## CARDIOVASCULAR DISORDERS

(G. GITARI)

### MAIN OBJECTIVE

To promote Health

**Prevent Illness** 

**Diagnose and** 

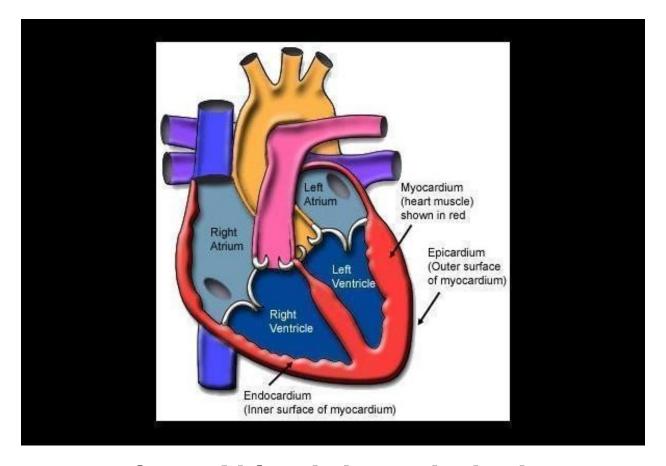
Manage patients suffering from common cardiovascular disorders

#### **COURSE OUTLINE (CONDITIONS)**

- Congestive cardiac failure (CCF)
- > Arteriosclerosis
- > Atherosclerosis
- Myocardial Infarction (MI)
- > Rheumatic Heart Disease (RHD)
- > Subacute Bacterial Endocarditis (SBE)
- > Acute Bacterial Myocarditis
- > Hypertension
- > Carotid Artery Disease
- > Varicose Veins
- > Transient Ischaemic Attacks (TIAs)

#### INTRODUCTION

#### ➤ Revision - A & P



o Flow of blood through the hearto Coronary arteries

#### **HEART DISEASE**

Heart diseases are a major cause of death but not all kinds are fatal

There are many causes as covered under various conditions

#### **DIAGNOSTIC TESTS**

✓ Electrocardiogram (ECG)

- ✓ X-rays
- √ Heart catheterization
  - o To determine blood pressures within the heart
  - o To take blood samples for determining oxygen content in various parts of the heart and the great vessels
- ✓ Angiogram
  - X-rays using dye to visualize the heart and blood vessels
- ✓ Radio isotopes (chemical elements) which
  - o Emit radiation, given IV and picked by camera
  - Aid in visualizing structural defects of the heart and the great vessels
- ✓ Echocardiography
  - To check structural defects of the heart and vessels using sound
- ✓ Phonocardiography
  - o For validating heart sounds

#### **CONGESTIVE CARDIAC FAILURE (CCF)**

## **Pathophysiology**

- Heart failure occurs when the amount of blood pumped by the heart is insufficient for the needs of the body
- The right ventricle fails to function properly, the right atrium becomes distended leading to stasis / congestion in the venous system
- ❖ The pressure in the superior and inferior cava rises; the veins in the neck are distended; the liver becomes engorged and the legs become oedematous.

#### **SPECIFIC SYMPTOMS**

- **▶ Left Sided Failure** 
  - Produces congestion in the lungs and pulmonary oedema
  - o Cardinal sign Dyspnoea
- **▶ Right Sided Failure**

- o Produces oedema of the lower part of the body and impaired circulation to the brain
- o Cardical sign Oedema
- > Pulmonary Oedema
  - o Accumulation of fluid within the lung tissue due to left sided failure (cardiac asthma) or an abrupt increase in the workload of the heart.
    - Symptoms
      - Severe dyspnoea
      - Coughing up blood-stained, frothy sputum
    - Acute pulmonary oedema can be fatal within a short period of time. It presents with
      - Sudden breathlessness / sense of suffocation
      - Cyanosis
      - Cold hands / grey appearance (colour of death)

- Distended neck (jugular)veins
- Tachycardia
- Incessant coughing
- Increasing mucoid sputum
- Noisy respiration as frothy, bloody mucus pours into bronchi and trachea

### **General Signs / Symptoms**

- > Tachycardia
- > Palpitations
- >Arrhythmia (irregular pulse)
- **>** Dyspnoea
- ➤ Distended abdomen with ascites and discomfort
- **≻**Cyanosis
- > Distended jugular veins
- ➤ Pitting oedema of feet and sacral area
- **≻Chest pain**
- ➤ Coughing, sometimes with bloody sputum (haemoptysis)
- ➤ Nausea and vomiting

### **≻Oliguria**

#### **GENERAL MANAGEMENT**

- AIM: To relieve symptoms and reduce venous return to the heart
- ✓ Positioning of the patient and oxygen administration
  - o To relieve dyspnoea and cyanosis
- √ Sedation
  - o to keep patient calm
- ✓ Rest
  - o physical, mental and emotional
- **✓ Diet:** 
  - Small, easily digested meals,
     limited intake of sodium and fluids
     depending on type of failure
- ✓ Care of the skin and turning
  - to avoid breakdown during immobility, especially for those with oedema
- ✓ Medication

- To decrease heart rate and improve strength of contractions of the heart muscle
  - Digoxin (digitalis)
- To relieve oedema and aid elimination of fluid causing pulmonary failure
  - Diuretics e.g. Frusemide (Lasix)
- o To relieve pain
  - Analgesics e.g. Morphine or other
- ✓ Rehabilitation
  - So that the patient can lead a useful, active life within the limitations imposed by the illness

#### **PREVENTION**

- ✓ Adequate exercise
- ✓ Avoidance of
  - o Obesity
  - o Tobacco smoking and
  - o Stresses that overwork the heart

✓ Early treatment of throat infections to prevent, and monitoring of children for early detection of heart disorders (e.g. RHD)

#### HOSPITAL MANAGEMENT

#### On admission

- ✓ Complete bed rest in upright position
- ✓ Oxygen administration if dyspnoeic or cyanosed
- ✓ Total nursing care
- ✓ Vital signs (TPR, BP) hourly while dyspnoeic, then gradually to four hourly
- ✓ Medication:
  - o Digitalis (Digoxin)
    - 0.125 mg 0.5 mg or as ordered
    - To slow the heart beat (pulse)
  - o Diuretics
    - Frusemide (Lasix)
      - 80 mg stat, then 20 40 mg / day

