#### Principles of history taking and physical examination

Key prerequisites for successful case history taking are: good listening, interpersonal skills and analytical ability. Are you and your clinical setting ready to receive the patient? From the moment the patient enters your surgery you must eliminate any distractions, whether in your mind or in your environment. It is particularly important with a new patient to form a good rapport and this, to a large extent, is formulated during case history taking.

#### What Is Differential Diagnosis?

This is a systematic process of generating hypotheses by evaluating all the data from a case history, physical examination and diagnostic tests in order to arrive at a point where you can identify the most probable diagnosis. —We use the specific findings from the history and examination of the patient to differentiate the relative probabilities of the potential conditions to create a short list of the most likely conditions, the differential diagnosis. To achieve this you can devise a screen with a series of questions using a structured form. Pause this procedure and focus on areas of relevance. This structured approach covers the following sections:

#### o General medical history

- History of present illnesses
- Operations
- Accidents
- Hospitalizations

#### o Drug history

#### o Dental history

#### o Family history

#### o Personal and social history

Depending on the structure of your registration form, if not already covered under the full case history, you should also include:

o Allergies and sensitivities (internal medications or topical).

o Medicines currently being taken.

o Chronic illness (e.g., diabetes, asthma, heart disease, blood disorders, infections).

o Might they be pregnant?

After the invitation to talk freely, you should be in a position to identify the **presenting complaint**, use a series of questions intended to provide you with key information for\_your evolving hypothesis and subsequent differential diagnosis. Remember these prompts using the acronym **SOCRATES**:

**Site**  $\rightarrow$  Please point to the area of pain (or symptoms). Is it always there?

**Onset**  $\rightarrow$  How did it start? Was it acute with a short duration, or insidiously over a long period? When did you first experience this?

**Character** (of pain or symptoms)  $\rightarrow$  Can you describe the sensation? Is it always like this?

**Radiations**  $\rightarrow$  Does the pain spread to any other locations? Does is start from another location? What does this other pain feel like?

Associated (manifestations or symptoms)  $\rightarrow$  What else is happening to you at the same time or just before or after? E.g., Locking of the jaw, headache, neck pain, sinusitis, stress?

**Timing**  $\rightarrow$  How long do the symptoms last? When do you get them? Is there a pattern?

**Exacerbating and relieving factors**  $\rightarrow$  What brings them on? What makes them worse? What makes them go away? What makes them better? For example, heat, cold, pressure or all of them? Anti-inflammatories or opiates?

**Severity**  $\rightarrow$  How bad is this for you? If pain is not the issue, then clarify. Is this getting better or worse?

#### **GENERAL MEDICAL HISTORY**

**o History of present illnesses;** The chronological narrative is the patient's account of any conditions they may suffer from at present, and whether they are under a formal treatment regime or are self-managed.

**o Operations;** Briefly inquire about any major operation the patient may have had, and the reasoning behind them, any complications arising, and the outcomes.

**o** Accidents and injuries; Inquire if the patient has suffered any *major* accidents or injuries, such as road traffic accidents (RTAs), work injuries, sports injuries or falls.

**o Hospitalizations;** Consider here the implications of a patient who has had TB or rheumatic fever.

#### **DENTAL HISTORY**

Find out how well the patient cares for their teeth, any procedures they have had, which practitioner the patient normally sees, how frequently they have check-ups, and how long ago was their last one.

#### **DRUG HISTORY**

Some detailed patient forms may have a list of medications which need to be recorded due to their influence on your procedures, and alerting you to possible interactions and side-effects.

These are some of the medicines about which you need to inquire:

o Anticoagulants: warfarin, heparin, aspirin

- o Anticonvulsants
- o Corticosteroids
- o Immunosuppressant's

o Heart medications: anti-arrhythmic, digoxin

Has the patient had any adverse drug reactions or allergies, such as an allergy to penicillin or non-steroidal anti-inflammatory drugs (NSAIDs)?

Does the patient suffer from pathology for which certain drugs may be contraindicated?

#### FAMILY HISTORY

One way of approaching this topic is to inquire about the state of health (or cause of death) of the patient's parents and siblings. Alternatively, ask them if they know of any conditions that run in their family such as:

Diabetes, Heart disease, Cancer (esp. Malignant melanoma, breast cancer), Blood disorders, S,eizures

#### PERSONAL AND SOCIAL HISTORY

In many instances, your patient's personal habits may have a significant bearing on their dental health. Inquire about:

Smoking habit. Alcohol consumption, Recreational drugs, including the chewing of tobacco, betel nuts and others, Diet, Occupation and possible environmental hazards, Sleep, Exercise and recreational activities.

#### GENERAL PHYSICAL EXAMINATION

Clinical examination of the body systems should be carried out in a logical, progressive manner. The accepted protocol is that you start with simple techniques such as observation and then progress to more specialized procedures.

Listen to their complaint  $\gg$  look  $\gg$  feel  $\gg$  move  $\gg$  investigate

#### VITAL SIGNS

- Pulse rate and rhythm
- Respiratory rate
- Blood pressure
- Temperature measurement
- Oximetry

# Pulse Rate and Rhythm

The pulse is normally taken at the radial artery just above the thumb.



Other possible sites are the brachial artery in the anterior and medial aspect of the elbow.

Measure the beats for 30 seconds and then multiply by two.

#### **Pulse rates:**

o Infants: 80 - 140 bpm

o Children: 70 - 110 bpm

o Adults: 60 - 100 bpm

**Respiratory Rate:** The normal respiration rate for an adult at rest is 12 to 20 breaths per minute. The patient must not be aware that you are evaluating their breathing rate as this will influence the measurement.

There are various inconspicuous techniques:

o Pretend you are taking the pulse whilst observing the lower parts of their thorax or abdomen.

o Pretend you are taking the pulse whilst resting the hand over the diaphragm.

Age	Respiratory rate (breaths per minute)	
nfant	30–60	1 10
Foddler	24–40	N 8
Preschooler	22–34	
School aged child	18–30	
Adolescent	12–16	

#### **Blood Pressure:**

Blood pressure may be taken using:

o Electronic sphygmomanometer; will provide you with accurate measurements of blood pressure and pulse rates simultaneously.

o Mercury sphygmomanometer



#### Normal ranges of blood pressure

	Systolic	Diastolic
Infants	75-100 mmHg	50-70 mmHg
Children	80-110 mmHg	50-80 mmHg
Adults	90-140 mmHg	60-90 mmHg

#### **Temperature:**

Temperature can be taken using various devices. In the mouth, it can be taken sublingually using a mercury or coloured alcohol-based device, or with an electronic type. The normal body temperature is  $37 \,^{\circ}$ C or  $98.6 \,^{\circ}$ F.

#### **Blood Oximetry:**

The saturation of oxygen can easily be measured using a digital oximeter. Normal range between 95-99%, below 94% the patient is in a state of hypoxia.



GENERAL INSPECTION OF THE PATIENT Nail and Hand Features

Nails:

o <u>Clubbing</u> (also known as drumstick fingers and watch-glass nails): chronic and serious diseases of lung, heart, bowel, cancer.



o <u>Leukonychia</u> (white discolouration on the nails): hypoalbuminaemia (liver, kidney, bowel).



o <u>Koilonychia</u> (spoon



shaped nails): iron deficiency anaemia.

o <u>Splinter haemorrhages</u> (tiny blood clots that tend to run vertically under the nails): infectious endocarditis, vasculitis.



o Pitting and onycholysis (detachment of the nail from the nail bed): psoriasis.



o <u>Beau's lines</u> (deep grooved lines that run from the side of the finger nail): Various systemic illnesses, fever, chemotherapy.



#### Then check the hands for:

o <u>Palmar erythema</u>: red warm palms (liver palms). Causes: liver diseases, oestrogens, pregnancy.



o <u>Heberden's nodes</u>: bony deformities in distal interphalangeal joints. Causes: osteoarthritis.



o Symmetrical soft swelling in proximal interphalangeal joints and deformity of the wrists. Causes: <u>rheumatoid arthritis</u>.



#### Examination of the Face, Eyes and Mouth

Look out for evidence of endocrine pathologies.

o Staring protruding eyes, restlessness  $\rightarrow$  hyperthyroidism.

o Cold, pale, puffy looking skin, loss of eyebrows → hypothyroidism.

o Moon face, thinning of skin, acne  $\rightarrow$  Cushing's disease (excess corticosteroids)

o Increased pigmentation, especially buccal membranes, creases and old scars  $\rightarrow$  Addison's disease (adrenal insufficiency).

o Gaps between teeth, broad mandible, large tongue, tunnel vision  $\rightarrow$  Acromegaly (pituitary adenoma, excess growth hormone).

o Yellow sclera  $\rightarrow$  jaundice (liver disease).

o Pale conjunctivae→ anaemia.

o <u>Corneal arcus</u> (a greyish-white ring occurring in the periphery of the cornea)



or <u>xanthelasmas</u> (soft, yellow-orange plaques on the eyelids)  $\rightarrow$  hypercholesterolemia.



o <u>Blue conjunctivae</u> and mucus membranes  $\rightarrow$  central cyanosis.



o Lack of saliva, creases on tongue, slow skin recoil when pinched  $\rightarrow$  dehydration, diabetes, Sjögren's syndrome.

o <u>Angular stomatitis</u>, cheilitis  $\rightarrow$  badly-fitting dentures, oral candidosis.



o Red \_beefy' tongue, soreness → <u>Deficiencies</u> of iron, B12, Niacin, folic acid.



o White coating on tongue  $\rightarrow$  enlarged filliform papillae, <u>candida</u>.



o White patches or plaques in buccal mucous membranes  $\rightarrow$  leukoplakia, lichen

#### planus, hyperkeratosis.



o Ulcers in oral mucous membranes → recurrent aphthous ulcers, Crohn's disease.



o Gingival disease associated with infections i.e., viruses, fungus, bacteria and hyperplastic gingiva due to medications (calcium channel blockers) and leukaemia.

#### **Inspection of the Neck**

Observe the neck from the front and in profile. Look out for:

o Neck swelling. Localized: lymph nodes, cysts, tumours. Generalised/diffuse: goitre, tonsils.

- o The cervical spine: shape, deformities, restrictions.
- o Muscle bulk, distribution and symmetry.
- o Skin lesions: pigmentation, erythematous, suppuration, excoriation
- o Scars: past trauma or surgery.

#### **Inspection of the Thorax**

The thorax is best observed sitting or standing so that anterior, lateral and posterior aspects can be examined. Look out for:

o Deformities of the thorax: barrel chest, pigeon chest, funnel chest



o <u>Deformities</u> of the <u>thoracic spine</u>: kyphosis, scoliosis, evidence of ankylosing spondylitis



o Scars: past trauma or surgery.

o The breathing pattern.

#### **Inspection of the Abdomen**

Observation of the abdomen should be performed with the patient lying supine on an examination couch. Look out for:

#### **Overall Shape of the Abdomen and Pelvic Region**

- o Abdominal swelling, and whether uniform or localised.
- o Skin lesions, scars and discolourations.
- o Prominent abdominal veins.
- o Evidence of hernia.



o Abnormal pulsations.

#### **Inspection of Upper and Lower Limbs**

Observe the arms and legs paying particular attention at the distal areas. Look out for:

- o The colour and perfusion.
- o Their shape and proportion in relation to the trunk.
- o The muscle bulk and symmetry.
- o The distribution of the hair and condition of the nails.
- o Evidence of skin infections, lesions, and swelling such as oedema.
- o If indicated, also palpate the peripheral pulses.

The above procedures should take the form of a quick scan of the entire body in a systemic way looking out for any overt signs of pathology. If you observe any suspect signs, examine that area more thoroughly. At this stage of the general examination procedure, it may appear to overlap with the system-specific examination

# ANEMIA

**Definition/description:** Anaemia refers to a decrease in the oxygen-carrying capacity of the blood, caused by decreased production of red blood cells, increased destruction of red blood cells, increased demand for iron, or formation of abnormal red blood cells.



**Common symptoms and signs of anaemia**: Symptoms include fatigue, breathlessness, palpitations, headache, tinnitus, anorexia, and bowel disturbances. Signs include pallor, retinal haemorrhages, and tachycardia, heart murmurs and cardiomegaly leading to heart failure in severe cases. In chronic iron deficiency, anaemic nail changes lead to spoon-shaped concave nails (koilonychia).



#### Classification of anaemia and haemoglobinopathies:

- o Haemolytic anaemia (autoimmune and non-autoimmune)
- o Iron-deficiency anaemia (microcytic anaemia)
- o Aplastic anaemia (normocytic anaemia)
- o Pernicious anaemia (macrocytic anaemia)
- o Sickle cell anaemia
- o Thalassaemia

# Anaemia

# □ Haemolytic anaemia

**Definition/description:** Haemolytic anaemia refers to the excessive intravascular or extravascular (in the spleen) destruction of red blood cells. The normal survival rate of RBCs is about 120 days. In haemolytic anaemia, it is much shorter.

**Causes** include autoimmune causes, infections, splenomegaly, drugs, RBC membrane disorders (spherocytosis), enzymopathies (deficiency of glucose-6-phosphate dehydrogenase) and haemoglobinopathies (sickle cell disease and thalassemia). Drugs which trigger haemolysis in G-6-PD deficiency include acetylsalicylic acid, ascorbic acid, dapsone and vitamin K. Fava bean ingestion in the diet is also associated with this form of anaemia. Malaria is the most common cause of anaemia in the developing world.

**Symptoms and signs** (of G-6-PD deficiency associated haemolytic anaemia):Symptoms include jaundice, palpitations, dyspnoea and dizziness. Signs include splenomegaly, cyanosis and Reynaud's phenomenon.



**Investigations:** A full blood count shows spherocytosis (sphere-shaped rather than biconcave disk shaped as normal). A direct antiglobulin test (Coomb's test) demonstrates the antigen responsible for RBC destruction. A rise in bilirubin and Lactate dehydrogenase (LDH), and urinary haemosiderin are other features of G-6-PD associated haemolytic anaemia.

**Management:** Identify the cause and administer the appropriate treatment. Steroids and splenectomy are useful. Raynaud's phenomenon can be avoided by keeping warm.

# □ Iron deficiency anaemia (microcytic anaemia)

**Definition/Description:** Iron deficiency anaemia is due to chronic blood loss. This is the most common form of anaemia.

**Cause:** An increased requirement for, or decrease in the intake of, iron can cause an iron deficiency. Chronic blood loss is the most common cause of iron deficiency anaemia.

