every 12 hours for 3 days</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mebendazole</td>
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<tr>
<td>Praziquantel</td>
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</tr>
</tbody>
</table>

**E. coli food poisoning**
- Fluid and electrolyte replacement
- +: Gentamicin
- -: Amoxicillin

**Prevention**
- Appropriate environmental and personal hygiene
  - - Use of sanitaire latrines or toilets
  - - Hand washing with soap and water
  - - Decontamination of water supplies
  - - Use of sanitary latrines or toilets
  - - Identify and treat chronic carriers among food handlers
  - Hygienic preparation and storage of food
  - - Ensure that food is cooked at temperatures sufficient to kill bacteria
- Refrigerate food whenever possible
- Encourage exclusive breastfeeding
- Encourage measures to reduce the burden of malnutrition (with its attendant predisposition to severe infections)
- Administer a pentavalent vaccine (A, B, C, D, and E) for persons at high of botulism
- Report new cases to public health authorities
**Prevention**
- Good personal and food hygiene
- Access to safe and potable water
- Regular deworming
- Adequate cooking of food and meats

**HUMAN IMMUNODEFICIENCY VIRUS INFECTION**

**Introduction**
Human Immunodeficiency Virus (HIV) is a retrovirus which infects primarily CD4 T cells (T helper cells)
Infection leads to a progressive destruction of the immune system with a consequent myriad of opportunistic infections and the development of certain malignancies

Acquired Immuno Deficiency Syndrome (AIDS) is defined as the presence of an AIDS-defining illness (see table 1) with a positive antibody test for HIV

**HIV transmission**
Sexual transmission through vaginal and anal sex is the commonest route globally and in Nigeria, accounting for about 80%
Transfusion of infected blood and blood products
Use of contaminated instruments; sharing needles, tattooing and occupational exposures
Mother-to-child transmission of HIV: from an infected mother to her baby during pregnancy, at delivery and, after birth through breast-feeding

**Clinical features**
Transient early acute symptoms: commonly “flu” -like illness, often not recognized in the first 2 - 3 weeks of HIV infection:
- Generalized lymphadenopathy
- Sore throat
- Fever
- Skin rash

Asymptomatic period:
The individual feels well despite on-going viral replication

**Initial symptoms:**
- Generalized lymphadenopathy
- Wasting syndrome/fever/night sweats
- Neurologic disease
- Early immune failure
- Oral thrush
- Herpes zoster
- Hairy leukoplakia
- AIDS (opportunistic infections)
- Recurrent bacterial pneumonias
- Pulmonary and extrapulmonary tuberculosis
- Pneumocystis carinii infection
- Viral infections including cytomegalovirus
- Other protozoan infections including cryptosporidium, cryptococcosis.
- Systemic fungal infections
- Other cancers (lymphomas, cervical cancer, etc.)

**Staging of HIV/AIDS**

**WHO Staging System for HIV Infection and Disease in Adults and Adolescents**

**Clinical Stage I:**
- Asymptomatic
- Generalised lymphadenopathy
- Performance scale 1: asymptomatic, normal activity

**Clinical Stage II:**
- Weight loss < 10% of body weight
- Minor mucocutaneous manifestations (seborrhoic dermatitis, prurigo, fungal nail infections, recurrent oral ulcerations, angular cheilitis)
- Herpes zoster within the last five years
- Recurrent upper respiratory tract infections (i.e. bacterial sinusitis)
- And/or performance scale 2: symptomatic, normal activity

**Clinical Stage III:**
- Weight loss > 10% of body weight
- Unexplained chronic diarrhoea, > 1 month
- Unexplained prolonged fever (intermittent or constant) > 1 month
- Oral candidiasis (thrush)
- Oral hairy leucoplakia
- Pulmonary tuberculosis within the past year
- Severe bacterial infections (i.e. pneumonia, pyomysitis)
- And/or performance scale 3: bedridden < 50% of the day during last month

**Clinical Stage IV:**
- HIV wasting syndrome
- Pneumocystis carinii pneumonia
- Toxoplasmosis of the brain
- Cryptosporidiosis with diarrhoea > 1 month
- Cryptococcosis, extrapulmonary
- Cytomegalovirus disease of an organ other than liver, spleen or lymph node (e.g. retinitis)
- Herpes simplex virus infection, mucocutaneous (> 1 month) or visceral
- Progressive multifocal leucoencephalopathy
- Any disseminated endemic mycosis
- Candidiasis of oesophagus, trachea, bronchi
- Atypical mycobacteriosis, disseminated or lungs
- Non-typhoid salmonella septicemia
- Extrapulmonary tuberculosis
- Lymphoma
- Kaposi sarcoma
- HIV encephalopathy
- And/or performance scale 4: bedridden > 50% of the day during last month
- Weight loss of > 10% plus either unexplained chronic diarrhoea > 1 month, or chronic weakness and unexplained prolonged fever > 1 month.

**WHO Improved Clinical Staging**

<table>
<thead>
<tr>
<th>Laboratory indices</th>
<th>Clinical stage</th>
</tr>
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<tbody>
<tr>
<td>Lymphocytes</td>
<td>CD4</td>
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<tr>
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<td>Stage 1</td>
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<td>Stage 2</td>
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<td>Stage 3</td>
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<td>Stage 4</td>
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<table>
<thead>
<tr>
<th>CDC classification</th>
<th>Stage A</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Asym. PGL</td>
</tr>
<tr>
<td>Stage C</td>
<td>AIDS indicator condition</td>
</tr>
<tr>
<td>Stage B</td>
<td>Symp. not A or C</td>
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<tr>
<td>Stage A</td>
<td>PGL</td>
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<td>&lt; 200</td>
<td>A3</td>
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<td>B3</td>
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<td>C3</td>
</tr>
</tbody>
</table>

**Differential diagnoses**
- Tuberculosis
- Malignancies
- Diabetes mellitus
- Other wasting syndromes
Non-infectious complications
myopathy, aseptic meningitis

Infectious complications
Wasting, peripheral neuropathy, progressive polyradiculopathy, HIV-associated dementia, cardiomyopathy

CD4 count (cells/mm$^3$)

<table>
<thead>
<tr>
<th>CD4 count (cells/mm$^3$)</th>
<th>Infectious complications</th>
<th>Non-infectious complications</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt; 500</td>
<td>Acute HIV, candid vaginitis</td>
<td>PGL, Guillain-Barre syndrome, myopathy, aseptic meningitis</td>
</tr>
<tr>
<td>200 - 500</td>
<td>Pneumococcal and other bacterial pneumonias, pulmonary TB, Herpes zoster, oropharyngeal candidiasis, oral hairy leukoplakia, Kaposi sarcoma</td>
<td>Cervical cancer, anaemia, lymphomas</td>
</tr>
<tr>
<td>&lt; 200</td>
<td>Milliary/extrapulmonary TB, pneumocystis carinii pneumonia (PCP), disseminated histoplasmosis and coccidiodomycosis, progressive multifocal leukoencephalopathy (PML)</td>
<td>Wasting, peripheral neuropathy, progressive polyradiculopathy, HIV-associated dementia, cardiomyopathy</td>
</tr>
<tr>
<td>&lt; 100</td>
<td>Disseminated herpes simplex, toxoplasmosis, cryptococcosis, cryptosporidiosis, chronic microsporidiosis, and oesophageal candidiasis</td>
<td></td>
</tr>
<tr>
<td>&lt; 50</td>
<td>Disseminated cytomegalovirus (CMV), disseminated Mycobacterium avium complex (MAC)</td>
<td>Central nervous system lymphomas</td>
</tr>
</tbody>
</table>

Complications
Investigations
Full Blood Count and differentials
VDRL or RPR
Tuberculin test (PPD)
Sputum smears for TB
Electrolytes, Urea and Creatinine
Blood glucose
Liver function tests
Lipid studies (fasting triglycerides, LDL, HDL)
HBV, HCV serology
Cervical (PAP) smears
CD4 T cell counts
HIV RNA level (viral load)
HIV DNA (paediatric diagnosis <18 months of age)
Genotype and phenotype assays for resistance testing

Treatment objectives
Clinical: prevent disease progression
Immunological: restore immunity
Virological: control or suppress viral replication
Public health: reduce infectivity

Criteria for initiating ART based on Nigerian ART guidelines
Adults and Adolescents
Initiation of therapy depends on availability of CD4 cell count testing
IFCD4 testing is available:
WHO Stage IV disease irrespective of CD4 cell count
WHO Stage III disease with CD4 cell count < 350/mm$^3$
WHO Stage I or II disease with CD4 cell counts ≥ 200/mm$^3$
IFCD4 testing is unavailable:
WHO Stage IV disease irrespective of total lymphocyte count (TLC)
WHO Stage III disease irrespective of TLC
WHO Stage II disease with a TLC ≥ 1200/mm$^3$
ATLC of ≥ 1200/mm$^3$ does not predict a CD4 cell count of ≥ 200/mm$^3$ in asymptomatic patients
TLC of ≥ 1200/mm$^3$ may not be used as criterion for the initiation of therapy in asymptomatic patients (WHO Stage I disease)

Children
Children are monitored using CD4 percentage (CD4 %) i.e. percentage of lymphocytes that are CD4 cells
CD4% of an HIV-negative child is around 40%
Diagnosis depends on the age of the child and availability of virological testing
Children < 18 months
Seroepidemiological diagnosis is unreliable as maternally-derived antibodies may persist for up to 15 - 18 months

Diagnosis of HIV has to be made by identifying HIV DNA using PCR
HIV-seropositive children aged <18 months
If HIV status is virologically-proven ART is recommended when the child has:
WHO Paediatric Stage III disease irrespective of CD4% WHO Paediatric Stage II disease, with consideration of using CD4 <20% to assist in decision making
Or:
WHO Paediatric Stage I (asymptomatic) and CD4 <20%
- If HIV-seropositive status is not virologically proven but CD4 cell assays are available, ART can be initiated when the child has:
WHO Stage II or III disease and CD4 <20%
WHO Paediatric Stage II or III disease and CD4 <20%
- In such cases, HIV antibody testing must be repeated at age 18 months to definitively confirm that the child is HIV infected
- Only children with confirmed infection should have ART therapy continued
HIV-seropositive children aged >18 months
ART can be initiated when child has:
WHO Paediatric Stage III disease (e.g. clinical AIDS) irrespective of CD4 count
WHO Paediatric Stage II disease with CD4 <15%
WHO Paediatric Stage I disease (e.g. asymptomatic appendix I) and CD4 <15% (Appendix I)

For children > 8 years adult criteria for initiation of therapy are applicable

Drug treatment
Preferred first line regimen (adults and adolescents)
- d4T/3TC/NVP or EFV

Alternative first line regimens
- TDF/3TC/NVP or EFZ
- ABC/3TC/NVP or EFZ

Children
Preferred first line regimen
- d4T/3TC/NVP or EFV
- TDF/3TC/NVP or EFZ

Alternative first line regimens for special category of adults
Pregnant women with CD4 count <250 cells/mm$^3$ or women who are likely to become pregnant
- ZDV / 3TC / NVP

Children
Children with tuberculosis require rifampicin-containing regimen for TB treatment
- (ZDV or dT4) + 3TC + NVP during non-rifampicin-containing intensive or continuation phase
- (ZDV or dT4) + 3TC + NVP during rifampicin-containing continuation phase

First line recommendations for HIV/TB patients
Adults/Adolescents and Pregnant Women:
- (ZDV or dT4) + 3TC + NVP during non-rifampicin-containing continuation phase
- (ZDV or dT4) + 3TC + NVP during rifampicin-containing intensive or continuation phase

Management of virological treatment failure
The three drugs reserved for the first line regimens are replaced with three totally new drugs-second line regimens
- If resistance testing cannot be done (see second line treatment regimen)

Where resistance testing is available, the failing drug may be identified and replaced
Recommended second line regimens

<table>
<thead>
<tr>
<th>Adults and adolescents</th>
<th>Second line</th>
</tr>
</thead>
<tbody>
<tr>
<td>First line</td>
<td>TDF/FTC/IDV/r or SQV/r or LPV/r</td>
</tr>
<tr>
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</tr>
<tr>
<td>ABC/3TC/NVP or EFV</td>
<td>Or: TDF/FTC/IDV/r or SQV/r or LPV/r</td>
</tr>
</tbody>
</table>

Note
The dose of ddl should be reduced from 400 mg to 250 mg when co-administering with TDF in an adult > 60 kg; reduce dose to 125 mg in adult < 60 kg.

Recommended second line regimens

<table>
<thead>
<tr>
<th>Children</th>
<th>Second line</th>
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</thead>
<tbody>
<tr>
<td>First line</td>
<td>TDF or ZDV/3TC/NVP or EFV</td>
</tr>
<tr>
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</tr>
<tr>
<td>LPV/r requires secure cold chain</td>
<td></td>
</tr>
</tbody>
</table>

All treatment failures at first and second level health facilities should be referred to a paediatric consultant.

Child dosages

- Didanosine (ddI): - 2 weeks - 8 months: 100 mg/m² orally twice daily
  - > 8 months: 120 mg/m² twice daily
  - Lamivudine (3TC)
  - < 1 month: 2 mg/kg orally twice daily
  - > 1 month: 4 mg/kg orally twice daily
  - Adolescents < 50 kg: 2 mg/kg orally twice daily
  - Stavudine (d4T)
  - 1 mg/kg orally twice daily up to a maximum of 40 mg per dose
  - Zalcitabine (ddC)
  - Not available
  - Zidovudine (ZDV)
  - 160 mg/m² orally every hours
  - Efavirenz (EFZ)
  - Taken orally once daily
  - 10 to <15 kg: 200 mg; 15 to < 20 kg 250 mg; 20 to <25 kg 300 mg; 25 to <32.5 kg 350 mg; 32.5 to < 40 kg 400 mg; >40 kg 600 mg

- Nevirapine (NVP)
  - 15 - 30 days: 5 mg/kg orally once daily for 14 days, then 120 mg/m² twice daily for 14 days, and 200 mg/m² twice daily
  - 1 month - 13 years: 120 mg/m² twice daily for 14 days, then 200 mg/m² twice daily
  - Indinavir (IDV)

- <4 years: not used
- 4 - 17 years: 500 mg/m² orally twice daily; (maximum 800 mg) three times daily
- Nelfinavir (NFV)
  - <1 year: 40 - 50 mg/kg orally three times daily; or 65 - 75 mg/kg twice daily
  - 1 - 13 years: 55 - 65 mg/kg twice daily
  - Lopinavir/ritonavir (Lop/r)
  - 7 kg to <15 kg: lopinavir 12 mg/kg, ritonavir 3 mg/kg orally twice daily with food
  - >15 - 40 kg: lopinavir 10 mg/kg, ritonavir 2.5 mg/kg orally twice daily
  - >40 kg lopinavir 400 mg, ritonavir 100 mg orally twice daily with food

Notable adverse drug reactions, caution and Contraindications

- Nevirapine (NVP)
  - Life-threatening skin rash (Stevens-Johnson syndrome); occurs in < 5% of patients, usually within 8 weeks of treatment
  - Dress syndrome (drug rash, eosinophilia and systemic symptoms); manifests as fever, atheralgia, etc
  - Hepatitis and jaundice reported
  - Efavirenz (EFV)
  - Morbilliform rash may appear; usually not life-threatening
  - CNS side effects in about 50% of patients (usually self-limiting)

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Note
- Refer to standard texts for possible drug-drug interactions in all cases.

Prevention
Mechanisms with established merit:
- Prevention of mother-to-child transmission (PMTCT)
- Prophylactic AZT/NVP or HAART
- Caesarean section

Mechanisms with anticipated (potential) merit:
- Reduction of viral load with HAART
- Post exposure prophylaxis following sexual exposure (rape)
- Sexual risk reduction
- Promotion of safer sex and low-risk behaviour
- Abstinence

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For these reasons, EFV is contraindicated in patients who already have psychiatric manifestations.

- Hallucinations
- Insomnia
- Abnormal dreams
- Somnolence
- Amnesia
- Abnormal thinking
- Confusion
- Euphoria

Gastrointestinal intolerance is fairly common: hypersalivation, nausea, abdominal discomfort

Stavudine (d4T)

- Nephrolithiasis with or without haematuria in 10 - 28% of patients; (fluid intake should be increased)
- Alopecia

Efavirenz (EFV)

- Nor major side effect but class side effects may occur
- Unconjugated hyperbilirubinaemia
- Gastrointestinal effects
- No effect on lipids

Lamivudine (3TC)

- No major side effect but class side effects may occur
- Life-threatening skin rash (Stevens-Johnson syndrome); occurs in < 5% of patients, usually within 8 weeks of treatment
- Lactic acidosis (a class adverse effect)

Nevirapine (NVP)

- Not available
- Life-threatening hypersensitivity in 3 - 9% of patients
- Lactic acidosis with or without hepatic steatosis

Indinavir (IDV)

- Class-specific events
- Nephrolithiasis with or without haematuria in 10 - 28% of patients; (fluid intake should be increased)
- Alopecia

Nelfinavir (NFV)

- Diarrhoea: 10 - 30% of patients; (should be managed with agents such as loperamide)
- Fat accumulation
- Hyperlipidaemia

Lopinavir/ritonavir (LPV/r)

- Well tolerated except for occasional class adverse reactions:
- Gastrointestinal
- Hepatic transaminis especially in patients with chronic hepatitis B or C
- Hyperlipidaemia
- Fat accumulation
- Hepatotoxicity
- Aesthenia

Abacavir (ABC)

- Dose-related pancreatitis; worse when combined with hydroxycarbamide (hydroxyurea)
- Peripheral neuropathy; worse if combined with d4T
- Lactic acidosis (a class adverse effect)

Didanosine (ddI)

- Taken orally once daily
- 10 to <15 kg: 200 mg; 15 to < 20 kg 250 mg; 20 to <25 kg 300 mg; 25 to <32.5 kg 350 mg; 32.5 to < 40 kg 400 mg; >40 kg 600 mg

Atazanavir (ATV)

- 1 - 13 years: 55 - 65 mg/kg twice daily
- >40 kg lopinavir 400 mg, ritonavir 100 mg orally twice daily with food

Efavirenz (EFZ)

- Life-threatening skin rash (Stevens-Johnson syndrome); occurs in < 5% of patients, usually within 8 weeks of treatment
- DRESS syndrome (drug rash, eosinophilia and systemic symptoms); manifests as fever, atheralgia, etc
- Hepatitis and jaundice reported
- Efavirenz (EFV)

- Morbilliform rash may appear; usually not life-threatening
- CNS side effects in about 50% of patients (usually self-limiting)
Repeated vomiting
Prostration
Impaired consciousness
Severe anaemia
Circulatory collapse
Hypoglycaemia
Pulmonary oedema
Abnormal bleeding
Jaundice
Haemoglobinuria
Febrile seizures
Renal failure
Hyperparasitaemia

Cerebral malaria
A severe form of malaria
Occurs usually in children and in non-immune adults
Manifests with diffuse and symmetric encephalopathy;
focal neurologic signs are unusual
Requires prompt and effective therapy to avoid fatality

Diagnosis of malaria
Absence of fever does not exclude a diagnosis of malaria
Microscopic diagnosis should not delay appropriate treatment if there is a clinical suspicion of severe malaria

Different diagnoses
Typhoid fever
Meningitis
Encephalitis
Septicaemia
Other causes of fever

Complications
Early:
Hypoglycaemia
Lactic acidosis
Haematological abnormalities
Liver dysfunction
Pneumonia
Septicaemia
Non-cardiogenic pulmonary oedema
Cerebral malaria
'Blackwater' fever
Acute tubular necrosis

In pregnancy
Anaemia
Preterm contractions/preterm labour
Abortions
Low birth weight
Intrauterine deaths
Congenital malaria

Late
Hyperreactive malaria splenomegaly
Quartan malaria nephropathy
Possible, Burkitt's lymphoma

Investigations
Blood smear for malaria parasites
Packed cell volume; haemoglobin concentration

White cell count with differentials
Blood sugar
Urinalysis
Electrolytes and Urea; Creatinine
Stool microscopy for ova; occult blood
Chest radiograph
Cerebrospinal fluid biochemistry; microscopy, culture and sensitivity

Treatment objectives
Eradicate parasitaemia
Prevent severe malaria
Attend to the immediate threats of life
Prevent complications
Provide personal protection against malaria
Provide chemoprophylaxis in susceptible groups

Drug treatment
Uncomplicated malaria
It is vital to prevent severe disease, therefore as soon as a presumptive diagnosis of malaria is made:
Insert artesunate suppository per rectum as a single dose
Re-insert if expelled; in young children the buttocks may need to be held or taped together for 10 minutes to ensure retention of the rectal dose

Artemisin-based combination therapy is the treatment of choice

Adult: 20 mg/kg of salt to a maximum of 1.2 g loading dose intravenously, diluted in 10 ml/kg isotonic fluid over 4 hours

- 8 hours after start of the loading dose: 10 mg/kg salt to a maximum of 600 mg over 4 hours, every 8 hours until the patient is able to take orally
- Then change to tablets 10 mg/kg for 7 days or give full dose of artemether-lumefantrine

Child: 20 mg/kg of salt as loading dose diluted in 10 mL/kg of 4.3% glucose in 0.18% saline or in 5% glucose over 4 hours 12 hours later, give 10 mg salt/kg as infusion over 4 hours, and every 8 hours until patient is able to take orally
Change to tablets 10 mg/kg every 8 hours to complete a total of 7 days

Or:
- Where intravenous access is not possible, give quinine dihydrochloride 20 mg/kg salt as loading dose, diluted to 60 -100 mg/ml intramuscularly in different sites
- 8 hours after loading dose, give 10 mg/kg 8 hourly until patient is able to take orally
- Thereafter, change to tablets 10 mg/kg 8 hourly for 7 days or give a full dose of artemether-lumefantrine

Or:
- Artesunate

Adult: 2.4 mg/kg intravenous bolus; repeat 1.2 mg/kg after 12 hours then 1.2 mg/kg daily for 7 days
Child: intravenous use reserved for specialists

- Once patient can tolerate oral medication give a full dose of artemether-lumefantrine

Or:
- Artesmer

Adult: 3.2 mg/kg intramuscular loading dose followed by 1.6 mg/kg daily for 6 days
Alternatively:
- Once patient can tolerate oral medication, give full dose of artemether-lumefantrine

Supportive measures
Paracetamol (oral/rectal) for symptomatic relief of fever
If temperature is >38.5 °C, wipe with wet towel, and fan to lower the temperature

- Nurse in cardiac position
- Give oxygen
- Furosemide 2 - 4 mg/kg intravenously
- Exclude anaemia as the cause of heart of the heart failure

Renal failure
- Give fluids if patient is dehydrated: 20 ml/kg of sodium chloride injection 0.9%, and challenge with furosemide 1 - 2 mg/kg
- Catheterize to monitor urinary output
- If no urine within the next 24 hours, refer for peritoneal or haemodialysis

Standard Treatment Guidelines for Nigeria 2008

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Prophylactic treatment
- Individualized based on the nature of the exposure
- Use of antitoxins
- Use of immunization

Prevention
- Personal protection
- Reduce the frequency of mosquito bites by avoiding exposure to mosquitoes at their peak feeding times
- Use insect repellents
- Put on suitable clothing
- Use insecticide-impregnated bed nets (ITN)

Chemoprophylaxis-
- Indicated for:
  - Children born to non-immune mothers in endemic areas
  - Pregnant women (see section on antenatal care)
  - Travellers to endemic areas
  - Mefloquine 5 mg base/kg weekly, giving an adult dose of 250 mg base/week
  - Onset: 1.5 mg of salt/kg administered daily (100 mg of salt daily)
- Contraindicated in children <8 years and in pregnant women
- Commence one week before departure and continue until 4 weeks after leaving the region
- Chemoprophylaxis is not recommended for individuals living in areas of intense transmission
- People with sickle cell anaemia should have regular chemoprophylaxis (see Sickle Cell Diseases)

RABIES

Introduction
- An acute disease of the CNS caused by a bullet-shaped rabdovirus that affects all mammals
- The virus is a single-stranded RNA virus found in animals, in all regions as urban rabies or sylvatic rabies
- Transmitted by infected secretions, usually saliva
- Most exposures are through bites of an infected animal; occasionally contact with a virus-containing aerosol or the ingestion or transplant of infected tissues may initiate the disease process
- Human infection is through contact with unimmunized domestic animals
- Dogs are the most important vectors worldwide

Clinical features
- There are four stages:
  - A non-specific prodrome of 1 - 4 days consisting of
  - Fever
  - Headache
  - Malaise
  - Myalgia
  - Anorexia
  - Nausea
  - Vomiting
  - Sore throat
  - Cough
- Paraesthesia
- An acute encephalitic stage
  - Excitement
  - Agitation
  - Confusion
  - Hallucinations
  - Combativeness
- Bizarre aberrations of thought
- Muscle spasms
- Meningismus
- Focal paralysis
- Hydrophobia
- Brainstem dysfunction
  - Diplopia
  - Facial paralysis
  - Optic neuritis
  - Difficulty with deglutition
  - Priapism
  - Spontaneous ejaculation
- Coma
- Death or recovery

Differential diagnosis
- Gullain-Barré syndrome
- Other causes of viral encephalitis
- Poliomyelitis
- Allergic encephalomyelitis

Complications
- Inappropriate secretion of ADH
- Diabetes insipidus
- Cardiac arrhythmias
- Adult Respiratory Distress Syndrome (ARDS)
- Gastro Intestinal (GI) bleeding
- Thrombocytopenia
- Paralytic ileus

Investigations
- Full Blood Count and differentials
- Urea and Electrolytes
- Culture of secretions
- Cerebro Spinal Fluid (CSF) analysis
- Serology
- Pulmonary Chain Reaction (PCR)

Treatment objectives
- Disinfect wound; avoid early suturing
- Provide passive immunization with antirabies

antiserum
- Provide active immunization with the vaccine

Non-drug treatment
- Wound care
- The wound or site of exposure should be:
  - Cleansed under running water
  - Washed for several minutes with soapy water
  - Disinfected and dressed simply
- It should not be sutured immediately

Drug treatment
- Unimmunized persons or those whose prophylaxis is probably incomplete
  - Rabies (cell mediated) vaccine
    - Adult: 1 ml by deep subcutaneous or intramuscular injection in the deltoid region on days 0, 3, 7, 14 and 30
    - Plus: Rabies immunoglobulin given on day 0
- Post-exposure prophylaxis (PEP)
  - For fully immunized persons:
    - Rabies (cell mediated) vaccine
      - Adult: 1 ml by deep subcutaneous or intramuscular injection in the deltoid region on days 1 and 3
      - Same as for adult
  - Should be initiated as soon as possible after exposure

The decision to initiate PEP should include:
- Whether rabies is known or suspected in the species and area associated with the exposure

Supportive measures
- Allay anxiety: reassure
- Other measures as appropriate for clinical situation

Notable adverse drug reactions, caution
- Concomitant chloroquine administration interferes with antibody response to rabies vaccine
- There are no specific contraindications

Prevention
- Pre-exposure prophylaxis
  - Should be offered to persons at high risk of exposure
  - Vets
  - Cave explorers
  - Laboratory workers who handle the rabies virus
  - Animal handlers
  - Workers in quarantine stations
  - Field workers who are likely to be bitten by infected wild animals
- Booster doses every 2 - 3 years for those at continued risk
- Certain port officials
- Bat handlers
- Persons living in (or travelling to) areas where rabies is enzootic and/or where there is limited access to prompt medical care
  - Those caring for patients caring for patients with rabies
  - Although there is no proven evidence of human-human transmission
  - Pregnancy is not a contraindication: if there is substantial risk of exposure, and rapid access to post-exposure prophylaxis is limited, give pre-exposure prophylaxis
  - Rabies vaccine:
    - 1 ml by deep subcutaneous or intramuscular injection in the deltoid region on days 0, 7 and 28
- TETANUS

Introduction
- A common, infectious disease affecting individuals of all ages and sexes, particularly the socio-economically deprived
- Neurologic disorder characterized by increased muscle tone and spasm that is caused by tetanospsamin, a powerful protein toxin elaborated by Clostridium tetani
- The bacteria are found in the soil, inanimate environment, animal faeces and occasionally in human faeces
- Portals of entry:
  - Umbilical stump
  - Female genital mutilation (FGM)
  - Male circumcision
  - Abortion sites
  - Penetrative wounds (e.g. nail puncture or intramuscular injection)
  - Head injury; scalp wounds
  - Traditional scarification (e.g. for tribal identity)
  - Trapeo-medicinal incisions
  - Post-operative surgical sites
  - Chronic otitis media