- Ethacrynic acid a rapidly acting loop diuretic IV (for pulmonary oedema)
  - 50 mg 100 mg stat
  - 25 mg / day /PO or
  - 0.5mg -1 mg / kg IV slowly (single dose - not to exceed 100 mg)
- To increase urinary output and reduce oedema
- o Analgesics for pain
  - Morphine 15 30 mg stat or as prescribed
- o Potassium chloride (Slow K 600 mg tabs) 1 2 tabs to replace loss
  - Ripe bananas can also be used
- √ Fluid input and output chart
- ✓ Daily weighing to monitor weight reduction after diuretics
- ✓ Diet: light, low salt, easily digestible
- ✓ Aperient during fluid restriction to stimulate bowel movement

- ✓ Turning and prevention of pressure to prevent bedsores because of oedema
- ✓ Follow up after discharge

#### **Home Care**

- ✓ Prolonged rest and light duty
- ✓ Light, low salt, high potassium diet (for right sided failure
- ✓ Continuation with drugs
- ✓ For women (child bearing age), health message on significance of cardiac disease in pregnancy
- ✓ Follow up at the cardiac / medical clinic

#### **DISEASES OF THE ARTERIES**

## ATHEROSCLEROSIS AND ARTERIOSCLEROSIS

#### **ATHEROSCLEROSIS**

A disease of the arteries in which fatty plaques develop on their inner walls, eventually obstructing blood

## flow and interfering with absorption of nutrients

#### **ARTERIOSCLEROSIS**

- Means hardening of the arteries with loss of elasticity within the medial layer of the walls of small arteries, causing impaired blood supply to the organs and severe elevation of blood pressure (i.e. hypertension)
- It is associated with atheroma and old age
- \*\* Although the processes of atherosclerosis and arteriosclerosis differ, rarely does one occur without the other \*\*

# ATHEROSCLEROSIS Pathophysiology

It is a disease of the arteries in which fatty plaques develop in the internal layer of the medium and

- large arteries e.g. the coronary arteries, eventually obstructing them
- The deposits interfere with absorption of nutrients by the endothelial cells that compose the lining of and protrude into the lumen, obstructing blood flow
- There is accumulation of smooth muscle cells, fat plaques (atheroma) with lipids, fatty acids, cholesterol, calcium crystals, carbohydrates, blood components and fibrous tissue
- Secondary changes can occur as a result of arterial dilatation and aneurysm formation

#### **TYPES**

- 1. Fatty Streak
  - a. A yellowish, smooth lesion which does not obstruct blood flow
  - b. Histology
    - i. Presence of cholesterol, fat deposits within the smooth

muscle cells and macrophages in the internal layer of the artery causes them to balloon out and are referred to as "foam cells"

- ii. Age incidence infancy to old age
- 2. Raised Fibrous Plaque
  - a. Is the characteristic lesion of atherosclerosis
  - b. It is a localized, yellowish-grey, elevated lump on the inner surface of the artery. It protrudes into the arterial lumen and obstructs blood flow
  - c. Histology
    - i. Involves:
      - Proliferation
         (multiplication) of smooth muscle cells and macrophages
      - Intracellular and extracellular fat deposits and

- Accumulation of connective tissue (collagen, elastic fibres)
- ii. Defective cells consist of
  - A fibrous cap / covering (of collagen, etc.)
  - 2. A central part with a mass of lipid material (cholesterol), cellular waste and plasma proteins
- iii. At autopsy, the centre of large plaques has a thick, yellow liquid atheroma (Greek word for gruel/uji)
- 3. Complicated Lesion
  - a. This is a fibrous cap with any of the following
    - i. Calcification
    - ii. Rupture of the plaque
    - iii. Haemorrhage into the plaque
    - iv. Thrombus formation
  - It is associated with signs and symptoms caused by complete occlusion of blood flow to an

- organ, especially in people with advanced disease
- c. The middle layer also undergoes pressure atrophy (wasting) and loss of elastic tissue, causing sufficient weakness leading to arterial dilatation (aneurysm). If untreated, it can result in fatal rupture and haemorrhage
- d. In smaller arteries, especially in the heart and the brain, the narrowing by plaques accompanied by thrombosis will lead to occlusion, causing myocardial infarction (MI) or cerebrovascular accident (CVA)

# **EFFECTS OF ATHEROSCLEROTIC PLAQUES**

- ➤ Narrowing the arterial lumen to cause ischaemia
- ➤ Sudden occluding of the lumen by thrombosis, causing infarction

- ▶ Providing a site for thrombosis then embolism
- > Forming an aneurysm, then rupturing

#### The effects occur most frequently in

- >The heart
- >The brain
- >The kidneys
- > Lower extremities and
- > Small intestine

#### **MANAGEMENT**

- ✓ Bed rest
- ✓ Provide warmth to cold extremities (if partial obstruction)
- ✓ Elevate the feet
- ✓ Moderate regular exercises of the limbs
- √ Skin care
- ✓ Low animal fat diet
- ✓ Medication
  - o Vasodilators
    - Pitressin tannate 1 ml (5 units)SC /IM

- Pitressin 1 ml (20 units) SC /IM
- o Antilipemics to decrease serum lipids (have also a vasodilating effect)
  - Nicotinic acid 1 2 g / X 3 / day with meals
- o Digoxin (digitalis) 0.125 mg 0.5 mg
  - Check pulse before administration
- ✓ Surgical intervention if indicated
  - o Amputation in case of gangrene due to thrombosis
  - o Vein grafting
  - o Endarterectomy surgical removal of atheromas
  - o Pre and Post operative care

## On Discharge

- ✓ Health education on
  - o Aeteology
  - o Prevention
  - o Signs / symptoms
  - o Follow up

## ARTERIOSCLEROSIS (THROMBOANGIITIS) OBLITERANS

### (BUERGER'S DISEASE)

- This is partial or complete obstruction of the arteries, especially within the extremities, by atheromatous plaques which reduce the diameter of the vessel
- The plaques cause irregular surface of the vessel wall, predisposing thrombus formation which further reduces the diameter
- The obstruction impedes blood flow to the extremities leading to o Ischaemia (if partial) or o Infarction (if complete)