**Symptoms and signs:** In the early stages of iron deficiency the condition may be asymptomatic. Fatigue and loss of energies are common in severe cases. Patients may have an abnormal desire to eat clay, dirt, paint, ice, etc. This abnormal craving is called pica. Glossitis, cheilitis and abnormal nails showing concavity (spoon-shaped nails called koilonychia) are common in advanced stages.

**Investigations:** Laboratory investigations include a FBC for the estimation of haemoglobin, haematocrit, mean corpuscular volume, and mean corpuscular haemoglobin concentrations. The FBC will show low values for all these in iron deficiency anaemia. In addition, microcytosis is present and the RBC distribution width is increased. Other investigations include serum iron (low), iron-binding capacity (increased) and serum ferritin (low) estimations.

Hematocrit	↓ to ↓ ↓ ↓
MCV	↓ to ↓ ↓ ↓
МСНС	Ŷ
Serum iron	↓ to ↓ ↓ ↓
Serum TIBC	Normal to <b>†</b>
Serum ferritin	↓ to ↓ ↓
Stainable iron in marrow	Absent
Reticulocytes	Normal to ↓

**Management:** Management protocols include the identification and elimination of the cause of blood loss, and administration of oral or parenteral iron therapy. Iron salts such as ferrous sulphate, gluconate or fumarate are used. Therapy should continue for up to six months after the correction of haemoglobin levels in order to refill iron stores in the tissues. The progress of treatments should be assessed by serial haemoglobin measurements until RBC levels reach normal values.

# □ Aplastic anaemia (normocytic anaemia).

**Definition/description:** A decrease in haematopoietic bone marrow leading to pancytopenia (involving all blood cells) results in aplastic anaemia.



**Cause:** These include idiopathic (60%), hereditary, viral hepatitis, irradiation, insecticides and drugs (sulphonamides, NSAIDs, antithyroids, etc).

**Symptoms and signs:** Symptoms of anaemia (due to a deficiency of RBCs) include bleeding tendencies, purpura (blood to pool under the skin),



haematuria, epistaxis, ecchymosis (very large bruised area), and gingival bleeding (due to thrombocytopenia). Susceptibility to infections (due to leucopoenia) is common. Headache and dyspnoea occur in a majority of patients.

Investigations: A FBC and estimation of erythropoietin (raised).

**Management:** Identification and removal of the cause, control of infection, bone marrow transplantation, haemopoietic stem cell transplantation and use of steroids are all used based on the severity of the condition.

#### □ Pernicious anaemia (macrocytic anaemia)

**Definition/Description:** Pernicious anaemia is characterised by the failure of intrinsic factor secretion in the stomach (due to an autoimmune process), which is responsible for the absorption of vitamin B12 (cobalamin).

**Cause:** An autoimmune disorder causes pernicious anaemia, resulting in permanent atrophy of the gastric mucosa. Total gastrotomy can also cause this form of anaemia. A higher incidence of pernicious anaemia occurs in individuals with blood group A. **Symptoms and signs:** General symptoms and signs of anaemia include neurological

symptoms such as paraesthesia of the fingers and toes, and dementia. Glossitis, periodic diarrhoea, weight loss, and mild jaundice due to haemolysis are also common.

Investigations: These include FBC (macrocytic),



serum B12 levels (low), detection of autoantibodies (parietal cells), and a gastric biopsy showing atrophic gastritis.



**Management:** This includes treatment with hydroxycobalamin, iron, potassium supplements, immunosuppressive drugs and blood transfusions.

# Haemoglobinopathies

# □ Sickle cell anaemia

**Definition/Description:** Sickle cell anaemia is an inherited disorder. Red blood cells become sickle-shaped when blood experiences lower oxygen tension (as in an unpressurised aircraft, or during GA administration), decreased pH, or dehydration. These changes result in erythrocytosis, increased RBC adhesion and blood viscosity, and increased vascular occlusion.

Cause: Sickle cell anaemia is inherited by an autosomal recessive means.

Symptoms and signs: Symptoms and signs include general symptoms of anaemia and lethargy, growth retardation, delayed puberty, increased susceptibility to

infection, leg ulceration, and infarcts in the spleen, lungs, kidneys, bowel, bones, and fingers. Often these features are precipitated by dehydration, excessive cooling, or infection.

Investigations: These include a FBC, blood film showing sickle-shaped cells



and haemoglobin electrophoresis.

Laboratory examinations	results	Values in this disease	Values in health
WBC count	increased	10000 -30000	5000 - 10000
RBC count	decreased	1-4 million/mm <sup>3</sup>	4 -6 million/mm <sup>3</sup>
Hb count	decreased	6 -8g/100ml	Male=13.5-17.5g/dl Female=11.5- 15.5g/dl
Haematocrit reading	decreased	10 -30%	45%
Reticulocyte count	increased	10-40%	1-2%
Hb electrophoresis	positive	HbS & HbF	HbA
Urine analysis	Albumin casts	positive	negative
Serum bilirubin	increased	1-3 mg/100 ml	0.2-0.8mg/100ml
Platelet count	increased	40000-50000/mm <sup>3</sup>	150000- 400000/mm <sup>3</sup>
Bone marrow exanination	Increased red cells	40-70%	<b>8-30%</b> 21

**Management:** Precipitating factors such as dehydration, infections and excessive cooling need to be avoided. Treatment includes folic acid supplements, hydration, a warm climate, antibiotics, analgesics, and blood transfusions.

#### □ Thalassaemias

**Definition/Description:** Thalassaemias are inherited disorders in which synthesis of one of the globin chains of the haemoglobin is either reduced or absent, resulting in haemolysis and anaemia.

Normally, haemoglobin is composed of four protein chains, two  $\alpha$  and two  $\beta$  globin chains. In thalassemia, patients have defects in either the  $\alpha$  or  $\beta$  globin chain, causing the production of abnormal red blood cells.

**Cause:** The thalassaemias are classified according to which chain of the haemoglobin molecule is affected. In  $\alpha$  thalassaemia (also known as thalassaemia major), production of the  $\alpha$  globin chain is affected, while in  $\beta$  thalassaemia (also known as thalassaemia minor) production of the  $\beta$  globin chain is affected. Deletion of one of the  $\alpha$  loci is more common in people of African or Asian descent, making them more likely to develop  $\alpha$  thalassaemia.  $\beta$  thalassaemias are common in Africans, Greeks and Italians.

**Symptoms and signs:** These include severe anaemia, failure to thrive and early death. Those who survive show a mongoloid appearance of the head and face due to bone marrow hyperplasia. Leg ulcerations and hepatosplenomegaly may also occur in these patients.



**Investigations:** Haemoglobin electrophoresis shows increased HbA in minor, and raised HbF in major thalassemia. Radiographs of the skull and phalanges show increased bone marrow cavities and a \_hair-on-end \_appearance of the skull vault. **Management:** Blood transfusions and iron chelating agents are administered to avoid iron overloading. A splenectomy may be conducted if hypersplenism is present.

#### **Dental Management Considerations**

o When classic symptoms and signs of anaemia exist, the patient should be sent for haematological screening tests.

o If anaemia is caused by any underlying systemic diseases (gastric carcinoma or peptic ulcer, for example) they must be identified early. Patients should be referred to a physician or specialist for diagnosis and treatment.

o There is no increased risk of dental complications requiring treatment precautions in patients with anaemia. In severe anaemia, the tendency for syncope should be considered.

o In aplastic anaemia, bleeding tendencies occur. A platelet count should be ordered prior to the commencement of any surgical procedure.

o In patients with aplastic anaemia, significant neutropenia may be present. In these patients the risk of infection is high. After surgical procedures, antibiotic cover is recommended for these patients.

o For any inhalational GA procedure, oxygenation is important for patients with aplastic anaemia and sickle cell anaemia.

o Patients with glucose-6-phosphate dehydrogenase deficiency anaemia, dental infections and drugs containing phenacetin may accentuate the rate of haemolysis.

These patients may also show drug sensitivity to sulphonamides, aspirin, and chloramphenicol.

o In sickle cell anaemia, long and complicated dental procedures should be avoided.

o In children with sickle cell anaemia, dental and periodontal tissues should be periodically checked for infections. Any foci of infection should be eliminated in order to avoid precipitation of a crisis attack.

o In sickle cell anaemia, local anaesthetic can be used for routine dental procedures, but epinephrine should be avoided in the anaesthetic agent.

o If necessary, sedation with diazepam can be used in patients with sickle cell anaemia.

o Prophylactic antibiotics for surgical procedures are recommended for patients with sickle cell anaemia.

o Liberal use of salicylates, acetaminophen (Tylenol) and codeine for pain control should be avoided in patients with sickle cell anaemia.

o Nitrous oxide-oxygen can be used with care: 50% oxygen with a high flow rate is recommended for use with thalassaemia patients.

# **Bleeding disorders**

# • Thrombocytopenia

**Definition/Description:** A bleeding disorder due to a circulating platelet (thrombocytes) count of below 50,000 per microlitre (a normal count is 150000 to 450000 per microlitre). Idiopathic thrombocytopenic purpura is a severe form of thrombocytopenia, probably due to an IgG antibody attack which follows a viral infection.



**Cause:** These include various diseases and conditions leading to decreased marrow production, decreased platelet survival, increased platelet consumption, platelet sequestration and platelet dilution. In the presence of autoimmune disorders, HIV disease, bone marrow tumours, leukaemia and lymphomas, thrombocytopenia can occur.

**Symptoms and signs:** Skin and mucous membranes show purpuric spots and ecchymoses, epistaxis, gastrointestinal bleeding, haematuria, headache and dizziness.



# **Oral Manifestations and Dental Management Considerations**

o In thrombocytopenia, oral mucous membranes my show purpuric spots. Often this is accompanied by purpuric spots on the skin.



o Spontaneous gingival bleeding is also a feature of a severe form of thrombocytopenia (as in idiopathic thrombocytopenia of autoimmune cause).



#### Dental management considerations

o Surgical procedures should not be undertaken unless the hematologist is involved with the diagnosis. For extractions, the platelet count should be at least 50,000/mm3 before the commencement of surgery. Transfusion of platelets may be required in some cases.

o In children with idiopathic thrombocytopenia, prednisone (4 mg/kg/day for 1 week orally) is recommended. This will increase the platelet count within 48 hours.

o If the patient is on long term steroid therapy, the dose of steroids needs to be doubled prior to the commencement of an invasive procedure.

**Investigations:** These include a FBC, WBC, detection of antibodies to platelets, bone marrow studies, bleeding time, platelet count, platelet adhesion and aggregation studies, and coagulation screen (to rule out factor deficiency syndromes). A simple test is the estimation of bleeding time, which can be performed in the clinic.

**Management:** Steroids, splenectomy and immunosuppressive drugs are used in the treatment of thrombocytopenia.

# • The Hemophiliac disorders (hemophilia A, B and Von Will brand's disease)

The —hemophilia's are a group of genetic disorders resulting in deficiency of one of the coagulation pathway factors. This group consists of three conditions: hemophiliaA, hemophilia B (Christmas disease) and Von Will brand's disease.

# Hemophilia A

**Definition/Description:** An inherited X–linked recessive disorder characterised by a deficiency of Factor VIII. This coagulation disorder affects males and is carried by females.

**Cause:** Genetic. The defective gene is located on the X chromosome (F8 gene). An affected male will not transmit the disorder to his sons, but all of his daughters will be carriers of the trait because they inherit his X chromosome. A female carrier will transmit the condition to half of her sons and the carrier state to half of her daughters. **Symptoms and signs:** Excessive bruising and haemarthroses are evident from very early in childhood. Swelling, pain and eventual deformity of the joints are common. Internal bleeding may occur. Spontaneous bleeding from oral soft tissues may occur in the severe form of the disorder. Excessive bleeding from trauma or surgery is common in these patients.



**Investigations:** Clinical history is suggestive. Screening tests include a prolonged partial thromboplastin test, (prolonged), bleeding time (normal), platelet count (normal) and specific tests for missing factors (Factor VIII assay).

**Management** includes the repeated administration of freeze dried or recombinant factor VIII concentrate (which is administered intravenously), oral antifibrinolytics (tranexamic acid or desmopressin, for example), and plasmapheresis (A procedure whereby blood plasma components are removed, treated, and returned to circulation).

# HemophiliaB (Christmas disease)

Factor IX is defective or deficient in hemophilia B. While less common than hemophilia A, clinical features are identical. Detection of defective or deficient Factor IX is diagnostic. Administration of purified factor IX products is required by these patients, administered intravenously.

# Von Will brand's disease

This is an autosomal dominant inherited disorder characterized by defective platelet function and a deficiency or abnormality of factor VIII.



Symptoms and signs include mucocutaneous bleeding and haemarthrosis.



**Investigations:** Family history, bleeding time (normal), platelet count (normal), platelet function tests (poor aggregation adhesion), and a factor VIII deficiency test are required.

# C)Laboratory tests:

 Bleeding time:assesse the function of platelets and their interaction with the vascular wall(bleeding stops within 4-8 min)
Platelet count: >50000/mm<sup>3</sup> rarely have significant clinical bleeding.
Activated partial thromboplastin time(aPTT):normal level 30-40 sec,it measures the intrinsic clotting system.
Prothrombin time(PT): Normal range 10-13sec,it measures the extrinsic clotting system.

Management: Administration of factor VIII concentrate.

# **Oral Manifestations and Dental Management Considerations**

o The level of deficiency of factor VIII is to be determined with the treating physician before invasive dental therapy.

o If multiple extractions are required, splints should be designed prior to surgical procedures taking place.

o Oral infections should be treated before surgical intervention.

o Prophylactic antibiotics prior to surgical procedures may be necessary for some patients.

o Postoperative antibiotic cover is indicated.

o Operative trauma to the tissues should be minimal.

o The majority of hemophiliacs patients need to be hospitalized.

o Missing factor needs to be administered intravenously prior to the surgical procedure.

o There are no contraindications to the use of local anesthetic.

o Aspirin or NSAIDs should be avoided.

# Dermatology

Skin is the most accessible organ of the body. Diseases of the skin are common. A sizable number of skin diseases manifest in oral mucosal lesions. Dental practitioners need to have an adequate knowledge of the range of skin diseases, some of which may

present oral manifestations.

# **Skin Diseases of Dental Interest**

#### □ Acne vulgaris

Acne vulgaris, commonly known as acne, refers to the formation of comedones, papules, pustules, cysts and nodules as a result of obstruction and inflammation of the hair follicles and the corresponding sebaceous glands (pilosabaceous units).