Clinical forms:
- Generalized tetanus
- Neonatal tetanus
- Localized tetanus
- Cephalic tetanus

Clinical features
- Generalized tetanus
  - Lock jaw
  - Dysphagia
  - Stiffness or pain in the neck, shoulder and back muscles
  - Rigid abdomen and stiff proximal limb muscles
  - The hands and feet are relatively spared
- Neonatal tetanus
  - Poor feeding
  - Rigidity
  - Spasms
**Chapter 11: Infectious Diseases/Infestations**

**Localized tetanus**
- Increased tone; spasms are restricted to the muscles near the wound
- Prognosis is excellent

**Cephalic tetanus**
- Follows head injury or ear infection
- Trismus
- Dysfunction of one or more cranial nerves, often the 7th nerve
- Mortality is high

**Diagnosis**
- Entirely clinical

**Differential diagnoses**
- Alveolar abscess
- Streptococcal poisoning
- Dystonic drug reactions
- Hyponatraemic tetani
- Meningitis/encephalitis
- Acute abdomen

**Complications**
- Autonomic dysfunction
- Labile or sustained hypertension
- Tachycardia
- Dysrhythmias
- Hyperpyrexia
- Profuse sweating
- Peripheral vasocorestriction
- Cardiac arrest
- Aspiration pneumonia
- Fractures
- Muscle rupture
- Deep vein thrombophlebitis
- Pulmonary embolism
- Decubitus ulcers
- Rhabdomyolysis

**Investigations**
- Wound swab for microscopy, culture and sensitivity
- Cerebrospinal fluid for biochemistry; microscopy, culture and sensitivity
- Full Blood Count; ESR
- Urinalysis; urine microscopy, culture and sensitivity
- Blood glucose
- Electrocardiography
- Serum Electrolytes, Urea and Creatinine
- Electromyography

**Treatment objectives**
- Eliminate the source of toxin
- Neutralize unbound toxin
- Prevent muscle spasms
- Monitor the patient’s condition and provide support (especially respiratory support) until recovery

**Non-drug treatment**
- Admit patient to a quiet room
- Protect airway
- Explore wounds
- Cleanse and thoroughly debride the wound

**Drug treatment**

**Provide intubation or tracheostomy for hypventilation**

**Physiotherapy**
- Monitor bowel, bladder and renal function
- Prevent decubitus ulcers

**Prevention**
- Benzylpenicillin (Penicillin G)
  - Adults: 0.6 - 2.4 g daily by slow intravenous injection or infusion in 2 - 4 divided doses; higher doses in severe infections
  - Child: 1 month - 18 years, 100 mg/kg in 4 divided doses every 6 hours; dose doubled in severe infections (maximum 2.4 g every 4 hours)
  - 1 - 4 weeks: 75 mg/kg daily in 3 divided doses, every 86 hours; dose doubled in severe infection
- Preterm neonate and neonate under 7 days: 25 mg/kg , every 12 hours; dose doubled in severe infection

**Or**
- Metronidazole
  - Adults: 500 mg intravenously, every 6 hours for 10 days
  - Child: neonate, initially 15 mg/kg by intravenous infusion then 7.5 mg/kg twice daily; 1 month - 12 years: 7.5 mg/kg (maximum 400 mg) every 8 hours; 12 - 18 years: 400 mg every 8 hours
- Antitoxin
  - Human tetanus immune globulin (TIG)
  - Adults: 250 units by intramuscular injection, increased to 500 units if:
    - The wound is older than 12 hours
    - There is risk of heavy contamination
    - Patient weighs more than 90 kg
- A second dose of 250 units should be given after 3 - 4 weeks if patient immunosuppressed or if active immunization with tetanus vaccine is contraindicated
- Administer antitoxin before manipulating the wound
- Control of muscle spasm
- Diazepam
  - Adults: 20 mg intravenously slowly stat and titrate up to 250 mg/day in infusion
  - Child: 1 month - 18 years: 100 - 300 micrograms/kg repeated every 1 - 4 hours by slow intravenous injection
  - Could also be administered by intravenous infusion or by naso duodenal tube as follows:
    - 3 - 10 mg/kg over 24 hours, adjusted according to response

**Or**
- Phenobarbital (dilute injection, 1 in 10 with water for injection)
  - Adults: 10 mg/kg intravenously at a rate of not more than 100 mg/minute, up to maximum total dose of 1g
  - Child: 5 - 10 mg/kg at a rate not more than 30 mg/minute
- Treat autonomic dysfunction with:
  - Vasopressors, chronotropic agents if necessary
- Hydration
  - To control insensitive and other fluid losses
  - Enteral or parenteral nutrition
  - As determined by clinical situation

**TUBERCULOSIS**

**Introduction**
- One of the oldest diseases known to affect humans, globally
- Nearly one third of the global population (i.e. 2 billion) people are infected with Mycobacterium tuberculosis and at risk of developing the disease
- More than 8 million people develop active tuberculosis (TB) every year; about 2 million die
- More than 90% of global TB cases and deaths occur in the developing world where 75% of cases are in the most economically productive age group (15 - 54 years)
- M. tuberculosis usually affects the lungs although in up to one third of cases other organs are involved
- If properly treated, TB caused by drug-susceptible strains is curable in virtually all cases; however if untreated it may be fatal within 5 years in more than half of cases
- Transmission usually takes place through the airborne spread of droplet nuclei produced by patients with infectious pulmonary TB and aerosolized by coughing
  - As many as 3,000 infectious nuclei per cough can be produced
  - Droplet nuclei could also by spread by sneezing and speaking
- Poverty and widening gap between rich and poor, hunger, neglect of the disease, the collapse of health infrastructure plus the impact of HIV pandemic

**Prevention**
- Surveillance and treatment
- Chemoprophylaxis
- Vector control by selective clearing of vegetation and use of insecticides

**Investigations**
- Peripherial blood film for the detection of trypanosomes
- Rapid Card Agglutination Trypanosomiasis Test (CATT) for antibody detection

**Diagnosis**
- Presumptive
  - Based on the clinical suspicion and history of exposure to the tsetse fly
  - A finding of the trypanosome in peripheral blood, lymph node aspirate or CSF is confirmatory

**Notable adverse drug reactions, caution and contraindications**
- Diazepam is adsorbed from plastics of infusion bags and giving sets; causes drowsiness and light headedness; hypotension
- Benzyl penicillin: hypersensitivity reactions
- Metronidazole: taste disturbances
- Phenobarbital: caution in renal and hepatic impairment

**TRYPANOSOMIASIS (Sleeping sickness)**

**Introduction**
- African trypanosomiasis is an acute or chronic disease caused by Trypanosoma brucei namely:
  - T. brucei rhodesiense (East Africa)
  - T. brucei gambiense (West Africa)

**Clinical features**
- Gambian Sleeping Sickness
  - Two clinical stages:
    - Early stage
    - CNS stage
    - Early stage:
      - Anodule or chancre following a bite
      - Fever
      - Headache
      - Dizziness
      - Weakness
      - Significant posterior cervical (Winterbottom sign) and nerve
    - CNS stage:
      - Labile or sustained hypertension
      - Tachycardia
      - Dysrhythmias
      - Hyperpyrexia
      - Profuse sweating
      - Peripheral vasoconstriction
      - Cardiac arrest
      - Aspiration pneumonia
      - Fractures
      - Muscle rupture
      - Deep vein thrombophlebitis
      - Pulmonary embolism
      - Decubitus ulcers
      - Rhabdomyolysis
    - Prognosis is excellent

**Investigations**
- Provide intubation or tracheostomy for hypventilation
- Physiotherapy
- Monitor bowel, bladder and renal function
- Prevent decubitus ulcers

**Prevention**
- Active immunization of all partially or un-immunized adults, those recovering from tetanus, all pregnant women, infants and un-immunized (missed) children
- Health education
- Improvement in socio-economic status

**Diagnosis**
- Entirely clinical

**Differential diagnoses**
- Alveolar abscess
- Streptococcal poisoning
- Dystonic drug reactions
- Hyponatraemic tetani
- Meningitis/encephalitis
- Acute abdomen

**Complications**
- Autonomic dysfunction
- Labile or sustained hypertension
- Tachycardia
- Dysrhythmias
- Hyperpyrexia
- Profuse sweating
- Peripheral vasocorestriction
- Cardiac arrest
- Aspiration pneumonia
- Fractures
- Muscle rupture
- Deep vein thrombophlebitis
- Pulmonary embolism
- Decubitus ulcers
- Rhabdomyolysis

**Investigations**
- Wound swab for microscopy, culture and sensitivity
- Cerebrospinal fluid for biochemistry; microscopy, culture and sensitivity
- Full Blood Count; ESR
- Urinalysis; urine microscopy, culture and sensitivity
- Blood glucose
- Electrocardiography
- Serum Electrolytes, Urea and Creatinine
- Electromyography

**Treatment objectives**
- Eliminate the source of toxin
- Neutralize unbound toxin
- Prevent muscle spasms
- Monitor the patient’s condition and provide support (especially respiratory support) until recovery

**Non-drug treatment**
- Admit patient to a quiet room
- Protect airway
- Explore wounds
- Cleanse and thoroughly debride the wound

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**Differential diagnoses**
- Malaria fever
- Meningitis
- Viral infections involving the CNS

**Treatment**
- Early stage
  - Suramin
    - Adult and child: 5 mg/kg on day 1, 10 mg/kg on day 3, and 20 mg/kg on days 5, 11, 17 and 30
  - Late stage
    - Melarsoprol
      - Adult: 2.0 - 3.6 mg/kg intravenously in 3 divided doses for 3 days, followed 1 week later with 3.6 mg/kg intravenously in 3 divided doses for 3 days
      - 10 - 21 days later: 3.6 mg/kg intravenously in 3 divided doses for 3 days

**CAUTION**
- Urine should be examined for casts and protein before and after treatment with suramin
- Lumbar puncture follow-up for at least 1 year after treatment with melarsoprol is required

**Prevention**
- Surveillance and treatment
- Chemoprophylaxis
- Vector control by selective clearing of vegetation and use of insecticides
Clinical features

Determinants of transmission: from exposure to infection (exogenous factors)
- The probability of contact with a case of TB
- The intimacy and duration of that contact
- Degree of infectiousness of the case

Determinants of developing TB: from infection to disease (endogenous factors)
- Innate susceptibility to disease
- Level of function of the individual's cell mediated immunity
- Age
  - Incidence highest during late adolescence and early childhood, women aged 25 - 34 years and the elderly
- Other diseases

The outcome of infection by M. tuberculosis is affected by the presence of:
- HIV co-infection
- Silicosis
- Chronic renal failure and haemodialysis
- Insulin dependent diabetes mellitus
- Immunosuppressive treatment
- Malnutrition
- Old, self-healed fibrotic TB lesions

Clinical features

Generally non-specific:
- Fever (low grade and intermittent)
- Night sweats
- Wasting
- Anorexia
- General malaise
- Weakness
- Cough (initially non-productive, subsequently productive of purulent and/or blood streaked sputum)
- Haemoptysis
- Chest pain
- Dyspnoea
- Adult respiratory distress syndrome (ARDS)
- Pallor
- Finger clubbing

Extrapulmonary TB

Lymph node TB
- Painless swelling of lymph nodes (usually cervical and supraventricular sites)
  - Usually discrete in early disease; may become inflamed and have a fistulous tract draining caseous material

Plural TB
- Fever
- Pleuritic chest pain
- Dyspnoea
- Dullness to percussion
- Absence of breath sounds

TB of the upper airways
- Nearsly always a complication of advanced cavitationary pulmonary TB
- May involve the larynx, pharynx and epiglottis
- Hoarseness
- Dysphagia
- Dysphonia
- Chronic productive cough

Genitourinary TB
- Urinary frequency
- Dysuria
- Haematuria
- Flank pain

Skeletal TB
- Weight bearing joints are affected: spine, hips and knees

Sporal TB (Pott's disease)
- Paraparesis
- Paraplegia

TB meningitis
- Headache
- Mental changes
- Confusion
- Lethargy
- Altered sensorium
- Neck rigidity
- Ocular nerve paresis
- Hydrocephalus

Gastrointestinal TB
- Commonly affects the terminal ileum and caecum
- Abdominal pain (may be similar to that of appendicitis)
- Diarrhoea
- Intestinal obstruction
- Haematochezia
- Palpable mass
- Fever
- Weight loss
- Night sweats
- TB peritonitis

Pericardial TB
- Fever
- Dull retrosternal pain
- Friction rub
- Cardiac tamponade

Military TB
- Fever
- Night sweats
- Anorexia
- Weakness
- Weight loss
- Cough

Hepatomegaly
- Splenomegaly
- Lymphadenopathy
- Choroidal tubercles (pathognomonic)
- Meningitis

There are no clinical findings specific for a diagnosis of pulmonary TB: a history of contact with a smear positive pulmonary TB case, respiratory symptoms for more than 2-3 weeks not responding to broad spectrum antibiotics, and weight loss, failure to thrive may suggest TB

Differential diagnoses
- Will vary depending on the system affected:
  - Asthma
  - Bronchiectasis
  - Whooping cough
  - Inhaled foreign body
  - Cardiac disease
  - Carcinomas
  - Intracranial space-occupying lesions
  - Osteoarthritis, etc

Investigations
- Sputum for AFB, microscopy, culture and sensitivity
- Chest radiograph
- Full Blood Count; ESR
- HIV screening
- Urinalysis; microscopy, culture and sensitivity
- CSF microscopy, culture, sensitivity; chemistry
- Drug susceptibility testing
- Others: IVP, bone biopsy, etc as indicated

Complications
- Lung abscess
- Destroyed lung syndrome
- Pressure effects from enlarged lymph nodes
- Obstructive uropathy
- Chronic kidney disease
- Infertility
- Skeletal deformities (varus and valgus; kyphosis, scoliosis)

Treatment objectives
- Cure the disease
- Prevent death from active TB or its late effects
- Prevent relapse of TB
- Decrease transmission of TB
- Prevent the development of acquired drug resistance

Treatment
- Regimen should include at least 4 drugs in the initiation phase
- Standardized regimens are the choice in settings where susceptibility testing of reserve drugs is not available

TYPHOID FEVER

Introduction
- A systemic disease characterized by fever and abdominal pain, caused by dissemination of Salmonella typhi or S. paratyphi
- Transmitted only through close contact with acutely infected individuals or chronic carriers (from ingestion of contaminated food or water)

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Incidence of chronic carriage is higher among women and persons with biliary abnormalities: gall stones, carcinoma of the gall bladder; also higher in persons with gastrointestinal malignancies

Clinical features
- Incubation period ranges from 3 - 21 days
- Prolonged fever (38.8 C to 40.5 C)
- A prodrome of non-specific symptoms:
  - Chills
  - Headache
  - Anorexia
  - Cough
  - Weakness
  - Sore throat
  - Dizziness
  - Muscle pains
- Gastro-intestinal: infection (exogenous factors)
- Genitourinary TB
- Abdominal pain

Investigations
- Full Blood Count
- Liver function tests
- Nearly always a complication of advanced cavitatory pulmonary TB case, respiratory symptoms for more than 2-3 weeks not responding to broad spectrum antibiotics, and weight loss, failure to thrive may suggest TB
- Values may be elevated
- Electrocardiography
- ST and T wave abnormalities may be present
- Serological tests
- Widal test gives high rates of false positives and negatives

Complications
- Asthma
- Bronchiectasis
- Whooping cough
- Inhaled foreign body
- Cardiac disease
- Carcinomas
- Intracranial space-occupying lesions
- Osteoarthritis, etc

Non-specific:
- Full Blood Count
- Leucopenia, neutropenia, leucocytosis can developearly, especially in children; late if complicated by intestinal perforation or secondary infection
- Liver function tests
- Values may be elevated
- Electrocardiography
- ST and T wave abnormalities may be present
- Serological tests
- Widal test gives high rates of false positives and negatives

Treatment objectives
- Eliminate S. typhi and S. paratyphi
CHAPTER 12: MUSCULOSKELETAL SYSTEM

BACK PAIN

Introduction
A common complaint which most adults will have had at some time or the other. Defined as pain in the back, at any site between the neck and the buttocks. Low back pain is the commonest; involves essentially the lumbosacral/coccygeal spine.

Most cases result from mechanical causes and usually last less than six weeks.

Causes include:
- Spondylosis
- Intra-spinal abscess
- Tumours (primary or secondary)
- Osteoporosis
- Osteomyelitis
- Trauma
- Pregnancy

Clinical features
Patients will complain of aches, pains, or sometimes peaky sensations. Pain is usually worsened on bending forward if due to a discopathy.

- Worsened when the intra-abdominal pressure is increased as in sneezing and coughing
- Worsened on extension of the back if it is due to apophysysal lesion
- Most back pains are from mechanical causes and are self-limiting
- There are danger or 'red flag' features that indicate more serious causes as infections, or malignancy
- Starting for the first time in persons aged 50 years and above
- Worsened at night
- Worse on lying supine
- Associated with constitutional disturbances such as fever, loss of weight, anorexia, anaemia
- Associated with radical pain
- Associated with structural abnormalities such as kyphosis or scoliosis

Differential diagnoses
- Pancreatic or gall bladder, stomach, or intestinal disorders with referred pain
- Retro-peritoneal tumours
- Alcoholics
- Aortic aneurysms
- Tuberculosis of the pleura, pericardium
- Metastatic bone disease
- Psychosomatic disorders
- Pelvic inflammatory disease

Complications
- Complications of underlying cause(s) or pressure effects on the spinal cord and nerve roots

Investigations

Full Blood Counts; ESR
- C-Reactive Protein
- Calcium, phosphate, alkaline phosphatase levels
- Radiograph of the lumbosacral spine, myelogram
- CT Scan
- MRI
- Bone densitometry

Treatment objectives
- Treating underlying cause
- Relieve pain
- Treat complications

Drug treatment
- Paracetamol
- Narcotic analgesics
  - Morphine 10 mg orally every 4 hours (if necessary)
- Antidepressants
  - Amitriptyline initially 25 mg orally daily
- Acupuncture
- Surgery

CHAPTER 12: MUSCULOSKELETAL SYSTEM

PREVENTION

Eliminate Salmonella by effective treatment of cases, improved sewage management, improved water treatment and improved food hygiene (production, transit, storage and utilization).

Typhoid immunization is recommended for those at risk.

- Not a substitute for scrupulous personal and environmental hygiene

Identify, and treat chronic carriers with amoxicillin or ciprofloxacin daily for 4-6 weeks.

- In patients with urolithiasis and schistosomiasis appropriate treatment should be instituted.
- Correct anatomic abnormalities associated with the disease surgically.
- Cholecystectomy may be required in some cases.
Diclofenac sodium
- 75 mg orally twice daily
Oral corticosteroids:
- Prednisolone
- 40 mg in divided doses for 3 days, tapered over 2 weeks
Intra-articular steroids:
- Triamcinolone
- 5 - 40 mg by intra-articular/intradermal injection according to patient's size (maximum 80 mg); may be repeated when relapse occurs
Methylprednisolone
- 4 - 80 mg (depending on patient's size) intra-articularly; may be repeated at intervals of 7 - 35 days

Injections should be looked for
- Affects mostly weight-bearing joints such as knees, ankles. Other joints such as hips (especially in sickle cell disease), hands and spine may be affected
- Presenting features are:
  - Peeks
  - Morning stiffness of short duration
  - Swelling
  - Creakiness while walking
  - Loss of function and deformity

Hyaluronate
- Injected into the joint (usually the knee), results in pain relief in 1 - 6 months, but increases inflammation in the short term

Gout
- 750/600 mg one tablet orally every 12 hours

Indications for surgery
- Intractable pain
- Deformity
- Disability

Alternative therapies
- Acupuncture
- Osteopathy
- Transcutaneous Electrical Nerve Stimulation (TENS)

Notable adverse drug reactions, caution and contraindications
- NSAIDs
  - Reduce weight
  - Cardiovascular disease
  - Ischaemic heart disease
  - Blood dyscrasias
  - Hypersensitivity reactions
  - Reduce dose in renal insufficiency
  - Methylprednisolone
  - - 40 mg in divided doses for 3 days, tapered over 2 weeks

Non-drug treatment
- Patient education
- Exercise
- Physiotherapy
- Hydrotherapy
- Occupational therapy
- Intra-articular lavage

Drug treatment
- Paracetamol
  - 500 mg 1 g orally every 8 hours
- NSAIDs
  - Orally or local application
  - Ibuprofen
- Adult: 400 - 800 mg orally every 8 hours
- Naproxen
- Adult: 500 mg orally every 12 hours
- Diclofenac sodium
- Adult: 75 - 150 mg orally in 2-3 divided doses daily

Narcotic analgesics
- Morphine
- Adult: 5 - 20 mg orally every 4 hours
- Anti-depressants for night pain
- Amitriptyline
- Adult: 25 - 75 mg orally daily in divided doses or as a single dose at bedtime
- Capsaicin cream
  - 0.075% cream, apply small amounts up to 3 - 4 times daily
- Intra-articular steroids

OSTEOARTHRITIS

Introduction
- A heterogenous group of diseases manifesting with symptoms and signs in the synovial joints, attributable to dysfunction of the articular cartilage and subchondral bone
- It is the end result of all forms of diseases in the joints
- When such changes occur in the intervertebral disc, it is called spondylosis

Clinical features
- Affects mostly females 40 years and above. If less than 40 years, underlying causes e.g. trauma or repetitive

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RHEUMATOID ARTHRITIS

Introduction
- A chronic inflammatory disease of unknown cause
- Possibly occurs as a result of auto-immunity
- Affects primarily the peripheral joints in a symmetric pattern; may affect other organs

Clinical features
- Clinical manifestations are usually preceded by constitutional symptoms such as fatigue, malaise, fever, weight loss, loss of appetite
- Joint involvements are characterized, serially or simultaneously, by the following

Significant joint morning stiffness
- Polyarthritis
- Arthritis of joints of the hands
- Bilaterally symmetrical arthritis
- Any joint could be affected but mostly the knees, ankles, hips, shoulders, elbows; not joints of the back

Other clinical features
- Rheumatoid nodules
- Lymph glands enlargement
- Anaemia
- Hepatosplenomegaly

Differential diagnoses
- Systemic Lupus Erythematosus
- Polyarticular gout

OSTEOARTHRITIS

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- When such changes occur in the intervertebral disc, it is called spondylosis

Clinical features
- Affects mostly females 40 years and above. If less than 40 years, underlying causes e.g. trauma or repetitive
Rheumatoid arthritis
Typhoid fever
Avascular necrosis
Premature atherosclerotic disease
Myocardial infarction

Full Blood Count: leucopaenia, thrombocytopaenia, anaemia
ESR, CRP
Urea, Electrolytes and Creatinine
LE cell test
Serology: ANA, Anti-ds DNA, Anti-SM; Ro/Ssa, La/SSB, Anti-Cardiolipin antibody
Radiographs of affected joints
Echocardiogram
MRI

SYSTEMIC LUPUS ERYTHEMATOSUS

Introduction
A chronic, multisystemic, auto-immune inflammatory disease that affects virtually any organ in the body
Typically runs a relapsing and remitting course
Affects mainly women of child-bearing age
Particularly common among Blacks and Asians, in whom it runs a more devastating course

Clinical features
Fever
Hot, painful and distended joint with pus
Markedly decreased range of motion

Typical presentations:
Fever
Marked weight loss
Loss of appetite

Onset usually preceded by constitutional symptoms:
Fever
Marked weight loss
Loss of appetite
Aches and pains all over the body

Typical characteristics are seen serially or simultaneously:
Joint pains
Malar rash
Discoideal rash
Photosensitivity
Mouth or pharyngeal ulcers
Pleurisy
Pericarditis
Renal failure
Nephritis
Nephritic syndrome
Seizures
Psychosis
Peripheral neuropathy
Transverse myelitis
Eye involvement
Recurrent abortions

Complications
Opportunistic infections
Avascular necrosis
Premature atherosclerotic disease
Myocardial infarction

Differential diagnoses
Malaria

SEPTIC ARTHRITIS

Introduction
An inflammation of synovial tissues by bacteria, with production of pus into the joint space
Also variously called suppurative, purulent or infective arthritis

Rapid, but may cause a lot of illness and early joint destruction or deformity
Septic arthritis is broadly categorized as:
Gonococcal
Non-gonococcal
S. aureus, streptococci, candida species, M. tuberculosis, HIV, hepatitis B virus

Clinical features
Frequency in most studies is about 2 - 10 cases per 100,000
May occur on its own, or in association with other forms of arthritis such as gout, rheumatoid arthritis and osteoarthritis
Causative organisms are mostly S. aureus, and streptococci. Other organisms include H.influenzae, Nesseria gonorrhoeae

Typical presentations:
Fever
Hot, painful and distended joint with pus
Markedly decreased range of motion
Occasionally, septic arthritis may present with a migratory polyarthritis and dermatitis, especially with gonococcal infection
Constitutional symptoms such as nausea, vomiting, headaches, loss of weight, loss of appetite may also be seen

Differential diagnoses
Malaria, fever
Acute gouty arthritis
Osteoarthritis
Rheumatoid arthritis

Complications
Irreversible joint destruction
Degenerative joint disease
Osteomyelitis
Soft tissue injury

Investigations
Full Blood Count and differentials
ESR
Blood cultures
Urethral, cervical and rectal cultures
Synovial fluid analysis
Main radiographs of affected regions
Ultrasonography

Treatment objectives
Initiate appropriate antibiotics therapy early to prevent joint damage
Prevent septicemia arising from the joint

Drug treatment
Antibiotic choice (based on culture report)
- Ceftriaxone 1 g intravenously every 24 hours
- Treatment may be continued for 4 weeks
- There can be a change to oral antibiotics after the first week
Joints infected with N. gonorrhoeae respond to 1 week of intravenous ceftriaxone followed by ciprofloxacin

Approved by: Dr. F. E. Okolie

Chapter 12: Musculoskeletal System

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500 mg orally twice a day for another 1 week

Surgical measures
- Needle aspiration
- Arthroscopic drainage and lavage
- Open drainage and lavage

Prevention
Effective treatment of the primary infective agents and other predisposing disease states e.g. sickle cell disease, complicated fractures
Attention to asepsis in joint manipulation procedures and during intra-articular diagnostic/therapeutic interventions

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Differential diagnoses
Malaria
Review existing laws on abortion with a view to promoting and protecting the overall wellbeing of mother and unborn child.

Chapter 13: Obstetrics and Gynaecology

ABORTION

Introduction
- Expulsion from the mother's uterus of a growing and developing embryo or foetus prior to the stage of viability (about 20 weeks), with foetal weight less than 50 g
- One of the leading causes of maternal mortality and morbidity in Nigeria
- May be:
  - Spontaneous
  - Occurring from natural causes
  - Induced
  - Brought about purposefully by drugs or mechanical means
- Accidental
- Due to a fall, blow or other injury
- Complete
- With complete expulsion or extraction from the mother of a foetus or embryo, and of any other products of conception
- Incomplete
- Parts of the products of conception have been expelled but some (usually the placenta) remain in the uterus
- Illegal (criminal)
- Termination of a pregnancy without legal justification
- Legal
- With or without medical justification but done in a manner that is legal
- Solitary
- A single experience of an abortion
- Habitual
- When a woman has had three or more consecutive, spontaneous abortions

Clinical features
- Threatened abortion:
  - Cramp-like pains
  - Slight show of blood
  - May or may not be followed by the expulsion of the foetus
  - Occurs during the first 20 weeks of intrauterine life ('pre-viability' period)
  - Imminent/incipient/impending abortion:
    - Copious vaginal bleeding
    - Uterine contractions
    - Cervical dilatation
- Inevitable abortion:
  - Rupture of the membranes in the presence of cervical dilatation in a pre-viable pregnancy
  - Amniotic/tubal abortion:
    - Abortion of pregnancy in the ampulla of the fallopian tube or the tube itself
    - Rupture of an oviduct, the seat of ectopic pregnancy
    - Extrusion of the products of pregnancy through the fimbriated end of the oviduct

Differential diagnoses
- Antepartum haemorrhage
- Ectopic pregnancy
- Hydatidiform mole
- Carcinoma of the cervix
- Rape

Investigations
- Pelvic ultrasound scan
- Abdominal radiograph
- Chest radiograph
- Microscopy, culture and sensitivity test of vaginal discharge
- Urinalysis; urine microscopy, culture and sensitivity
- Full Blood Count
- Blood Group

Complications
- Endometritis
- Parametritis
- Peritonitis
- Haemorrhage
- HIV infection
- Secondary infertility
- Perforation of the uterus and/or intestines
- Rupture of the bladder

Treatment objectives
- Restore haemostasis
- Prevent/treat complications
- Provide health education

Nursing care
- Psychological support
- Personal hygiene

Drug treatment
- Treat infection(s)
- Replace fluid, electrolyte, and blood losses
- Complete incomplete abortion
- Surgical correction of complication(s)

Prevention
- Promote personal and family understanding of basic reproductive health
  - Universal basic education
  - Girl child education
  - Mental instruction
  - Protect vulnerable groups (young females) from undue exposure to their male folks
  - Pregnant women
  - In school
  - Within peer groups
  - Legislation against street hawking for vulnerable groups
  - Provide access to Primary Health Care and referral to efficient and effective higher levels of care
  - Enforce existing laws on the criminality of abortion

ANTENATAL CARE (ANC)

Introduction
ANC is clinical assessment of mother and foetus, with an overall goal of obtaining the best possible outcomes for both.