## Age / Gender

- >45 70 years,
- **➤ More frequently in men than women**

## **Predisposing Factors**

➤ Cigarette smoking and tobacco chewing

- > High fat diets
- **>** Sedentary lifestyle
- > Hypertension
- > Diabetes mellitus

### **Causes / Aetiology**

- > Errors in fat metabolism
- ➤ Coagulation defects
- > Heredity
- > Reduced blood supply to arterial wall
- ➤ Stress related catecholamine (norepinephrine - a neurohormone) which slows the heart rate
- ➤ Chemicals Tar, nicotine (constricts peripheral vessels and increases force of flow), and carbon monoxide from cigarettes
- **≻Trauma**
- > Viral infections

## **Pathophysiology**

An atherosclerotic plaque forms, causing thickening of the intima, degenerative changes in the middle layer of the artery, resulting in platelet aggregation leading to thrombus formation at the site

## **Commonly Affected Arterial Sites**

- > Superficial femeral
- > Proximal popliteal
- > Aortoiliac
- **≻Tibial**
- > Peroneal

## **Compensatory Mechanisms**

A network of collateral circulation may develop, allowing the vessels below the obstruction to fill

#### **Clinical Manifestations**

- These result from decreased oxygen supply to the peripheral tissues
- > Intermittent Claudication
  - o Limping caused by interference with blood supply to the legs
  - o The patient complains of cramplike pain after walking a certain

# distance, which disappears if he rests at that point

- o The cramping pain results from the build-up of metabolic wastes within the muscle tissue.
  - An exercising muscle demands more oxygen, but because arterial flow is decreased, less oxygenated blood reaches the tissues.
  - Waste products of anaerobic metabolism build up in the muscle.
  - There is local irritation by these toxic wastes, causing the ischaemic pain.
  - When the patient rests, the metabolites are washed away and
  - The pain goes away
- Mechanism of Intermittent Claudication
  - Activity / Exercise
  - Increased tissue oxygen demand

- Impaired O<sub>2</sub> supply secondary to obstruction
- Shift to anaerobic metabolism
- Build-up of metabolic wastes (e.g. Lactic acid)
- Local irritation of peripheral nerve endings
- Pain in extremity
- Rest
- Decreased tissue 0<sub>2</sub> demand
- Return to anaerobic metabolism
- Elimination of metabolic wastes
- Relief of pain

## > Resting Pain

- o The pain is described as
  - Burning
  - Numbness
  - Tingling of toes
- o It may be as a result of ischaemic neuritis

- o It occurs at night and interrupts sleep
- o It is alleviated by standing or walking
- ➤ Pallor and redness (mottling) of extremities
- ➤ Gangrene if occlusion has been present for several days
  - o Pre-gangrenous state
    - Tissue is deep purple and not affected by change of position or pressure
  - o Gangrenous state
    - Area looks black, dried and hard
  - o Patient may not be aware as sensation is absent
- **>**Skin changes

- o Cool and dry
- o Smooth, thin and shiny
- o Reduced or absent hair growth
- o Nail thickening
- > Uneven temperature
  - Patient may complain of a "cold foot" which does not change when exposed to warmth
- > Unequal muscle bulk and tone
- ➤ Peripheral pulses are diminished or absent

## **Diagnosis**

- ✓ Segmental systolic pressures are taken using pneumatic (air) cuffs in each arm, thigh, calf and ankle
- ✓A difference of 20 mmHg or more between segments indicates occlusion

#### **MANAGEMENT**

## **Treatment is palliative**

**Aim:** To reduce risk factors to halt progression of the disease

- √ Stopping smoking
- ✓ Exercises especially walking
- ✓ Low fat diet
- ✓ Drugs / Medication
  - o Pentoxyfylline (Trental)
    - Decreases blood viscosity and alleviates ischaemic symptoms
    - Oral dose: 300 mg 600 mg daily
    - IV dose: 100 mg 150 mg / hr
      - (Maximum 1.2 g / 24 hrs)
  - o Nifedipine (Adalat)
    - Peripheral vasodilator

Dose: 10 mg - 20 mg twice / thrice per day

## o Antilipemics

- To slow atherosclerotic process by decreasing serum lipids
- Nicotinic acid (Niacin, Nicotinamide)
  - Oral: 1.0 g 2.0 g X 3 / day
     with meals

## o Antiplatelets

- To decrease risk of platelet aggregation and thrombus formation e.g.
- Aspirin 80 mg -325 mg daily

## ✓ Safety and hygiene:

- To prevent injury and infection of extremities by
  - Treatment of infections

- Surgical cleaning of wounds
- Post operative prophylactic antibiotics
- ✓ Surgery
  - o Peripheral arterial bypass surgery
    - A synthetic graft (Dacron) is anastomosed to a patent arterial source both proximal and distal to the lesion
    - Complications
      - Infection may lead to death
      - Graft failure
      - Amputation
- √ Thrombolysis (Dissolution of clots)
  - Anticoagulants (fibrinolytic agents) in severe occlusion and high risk surgery e.g.

- o Heparin, Warfarin
- ✓ Percutaneous Transluminal Angioplasty (PTA)
  - A small catheter with an inflatable balloon is passed through the vessel to compress the lesion and widen the lumen, followed by
     Anticoagulant therapy

## **Nursing Management**

**Aim:** To minimize complications

- √ Evaluate and modify activity
- ✓ Medication administration
- ✓ Support lifestyle changes to reduce risk factors
- ✓In surgery, maintain and monitor peripheral circulation by observing o Colour

- o Temperature
- o Muscle size and symmetry
- o Skin for
  - Ulceration
  - Slow healing
  - Necrosis
- o Intermittent claudication
- o Resting pain

## **On Discharge**

> Follow up in cardiac clinic

## **Prognosis**

- Depends on
  - o Severity and
  - o Association with another underlying disease

**CORONARY HEART DISEASE (CHD) Definition** 

CHD is myocardial impairment due to an imbalance between coronary blood flow and myocardial oxygen requirements caused by changes in the coronary circulation.