Acne is common in adolescents. Clinical appearance is diagnostic. These often remit in the 20s but may continue into the 40s, particularly in women. Treatment includes topical and systemic agents which reduce the secretion of sebum and inflammation. Topical antibacterial agents (Benzoil peroxide gel), topical comedolytic and exfoliant agents (tretinoin cream), oral antibiotics (doxycycline, tetracycline) and oral retinoids (isotretinoin) are effective.

□ **Perioral dermatitis:** Perioral dermatitis is an erythematous papulopustular facial (perioral) eruption which resembles acne. The exact cause is not known. Topical applications of steroids, and exposure to fluorides in water or tooth paste are sometimes associated with this disorder. Though it is not due to infections, treatment with systemic tetracyclines seems to be effective.



□ Atopic dermatitis (AD): Atopic dermatitis refers to an immune-mediated inflammatory condition of the skin characterized by pruritus. Skin lesions range from

inflammatory condition of the skin characterized by pruritus. Skin lesions range from mild erythema to lichenification. Environmental triggers include food (milk, soy, eggs, wheat, etc) airborne allergens (dust mites, dander etc), and *Staphylococcus aureus* colonization in the skin.



Genetic predisposition may also be implicated. Treatment involves the use of moisturizers, topical steroids and the avoidance of triggers.

 $\Box$  Contact dermatitis(CD): Contact dermatitis refers to acute inflammation of the skin caused by irritants or allergens and is characterized by pruritus, pain with erythematous or blistering lesions usually found on hands or other exposed surfaces. The majority of CD cases are examples of <u>irritant contact dermatitis</u> (ICD) in those who have had contact with acids, alkalies, etc.



<u>Allergic contact dermatitis</u> cases are hypersensitivity responses to allergens such as chemicals used in shoe industry, cosmetics, dyes, metal compounds and plants.



Irritant contact dermatitis is predominantly painful whereas allergic contact dermatitis is predominantly pruritic. Treatment involves symptomatic treatment and avoiding contact with triggers. Topical application of steroids (oral steroids for severe cases of ACD) and antihistamines are effective for ACD cases.

#### □ Seborrheic dermatitis (SD)

Seborrheic dermatitis (SD) refers to inflammation of the skin with a high density of sebaceous glands on the face, scalp and upper extremities. A normal skin organism called *Pityrosporum ovale* is believed to be associated with the condition. Dandruff, yellow, greasy scaling along the hair line, and pruritus are common in SD.



Treatment involves the use of tar shampoos and topical corticosteroids.

#### □ Skin cancer

People with fair and light skin are susceptible to developing <u>squamous cell and basal</u> <u>cell carcinomas</u> due to chronic sun exposure. Local spread and distance spread are common.



Treatment involves surgical dissection, cryosurgery, or occasionally radiotherapy. Basal cell carcinoma is a slow-growing, superficial, malignant. Local growth is destructive but distant metastasis is uncommon. Treatment involves surgery, cryosurgery or occasionally, radiation therapy.

□ **Melanoma:** Melanoma (malignant melanoma) arises from melanocytes in pigmented areas such as the skin, mouth, eyes and CNS.

□ **Bullous Pemphigoid:** Bullous pemphigoid is an autoimmune disorder of the skin producing chronic bullous lesions, particularly in the elderly. Tense bullae appear on the normally appearing reddened skin. Corticosteroids are effective.



#### □ Pemphigus vulgaris

Pemphigus vulgaris is an autoimmune disorder of the skin characterised by intraepidermal bullae and extensive erosions on healthy looking skin and mucous membranes. Treatment includes systemic use of corticosteroids, and sometimes with immunosuppressants. Maintenance therapy is essential.



#### □ Herpes simplex virus infections

Herpes simplex viruses are known to cause recurrent infections of the skin and mucous membranes. Mucocutaneous infections cause clusters of small painful vesicles on an erythematous base. HSV-1 causes oral lesions, whereas HSV-2 causes genital lesions. HSV-1 causes acute herpetic gingivostomatitis and (recurrent) herpes. Severe infections are treated with acyclovir or famciclovir and analgesics.

![](_page_26_Picture_2.jpeg)

#### □ Chickenpox

Chickenpox is an acute, systemic, and usually childhood, infection caused by the varicella-zoster virus (VZV). Usually chickenpox starts with mild constitutional symptoms, followed by skin lesions appearing in crops. These include macules, papules, vesicles, and crusting lesions. Generally the infection is self-limiting. Symptomatic treatment for pruritus, pain and fever may be necessary. The severe form (common in adults) requires antiviral treatment with acyclovir or famciclovir.

![](_page_26_Picture_5.jpeg)

#### □ Shingles (Herpes zoster)

Herpes zoster (shingles) results when varicella-zoster virus reactivates from its latent state in a posterior dorsal root ganglion. Pain along the affected nerve is the initial symptom. This is soon followed by the appearance of vesicular eruptions. Treatment includes antiviral agents, and corticosteroids in the severe form of the disease. Postherpetic neuralgia may occur in elderly patients at a later stage.

![](_page_26_Picture_8.jpeg)

#### □ Candidiasis

Infection by Candida species (commonly *C. albicans*). Skin infection can occur anywhere. Skin folds are the most commonly involved sites. Infection can also involve mucocutaneous sites. Treatment involves antifungal agents and drying agents.

![](_page_27_Picture_4.jpeg)

#### □ Molluscum contagiosum

Molluscum contagiosum is characterised by clusters of smooth, waxy or pearly umbilicated papules of 1-5 mm in diameter caused by the molluscum contagiosum virus. Lesions occur frequently on the face, trunk, pubis, penis and vulva. Treatment involves trichloroacetic acid application or the use of electrotherapy, cryosurgery, or laser surgery.

![](_page_27_Picture_7.jpeg)

#### □ Warts (Verruca vulgaris)

Skin warts are common, benign epidermal lesions associated with human papilloma virus (HPV) infection. They are asymptomatic and appear as flat, verrucous or cauliflower-like lesions on the skin. Palmar and plantar warts, periungual warts, flat warts, filiform warts and genital warts are common. Treatment involves the use of an irritant such as trichloroacetic acid, salicylic acid or podophyllum resin.

![](_page_27_Picture_10.jpeg)

Department of Oral Maxillofacial Surgery Lecture (4)

![](_page_28_Picture_2.jpeg)

#### □ Vitiligo

Vitiligo is the loss of skin melanocytes that cause areas of skin depigmentation of varying sizes. The cause of vitiligo is unknown. Autoimmunity is associated in about 30 % of cases. Diagnosis is clinical. Topical corticosteroids, psoralens plus ultraviolet A are used in the treatment of vitiligo.

![](_page_28_Picture_5.jpeg)

□ Lichen planus (LP); Lichen planus is a chronic recurrent inflammatory condition characterised by small, flat, polygonal, violaceous papules and scales. Oral reticular, erosive or bullous lesions are also common (in 50% of skin LP). Skin lesions are itchy and symmetrically distributed on the flexor surfaces of wrists, legs and lower back. Causes of LP are uncertain. Erosive oral lichen planus may carry malignant potential.

![](_page_28_Picture_7.jpeg)

#### **Psoriasis**

Psoriasis is an inflammatory disease characterised by well circumscribed erythematous papules and plaques covered with silvery scales. The cause is unknown. Triggers of the disorder include trauma, infection and certain drugs. Mild itching may be present. Treatment involves the use of emollients, vitamin D analogues, retinoids, tar, anthralin, corticosteroids, and phototherapy. Methotrexate and immunosuppressants are used for severe cases. Department of Oral Maxillofacial Surgery

#### 3rd Year General Medicine

![](_page_29_Picture_2.jpeg)

![](_page_29_Picture_3.jpeg)

#### **Erythema multiforme**

Erythema multiforme is an inflammatory reaction characterised by -target or -iris lesions on the skin. Onset is sudden with erythematous macules, papules, weals, vesicles and/or bullae on the distal extremities including the palms and soles. The face is also frequently involved. Lesions occur as a reaction to a drug or an infectious agent such as herpes simplex virus. Topical and systemic corticosteroids are effective for severe symptoms. Antiviral therapy is recommended if recurrences are frequent.

![](_page_29_Picture_6.jpeg)

#### Sore Throat and Sore Mouth

#### Acute pharyngitis (sore throat)

**Definition/description:** Acute pharyngitis is a generalized (acute) inflammation of the whole pharynx (including tonsils).

**Cause:** Causes include viral (40-60%), bacterial (5-30%), fungal (in immunocompromised patients), and non-infectious such as allergy, gastroesophageal reflux disease, post –nasal drip, chemical injury, smoking and trauma during endotracheal intubation. Glandular fever (infectious mononucleosis) caused by EBV also presents with sore throat in the initial stages of the disease. Streptococcal sore throat may present tonsillar exudates.

![](_page_30_Figure_5.jpeg)

![](_page_30_Picture_6.jpeg)

**Symptoms and signs:** The majority of patients develop only the sore throat. Other features include fever, headache, malaise, anorexia and loss of appetite. Palatal haemorrhagic spots may be seen in some cases. Anterior cervical lymph node enlargement occurs. Generalized lymphadenopathy is seen in cases with glandular fever.

![](_page_31_Picture_2.jpeg)

**Investigations:** Clinical. Throat swabs in resistant cases are useful. Conduct a monospot test if glandular fever is suspected.

**Management:** Analgesics or antipyretics are used for pain relief and fever. Antibiotics in severe cases (penicillin-based antibiotics are not to be used for those with glandular fever). Tonsillectomy is recommended for those patients with large tonsils (causing sleep apnoea) and for those with tonsillitis for more than two years.

**Complications:** These include rheumatic fever, glomerulonephritis, (due to betahaemolytic streptococcal pharyngitis) and parapharyngeal abscesses.

# Sore Mouth Or Chronic Atrophic Candidiasis

**Oral Candidiasis:** One of the most common manifestations of human candida infection is oral candidiasis. Symptomatic oral manifestations of oral candidiasis generally include white and red lesions. White lesions include acute pseudomembranous candidiasis (thrush), candidal leukoplakia (chronic hyperplastic candidiasis) and mucocutaneous candidiasis.

![](_page_31_Picture_8.jpeg)

Acute pseudomembranous candidiasis

leukoplakia

![](_page_32_Picture_1.jpeg)

chronic hyperplastic candidiasis Oral candidiasis Red lesions of candidiasis include erythematous (or atrophic) candidiasis comprising denture-related stomatitis, median rhomboid glossitis and antibiotic- or steroid-related candidiasis.

![](_page_32_Picture_3.jpeg)

Red lesion

**Oral symptoms and signs:** Pseudomembranous candidiasis is characterised by white curd–like mucosal plaques. These can be easily rubbed off with gauze to leave a raw, erythematous and bleeding base.

![](_page_32_Picture_6.jpeg)

Pseudomembranous candidiasis

In chronic hyperplastic candidiasis, the patch is a persistent lesion with white or speckled red/white appearance. This lesion cannot be rubbed off due to the keratotic nature of its development.

![](_page_33_Picture_1.jpeg)

chronic hyperplastic candidiasis

Chronic mucocutaneous candidiasis is often seen in patients with endocrinopathies and immune disorders. In these patients, oral white patches are irremovable. Skin, nails and other mucosal regions are also involved in this condition.

![](_page_33_Picture_4.jpeg)

Erythematous candidiasis is seen in long term antibiotic or steroid users. Wide spread erythema and soreness of the oral mucosa are common in these patients. Median rhomboid glossitis or glossal central papillary atrophy shows a rhomboid, central depapillated area anterior to the circumvallate papillae on the tongue. Occasionally lesions may be lobulated.

![](_page_33_Picture_6.jpeg)

Denture related stomatitis (also known as denture **sore mouth or chronic atrophic candidiasis**) presents mild erythema of the mucosa beneath the dental appliance. In most cases this occurs in upper, full denture wearers. Papillary hyperplasia on the vault of the palate may be seen in this condition and which requires surgical intervention before the denture is replaced. **Angular cheilitis** may be associated with this condition.

![](_page_34_Picture_1.jpeg)

**Investigations:** Clinical findings are suggestive of candidal involvement for cutaneous and mucosal lesions. Confirmatory tests include microscopic identification of the organisms in large numbers in smears or tissues. Culture studies or serology and skin tests are also useful.

**Management:** Antifungal treatment (Nystatin or Amphotericin B topical application) and identification and elimination of predisposing factors are essential therapeutic measures in the treatment of oral candidiasis. In addition, systemic antifungal treatment may be required for candidiasis of the gastrointestinal tract and other mucosal sites.

# **Management of Oral Candidiasis**

- Topical agents
  - Clotrimazole troches 10 mg
  - Clotrimazole 1% cream
  - Nystatin oral suspension 100,000 units/ml
  - Nystatin pastilles 100,000 units
- Systemic agents
  - Fluconazole 100mg
  - Itraconazole oral suspension 10mg/10ml
  - Amphotericin B, Voriconazole

April 200

NWFTC 📰

# Cardiovascular diseases

#### Introduction

The components of the cardiovascular system are the heart, blood vessels and blood. From a dental management point of view, dental practitioners are expected to have an adequate knowledge of some of the more common manifestations of cardiovascular diseases and their impact on oral health.

#### **Common Cardiovascular Symptoms**

# Heart Disease Symptoms

![](_page_35_Picture_7.jpeg)

ain in chest Lorem ipsum dolor sit amet, consectetur adipiscing elit. Donec iaculis interdum purus curabitu vquis.

# Trouble breathing

Lorem ipsum dolor sit amet, consectetur adipiscing elit. Donec iaculis interdum purus curabitu vquis.

![](_page_35_Picture_11.jpeg)

# Palpitations

Lorem ipsum dolor sit amet, consectetur adipiscing elit. Donec iaculis interdum purus curabitu vquis.

# Swelling of feet

![](_page_35_Picture_16.jpeg)

Lorem ipsum dolor sit amet, consectetur adipiscing elit. Donec iaculis interdum purus curabitu vquis.

# Feeling weak

Lorem ipsum dolor sit amet, consectetur adipiscing elit. Donec iaculis interdum purus curabitu vquis.

![](_page_35_Picture_20.jpeg)

![](_page_35_Picture_21.jpeg)

Lorem ipsum dolor sit amet, consectetur adipiscing elit. Donec iaculis interdum purus curabitu vquis.