An excellent example of preventive health care, as it deals mainly with normal individuals with an emphasis on the practice of health promotion.

Availability, accessibility and utilization of ANC remain poor in Nigeria as in many other developing nations.

Aims of antenatal care
- Assessment and management of maternal risk and symptoms
- Assessment and management of foetal risk
- Prenatal diagnosis and management of foetal abnormality
- Diagnosis and management of perinatal complications
- Decisions regarding timing and mode of delivery
- Parental education regarding pregnancy and childbirth
- Parental education regarding child-rearing

Providers of antenatal care
- Community care, supervised predominantly by the midwife
- Shared care between the woman's general practitioner, midwife and obstetrician, with visits interspersed between all health professionals concerned

Hospital-only care:
- In cases where there is increased risk to the mother, foetus, or both
- Specialized care component

Previously, antenatal visits were:
- Best available evidence indicates that there is no difference in outcome between a four-visit schedule and a twelve-visit schedule
- Current trends favour fewer visits, while establishing clearly defined objectives to be achieved at each visit
- Pre-conception visit
- 1st ANC visit
Chapter 13: Obstetrics and Gynaecology

Standard Treatment Guidelines for Nigeria 2008

- Iron
- Folic acid
- Tetanus toxoid (2nd injection)
- Antimalarials

Keep complete clinic records of all transactions of the visit.
2nd ANC visit
- Should be close to, or at 26th week
- Expected to take about 20 minutes
- Activities during the visit should include:
  - Review of history for any changes
  - Assessment of adherence to routine ANC medicines

Assess for referral
- Update the risk status and refer if the need arises
- Physical examination
- General examination: pallor, oedema
- Blood pressure
- SFH

Investigations
Urinalysis for bacteriuria, proteinuria
For nulliparous women and those with a history of hypertension or pre-eclampsia/eclampsia
Haemoglobin concentration/packed cell volume

Only if there is evidence of anaemia

Interventions
Iron
- Folic acid
- Malaria prophylaxis
- Intermittent treatment with sulfadoxine/pyrimethamine
- One full treatment dose in the 2nd and 3rd trimesters
- Last dose not later than 1 month before the Expected Date of Delivery

Or:
- Proguanil 100 - 200 mg orally daily
- Maintain complete clinic records as well as ANC card records

3rd ANC visit
- Should be around the 32nd week
- Expected to take about 20 minutes
- Activities during the visit:
  - Review history for any changes
  - Assess adherence to routine ANC medicines
  - Extra attention to advice on:
    - What to do if labour occurs
    - What to do if membranes rupture
    - Birth spacing and counselling on contraception
  - Assess for referral
  - Physical examination
    - General examination: pallor, oedema, dyspnecia
    - Breast examination
    - Blood pressure
    - Abdomen: SFH palpation for twin gestation

Investigations
- Haemoglobin concentration/packed cell volume
- Compulsory for all in this visit
- Urinalysis: bacteriuria, proteinuria; for nulliparous and those with hypertension, pre-eclampsia/eclampsia

Interventions
- Iron
- Folic acid
- Malaria prophylaxis
- Advice, questions and answers; scheduling next appointment
- Maintain complete records: clinic as well as ANC card records

4th ANC visit
- The final visit before labour or delivery
- Should take place about or between the 36th - 38th weeks
- Activities during the visit include:
  - Review history for any changes
  - Assessment of adherence to routine ANC medicines
  - Physical examination
  - General examination
  - Blood pressure
  - SFH, foetal lie and presentation; presence of multiple pregnancies
  - Advise on the concept of prolonged pregnancy and the need to present if still not in labour by the 41st week

Investigations
Urinalysis: proteinuria; only in nullipara, hypertension, pre-eclampsia/eclampsia

Assess for referral

Interventions
Iron
- Folic acid
- Malaria prophylaxis
- Advice, questions and answers; scheduling next appointment
- Maintain complete records: clinic as well as ANC card records

Malaria treatment for breakthrough episodes
- Quinine is safe and can be used in all trimesters
- Artemisinin-based combinations are safe in the 2nd and 3rd trimesters
- Artemether-lumefantrine is considered safe risk
- Should hold within 1 week postpartum
- Offer contraception
- Complete treatment prophylaxis with malaria toxoid
- Continue interventions: iron, folic acid and malaria prophylaxis

ANAEMIA IN PREGNANCY

Introduction
- Anaemia is the most common complication of pregnancy in Sub-Saharan Africa
- It is a diminution below normal of the total circulating haemoglobin mass

World Health Organization definition of anaemia
- Haemoglobin concentration less than 11 g/dL or a haematocrit less than 33% in peripheral blood

For practical purposes in developing and tropical countries a haemoglobin concentration of 10 g/dL or haematocrit of 30% is taken as cut off
- Below these levels there may be adverse foetal and maternal outcomes

Classification
- Mild: PCV 25 - 29%
- Moderate: PCV 20 - 24%
- Severe: PCV < 20%

Clinical presentation
- Varies; depends on the severity
- May be asymptomatic or symptomatic

Symptoms
- Generalised weakness
- Lassitude
- Easy fatigability
- Headaches
- Dyspnoea on mild exertion
- Ankle swelling

Signs
- Pallor
- Jaundice may or may not be present
- Pedal oedema
- Tachypnoea
- Tachycardia
- Ankle swelling
- Pallor
- Jaundice may or may not be present
- Pedal oedema
- Tachypnoea
- Tachycardia
- Ankle swelling

Physiological demands of pregnancy
- Excessive red cell haemolysis as in malaria, haemoglobinopathies
- Infections: urinary tract infection, HIV/AIDS

Differential diagnoses
- Nutritional deficiencies
  - Iron, folic acid, protein, vitamin C; trace elements, and rarely vitamin B12
  - Physiological demands of pregnancy
  - Excessive red cell haemolysis as in malaria, haemoglobinopathies
  - Infections: urinary tract infection, HIV/AIDS

Hepatomegaly: not invariable
- Splenomegaly: not invariable

Anaemic heart failure in extreme cases

Postnatal visit
- Scheduled within 1 week postpartum

Offer contraception
- Complete tetanus prophylaxis with tetanus toxoid
- Continue interventions: iron, folic acid and malaria prophylaxis

Complications
- Maternal
  - Abortion
  - Cardiac failure
  - Reduced ability to tolerate blood loss at delivery

- Diminished resistance to infection
Principles of management
Examination Under Anaesthesia
Stages I to IIA
Surgery- Radiotherapy- Surgery plus radiotherapy- Chemo-radiation
Treatment options will depend on
The skill of the surgeon
Availability of facilities
The stage of the disease
Follow up
Age of the patient
Ability of available personnel to manage untoward effects of the modality of treatment chosen
Antihistamine (chlorphenamine injection),
Prevention
Surgery or radiotherapy (as primary modes of treatment respectively)- Radiotherapy can be used as primary mode of treatment in all stages of the disease
This is for life
Investigations
Chapter 13: Obstetrics and Gynaecology
Intramuscular injection
- 250 mg daily; after a negative test dose of 25 mg
Intravenous
- If the total calculated dose of iron dextran is less than 1,500 mg it can be given over 8 hours in one litre of sodium chloride 0.9%
- If greater than 1,500 mg, it should be given in divided doses daily, not exceeding 1,500 mg/day
Antihistamine (chlorphenamine injection), epinephrine and hydrocortisone injection must be available; iron dextran could cause severe anaphylaxis
Blood transfusion
- Consider as from the 37th week for mild anaemia and from the 32nd week for moderate anaemia
- Usually, packed cells under furomide cover
Indications:
- Severe anaemia irrespective of gestational age
- Cardiac failure
- Moderate anaemia detected in labour or during an abortion, or co-existing with other conditions such as sepis, renal failure, haemorrhage or eclampsia
Prevention
Counselling on contraception; adequate spacing of pregnancies
Malaria prophylaxis in pregnancy
Chlamydia prophylaxis against helminthiasis
Prompt and appropriate treatment of febrile illnesses in pregnancy
Improvement in the socioeconomic status of the people
- Provision of accessible and affordable maternity care facilities
CANCER OF THE CERVIX
Introduction
The second most common malignancy and the leading cause of death among women in developing countries
- 75% of the patients present in advanced stages; lack of organized screening programmes for detection of the pre-clinical stages in many countries
Aetiology/risk factors
Aetiology not known but several risk factors have been implicated:
- Early sexual exposure
- Multiple sexual partners
- A promiscuous male partner
- History of sexually transmitted infections particularly Human Papilloma Virus infection;
- Herpes simplex type 2; chlamydiae
- Early first child birth
- High parity
Intravenous urography
- Low socio-economic status
- Smoking
- Micronutrient deficiency
- Oral contraceptive usage
- Poor sexual hygiene
Clinical features
Two age groups with highest incidence: 35 - 40 years; 45 - 55 years
- May be asymptomatic
- Picked up in the early stage by routine PAP smear screening
Abnormal vaginal bleeding
- Postcoital
- Contact
- Spontaneous
- Inter-menstrual
- Post-menopausal
Vaginal discharge
- Becomes offensive in advanced disease
- Pyometria with uterine enlargement
- Haemorrhagic, ulcerative or fungating lesion on the cervix, with extension on to the vagina wall in advanced stages
- Vesico-vaginal fistula in advanced stages
- Recto-vaginal fistula in advanced stages
- Cachexia
- The presence of a lesion on the cervix
Presumptive Diagnosis
Based on:
- Typical history of risk factors
- Histological confirmation of malignancy
Diagnósticos diferenciales
Endometrial cancer
Endometrial hyperplasia
Endometrial polyps
Endometritis: particularly atrophic
Choriocarcinoma
Cervicitis
Cervical polyps
Cervical erosion
Vaginal lesions: vaginitis, vaginal malignancy
Functioning tumours of the ovary leading to endometrial hyperplasia and vaginal bleeding
Iatrogenic: hormonal drugs and IUCD in-situ
Blood disorders: bleeding dyscrasias, leukaemia
Investigations
Packed cell volume; haemoglobin concentration
Urinalysis
Blood Group
Electrolytes and Urea
Liver function tests
Midstream urine specimen for microscopy, culture and sensitivity
Chest radiograph
HIV screening
Cardiac disease in pregnancy
Introduction
A rare but potentially serious clinical entity
Occurs in about 1% of all pregnancies
Incidence and prevalence of all heart disease varies from place to place
- Rheumatic heart disease is more commonly found in less affluent societies while congenital heart disease now accounts for approximately 50% of cardiac diseases in pregnancy in the UK
Types of cardiac diseases in pregnancy
Acquired
- Rheumatic heart diseases
- Mitral > Aortic > Tricuspid > Pulmonary
Cardiomyopathies
- Particularly peripartum cardiomyopathy which could be either congestive or obstructive
Pre-existing hypertensive heart disease
Ischaemic heart disease
Congenital
Acanthotic heart disease


Chapter 13: Obstetrics and Gynaecology

- Atrial septal defect, ventricular septal defect, patent ductus arteriosus, etc
- Cyanotic heart disease
- Tetralogy of Fallot, Eisenmenger's syndrome

Acquired forms of cardiac disease appear to be more lethal in association with pregnancy, in women aged 25 years or more, and in third or later pregnancies

Congenital malformations are more prevalent in younger women and in those of lower parity

Clinical features

Severity of heart disease in pregnancy

The New York Heart Association Guidelines (1965) is used.

- Relies on the cardiac response to physical activity; may not bear any relationship to the extent of the lesion present

Class 1
- No limitation of physical activity

Class 2
- Slight to moderate limitation of physical activity: ordinary day-to-day activities cause dyspnoea

Class 3
- Marked limitation of activity. Minimal exertion causes dyspnoea

Class 4
- Symptoms at rest; unable to carry out any physical activity without dyspnoea; orthopnoea may be present

Other symptoms

Palpitations
Nasal stuffiness
Dizziness; light headedness; syncope
Epigastric or subxiphoid pain; bloating, heartburn
Heat intolerance, sweating and flushing

Signs

Pleuritic facies
Odema (legs; occasionally hands and face)
Varicose veins
Bounding pulses and capillary pulsations
Capillary telangiectasia
Prominent jugular venous pulsations
Lateral displacement of cardiac apex
Sinus tachycardia, ectopic beats
Third heart sound
Widely split S1 and S2, heart sounds

Murmurs
Crepitations

Investigations

Full Blood Count
Serum Electrolytes, Urea and Creatinine
Urinalysis
Blood Glucose
Echocardiography (Doppler)
Electrocardiography

Serial blood cultures (if infective endocarditis is suspected)

Chest radiograph is better avoided in pregnancy

Management

Pre-pregnancy

- Fully evaluate patient in conjunction with a cardiologist
- Surgically correct any defect that is amenable
- Counsel on the following points:
  - Risk of maternal death
  - Possible reduction of maternal life expectancy
  - Risk of foetus developing congenital heart disease;
  - Foetal growth restriction
  - Possibility of pre-term labour
  - Need for frequent hospital attendance; possibly admission
  - Need for intensive maternal and foetal monitoring in labour

Antenatal Care

- Joint management with the cardiologist
- Extreme vigilance: most features of cardiac failure are present in pregnancy
- Watch out for respiratory tract infection or urinary tract infection and treat aggressively
- Watch out for anaemia, obesity and multiple gestations for intensive care. Intensive care also required when other medical or psychological conditions co-exist
- Examination:
  - Ankle and sacral oedema
  - Pulse rate and rhythm
  - Blood pressure
  - Jugular venous pressure
  - Basal crepitations
  - Symphysio-fundal height (SFH) measurement
- Competent dental care:
  - Full inspection
  - Advise on oral hygiene
- Dental treatment e.g. tooth extraction should be done under antibiotic cover to prevent infective endocarditis
- Admissions:
  - Individualised; usually when complications or intercurrent illnesses occur

Supportive measures

- Elastic stockings or tights to prevent pooling of blood in the veins of the lower limb
- Anticoagulation
  - Indicated for example in patients with congenital heart disease, with pulmonary hypertension; artificial valves
  - Replacements; those with atrial fibrillation
  - Heparin safer in pregnancy; warfarin is teratogenic
- Termination of pregnancy and sterilization
- Best option in severe debilitating cases
- Manage as if non-pregnant (in conjunction with a cardiologist)

Foetal surveillance:

- Ultrasound scan particularly for cardiac anomaly at 22 weeks
- Delivery:
  - Either for maternal or foetal indications
- Cardiac surgery in pregnancy if indicated
- Avoid induction of labour if possible
- Prophylactic antibiotics to prevent bacterial endocarditis

Cyanotic congenital heart disease

- Careful fluid balance
- Avoid the supine position

Foetal growth restriction in > 10% of cases
Pre-maturity
Small for gestation age
Intrauterine growth restriction
Intrauterine foetal death
Brain damage
Death

ECLAMPSIA

Introduction

- The occurrence of generalized convulsions, associated with signs of pre-eclampsia during pregnancy, labour, or within 7 days of delivery; not caused by epilepsy or other convulsive disorders
- Referred to as atypical eclampsia if it occurs
- In the absence of high blood pressure
- After 7 days post-partum

Incidence is widely variable. Worldwide range reported to be 1 in 100 - 1 in 3,448 pregnancies

In Nigeria, it is commoner among unbooked patients

Management

- Manage in conjunction with the physician

Treatmeent objectives

- Stabilise the patient

Other symptoms

- Palpitations
- Nasal stuffiness
- Dizziness; light headedness; syncope
- Epigastric or subxiphoid pain; bloating, heartburn
- Heat intolerance, sweating and flushing

Elastics: stockings or tights to prevent pooling of blood in the veins of the lower limb

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- Indicated for example in patients with congenital heart disease, with pulmonary hypertension; artificial valve replacements; those with atrial fibrillation
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Aetiology

- Not exactly known. Its precursor is pre-eclampsia
- A disease of primigravidae, or multigravidae with pregnancy for a new consort

Clinical features

- Generalized tonic-clonic seizures, usually heralded by:
  - Headaches
  - Dizziness and blurring of vision
  - Nausea and vomiting
  - Epigastric pain
  - Rapidly progressive oedema

- Exaggerated tendon reflexes
- Oliguria
- Hypertension
- Worsening proteinuria

Complications

Maternal

- Cerebral haemorrhage
- Disseminated intravascular coagulopathy
- Renal failure
- Cardiopulmonary failure
- Liver dysfunction (as in HELLP syndrome)
- Fatality

Foetal

- Prematurity
- Intrauterine growth restriction
- Intrauterine foetal death
- Brain damage
- Death

Differential diagnoses

- Idiopathic epilepsy: sometimes accompanied by transient proteinuria
- Cerebral malaria: sometimes accompanied by hypertension and albuminuria
- Pneumococcal meningitis
- Hyper and/or hypo-glycaemia, particularly among diabetics
- Terminal phase of severe anaemia
- Terminal phase of hepatic failure
- Severe infections and septicamia
- Others:
  - Uraemia
  - Brain tumours or abscesses
  - Cerebral haemorrhage
  - Poisoning (accidental or intentional)
  - Hysteria

Investigations

- Haemoglobin concentration/haematoctrit
- Bedside crude clotting time
- Haemoglobin genotype
- Platelet count
- Blood Group
- Serum Urea and Electrolytes; Creatinine
- Liver function tests
- Urinalysis

Management

- Manage in conjunction with the physician

Treatmeent objectives

- Stabilise the patient
Chapter 13: Obstetrics and Gynaecology

**Complications**

- Stabilization
  - Control (and prevent further) fits
  - Control blood pressure
  - Maintain the airway
  - Ensure adequate urinary output

- Monitor
  - Controlling fits
    - Intravenous diazepam
      - 10 mg stat to abort seizures or prevent fits during examination; then
        - Intra venous infusion of glucose 5% in water with 40 mg of diazepam added, and titrated against the patient's level of consciousness
  - Magnesium sulfate (see details below)

- Treatment packs are contained in cardboard boxes containing magnesium sulfate for the loading dose, 24-hour maintenance therapy and treatment of one (recurrent) convulsion. Syringes, swabs, drip sets and fluids also contained in treatment packs;
- Calcium gluconate should always be available to manage toxicity
- Intra venous infusion of magnesium sulfate
  - Loading dose: 4 g by slow intra venous injection over a period not less than 5 minutes (preferably over 10 - 15 minutes)
  - Maintenance: 10 g in litre of sodium chloride 0.9%, given by intra venous infusion at a rate of 1 g per hour
- The intramuscular magnesium sulfate (Pritchard) regimen
  - Loading dose: 4 g by slow intra venous injection over a period not less than 5 minutes, then 10 g intramuscularly, 5 g by deep intra muscular injection into each buttock
  - Maintenance therapy: 5 g by deep intra muscular injection, 2.5 g in each buttock every 4 hours
  - Continue for 24 hours after last convulsion, or delivery.

- Recurrent convulsions
  - Magnesium sulfate
    - 2 - 4 g intravenously over 5 minutes
  - Give lower dose (2 g) if the patient is small and/or weight is less than 70 kg

- Monitoring during magnesium sulfate therapy
  - Continue with intra venous infusion or give the next intramuscular dose only if - Patellar reflexes are normal
  - Respiratory rate is > 16 cycles/minute
  - Urine output is > 25 mL/hour (or > 100 mL in 4 hours)
  - Consider reducing the dose if
    - Renal function is impaired
    - Respiratory depression occurs
  - Urine output is < 100 mL in 4 hours
  - More frequent monitoring is required in the first two hours

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  - Maintenance therapy: 5 g by deep intra muscular injection, 2.5 g in each buttock every 4 hours
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- Recent convulsions
  - Magnesium sulfate
    - 2 - 4 g intravenously over 5 minutes
  - Give lower dose (2 g) if the patient is small and/or weight is less than 70 kg

- Monitoring during magnesium sulfate therapy
  - Continue with intra venous infusion or give the next intramuscular dose only if - Patellar reflexes are normal
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  - More frequent monitoring is required in the first two hours

- ultrasound for another pelvic pathology
- Complications
  - Shock
  - Sterility (with the loss of both tubes)
  - Often requires blood transfusion (with its attendant cost and risk of blood-borne infections)
  - 5 - 20% risk of having another ectopic gestation

- Fatality
  - Requires a high index of suspicion particularly in the case of atypical, slow-leaking or chronic ectopic gestation where diagnosis could be difficult

- Differential diagnoses
  - For unruptured ectopic pregnancy:
    - Acute pelvic inflammatory disease
    - Adnexal torsion
    - Incomplete abortion
    - Endometriosis
    - Degenerating uterine fibroid
    - Acute appendicitis
    - Accidental ovarian cysts

- Investigations
  - Haemoglobin concentration/packed cell volume
    - Blood grouping and cross matching
    - Urinalysis
    - Ultrasound scan of the pelvis/abdomen
  - Serum h-B-HCG (where available) especially in silent cases
  - Paracentesis abdominis (should be considered)
  - Laparoscopy
  - Final arbiter when the diagnosis is in doubt

- Treatment objectives
  - Depend on the clinical subset
  - Preserve maternal life

- Acute ectopic
  - Immediate resuscitation (fluids/blood)
  - Stop haemorrhage; by surgery
  - Replace lost blood

- General principles and treatment modalities
  - Surgery
    - Salpingectomy (total or partial) for ruptured ectopic pregnancy
    - Partial salpingectomy if the remaining segment of the tube is about 4 cm long; this could be used for reconstructive surgery subsequently
    - Salpingostomy for unruptured cases
  - Non-surgical options
    - Used in unruptured cases: expectant management and medical agents
    - Expectant management
      - Monitor pregnancy by -hCG levels
    - Medical treatment
      - Methotrexate
      - Administered systemically or locally to induce

- **ECTOPIC PREGNANCY**

- **Introduction**
  - Pregnancy in which the conceptus implants either outside the uterus (falllopian tube, ovary or abdominal cavity) or in an abnormal position within the uterus (cornua, cervix, angular and rudimentary horn)
  - The most common surgical emergency in women in many developing countries
  - A substantial cause of maternal mortality
  - Rapidly with which haemorrhage and shock occur
  - Pre-rupture diagnosis is elusive, with consequent delay in surgical management

- **Clinical features**
  - The clinical subsets include:
    - Acute ectopic gestation
      - 25% or less of cases
    - Sub-acute ectopic gestation
      - 75% of cases
  - “Silent” ectopic/chronic ectopic gestation
  - Acute Ectopic Gestation
    - Amenorrhoea
      - Features of acute abdomen particularly lower abdominal pain
    - Vaginal bleeding or brownish discharge
      - Severe pallor
    - Shoulder tip pain
      - Difficulty with sitting on hard surfaces
      - Features of shock with cardiovascular collapse: hypotension and tachycardia
      - The uterus is slightly enlarged with tenderness on one side
      - Some advise that examination should be avoided if there is a strong suspicion of an ectopic pregnancy
      - Positive cervical excitation tenderness
  - Sub-acute Ectopic Gestation
    - Slow-leaking ectopic prior to rupture, with most of the signs and symptoms of acute ectopic gestation but in the mildest form
    - Asymptomatic
      - May just be picked up during a pelvic examination in the course of booking or antenatal clinic, or found on
dissolution of trophoblastic tissue (Ru 486)
- Hyperosmolar glucose solution, potassium chloride
- Auto transfusion
- During surgery for ectopic gestation; very important in developing countries
- Inadequate blood banking services
- The risks of transfusion with donated blood are avoided
- Use only fresh blood
- Counsel for contraception and advise to report immediately to the hospital if a pregnancy is suspected so that its site can be confirmed

HYPEREMESIS GRAVIDARUM
Introduction
A clinical situation in which vomiting in early pregnancy considered to be physiological becomes persistent or severe enough to disturb the patient's health and/or require hospitalization
- Occurs in approximately a third to 50% of women
- Often the first sign of pregnancy, beginning at about the 6th week and stops spontaneously before the 14th week
- Generally limited to the early morning but may occur at other times of the day
- Cause is essentially unknown, but hypotheses include
  - Hormonal: Increased sensitivity to placental hormones such as hCG, estrogen or progesterone
  - Psychogenic: The woman thinks she should have early morning sickness because generations before her have had it

Clinical features
- Persistent and severe vomiting that leads to electrolyte and nutritional derangements

Differential diagnoses
It is a diagnosis of exclusion. Concerted effort must be made to exclude the under listed causes of pathological vomiting:
- Multiple gestations
- Hydatidiform mole
- Malaria in pregnancy
- Gastrointestinal disorders:
  - Heartburn due to hiatus hernia: a common cause of vomiting in late pregnancy
- Enteritis
- Appendicitis
- Peptic ulcer disease
- Hepatitis
- Acute fatty liver of pregnancy
- pancreatitis
- Cholecsyctitis
- Urinary tract disorders: pyelonephritis

Acute polyhydramnios
- Commonly associated with monozygotic twinning and diabetic pregnancies
- Pre-eclampsia
- Accidents to ovarian cysts
- Ketoacidosis, electrolyte imbalance (alkalosis and hypokalaemia); vitamin deficiencies
  In neglected or poorly managed cases: Severe weight loss
  - Tachycardia
  - Hypotension
  - Oliguria
  - Neurologic disorders from vitamin B deficiency
  - Retinal haemorrhages
  - Jaundice (from hepatic necrosis)
  - Oesophageal tears and spontaneous rupture of the oesophagus
  - Mendelson's syndrome
  - Foetal loss
  - Maternal mortality

Investigations
- Full Blood Count with differentials
- Urea, Electrolytes and Creatinine
- Liver function tests
- Midstream urine for microscopy, culture and sensitivity
- Urinalysis for ketones
- Blood film for malaria parasites
- Ultrasound scan of the pelvis/abdomen

Management
- Admit: Start intake-output monitoring
- Intravenous fluid therapy to:
  - Correct electrolyte disturbances
  - Provide calories
  - Rehydrate the patient
  - Anti-emetics
  - Those which have been proven not to be teratogenic:
    - Meclizine 25 mg orally
    - Cyclizine 50 mg orally
    - Promethazine 25 mg orally
    - All of these are taken three times daily
  - Total parenteral nutrition
    - In severe cases
    - In persistent and intractable cases with significant maternal complications, termination of pregnancy may be considered

IMMUNIZATION SCHEDULES
Introduction
Tetanus immunization for the pregnant woman is geared towards protecting the mother (and baby) against tetanus

Tetanus Immunization Schedule in Pregnancy

<table>
<thead>
<tr>
<th>TIMING OF IMMUNIZATION</th>
<th>PROTECTION OFFERED</th>
</tr>
</thead>
<tbody>
<tr>
<td>1st dose at booking or on 1st contact</td>
<td>Confers no protection</td>
</tr>
<tr>
<td>2nd dose at 4 weeks after 1st dose</td>
<td>Confers protection for 3 years</td>
</tr>
<tr>
<td>3rd dose at 6 months after 2nd dose</td>
<td>Confers protection for 5 years</td>
</tr>
<tr>
<td>4th dose at 1 year after 3rd dose or in next pregnancy</td>
<td>Confers protection for 10 years</td>
</tr>
<tr>
<td>5th dose at 1 year after 4th dose or in next pregnancy</td>
<td>Confers protection for life</td>
</tr>
</tbody>
</table>

Immunization and Vitamin A Schedule

At Delivery
- Vitamin A to Mother
- BCG; POLIO; HBV

At Birth
- DPT; POLIO; HBV

6 Weeks
- DPT; POLIO

10 Weeks
- DPT; POLIO

14 Weeks
- DPT; POLIO

9 Months
- MEASLES; YELLOW FEVER; 1st Dose Vitamin A

15 Months
- Vitamin A

JAUNDICE IN PREGNANCY
Introduction
Usually indicates a liver/biliary disorder and becomes clinically apparent when the serum bilirubin exceeds 2 - 2.5 mg/dL.