#### **Manifestations**

- Myocardial ischaemia (Angina Pectoris)
- Myocardial Infarction (M I myocardial cell death)

#### **Forms**

- 1. Acute (cardiac arrest)
- 2. Chronic
- 3. Silent M I (when people just die)

#### Causes

- > Organic disease
  - o Associated with changes in structure e.g.
    - Atherosclerosis
    - Thrombosis
- > Functional changes

- o No physical cause can be found e.g.
  - Coronary artery spasm

#### CHD also includes

- 4. Arteriosclerosis
- 5. Atherosclerosis
- 6. Pulmonary embolism

It causes occlusion of blood flow, either partial, leading to ischaemia, or complete, leading to infarction

Ischaemia is reversible, but if myocardial blood flow is not increased or myocardial oxygen demands reduced, ischaemia progresses to myocardial infarction.

## ANGINA PECTORIS Definition

A clinical syndrome produced by a reduction in the blood supply to the heart muscle, caused by a narrowing or partial occlusion of the coronary arteries

## **Pathophysiology**

- When a muscle has to work with deficient oxygen, a severe cramping pain occurs (see Claudication)
- This pain, due to oxygen deficiency, is common to all muscles

## Signs / Symptoms

- ➤ Severe gripping pain behind the sternum, which radiates down the left arm, right arm, neck and sometimes upper abdomen
  - o It may be triggered by bending or climbing stairs especially in cold weather
- >The patient stops, and after about a minute or so, the pain goes away
- **▶**Tightness in the chest
- > Profuse perspiration
- **▶** Pallor
- > Flushing of the face
- **>** Dyspnoea

#### Causes

- ➤ Arteriosclerosis of coronary arteries which become too narrowed to provide adequate blood supply to the heart muscle
- ➤ High blood pressure leading to enlargement of the heart and greater oxygen requirements
- > Aortic aneurysms or stenosis
- > Severe anaemia (with ischaemia)

#### **MANAGEMENT**

- ✓ Avoidance of
  - o Sudden physical exertion
  - o Emotional upsets
  - o Exposure to cold
- **✓ Drugs** 
  - o Vasodilators
    - Glyceryl nitrate (Trinitrin)
    - 0.5 mg (tablet) chewed or dissolved under the tongue
  - Beta-blockers to block the constricting effects of adrenaline and slow the heart rate e.g.

- Propranolol (Inderal): 10 30 mg 3 - 4 times / day before meals and at bedtime
- o Sedatives: to relieve anxiety
  - Diazepam: 4 30 mg (or as prescribed)

#### ✓ Diet:

- o low calorie for the obese to reduce weight and strain on the heart
- o Light meals as heavy meals increase workload on the heart
- ✓ Treatment for hypertension

#### **MYOCARDIAL INFARCTION (M I)**

## (HEART ATTACK) Definition

- Myocardial infarction is an area of necrotic tissue in the myocardium resulting from obstructed blood supply from the coronary artery to the area
- M I s can occur in any chamber and in the layers of the heart, but

# most occur in the left ventricle because its oxygen need is very great

#### **Clinical Features**

- ➤ Either severe prolonged chest pain or burning sensation in the chest or
- ➤ Crushing pain of sudden onset (15 30 minutes) with cardiac arrest
- **≻**Pallor
- > Diaphoresis
- > Anxiety
- ➤ Nausea and vomiting / indigestion
- ➤ Syncope (sudden fainting from loss of strength)
- **>** Signs of shock
  - o Tachycardia
  - o Hypotension
  - o Cold, clammy skin
- > Palpitations
- > Arrhythmia
- **>** Dyspnoea

#### **Diagnosis**

√ History of

- o Severe, sudden chest pain or
- o Persistent pain
- ✓ Electrocardiogram (ECG)
- ✓ Serum enzyme (transaminases) changes indicating muscle necrosis

#### MANAGEMENT

## First Aid (Pre-Hospital care)

- ✓ Relief of pain
- ✓ Restoration of myocardial blood flow through resuscitation to reduce infarct size
- ✓ Oxygen administration to relieve dyspnoea
- ✓ Prevention of shock and complications
- \*\* About half of deaths occur outside the hospital, THEREFORE,

Any patient experiencing worsening effort or spontaneous angina or angina for the first time is provisionally diagnosed as acute MI until proven otherwise, and should

# receive medical attention as soon as possible \*\*

## **In Hospital**

### On admission

- √ Brief history is taken
- ✓ Complete bed rest
- ✓ Oxygen administration for hypoxia 2 -4 litres / min of pure oxygen by mask for 2 - 3 days
- √ Total nursing care
- ✓ Cardiac monitoring ECG
- ✓ Vital signs and apex beat
- ✓ Intravenous line is established for emergency drug administration
- **✓ Drugs** 
  - o Pain relief
    - Morphine (is the drug of choice) 10 mg - 15 mg prn
      - Relieves pain, anxiety and restlessness
      - Reduces activity of the autonomic nervous system,

- hence reducing myocardial oxygen consumption
- Dilates arteries and veins
- Reduces work of breathing
- Slows the heart rate
- Diamorphine
  - 5 10 mg SC or lesser dose
     IV
- o Vasodilator: to reduce artery spasm
  - Nitroglycerin: 0.5 mg in tablet form, chewed or dissolved under the tongue
  - Isosorbide dinitrate (Isordil):2.5 mg 30 mg PO qid chewed
- o Anticoagulants: to stop or slow progression of thrombi and prevent new ones forming and to prevent pulmonary embolism
  - Heparin: 5000 units SC 8 12 hourly until 2 - 3 days before discharge. Higher doses may b given to those with high risk of embolism

- Aspirin: 80 mg 325 mg daily for those without history of PUD
- o Prevention of Arrhythmias
  - IV Lidocaine: 1 mg / kg stat, then
  - 2 mg / min as prescribed, then
  - Continuous infusion 2 4 mg in 24 hours
- o Thrombolysis: to limit infarct size
  - Streptokinase IV will lyse all clots in the body
    - 10,000 30, 000 units initially, then
    - 3,000 4,000 units for 2 hours until the clot lyses
    - Infusion is continued for 15
       60 minutes after lysis to maintain patency
    - Total dose : 150,000 500,000 units
- o Heparin is then introduced later, followe by
- o Warfarin orally

- ✓ Ambulation progression is ordered by the physician
- ✓ Observe for and prevent injury in case of bleeding

## **Prognosis**

- ▶ 60 % of deaths occur within the first hour
- ➤ In hospital, most deaths are to heart failure and shock within 3 - 4 days after the onset of MI