**Chest pain:** Chest pain is one of the major symptoms of heart disease. Pain can also originate from lungs, oesophagus and thorax. Chest pain of cardiac origin occurs in angina pectoris, myocardial infarction, thoracic aortic dissection, pericarditis and myocarditis. Non-cardiac causes of chest pain include tension pneumothorax, oesophageal rupture, pulmonary embolism, pneumonia, pancreatitis, thoracic malignancies, gastroesophageal reflux, peptic ulcers, costochondritis, biliary tract disorders, herpes zoster infection involving the thorax, and chest trauma.
### 

# Quality of the chest pain due to:

o Angina and myocardial infarction, feels tight and crushing.

o Pericarditis and pulmonary in origin is sharp and worse on inspiration (pleuritic pain).

o Gastroesophageal reflux disease (GORD) has a burning quality.

o Peptic-acid disorders are deep and of a biting, gnawing or chewing quality.

# Location of the Chest Pain:

o Pain of IHD and GORD are retrosternal in location and can radiate to the left arm and the jaw.



o Pericarditis pain may radiate to the shoulders,

o Pain due to aortic dissection may radiate to the back.

o Pain of pulmonary origin can be located anywhere in the thorax.

# **Precipitating Factors:**

o Angina is precipitated by exercise, emotion, heavy meals or cold weather.

o If pain occurs at rest for more than 30 minutes, it should be considered as a pain of myocardial infarction until proven otherwise.

o Pain of GORD is associated with meals and a change in posture (bending down, for example).

o Pulmonary pain worsens with movements of the thorax.

# **Relieving Factors:**

o Both angina and pain due to oesophageal spasm are relieved by glyceryl trinitrate

(GTN) due to its action on smooth muscles.

o GORD pain is relieved by antacids. Antacids have no effect on pain of cardiac origin.

o Pericarditic pain is relieved by sitting forwards.

**Breathlessness:** Breathlessness (short of breath, or dyspnoea) is a normal symptom after heavy exertion, but becomes pathological if it occurs in clinical situations.

Non-cardiac causes of breathlessness include asthma, pneumonia, interstitial lung disease, chronic obstructive pulmonary disease, or psychogenic factors.

**Palpitations:** A patient's perception of cardiac activity as a racing, fluttering or skipping sensation is generally referred to as palpitations. Anxiety, exercise and febrile illness often cause heightened awareness of normal cardiac activity, however, these experiences are not to be regarded as examples of palpitations. Palpitations generally result from cardiac arrhythmia caused by premature atrial or ventricular contractions, and these are harmless. Coffee, alcohol, epinephrine, and theophylline are known to trigger palpitations.

**Postural hypotension:** Postural hypotension (orthostatic hypotension) results in faintness, dizziness and blurred vision within a few seconds of standing due to an excessive fall in blood pressure. This can also occur due to vagal stimulation after urination or defecation.

**Syncope:** Syncope is characterized by a brief loss of consciousness with a loss of postural tone followed by spontaneous recovery. The patient becomes pale, motionless and hypotensive with a weak pulse, cool extremities and shallow respiration.

**Ankle swelling:** Right heart failure is one of the major cardiac causes of ankle swelling. Other causes include varicose veins, venous insufficiency, lymphatic obstruction, surgery of the foot, burns, hypertension and insect bites or stings. Certain birth control pills, calcium channel blockers (e.g., nifedipine, amlodipine, diltiazem) and steroids may trigger ankle swelling. Ankle oedema during pregnancy is also common. Long periods of standing and long aeroplane flights.



### Department of OMS Lecture (6) P 2 3rd Year General Medicine

#### • Examination of the cardiovascular system

The major elements of a cardiovascular examination include observation, palpation and auscultation. Percussion of the chest was previously used to determine the size of the heart. This test was found to be unreliable and was abandoned. X-rays are now used to determine the size of the heart.

#### Methods

Ask the patient to strip to the waist. Female patients can cover up until that part of the chest needs to be examined. Inform the patient of what you are about to do. The patient should rest in a supine position with the upper body elevated at an angle of 30-45 degrees.



Assessment of the pulse and blood pressure are essential elements of the CVS examination.

**Inspection:** Look for tachypnoea (heart failure), malar flush showing a bluish tinge on the cheeks (mitral stenosis), xanthelasma and corneal arcus (hyperlipidaemia), cyanosis (heart failure), forceful carotid pulsations (aortic regurgitation), ankle oedema (heart failure), splinter haemorrhages in the nail bed under the nail (infective endocarditis), clubbing (congenital cyanotic heart disease, respiratory disease), pallor, (anaemia, congenital heart disease), surgical scars on the chest and chest deformities.





**Palpation:** Palpate the right radial pulse. Assess the rate (60-100 beats per minute) and rhythm.

# Radial Pulse

- The wrist is held in semi-flexed & semipronated position.
- Place pad of index finger, middle finger & ring finger on the radial artery against the lower end of radius.
- To detect a collapsing pulse, feel the pulse with the base of your fingers , then raise the patient's hand above his head.
- Palpate both radial pulse simultaneously to assess radio-radial delay.
- Palpate femoral & radial pulse simultaneously to assess radio-femoral delay.



Radial and femoral pulse should be recorded to detect if there is any delay, as found in coarctation of the aorta.



Carotid pulse should be palpated for its character.



Abnormal pulse characteristics can indicate valvular disease. Collapsing pulse is a sign of aortic regurgitation. This is assessed by raising the patient's left arm and with both hands, holding the forearm (one on the wrist and the other lower down) with your fingers on the ulnar side of forearm. If you feel the pulse vibrating back down your arm, it is indicative of collapsing pulse.



### Auscultation

Use a stethoscope with both a bell (which is better for low pitched sounds) and a diaphragm (used for high pitched sounds). Always feel the carotid pulse during auscultation of the heart. This will give you an idea of the part of the cardiac cycle (systole or diastole) in which the murmur occurs.



There are normally two heart sounds:

o **The first heart sound** (due to the closure of the mitral and tricuspid valves) is best heard at the apex. This signals the start of a systole. Auscultate the apex (using the bell) in the left lateral position.

o **The second heart sound** (due to the closure of the aortic and pulmonary valves) signals the start of the diastole. (Generally diastole is longer than systole).

The second heart sound can be best heard at the left sternal edge in the second intercostal space.

o The first heart sound (S1) is generally lower pitched than the second heart sound (S2). S1 is the \_lubb' and S2 is the \_dup'.

### **Common Investigations In Cardiology**

Common investigations in cardiology include chest x-rays, electrocardiograms (ECGs), exercise ECGs, ambulatory ECGs and BP monitoring. Echocardiography and angiography are also used in detecting coronary artery disease and other disorders involving the heart muscles and valves.

**Chest x-ray**: In cardiology, an x-ray of the chest is useful in identifying cardiomegaly.

**Electrocardiogram (ECG):** An ECG is used to measure the rate and regularity of heartbeats as well as the size and position of the chambers, the presence of any damage to the heart, and the effects of drugs or devices used to regulate the heart, such as a pacemaker.



**Ambulatory electrocardiography (AECG):** An ambulatory electrocardiogram records the electrical activity of the heart during usual activities. The most common type is the continuous recorder (such as the Holter monitor) which provides a 24- to

72-hour record of the electrical signals from the heart. Ambulatory electrocardiography is used to detect, characterise and document cardiac arrhythmias.



**Echocardiography** (cardiac ultrasound scanning): This is useful in assessing ventricular function and valvular abnormalities. It can detect blood clots inside the heart, pericardial fluid build-up, and problems with the aorta.

A type of echocardiography called Doppler ultrasound provides information on the blood flow through the chambers and valves of the heart.



**Angiography:** Angiography is an imaging technique used to visualise the lumen of arteries, veins and the heart chambers. This is done by injecting a radio-opaque contrast agent into the blood vessels and imaging using x-ray based techniques such as fluoroscopy.



Arterial blood gasses: Distributions of PaO2

The normal arterial values are:

. PaO2 10-13 kPa (values fall with age) . PaCO2 4.7-6.0 kPa

. pH 7.35–7.45 . Standard HCO3 23–27 mmol/l

**Estimation of cardiac enzymes**: These include creatinine kinase, transaminases and lactate dehydrogenase. These enzymes are useful in evaluating the status of cardiac disease. Troponin levels provide useful information on cardiac muscle damage, as seen in myocardial infarction.

**D-dimer (ELISA):** Helpful to exclude pulmonary embolism in patients with low pretest clinical probability or nondiagnostic lung scan.

# **Cardiovascular Diseases of Dental Interest**

### **Ischemic Heart Disease**

### DEFINITION

Ischemic heart disease (IHD) describes the condition in which atherosclerotic plaque is present in the coronary arteries, giving way to coronary artery disease (CAD).



#### Ischemic heart disease risk factors

- Hypertension.
- o Smoking.
- Diabetes mellitus.
- Family history (1st-degree relative <60yrs old with IHD).
- o Hyperlipidemia.
- Male gender

### • Angina (Angina pectoris)

**Definition/Description:** Angina, also called angina pectoris, is a common symptom characterized by pain of cardiac origin as a result of Ischemic heart disease (IHD). IHD causes an imbalance between the myocardial oxygen supply and demand. **Causes:** A reduction of the coronary arterial luminal diameter (by 70-90%) due to Ischemic heart disease (IHD) causes angina.

Non-IHD diseases that can cause angina include coronary artery spasm (Prinzmetal's angina), aortic stenosis, and cardiomyopathy.



**Symptoms and signs:** These include severe central chest pain (gripping, constricting, crushing or tightness) often with shortness of breath, faintness and pain radiating to the left (and sometimes right) arm, and into the neck and jaw. Pain is typically induced by exercise, emotion, a heavy meal and cold weather, and is relieved by rest and nitrates. This is called **stable angina**.



If angina is of recent onset, severe and rapidly worsening on minimal or no exertion, and lasts longer than a few minutes, it is considered as **unstable angina**. This is usually a forerunner of myocardial infarction (MI). The pain of myocardial infarction is of similar character and site to stable angina, but is more prolonged, more severe, and accompanied by nausea, vomiting, breathlessness, sweating, and abnormal heart rate and rhythm. This pain is not relieved by nitrates. Signs of angina are sometimes absent. Risk factors such as hypertension may be identified in a majority of cases.

**Investigations: Electrocardiogram** (<u>ECG</u>). This reading is abnormal during an attack, and often normal at rest. If the ECG has been recorded as normal during rest (in a patient with suspected angina), a treadmill exercise ECG or radionuclide scanning is usually considered.





Unstable Angina

ECG and BP are monitored during the recovery period after exercise. <u>Coronary</u> <u>angiography</u> is considered for those who may require angioplasty or bypass surgery.



A full blood count (FBC) and erythrocyte sedimentation rate (ESR) are required to exclude non-atheromatous causes of angina.

**Management:** Recognition and correction of risk factors such as hypertension, smoking, obesity, diabetes and Hyperlipidemia are the key factors in the management of angina.

Drug therapy for angina includes aspirin (75 mg daily), nitrates (isosorbide mononitrate 20 mg bd),  $\beta$ -blockers (atenolol 50 mg daily), and calcium channel antagonists (nifedipine 10 mg tid or amlodipine 5-20 mg/day, or ATP-sensitive potassium channel activators (nicorandil 10-20 mg bd).

Surgery includes coronary angioplasty revascularisation (with stents) for proximal arterial stenosis, and coronary artery bypass surgery for triple coronary artery disease.

### **Oral Manifestations and Dental Management Considerations**

o During the attack, the patient may feel acute pain in the jaw.

o Patients with stable angina can be treated with LA containing 2% lidocaine and adrenaline 1:80,000. Good practice is to take GTN before the commencement of treatment.

o If a patient with history of angina experiences chest pain during dental procedures:

- Treatment must be stopped immediately and the patient must be seated upright, given GTN (sublingual) and administered oxygen. Vital signs should be monitored.

- Pain should be relieved within 3-4 minutes. If pain persists (a symptom of MI), medical help must be summoned.

- Patient should be given 300 mg aspirin to chew and the administration of oxygen continued.

### • Myocardial infarction (MI)= heart attack

**Definition/description:** Death (necrosis) of a part of the heart muscle due to total occlusion of the coronary artery results in myocardial infarction.

**Cause:** Embolism following rupture of atheromatous coronary artery plaque is the major cause of myocardial infarction. MI is common in the winter months, and may be precipitated by vigorous exercise, major surgery or infections.



**Symptoms:** These include severe, crushing, central chest pain, often radiating to the neck, left arm and mandible, and not relieved by nitrates. Pain is usually associated with nausea, sweating, breathlessness and vomiting. In some cases symptoms do not occur and infarction is discovered incidentally when an ECG is performed at a later date. In this situation the infarct is called a **silent infarct**. Sudden death is due to ventricular fibrillation in 50% of heart attack patients.

**Signs:** Common signs of myocardial infarction include pallor, circulatory shock, tachycardia, low blood pressure, cyanosis and gallop rhythm. Gallop rhythm usually refers to the abnormal rhythm of the heart on auscultation. It includes three or four sounds, thus resembling the sound of a galloping horse.

**Investigations:** These include abnormal ECG findings and plasma cardiac enzymes such as creatine kinase, transaminases and lactate dehydrogenase (elevated). The troponin level is also elevated in MI. Chest x-rays are used to identify pulmonary oedema, and aortic dissections are required.

**Management:** Therapeutic management of MI includes aspirin (300 mg soluble) as soon as possible and opiates (diamorphine 2.5 mg IV with an antiemetic (metoclopramide 10 mg IV). Immediate transfer of the patient to ICU is required. ECG recording, bed rest with high flow oxygen administration, thrombolytic therapy (subcutaneous heparin 5000 IU eight hourly) and monitoring blood cardiac enzymes are other essential measures.

#### **Oral Manifestations and Dental Management Considerations**

o During the attack, patients may feel acute pain in the jaw

o Dental intervention in patients with myocardial infarction can precipitate arrhythmias. For those patients who have had a recent MI attack (in the last 6 months), elective surgical procedures are to be deferred. Simple emergency procedures under LA may be undertaken in consultation with the cardiologist.

o Patients who have had recent angioplasty, or a Coronary Artery Bypass Graft (CABG)

- Elective procedures need to be modified. LA with adrenaline/epinephrine is contraindicated for CABG patients, as arrhythmias may be precipitated by these agents. Emergency procedures need to be carried out in a hospital setting.

- Consultation with the cardiologist or cardiac surgeon is essential.

# **Cardiovascular Diseases of Dental Interest**

#### Heart failure (cardiac failure)

**Definition/description:** Heart failure is a clinical syndrome characterised by a change in the pumping function of the heart accompanied by typical symptoms such as shortness of breath or weakness.

**Cause**: These include ischaemic heart disease, hypertension, valvular heart disease, arrhythmias, pulmonary embolism, anaemia, thyrotoxicosis, myocarditis, cardiomyopathy, infective endocarditis, and thiamine deficiency.