Many indicators of liver disease in the non-pregnant state are normal findings in pregnancy. These include:
- Spider naevi
- Decreased plasma albumin
- Increased alkaline phosphatase
- Increased serum lipids
- Prothrombin time, transaminases and bilirubin are unaltered in normal pregnancy
- Jaundice occurs in about 1 in 1,500 - 2,000 pregnancies

Aetiology

Aetiology peculiar to pregnancy

- Hyperemesis gravidarum
- Pre-eclampsia and eclampsia as seen with HELLP syndrome
- Acute yellow atrophy (acute fatty liver in pregnancy; acute hepatic failure)
- Intra-hepatic cholestasis of pregnancy
- Cholestasis in pregnancy
- Gallstones

Aetiology not peculiar to pregnancy

- Viral hepatitis
- Haemolytic jaundice
- Adverse reactions to drugs e.g. chlorpromazine, tetracycline
- Congenital hyperbilirubinaeas such as Dubin-Johnson syndrome
- Liver cirrhosis

Clinical features

Acute yellow atrophy

A rare and serious disorder associated with high mortality
- Common in the order of 1: 10,000 pregnancies
- Unknown aetiology
- Typically noted in primigravidae, occurring after the 30th week or few days after birth
- The jaundice is classically obstructive
- Onset usually sudden with:
  - Abdominal pain (right upper quadrant)
  - Headaches
  - Nausea and vomiting
  - Progressive jaundice
- Encephalopathy

Histo1gy

Peribiliar fatty infiltration of the liver cells

There is no place for liver biopsy because of bleeding complications

Management

- Early diagnosis is mandatory
- Clinical features with evidence of deranged LFTs and renal failure
- Transfusion with blood, fresh frozen plasma, platelets as indicated
- Dialysis

Complications

Disseminated intravascular coagulopathy

Hypotension

Significant risk of maternal and foetal death due to:
- Maternal liver failure
- Metabolic disturbance
- Encephalopathy
- Overwhelming haemorrhage associated with clotting defects

Management

Careful maternal follow-up with LFTs and wellbeing (CTG) monitoring

If all is well induce at 38 weeks

Adult:
- Risk of recurrence is 50%

Child
- None is required

Asevere attack may influence foetal outcome

Hepatitis A virus does not affect the foetus

- Increased serum lipids
- Decrease itching and normalize liver function

Clinical features

Common in certain southern American countries particularly Chile

Presents commonly in late third trimester, after 36 weeks

Clinically significant because of its association with IUAR and IUPD (mechanism unclear)

It is not as a rule associated with maternal complications

Cholestasis of pregnancy

Uncommon, in the order of 1: 2,000 pregnancies

Common in certain southern American countries particularly Chile

Presents commonly in late third trimester, after 36 weeks

Clinically significant because of its association with IUAR and IUPD (mechanism unclear)

It is not as a rule associated with maternal complications

Clinical features

Generalized pruritus

Occurs foetal movements

Upper abdominal pain

Dark urine

Steatorrhea

Occasionally there is jaundice (particularly in the later stages of the disease)

Investigations

Liver function tests:
- Mildly deranged
- Serum bilirubin and bile salts may be elevated

Differential diagnoses

Viral hepatitis

Early HELPP syndrome

Acute fatty liver

Management

Careful maternal follow-up with LFTs

Foetal surveillance: by growth (serial USS biometry) and wellbeing (CTG) monitoring

If all is well induce at 38 weeks

Management of associated pruritus

(Difficult to manage)

Topical agents offer little help

- To bind bile salts
- Vitamin K
- To decrease bleeding tendencies
- (Colestyramine binds fat soluble vitamins)
- May offer brief respite

Ursodeoxycholic acid and colestyramine (orally)

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- Decrease itching and normalize liver function

Adult:
- 10 - 15 mg/kg daily in 2 - 4 divided doses

Child
- 1 month - 18 years: 10 - 15 mg/kg twice daily; total dose may be given in 3 divided doses

Recurrent

- Quite high

Prognosis

- Good

- Complete recovery in days to weeks

Dubin-Johnson syndrome

Intermittent bilirubinemia (conjugated)

Often chronic and familial

No itching, usually asymptomatic

Caue is unknown

Treatment

None is required

Intra-hepatic cholestasis of pregnancy

Also termed ‘recurrent obstructive jaundice’ or ‘idiopathic cholestasis’

Thought to be due to the effect of high estrogen levels on the liver, which results in decreased conjugation of bilirubin

At rare condition

- Incidence of 1: 500 pregnancies

- Measurably common seen in Scandinavians

- Its exact etiology is unknown

Clinical features

- Intense pruritis due to retention of bile salts

- The most common presenting symptom and may occur in the absence of other symptoms

- Onset of symptoms usually in the third trimester

Jaundice is not often seen

Investigations

Bilirubinuria

Elevated bile acids

Elevated alkaline phosphatase

Elevated liver transferase enzymes

Prothrombin time

Always exclude viral disease, gallstones and treatment with chlorpromazine

Complications

Maternal

Haemorrhage

Preterm labour

Steatorrhea

Foetal

Foetal distress

Still-birth

Perinatal death

Prematurity and its problems

Meconium staining of the liquor

Management

Careful maternal follow-up with LFTs

Foetal surveillance: by growth (serial USS biometry)

and wellbeing (CTG) monitoring

If all is well, induce at 38 weeks

Management of pruritus

- See Cholestasis of pregnancy

Recurrent

- Risk of recurrence is 50%

Can be precipitated by oestrogen-containing oral contraceptive pills

Viral hepatitis

The most common cause of jaundice in pregnancy, accounting for about 40% of the causes

Incidence during pregnancy is probably no more than in the normal population

Pregnancy does not alter the course of the disease

Hepatitis A virus does not affect the foetus

- Unlike other hepatotrophic viral infections, which carry a significant risk of vertical transmission (particularly in the third trimester)

A severe attack may influence foetal outcome

- Slight increase in premature labour and stillbirths (as seen in any severe medical illness)

Treatment

Avoid any further damage to the liver by drugs

Bed rest

Adequate nutrition

- Improved nutrition

- Dietary advice

- Avoid breastfeeding

If hepatitis B is present then the infant requires protection with immunoglobulins against HBsAg

- Hepatitis B immunoglobulin by intramuscular injection

Neonate:
- 200 units as soon as possible after birth

Child
- 1 month - 5 years: 200 units; 5 - 10 years: 300 units; 10 - 18 years: 500 units

Avoid breastfeeding

Delivery room personnel must exercise great care in dealing with these patients, as all their body fluids are highly infectious

Immediate delivery if hepatitis becomes fulminating

PELVIC INFLAMMATORY DISEASE

Introduction

- Ascending pelvic infection involving the upper genital tract
- Usually involves sexually transmitted organisms e.g. Neissera gonorrhoeae and Chlamydia trachomatis
- It may also be caused by organisms endogenous to the lower genital tract
- In severe cases, organisms may migrate via the peritoneum to the upper abdomen causing perihitic adhesions: the so-called “violin strings” (Fitz-Hugh-Curtis syndrome)
- Responsible for significant morbidity in women, accounting for about 30% of all gynaecological admissions in sub-Saharan Africa

It is thought that 3% of women have Pelvic
Inflammatory Disease (PID) during their lifetime

**Risk factors**

- Peak incidence between 15 - 25 years
- Multiplicity of sexual partners
- Use of intrauterine contraceptive devices
- Usually within the first 4 months of use

**Clinical features**

**Major criteria** (the Westrom triad):
- Cervical excitation tenderness
- Adnexal tenderness

**Minor criteria**
- Fever (38°C)
- Infection of the pelvic inflammatory disease (PID)
- Purulent vaginal discharge
- Adnexal mass

**Diagnosis**

Based on the presence of the Westrom triad of symptomatology **plus** one of the minor criteria

Confirmation by demonstration of causative organism(s) on microscopy, culture, and sensitivity testing

**Differential diagnoses**

- Acute appendicitis
- Ovarian cyst accident
- Endometriosis
- Urinary tract infections
- Renal disorders (e.g. nephrolithiasis)
- Pelvic adhesions
- Lower lobe pneumonia
- Ecopic gestation

**Complications**

- Pelvic abscess
- Septicaemia
- Chronic pelvic pain
- Ecopic gestation
- Infertility
- Fitz-Hugh-Curtis syndrome
- Recurrence (about 25% rates)

**Investigations**

- Packed cell volume
- Haemoglobin genotype
- Blood Group
- White Blood Cell count
- Electrolytes and Urea
- Midstream urine microscopy, culture and sensitivity
- Endocervical swab
- High vaginal swab culture: to exclude trichomoniasis, bacterial vaginosis
- Urethral swab
- Ultrasound scan: to exclude cyesis, ectopic gestation, adnexal mass (e.g. ovarian mass)

**Indications for admission**

- Uncertainty of diagnosis with or without clinical findings
- Intolerance of oral medication or non-response to outpatient therapy
- Presence of a pelvic mass
- Presence of an intrauterine device
- Upper abdominal pain
- Adherence to the treatment
- Pregnancy
- Nulliparity

**Treatment objectives**

- Rehydrate adequately
- Indicate the infecting organism(s)
- Prevent complications

**Drug treatment**

- Appropriate antibiotics for an adequate period
  - The antibiotic chosen should cover all possible causative organisms while awaiting culture/sensitivity results

- Outpatient therapy while awaiting culture results:
  - Ceftriaxone (or equivalent cephalosporin)
  - 1 g intramuscularly stat
  - Plus:
    - Doxycycline
      - 100 mg orally every 12 hours for 14 days
      - Plus or minus:
        - Metronidazole
        - 400 mg orally every 12 hours for 14 days
        - If no response in 48 - 72 hours
        - Admit, re-evaluate and give appropriate intravenous therapy

- Inpatient triple therapy:
  - Ceftriaxone/doxycycline/metronidazole
  - Or:
    - Clindamycin/gentamicin/metronidazole

- Triple antibiotic regimen to be continued for 48 hours after the patient improves clinically

- Subsequently, the patient should continue therapy with:
  - Doxycycline
    - 100 mg orally every 12 hours
    - Plus:
      - Metronidazole
      - 400 mg orally every 8 hours for 10-14 days

**Prevention**

- Encourage the use of barrier contraceptive with spermicides
- Modify risky sexual behaviour: avoid multiplicity of sexual partners
- Contact tracing: to break the existing chain of infection and prevent recurrence
- Prompt diagnosis and treatment to prevent long term complications

**R.A.P.E**

**Introduction**

Performance of the act of sexual intercourse by force, duress, intimidation or without legal consent (as with a minor)

A growing social disorder afflicting the poor and rich, alike, with devastating and longstanding emotional consequences for the afflicted, family and society at large.

An enormous societal problem that appears to be poorly recognized and grossly under-reported.

An average of one in five adult women may have experienced sexual assault during her lifetime.

Adult women are much more likely to be raped by a spouse, ex-spouse, or acquaintance than by a stranger.

The girl-child is much more likely to be raped by her father, uncle, brother, cousin, neighbour, school teacher, family driver, security personnel, and even faith-based instructor.

Mental illness, alcohol and drug abuse appear to be predisposing factors; neglect and inattention to the needs of the girl-child also contribute.

**Clinical features**

Indirect presentation

- Vague symptoms
  - Physical features:
    - Perineal pain
    - Bleeding per vaginam
    - Bruised face/body
    - Arthritis
    - Disordered gait
  - Psychological symptoms/disorders
    - Sadness
    - Depression
    - Reluctance to answer simple questions
    - Avoidance of eye contact
- School/work absenteeism

**Differential diagnoses**

- Vaginitis
- Threatened abortion
- Domestic violence
- Alcoholism
- Drug abuse
- Depression

**Investigations**

- Early
  - Vaginal/perineal swab for microscopy, culture and sensitivity
  - Semen: DNA analysis

- Late
  - Urinalysis; urine microscopy, culture and sensitivity
  - Pregnancy test (blood)
  - HIV screening

**Treatment objectives**

- Evaluate safety of the patient
- Assess and treat physical injuries
- Provide emotional support
- Assess and deal with the risk of sexually transmitted infections and pregnancy

**It is important to document clinical findings**

**Non-drug measures**

- Reassure patient
- Provide information about legal services
- Drug treatment
  - Treat physical injury (as appropriate)
  - Treat STIs, UTI (as appropriate)
  - Treat HIV infection (if detected); Post-exposure prophylaxis if clinical situation so requires
- Prevention
  - See section on HIV infection
  - Manage pregnancy (as appropriate)
  - Treat depression (if present)
CHAPTER 14: RESPIRATORY SYSTEM

ACUTE EPIGLOTTITIS

Introduction
A life-threatening, rapidly progressive cellulitis of the epiglottis that may cause complete airway obstruction.

Most common in children, in whom Haemophilus influenzae is the most common pathogen.

In adults, is often caused by Strept. pneumoniae and group A streptococci.

Clinical features

Nasal obstruction (usually alternating)

Supportive measures

Hospitalization may be necessary.

Drug treatment

Ceftriaxone: avoid in pregnancy and in patients with renal impairment.

Notable adverse drug reactions, caution

Cefuroxime: avoid in pregnancy and in patients with renal impairment.

Ceftriaxone: rash, fever, gastrointestinal disturbances.

Prevention

Complete airways obstruction and asphyxiation.

Investigations

Lateral X-ray of the neck.

“Thumb sign” appearance of the enlarged epiglottis.

Blood culture.

Do not view the epiglottis using a tongue depressor: this may cause laryngospasm, with complete respiratory obstruction.

Treatment objectives

Safeguard the airway.

Control infection.

Drug treatment

Cefuroxime.

Adult: 250 mg orally every 12 hours for 5 - 10 days.

Child: 125 mg orally every 12 hours for 5 - 10 days.

Or: Ceftriaxone.

Adult: 250 - 500 mg intramuscularly or intravenously for 5 - 10 days.

Child: neonate, infuse over 60 minutes, 20 - 50 mg/kg daily (maximum 50 mg/kg daily).

Child: under 50 kg: 20 - 50 mg/kg daily by deep intramuscular injection or by intravenous injection over 2 - 4 minutes, or by intravenous infusion; up to 80 mg/kg daily in severe infections.

Supportive measures

Oxygen.

Steam inhalation.

Nasotracheal intubation may be required.

Notable adverse drug reactions, caution

Cefuroxime: avoid in pregnancy and in patients with renal impairment.

Ceftriaxone: rash, fever, gastrointestinal disturbances.

Dose reduction in the elderly.

Haemophilus influenzae vaccine.

Should be available as part of childhood immunization.

Cough: unproductive, or productive of scanty sputum.

Wheezing.

Tachypnoea.

Tachycardia.

Effects of nebulized epinephrine last 2 - 3 hours; the child should be monitored carefully for recurrence of the obstruction.

Treatment objectives

Relieve nasal mucosal oedema and obstruction.

Relieve pain/discomfort.

Treat complications.

Drug treatment

Analgesics.

- Paracetamol.

Adult: 1 g orally three times daily to relieve headaches or fever.

Child 1 - 5 years: 120 - 250 mg; 6 - 12 years: 250 - 500 mg; 12 - 18 years: 500 mg 4 - 6 hourly (maximum 4 doses in 24 hours).

- Antipyretics.

- Only if secondary bacterial infection occurs.

Supportive measures

Steam inhalation with a drop of eucalyptus oil.

Notable adverse drug reactions

Paracetamol: raised liver enzymes, renal papillary necrosis.

BRONCHIAL ASTHMA

Introduction

Chronic inflammatory disease of the airways that is characterized by hyper-responsiveness of the tracheo-bronchial tree to a multiplicity of stimuli.

Caution

- Effects of nebulized epinephrine last 2 - 3 hours; the child should be monitored carefully for recurrence of the obstruction.

ACUTE RHINITIS (Common cold)

Introduction

Inflammation of the mucosal surface of the nose, most commonly due to infection with respiratory viruses.

Clinical features

Tickling sensation in the nose associated with itching of the nose and palate.

Watery nasal discharge (rhinorrhoea), which may later become purulent.

Sneezing.

Headaches.

Nasal obstruction (usually alternating).

Differential diagnoses

Chronic bronchitis.

Left ventricular failure.

Glottic dysfunction with respiratory obstruction.

Recurrent pulmonary emboli.

Eosinophilic pneumonia.

Carcinoid tumour.

Complications

Spontaneous pneumothorax.

Pneumo-mediastinum.

Atelectasis.

Investigations

Diagnosis is based on:

Airway reversibility to inhaled β2 adrenergic agonist.

Isocapnoeic response to hyperventilation of cold air.

Sputum eosinophilia.

Chest radiograph: hyperinflation.

Treatment objectives

Achieve a stable asymptomatic state.

Maintain the best pulmonary function possible.

Drug treatment

Acute asthma episodes:

Nebulised salbutamol.

Adult and child over 18 months: 2.5 mg repeated up to 4 times daily; may be increased to 5 mg if necessary.

Child under 18 months: 1.25 - 2.5 mg up to 4 times daily.

- More frequent administration may be needed in severe cases.

Intravenous aminophylline.

Adult: 250 - 500 mg slowly (with close monitoring) over 20 minutes.

Child 1 month - 18 years: by intravenous injection of 5 mg/kg (maximum 500 mg), and then by intravenous infusion.

Intravenous steroids.

- Adequate hydration.

- Oxygen.

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Manifests physiologically by wide-spread airway narrowing and clinically by paroxysmal attacks of dyspnoea, cough and wheezing.

Acute episodes are interspersed with symptom-free periods.

Clinical features

Episodic dyspnoea.

Cough: unproductive, or productive of scanty sputum.

Wheezing.

Tachypnoea.

Tachycardia.

Pulse paradoxus in severe attacks.

Mildly raised blood pressure.

Rhonchi: inspiratory and expiratory.

Prolonged expiration.

Silent chest (an ominous sign).

Differential diagnoses

Chronic bronchitis.

Left ventricular failure.

Glottic dysfunction with respiratory obstruction.

Recurrent pulmonary emboli.

Eosinophilic pneumonia.

Carcinoid tumour.

Complications

Spontaneous pneumothorax.

Pneumo-mediastinum.

Atelectasis.

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Intravenous steroids.

- Adequate hydration.

- Oxygen.
### Supplemental oxygen

- Hydration
- Education on care and precipitating factors

### Notable adverse reactions, caution

- In all cases, prescribers/dispensers should consult product literature to confirm the strengths of various aerosol preparations
- Aminophylline
- Do not exceed 500 mg in 24 hours because of the risk of cardiac arrhythmias
- In case of CNS stimulation with insomnia and convulsions

### Bronchitis

**Introduction**

- Abnormal and permanent dilatation of medium sized bronchi
- A consequence of inflammation and destruction of the structural components of the bronchial wall, caused by bacterial or viral infections

**Clinical features**

- Persistent or recurrent cough
- Purulent fetid sputum
- Haemoptysis
- Pleuritic chest pain
- With or without a history of preceding pneumonic illness
- Digital clubbing
- Crepitations, rhonchi and wheezes
- Cor pulmonale and right ventricular failure in chronically hypoxic patients

**Differential diagnosis**

- Pulmonary tuberculosis
- Lung abscess
- Chronic bronchitis
- Bullous emphysema

**Complications**

- Massive haemoptysis
- Lung abscess
- Myotic brain abscess
- Pulmonary amyloidosis
- Ventilatory failure
- Cor pulmonale and right ventricular failure

**Investigations**

- Chest radiograph: cystic spaces with air-fluid levels
- Bronchography: saccular, cylindrical or varicose

**Chronic obstructive airways disease (COAD)**

- Two extreme types of COAD are recognized although there is a lot of overlap

**Clinical features**

- Depending on the predominant syndromes, could be described as follows:
  - **Pink puffers**: Slowly progressive dyspnoea
  - **Cough with scanty sputum**: Aesthetic features
  - **Barrel-shaped chest**: Wheeze
  - **These patients mainly have emphysema**

- **Blue bloaters**: Prolonged periods of cough and copious sputum

**CHEST PAIN**

**Introduction**

- A common clinical symptom that may or may not have significant clinical implications

**Clinical features (with differential diagnoses)**

- Sharp, lancinating lateral chest pain, worse with breathing and coughing: pleurisy
- Dull aching lateral chest pain: chest wall pain, pleural effusion

- Central chest pain precipitated by a dry harking cough: suggestive of tracheitis or tracheobronchitis
- Central chest discomfort/pain with sensation of heaviness or chest compression: suggestive of myocardial ischemia
- Lateral burning chest pain associated with tenderness on physical contact: Bornholm’s disease

**Investigations**

- Chest radiography
- Electrocardiography
- Echocardiography

**Treatment objectives**

- Treat primary cause
- Relieve pain

**Drug treatment**

- Non narcotic analgesics
  - Paracetamol
- Adult: 1 g orally every 8 hours
- Child 1 - 3 months: 30 - 60 mg every 8 hours; 3 - 12 months: up to 120 mg every 4 - 6 hours; 1 - 5 years: 120 - 250 mg every 4 - 6 hours; 6 - 12 years: 250 - 500 mg every 4 - 6 hours; 12 - 18 years: 500 mg every 4 - 6 hours
- Non-steroidal analgesics
  - Diclofenac sodium
  - Adult: 25 - 50 mg orally three times (depending on severity)
  - Child 6 months - 18 years: 0.3 - 1 mg/kg by mouth or by rectum 3 times daily (maximum total dose 150 mg daily)

**Pain of more serious aetiology e.g. pain of lower or upper respiratory tract infection, or pain of myocardial ischaemia**

- Refer to an appropriate specialist
Supportive measures
- Assisted ventilation
- Hydration
- Pulmonary physiotherapy

Differential diagnoses
- Avoidance of cigarette smoking
- Avoid / remove atmospheric pollutants

COUGH
Introduction
The explosive expiration that clears the tracheobronchial tree of secretions and foreign particles or noxious gaseous materials
- A defensive reflex reaction
- Comes to medical attention only when it becomes troublesome, affects life style and/or when there is concern about its cause

Clinical features
- Cough may be:
  - Acute or chronic
  - Seasonal
- Associated with breathlessness and or wheezing
- Productive of sputum: note colour, smell; haemoptysis
- Associated with fever
- Associated with chest pain: note location and character of pain
- Associated with risk factors, e.g. cigarette smoking
- Associated with the use of drugs for other illnesses
- Associated with other constitutional symptoms

Differential diagnoses
- Triggers of cough may rise from the upper or lower airways, or lung parenchyma
- Upper airways:
  - Inhaled irritants: dust, fumes, smoke
- Upper airways secretion
- Antigens
- Lower airways:
  - Inflammation
  - Viral bronchitis
  - Bronchiectasis
- Bacterial infection
- Bronchial asthma
- Endobronchial tuberculosis
- Bronchial infiltration/compression
- Parenchymal lung disease
- Pneumonia
- Infections
- Interstitial or endobronchial oedema due to heart disease

Drugs:
- ACE inhibitors
- Lung abscess
- Interventions
- Macroscopic and microscopic examination of sputum
- Sputum culture

Non-drug measures
- Exclude tuberculosis if cough is chronic
- Sputum cytology for malignant cells
- Chest radiograph where indicated
- HIV screen if history and clinical features are suggestive
- Treatment objectives
- Identify and treat the underlying cause(s)
- Abolish cough

Non-drug measures
- Adequate hydration to prevent inspissation
- Encourage expectoration for productive cough
- Do not use antussives unless cough is dry, unproductive and distressing

Drug treatment
- Cough suppressants: for dry, unproductive cough
- - Codeine cough linctus

Dyspnoea
Introduction
An abnormal and uncomfortable awareness of breathing
- Effort of breathing is out of proportion with exertion needs
- Patients often have difficulties in describing the discomfort of dyspnoea

Clinical features
- May depend on the underlying cause(s) of dyspnoea

Differential diagnoses
- Pulmonary:
  - Obstructive airways disease: asthma, chronic bronchitis, emphysema
- Parenchymal lung disease: pneumonia, pneumoconiosis, pulmonary fibrosis
- Pulmonary vascular obstruction: pulmonary emboli
- Chest wall disorders: respiratory muscle paralysis, kyphoscoliosis

Cardiogenic:
- Congestive cardiac failure
- Left ventricular failure

Metabolic:
- Diabetic ketoacidosis

Neurogenic:
- Anxiety

Treatment objectives
- Treat cause(s) of dyspnoea
- Restore normal respiration

Non-drug treatment
- Oxygen in appropriate concentration
- Other treatment will depend on the underlying/precipitating cause

Lung abscess
Introduction
Suppurative of the lung parenchyma
- May be due to:
  - Infection by aspirated oro-pharyngeal anaerobes
  - Inadequately treated pneumonia caused by Staphylococcus aureus, Mycobacterium tuberculosis
  - Bronchial obstruction

Clinical features
- Symptoms are indolent lasting several weeks:
  - Cough, with purulent offensive sputum
  - Fever, chills
  - Night sweats

Differential diagnoses
- Localized bronchiectasis
- Pneumonia
- Tuberculosis

Complications
- Cerebral abscess
- Empyema
- Pulmonary amyloid

Investigations
- Sputum: Gram stain and culture (during symptom exacerbation)
- Electrocardiogram
- Airways reversibility test
- Maintain optimal level of oxygenation and ventilation
- Supplemental oxygen, at 24 - 28% or 1 - 2 litres/minute
- Treat infections
- Reverse airways obstruction
- Clear airways secretions

Drug treatment
- Long acting β - agonist
  - See bronchial asthma
- Theophylline
  - Aminophylline (see bronchial asthma)
- Antibiotics (when necessary to control infection)
  - Erythromycin

Adults and child over 8 years: 250 - 500 mg orally every 6 hours, or 500 mg - 1 g every 12 hours (up to 4 g daily in severe infections)
- Child: 2 - 8 years: 250 mg orally every 6 hours
- Up to 2 years: 125 mg every 6 hours
  - Co-amoxiclavulanate

Adults: 500/125 mg orally every 12 hours
Child 1 month - 1 year: 0.25 mL/kg of 125/31 mg suspension orally every 8 hours; dose doubled in severe infections
  - 1 - 6 years: 5 mL of 250/62 mg suspension every 8 hours; dose doubled in severe infections
  - 6 - 12 years: 5 mL of 250/62 mg suspension every 8 hours; dose doubled in severe infections
- Restorils mg strength tablet every 8 hours, daily increased in severe infection to one 500/125 strength tablet every 8 hours daily

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Exclude tuberculosis if cough is chronic
- Sputum cytology for malignant cells
- Chest radiograph where indicated
- HIV screen if history and clinical features are suggestive
- Treatment objectives
- Identify and treat the underlying cause(s)
- Abolish cough

Non-drug measures
- Adequate hydration to prevent inspissation
- Encourage expectoration for productive cough
- Do not use antussives unless cough is dry, unproductive and distressing

Drug treatment
- Cough suppressants: for dry, unproductive cough
  - Codeine cough linctus

Adults: 5 - 10 mL, 3 - 4 times daily
years: half adult dose
Or: Amoxicillin/clavulanic acid
Adult: 1 g/200 mg orally every 8 hours for 7 - 10 days (Definitive antibiotic therapy should be based on culture and sensitivity results)
- Good dental care
- Adequate treatment of acute pneumonia
- Prevent pneumonia with vaccination in persons at risk
- HIV infected patients who are still capable of responding to a vaccine challenge
- Patients with recurrent sinusopulmonary infection
- Patients with or acquired hypogammaglobulinaemia

PNEUMONIA
Introduction
An inflammation of the lung parenchyma
Various bacterial species, fungi and viruses may cause pneumonia
The setting in which infection is acquired could be a predictor of the infecting pathogen
Streptococcus pneumoniae is the most common pathogen in community-acquired pneumonia
Other causative organisms:
- Haemophilus influenzae
- Mycoplasma pneumoniae
- Pseudomonas aeruginosa (usually implicated in nosocomial pneumonia)

Clinical features
Typical pneumonia:
- Sudden onset fever, chills and rigors
- Cough with purulent sputum production
- Pleuritic chest pain
- Breathlessness with short inspiratory efforts
Signs:
- Fever
- Herpes labialis
- Tachypnoea
- Signs of lung consolidation
- Pleural friction rubs
Atypical pneumonia:
- Gradual onset
- Dry cough
- Prominent extra-pulmonary symptoms
- Headache
- Sore throat
- Fatigue
- Myalgia
- Chest crackles or rales

Differential diagnoses
Pulmonary embolism
- Haemorrhage

Complications
Lung abscess
- Pleural effusion
- Empyema thoracis
- Septicaemia
- Endocarditis
- Meningitis

Investigations
- Spirometry examination
- Haematochemical evaluation
- Sputum culture
- Chest radiograph
- Blood cultures
- Serologic studies