## **Complications of MI**

- > Arrhythmias
- ➤ Heart failure depending on the size of the infarct
  - o The dead heart muscle is replaced by a fibrous scar.
  - o If it is too large, the force of the heartbeat may be adversely affected and the heart will fail
- > Pulmonary embolism
- > Structural heart problems

## **On Discharge**

- ✓ Rehabilitation depending on effect of MI
- ✓ To continue with medication if any
- ✓ Advice on
  - o Exercise
  - o Prevention

### **PULMONARY HEART DISEASE**

- The right ventricle has to propel blood through the pulmonary circulation.
- When this circulation is impeded, a strain is imposed on the work of the heart, affecting the lungs

### **PULMONARY EMBOLISM**

- ➤ Occurs mostly due to a detached blood clot (thrombus) from thrombosis of a deep vein (DVT) in the leg or the pelvis, travels in the veins and lodges in the pulmonary artery or its branches
- ➤ Blood flow to the lungs and oxygenation is reduced or stopped

- ➤ A massive pulmonary embolus causes infarction of the lung or parts of the lung depending on the site causing
  - o Sudden tightness in the chest
  - o Breathlessness
  - o Collapse or
  - o Death
- ➤ Smaller emboli might cause very little symptoms immediately but later are associated with
  - o Increasing breathlessness
  - o Haemoptysis

### **Prevention**

- ✓ Minimize risk of venous thrombosis by
  - o Early movement of legs postoperatively
  - o Early exercises in the elderly
- ✓ Anticoagulant therapy if there is evidence of thrombosis
  - o Heparin
  - o Phenindione (Dindevan)

- Initial dose: 100mg 150 mg x2 on day Day One
- Then dose adjusted to prothrombin time

### **MANAGEMENT**

- ✓ Emergency surgery may have to be done (if there is time)
- ✓ Oxygen to relieve dyspnoea
- ✓ Sedatives for pain
  - o Pethidine: 50 mg 100 mg
  - o Morphine: 10 mg 30 mg
- √ Total nursing care

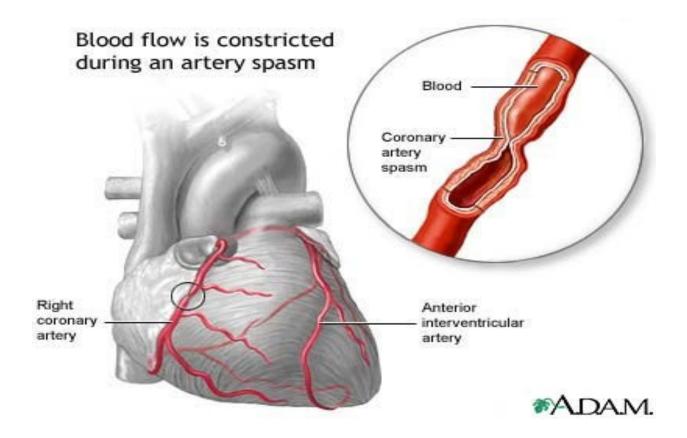
### **CORE PULMONALE**

- This is a form of heart failure occurring in patients with lung disease (COPD) e.g.
  - o Chronic bronchitis
  - o Emphysema
- The lungs cannot supply enough oxygen
  - o This affects the heart
  - o The heart may fail

- Heart failure makes worse an already impaired pulmonary function
- The patient becomes very cyanosed
- Oedema and ascites are common
- Diuretics may be given but relief will depend on the state of the lungs

### **CORONARY ARTERY SPASM**

❖ The smooth muscle fibres in the coronary arterial walls contract and temporarily narrow the vessel lumen (see functional cause of CHD), resulting in myocardial ischaemia, and may contribute to the development and size of myocardial infarction (MI) (see Nitroglycerin in MI)



### **Causes**

- **>** Smoking
- ➤ High blood cholesterol
- > Hypertension

## **Predisposing Factors / Triggers**

- > Alcohol withdrawal
- > Emotional stress
- > Vasoconstrictors
- ➤ Stimulant drugs e.g. amphetamines, cocaine

## **Cause of Coronary Heart Disease**

### Unknown

## **Predisposing and Risk Factors**

- **≻**Major
  - o Cigarette smoking
  - o Hypertension
  - o High fat diets
  - o Emotional stress
- ➤ Unavoidable Risk Factors
  - o Age:
    - In men increasing age
    - In women menopause
  - o Gender
  - o Family history of CHD
  - o Ethnic background
- > Avoidable Risk Factors
  - NB: Risk factors of CHD can be modified by controlling avoidable risk factors
  - o Cigarette smoking
  - o Physical inactivity (sedentary lifestyle)

- o Obesity
- o Emotional stress
  - Increases need for oxygen, amounts of food eaten, cigarette and alcohol use
- o Oestrogen / Progestin therapy
  - Risk increases with age and smoking
- o Alcohol
  - Raises heart rate and increases myocardial oxygen consumption

### **▶ Risk Factor Combination**

o The greater the number of risk factors combined, the greater is the risk of developing coronary heart disease (CHD)

### RHEUMATIC HEART DISEASE

### RHEUMATIC FEVER

RF is a connective tissue hypersensitivity disorder resulting from a reaction to one or more antigenic metabolic products of

- o Group A beta-haemolytic streptococcus
- o Rheumatic chorea (St Vitus`s dance) or childhood
- o Haemolytic streptococcus infection following diphtheria
- o Immune responses against
  Hepatitis B streptococcus pyogens
  or malaria

## **Immunologic Injury (Response)**

- The body has many powerful immunologic defences against infection. The result of activating these defence mechanisms can be
  - o Inflammation
  - o Cell infiltration (increase in WBCs)
  - o Tissue destruction or
  - o Death,
  - e.g. Rheumatic fever with Group A streptococcus infections

- Antibodies produced against these antigens form complexes with the streptococci and initiate an inflammatory process with the heart muscle and valves
- Infections by other microorganisms can also lead to the formation of antigen - antibody complexes and cause further disease e.g.
  - o Basement membranes of the renal glomerulus and blood vessels
  - o The cascade and inflammation are activated at these sites, resulting in immunologic damage to body tissue e.g.
    - Acute glomerulo-nephritis (AGN) and
    - Vasculitis