**Symptoms:** Dyspnea on exertion, orthopnea, paroxysmal nocturnal dyspnoea, ankle swelling, fatigue and lethargy are common symptoms of cardiac failure.



**Signs:** These include ankle and sacral oedema, jugular venous distension, basal crepitations, hepatomegaly and gallop rhythm.

**Investigations**: Chest x-ray, ECG, echocardiography, and radionuclide imaging are used in the diagnosis of heart failure. Blood tests such as a complete blood count, blood creatinine, blood glucose, albumin, liver function tests, and thyroid function tests are also necessary.

**Management:** Management includes dietary salt restriction, diuretics, digitalis preparations (digoxin), vasodilators,  $\beta$ -blockers, and ACE inhibitors. If thyrotoxicosis is the cause, treatment is directed to correct it.

#### Lecture (6) P 4 3rd Year General Medicine

### **Cardiac arrhythmias**

**Definition/description:** Loss of rhythm resulting in irregularity of the heartbeat is called arrhythmia.



Cardiac arrhythmias are divided in to two categories: (1) supraventricular and ventricular arrhythmias, and (2) bradyarrhythmias and tachyarrhythmias.

Cardiac arrhythmias include isolated ectopic beats, bradycardia and tachycardia. Cardiac arrest can occur as a result of arrhythmias.

**Causes**: Cardiac arrhythmias are associated with a range of diseases of cardiac and noncardiac origin. These include ischaemic heart disease, myocardial infarction, rheumatic heart disease, congestive heart failure, pneumonia, obstructive lung disease, thyrotoxicosis, systemic infections, drug-related side effects, and electrolytic imbalances.

**Symptoms and signs:** Cardiac arrhythmias can cause palpitations and collapse. Irregularly irregular pulse and rapid atrial rhythm can be considered as signs of atrial fibrillation. The rate at the apex is faster than that at the radial artery.

A sinus rate greater than 100 beats per minute is considered as tachycardia. A sinus rate of less than 60 bpm is considered to be bradycardia.

#### **Rheumatic Fever (RF)**

**Definition** /description: Rheumatic fever is an acute inflammatory disease of the joints and heart caused by an autoimmune disorder which is preceded by a

streptococcal infection of the throat. In RF, heart valves can be damaged and become vulnerable to infective endocarditis.

**Cause:**  $\beta$ -haemolytic streptococcal infection of the throat results in producing cross reacting antibodies which then attack various normal body tissues including the heart valves, joints and skin.



Rheumatic nodules called Aschoff's nodules can occur on heart valves (the mitral valve in particular) causing valve incompetency.

**Symptoms and signs**: These include fever, polyarthritis, pericarditic pain, heart murmurs and heart failure.



**Diagnosis/Investigations:** These include microbiological confirmation of the causative organism via a throat swab. Other investigations include detection of the antistreptolysin antibody, ECGs, and ESR.

Major Criteria	Minor Criteria		
J: Joints (polyarthritis, hot/swollen joints)	P: Previous rheumatic fever		
. Heart (carditis, valve damage)	E: ECG with PR prolongation		
N: Nodules (subcutaneous, extensor surfaces)	A: Arthralgias		
E: Erythema marginatum (painless rash)	C: CRP and ESR elevated		
S: Sydenham chorea (flinching movement disorder)	E: Elevated temperature		

Management: Bed rest, analgesia and appropriate antibiotics are recommended.

### • Hypertension

**Definition/description:** Hypertension is characterised by the sustained elevation of resting systolic blood pressure (BP  $\geq$ 140 mmHg), diastolic blood pressure (BP  $\geq$ 90 mmHg) or both. Two types of hypertension are known: primary (85-95% of cases) and secondary hypertension.

**Cause:** Although in the majority of cases no obvious cause can be found, obesity, dietary salt, stress, and hereditary factors are frequently associated with hypertension. This is called **essential or primary hypertension**.

**Secondary hypertension,** on the other hand, is associated with glomerulonephritis, pyelonephritis, Cushing's syndrome, primary aldosteronism, hyperthyroidism, myxoedema, coarctation of the aorta, alcohol abuse, contraceptive pills, pregnancy and drugs such as prednisolone.

**Symptoms and signs:** The majority of patients with hypertension are asymptomatic. Occasionally, patients may complain of headache, dizziness, flushed face, fatigue, nose bleeds, and nervousness.

**Complications** may include cardiac failure, myocardial infarction, renal failure and retinal symptoms and signs. High BP is a consistent sign in these situations.

**Diagnosis/investigations:** Physical examination includes measurements such as weight, height, waist circumference, fundoscopic examination for retinopathy, auscultation of the neck for bruits, and a full cardiac, respiratory and neurological examination. If the blood pressure is severe and diagnosed for the first time in a young patient, testing for target organ damage should be carried out. Tests include

urinalysis, spot urine albumin/creatinine ratio, lipid profile, blood glucose estimation, and ECGs.

**Management:** Primary hypertension has no cure! Causes of secondary hypertension can be corrected. Treatment is aimed at reducing BP lower than 140/90 mmHg for those without signs of target organ disorders, and 130/80 for those with renal disease or diabetes.

Life style modifications include regular physical exercise (30 minutes a day most days of the week), weight loss down to a body mass index between 18.5 and 24.9, smoking cessation, a diet rich in fruits and vegetables and low-fat dairy products, a low salt intake (NaCl less than 6 g/day), and alcohol less than 30 ml/day for men and 15 ml/day for women. These life style modifications may be adequate for those who have no signs of target organ manifestations of hypertension. No medications are required as long as their BP is controlled.

Antihypertensive drugs are required for patients whose BP remains above 140/90 after 6 months of life style modifications.

Antihypertensive drugs include diuretics,  $\beta$ -blockers, calcium channel blockers, ACE inhibitors (Angiotensin-Converting Enzyme Inhibitor), and angiotensin-II receptor blockers.

### **Oral Manifestations and Dental Management Considerations**

o There are no specific oral manifestations of hypertension.

o The practitioner should be aware of oral side-effects of antihypertensive drugs. These include xerostomia, gingival hyperplasia (with nifedipine), salivary gland swelling (with clonidine), and increased post-operative bleeding (for those patients on aspirin).

o There are no dental treatment restrictions (including the use of vasoconstrictors) for those patients with mild to moderate but controlled hypertension. For those with severe hypertension, only emergency dental procedures can be undertaken. Consultation with the physician is necessary.

o Stressful situations should be avoided. Vasoconstrictors in LA need to be avoided for patients with severe hypertension (180/100 mmHg and above).

# **Renal Diseases**

### Introduction

Renal diseases are common. Infections, autoimmune disorders, diabetes, hypertension and other diseases can cause kidney damage. These patients require special dental management.



### **Common Symptoms of Renal Disease**

Common symptoms of kidney disease may include any of the following:

Nocturia, loin pain, polyurea, oliguria, fatigue, mild fever, incontinence, chills, altered mental states, peripheral neuropathy, nausea, vomiting, anorexia, pruritus, oedema, hypertension and anemia.



### **Examination of the Kidneys**

Kidneys are physically examined using a bimanual palpation technique.

# **Examination of Kidney**

Patient take a deep breath.
Feel lower pole of kidney and try to capture it between your hands.

### **Common Investigations in Renal Disease**

Common investigations for renal diseases include the following:

o **Urinalysis** - This test is done by stick testing for proteins, blood, glucose, ketones and ph.

o Microscopy for bacteria, white blood cells and casts.

o Biochemistry for creatinine, urea, calcium, urate and glucose.

o **Autoantibodies;** antineutrophil cytoplasmic antibody (ANCA, ANF) can also be detected in the renal involvement of vasculitis.

- o Ultrasounds are carried out for kidney size and to detect any obstructions.
- o X-rays for size, shape and position of the kidneys and suspected kidney stones.
- o Pyelography.
- o Radionuclide scans are done to asses asymmetrical kidney function.
- o Kidney biopsies are done for final diagnosis.
- o **Renal angiography** is carried out for renovascular disease

### **Renal Diseases of Dental Interest**

### • Urinary tract infections (UTIs)

**Definition/description:** A common bacterial infection of the urinary tract which sometimes can involve the kidneys (pyelonephritis), bladder (cystitis) or prostate (prostitis). Fifty percent of women are infected with urinary tract infections and become symptomatic at some time during their lives.

**Cause:** Bacteria involved include *E. coli, Enterobacter spp, Klebsiella spp, Proteus spp, Pseudomonas aeroginosa, Staph spp,* enterococci, streptococcus groups B, D, and G and *Strep. viridians.* 

**Symptoms and signs:** These include fever, incontinence, dysuria, chills, frequent urination, suprapubic tenderness (cystitis) or tenderness over the renal angle (pyelonephritis), and haematuria.

**Investigations:** Urinalysis for microscopy and culture. Blood pressure recording and levels of plasma electrolytes and creatinine are commonly investigated in UTIs.

**Management:** Plenty of water to drink, appropriate antibiotics and symptomatic treatment for fever are the routine management protocols undertaken in UTIs.

# • Acute glomerulonephritis (GN)

**Definition/description:** Glomerulonephritis (GN) is a complex inflammatory disease of the glomeruli which can be caused by several factors and may manifest as acute GN, nephrotic syndrome and chronic GN.



**Causes:** Acute GN is caused by a preceding infection of *Streptococcus pyogenes* (presenting as a sore throat in children, for example). Occasionally this may follow viral infections (including hepatitis B virus infection) and renal involvement in multisystem disorders.

**Symptoms and signs:** Acute GN is of sudden onset. Symptoms and signs include headache, hypertension, vomiting, loin pain, facial oedema in the morning, haematuria, proteinuria, and uraemia and a reduced amount of urine.

Investigations: Investigations for acute GN include the following:

1. Urine examination includes microscopy for RBC casts and dysmorphic RBC, urine 24-hour protein excretion, and creatinine clearance.

2. Blood examination includes serum for urea and electrolytes (U&E), full blood count (FBC), erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), albumin, antinuclear antibody (ANA), antineutrophil cytoplasmic antibody (ANCA), DNA binding, complement (C3 and C4), antistreptolysin O (ASO), and HBsAg.

3. Culture is carried out from samples of blood, throat swabs, ear swabs (if otitis is present) and skin swabs (if cellulitis is present).

4. Chest x-ray

5. Renal ultrasound.

6. Renal biopsy.

**Management:** Bed rest, fluids, antibiotics, and management of hypertension are essential measures. As a complication, acute GN may progress to chronic GN. In chronic GN, the kidneys are small, scarred and unable to function adequately. Clinically, symptoms and signs include those of chronic renal failure, including hypertension and anemia.

### • Nephrotic syndrome

**Definition/description:** Nephrotic syndrome is a form of glomerulonephritis characterised by the heavy leaking of plasma proteins into the urine, resulting in hypoalbuminaemia.

**Cause:** Glomerulonephritis (GN), diabetes, SLE, infections, amyloidosis, drugs such as NSAIDs or penicillamine, and malignancies (such as lymphoma or leukaemia) may cause nephrotic syndrome.

**Symptoms and signs:** These include peripheral oedema and swelling of the eyelids, ascites, pleural effusion and frothy urine due to the presence of protein (proteinuria). **Investigations:** These include:

1. A 24-hour urine sample for creatinine clearance and protein.

2. Microscopy for RBC and casts

3. Blood examination for U&E and RBC, cholesterol, FBC, ESR, CRP, Ig level, ANA, ANCA, C3and C4.

4. Renal venogram (a diagnostic procedure that uses x-rays and intravenous (IV) contrast dye to visualise the veins within the kidneys and those carrying blood away from the kidneys) or Doppler ultrasound to assess the size, shape and location of the kidneys.

**Management:** Diuretics for oedema, However, a high-protein diet isn't recommended for nephrotic syndrome, infusions of salt–poor albumin to raise serum albumin levels, and corticosteroids or immunosuppressive drugs. Venous thromboses and pulmonary embolism may occur as complications of nephrotic syndrome. Anticoagulation (with streptokinase) may become necessary.

# • Renal Failure (RF): acute and chronic RF

**Definition/description:** Renal failure is the loss of renal function leading to uraemia. Two forms exist: acute and chronic RF. chronic RF is characterised by a gradual and permanent loss of renal function.

**Cause:** These include diabetes mellitus, GN, pyelonephritis, hypertension, renal stones, bladder outlet obstruction, and connective tissue disease.

**Symptoms and signs: Acute renal failure** (ARF) is also called acute kidney injury (AKI). This is characterised by a rapidly progressive loss of renal function resulting in oliguria, and fluid and electrolyte imbalances. Acute RF involves rapid deterioration of renal function within hours or days.

**Causes of Acute renal failure** include injuries (due to accidents), complications of surgeries (such as bypass surgery on the heart restricting blood flow to the kidneys for extended periods of time), chemicals and accidental overdoses of drugs.

Renal failure accompanied by noticeable symptoms is termed uraemia which is characterised by high levels of urea in the blood. The term uraemia is used for the illness accompanying kidney failure.



**In chronic RF** any of the following symptoms and signs may be encountered: Apathy, confusion, drowsiness, ammoniacal breath odour, brown-coated tongue, metabolic acidosis leading to over breathing, anorexia, nausea, vomiting, bleeding or bruising tendencies, anemia, polyuria, peripheral oedema, increased pigmentation, ascites, pleural effusion and pericarditis.

Table 3: Causes of Chronic Renal Failure					
Pathologic	Physiologic				
Glomerulonephritis	Hypertension				
Interstitial nephritis	Diabetes mellitus				
Tumors	Chronic urinary tract				
	infections				
Transplant rejection	Congenital abnormalities				
Congenital disease	Vascular disease				
	HIV				

**Investigations:** Biochemical: increased urea and creatinine, hyperkalaemia (abnormally high levels of potassium in the circulating blood), hypocalcaemia (abnormally low concentrations of calcium in the circulating blood), hyperphosphataemia (abnormally high levels of phosphates in the circulating blood), and hyponatraemia (abnormally low concentrations of sodium ions in the circulating blood). Urine examination includes microscopy for casts, protein analysis, specific gravity and creatinine clearance. Radiology and biopsy includes plain x-ray, retrograde pyelography and renal biopsy.

**Management:** Management of BP is an essential part of the treatment. BP should be maintained at less than 130/80 mmHg in chronic renal failure (CRF), and for those CRF patients with diabetes, BP should be maintained at less than 120/70mmHg. Vitamin D, avoidance of nephrotoxic drugs such as tetracyclines, a diet low in protein and salt, and renal dialysis (haemodialysis or chronic ambulatory peritoneal dialysis) are the recommended management strategies for CRF.