Treatment objectives
- Eliminate the infection
- Return to normal lung function

Drug treatment
- Antibiotics
- Co-amoxiclavulanate

Adult: 1 g/200 mg orally every 12 hours for 5 - 7 days
Child: neonate and premature infants, 25 mg/kg every 6 - 12 hours; infants up to 3 months, 25 mg/kg every 8 hours; 3 months to 12 years, 25 mg/kg every 8 hours increased to 25 mg/kg every 6 hours in more severe infections
Or:
- Benzyl penicillin
  Adult: initially 1.2 g (2 million units) intravenously every 6 hours
  Child: preterm and neonate under 7 days, 25 mg/kg by intramuscular injection or by slow intravenous injection or infusion every 12 hours; dose doubled in severe infection

Sudden death
Sudden onset dyspnoea

Tachypnoea
Tachycardia

Small volume pulse
Hypotension
Circulatory collapse
Raised jugular venous pressure

Small-to-moderate embolus:
- Cough
- Pleurtic chest pain
- Haemoptysis

Pulmonary embolism
Introduction
Occurs when a venous thrombus is dislodged from its site of formation (thrombotic embolus) or a fat globule from a long bone fracture or crush tissue injury or even a tumour fragment (non-thrombotic embolism), is carried in the blood stream to the pulmonary arterial circulation causing obstruction to alveolar perfusion

Clinical features
Massive embolus in main pulmonary artery:
- Sudden death
- Sudden onset dyspnoea
- Tachycardia

Non-drug treatment
- Temporary measures:
  - Supplemenal oxygen
  - Psychological support

Drug treatment
Anticoagulants
- Heparin

Adult: 5,000 units (10,000 in severe pulmonary embolism) loading dose then continuous infusion at a rate of 15 - 25 units/kg/hour
Child: neonate, initially 75 units/kg (50 units/kg if under 35 weeks post-menstrual age), then 25 units/kg/hour by intravenous injection, adjusted according to APTT
1 month - 1 year: same as for neonate
1 year - 18 years: initially 75 units/kg by intravenous injection, then 20 units/kg/hour by continuous intravenous infusion, adjusted according to APTT
Or:
- Enoxaparin

Adult: 1.5 mg/kg (or 150 units/kg) by subcutaneous injection every 24 hours, for at least 5 days (until adequate oral anticoagulation is established)
Child: neonate, 1.5 - 2 mg/kg by subcutaneous injection twice daily; 1 - 2 months: 1.5 mg/kg twice daily; 2 months - 18 years: 1 mg/kg twice daily
- Warfarin

Adult: initially 10 mg orally daily for 2 days
Child: neonate (under specialist advice), 200 micrograms/kg once daily as a single dose on first day, then on the following 2 days
1 month - 18 years: 200 micrograms/kg (maximum 10 mg) as a single dose on first day, reduced to 100 micrograms/kg (maximum 5 mg) once daily for following 2 days
- Usual maintenance dose: 100 - 300 micrograms/kg once daily
- Subsequent doses depend on prothrombin time (INR)
- Thrombolytic agents
- Recombinant tissue plasminogen activator

Adult: 10 mg by intravenous injection given over 1 - 2 minutes; then intravenous infusion of 90 mg given over 2 hours
- Not exceeding 1.5 mg/kg in persons less than 65 kg
CHAPTER 15: INJURIES AND ACUTE TRAUMA

**Bites and Stings**

**Introduction**

Bites occur from:

- Humans
- Domestic animals such as cats and dogs
- Wild animals e.g. snakes, sharks and crocodiles
- Stings often occur from:
  - Bees, wasps and other insects
  - Marine invertebrates such as the jellyfish, corals, scorpions and anemones
- The microbiology of bite wound infections reflects the oro-pharyngeal flora of the biting animal
- Organisms from the soil, skin of the animal and victims, animal feaces may also be present

**Clinical features**

Depend on the type of injury, and the delay before presentation in hospital

- Bites from common domestic animals usually result in bruises, lacerations and haemorrhage;
- Rabies may complicate dog bites

**Dog bites**

- Responsible for 80% of bite wounds
- Bacteriology usually mixed
- Alpha haemolytic streptococci, pasteurella species, staphylococci, Eikenella corrodens, actinomyces, fusobacterium, prevotella, popyhomonas species, Capnocytophaga canimorsus
- 15 - 20 % of wounds become infected
- Lower limbs are most commonly affected

Infections occur 8 - 24 hours after bite and may manifest as:

- Pain
- Fever
- Lymphadenopathy
- Cellulitis
- If the canine tooth penetrates synovium or bone:
  - Septic arthritis
  - Osteomyelitis

**Cat bites**

- Less common
- More than 50% result in infection
- Females are more affected than males
- The hands and arms are more commonly affected
- Usual organisms include *P. multocida* and those ones following dog bites

**Rats, mice, gerbils and animals that prey on them**

- May transmit *Streptobacillus moniliformis* or *Spirillium minor*
- Usually affect hunters or laboratory handlers of rats
- Manifests as:
  - Fever
  - Chills

**Human bites**

- May be:
  - Self-inflicted
  - Sustained by medical personnel caring for patients
  - Sustained during fights, rapes or during sexual activity
- May become infected more than bites from other animals

- The oral microflora include multiple species of aerobic and anaerobic bacteria
- Those of hospitalized and debilitated patients often
- Include *Enterobacteriaceae*
- HIV, HBV have been reported due to human bites

**Snake bites**

- In Africa, often occur among farmers who walk unshod
- Bites occur from:
  - Poisonous snakes belong to the families of:
    - Viperidae: *Viperidae* and *crotalidae*
    - Elapidae: *Elapidae* (e.g. cobras)
    - Hydroidae (sea snakes)
- Stings often occur from:
  - Bees, wasps and other insects
  - Marine invertebrates such as the jellyfish, corals, scorpions and anemones
- They leave their stinging apparatus behind in the skin
- The symptoms that follow bee stings are those due to anaphylaxis to their venom

**Infections occur 8 - 24 hours after bite and may manifest as:**

- Pain
- Swelling
- Bruising
- Tender enlargement of regional lymph nodes
- Systemic effects:
  - Early anaphylactoid symptoms
  - Transient hypotension with syncope
  - Angioedema
  - Urticaria
  - Abdominal colic
  - Diarrhoea
  - Vomiting
  - Late persistent or recurrent hypotension
  - Electrocardiograph abnormalities
  - Spontaneous systemic bleeding
  - Coagulopathy
  - Adult respiratory distress syndrome
  - Acute renal failure

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- Inhibition of peripheral nerve impulses
- Multisystem effects
- Rhabdomyolysis
- Haemolysis
- Blood vessel damage
- Neurotoxic effects
- Snake bite wounds may become secondarily infected with:
  - *Clostridium tetani*, causing tetanus
  - *Clostridium welchi*, causing gas gangrene

**Indications for antivenom treatment**

- Hypotension
- Vomiting
- Hand or foot bite swellings extending beyond the wrist or ankle within 4 hours of the bite
- Electrocardiograph abnormalities

**Sharks and crocodiles**

- Cause death by:
  - Tissue destruction
  - Crush syndrome
  - Haemorrhage
- Infection

- Bees and wasps
- Are the most common causes of stings
- They leave their stinging apparatus behind in the skin
- The symptoms that follow bee stings are those due to anaphylaxis to their venom

- Marine invertebrates
- Have specialized organelles called nematocysts for poisoning and capturing prey
- May cause serious ill health and death

**Initial assessment**

- Careful history
- Contact local authorities to determine if the specie is rabid; if possible locate animal for observation
- Antibiotic allergy, immunization of patient and other morbid condition(s) should be documented
- Inspect wound for evidence of infection.
- Conduct general physical examination, including vital signs

**Investigations**

- Depend on the type of injury, the clinical presentation and the onset/type of complications:
  - Full Blood Count
  - Electrolytes and Urea
  - Blood clotting profile
  - Arterial blood gas estimations
  - Chest radiographs
  - Wound and blood cultures

**Treatment objectives**

- Neutralize envenomation
- Limit systemic effects
- Local wound care
- Prevent onset of complications
Chapter 15: Injuries and Acute Trauma

Adrenaline (epinephrine), hydrocortisone must be immediately on hand for the treatment of anaphylaxis if it occurs

**Prevention**
- Appropriate clothing and footwear while outdoors
- Attention and care to observe general safety measures

**BURNS**

**Introduction**
A common form of trauma in our environment
Involves coagulative necrosis of tissue cells following various insults
- Flames
- Chemicals
- Electricity
- Friction
- Cold or hot fluids

The various types occur with varying frequencies in various segments of the population
- For example scalds occur with great frequency in children while flame burns occur commonly in young adults

**Clinical features (and complications)**
Extensive skin loss with dehydration
Airway burns leading to dyspnoea, tachypnoea, stridor, hypoxia, hypercarbia, airway obstruction and death
Breathing difficulties from circumferential chest burns
Acute respiratory distress syndrome, acute lung injury and pulmonary oedema
Massive fluid losses from evaporation and interstitial fluid shifts leading to hypovolaemic shock
Acute renal failure from pre renal failure, acute tubular necrosis, and the crush syndrome
Electrolyte abnormalities: hyper or hypokalaemia with cardiac dysrhythmias and/or arrest
Anaemia from destruction of red cells. Also nutritional anaemia

**Hypothermia**
Immune dysfunction
Burns wound sepsis and septicemia
Tetanus
Acute gastric dilatation
Stress ulcerations in the gastrointestinal system
Limb compartment syndrome

**Crush syndrome**
Deep vein thrombosis
Systemic Inflammatory Response Syndrome (SIRS)
Organ Dysfunction Syndrome (MODS)

**Investigations**
- Full Blood Count
- Electrolytes and Urea
- Grouping and cross-matching
- Arterial blood gases
- Chest radiograph

**Treatment**
Copiously irrigate the wound with cold water (not ice cold) for 10–15 minutes
Avoid hypothermia and the use of agents such as raw eggs and palm oil
- They are not useful and may promote wound sepsis

**In the hospital perform a quick primary survey**
- **Check:**
  - Airway
  - Breathing
  - Circulation
  - Disability
  - Exposure
- Correct problems identified
- Give patient 100% oxygen

**Posterior**
Pass an endotracheal tube if there is risk of airway obstruction
Obtain specimens for investigations as detailed above
Determine percentage total body surface area (TBSA) burned
- Wallace rule of nines is recommended in adults
- In children there are several charts e.g. Lund and Browder charts

Calculate the total fluid requirement in the first 24 hours using appropriate formulae
- We recommend the Parkland's 
- Obtain the AMPLE history

**Allergies, Medications, Past medical history, pregnancy, Last meal**

**Environment** (including details of the incident)

**Prevention**
Administer tetanus prophylaxis depending on immune status
Apply relevant splintage

Wound swab for microscopy, culture and sensitivity
Blood culture
Intracompartmental pressure monitoring

**Treatment objectives**
At the scene: to stop the burning process or remove victim from the burn situation
Transport the patient to hospital as soon as possible

**In the hospital identify life threatening injuries and treat**
- Perform a detailed survey
- Restore patient's physiology as much as possible
- Promote wound healing
- Prevent complications

**Rehabilitation**

**Prevention**
- Commence prophylaxis against deep venous thrombosis
- Physiotherapy
- Decide whether patient should go to a burns unit or burns centre following standard criteria

**Drug treatment**
- Oxygen
- Tetanus toxoid
- Anti tetanus serum, antitetanus globulin as appropriate
- Narcotic analgesics e.g. morphine, pethidine, tramadol
- Nonsteroidal anti inflammatory analgesics e.g. diclofenac
- H1 receptor antagonists e.g. ranitidine
- Prophylactic antibiotics e.g. cephalosporins

**Topical wound dressing agents e.g. with zinc oxide based creams, antibiotic-containing dressings**

**Prevention**
Health education to promote healthy life style and avoidance of risky behaviour
Installation of fire warning systems such as smoke detectors in buildings
- Control of petroleum products
- An efficient fire service
- Fire protocols in all establishments

**DISASTER PLAN**

**Introduction**
A disaster is an event which causes serious disruption to community life, threatens or causes death or injury in that community, and/or damage to property

It is beyond the day-to-day capacity of the prescribed statutory authorities and requires special resources other than those normally available to those authorities
Could arise from natural causes such as cyclones, earthquakes and tsunamis or from man-made situations such as plane crashes and wars

**Human/Rapid or no warning**
- Only well-prepared systems will be able to limit the damages and losses that follow disasters
- The effectiveness and quality of response to a disaster is highly dependent on the level of preparation
- An ill-prepared system will lead to an ineffective and uncoordinated response
- Apart from an effective response, other advantages of preparation include cost savings and an improved and alert system

**There are four phases of disaster management:**
- **Prevention**
- **Preparation**
- **Response**
- **Recovery**

**Prevention**
Essentially the evolution and implementation of strategies to prevent or mitigate the impact of disasters
**Preparation**
Involve system upgrade, overhaul, protocol design, implementation and quality assessment for disaster management.

**Response**
Involves the interaction of the various emergency response agencies to the disaster to save as many casualties as possible; quick transfer to hospitals, coordination of the hospitals and creation of temporary shelters.

**Recovery**
A phase that involves rebuilding, reconstruction and rehabilitation, with a goal to restoring the community to its pre-event state or as close to it as possible.

For a disaster plan to be effective it needs to involve all the stakeholders in its design.

Disaster plan is necessary at various levels of health care and political terrain: national, regional, state and local government levels.

There should be disaster plans within organizations such as the hospitals, fire service, Army, Air force and Navy; the Ministries of health, the police and the Emergency Medical Service (EMS).

There is need for a coordinating agency such as the National Emergency Management Agency (NEMA) to supervise, monitor and coordinate inter-agency procedures, protocols, joint training sessions and drills.

Personnel in all the relevant response agencies must be familiar with the policies, protocols and procedures to be implemented following a disaster.

**Training and retraining is essential**

**The hospital disaster plan**
There should be a Disaster Committee in the hospital which should:
- Design a disaster plan for the hospital.
- Put in place procedures and protocols to be implemented in a disaster situation.
- Supervise staff training for disaster management.
- Be engaged in capacity building.
- Promote staff awareness regarding disaster prevention and preparation.
- Promote inter-departmental interaction regarding disaster management.
- Determine staff competency levels in disaster management.
- Allocate staff roles in disaster management.
- Ensure regular drills, seminars, tabletop exercises, computer simulations and interactions on disasters.
- Ensure stockpile of drugs and equipment to be mobilized in disaster situation.
- Ensure quality assurance and audit.
- Promote inter-hospital and inter-agency interaction within the municipality with regard to disaster management.

**Committee composition**
The committee should be composed of the following:
- The Hospital Trauma Director
- The Emergency Department Chief
- The Chief of Surgery
- The Chief of Anaesthesia
- The Chief of Nursing services
- The Head of Security
- The Head of Stores
- The Head of Pharmacy
- The representative of the Hospital Manager

The disaster protocol in the hospital should address the following principal issues:
- Who activates the disaster protocol?
- What are the criteria for activation?
- Information relay to critical departments: laboratories, blood bank, theatres, ICU, radiology, anaesthesia, Emergency Department (ED) Management, Hospital Management, Portage and Security.
- Pattern of staff call up to the Emergency Department in a disaster situation.
- Method of staff call.
- Pre-determined plan for Emergency Department evacuation.
- Information to the staff.
- Departmental disaster procedures.
- Logistic issues in a disaster situation.
- “Standing down” criteria and procedure.

**Head Injury**

**Introduction**
The term refers to any injury to the head.
- Includes bruises and lacerations to the scalp.
- For practical purposes it is preferable to talk of:
  - Traumatic brain injury (TBI).
  - Craniofacial injury.
  - Craniofaciocerebral injury.

- This section will focus on TBI.
- TBI is common in trauma patients.
- Present in up to 50% of multiply injured patients.
- Isolated TBI is uncommon.
- In up to 50% of cases of severe TBI there is multisystem trauma.

**Classification**
Can be considered from the point of view of:
- Mechanism of injury.
- Severity of injury.
- Morphology.
- Mechanism:
  - Blunt or penetrating.
  - Depends on the patient’s position on the Glasgow Coma Scale (GCS).

**Pathophysiology**
The brain is covered by the meninges: dura, arachnoid and pia mater with the subdural and the subarachnoid spaces.
- The normal circulating volume of CSF is 140 mL.
- The brain normally regulates its blood flow by a process of autoregulation, which is for the most time undisturbed in TBI.
- Normal CBF is 800 mL/min or 20% of total cardiac output.
- CBF = CPP/CVR = 50 mL/100 g of brain tissue/min.
- CPP is Cerebral Perfusion Pressure.
- CVR is Cerebral Vascular Resistance.
- CBF = MAP - ICP.
- MAP is Mean Arterial Pressure.
- ICP is Intracranial Pressure.
- The normal ICP is 10 mmHg (136 mm HgO).
- Changes in intracranial volume result in compensation, with alterations in CSF volume and blood volume within the cranium but with minimal change in intracranial pressure.
- At some point minimal changes in volume result in geometric increases in ICP (The Monro-Kellie doctrine), and decompression occurs.
  - An expanding intracranial mass (such as a subdural haematoma) leads to:
    - Unal herniation through the incisura in the tentorium with compression of the oculomotor nerve and the motor tracts in the mid brain.
    - This leads to ipsilateral pupillary dilation and contralateral hemiparesis or hemiplegia.
    - In the Kernohan’s notch syndrome which occasionally occurs there is ipsilateral papillary dilatation and hemiparesis.

With progressive expansion of an intracranial mass the cerebellar tonsils eventually herniate through the foramen magnum (coning).

**Clinical features**
These patients may present with:
- Features of multisystem trauma.
- Altered level of consciousness.
- Skull fractures and mass effect from intracranial lesions.
- Features of raised intracranial pressure.
- Headaches.
- Nausea.
- Projectile vomiting.
- Drowsiness.
- Papilloedema.

**Complications of TBI**
- A lucid interval (often occurs in extradural haematoma).
- Post injury, the patients maintain a satisfactory level of consciousness until suddenly consciousness is lost.

**Extradural haematoma**
- Rare; overall, occurs in less than 1% of head injuries.
- More common in young patients.
- Often results from torn middle meningeal vessels.
- CT shows a biconvex or lenticular opacity.

**Subdural haematoma**
- More common.
- Occurs in 20 - 30% of severe head injuries, more commonly in the elderly (due to brain atrophy).
- Results from torn bridging veins.
- The opacity on CT follows the contour of the brain.

**Basal skull fracture**
May be suggested by:
- Periorbital ecchymosis (raccoon eyes).
- Retrauorical ecchymosis (Battel sign).
- CSF leaks.
- Facial nerve palsy.

**Complications of TBI**
- Early:
  - Coma.
  - Post concussion headaches.
  - Post traumatic amnesia.
  - Retrograde amnesia.
  - Abnormalities of salt and water metabolism such as diabetes insipidus and syndrome of inappropriate ADH.
  - Anterior pituitary dysfunction such as ACTH abnormalities and poor cortisol stress response.
- Late:
  - Chronic subdural haematoma.
  - Infections such as meningitis and brain abscess.
  - Hydrocephalus.
  - Epilepsy.
  - CSF leaks.
  - Carotico-cavernous fistulae.
  - Traumatic aneurysms.
  - Chronic headaches.
  - Personality changes.

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13 - 12: mild
8 or less: severe
- Morphology:
  - Skull fractures
  - Intracranial lesions
  - - Fractures could involve the vault or base of the skull.
  - - Fractures may be linear, stellate, depressed or non-depressed; open or closed.
  - - Basilar fractures may be with or without CSF leaks and also with or without facial nerve palsy.
  - - Intracranial lesions may be focal or diffuse.
  - - Focal lesions include epidural, subdural and intracerebral haematomas.
  - - Diffuse lesions include concussions and diffuse axonal injury (DAI).

- This is associated with hypertension and bradycardia (Cushing’s reflex).
- Sequentially apnoea, arrhythmias, hypotension and death ensue.

- Clinical features
  - These patients may present with:
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    - CSF leaks.
    - Carotico-cavernous fistulae.
    - Traumatic aneurysms.
    - Chronic headaches.
    - Personality changes.
Chapter 15: Injuries and Acute Trauma

Treatment objectives
- Identify life threatening injuries and treat
- Limit primary injury
- Prevent secondary brain injury
- Provide critical care
- Rehabilitate

Primary survey
- Assess airway and maintain patency
- Suctioning and manoeuvres to elevate the tongue (jaw thrust and chin lift) may be useful
- A patent airway is important in optimizing outcomes in TBI
- Ventilation is next addressed
- Administer 100% oxygen
- Hypoxia is one of the causes of secondary head injury and must be avoided
- Hypotension is a cause of secondary brain injury and must be avoided
- Intra venous lines should be set up; administer crystalloids
- Assess the GCS and the state of the pupils
- Expose the patient to perform a quick general examination but avoid hypothermia.

Secondary Survey:
- (See section on multiple injuries)
- Secondary brain injury
- Cerebral injury that is not present at the time of the primary insult but develops in response to subsequent intracranial or extracranial events

Extracranial causes:
- Hypoxia
- Hypotension
- Seizures
- Hyperthermia
- Hyponatraemia
- Hypoglycaemia
- Hyperglycaemia
- Intracranial causes:
- Extradural haematoma
- Subdural haematoma
- Intracerebral haematoma
- Cerebral oedema
- Cerebral contusion
- Hydrocephalus
- Meningitis
- Brain abscess

CT scan in TBI
- Has revolutionized the management of traumatic brain injury as it can readily diagnose intracranial haematomas and skull fractures
- In trauma it is advisable to do a non-contrast CT scan

Indications for CT scan
- GCS of 14 or less
- GCS of 15 with:
  - Loss of consciousness >5 minutes
  - Amnesia for injury
  - Focal neurological deficit
  - Signs of calvarial or basal skull fracture

Intracranial pressure monitoring
- Best done through a ventriculostomy catheter, with or without concomitant intraparenchymal transducer

Indications for ICP monitoring in TBI
- Patients with post resuscitation GCS of 8 or less
- Intubated patients in ICU
- Patients with intracranial haematomas but are adjudged not to need surgery

Emergency management of raised intracranial pressure
Endotracheal intubation
- Controlled ventilation to a pCO2 of 35 mmHg

Volume resuscitation
- Maintain normal blood pressure

Narcotic sedation
Neuromuscular blockade
Bolus mannitol (1g/kg)
- See Meningitis

Head up tilt at 30 degrees
Controlled hypothermia

Surgery in TBI
- Often indicated in head injury for the evacuation of intracranial haematomas or elevation of depressed skull fractures
- Indications may depend on the centre and the neurosurgeon, but all agree that an intracranial haematoma causing significant mass effect should be removed
- A midline shift of more than 5 mm is considered significant

Indications for surgery will depend on:
- The neurological status of the patient
- Findings on CT
- Extent of intracranial injury
- Intracranial pressure

The procedures include:
- Burr holes
- Craniotomy
- Craniectomy
- Elevation of depressed skull fractures
- Drainage in TBI
- Diuretics to reduce intracranial pressure e.g. mannitol (see Meningitis)
- Sedatives e.g. diazepam (see Tetanus)
- Muscle relaxants e.g. diazepam, suxamethonium
- Mannitol 20% and tamsulos et al. e.g. phenytoin, phenobarbital (see Epilepsy)
- Antibiotics as appropriate

Multiple Injuries
Introduction
- The multiply injured patient is that patient with injury to more than one organ system
- Often victims of motor vehicle crashes, motor bike accidents, pedestrians hit by cars, or falls from heights
- Present a challenge to the managing team in terms of priority of medical intervention
- If the priorities are not well ordered the results can be catastrophic
- Difficult to outline clinical features for these patients as virtually any injury is possible

Treatment objectives
- Identify life threatening injuries and treat
- Identify all injuries, institute primary management and limit progress of injuries and further tissue damage
- Restore patient's physiology paying special attention to the triad of hypothermia, acidosis and coagulopathy
- Format a prioritized plan of definitive treatment and rehabilitation

Management
- Advanced trauma life support (ATLS) principles should apply
- Patient should be received by a trauma team consisting of at least:
  - A trauma team leader
  - An airway and a procedure doctor
  - Two nurses in similar capacity
  - A radiographer
  - A scrub nurse
  - A social worker
- It is important that hospitals which regularly manage trauma patients should maintain a standing trauma team on a 24-hour basis
- This helps to optimize outcomes in patient management

Prehospital INFORMATION
- The trauma team needs this information from theprehospital team

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- Relayed in the MIST format, preferably before the patient's arrival to enable adequate preparation to be made beforehand

M: Mechanism of injury
- I: Injuries sustained
- P: Prehospital vital signs: pulse, blood pressure, respiratory rate, oxygen saturation, temperature
- T: Treatment given e.g. cervical collar, intravenous fluids etc

Primary survey
- Quick survey to identify life threatening injuries and treat
  - Airway
  - Talking? Assume airway is alright. If not suction, Guedel's airways
  - Careful with airway manoeuvres such as the jaw thrust and chin lift
  - Always protect the cervical spine
  - Apply rigid cervical collar
  - May need endotracheal intubation.

Breathing
- Check the breathing, respiratory rate, oxygen saturation
- Examine the chest:
  - Tension pneumothorax? Haemothorax? Flail chest?
- Chest tube decompression?
  - Always obtain a chest radiograph before decompression if possible
  - Perform arterial blood gas estimations

Circulation
- Equally important in optimizing outcomes
- Hypotension is a cause of secondary brain injury and must be avoided

- Intravenous lines should be set up; administer crystalloids
- Assess the GCS and the state of the pupils

- Expose the patient to perform a quick general examination but avoid hypothermia.

- Secondary Survey:
  - (See section on multiple injuries)
  - Secondary brain injury
  - Cerebral injury that is not present at the time of the primary insult but develops in response to subsequent intracranial or extracranial events

- Extracranial causes:
  - Hypoxia
  - Hypotension
  - Seizures
  - Hyperthermia
  - Hyponatraemia
  - Hypoglycaemia
  - Hyperglycaemia

- Intracranial causes:
  - Extradural haematoma
  - Subdural haematoma
  - Intracerebral haematoma
  - Cerebral oedema
  - Cerebral contusion
  - Hydrocephalus
  - Meningitis
  - Brain abscess

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- Has revolutionized the management of traumatic brain injury as it can readily diagnose intracranial
Chapter 15: Injuries and Acute Trauma

 Secondary survey

 This is a total body examination to detect injuries sustained
 Involves obtaining the AMPLE history (allergies, medications, past medical history, pregnancy, last meal, environment including details of the accident)
 - Check for scalp haematoma, lacerations, skull fractures, CSF leaks (rhinorrhoea, otorrhoea); facial fractures, raccoon eyes
 - Remove contact lenses; examine pupils, oral examination; Battle sign
 - Neck:
   - Perform a careful neck examination
   - Leave in collar if there is a high index of suspicion for cervical injury
 - Chest:
   - Inspect for dyspnoea, tachypnoea, chest movements, flail chest, open pneumothorax or obvious penetration
   - Palpate for chest expansion, crepitus (subcutaneous emphysema) and rib fractures
 - Assess position of the trachea and determine any tracheal shift
 - Determine percussion notes in both lung fields (dull in haemorrhax and hyperresonant in pneumothorax)
 - Auscultate for breath sounds and air entry
 Abdomen:
 - Examination findings often unreliable in the multiply injured patient
 - This may be as a result of altered sensorium due to head injury, inebriation or drugs, neurological injury, or distracting injury
 - There is need to augment examination with bedside investigations like FAST and DPL (Diagnostic Peritoneal Lavage) if indicated
 - In the haemodynamically stable patient the best imaging modality is the CT scan with contrast

 Head:
 - Check for scalp haematoma, lacerations, skull fractures, CSF leaks (rhinorrhoea, otorrhoea); facial fractures, raccoon eyes
 - Remove contact lenses; examine pupils, oral examination; Battle sign
 - Neck:
   - Perform a careful neck examination
   - Leave in collar if there is a high index of suspicion for cervical injury

 Acute abdomen

 An abdominal condition of sudden onset requiring immediate (urgent) attention

 Aetiology

 Surgical:
 - Inflammatory/infective conditions:
   - Acute appendicitis: the commonest cause of acute abdomen
   - Acute salpingitis: a common cause in sexually active young females
   - Acute cholecystitis
   - Acute pancreatitis
   - Acute diverticulitis: not very common in this environment

 These conditions usually begin with a localized peritonitis which progresses to generalized peritonitis if left untreated.