### **Cause of Rheumatic Fever**

➤ Rheumatic fever usually follows a streptococcal sore throat, gives no immunity and recurrences may occur

## **Susceptibles**

➤ Young children, and rarely after the age of 25 years

### **Effect**

- The inflammatory response to the infection can permanently damage
  - o The Valves of the heart -Endocarditis
  - o The Myocardium Myocarditis
  - o The Pericardium Pericarditis
- During the acute phase, the joints are swollen but temporarily
  - o \*\* It is said that "Rheumatic fever licks the joints but bites the heart"\*\*

## Signs / Symptoms

- >H/O a sore throat 7 21 days earlier
- > General malaise
- > Fever
- > Diaphoresis
- ➤ Characteristic pain in joints (is diagnostic)

- o Flitting pain on the affected joints and
- o Different joints affected at different times
- ➤ Swelling and tenderness of the affected joints but they never suppurate

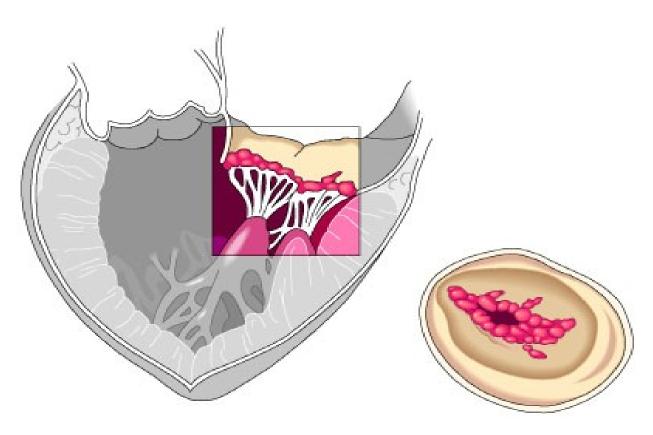
EFFECTS OF RHEUMATIC FEVER ON THE HEART

(RHEUMATIC HEART DISEASE - RHD)

### RHEUMATIC ENDOCARDITIS

- Is inflammation of the endocardium which affects the valves, and is the commonest in young and middle aged people
- Endocarditis results in lesions called vegetations on the valves. They are small clots (thrombi) which look like a row of beads on the valves composed of
  - o Fibrin
  - o Red cells and

### o Platelets



- Fibrosis and thickening make the tendonous chords (chordae tendinae)
- The vegetations rarely break off to become emboli
- The valves become swollen and distorted due to inflammation, leading to alterations in heart sounds and a blowing murmur on auscultation

### RHEUMATIC MYOCARDITIS

In the acute phase, the myocardium is affected, leading to acute myocarditis

NB Death in acute rheumatic heart disease is usually due to failure of the heart muscle

An Unusually rapid and irregular pulse is the most important sign of acute myocarditis.

### RHEUMATIC PERICARDITIS

- Inflammation of the pericardium occurs in the more severe cases of acute rheumatic fever
- It may be
  - o Dry with a friction rub at the left sternal edge in the 4<sup>th</sup> intercostals space where the pericardium comes in contact with the left chest wall or
  - o Wet with pericardial effusion.
    - If too much, fluid will press on the heart (i.e. tamponade)

- Other causes of tamponade include
  - Heart surgery
  - Tumours in the heart
  - M I
  - Injury
  - Lung cancer

### Forms Acute

**Chronic** 

### **ACUTE RHEUMATIC HEART DISEASE**

- Usually caused by rheumatic fever
- May also occur in Rheumatic chorea (a condition in which there are involuntary, rapid, brief, jerky movements occurring at an irregular rate in the extremities, and sometimes a grimace)

## Signs of heart involvement

- ➤ Very rapid, irregular pulse one the few signs of heart damage
- > Heart murmurs

- ➤ Fibrous, tender and painful nodules around the joints and tendons, usually behind the elbows, on the back of the scalp or ankles. They denote a severe attack affecting the heart
- ➤ Raised erythrocyte sedimentation rate (ESR)

## **Management**

- ✓ Complete bed rest.
  - o Gently persuade children to stay in bed
- ✓ Total nursing care in the acute stage
- ✓ Minimize boredom
  - Encourage parents and family to visit but explain to them the need for rest
- √ Relief of pain and swelling by
  - o Wrapping of joints with warm cotton wool
- ✓ Keep off the weight of bed clothes from joints with a bed cradle
- **√** Diet

- o Light until acute symptoms subside
- ✓ Drug: There is no specific cure yet o Relief of pain
  - Calcium aspirin: 1 g 4 hourly with food or milk
    - Observe for side effects tinnitus, deafness, nausea / vomiting
    - They subside when the drug is withdrawn
    - Protect from injury due to bleeding
  - Brufen
  - Steroids for inflammation relieve and reduce pain but may not prevent damage to the valves
    - Hydrocortisone: 20 mg 2/3 times / day
    - Prednisone: 4 30 mg OD
  - o Antibiotics for streptococcal infection depending on weight
    - Primary Prophylaxis (Treatment of acute infection)

- Oral Penicillin V: 125mg 250 mg tds X 10 days or
- Amoxil or
- IM Benzathine Penicillin:
  - o Single dose: 0.6 MU to 1.2 MU
- If allergic to Penicillin
  - o Sulphadiazine: 250 mg -500 mg tds X 10 days or
  - o Erythromycin: 125 mg 250 mg tds X 10 days

### o Convalescence

 When the pulse rate, temperature and ESR return to normal, the patient is allowed to sit out of bed

## o After Discharge

- Follow up in cardiac clinic for medication
- Secondary Prophylaxis (To prevent further episodes
  - Oral Penicillin V bd or
  - IM Benzathine Penicillin every 4 weeks for at least 5

# years or till the age of 25 years, whichever is longer

\*\* Patient should be reviewed every 6 months \*\*

## CHRONIC RHEUMATIC VALVULAR DISEASE

- Follows acute stage
- May be many years before the effects are noticed
- Chronic inflammation causes thickening, distortion and loss of elasticity and function of the valves

### **Two Main Effects**

- ➤ The valves adhere together, causing narrowing (stenosis) of the valve opening, especially the Mitral Valve (i.e. Mitral Stenosis) and obstruction of blood flow
- ➤ Loss of elasticity and distortion, causing the valve not to close properly (i.e. Valve Incompetence)