### **Dental Management and Considerations**

- The dentist should consult the physician prior to treating patients with chronic renal failure.
- Dental extractions should be scheduled to follow recent renal dialysis, after the heparin effect has worn off.
- RF patients on immunosuppressive agents and corticosteroids are predisposed to oral infections.
- Nephrotoxic agents are to be avoided (tetracyclines, for example).
- Those CRF patients on dialysis often carry hospital viruses. Dentists should be aware of these patients contracting cytomegalovirus (CMV) and Epstein-Barr virus (EBV) infections and strict infection control protocols must be followed.

Lecture (8)

# Vitamins

Vitamins are a class of organic compounds categorized as essential nutrients. They are required by the body in very small amounts. They fall in the category of micronutrients. Vitamins do not yield energy but enable the body to use other nutrients. Since the body is generally unable to synthesize them (at least in sufficient amounts) they must be provided by food. A well balanced diet supplies in most instances the vitamin needs of a healthy person.

### Vitamins are classified as:

- 1. Water soluble (vitamin B group and C)
- 2. Fat soluble (vitamin A, D, E and K).

**The B vitamins** include biotin, folate, niacin, pantothenic acid, riboflavin, thiamine, pyridoxine and B12.

Vitamins	Food source	
(Vitamin A (Retinol)	Cod liver oil, carrots	
Vitamin B <sub>1</sub> ( <u>Thiamine</u> )	Rice bran	
Vitamin C (Ascorbic acid)	Citrus, most fresh foods	
Vitamin D ( <u>Calciferol</u> )	Cod liver oil	
Vitamin B <sub>2</sub> (Riboflavin	Meat, eggs	
Vitamin E ( <u>Tocopherol</u> )	Wheat germ oil, unrefined vegetable oils	
Vitamin B <sub>12</sub> (Cobalamins)	Liver, eggs, animal products	
Vitamin K (Phylloquinone/phytol naphthoquinone)	Leafy green vegetables	
Vitamin B <sub>5</sub> ( <u>Pantothenic acid</u> )	Meats, whole grains, in many foods	
Vitamin B <sub>7</sub> (Biotin)	Meats, dairy products, eggs	
Vitamin B <sub>6</sub> (Pyridoxine)	Meat, dairy products	
Vitamin B <sub>3</sub> (Niacin)	Meat, eggs, grains	
Vitamin B <sub>9</sub> (Folic acid)	Leafy green vegetables	

# • Biotin (B7)

Biotin acts as a coenzyme which assists in the metabolism of carbohydrates and fats. Principal sources of biotin include liver, kidney, egg yolk, yeast, cauliflower, nuts and legumes. Deficiency of biotin results in dermatitis, glossitis and metabolic acidosis.



RDA; 30 mcg

### • Folate (folic acid)

Folates are necessary for RBC maturation and the synthesis of purines and pyrimidines. They are required for the development of the foetal nervous system. Folate is available in leafy vegetables, fruits, enriched cereals and breads, and meats. Its bioavailability is greater when it is added to enriched grain foods. Prolonged cooking destroys folate.

Folate deficiency is common. This occurs as a result of inadequate intake or malabsorption.

Folate deficiency causes megaloblastic anaemia and neural tube birth defects.





### • Niacin (nicotinic acid, niacinamide)

**Niacin (B3)** is vital in cell metabolism. Principal sources include red meat, fish, poultry, milk, legumes and enriched bread and cereals.

A deficiency of niacin results in pellagra.

This is characterized by dermatitis, diarrhea and central nervous system dysfunction (Dementia) . Untreated pellagra will lead to death, sometimes called the "fourth D





RDA; 16mg M, 14mg F

### • Pantothenic acid (Vit B5)

Deficiency; result in dermatitis, hair loss & wt. loss.

RDA; 5mg



### • Riboflavin (vitamin B2)

Riboflavin is necessary for many aspects of carbohydrate and protein metabolism, and the integrity of mucous membranes. Principal sources of riboflavin include milk, cheese, meat, liver, eggs and enriched bread and cereals.

Deficiency in riboflavin results in cheilosis, angular stomatitis and corneal vascularization.



RDA; 1.3mg M, 1.1mg F

### • Thiamin (vitamin B1)

Thiamin is required for carbohydrate, fat, amino acid, glucose and alcohol metabolism.

Principal sources of B1 include, liver, whole grains and nuts. Thiamine is also necessary for myocardial function, and central and peripheral nerve cell function.

A deficiency of thiamine includes beriberi, characterized by peripheral neuropathy, and heart failure.



RDA; 1.2mg M, 1,1mg F

# • Vitamin B6 group (pyridoxine, pyridoxal and pyridoxamine)

The vitamin B6 group is necessary for many aspects of nitrogen metabolism, nucleic acid biosynthesis, linoleic acid, and lipid and carbohydrate metabolism.

Principal sources include liver, whole grain, fish and legumes.

Deficiencies result in seizures, anaemias, neuropathies and seborrhoeic dermatitis.



RDA; 1.3mg

# • Vitamin B12 (cobalamins)

Vitamin B12 is necessary for RBC maturation, neural functioning, DNA synthesis, and repair.

Principal sources include beef, pork, fish, poultry, eggs and fortified cereals.

A deficiency results in megaloblastic anaemia, neurologic deficits such as paraesthesia, and ataxia.



RDA; 2.4 mcg

### • Vitamin C (ascorbic acid)

Vitamin C is required for collagen formation, hormone and amino acid formation, and wound healing.

Principal sources include citrus fruits, tomatoes, potatoes, broccoli, strawberries and capsicums (sweet peppers).

A deficiency in vitamin C results in scurvy, characterized by hemorrhages, loose teeth, gingivitis and bone defects.



RDA; 90mg M, 75mg F

### • Vitamin A (retinol)

Vitamin A is necessary for the formation of rhodopsin (a photoreceptor pigment in the retina), epithelial integrity and lysosome stability.

Principal sources of this vitamin include fish liver oils, liver, egg yolks, butter and vitamin A fortified dairy products.

Deficiency includes night blindness, xerophthalmia and keratomalacia.



Xerophthalmia RDA; 900 mcg M, 700 mcg F keratomalacia

### • Vitamin D (cholecalciferol, ergocalciferol)

Vitamin D is necessary for calcium and phosphorus absorption and for resorption, mineralization and maturation of bones.

Sources include ultraviolet radiation of the skin, fortified milk, fish liver oils, butter and eggs.

Deficiencies include rickets in children and osteomalacia in adults.



RDA; 600 IU

### • Vitamin K

Vitamin K is necessary for the formation of prothrombin, other coagulation factors and bone proteins.

Sources include green leafy vegetables, pork, liver, vegetable oils and soy beans. Vitamin K deficiency results in bleeding due to prothrombin decrease and other clotting factor deficiencies.



RDA; 120mcg M, 90mcg F

# • Vitamin E (Alpha tocopherol)

Vitamin E is necessary as an intracellular antioxidant, and as a scavenger of free radicals in biologic membranes.

Deficiency of vitamin E results in RBC hemolysis and neurologic deficits.



RDA; 15mg (22 IU)

Lecture (9)

#### Poisoning, Overdose, Antidotes

Poisoning refers to the development of dose-related adverse effects following exposure to chemicals, drugs, or other xenobiotic. To paraphrase Paracelsus, the dose makes the poison. Although most poisons have predictable dose-related effects, individual responses to a given dose may vary because of genetic polymorphism, enzymatic induction or inhibition in the presence of other xenobiotics, or acquired tolerance. Poisoning may be local (e.g., skin, eyes, or lungs) or systemic depending on the route of exposure, the chemical and physical properties of the poison, and its mechanism of action. The severity and reversibility of poisoning also depend on the functional reserve of the individual or target organ, which is influenced by age and preexisting disease.

#### **EPIDEMIOLOGY**

More than 5 million poison exposures occur in the United States each year. Most are acute, are accidental (unintentional), involve a single agent, occur in the home, result in minor or no toxicity, and involve children <6 years of age. Pharmaceuticals are involved in 47% of exposures and in 84% of serious or fatal poisonings. About 20-25% of exposures require bedside health-professional evaluation, and 5% of all exposures require hospitalization.

Overall, the mortality rate is low: <1% of all poisoning exposures. It is significantly higher (1-2%) among hospitalized patients with intentional (suicidal) overdose or complications from drugs of abuse, who account for the majority of serious poisonings.

Acetaminophen is the pharmaceutical agent most often implicated in fatal poisoning. Overall, carbon monoxide is the leading cause of death from poisoning, patients with such poisoning are typically dead when discovered.

### DIAGNOSIS

Although poisoning can mimic other illnesses, the correct diagnosis can usually be established by the history, physical examination, routine and toxicologic laboratory evaluations, and characteristic clinical course.

### [1] HISTORY

The *history* should include the time, route, duration, and circumstances (location, surrounding events, and intent) of exposure; the name and amount of each drug, chemical, or ingredient involved; the time of onset, nature, and severity of symptoms;

the time and type of first-aid measures provided; and the medical and psychiatric history.

Relevant information may be available from family, friends, paramedics, police, pharmacists, physicians, and employers, who should be questioned regarding the patient's habits, hobbies, behavioral changes, available medications, and antecedent events.

### [2] PHYSICAL EXAMINATION AND CLINICAL COURSE

The *physical examination* should focus initially on vital signs, the cardiopulmonary system, and neurologic status. The patient should also be examined for evidence of trauma and underlying illnesses. Examination of the eyes (for nystagmus and pupil size and reactivity), abdomen (for bowel activity and bladder size), and skin (for burns, bullae, color, warmth, moisture, pressure sores, and puncture marks) may reveal findings of diagnostic value. Measuring core temperature is especially important, even in difficult or combative patients, since temperature elevation is the most reliable prognosticator of poor outcome in poisoning from stimulants (e.g., cocaine) or drug withdrawal (e.g., alcohol or GHB). The final step is to attempt to identify the particular agent involved by looking for unique or relatively poison-specific physical or ancillary test abnormalities.

### [3] LABORATORY ASSESSMENT

Laboratory assessment may be helpful in the differential diagnosis.

The electrocardiogram (ECG) can be useful for rapid diagnostic purposes.

*Radiologic studies* may occasionally be useful.

*Toxicologic analysis* of urine and blood (and occasionally of gastric contents and chemical samples) can sometimes confirm or rule out suspected poisoning.

*Quantitative serum tests* are useful for evaluation of patients poisoned with acetaminophen, alcohols (including ethylene glycol and methanol),anticonvulsants, barbiturates, digoxin, heavy metals, iron, lithium, salicylate, and theophylline as well as for the presence of carboxyhemoglobin and methemoglobin.

The response to antidotes is sometimes useful for diagnostic purposes.

### TREATMENT

Treatment goals include support of vital signs, prevention of further poison absorption (decontamination), enhancement of poison elimination, administration of specific antidotes, and prevention of reexposure (**Table 1**). Specific treatment depends on the

identity of the poison, the route and amount of exposure, the time of presentation relative to the time of exposure, and the severity of poisoning.

Knowledge of the offending agents' pharmacokinetics and pharmacodynamics is essential.

	, , , , , , , , , , , , , , , , , , ,					
Supportive Care						
Airway protection	Treatment of seizures					
Oxygenation/ventilation	Correction of temperature abnormalities					
Treatment of arrhythmias	Correction of metabolic derangements					
Hemodynamic support	Prevention of secondary complications					
Prevention of Further Poison Absorption						
Gastrointestinal decontamination	Decontamination of other sites					
Gastric lavage	Eye decontamination					
Activated charcoal	Skin decontamination					
Whole-bowel irrigation	Body cavity evacuation					
Dilution						
Endoscopic/surgical removal						
Enhancement of Poison Elimination	n					
Multiple-dose activated charcoal	Extracorporeal removal					
administration	Hemodialysis					
Alteration of urinary pH	Hemoperfusion					
Chelation	Hemofiltration					
	Hemofiltration					
	Hemofiltration Plasmapheresis					
	Hemofiltration Plasmapheresis Exchange transfusion					
	Hemofiltration Plasmapheresis Exchange transfusion Hyperbaric oxygenation					
Administration of Antidotes	Hemofiltration Plasmapheresis Exchange transfusion Hyperbaric oxygenation					
Administration of Antidotes Neutralization by antibodies	Hemofiltration Plasmapheresis Exchange transfusion Hyperbaric oxygenation Metabolic antagonism					
Administration of Antidotes Neutralization by antibodies Neutralization by chemical binding	Hemofiltration Plasmapheresis Exchange transfusion Hyperbaric oxygenation Metabolic antagonism Physiologic antagonism					
Administration of Antidotes Neutralization by antibodies Neutralization by chemical binding Prevention of Reexposure	Hemofiltration Plasmapheresis Exchange transfusion Hyperbaric oxygenation Metabolic antagonism Physiologic antagonism					
Administration of Antidotes Neutralization by antibodies Neutralization by chemical binding Prevention of Reexposure Adult education	Hemofiltration Plasmapheresis Exchange transfusion Hyperbaric oxygenation Metabolic antagonism Physiologic antagonism Notification of regulatory agencies					

### **TABLE 1. Fundamentals of Poisoning Management**

### **OVERDOSE**

An overdose is a biological response to when the human body receives too much of a substance or mix of substances. An overdose can be intentional or accidental. People can overdose on illicit drugs, alcohol, prescription medications, and many other substances. In many cases, overdoses are fatal, although most individuals who have

overdosed can be saved if medical treatment is provided quickly enough. However, the most common cause of death during any chemical overdose is respiratory failure.

### Drug Overdose Causes

The cause of a drug overdose is either by accidental overuse or by intentional misuse. Accidental overdoses result from either a young child or an adult with impaired mental abilities swallowing a medication left within their grasp. An adult (especially seniors or people taking many medications) can mistakenly ingest the incorrect medication or take the wrong dose of a medication. Purposeful overdoses are for a desired effect, either to get high or to harm oneself.

### **Drug Overdose Symptoms**

- Problems with vital signs (temperature, pulse rate, respiratory rate, blood pressure) are possible and can be life threatening. Vital sign values can be increased, decreased, or completely absent.
- Sleepiness, confusion, and coma (when someone cannot be aroused) are common and can be dangerous if the person breathes vomit into the lungs (aspirated).
- Skin can be cool and sweaty, or hot and dry.
- Chest pain is possible and can be caused by heart or lung damage. Shortness of breath may occur. Breathing may get rapid, slow, deep, or shallow.
- Abdominal pain, nausea, vomiting, and diarrhea are possible. Vomiting blood, or blood in bowel movements, can be life threatening.
- Specific drugs can damage specific organs, depending on the drug.