 Perforation of hollow viscera:
 - Perforated chronic duodenal ulcer
 - Perforated typhoid ileitis: a common cause in this environment
 - Traumatic gastrointestinal perforation
 - Perforated gastrointestinal malignancies

 Intestinal obstruction:
 - Strangulated external and internal hernias
 - Intussusception
 - Peritoneal adhesions and bands (congenital or acquired)
 - Gastrointestinal tumours
   - Intra-abdominal haemorrhage
   - Trauma (injury to solid viscera e.g. spleen and liver)
   - Ruptured abdominal aortic aneurysm
   - Haemorrhage from tumours (e.g. primary liver cell carcinoma)

 Obstruction to urinary/biliary tract:
 - These usually present as colics due to stones
   - Ureteric colic
   - Biliary colic
   - Gynaecologic (outside those listed above)
   - Bleeding Graffian follicle
   - Twisted ovarian cyst
   - Ecopic pregnancy
   - Salpingitis
   - Degenerating fibroids

 Non-specific abdominal pain:
 - Includes a variety of conditions that do not come under the above causes
 - Medical:
   - Should always be borne in mind so as to avoid unnecessary surgery

 Haebrrocystogram to confirm urethral rupture
 - If not contraindicated pass an indwelling urethral catheter to monitor urinary output and tissue perfusion
 - Haematuria is suggestive of bladder or kidney injury

 Lower limb examination:
 - Check for obvious lacerations, deformity, fractures and dislocations
 - Undertake an appropriate neurovascular assessment
 - Assess muscle power in each limb
 - Same as for lower limb

 'LOG ROLL'

 The patient is now log rolled by four persons so as to examine the back

 The spine is examined from the occiput to the sacrum and is palpated for abnormalities.

 "A high riding prostate is suggestive of urethral rupture"

 Return patient to the supine position

 Neurological examination:

 - Perform a detailed neurological examination as indicated

 Clinical features

 Acute abdominal pain

 Note the following:
 - Location
 - Onset and progression
 - Nature and character
 - Aggravating and relieving factors
 - Abdominal distension
 - A past history of similar pain suggests complication of an underlying condition
 - In typhoid perforation, fever precedes abdominal pain, while the reverse is true for acute appendicitis

 Gynaecologic history:

 - In every female, the following should be ascertained
   - Last menstrual period: this will help in the suspicion of ectopic gestation and bleeding Graffian follicle

 Urinary symptoms:

 - Ascertain the presence or absence of the following
   - Pain on micturition
   - Pus in urine or cloudy urine
   - Loin pain

 Medical:

 - Diabetes mellitus
 - Sickle cell disease

 Infectious and infestations:
 - Lower lobe pneumonia
 - Gastroenteritis
 - Malaria

 Parasitic infestations

 Acute abdominal pain

 Note the following:
 - Location
 - Onset and progression
 - Nature and character
 - Aggravating and relieving factors
 - Abdominal distension
 - A past history of similar pain suggests complication of an underlying condition
 - In typhoid perforation, fever precedes abdominal pain, while the reverse is true for acute appendicitis

 Nausea and vomiting:

 - A frequent finding
 - Common in intestinal obstruction

 Altered bowel habits:

 - Diarrhoea may suggest an infective/inflammatory condition
 - Constipation occurs in intestinal obstruction and late in peritonitis
 - The presence or absence of blood, mucus in stool should be ascertained

 Fever:

 - An early feature in inflammatory/infective conditions
 - A late feature in most other causes of acute abdomen

 Gynaecologic (outside those listed above)
 - Bleeding Graffian follicle
 - Twisted ovarian cyst
 - Ecopic pregnancy
 - Salpingitis
 - Degenerating fibroids

 Non-specific abdominal pain:
 - Includes a variety of conditions that do not come under the above causes
 - Medical:
   - Should always be borne in mind so as to avoid unnecessary surgery

 Previous medical history:

 - Diabetes mellitus
 - Sickle cell disease

 Physical examination:

 General examination

 - Dehydration
 - Temperature (the exact temperature should be taken with a thermometer: oral, axillary or rectal temperature)

 - Pallor

 Standard Treatment Guidelines for Nigeria 2008

 Metabolic disorders:
 - Diabetes mellitus
 - Porphyria

 Haematologic conditions:
 - Sickle cell disease
 - Leukaemia

 Infections and infestations:
 - Lower lobe pneumonia
 - Gastroenteritis
 - Malaria
 - Parasitic infestations

 Acute abdomen

 Note the following:
 - Location
 - Onset and progression
 - Nature and character
 - Aggravating and relieving factors
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 Gynaecologic history:

 - In every female, the following should be ascertained
   - Last menstrual period: this will help in the suspicion of ectopic gestation and bleeding Graffian follicle

 Urinary symptoms:

 - Ascertain the presence or absence of the following
   - Pain on micturition
   - Pus in urine or cloudy urine
   - Urethral discharge

 Medical:

 - Diabetes mellitus
 - Sickle cell disease

 Infectious and infestations:
 - Lower lobe pneumonia
 - Gastroenteritis
 - Malaria

 Parasitic infestations
Evidence of adequate resuscitation
- Central venous pressure
- Pulmonary capillary wedge pressure
- Urine output, volume, colour
- Hydration status
- Skin turgor
- Sensorium

Biochemical tests:
- Urinalysis: test the urine for sugar, protein, ketones, etc.
- Random blood sugar to exclude diabetes mellitus
- Serum electrolytes and urea; correction may be needed
- Serum amylase to exclude acute pancreatitis

Haematological tests:
- Haemogram to exclude anaemia
- Packed cell volume may not be reliable because of haemoconcentration from dehydration
- If there is suspicion of sickle cell disease, the haemoglobin genotype should be obtained
- A complete blood count may show evidence of acute infection (leucocytosis, neutrophilia)
- Blood should be grouped, and compatible blood cross-matched and made ready

Other investigations:
- Computed tomography may be needed when there is diagnostic confusion
- Cultures: any suspicious fluid and materials should be obtained and sent for microbiology and culture (e.g. vaginal discharge, peritoneal fluid)

Differential diagnoses
- Follow a detailed evaluation (as above) and make a reasonable (probable) list of not more than 3 - 5 differential diagnoses

General measures
- Resuscitation
  - Rehydration and correction of electrolyte derangements
  - Correct shock by giving crystalloids (sodium chloride 0.9%, Ringer's lactate) or colloid (e.g. dextran)
  - Maintenance fluids are calculated based on degree of dehydration
  - Correct electrolyte deficits (especially potassium)
  - Nasogastric decompression: the largest possible size of tube for patient
  - Aspirate intermittently using low pressure suction or large syringe
- Urethral catheterization (to monitor urine output)
- Correct anaemia (by blood transfusion)
- Commence broad spectrum, intravenous antibiotics

Chest:
- Against likely microorganisms
- Do not give aminoglycosides until urine output is adequate
- Monitor the following parameters to ensure adequate rehydration:
  - Should help to ascertain the cause of pain in a proportion of the patients (e.g. cholecytitis, gynaecologic conditions, urinary calculi, and degenerating masses)

Intestinal obstruction
- A condition in which there is failure of onward propulsion of intestinal contents
- A common surgical emergency

Antimicrobial prophylaxis in surgery

Introduction
- Postoperative surgical site infection (wound infection) is a rather common, but undesirable occurrence in this environment
- Surgical site infection tends to increase postoperative morbidity and may lead to mortality
- Efforts therefore need to be made to prevent surgical site infection

Antibiotic prophylaxis is not a substitute for adherence to basic principles of surgical asepsis and meticulous attention to technical details

Objective of antibiotic prophylaxis
- To prevent postoperative infection in susceptible patients

Principles of antibiotic prophylaxis
- Should be used only where there is a high risk of bacterial contamination
- Intravenous route is preferred to achieve optimum effect
- Should be given not >2 hours before surgical incision

Complications
- Antibiotic misuse
- Antibiotic resistance
- Complications of antibiotics (e.g. pseudomembranous colitis)
- False sense of surgical security

Antibiotic prophylaxis should be effective and efficient

INTESTINAL OBSTRUCTION

Introduction
- A condition in which there is failure of onward propulsion of intestinal contents

Aetiology
- Mechanical (dynamic):
  - Extra-luminal (compression from outside the intestinal wall)
General measures

Haematological:
- Haemogram: Complete blood count (leucocytosis and neutrophilia suggest strangulation)
- Group and cross match blood and store appropriately

Ultrasonography
- May be helpful in some instances to identify the cause of obstruction
- In difficult cases, other investigations may be necessary depending on the presentation and clinical suspicion
- Avoid contrast studies (as much as possible) in acute intestinal obstruction

Resuscitate:
- Rehydrate and correct electrolyte deficits (especially potassium)
- Nasogastric decompression using a wide bore nasogastric tube

Mechanical obstruction
- Gram negatives, gram positives

Should only be embarked upon after adequate resuscitation
- Most of the causes will require laparotomy
- Treat identified cause on its merits:
  - Gangrenous or perforated bowel: resect
  - Re-anastomose if patient is fit
  - Bring ends out as stomas if patient is too ill

Vomiting: usually bilious and occurs early in small intestinal obstruction
- A late symptom in large intestinal obstruction
- May be faeculent in advanced obstruction
- Constipation: occurs early in large intestinal obstruction and late in small intestinal obstruction
- Obstipation (non-passage of faeces or flatus) signifies complete obstruction
- Stools may be blood-stained (intussusception, volvulus, strangulation)
- Diarrhoea: may be present in the face of obstruction (spurious diarrhoea)
- Fever: signifies strangulation or perforation

Signs:
- General:
  - Dehydration
  - Pyrexia
  - Pallor
- Cardiorespiratory: assess the following
  - Lung fields
  - Pulse rate
  - Blood pressure
- Abdomen:
  - Distension: usually marked in large intestinal obstruction
  - Visible peristalsis
  - Only the intestinal lumen is affected; there is no evidence of strangulation

Strangulated obstruction
- Vascular compromise has occurred and may progress to gangrene and/or perforation

Closed loop obstruction
- A segment of intestine is blocked at 2 ends (e.g. colonic obstruction with competent ileocaecal valve, intestinal volvulus)
- Dangerous because the risk of perforation is high
- Irrespective of the cause or type of obstruction, the symptoms, signs and physiologic consequences are the result of the following
  - Stasis proximal to the level of obstruction (gases, fluid)
  - Dilatation above level of obstruction
  - Increased secretion from the involved segment(s)
- Compression of the veins and later arteries leading to ischaemia, gangrene, necrosis and perforation

The end results are:
- Dehydration
- Electrolyte derangements
- Anaemia
- Peritonitis
- Septicaemia

Clinical features
- Colicky abdominal pain: not a prominent symptom in adynamic obstruction
- Abdominal distension

Chapter 16: Surgical Care and Associated Disorders

PREOPERATIVE EVALUATION AND POSTOPERATIVE CARE

Haematological:
- Haemogram
- Complete blood count (leucocytosis and neutrophilia suggest strangulation)
- Group and cross match blood and store appropriately
- Ultrasoundography
- Useful in intussusception, suspected intra-abdominal tumours

Laparoscopy:
- May be helpful in some instances to identify the cause of obstruction
- In difficult cases, other investigations may be necessary depending on the presentation and clinical suspicion
- Avoid contrast studies (as much as possible) in acute intestinal obstruction

General measures
- Resuscitate:
  - Rehydrate and correct electrolyte deficits (especially potassium)
  - Nasogastric decompression using a wide bore nasogastric tube
  - Urinary catheterization to monitor urine output
  - Broad-spectrum intravenous antibiotics (anaerobes, gram negatives, gram positives)
  - Correct any anemia by blood transfusion

Definitive treatment
- Should only be embarked upon after adequate resuscitation

Mechanical obstruction
- Most of the causes will require laparotomy
- Treat identified cause on its merits:
  - Gangrenous or perforated bowel: resect
  - Small intestine:
    - Re-anastomose if patient is fit
    - Bring ends out as stomas if patient is too ill
- Large intestine:
  - Re-anastomose if on right side
  - Evacuate any peritoneal collection
  - Bring ends out as stomas if on left side
- Suspicious lesions: take specimens for histopathology

Non-mechanical (adynamic) obstruction
- Treating accordingly


Preoperative Evaluation

Introduction
The assessment of a patient before surgery to ensure that the patient is in optimal physiologic state and fitness for the surgical procedure
A most important aspect of the care of a surgical patient
No elective operation should be carried out without an adequate preoperative assessment
- In the emergency situation, all efforts must be made to ensure that the patient can withstand anaesthesia and the surgical procedure
- Occasionally (e.g. with severe on-going haemorrhage, airway obstruction) resuscitation, anaesthesia and surgery may commence simultaneously

Objectives of preoperative evaluation
- To detect any fluid and electrolyte derangements
- To detect any haematological derangements (e.g. anaemia, bleeding diathesis, sickle cell disease)
- To detect any coexisting medical conditions that may adversely affect the outcome of anaesthesia and surgery
- To detect any coexisting medical conditions that may adversely affect the outcome of anaesthesia and surgery
- All patients scheduled to have surgery should be in a haemodynamically stable condition before surgery

The above may not always be possible, but efforts must be made to improve cardiopulmonary and renal function
- Correct any detected abnormality
- Patient evaluation and correction of abnormalities may need to be done in conjunction with others: the anaesthetist, physician, paediatrician etc

Clinical evaluation
- Efforts should be made to identify the following by history and physical examination:
  - Cardiopulmonary disorders:
    - Cough
    - Chronic obstructive airways disease
    - Chronic obstructive airways disease
    - Hypertension
    - Cardiac failure
    - Metabolic disorders:
      - Diabetes mellitus
      - Haematologic disorders:
        - Sickle cell disease
        - Sickle cell disease
        - Allergy:
          - Drug allergies (e.g. penicillins, tetracyclines, antibiotics)
          - Drug allergies (e.g. penicillins, tetracyclines, antibiotics)
          - Propranolol, diuretics, steroids and other hormonal agents; prednisolone, oral contraceptives; tricyclic antidepressants
          - Social habits:
            - Cigarette smoking, alcohol use
            - Cigarette smoking, alcohol use
            - Previous anaesthetic experience:
              - How long ago, type of anaesthesia

Investigations
- Cardiopulmonary:
  - Chest radiograph: especially for patients 60 years and above, and those with chest infection
  - Look for evidence of chest infection and cardiomegaly
- Electrocardiogram: especially for patients over 60 years and those with heart disease or hypertension
- Pulmonary function tests may be necessary in patients with obstructive airways disease

Metabolic:
Airways management

The cardiopulmonary status (pulse rate, blood pressure, respiration) needs to be monitored very closely (every 15 minutes) in order to promptly detect any abnormality. Where available, electronic monitors with an alarm should be used.

The patient may still be under some effect of anaesthesia. Prevent the tongue from falling backwards by positioning it or using a nurse’s tongue depressor. This can occlude the airway. Suction should be continued as required.

Analgesia

Pain is a most undesirable effect of surgery. Patients should not be allowed to suffer from pain unduly. The appropriate analgesic technique should be chosen for the nature of surgical procedure performed. Adequate analgesia will ensure early ambulation and help to limit atelectasis.

Consent for surgery

Details of the surgery should always be explained to the patient (or relatives) in very simple language before surgery. Include a mention of the possible/common complications.

Postoperative Care

- Intramuscular vitamin K (10 mg daily), at least 48 - 72 hours before surgery
- Haemogram/packed cell volume
- Haemoglobin genotype
- Clotting profile (prothrombin time and kaolin cephalin clotting time) where there is suspicion of bleeding diathesis e.g. in jaundiced patients

- Other investigations as may be indicated by individual clinical circumstances

Correction of abnormalities and preparation for surgery

Cardiopulmonary:
- Rehydrate patient adequately, using appropriate fluids
- Control blood pressure
- Treat/control chest infections with appropriate antibiotics
- Control obstructive airways disease

Metabolic conditions and derangements:
- Correct electrolyte deficits, especially hypokalaemia
- Acidosis is usually corrected by adequate rehydration (provided the patient has no renal disease)

Diabetes should be controlled
- Patients already controlled will need their therapy to be converted to soluble insulin for long surgical procedures (this should be done in conjunction with the physician and anaesthetist)

Haematological:
- Correct anaemia
- Cause(s) of anaemia should be identified and treated
- The minimum haemogram for a patient undergoing elective surgery should be 10 g/dL.
- Haemogram 6 - 9 g/dL: correction may be achieved by haematinics; redserelude surgery
- Haemogram <6 g/dL: correction may require blood transfusion

Emergency surgery: correct anaemia by blood transfusion
- Blood transfusion should be avoided as much as practicable.
- Patients with sickle cell anaemia: haemogram should be brought up to 8 g/dL.
- These patients must be adequately hydrated to avoid sickling and sludging within the bloodstream.
- Short day case procedure: imperative to admit the patient with sickle cell anaemia at least a day before surgery to achieve adequate hydration.

Suspected bleeding diathesis
- Intramuscular vitamin K (10 mg daily), at least 48 - 72 hours before surgery
- For major surgery, blood should be grouped, cross-matched and stored

Other disorders:

Any associated medical condition should be treated / controlled before embarking on surgery
- This should be done in conjunction with the physician as much as possible
- Patients who require nutritional rehabilitation
- If surgery is elective reschedule it, and give adequate time to achieve improved nutritional status, otherwise morbidity and mortality may be increased

High-risk patients:
- At high risk of developing postoperative complications
- Deliberate and meticulous efforts should always be made to adequately evaluate them and ensure optimal fitness for surgery
- Elderly patients (age >60 years): - risk of deep vein thrombosis, atelectasis
- Obesity (risk of deep vein thrombosis, atelectasis, haemorrhage)
- Women on oral contraceptive pills-risk of deep vein thrombosis
- Co-existing chronic medical conditions-risk of wide ranging complications
- Sickle cell anaemia-risk of sickling crises, deep vein thrombosis

Consent for surgery

Details of the surgery should always be explained to the patient (or relatives) in very simple language before surgery.

Obtaining consent should be done by the surgeon himself.

Postoperative Care

Introduction

Meticulous and efficient care in the postoperative period is paramount for adequate patient recovery and success of surgery.

A well-planned and supervised postoperative care ensures a smooth recovery, and helps to prevent or limit postoperative morbidity and mortality.

Preoperative, intraoperative and postoperative care is a continuum and interlinked.
- Many of the instructions and therapy started in the preoperative period may need to be continued into the postoperative period.

The surgeon himself must be involved in the postoperative care and not leave it to others, who may not have much ideas or information about the surgery.

Initial recovery

- 4 - 6 hours before surgery
- First 4 - 6 hours after a major surgery and general anaesthesia are critical
- The patient is still drowsy and recovering from the effects of anaesthesia

The cardiopulmonary status (pulse rate, blood pressure, respiration) needs to be monitored very closely (every 15 minutes) in order to promptly detect any abnormality.

Where available, electronic monitors with an alarm should be used.

Airways management

The patient may still be under some effect of anaesthesia.
- Airways need to be kept patent
- Prevent the tongue from falling backwards by positioning patient in the left lateral position.
- The neck should be prevented from falling on itself as this can occlude the airway.

Secretions should also be cleared using a low-pressure suction.

Nursing position

Different operations require specific positioning in the postoperative period to reduce venous pressures, keep airway patent, enhance drainage etc.

The surgeon should be conversant with the specific positions and give appropriate instructions.

Analgesia

Pain is a most undesirable effect of surgery.

Patients should not be allowed to suffer from pain unduly. The appropriate analgesic technique should be chosen for the nature of surgical procedure performed.

Adequate analgesia will ensure early ambulation and help to limit atelectasis.

Minor/moderate surgery

Patient taking orally:
- Paracetamol
- Non steroidal antiinflammatory drugs

Patient not taking orally:
- Injectable nonsteroidal antiinflammatory drugs (e.g. diclofenac sodium)

Major surgery:
- Parenteral analgesics
- Narcotic analgesics (e.g. morphine)
- NSAIDs (e.g. diclofenac sodium)

Nasogastric decompression

The stomach may need to be kept decompressed for 24 - 48 hours, particularly following gastrointestinal surgery.

Decompression prevents abdominal distension and tension on abdominal fascial closure.

It also prevents splinting of the diaphragm and atelectasis.

The widest possible bore of nasogastric tube for patient's age should be chosen.

The nasogastric tube should be removed as soon as it is no longer needed, evidenced by:
- Progressively diminishing effluent (<500 mL/24 hours in an adult)
- Change from bilious colour to clear colour of gastric juice

Fluid and electrolyte balance

Ensure that the patient receives adequate amounts of intravenous fluids if oral intake is prohibited.

Check an appropriate fluid to provide enough calories and electrolytes.

Glucose 5% in sodium chloride 0.9% or lactated Ringer's solution is appropriate for most adults.

After the 48 hours, the daily requirement of potassium should be provided if oral intake is still prohibited, especially if nasogastric drainage is ongoing.

- This should be in form of potassium chloride added to intravenous fluids.
- Assess fluid and electrolyte balance on a daily basis and correct deficits.
- All intake (intravenous fluids, drugs, blood etc.) and output (urine, nasogastric drainage, other tubes etc.) as well as insensible losses should be carefully recorded.

Nutrition

Following major surgery, adequate nutrition should be provided for the patient, particularly if oral intake is going to be prohibited for more than 48 - 72 hours.

- This can be done in the form of parenteral nutrition.

Chest physiotherapy

Bedridden patients and patients who have had chest or upper abdominal surgery are prone to basal atelectasis and hypostatic pneumonia.

- These should be prevented by appropriate chest physiotherapy.
- Ensure adequate analgesia to enhance chest excursion.
- Encourage coughing and expectoration, with a hand supporting any abdominal wound.
- Periodic chest percussion to loosen bronchial secretions.
- Ambulate as early as possible.

Mobilization and ambulation

Mobilize and ambulate patients as early as is practicable to avoid the complications of prolonged recumbency.

Ambulation should be gradual; prop up in bed, sit out of bed, short walks etc.

Early ambulation should help prevent hypostatic pneumonia and deep vein thrombosis (very important in obese and elderly patients).

Antibiotics

Appropriate antibiotics as indicated.

Irational or indiscriminate use is not to be encouraged.

Wound care

Specific surgical wounds are cared for in different ways.

Clean surgeries: do not open wound (unless indicated) until day 5 - 7.

Inspect wounds immediately if there are features suggestive of surgical site (wound) infection.
- Undue pain
- Undue swelling
- Discharge of serosanguinous fluid or pus

Infected wounds:
- Wound swab for microbiological culture and sensitivity tests.

Adequate local wound care

Appropriate antibiotics

If there are systemic features (e.g. fever, anoxia)
Complications

Early complications:
- Immune reactions
- ABO incompatibility
- Rhesus incompatibility
- Febrile reactions
- Allergic reactions
- Reactions to plasma proteins

Biochemical complications:
- Hyperkalaemia
- Citrate toxicity (hypocalcaemia)
- Haemoglobinuria

Infective complications:
- Bacteraemia
- Transfusion of parasites (e.g. malaria)
- Transfusion of viruses (HIV, Hepatitis B, C, D)

Physical complications:
- Volume overload
- Air embolism
- Hypothermia

Complications of massive blood transfusion

Massive transfusion refers to the single transfusion of 50 - 100% of the equivalent of an individual's blood volume in less than 24 hours.

- 2.5 - 5 litres in adults and 40 - 80 mL/kg body weight in children

The complications are related to:
- Volume overload
- Transfusion of old blood
- Electrolyte derangements (especially potassium and calcium)
- Transmission of infections
- Delayed complications: Haemosiderosis
- Post transfusion purpura

Autologous transfusion

Transfusion of the patients' own blood

Advantages
- Reduced risk of transmitting communicable diseases
- Overcomes the problem of shortage of blood

Types and methods
- Pre-deposit blood
- Usually best done in conjunction with haematology staff
- The patient donates one unit of blood at a time (e.g. weekly) several weeks before the elective surgery
- Following donation, the patient is given haematinics, and sometimes erythropoietin to enhance bone marrow function; the blood is stored for later use
- Pre-operative isovolaemic haemodilution
- Just before elective surgery, 1 - 2 units of blood are taken from the patient and replaced by volume expanders such as Ringer's lactate, sodium chloride 0.9%, or colloid
- The blood taken is transfused intraoperatively after all haemostasis has been secured

Intraoperative blood salvage

CONTRAINDICATIONS TO AUTOLOGOUS TRANSFUSION

- Pregnancy
- Chronic medical conditions
- Cancer
- Situations where the blood may have become contaminated (this is for intraoperative blood salvage)

Children:
- Other sources of blood

Umbilical cord blood

Alternatives to blood transfusion

Since blood transfusion is attended by several untoward effects and complications, efforts are continuously being made to identify alternatives to transfusion.

- Most of these are experimental at the moment and are not practicable in the clinical setting

MEASLES (Rubella)

Introduction
- An acute viral infection caused by an RNA virus of the genus Morbillivirus in the family Paramyxoviridae
- Only one serotype is known
- Endemic throughout the world
- 30 - 40 million cases and 745,000 deaths for the year 2000
- 50 - 60% of estimated deaths due to vaccine-preventable diseases
- Also a major cause of preventable blindness

Transmission is by droplet infection during the prodromal stage.