## resulting in leakage and regurgitation of blood

# All heart valves may be affected, but the most commonly damaged are

- >The Mitral Valve leading to
  - o Mitral stenosis or
  - o Mitral incompetence
- > The Aortic Valve leading to
  - o Aortic stenosis or
  - Aortic incompetence (characterized by Water-hammer pulse - quick sharp strokes that suddenly collapse)
- >\*\* These may cause few symptoms until the heart fails \*\*

## Signs / Symptoms

- ➤ Characteristic heart murmurs heard with a stethoscope on a routine medical examination
- ➤ Enlargement of the heart (hypertrophy) and dilatation which

take place to overcome the strain on the heart

- > Severe breathlessness
- >\*\* Symptoms may be mild with a long life or severe leading to heart failure and death within a few years

### **MANAGEMENT**

- ✓ The patient is advised to avoid any undue exertion which might put a strain on the heart, precipitating heart failure
- √ Heart Surgery (OHS)
  - o Mitral Valvotomy
    - The valve is cut and dilated to relieve obstruction and engorgement in the lungs, to improve breathing and exercise tolerance
    - o Mitral Valvectomy
      - A severely damaged and shrunken mitral valve is cut off and replaced with an artificial valve

## **After Surgery**

- ✓ Prophylactic anticoagulants to prevent clot formation
- ✓ Prophylactic antibiotics before dental treatment and surgery to prevent infections

## **Complications**

- > Heart failure
- > Embolism leading to
  - o CVA or
  - o Pulmonary embolism

## SUBACUTE BACTERIAL ENDOCARDITIS Cause

- > Streptococcus viridians
  - o Never attacks normal valves but only those already diseased e.g.
    - Rheumatic endocarditis
    - Congenital heart disease
  - o It is commonly found in mouth and teeth, therefore bacterial endocarditis occurs after dental extraction or treatment which

# releases bacteria into the blood stream(bacteraemia)

## **Pathology**

- The organisms settle on the valves and cause larger vegetations than those in rheumatic endocarditis
- The vegetations dislodge easily, causing emboli in the blood stream

## Signs / Symptoms

- ▶ Prolonged pyrexia of unknown origin (PUO)
- ➤ Signs of septicaemia o Fever with rigors and sweating
- > General malaise
- ➤ Signs of embolism depending on organ affected
  - o Brain
    - Cerebral vascular accident (CVA)
  - o Kidneys
    - Pain in the loins
    - Haematuria

### o Skin

- Haemorrhages into the skin (petechiae and purpura) and nails
- ➤ Heart murmurs due to the changes in the valves

## **Diagnosis**

## **Apart from PUO**

- ➤ A history of recent dental treatment or RHD
- > Heart murmur
- > Blood culture for bacteria

### **TREATMENT**

- ✓ Penicillin is the drug of choice in large doses
  - o 12 MU / day in divided doses for at least 6 (six) weeks
- ✓ Other antibiotics may be used if bacteria is not sensitive to penicillin

## **Nursing Care**

✓ As for other heart conditions

### **Prevention**

- ✓ Prophylactic antibiotic before dental or other surgery
- ✓ Infected teeth should be removed to prevent further attacks

### **BACTERIAL MYOCARDITIS**

- > May be acute or chronic
- ➤ May occur at any age
- ➤ May be asymptomatic and recover spontaneously or
- ➤ Diffuse leading to severe cardiac failure

## **NB:** Other causes of myocarditis are

- **>Viruses**
- ▶ Parasites (protozoa)
- **≻** Radiation
- Chemical poisonso Some are idiopathic

### **ACUTE MYOCARDITIS**

➤ Is commonly associated with viral infections

- ➤The myocardium is oedematous and infiltrated with lymphocytes and macrophages but may be undamaged
- Bacteria myocarditis following a streptococcal infection of the endocardium, myocaridium and pericardium in addition to rheumatic fever causes disability and mortality due to
  - o Alteration of heart fibres
  - o Necrosis of heart tissue causingCCF after the infection is gone
  - o Scar formation (fibrosis)
  - Hypertrophy from cardiac overload and decreased muscle fibre capacity
  - o Permanent damage of the muscle leading to CCF

### **Clinical Features**

- **➤ Non specific symptoms** 
  - o Dyspnoea
  - o Palpitations
  - o Fever

- **➤ Muscle fibre degeneration causes** 
  - o Right and left sided heart failure
  - o Cardiomegaly
  - o Distended neck veins
  - o Tachycardia
  - o Arrhythmias
  - o Systolic murmurs
  - o Pericardial friction rub if pericarditis is present
  - o Signs of infection increasedWBCs, fever etc.

### **MANAGEMENT / TREATMENT**

- ✓ Antibiotics to cure the infection according to C & S of the organism
- ✓ Complete bed rest to conserve energy and decrease myocardial oxygen consumption
- **✓TNC** in the acute phase
- ✓ Oxygen therapy
- ✓ Vital signs especially pulse rate and rhythm
- ✓ Digitalis (Digoxin) as necessary

- ✓ Pericardiocentesis for pericardial effusion / tamponade
- √ For arrhythmias
  - o Procainamide 250 mg orally or in continuous IV infusion of 5 % dextrose or
  - o Lidocaine 1 mg / kg in slow IV drip
- ✓ Corticosteroids for inflammation
- ✓ Anticoagulant therapy for thromboembolism

## **Prognosis**

Majority of patients recover spontaneously with the exception of

- o Rheumatic fever
- o Diphtheria
- o Protozoal infection

### **HYPERTENSION**

An increase in blood pressure places an extra burden on the heart as it works harder to force the blood through the blood vessels This results in an increase in the size of the heart (hypertrophy) and impaired function

## **Pathology**

**Effects of increased pressure in the arteries include** 

- 1. Overworked Left ventricle
  - a. The heart hypertrophies and dilates (aneurysm) to counteract the extra pressure in the arteries
  - b. Excess pressure leads to heart failure
- 2. Renal Arteries
  - a. They become thickened and narrowed leading to diminished blood supply, chronic renal failure and uraemia
- 3. Rupture of Cerebral arteries
  - a. Leads to cerebrovascular accident (CVA)
- 4. Sclerotic Changes in the arteries lead to
  - a. Angina pectoris