### **Drug Overdose Treatment**

Treatment will be dictated by the specific drug taken in the overdose. Information provided about amount, time, and underlying medical problems will be very helpful.

- On rare occasions, the stomach may be washed out by <u>gastric lavage</u> (<u>stomach</u> <u>pumping</u>) to mechanically remove unabsorbed drugs from the stomach.
- <u>Activated charcoal</u>, also known as activated carbon, may be given to help bind drugs and keep them in the stomach and intestines. This reduces the amount absorbed into the blood. The drug, bound to the charcoal, is then expelled in the stool. Often, a cathartic is given with the charcoal so that the person more quickly evacuates stool from their bowels. Activated charcoal is a fine black powder made from bone char, coconut shells, peat, petroleum coke, coal, olive pits



• Agitated or violent people may need physical restraint and sometimes <u>sedating</u> <u>medications</u> in the emergency department until the effects of the drugs wear off.

Lecture (9)

#### ANTIDOTES

Antidotes counteract the effects of poisons by **<u>neutralizing</u>** them (e.g., antibodyantigen reactions, chelation, chemical binding) or by **<u>antagonizing</u>** their physiologic effects (e.g., activation of opposing nervous system activity, provision of a competitive metabolic or receptor substrate). Poisons or conditions with specific antidotes include acetaminophen, anticholinergic agents, anticoagulants, benzodiazepines, beta blockers, calcium channel blockers, carbon monoxide, cardiac glycosides, cholinergic agents, cyanide, drug-induced dystonic reactions, ethylene glycol, fluoride, heavy metals, hypoglycemic agents, isoniazid, membrane-active agents, methemoglobinemia, opioids, sympathomimetics, and a variety of envenomations.

Antidotes can significantly reduce morbidity and mortality rates but are potentially toxic if used for inappropriate reasons. Since their safe use requires correct identification of a specific poisoning or syndrome.

	Antidote	Indications		Antidote	Indications
	Acetylcysteine	Paracetamol		Calcium folinate	Methotrexate
	Acetylcysteine	Faracetainor		Cyproheptadine	Serotonin syndrome
	Activated charcoal	Many oral poisons		Dontrolono	Neuroleptic malignant
	Calcium gluconate ge	el Hydrofluoric acid		Dantiolene	syndrome
	Dicobaltedetate	Cvanide		Desferrioxamine	Iron
	Flumazenil	Benzodiazepines		Digoxin-specific antibody fragments	Digoxin
Ι.	Glucagon	Beta blockers		Ethanol (oral)	Ethylene glycol, methano
	Hydroxocobalamin	Cvanide		Ethanol (intravenous)	Ethylene glycol, methanol
	later linid			Fomepizole	Ethylene glycol, methano
	Intralipid	Local anesthetics		Octreotide	Sulphonylureas
N	lethylthioninium chlor (methylene blue)	ide Methaemoglobinemia		Phytomenadione (iv) (vitamin K)	Anticoagulants
	Naloxone	Opioids		Polyethylene glycol	Gut decontamination: Iron lithium
	Sodium nitrite Sodium thiosulphate	e Cyanide		Pralidoxime	Organophosphate insecticide
Γ	Antidote	Indications	<u> </u>		
	Botulinum antitoxin	Botulism			
3	Sodium calcium edetate	Lead			
	Succimer	Heavy metal poisoning			

#### List of antidotes
#### Liver diseases

#### Introduction

Liver diseases, are common. From the dental practice point of view, disorders of this organ is important because of their direct and indirect relevance to oral health. Liver diseases are caused by viral infections, alcohol, autoimmunity, malignancy or genetic disorders.

#### General Signs and Symptoms of Liver Disease

**Jaundice:** Also called icterus, jaundice is characterized by yellow pigmentation of the skin, oral mucous membrane and sclera due to deposition of bilirubin. Jaundice is detectable when bilirubin is above 30-60 mmol/L. Jaundice is often seen in liver disease such as hepatitis or liver cancer. It may also indicate obstruction of the biliary tract by, for example, gallstones or pancreatic cancer. Less commonly, jaundice is congenital in origin (e.g., biliary atresia).





**Spider angioma** (spider nevi): These are small arterial dilatations on the skin of the face and neck. They contain a central red spot and reddish extensions which radiate outwards like a spider's web. They occur in healthy adults and young children. Having more than five spider nevi generally indicates liver disease.



**Palmar erythema:** Redness of the palms is called palmar erythema. These are often referred to as liver palms. Palmar erythema also occurs in pregnancy, polycythemia and thyrotoxicosis.



**Dupuytren"s contracture:** This condition is characterized by fixed flexion of the little, and sometimes ring, finger due to thickening of the palmar fascia. Dupuytren's contracture has a genetic background in some cases. It also occurs in patients with diabetes, alcoholism and epilepsy.



**Finger clubbing:** Finger (nail) clubbing is characterised by the loss of the normal angle at the bed of the nails. This is a common feature in liver cirrhosis (primary biliary cirrhosis in particular).



**Ascites:** Ascites is characterised by the accumulation of serous fluid in the peritoneal cavity. This causes abdominal distension and bulging flanks.



Leukonychia: White discolouration appearing on the nails is called leukonychia.



**Terry's nail:** This is a physical finding in which fingernails and/or toenails appear white with a brown or pink arc at the tip of the nails.



**Caput medusae:** This is characterised by a pattern of dilated cutaneous veins radiating from the umbilical area.



Ankle oedema: Abnormal accumulation of interstitial fluid in the tissues around the ankles causes ankle oedema. Under these circumstances ankle oedema is generally of pitting dependant type. Non-pitting oedema of the lower limbs is seen in hypothyroidism, lymphoedema and allergy.



Petechiae and ecchymosis: Petechiae are minor haemorrhagic spots on the skin and mucous membranes which are smaller than 3 mm in diameter. They are usually caused by thrombocytopenia or deficiency of clotting factors. Ecchymosis is a subcutaneous collection of blood as a result of haemorrhage. These lesions are larger than petechiae.



#### **Investigations in Liver Disease**

Depending on the type and severity of liver disease, any of the following investigations may be necessary:

- 1. Full Blood Count (FBC) for platelet count and microcytosis
- 2. Liver Function Tests for liver dysfunction Test Normal Range 0-1 MG/DL **Bilirubin Total** Conjugated (D. Bilirubin) 0-0.35 MG/DL Unconjugated (I.D. Bilirubin) 0.2-0.65 MG/DL SGOT 10-40 IU/L SGPT 10-40 IU/L **Alkaline Phosphatase** 40-112 U/L **Total Protein** 6-8.5 GM/DL Albumin 3.5-5 GM/DL
- 2-3.5 GM/DL Globulin 3. Serological investigations for viral hepatitis:
- a. Anti-HBs Ag (previous HBV infection or immunisation)
- b. IgM Anti-HBcAg recent HBV infection
- c. IgM Anti-HAV recent HAV infection
- d. Anti-HDV previous delta virus infection

Lecture (10)

- e. Anti-HCV chronic hepatitis C infection
- f. HCV-RNA active HCV infection

4. Radiology for detection of air within the biliary tree and for the presence of gallstones

- 5. CT scan for lesions within the liver parenchyma
- 6. MRI for lesions of the liver, biliary tree and pancreas
- 7. Angiography of the hepatic artery for hepatic tumours and portal hypertension
- 8. Endoscopy for gastroesophageal varices
- 9. Liver biopsy for the staging of most hepatic disorders

## Liver Diseases of Dental Interest

### 1. Viral Hepatitis

Viral hepatitis is inflammation of the liver caused by a range of viruses. The disease is seen in acute and chronic forms. There are six types of viral hepatitis -A and E, which are transmitted by the faeco-oral route, and B, C, D and G, which are blood-borne infections. Other viruses that can cause hepatitis include Epstein-Barr virus (EBV), herpes simplex virus (HSV), cytomegalovirus (CMV), and yellow fever virus.

#### Hepatitis A virus infection

Hepatitis A viruses are RNA viruses transmitted through the fecal-oral route. Risk groups for hepatitis A virus infection include food handlers and day care workers with poor hygiene. Incubation of the virus is between <u>15 and50 days</u>. There is no carrier state for this infection. Prophylaxis is through immunoglobulin (Ig) and vaccine. Immunity following infection is probably for life.



### Hepatitis E virus infection

Hepatitis E viruses are defective RNA viruses transmitted through fecal-oral routes. Risk groups for hepatitis E virus infections include travellers to endemic areas such as India, Asia, Africa and Central America. Incubation of the virus is 15-64 days. There is no carrier state and no prophylaxis available for this infection. Immunity following this infection may last a lifetime.

### Hepatitis B virus infection

Hepatitis B viruses are DNA viruses. Hepatitis B is not transmitted by the faeco-oral route but is a blood-borne agent, transmitted by inoculation. percutaneous, sexual or perinatal routes. Risk groups for hepatitis B virus infections include IV drug users, healthcare workers dealing with blood, hemodialysis patients, male homosexuals, heterosexuals with multiple partners, and recipients of blood transfusions. Incubation of the virus is <u>30-180 days</u>.

There is a carrier state for this infection. Prophylaxis is through Hepatitis B immunoglobulin (HbIg) and vaccine (Three doses at 0, 1 and 6 months are required for complete protection).. Immunity following infection is probably lifelong.



### Hepatitis C virus infection

Hepatitis viruses are RNA viruses transmitted through percutaneous, and occasionally sexual or perinatal routes. Risk groups for hepatitis C virus infections include IV drug users, healthcare workers dealing with blood, hemodialysis patients, and recipients of blood transfusions. Incubation of the virus is 15-160 days. There is carrier state for this infection (50-80%). There is no prophylaxis for this infection and no vaccine is available.

*Hepatitis G (HGV)* HGV has a similar role to HCV.

#### Hepatitis D virus infection

Hepatitis D viruses are defective RNA viruses transmitted through percutaneous, sexual or perinatal routes. This virus infects with hepatitis B virus. Risk groups for hepatitis B virus infections include IV drug users, healthcare workers dealing with blood, hemodialysis patients, male homosexuals, heterosexuals with multiple partners and recipients of blood transfusion. Incubation of the virus is 21-140 days. There is a carrier state for this infection. It gives rise to a more severe form of hepatitis.

Prophylaxis is not available but HBV vaccine offers some immunity for susceptible persons.

#### **Clinical features of viral hepatitis**

There are no differences in clinical features between the types of viruses involved. Generally patients complain of \_flu' like symptoms in the early phase of infection. Three stages of infection are often noticed:

- 1. **Preicteric phase:** This phase is characterized by anorexia, nausea, vomiting, fatigue, myalgia, malaise and fever (<u>1-2 weeks</u> before the onset of jaundice). With Hepatitis B virus infection, 5-10% of sufferers develop arthralgia, rash and angioedema.
- 2. Icteric phase: In this phase, clinical features include the appearance of jaundice and right upper quadrant pain, with anorexia, nausea and vomiting. Hepatomegaly and splenomegaly also become palpable. This phase lasts from between <u>2 and 8</u> weeks in 20 to 50% of patients.
- **3. Posticteric phase:** In this phase, symptoms disappear but hepatomegaly persists for some time. Recovery is achieved in <u>four months</u> after the onset of jaundice.

**Diagnosis/investigations**: History, physical findings and blood tests for liver enzymes (elevated) bilirubin (raised), prothrombin time (elevated), alkaline phosphatase (elevated) and WBCs (increased) are helpful in the diagnosis of the disease. Serological investigations for viral hepatitis.

#### **Dental Management Considerations of Viral Hepatitis**

- Identification of carriers of HBV, HCV and HDV is essential.

- *Patients with active hepatitis*: Consultation immediately with their physician is essential. No dental treatment should be given unless it is urgent. No hepatotoxic drugs are to be prescribed for these patients.

- *Dentists who are hepatitis virus carriers:* Dentists should adhere strictly to professional ethics and practice guidelines, and to standard precautions in the operatory. Periodically, the dentist should test his/her HBsAg status. Until seroconversion has occurred, dental practice should be discontinued.

#### • Alcoholic liver disease (ALD)

Alcoholic liver disease (ALD) refers to liver damage and its function as a result of alcohol abuse. <u>Classification of ALD</u>

1. Fatty liver: is the mildest form of reversible liver injury.

2. Alcoholic hepatitis: features hepatocellular damage. Jaundice, fever and ascites are common at this stage.

3. Perivenular sclerosis: leads to liver cirrhosis.

4. <u>Cirrhosis:</u> displays fibrosis and nodule formation. Cirrhosis is irreversible and accompanies portal hypertension in most cases.

Alcoholic Liver Disease



**Symptoms and Signs:** Suggestive of ALD, these include oedematous puffy face, traumatic or unexplained injuries and scars, memory deficits, slurred speech, jaundice of sclera and oral mucosa, spider angiomas, ascites, white nails or transverse pale bands on nails, ankle oedema, petechiae, ecchymoses, prolonged bleeding, parotid gland enlargement and a sweet, musty breath odour.

#### **Diagnosis/Investigations:**

History and physical examination are suggestive of alcohol abuse.

**Management:** Cessation of alcohol use is the main form of management of ALD. A calorie rich diet, the use of antioxidants, and steroids in severe cases, are used to treat ALD. A liver transplant is the ultimate measure when all other modalities have failed. **Dental Management Considerations** 

o Referral or consultation with a physician to check current health status, medications, laboratory values and to discuss management issues.

o Laboratory screening for FBC, AST, ALT, bleeding time (BT), thrombin time (TT) and prothrombin time (PT)

o Avoidance of drugs metabolised by the liver is essential.

#### • Liver cirrhosis

**Definition/description:** Liver cirrhosis is a consequence of chronic liver disease characterized by replacement of liver tissue by fibrosis, scar tissue and nodules leading to loss of function.



Cirrhosis causes scaring and nodules to form throughout the liver.

#### • Liver cancer (Hepatocellular carcinoma)

**Definition/description:** A malignant tumor of the hepatocytes is hepatocellular carcinoma. This is one of the most common tumors worldwide.

**Causes:** These include chronic HBV or HCV carriage, and cirrhosis from any cause. Hemochromatosis (excess iron absorption and storage, a genetic disorder) is also known to cause liver cancer.



Management: includes resection or liver transplant, chemotherapy and opiates for pain.

## Diseases of the endocrine glands/ introduction.

### Hypothalamus/ pituitary/ thyroid/ parathyroid/ adrenals/ gonads.

#### Introduction

Endocrine and metabolic disorders are relevant to dentists because some disorders present systemic manifestations and require especial management modifications.