Incubation period: 9 - 11 days

Time of exposure to appearance of rash: about 14 days

Clinical features
- The essential lesion is found on the skin, mucous membranes of the nasopharynx, bronchi, intestinal tract and conjunctivae

Three stages:
- Incubation period
- Prodromal stage with an enanthem
- Final stage

Incubation period: 2 - 3 days

Prodromal stage:
- Mild fever; 10 - 11 days
- Low grade to moderate fever
- Dry cough
- Coryza
- Conjunctivitis
- Koplik spots
- Photophobia

Final stage:
- Temperature rises abruptly as the rash appears
- Rash begins from the upper lateral part of the neck, behind the ears, along the hairline and posterior parts of the cheek then spreads to the rest of the body
- Rash fades in the same pattern in 3 - 4 days

Associated lymphadenopathy

Differential diagnoses
- Rubella
- Roseola infantum
- Infections from Echovirus, Coxsackie Virus and Adenovirus
- Infectious mononucleosis
- Toxoplasmosis
- Meningococcaemia
- Scarlet fever
- Rickettsial diseases
- Kawasaki disease
- Serum sickness
- Drug rashes
### Complications

<table>
<thead>
<tr>
<th>Condition</th>
<th>Description</th>
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<tbody>
<tr>
<td>Diarrhoea</td>
<td>An acute infectious disease of humans (particularly children) caused by any of three serotypes of poliovirus P1, P2, and P3.</td>
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<tr>
<td>Otitis media</td>
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<td>Pneumonia</td>
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<td>Laryngo-tracheobronchitis</td>
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<td>Encephalitis</td>
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<tr>
<td>Blindness</td>
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<tr>
<td>Subacute sclerosing panencephalitis</td>
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<tr>
<td>Investigations</td>
<td></td>
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<tr>
<td>Location of paralysis</td>
<td>Depends on region affected.</td>
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<tr>
<td>Abnormal sensation</td>
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<td>Hypertension</td>
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<tr>
<td>Hypercalcaemia</td>
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<tr>
<td>Nephrocalcinosis</td>
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<td>Vascular lesions</td>
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<td>Myocarditis</td>
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<td>Pulmonary oedema</td>
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<td>Pulmonary embolism</td>
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<td>Guillain-Barré syndrome</td>
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<td>Polio toxicity</td>
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<td>Cranial nerve Herpes zoster</td>
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<td>Post-diphtheric neuropathy</td>
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<tr>
<td>Rabies</td>
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<td>Tetanus</td>
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<td>Botulism</td>
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<tr>
<td>Encephalomyelitis</td>
<td></td>
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<tr>
<td>Acute porphyrias</td>
<td></td>
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<tr>
<td>Hysteria and malingering</td>
<td></td>
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<tr>
<td>Conditions causing pseudoparalysis</td>
<td></td>
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<tr>
<td>Unrecognized trauma</td>
<td></td>
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<tr>
<td>Transient toxic synovitis</td>
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<td>Acute osteomyelitis</td>
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<td>Acute rheumatic fever</td>
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<td>Scurvy</td>
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<tr>
<td>Congenital syphilis</td>
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<tr>
<td>Pulmonary embolism</td>
<td></td>
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<tr>
<td>Poliomyelitis</td>
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### Pathogenesis

- Entry into mouth (via faecally-contaminated food/water)
- Replication in pharynx, gastrointestinal tract, local lymphatics
- Haematologic spread to lymphatics and central nervous system
- Viral spread along nerve fibres
- Destruction of motor neurons

### Clinical features

- Incubation period: 6 - 20 days, with a range of 3 - 35 days
- Asymptomatic infection: 95%
- Minor non-specific symptoms: 4 - 8%
- Symptoms occur in less than 2%
- Slight fever
- Headache
- Malaise
- Sore throat

### Differential diagnoses

- Guillain-Barré syndrome
  - Lead toxicity
  - Cranial nerve Herpes zoster
  - Post-diphtheric neuropathy
  - Arthropod borne viral encephalitis
  - Rabies
  - Tetanus
  - Botulism
- Encephalomyelitis: demyelinating type
- Neoplasms in and around the spinal cord
- Polyneuritis: inflammatory type
- Polyneuritis: degenerative type
- Anti-GM1 ganglioside neuropathy

### Complications

- Multiple intestinal erosions
- Acute gastric dilatation
- Hyperparathyroidism
- Hypercalcaemia
- Nephrocalcinosis
- Vascular lesions
- Myocarditis
- Pulmonary oedema

### Treatment objectives

- Relieve symptoms
- Hydrate adequately
- Treat secondary bacterial infection
- Prevent complications

### Prevention

- Vaccination
  - The only effective method of prevention
  - Given at:
    - Birth
    - 6 weeks
    - 10 weeks
    - 14 weeks
  - Highly effective
  - 50% immune after 1 dose

### Drug treatment

- Beclometasone 5 - 10 mg orally 2.5 - 5 mg subcutaneously
- AlỞs 10 mg orally 5 - 10 mg subcutaneously
- Phenobarbitone 5 - 10 mg orally
- Diazepam 1 - 2 mg subcutaneously

### Notable adverse drug reactions

- Nausea, vomiting, drowsiness, bulging fontanelle, diplopia, papilloedema and cranial nerve palsies

### Hygienic practices

- To prevent / limit contamination of food and water by the virus

### Standard Treatment Guidelines for Nigeria 2008

- Poliomyelitis: Paralysis of limbs, muscles of respiration and swallowing which can be fatal
- Investigations
  - Viral isolation from stool, pharynx or cerebrospinal fluid
  - If the virus is isolated from a person with acute flaccid paralysis, it must be tested further, using fingerprinting or genomic sequencing to determine if it is the wild type or vaccine type
  - Serology: a fourfold rise in antibody may be demonstrated

### Non-drug treatment

- Allay fear
- Minimize ensuing skeletal deformities
- Anticipate and treat complications
- Prepare the child and family for a prolonged management of permanent disability if it seems likely

### Paralytic polio

- Symptomatic fever
- Headache
- Malaise
- Sore throat
- Muscle tenderness and spasms in any part of the body
- Neck pain and stiffness

### Paralytic polio

- 3 types depending on the level of involvement
  - Spinal in 79%
  - Bulbar polio: 2%
  - Bulbospinal: polio 19%
- Fever 5 - 7 days before other symptoms
- Headache
- Stiffness and back
- Asymmetric muscle weakness
- Rapid onset

### Paralytic polio

- Virus isolation from stool, pharynx or cerebrospinal fluid
- If the virus is isolated from a person with acute flaccid paralysis, it must be tested further, using fingerprinting or genomic sequencing to determine if it is the wild type or vaccine type
- Serology: a fourfold rise in antibody may be demonstrated

### Non-drug treatment

- Allay fear
- Minimize ensuing skeletal deformities
- Anticipate and treat complications
- Prepare the child and family for a prolonged management of permanent disability if it seems likely

### Paralytic polio

- Relief of symptoms
- Hydration
- Treatment of secondary bacterial infection
- Prevention of complications

### Drug treatment

- Beclometasone 5 - 10 mg orally 2.5 - 5 mg subcutaneously
- Al-Origin 10 mg orally 5 - 10 mg subcutaneously
- Phenobarbitone 5 - 10 mg orally
- Diazepam 1 - 2 mg subcutaneously

### Notable adverse drug reactions

- Nausea, vomiting, drowsiness, bulging fontanelle, diplopia, papilloedema and cranial nerve palsies

### Hygienic practices

- To prevent / limit contamination of food and water by the virus

### Prevention

- Vaccination
  - The only effective method of prevention
  - Oral Polio Vaccine
  - Given at:
    - Birth
    - 6 weeks
    - 10 weeks
    - 14 weeks
  - Highly effective
  - 50% immune after 1 dose
  - >95% immune after 3 doses
Chapter 17: Paediatric Perspectives

**Inactivated Polio Vaccine**

Given at:
- 2 months
- 4 months
- 12 months

- Highly effective
- >90% immune after 2 doses
- >99% immune after 3 doses
- Duration of immunity not known with certainty

**Notable adverse drug reactions, caution and contraindications**

Oral polio vaccine:
- Paralytic poliomyelitis

Should not be administered to persons who are immunocompromised (it is a live vaccine)

Contra indicated in:
- Persons with history of severe allergic reaction to a vaccine component or following prior dose
- Moderate or severe acute illness

**VITAMIN A DEFICIENCY**

Introduction

Vitamin A was the first fat-soluble vitamin to be discovered.

It comprises a family of compounds called the retinoids.

In nature, the active retinoids occur in 3 forms:
- Alcohol (retinol), aldehyde (retinal or retinaldehyde) and acid (retinoic acid)

In the human body, retinol is the predominant form, and 11-cis-retinol is the active form.

Retinol-binding protein (RBP) binds vitamin A and regulates its absorption and metabolism.

Vitamin A is essential for:
- Vision (especially dark adaptation)
- Immune response
- Epithelial cell growth and repair
- Bone growth
- Reproduction
- Maintenance of the surface linings of the eyes
- Epithelial integrity of respiratory, urinary, and intestinal tracts
- Embryonic development
- Regulation of adult genes

It functions as an activator of gene expression by retinoid alpha-receptor transcription factor and ligand-dependent transcription factor.

Deficiency of vitamin A is found among malnourished children, the elderly, and chronically ill populations in the United States, but it is more prevalent in developing countries.

Among the first signs of vitamin A deficiency (VAD) are:
- Abnormal dark adaptation
- Dry skin and dry hair
- Broken fingernails
- Decreased resistance to infections

**Epidemiology**

An estimated 250 million children in developing countries are at risk for vitamin deficiency syndromes.

The most widely affected group includes up to 10 million malnourished children who develop xerophthalmia and have an increased risk of complications and death from measles.

Each year 250,000 - 500,000 children become blind because of VAD.

Improving the vitamin A status of children (aged 6 - 59 months) with deficiencies can reduce rates of death from measles by 50%; from diarrhoea by 33%, and from all causes of mortality by 23%

**Pathophysiology**

Vitamin A deficiency may be secondary to:
- Decreased ingestion
- Defective absorption and altered metabolism
- Increased requirements

An adult liver can store up to a year's reserve of vitamin A, whereas a child's liver may have enough stores to last only several weeks.

Serum retinol concentration reflects an individual's vitamin A status.

Because serum retinol is homeostatically controlled, its levels do not drop until the body's stores are significantly limited.

The serum concentration of retinol is affected by several factors:
- Synthesis of Retinol Binding Protein in the liver
- Infection
- Nutritional status
- Adequate levels of other nutrients such as zinc and iron

**Recommended Daily Allowance**

**Child:**
- Infant (1 year or younger): 375 micrograms
- Child 1 - 3 years: 400 micrograms
- Child 4 - 6 years: 500 micrograms
- Child 7 - 10 years: 700 micrograms
- All males older than 10 years: 1000 micrograms
- All females older than 10 years: 800 micrograms

**Adult:**
- 3,000 microgram (10,000 IU) orally once daily for a minimum of 2 days
- Has been shown to reduce child mortality rates by 35 - 70%

**Iron panel**
- Useful because iron deficiency can affect the metabolism of vitamin A
- Serum albumin
- Levels are indirect measures of levels of vitamin A
- Full Blood Count with differentials
- If anaemia, infection, or sepsis is a possibility
- Serum electrolytes
- Liver function tests
- To evaluate bone growth and excessive deposition of periosteal bone

Clinical features

VAD may be asymptomatic.

Increased risk of respiratory and diarrhoeal infections.

Decreased growth rate.

Retarded bone development.

Increased fatigue as a manifestation of VAD anaemia.

Bitot spots.

Poor dark adaptation (nyctalopia).

Dry skin.

Dry hair.

Precarious.

Broken fingernails.

Keratomalacia.

Follicular hyperkeratosis (phrynoderma) from blockage of hair follicles with plugs of keratin.

Excessive deposition of periosteal bone secondary to reduced osteoclastic activity.

Anemia.

Keratinization of mucous membranes.

**Differential diagnoses**

- Cataract.
- Refractive errors.
- Zinc deficiency.

**Complications**

- Blindness.
- Corneal ulceration.

**Investigations**

- Serum retinol.
- - Costly but is a direct measure.
- - A value of less than 0.7 mg/L in children younger than 12 years is considered low.

- Serum RBP.
- - Easier and less expensive to perform than retinol.
- - Less accurate because levels are affected by serum protein concentrations; types of RBP cannot be differentiated.

- Serum zinc.
- - Useful because zinc deficiency interferes with RBP production.

**Drug treatment**

Daily oral supplements of vitamin A.

**Child:**
- Less than, or 3 years: 600 microgram (2,000 IU) orally once daily.
- 4 - 8 years: 900 microgram (3,000 IU) orally once daily.
- 9 - 13 years: 1,700 microgram (5,665 IU) orally once daily.
- All males older than 10 years: 2,800 microgram (9,335 IU) orally once daily.
- All females older than 10 years: 2,000 microgram (6,665 IU) orally once daily.

**Adult:**
- all ages: 3,000 microgram (10,000 IU) orally once daily.

**Severe disease**
- 60,000 microgram (200,000 IU) orally for a minimum of 2 days.
- Has been shown to reduce child mortality rates by 35 - 70%.

**Notable adverse drug reactions, caution**

Risk of teratogenicity increases in pregnant women at doses >800 micrograms/day (not recommended at these doses).
CHAPTER 18: EMERGENCIES

ACUTE LEFT VENTRICULAR FAILURE

Introduction
Sudden diminution in the function of the left ventricle
Pulmonary capillary and venous pressure increase beyond plasma oncotic pressure
There is resultant accumulation of oedema fluid in the pulmonary interstitial spaces and alveoli

Aetiology
Insipid left ventricular failure secondary to hypertension
Arhythmias
Myocardial infarction

Clinical features
Dyspnoea
Orthopnoea
Paroxysmal nocturnal dyspnoea
Cough
Hemoptysis
Restlessness
Wheeze
Hypoxia

Differential diagnoses
Pulmonary thromboembolism
Bronchial asthma
Pulmonary tuberculosis
Cardiac tamponade

Complications
Right-sided heart failure
Acute renal failure
Myocardial infarction

Investigations
Electrocardiography
Plain chest radiograph
Echocardiography
Cardiac catheterization
Pulmonary function tests
Arterial blood gases
Electrolyte, Urea and Creatinine

Treatment objectives
To improve pump performance of the failing ventricle
To reduce the cardiac workload
To control salt and water retention

Non-drug treatment
As in hypertension

Drug treatment
Diuretics
- Furosemide
Adult: 40 - 80 mg by slow intravenous injection stat
- Then 40 - 160 mg orally or intravenously daily in 1 or 2 divided doses for maintenance
Child: neonate, 0.5 - 1 mg/kg by slow intravenous injection every 12 - 24 hours (every 24 hours if post-menstrual age is under 31 weeks)
1 month - 12 years: 0.5 - 1 mg/kg (maximum 4 mg/kg), repeated every 8 hours as necessary
12 - 18 years: 20 - 40 mg every 8 hours; higher doses may be necessary in resistant cases
Angiotensin converting enzyme inhibitors
- Captopril
Adult: 6.25 - 12.5 mg daily orally, then 25 mg in divided doses daily (maximum 150 mg daily) for maintenance
Child: not licensed for use in children
Or:
- Lisinopril
Adult: 2.5 mg orally daily, 5 - 20 mg daily for maintenance
Child: neonate, initially 10 micrograms/kg orally once daily; monitor blood pressure carefully for 1 - 2 hours, increased as necessary up to 500 micrograms/kg daily in 1 - 3 divided doses
1 month - 12 years: initially 100 micrograms/kg orally once daily, monitor blood pressure carefully for 1 - 2 hours, increased as necessary up to a maximum of 1 mg/kg daily in 1 - 2 divided doses
12 - 18 years: initially 2.5 mg daily, monitor blood pressure carefully for 1 - 2 hours; usual maintenance dose 10 - 20 mg daily in 1 - 2 divided doses (maximum 40 mg daily if body weight is >50 kg)
May require morphine
Adult: 5 - 10 mg orally, subcutaneously or intramuscularly (usually a single initial dose)
Child: not listed for this indication
Digoxin
Adult: 125 - 250 micrograms orally daily may be required
Aminophylline
Adult: up to 250 mg by slow intravenous injection stat
Supportive measures
Oxygen
- Nurse in cardiac position

Notable adverse drug reactions, caution and contraindications
- Use ACE inhibitors, and aminophylline and digoxin with caution
- Monitor potassium levels closely
- Monitor fluid input and output

Prevention
Adequate control of hypertension

CARDIAC ARREST

Introduction
Sudden cessation of cardiac pump function
If there is no spontaneous reversal or resuscitative measure, death results
Commonest cause of cardiovascular deaths among causations
Peaks between ages 0 - 6 months and 45 - 75 years

Aetiology
Congenital and acquired structural defects of the heart
Abnormal electrical activities of the heart

Clinical features
Usually sudden collapse
Unrecordable blood pressure
Loss of peripheral pulses
Cessation of respiration
May be asymptomatic
Complaints may be non-specific
Presentation may be that of underlying cause

Differential diagnoses
Syncope
Seizures

Complications
Death
Sequela involving the vital organs
- Acute renal failure
- Myocardial infarction
- Cerebrovascular accident

Investigations (after the initial rapid assessment and resuscitation)
Electrocardiography
Echocardiography
Urea, Electrolytes and Creatinine
Lipid profile
Blood gases
Chest radiograph

Treatment objectives
Prompt restoration of cardiac and respiratory function
Monitoring of impact of cardiac arrest on the various associated organs
Intervention to restore normal functions
Formulation of a broader and more comprehensive diagnostic and treatment plan
Eliminate/control aetiological factor(s) in order to reduce morbidity/prevent mortality

Non-drug treatment
Ensure clear airway by tilting the head backwards, lifting the chin and exploring to remove foreign bodies/dentures
- Remove wears/ornaments which may negate the above

Basic life support (CPR)
Ensure that patient is lying on a firm/hard surface
Cardiac massage (80 - 100 per minute)
- Assisted ventilation using a masked ambu bag
- Twice in succession for every 15 cardiac massages (once every 5 massage when 2 people are in attendance)
- Watch out for spontaneous respiration during this exercise

Advanced life support
Intubation with an endotracheal tube
Defibrillation/cardioversion for patients with ventricular fibrillation/ventricular tachycardia

Inflammatory, infiltrative, neoplastic and degenerative processes
Fluids and electrolyte imbalances
Drugs and other substances of abuse
Sudden infant death syndrome

Miscellaneous

Prevention
Eat foods rich in vitamin A, in adequate amounts
Family and community health education

Contraindicated in
- Documented hypersensitivity
- Hypervitaminosis A
Parenteral vitamin A in infants of low birth weight may be associated with:
- Thrombocytopenia
- Renal dysfunction
- Hepatomegaly
- Cholestasis
- Ascites
- Hypotension
Metabolic acidosis (E-Ferol syndrome)
Clinical features
- Promoting potassium loss
- Limiting exogenous potassium intake
- Discontinuation of anti-kaliuretic drugs
- Shifting potassium into cells

Clinical features
- Shifting potassium into cells
- Right-to-left shunting of un-oxygenated blood
- Infusion of fresh and salt water
- Hypothesis of tissue hypoxia

Clinical features
- If alive, patient is unconscious and not breathing
- Hypoxemia and tissue hypoxia
- Acidosis
- Hypothermia
- Pneumonia
- Acute renal failure
- Hemolysis

Complications of near-drowning
- Hypoxic brain injury with cerebral edema (which may occur within 24 hours)
- Cardiac arrhythmias
- Dehydration
- Acute Respiratory Distress Syndrome (ARDS)
- Acute renal failure
- Disseminated Intravascular Coagulopathy

Investigations
- Full Blood Count; ESR
- Chest radiograph
- Electrolytes, Urea and Creatinine
- Liver function tests
- Acid base status evaluation
- Arterial blood gases
- Skull and spine radiographs
- CT Scan (if available)

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- Arterial blood gases
- Skull and spine radiographs
- CT Scan (if available)

DROWNING AND NEAR-DROWNING
Introduction
Refers to death by suffocation due to immersion in water.

Contributory factors
- Swimming in deep waters
- Failing unexpectedly into water
- Not being able to swim
- Breath-holding swimming and diving
- Alcohol consumption
- High water temperatures
- Easy, illicit access to pools
- Inadequate pool and spa covers
- Other antiarrhythmic drugs if necessary

Pathophysiology
- Inhalation of water results in ventilation-perfusion imbalance with hypoxaemia and pulmonary oedema

- Absorption of hypotonic fresh water results in collapse of the alveoli, resulting in right-to-left shunting of un-oxygenated blood

- Absorption of hypertonic salt water results in alveolar oedema, but the overall effects are the same for both inhalation of fresh and salt water

- Infection may develop subsequently and is more likely when contaminated water is inhaled

- Hemolysis

- Complications of near-drowning

- Hypoxic brain injury with cerebral edema (which may occur within 24 hours)

- Cardiac arrhythmias

- Dehydration

- Acute Respiratory Distress Syndrome (ARDS)

- Acute renal failure

- Disseminated Intravascular Coagulopathy

- Investigations

- Full Blood Count; ESR

- Chest radiograph

- Electrolytes, Urea and Creatinine

- Liver function tests

- Acid base status evaluation

- Arterial blood gases

- Skull and spine radiographs

- CT Scan (if available)

- Treatment objectives

- Immediate resuscitation and stabilization to prevent or minimize complications

- Non-drug measures

- Airway management

- Immobilize the cervical spine, as trauma may be present

- Treat hypothermia vigorously

- Endotracheal intubation with mechanical ventilation and positive end-expiratory pressure if patient is apneic or in severe respiratory distress or has oxygen-resistant hypoxemia

- Admission for observation for at least 24 hours if any of the complications are observed even if briefly

- Drug treatment

- Ventilate with 100% oxygen

- Mix an intravenous infusion with 0.9% saline or lactated Ringer's solution

- Manage pulmonary complications with the administration of 100% oxygen initially, titrated thereafter as required

- Review arterial blood gases

- Bronchodilators if bronchospasm is present

- Manage metabolic acidosis: give NaHCO3, if pH is persistently less than 7.2

ELECTROLYTE ABNORMALITIES
Introduction
Detection of deranged electrolytes and fluid balance does not constitute a diagnosis

- Efforts should be made to determine the underlying causes in every case

- Hyperkalaemia

- Plasma K concentration > 5 mmoles/L

Aetiology
- Usually occurs as a result of potassium release from cells

- Decreased renal excretion of K as in renal failure

- Decreased potassium secretion: Impaired sodium reabsorption in

- Primary hypoadosteronism

- Adrenal insufficiency

- Secondary hypoadosteronism

- Medications such as ACE inhibitors, NSAIDs and heparin

- Enhanced chloride reabsorption (chloride shunt) as seen in Gordon's syndrome

- Clinical features

- Weakness, flaccid paralysis, metabolic acidosis

- ECG changes

- Increased T wave amplitude

- Peaked T waves

- Prolonged PR intervals, QRS duration

- Atrioventricular conduction delays

- Loss of P waves

- Ventricular fibrillation or asystole

Investigations
- Serum Urea, Electrolytes and Creatinine

- Other renal function tests

- Acid base balance

Treatment objectives
- Correction of hyperkalaemia

- Preservation of cardiac function

- Treatment of underlying cause(s)

Management
- Depends on the degree of hyperkalaemia, associated physical features and ECG changes

- The measures are aimed at:

- Promoting potassium loss

- Limiting exogenous potassium intake

- Discontinuation of anti-kaliuretic drugs

- Shifting potassium into cells

- Drug treatment

- Calcium gluconate

- 10 ml of 10% solution intravenously over 2-3 minutes

- Insulin plus glucose infusion

- 1-20 units of regular insulin plus 25-50 g of glucose given as 10 units in 100 ml of 50% glucose

- Other alternatives to cause influx of potassium:

- Sodium bicarbonate (134 mmoles/L) if there is metabolic acidosis

- See Cardiac Arrest

- When contaminated water is inhaled

- Parenteral/nebulised salbutamol (see Bronchial asthma)

- Appropriate management of pulmonary oedema

- Cerebral oedema

- Further defibrillation

- Intravenous line

- Repeat defibrillation

- Monitor arterial blood gases

- Full Blood Count; ESR

- Calculation of water deficit

- Deficit = (Plasma Na+ - 140) X (0.5 (males) or 0.4 (females) X body weight in kg

- Water replacement in glomerulo nephropathy

- Mineralocorticoid excess (primary deficit should be corrected slowly over 48-72 hours to prevent cerebral oedema

Chapter 18: Emergencies

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- The most rapid and effective way of lowering plasma potassium concentration

- Reserved for patients in renal failure and those with severe hyperkalaemia unresponsive to more conservative measures

- Sodium polystyrene sulphonate (a cation exchange resin)

- Removal of potassium with diuretics (loop plus thiazide diuretics in combination)

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- Sodium polystyrene sulphonate (a cation exchange resin)

- Removal of potassium with diuretics (loop plus thiazide diuretics in combination)

- Sodium polystyrene sulphonate (a cation exchange resin)

- Removal of potassium with diuretics (loop plus thiazide diuretics in combination)
Chapter 18: Emergencies

Hyponatraemia

Introduction
Plasma potassium less than 3.5 mmol/Litre
Mostly associated with increase in potassium loss
Increased renal loss:
- Disturbs and salt-waste and secondary hyperaldosteronism
- Calcification and distal delivery of non-reabsorbable anions
- Amphotericin B
- Cushing’s syndrome, Bartter’s syndrome
- Increased non-renalin loss:
  - GIT loss (diarrhoea, intemgutary sweat)
- Redistribution into cells:
  - Metabolic alkalosis
  - Drugs
  - Adrenergic agonists
  - Adrenergic antagonist
- Decreased intake:
  - Starvation
Clinical features
- Vary between patients and depend on the level of potassium loss
  - Serum K <3mmol/Litre:
    - Fatigue
    - Myalgia
    - Weakness of the lower extremities
    - More severe hyponatraemia results in:
      - Progressive weakness
      - Hypoventilation
      - Complete paralysis
- ECG changes are due to ventricular depolarisation and do not correlate with the plasma potassium levels
  - Flattening/inversion of the T wave
  - A prominent U wave
  - ST segment depression
  - Prolonged QT interval
  - Severe depletion results in prolonged PR interval
  - Decreased voltage and widening of the QRS complex
Investigations
- Electrocardiography
- Electrolytes, Urea and Creatinine
- Acid-base status
- Identifying the underlying disease

Treatment objectives
- Correction of potassium deficit
- Minimize/stop on-going loss
- Drug treatment (oral route preferred)
  - Potassium chloride
  - Doses depend on deficits, on-going losses and renal status
  - Intravenous potassium (given in an infusion)
  - Do not exceed 20 mmol/L

Calculation of potassium requirement
- Deficit body weight (kg) 0.3
- Add daily requirement of potassium and correct over 3 days
Caution
- Oral potassium supplements should be taken in an erect position or sitting upright and with plenty of water to avoid oesophageal erosions

Hypokalaemia

Introduction
Different types with varied aetiologies
- Pseudo-hyponatraemia:
  - Hyperglycaemia, infusion of mannitol

HYPERTENSIVE EMERGENCIES

Introduction
Severely elevated blood pressure (>200/120 mmHg) with evidence of target organ damage such as:
- Neurologic: altered consciousness
- Cardiovascular: myocardial ischaemia, left ventricular failure
- Renal failure
- Drugs:
  - Sodium niprusside
  - Amount of sodium = (desired concentration -- actual concentration) X body weight X 0.6

Aetiology
- Improperly managed hypertension
- Renal vascular disease
- Pheochromocytoma
- Accelerated essential hypertension

Clinical features
- Cerebral oedema
- May be asymptomatic
- Otherwise nausea, malaise, headache, lethargy, confusion, and altered consciousness
- Coma when plasma sodium is less than 120 millimoles per litre

Diffenrential diagnoses
- Congestive cardiac failure
- Hepatic cirrhosis
- Nephritic syndrome

Investigations
- Directed at establishing the cause and severity of hyponatraemia

Objective
- To correct plasma sodium concentration by restricting water intake and promoting water loss
- To correct the underlying disorder

Management
- Mild asymptomatic hyponatraemia requires no treatment
- Treatment objectives
  - Prompt but gradual reduction in mean arterial pressure by not more than 25% within the first 2 hours
  - Further reduction of BP to (not less than) 160/100 mmHg within 2 to 6 hours
  - Lower pressures may be indicated for patients with aortic dissection
  - Initiate/re-initiate long term therapy to normotensive levels

Drug treatment
- Sodium nppirsudse
  - 0.3 micrograms/kg/min intravenously initially, 0.5 - 6 micrograms/kg/min maintenance (maximum of 6 micrograms/kg/min)

Notable adverse drug reactions, caution
- Stop infusion if response is unsatisfactory after 10 minutes at maximum dose
- May occur in a fasting state or may be post-prandial
- Associated with quinidine, salicylates and sulphonamide use
- After overnight fast
- Reduced meal(s)
- During exercise
- During intensive insulin therapy
- May follow weight loss
- May follow alcohol ingestion
- Reduced insulin clearance
- Secondary to non-ß cell tumours/insulinoma

Clinical features
- The two types are neuroglycopenic and neurogenic
- Neuropenic manifestations:
  - Palpitations
  - Tremors
  - Anxiety
  - Sweating
  - Hunger

Hypoglycemia

Introduction
Blood glucose level less than 2.5 mmol/L (45 mg/dL)
- May occur in a fasting state or may be post-prandial
- Most commonly iatrogenic
- Associated with quinidine, salicylates and sulphonamide use
- After overnight fast
- Reduced meal(s)
- During exercise
- Can be due to intensive insulin therapy
- May follow weight loss
- May follow alcohol ingestion
- Reduced insulin clearance
- Sepsis
- Secondary to non-ß cell tumours/insulinoma

Clinical features
- The two types are neuroglycopenic and neurogenic
- Neuropenic manifestations:
  - Palpitations
  - Sweating
  - Hunger

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Mild hypokalaemia with ECF volume contraction:
- Sodium repletion with isotonic saline infusion

Hypokalaemia associated oedematous states:
- Restriction of both sodium and water intake

For severe cases which are symptomatic (plasma sodium concentration <115 mmol/L):
- Hypertensive saline to raise sodium concentration by 1 - 2 mmol/L/hour for the first 3 hours, but not more than 12 mmol/L/during the first 24 hours
- Calculation of the total amount of sodium to administer
- Amount of sodium = (desired concentration -- actual concentration) X body weight X 0.6

Hypertensive encephalopathy
- Severe headaches, malaise, vomiting, dizziness, blurred vision, chest pain, palpitations, dyspnoea, oliguria
- Fundoscopic changes
- Evidence of left ventricular failure
- Changes in level of consciousness

Target organ damage
- Cerebrovascular accident
- Myocardial infarction
- Cardiac failure
- Renal failure

Investigations
- Plain chest radiograph
- Echocardiography
- Full Blood Count
- Urea Electrolytes and Creatinine
- Urinalysis
- Echocardiography

Treatment objectives
Chapter 18: Emergencies

Standard Treatment Guidelines for Nigeria 2008

Paresthesia
Neuroglycopenic manifestations:
Confusion
Fatigue
Seizures
Loss of consciousness
Death

Diagnosis
The Whipples's triad provides a framework for diagnosis of hypoglycaemia:
Symptoms of hypoglycaemia
Low plasma glucose concentration (<2.5 mmole/L)
Alleviation of hypoglycaemic symptoms after glucose administration

Differential diagnoses
Other causes of acute confusional state

Investigations
Random blood sugar on presentation
Other tests to confirm the cause of hypoglycaemia

Treatment objectives
Prompt restoration of normal blood glucose level
Elimination of the poison or decontamination
Enhancing systemic clearance

Supportive measures
Remove the patient from the toxic environment
Provide fresh air and oxygen (respiratory support)
Maintain therapy with oral thyroxine in a dose of 50 micrograms per day

Clinical evaluation
Obtain a good history of the drug ingestion/exposure
- Amount, time, etc
- Circumstances surrounding the event (from the patient, relatives and other eyewitnesses)

Clinical presentation
The ingestion by, or exposure of a patient to excessive doses of a medicine or other substances may cause harm
This may be:
Self poisoning (may be suicidal)
Accidental
Homicidal

POISONING
Introduction
The ingestion by, or exposure of a patient to excessive doses of a medicine or other substances may cause harm
This may be:
Self poisoning (may be suicidal)
Accidental
Homicidal

Clinical presentation
Determined (amongst others) by:
Type of drug
Inherent toxicity
Dose and duration following exposure
Concurrent therapy
Co-existing disease states etc

This guideline provides only a brief overview.