The hormones are produced at three levels, i.e. hypothalamus (releasing hormones), pituitary (trophic or stimulating hormones) and target organ such as thyroid, parathyroid, adrenals, gonads etc. (active hormones or prohormones) Figure 1.



#### **Common Symptoms in Endocrine Diseases**

In endocrine diseases, symptoms of over- or under-secretion of hormones are encountered. Some general symptoms include weight loss or gain, excessive hair loss or growth, skin pigmentation, fertility or menstrual problems, dwarfism, slow mental activity, disturbances in the heart rate and rhythm, gigantism, and chronic fatigue.

#### **Common Investigations in Endocrine Diseases**

Investigations into endocrine diseases include thyroid function tests, isotope scanning, fine needle cytology, plain x-rays of the appropriate anatomic region, ultrasound, CT scan, MRI, biochemical investigations such as estimation of levels of hormones, blood glucose, serum cortisol levels, urea and electrolytes, vitamin D levels, serum calcium and phosphate levels.

### **Diseases of the Parathyroid Gland**

Diseases of the parathyroid glands include primary hyperparathyroidism, secondary hyperparathyroidism, hypoparathyroidism and pseudohypoparathyroidism.



Anatomy of the Thyroid and Parathyroid Glands

#### Primary hyperparathyroidism

• **Definition/description:** High levels of parathyroid hormone (PTH) (14 to 65 pg/mL) due to glandular pathology results in primary hyperparathyroidism.

Cause: Adenoma or hyperplasia of the parathyroid gland.

Symptoms and signs: Often, patients are asymptomatic. When symptomatic, features

include polyuria, excessive thirst (due to hypercalcaemia), anorexia, weakness, constipation, vomiting, renal colic, backache, hypertension, renal stones, peptic ulceration, giant cell tumour of the bone, and pancreatitis.

**Investigations:** These include serum calcium (>10.5 mg/dL,), phosphate (raised), alkaline phosphatise (raised), bicarbonate, vitamin D, x-rays of the hands and skull (\_pepper-pot' like erosions of skull), brown tumours of the bone, abdominal x-rays for renal stones, ultrasound of the neck, radioactive thallium-technetium subtraction scan and immunoassays for PTH.

**Management:** This includes surgery for adenoma and hyperplasia of the parathyroid gland. In addition, administration of vitamin D in severe cases may be necessary.

#### • Secondary hyperparathyroidism

**Definition/Description:** Prolonged hypocalcaemia associated with renal failure and deficiency of dietary vitamin D can lead to secondary hyperparathyroidism. Stimulated PTH can also result in secondary hyperparathyroidism. Treatment includes correction of the underlying cause.

#### • Hypoparathyroidism

**Definition/description:** Hypoparathyroidism may be either primary due to autoimmune disease, or secondary as a result of thyroid surgery.

**Symptoms and signs** include peri-oral and peripheral paraesthesia, cramps, and abnormalities of the hair, nails and teeth in chronic cases. Tetany in acute cases is characterised by tingling in the extremities, spasms in the hands, facial twitching.



**Investigations** include estimation of serum calcium (low), phosphate (raised), and alkaline phosphatase (normal) levels. Skull x-ray shows basal ganglion calcifications, and plasma PTH levels are low.

**Management** includes IV administration of calcium gluconate and in acute cases, long term alfacalcidol.

#### Pseudohypoparathyroidism

Definition/description: This is an inherited disorder with resistance to PTH.

**Symptoms/signs** include a short stature, mental retardation, \_moon' face, cerebral calcifications, short 4th and 5th metacarpals and hypothyroidism.

Management: Treatment is as for primary hypoparathyroidism.

## **Diseases of the Thyroid Gland**

### • Hyperthyroidism (Thyrotoxicosis)

**Definition/description:** Also known as thyrotoxicosis, this disorder is characterised by over-production of thyroid hormone. Thyroid hormone exists in two main forms: thyroxine (T4) and triiodothyronine (T3).



**Cause:** A common cause of hyperthyroidism is an autoimmune disorder (Graves' disease), where antibodies stimulate the thyroid to secrete excess hormone. Less often, a nodule within the multinodular goitre, or a thyroid adenoma producing excessive thyroxin is responsible.

**Symptoms:** Common symptoms include sweating, heat intolerance, sleep disturbances, irritability, amenorrhoea, palpitations, weight loss, increased appetite, and anxiety.

**Signs** include tachycardia, atrial fibrillation, exophthalmos, fine tremor, goiter, and peritibial myxoedema.



**Investigations:** T3 is raised (80-220 ng/dL). T4 or (fT4) is also usually raised (5.0 to  $12.0\mu$ g/dL). Thyroid Stimulating Hormone (TSH) (0.5 to 5.0 mIU/L ) may be

suppressed in those with multinodular goitre. The presence of thyroid autoantibodies may be detected.

Management includes antithyroid drugs (Carbimazole or propylthiouracil) and surgery.

### • Hypothyroidism

**Definition/Description:** Underproduction of thyroid hormone results in hypothyroidism.

**Causes** include **an** iodine deficiency, (the most common cause worldwide) or an autoimmune disorder (Hashimoto's disease). Thyroidectomy or radiation of the gland, and occasionally hypopituitarism, may be etiologically associated.

**Symptoms** include weight gain, cold intolerance, depression, tiredness and constipation.

Signs include the slow relaxation of tendon reflexes, myxoedema (deposition of subcutaneous mucopolysaccharides), hair loss, a hoarse voice, cold skin and bradycardia.



**Investigations** include estimation of T4 (low) and TSH (high) levels. Tests for cholesterol (hypercholesterolemia) and anaemia are necessary. Thyroid autoantibodies in the autoimmune variant can be detected.

Management: Thyroxin replacement is the treatment of choice.

## **Diseases of the Pituitary Gland**

### • Hypopituitarism

**Definition/description**: A condition characterised by the deficiency of anterior or posterior pituitary hormones is called hypopituitarism.

### • Pituitary dwarfism

Hypopituitarism in children typically results in short stature with normal proportions, and slow growth due to a deficiency in the growth hormone. This condition is called **pituitary dwarfism.** 



**Causes** include anterior pituitary tumours, surgery on the pituitary for tumours, past head injury, tuberculosis, sarcoidosis, and radiation.

**Symptoms and signs** include myxoedema, infertility, amenorrhoea, depression, signs of hypoglycaemia, muscle weakness, and short stature.

**Investigations** include CT scan, MRI, glucose, T4 and TSH levels, prolactin, gonadotrophins, cortisol and testosterone levels.

**Management:** Hormone replacement therapy; recombinant growth hormone (GH) is recommended for children with a deficiency of growth hormone.

### • Acromegaly and gigantism

**Definition/description:** Acromegaly and gigantism are syndromes that result when the pituitary gland produces excess growth hormone (GH). Nearly always this is due to a pituitary adenoma. If this occurs after epiphyseal plate closure at puberty, the condition is called **acromegaly**; if before the closure of the epiphyses, the result is **gigantism**.



**Symptoms and signs in acromegaly:** Excess secretion of growth hormone usually starts between the ages of 20 and 40. Headache is common due to the pituitary tumour. Other features include coarsening of the facial features, enlargement of the extremities, a husky voice, excessive sweating and offensive body odour,

hypertension, joint symptoms of degenerative arthritis, peripheral neuropathy, impaired glucose intolerance, menstrual irregularities and heart failure. These patients are at a higher risk of developing gastrointestinal cancers.



**Symptoms and signs in gigantism:** This is a rare condition. Excess growth hormone secretion begins in childhood before the closure of epiphyses. Bone growth is faster but does not show major deformities. Soft tissue swellings are common. Peripheral nerves are enlarged. A eunuchoid habitus (partially resembling, or having the general characteristics of a eunuch) is often present.

## **Diabetes insipidus (DI)**

**Definition:** Diabetes insipidus (DI) is characterized by an inability to produce concentrated urine due to the complete or partial deficiency of antidiuretic hormone (ADH), or renal resistance to the ADH action. These are respectively known as cranial diabetes insipidus and nephrogenic diabetes insipidus.



**Causes:** Causes of cranial DI can be idiopathic, a head injury, or sarcoidosis. Nephrogenic DI may be caused by drugs, renal disease and glycosuria.

**Symptoms and signs:** These include polyuria resulting in large volumes of pale coloured urine. Frequent urination, nocturia and polydipsia and dehydration are other common symptoms.

In patients with diabetes insipidus, osseous infiltrates are often found on the skull and

jaws and can be identified on conventional dental radiographs. Loose teeth are another feature of DI. Due to excessive thirst, children with DI drink large amounts of water (fluoridated at optimal level) which can result in dental fluorosis.

**Investigations:** Estimation of urine osmolality and a 24-hour urine output is required to confirm polyuria.

**Management** includes administration of desmopressin for cranial DI, and treatment of the underlying cause for nephrogenic DI. Underlying causes should be identified and treated.

### **Diseases of the Adrenal Gland**

• Adrenocortical Excess (Cushing's syndrome); hyperadrenalism

**Definition/description:** Cushing's syndrome refers to the clinical picture resulting from circulating cortisol excess from any cause, whereas **Cushing's disease** results from hyperfunction of the adrenal cortex from pituitary Adrenocorticotropic Hormone (ACTH) excess, usually due to pituitary adenoma.

Cause: Hyperfunction of the adrenal cortex



# Symptoms/Signs:

• Adrenal insufficiency: Primary adrenal insufficiency (Addison's disease) and secondary adrenal insufficiency

**Definition/Description:** A disease of the adrenal glands causing primary adrenal insufficiency is called **Addison**"s disease.

**Cause: Primary adrenal insufficiency** involves autoimmune destruction of the glands in about 80% of cases. Other causes include TB, metastatic disease and hypoparathyroidism, diabetes mellitus and Graves' disease.

**Secondary adrenal insufficiency** results from panhypopituitarism, a lack of ACTH, and in those patients who are receiving steroids.

#### Symptoms/Signs:



Weight loss/anorexia

Pigmentation of feet

#### Treatment

Addison's disease is treated with medications that serve to replace the hormones that the body cannot produce. These drugs include hydrocortisone, which is used to replace cortisol, and mineralocorticoid, which is used to replace aldosterone.

#### **Diabetes mellitus**

**Definition/description:** Diabetes mellitus (DM) is a group of disorders characterized by persistent hyperglycaemia due to a deficiency of endogenous insulin, or resistance to insulin action.

### Two types of DM exist: type 1 and type 2 DM.

**Type 1** is insulin dependent. Usually it is found in children, who are often prone to ketosis. Approximately 5-10% of the people who have diabetes have type 1.

**Type 2** is non-insulin dependent, and usually occurs in obese older adults. About 90-95% of people with diabetes have type 2.

**Cause:** An autoimmune process resulting in  $\beta$ -cell destruction of the pancreas is the cause of type1 DM (that stops your body from making insulin). Obesity and genetic component are associated with type 2 DM (your body doesn't use insulin well and can't keep blood sugar at normal levels). Other factors associated with type 2 DM include drugs such as corticosteroid therapy and thiazides, as well as pancreatic disease, Cushing's disease, acromegaly and thyrotoxicosis.

**Symptoms and signs** include irritability, tiredness, thirst, a dry mouth, weight loss, nocturia, blurring of vision, hyperphagia (excessive hunger and eating), dehydration, ketonuria, hyperventilation, ketone breath, obesity, lethargy, increased susceptibility to infections (such as pruritus vulvae), and delayed wound healing. These are presenting symptoms of type 2 DM. Polyphagia, polydypsia (excessive thirst) and polyuria (the three \_P's) are classic symptoms of Type 1 DM.

Complications of DM: DM can damage a variety of organs.

o **Kidney Damage:** microalbuminuria due to kidney damage (glomerulosclerosis or chronic pyelonephritis).

o **Ocular damage**: background microaneurysms (dots) and haemorrhages (blots), cataracts, vitreous haemorrhages, cotton wool spots and neovascularisation are ocular complications of DM.

o Heart: chronic heart disease (CHD) is common (diabetic cardiomyopathy).

o **Circulation:** atheroma of large vessels causing intermittent claudication and stroke. Small vessel disease may cause distal gangrene.

o **Nervous system**: peripheral neuropathy is common in a glove and stocking distribution.

o **Transient diabetes** as a complication of pregnancy is known as gestational diabetes.

#### Investigations for DM include:

**1. Urine** examination by glucose strips or dipstick method. If glycosuria is detected, the patient should be tested for blood glucose levels.



**2. Random blood glucose and fasting blood glucose tests**. A random blood glucose level of >14 mmol/l and a fasting glucose level of >7 mmol/l (>120 mg/dl) are diagnostic. Typical blood glucose levels that people with diabetes aim at:

•Before eating food: 80 to 130 mg/dL

• About 2 hours after eating food: less than 180 mg/dL

An oral glucose tolerance test (OGTT) At 2 hours, a blood sugar level of 140 mg/dL or lower is considered normal, 140 to 199 mg/dL indicates you have prediabetes, and 200 mg/dL or higher indicates you have diabetes.

**3. HbA1c** (glycosylated haemoglobin) measurements, measures the amount of blood sugar (glucose) attached to hemoglobin (which provide an accurate measurement of glycaemic control over a period of three months).

Normal: HbA1c below 5.7%

Prediabetes: HbA1c between 5.7% and 6.4%

Diabetes: HbA1c of 6.5% or higher.

**4.** Tests for the evaluation of serum **cholesterol** (for hypercholesterolemia), creatinine, microalbuminuria for renal disease, **fundoscopy** for diabetic retinopathy, hypertension and peripheral neuropathy.

#### Management:

**Type 1 DM:** If ketoacidosis (occurs when body produces high levels of blood acids called ketones that results in changes in blood pH) is present, immediate administration of IV fluids and insulin is essential. In patients without ketoacidosis (with hyperglycemia and other symptoms), insulin therapy is to be given. Short acting insulin preparations (e.g., Humalog) before meals and long acting (e.g., Humulin I) before bedtime are recommended. Insulin pumps are also available. Dietary control and patient education are also important factors.

Type 2 DM: dietary control, physical exercise, reduction in weight and restricted

carbohydrate intake are essential. Treatment with metoformin is necessary for those obese patients who do not respond to dietary control. Some type 2 patients may need insulin to achieve adequate glycaemic control.



Dipeptidyl peptidase-4 (DPP-4) inhibitors are a relatively new class of oral diabetes drugs. Also known as gliptins, they are usually prescribed for people with type 2 diabetes who have not responded well to drugs such as metformin and sulphonylureas. Medicines in the DPP-4 inhibitor class include sitagliptin, saxagliptin, linagliptin, vildagliptin and alogliptin