Practitioners are advised to seek advice from experts, standard texts in medicine and toxicology, in the absence of a Poison Information Centre

Principles of management of poisoning
Verify, validate or confirm all of the events related to the poisoning
Take good clinical history
- Information from relatives, friends, emergency services personnel may be very useful especially where the patient is unwilling or unable to provide useful information
Emergency stabilization
Quick clinical evaluation
Elimination of the poison or decontamination
Enhancing systemic clearance
Administration of antidotes
Supportive measures
Observation
Disposition
Emergency stabilization

Life-saving measures take priority over all other decontamination techniques
The following ABC approach is recommended:
A  Establish a clear Airway
B  Ensure adequate Breathing and ventilation
C  Ensure adequate Circulation
D  Address Drug-induced depression of the central nervous and respiratory systems
E  Correct any Electrolyte and metabolic abnormalities

Clinical evaluation
A quick clinical evaluation should be carried to:
- Obtain a good history of the drug ingestion/exposure
- Amount, time, etc
- Circumstances surrounding the event (from the patient, relations and other eyewitnesses)

The patient may have no symptoms when seen early in the course of the poisoning
A thorough physical examination may further provide clues on the drug class causing toxicity e.g pinpoint pupils with opioid overdose
- The absence of a significant sign does not negate the diagnosis
- Clinical laboratory patient data e.g. urine drug screens
- Useful in patients with coma of unknown aetiology

Elimination of poisons (or Decontamination)
The removal of the offending substance from the patient
The presumption is that both the dose and duration of exposure are determinants of toxicity, and limiting continued exposure is beneficial

Remove the patient from the toxic environment
Provide fresh air and oxygen (respiratory decontamination)

Flushing the areas (e.g. skin and eyes) with large volumes of fluid to remove the toxic substance
Gastrointestinal decontamination:
Emesis or lavage to evacuate the gastric contents
Clinical features

- Naloxone for opioids
- N-acetylcysteine for paracetamol

Antidotes

- Flumazenil, a competitive benzodiazepine receptor antagonist, can reverse CNS and respiratory depression
- Give 0.1 mg intravenously at 1 minute intervals until desired effect is achieved
- Notable adverse drug reactions
- Flumazenil with tricyclic antidepressants can cause seizures

Prevention of Drug Poisoning

- Keep all medicine out of reach when not needed
- Label all medicines appropriately
- Kerosene poisoning prevention
- Keep kerosene and other hydrocarbons away from children

Use dedicate on tenants kerosene and other hydrocarbon

Co-poison prevention

1. Keep working generator safely away from explosions
2. Do not run mobile engine/vehicles within explosions
3. Enact and enforce laws for safe engine/generator purchasing and use

Carbon monoxide poisoning

- Usually due to inhalation of smoke, car or generator exhaust fumes caused by incomplete combustion in a confined space
- Carbon monoxide binds to haemoglobin, myoglobin and to mitochondria, inhibiting cellular respiration
- Toxic effects of carbon monoxide are related to hypoxia

Clinical features

- Dyspnoea
- Tachypnoea
- Headache
- Emotional lability
- Confusion
- Impaired judgement
- Clumsiness
- Syncope
- Nausea, vomiting and diarrhoea may occur

Cardiovascular manifestations:
CHAPTER 19: THERAPEUTICS

PRESCRIPTION WRITING

Introduction
The writing of a prescription is the culmination of a clinical encounter with a patient. The decision to issue a prescription follows a complex process of professional analysis and must be based on the following considerations:

- Knowledge of the patient's clinical state
- Factors likely to influence the drug's pharmacokinetics and pharmacodynamics; the efficacy, safety and cost of the drug

Rational prescribing entails the following process with various steps:

Step 1: Define the patient's problem
Step 2: Specify the therapeutic objectives
Step 3: Verify whether your proposed treatment is suitable for this patient
Step 4: Start the treatment
Step 5: Give information, instructions and warnings
Step 6: Monitor (and/or stop) the treatment

Details of this process will be found in the WHO's "Guide to Good Prescribing." A prescription order should specify:

- What is to be administered
- To whom
- By whom prescribed
- How much should be taken (the amount e.g. in milligrams, grams)
- How often (frequency)
- The route of administration
- Duration of therapy

A prescription order is essential as a medico-legal document.

Identity of prescriber:
- Name
- Address/institution of prescriber
- Telephone number

Date of prescription:
- Near top/beginning of left margin of a chart order

Identity of patient:
- Name

Glucocorticoids are ineffective

Organophosphate/insecticide poisoning

Introduction
These substances irreversibly inhibit acetylcholinesterase and cause accumulation of acetylcholine at muscarinic and nicotinic synapses and in the CNS.

Clinical features
- Onset from exposure to toxicity is between 30 minutes - 2 hours
- Muscarinic effects: Nausea, vomiting, abdominal cramps, increased urinary frequency; urinary and fecal incontinence
- Increased bronchial secretions

Non-drug treatment
- Remove from carbon monoxide exposure; move to fresh air
- Oxygen administration
- Ventilatory support
- Remove contaminated clothing
- Wash skin with soap and water
- Ventilatory support
- Atropine

Adult: 0.5 - 2 mg intramuscularly every 5 - 15 minutes until bronchial and other secretions have dried
Child: 20 micrograms/kg (maximum 2 mg) intramuscularly or intravenously depending on the severity of poisoning, every 5 - 10 minutes until the skin becomes flushed and dry, pupils dilate and tachycardia develops

- Effective for muscarinic symptoms

Plus:
- Pralidoxime
- Diluted to 10 - 15 mL with water for injection and administered by slow intravenous injection over 5 - 10 minutes

Adult: 1 - 2 g; can be repeated in 30 minutes
Child: initially 30 mg/kg, then either 30 mg/kg every 4 hours or by intravenous infusion, 8 - 10 mg/kg/hour (usual maximum 12 g in 24 hours)

Treat seizures with intravenous diazepam 10 mg stat

Investigations

- Standard Treatment Guidelines for Nigeria 2008
- Full Blood Count and ESR
- Serum Urea, Electrolytes and Creatinine
- Liver function tests
- Acid-base status
- Blood gases

Non-drug treatment

- Remove contaminated clothing
- Wash skin with soap and water

Elimination is slow - by hepatic metabolism

- Definitions of this process will be found in the WHO's "Guide to Good Prescribing"
- Age (especially in children)
- Gender
- Address of patient
- Hospital number

Elements specifying medication:
- Name of medication (generic name)
- Strength (metric units) and quantity
- Dosage
- Frequency
- Duration
- Directions for use (drug- and patient-specific)
- Refill instructions
- Waiver of requirements for child-proof containers
- Additional labelling instructions

Prescriber's signature and other identification data e.g. code. Prescriptions may be hand written or computerized:
- Hand written prescriptions should be written in indelible ink and the hand writing should be legible (important, to avoid medication errors)
- Any alteration(s) made in a computer-issued prescription should be duly endorsed

Abbreviations
Only standard, official abbreviations should be used.

The following are some notable abbreviations

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Meaning</th>
</tr>
</thead>
<tbody>
<tr>
<td>a.c.</td>
<td>ante cibum (before food)</td>
</tr>
<tr>
<td>b.d.</td>
<td>bis die (twice daily)</td>
</tr>
<tr>
<td>o.d.</td>
<td>omni die (every day)</td>
</tr>
<tr>
<td>o.m.</td>
<td>omni mane (every morning)</td>
</tr>
<tr>
<td>p.c.</td>
<td>post cibum (after food)</td>
</tr>
<tr>
<td>q.d.</td>
<td>quarter die sumendum (to be taken four times daily)</td>
</tr>
<tr>
<td>q.d.h.</td>
<td>quarter quaque hora (every four hours)</td>
</tr>
<tr>
<td>stat</td>
<td>immediately</td>
</tr>
<tr>
<td>t.d.s.</td>
<td>ter die sumendum (to be taken three times daily)</td>
</tr>
<tr>
<td>t.i.d.</td>
<td>ter in die (three times daily)</td>
</tr>
</tbody>
</table>

NOTE
Avoid abbreviations of drug names

Doses should be written in the metric system or in international units (IU) when metric doses are not practicable

If a drug is to be administered as 'required', specify the minimum dose interval and the total amount of drug to be administered

Avoid unnecessary use of decimal points

<table>
<thead>
<tr>
<th>Value</th>
<th>Abbreviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 mg</td>
<td>1.0 mg</td>
</tr>
<tr>
<td>&gt;1 g</td>
<td>g</td>
</tr>
<tr>
<td>&lt;1 g</td>
<td>0.5 g</td>
</tr>
</tbody>
</table>

- Millilitre (mL) should be used for volume and not cubic centimetre, c.c or cm³

Prescription for special cases
Special precaution should be taken in children (especially neonates and infants), and the elderly when considering drug therapy
- There are differences in drug handling (pharmacokinetics) and sensitivity in drug response (pharmacodynamics) in the different age groups

Particular care should also be taken when prescribing for pregnant women

Precaution should also be taken in clinical states associated with organ system failure (renal, hepatic) where dosage adjustment may be required

Children (including neonates and infants)
There are notable differences in the proportions and constituents of body fluids between adults and children

The immature enzyme systems result in poor oxidation and conjugation and may cause adverse effects
- Grey Baby syndrome with chloramphenicol is an example

Drugs predominantly excreted by the kidneys e.g. aminoglycosides, penicillins may require dose reduction
- Use appropriate formulations for various routes e.g. rectal route (for diazepam, theophylline) in the uncooperative child

(See appendix IV for calculation of dose requirements for children)

The Elderly
Persons 65 years or over: a growing segment of the Nigerian population

A number of factors interplay to increase the incidence of adverse drug reactions in this group of patients
- Bodily changes affecting drug handling and tissue response

- The increasing number of medicines prescribed to treat multiple diseases, each with a potential to cause an adverse drug reaction as well as a drug-drug interaction
- Poor adherence to therapy due to factors inherent in the elderly

Dosage reduction may be required for some drugs because of
- Changes in volume of distribution
- Reduced metabolism
- Reduced renal elimination

Particular care is necessary in administration of drugs where sensitivity in the elderly is increased e.g.
- Hypno-sedatives
- Neuroleptics
- Diuretics

Where no drug is needed avoid unnecessary prescriptions.

Relevant drugs should be prescribed in the appropriate dose and monitored closely

Consideration should be given to the formulation that is most appropriate in the clinical circumstances

The possibility of drug-drug interactions should always be borne in mind

Pregnancy and Lactation
Changes in fluid and tissue composition occur during pregnancy

Reduced gastrointestinal motility delays gastric emptying and may delay drug absorption after oral administration

Vasodilation may result in enhanced absorption following drug administration by the intramuscular route

There is increased volume of distribution, increased hepatic metabolism and increased elimination of drugs

Extreme care must be taken when administer drugs with teratogenic potential to women in the reproductive age group (See appendix IV)

Some drugs may cause harm to infants when administered to nursing mothers (see appendix V)

Other drugs e.g. bromocriptine inhibit lactation

Drugs excreted significantly in milk and likely to cause toxicity are shown in appendix V

ADVERSE DRUG REACTIONS
Introduction
The use of medicines is inextricably linked to unintended responses

The safe use of medicines is therefore an important consideration in therapy

In this text the following WHO definitions will apply

Adverse drug reaction
A response to a medicine which is noxious and unintended

- Occurs at doses normally used in man for the prophylaxis, diagnosis or therapy of disease, or for the modification of physiologic function

Adverse drug event
Any untoward medical occurrence that may present during treatment with a pharmaceutical product, but which does not necessarily have a causal relationship with the treatment

A serious adverse event (experience, or reaction)
Any untoward medical occurrence that at any dose
- Results in death
- Is life-threatening
- Requires patient hospitalization or prolongs existing hospitalization
- Results in persistent or significant disability/incapacity
- Causes a congenital anomaly or birth defect
- Requires an intervention to prevent permanent impairment or damage

Side effect
Any unintended effect of a pharmaceutical product occurring at doses normally used in humans

- Is related to the pharmacological properties of the drug
- There is need to have a high index of suspicion during therapy so as to recognize and adequately manage adverse effects

Report any suspected adverse response to a drug to the hospitals’ Adverse Reaction Registry or directly to the National Agency for Food and Drug Administration and Control (NAFDAC), Abuja

A sample of the Yellow Form is shown in Appendix VI

Analysis of such reports enables appropriate decisions to ensure safe and judicious use of medicines

In the text a number of known adverse reactions are listed for medicines used for the treatment of the stated diseases
- This list is by no means complete or comprehensive
- There may be unknown adverse reactions peculiar to our population

Particular care should also be taken when prescribing for pregnant women

(Pregnancy and Lactation)

Standard Treatment Guidelines for Nigeria 2008

Chapter 19: Therapeutics

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- Persons 65 years or over: a growing segment of the Nigerian population
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- This list is by no means complete or comprehensive
- There may be unknown adverse reactions peculiar to our population
### List of Notifiable Diseases

1. AIDS
2. Anthrax (human)
3. Brucellosis (human)
4. Cerebro-spinal meningitis
5. Chicken pox
6. Cholera
7. Diarrhoea (simple without blood)
8. Diarrhoea with blood (dysentery)
9. Diphtheria
10. Dracunculiasis
11. Filariasis
12. Food poisoning
13. Gonorrhoea
14. Hepatitis
15. Lassa Fever
16. Leprosy
17. Louse-borne typhus fever
18. Malaria
19. Measles
20. Onchocerciasis (River blindness)
21. Ophthalmia neonatorum
22. Pertussis (Whooping cough)
23. Plague
24. Pneumonia
25. Poliomyelitis
26. Rabies (human)
27. Schistosomiasis
28. Smallpox
29. Syphilis
30. Other sexually transmitted diseases (STD)
31. Tetanus (other)
32. Tetanus (neonatal)
33. Trachoma
34. Trypanosomiasis (sleeping sickness)
35. Tuberculosis
36. Typhoid and paratyphoid fevers
37. Viral influenza
38. Yaws
39. Yellow fever

### List of emergency and immediate notifiable disease

1. AIDS (Acquired Immune Deficiency syndrome)
2. Acute Flaccid Paralysis
3. Anthrax
4. Cerebro-spinal Meningitis (CSM)
5. Cholera
6. Lassa fever
7. Plague
8. Rabies (human)
9. Smallpox
10. Typhoid and paratyphoid fevers
11. Yellow fever
APPENDIX 1

WHO CLINICAL STAGING OF HIV FOR INFANTS AND CHILDREN WITH ESTABLISHED HIV INFECTION

Clinical Stage 1
Asymptomatic
Persistent generalized lymphadenopathy

Clinical Stage 2 (I)
Unexplained persistent hepatosplenomegaly
Papular pruritic eruptions
Fungal nail infections
Angular cheilitis
Linear gingival erythema
Extensive molluscum contagiosum
Recurrence of oral ulceration
Unexplained persistent parotid enlargement
Herpes zoster
Recurrent or chronic upper respiratory tract infections (otitis media, otitis media, sinusitis, tonsillitis)

Clinical Stage 3 (I)
Unexplained moderate malnutrition or wasting not adequately responding to standard therapy
Unexplained persistent diarrhoea (14 days or more)
Unexplained fever (above 37.6 °C, intermittent or constant, for longer than one month)
Oral hairy leukoplakia
Acute necrotizing ulcerative gingivitis or periodontitis
Lymph node tuberculosis
Pulmonary tuberculosis
Severe recurrent bacterial pneumonia
Symptomatic lymphoid interstitial pneumonitis
Chronic HIV-associated lung disease including bronchiectasis
Unexplained anaemia (<8.0 g/dl), neutropenia (<0.5 x 10^9/L) and or chronic thrombocytopenia

Clinical stage 4 (i, ii)
Unexplained severe wasting, stunting or severe malnutrition not responding to standard therapy
Pneumocystis pneumonia
Recurrent severe bacterial infections (e.g., empyema, pyomyositis, bone or joint infection, meningitis, but excluding pneumonia)
Chronic herpes simplex infection; (orolabial or cutaneous of more than one month’s duration, or visceral at any site
Extrapulmonary tuberculosis
Kaposi sarcoma
Oesophageal candidiasis (or Candida of trachea, bronchi or lungs)
Cytomegalovirus infection; retinitis or cytomegalovirus infection affecting another organ, with onset at age over 1 month
Central nervous system toxoplasmosis (after the neonatal period)
Extrapulmonary cryptococcosis (including meningitis)
HIV encephalopathy
Disseminated endemic mycosis (extrapulmonary histoplasmosis, coccidiodymycosis)
Chronic cryptococcosis (with diarrhoea)
Chronic isosporiasis
Disseminated non-tuberculous mycobacteria infection
Cerebral or B cell non-Hodgkin lymphoma
Progressive multifocal leukoencephalopathy
HIV-associated cardiomyopathy or nephropathy

(1) Unexplained refers to where the condition is not explained by other causes

(2) Some additional specific conditions can be included in regional classifications (e.g., Disseminated Pneumocystis in Asia, HIV-associated rectovaginal fistula in Africa), and reactivation of American trypanosomiasis

APPENDIX II:

WHO NEW ANTENATAL CARE MODEL CLASSIFYING FORM 2001

Criteria for classifying women for the basic component of the new antenatal care model

Name of patient: ____________________________
Address: ____________________________
Telephone: ____________________________

Name of patient: ____________________________
Address: ____________________________
Telephone: ____________________________

INSTRUCTIONS: Answer all of the following questions by placing a cross mark in the corresponding box.

OBSTETRIC HISTORY

1. Previous stillbirth or neonatal loss?  
   No  Yes
2. History of 3 or more consecutive spontaneous abortions?  
   No  Yes
3. Birthweight of last baby < 2500g?  
   No  Yes
4. Birthweight of last baby > 4500g?  
   No  Yes
5. Last pregnancy: hospital admission for hypertensive or pre-eclampsia/eclampsia?  
   No  Yes
6. Previous surgery on reproductive tract?  
   (Myomectomy, removal of septum, cone biopsy, classical CS, cervical cerclage)  
   No  Yes

CURRENT PREGNANCY

7. Diagnosed or suspected multiple pregnancy?  
   No  Yes
8. Age less than 16 years?  
   No  Yes
9. Age more than 49 years?  
   No  Yes
10. Immunization Rh (-) in current or in previous pregnancy?  
   No  Yes
11. Vaginal bleeding?  
   No  Yes
12. Pelvic mass?  
   No  Yes
13. Diastolic blood pressure 90mm Hg or more at booking?  
   No  Yes

GENERAL MEDICAL

14. Insulin-dependent diabetes mellitus?  
   No  Yes
15. Renal disease?  
   No  Yes
16. Cardiovascular disease?  
   No  Yes
17. Known ‘substance’ abuse (including heavy alcohol drinking)?  
   No  Yes
18. Any other severe medical disease or condition?  
   Please specify: ____________________________

A “Yes” to any ONE of the above questions (i.e. ONE shaded box marked with a cross) means that the woman is not eligible for the basic component of the new antenatal care model.

Is the woman eligible? (circle)  NO  YES

If NO, she is referred to ____________________________

Date ____________________________
Name ____________________________
Signature ____________________________

(staff responsible for ANC)
APPENDIX III

CALCULATION OF DOSAGE REQUIREMENTS IN CHILDREN

Introduction
Medicine doses are generally based on body weight (in kilogram) or the following age ranges:
First one month (neonate)
Up to 1 year (infant)
1 - 5 years
6 - 12 years

Unless the age is specified, the term child includes persons aged 12 years and below.

Dose Calculation
Calculated based on body weight (in kilogram) or the body surface area (in m²). Use this rather than attempting to calculate doses on the basis of doses used in adults.

Body Surface Area (BSA) estimates are more accurate for calculation of paediatric doses—many physiological phenomena correlate better to BSA.

For most medicines the adult maximum dose should not be exceeded.

For example, if the dose is stated as 4 mg/kg (max. 180 mg), a child weighing 10 kg should receive 40 mg but a child weighing 50 kg should receive 180 mg and not 200 mg.

Young children may require higher doses per kilogram than adults because of their higher metabolic rate;
Calculation by body weight in an overweight child may result in much higher doses being administered than necessary. Such doses should be calculated based on ideal body weight in relation to height and age.

See table below.

<table>
<thead>
<tr>
<th>Age</th>
<th>Ideal body-weight Kg</th>
<th>Height cm</th>
<th>Body Surface /m²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Newborn*</td>
<td>3.5</td>
<td>50</td>
<td>0.23</td>
</tr>
<tr>
<td>1 Month*</td>
<td>4.2</td>
<td>55</td>
<td>0.26</td>
</tr>
<tr>
<td>3 Month*</td>
<td>5.6</td>
<td>59</td>
<td>0.32</td>
</tr>
<tr>
<td>6 Month</td>
<td>7.7</td>
<td>67</td>
<td>0.40</td>
</tr>
<tr>
<td>1 year</td>
<td>10</td>
<td>76</td>
<td>0.47</td>
</tr>
<tr>
<td>3 years</td>
<td>15</td>
<td>94</td>
<td>0.62</td>
</tr>
<tr>
<td>5 years</td>
<td>18</td>
<td>108</td>
<td>0.73</td>
</tr>
<tr>
<td>7 years</td>
<td>23</td>
<td>120</td>
<td>0.88</td>
</tr>
<tr>
<td>12 years</td>
<td>39</td>
<td>148</td>
<td>1.25</td>
</tr>
</tbody>
</table>

* The figures relate to full term and not preterm infants who may need reduced dosage according to their clinical condition.
## APPENDIX IV:

### MEDICINES WITH TERATOGENIC POTENTIAL

<table>
<thead>
<tr>
<th>Medicine</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antiepileptics</td>
<td>Risk of teratogenicity greater if more than one medicine used</td>
</tr>
<tr>
<td>Bleomycin</td>
<td>Avoid (teratogenic and carcinogenic in animal studies)</td>
</tr>
<tr>
<td>Carboplatin</td>
<td>Avoid (teratogenic and embryotoxic in animal studies)</td>
</tr>
<tr>
<td>Ciplitin</td>
<td>Avoid (teratogenic and embryotoxic in animal studies)</td>
</tr>
<tr>
<td>Co-trimoxazole</td>
<td>Teratogenic risk (trimethoprim - a folate antagonist)</td>
</tr>
<tr>
<td>Cytarabine</td>
<td>Avoid (teratogenic in animal studies)</td>
</tr>
<tr>
<td>Daunorubicin</td>
<td>Avoid (teratogenic and carcinogenic in animal studies)</td>
</tr>
<tr>
<td>Doxorubicin</td>
<td>Avoid (teratogenic and toxic in animal studies)</td>
</tr>
<tr>
<td>Sulfadoxine/pyrimethamine</td>
<td>Possible teratogenic risk (pyrimethamine is a folate antagonist)</td>
</tr>
<tr>
<td>Hydroxocarbamide(hydroxyurea)</td>
<td>Avoid (fetotoxicity and teratogenicity in animal studies)</td>
</tr>
<tr>
<td>Idouridine</td>
<td>Teratogenic in animal studies</td>
</tr>
<tr>
<td>Isoretinoin</td>
<td>Teratogenic</td>
</tr>
<tr>
<td>Lithium salts</td>
<td>Avoid if possible (risk of teratogenicity)</td>
</tr>
<tr>
<td>Phenytoin</td>
<td>Congenital malformation (screening advised)</td>
</tr>
<tr>
<td>Trimenol</td>
<td>Teratogenic</td>
</tr>
<tr>
<td>Trimethoprim</td>
<td>Teratogenic risk (folate antagonist)</td>
</tr>
<tr>
<td>Vinblastine</td>
<td>Avoid (limited experience suggest fetal harm; teratogenic in animal studies)</td>
</tr>
<tr>
<td>Vincristine</td>
<td>Avoid (teratogenicity and fetal loss in animal studies)</td>
</tr>
</tbody>
</table>

## APPENDIX V:

### MEDICINES THAT COULD CAUSE HARM WHEN ADMINISTERED TO BREASTFEEDING MOTHERS

<table>
<thead>
<tr>
<th>Medicine</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abacavir</td>
<td>Breastfeeding not advised in HIV infection</td>
</tr>
<tr>
<td>Acetazolamide</td>
<td>Amount too small to be harmful</td>
</tr>
<tr>
<td>Alcohol</td>
<td>Large amounts may affect infant and reduce milk consumption</td>
</tr>
<tr>
<td>Amantadine</td>
<td>Avoid; present in milk; toxicity in infants reported</td>
</tr>
<tr>
<td>Amiloride</td>
<td>Manufacturer advises avoid</td>
</tr>
<tr>
<td>Aminophylline</td>
<td>Present in milk- irritability in infants reported</td>
</tr>
<tr>
<td>Amaprylline</td>
<td>Manufacturers advise avoid</td>
</tr>
<tr>
<td>Amlodipine</td>
<td>Manufacturers advise avoid</td>
</tr>
<tr>
<td>Amoxicillin</td>
<td>Trace amounts in milk</td>
</tr>
<tr>
<td>Amphetamines</td>
<td>Significant amount in milk</td>
</tr>
<tr>
<td>Ampronavir</td>
<td>Breast feeding not advised in HIV infection</td>
</tr>
<tr>
<td>Aspirin</td>
<td>Avoid; possible risk of Reye's syndrome; regular use of high doses could impair platelet function and produce hypoprophrominemia in infants if neonatal vitamin K stores low</td>
</tr>
<tr>
<td>Androgens</td>
<td>Avoid. May cause masculinization in the female infant or precocious development in the male infant; high doses suppress lactation</td>
</tr>
<tr>
<td>Anticoagulants, oral</td>
<td>Risk of haemorrhage; increased by Vitamin K deficiency; warfarin appears safe but phenindione should be avoided</td>
</tr>
<tr>
<td>Antihistamines</td>
<td>Significant amounts of some antihistamines present in milk, although not known to be harmful</td>
</tr>
</tbody>
</table>
# Form for Reporting of Suspected Adverse Drug Reactions

**National Pharmacovigilance Centre (NPC) Nigeria**

**Form for Reporting of Suspected Adverse Drug Reactions**

**IN STRICT CONFIDENCE**

**National Agency for Food and Drug Administration & Control (NAFDAC), Headquarters Office**

**Plot 2032 Olusegun Obasanjo Way**

**Wuse Zone 7 Abuja**

**Tel:** 08068999571 **or Fax:** 09-5241108

## 1. Patient's Details

- **Full Name or Initials:**
- **Patient Record No.:**
- **AGE/DATE OF BIRTH:**
- **SEX:**
  - M
  - F
- **WEIGHT (kg):**
- **HOSPITAL/Treatment Centre:**

## 2. Adverse Drug Reaction (ADR)

### A. Description

<table>
<thead>
<tr>
<th>C. OUTCOME OF REACTION</th>
<th>TICK AS APPROPRIATE</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Recovered fully</td>
</tr>
<tr>
<td></td>
<td>Recovered with disability (Specify)</td>
</tr>
<tr>
<td></td>
<td>Congenital Abnormality (Specify)</td>
</tr>
<tr>
<td></td>
<td>Life Threatening (Specify)</td>
</tr>
<tr>
<td></td>
<td>Death</td>
</tr>
<tr>
<td></td>
<td>Others (specify)</td>
</tr>
</tbody>
</table>

- **DATE Reaction Started**
- **DATE Reaction Stopped**

### B. Was Patient Admitted Due to ADR

- **Yes**
- **No**

## 3. Suspected Drug (Including Biologics Traditional/Herbal Medicines & Cosmetics)

### A. Drug Details

- **Brand Name:**
- **Generic Name:**
- **NAFDAC No.:**
- **Batch No.:**
- **Expiry Date:**

- **Name & Address of Manufacturer:**

### B. Indications for Use

- **Dosage**
- **Route of Administration**
- **Date Started**
- **Date Stopped**

## 4. Concomitant Medicines

- **(All medicines taken within the last 3 months including herbal and self medication)**

### Brand or Generic Name

- **Dosage**
- **Route**
- **Date Started**
- **Date Stopped**

### Reason for Use

## 5. Source of Report

- **Name of Reporter:**
- **Address:**
- **Profession:**
- **Signature:**
- **Tel No/E-mail:**

*MANDATORY FIELDS*