- b. Myocardial infarction (MI)
- c. Cerebral thrombosis

### **TYPES**

### 1. ESSENTIAL HYPERTENSION

- Is benign
- Onset 25 55 years
- No obvious cause is known

## **Predisposing Factors**

➤ Anxiety and stress are thought to play a role in release of pressor hormone (angiotensin) from the kidneys that causes chronic vasoconstriction, thereby raising blood pressure

## 2. SECONDARY HYPERTENSION

- Results from other conditions e.g.
  - o Kidney disease
  - o Certain endocrine disorders
    - Phaeochromocytoma a tumour of the adrenal medulla which produces large amounts of adrenaline

# leading to increase in blood pressure

### 3. MALIGNANT HYPERTENSION

## Affects much younger people

- The progress of the disease is much more rapid and death may occur within a few years of onset,
- Frequently complicates to renal failure

## **Diagnosis**

- ✓ Is through diastolic pressure
  - o Mild: 90 104 mr On 3 separate
  - o Moderate 105 11+ iiming
  - o Severe: 115 mmHg + Three readings not necessary
- ✓ WHO Definition BP above 160 / 95 mmHg

## **Signs & Symptoms**

- ✓ Blood pressure above 140 / 90 mmHg
- √ Headache
- **✓ Dizziness**

- √ Tinnitus (ringing in the ears)
- ✓ Epistaxis (nose bleeding)
- √ Breathlessness
- ✓ Angina
- √ If severe
  - o Cardiac asthma
  - o Severe headaches
  - o Vomiting
  - o Convulsions
  - o Papilloedema
  - o Visual disturbances
  - Hypertensive encephalopathy (cerebral irritation without localized lesion)
  - o Cerebrovascular accident (CVA)
  - o Paralysis

#### **MANAGEMENT**

- ✓ Admission and bed rest
- ✓ Observations of vital signs (4 hourly BP chart)
- ✓ Investigations
  - o Urine
    - Urinalysis

- Microscopy
- Creatinine clearance test (from metabolism of creatine and phosphor- creatine, in skeletal muscle)
- ✓ Medication for
  - o Reduction of blood pressure and stabilization
    - Beta-blockers: to block the constricting effects of adrenaline on blood vessels and slow the heart rate
      - Propranolol (Inderal):
         o 10 30 mg tds
    - Diuretics: to increase urinary output
      - Frusemide (Lasix): 20 mg -40 mg OD with Potassium supplements
      - Spironolactone (Aldactone): a potassium sparing diuretic: 2 mg - 4 mg daily

- Methyldopa (Aldomet): to reduce formation of adrenaline (for moderate hypertension)
  - 0.5 g 4.0 g daily PO (can also be given IV)
  - \*\* Observe for postural hypotension \*\*
- Hydralazine (Apresoline):
  - Decreases peripheral resistance and improves cardiac output
  - For moderate to severe hypertension

## **DEEP VENOUS THROMBOSIS (DVT)**

Any vessel may be the site of thrombosis, but it occurs most commonly in a deep vein (pelvic or lower limb) in bed-ridden patients where blood is moving slowly

#### **Causes of Thrombosis**

➤ Injury to the blood vessel leading to phlebitis by o Accidents

- o Operations
- o Intravenous injections
- > Increased viscosity of blood e.g. in
  - o Surgical operations
  - o Labour
  - o Pregnancy after contraceptive pills
- > Stasis of blood flow in
  - o Elderly and inactive postoperative patients
  - o Use of knee pillows
  - o Tight bandages on legs
  - o CCF

## Signs / Symptoms

- ➤These may not be noticed until it becomes pulmonary embolism when a clot lodges in the lungs
  - o Heaviness on the calf muscle
  - o Slight pain
- ➤These two may be slight but a potentially dangerous clot may be present

- o Low grade fever (after an operation)
- o Malaise

#### **Prevention**

- ✓ Early movement and massage of lower limbs to prevent slowing of circulation
- ✓ Early ambulation and exercises post op
- ✓ Supportive elastic bandages/crepe bandage for phlebitis
- √ Firm elastic stockings

#### **MANAGEMENT**

- ✓ Bed rest to prevent dislodging of the clot
- ✓ Elevation of the limb
- ✓ Immobilizing of the limb with a splint or sandbags
- ✓ A bed cradle to take off the weight of bed clothes
- ✓ Anticoagulants (& monitoring of prothrombin time)

## o Heparin

- As a continuous infusion of 10,000 units (100 mg) in 500 ml Dextrose / Saline (every 100 units are equivalent to 1.0 mg)
- 20,000 40,000 units in 24 hours
  - Initial dose: 15,000 units
  - Maintenance dose: 5,000 10,000 units 4 6 hourly
- Contraindication
  - Peptic ulcer
  - Severe hypertension (risk of bleeding in CVA)
- Antidote
- Protamin sulphate: 5 ml IV
   Warfarin
  - 3 10 mg daily
  - Contraindication
    - Peptic ulcer
    - Severe hypertension
  - Antidote:
    - Vitamin K
- √ Vital observations (TOR/Bp)

- ✓ Care of the bowels to prevent constipation and straining while passing stool
- ✓ TNC while confined to bed
- ✓ Later, patient is allowed out of bed wearing a firm elastic bandage or an elastic stocking before getting out of bed
- ✓ Discharge when patient improves

#### **CARDIAC SURGERY**

#### **Indications**

- **➤ Mitral stenosis / Regurgitation**
- **≻**Aortic stenosis / Insufficiency
- > Coronary occlusion
- > Ventricular aneurysms
- ➤ Cardiac tamponade (to remove or cut part of the pericardium)

## **Pre-Operative Care**

- ✓ Vital signs and weight
- ✓ Coughing and deep breathing exercises are taught and practiced

- ✓ Inhalation therapy to improve pulmonary function
- ✓ Coagulation studies
- ✓ Routine laboratory tests
- √ Chest X-ray
- ✓ Electrocardiograph (ECG)
- ✓ Echocardiography using sound to evaluate valvular defects
- ✓ Cardiac catheterization

# Types of Procedures (Open or Closed Heart Surgery)

- ✓ Mitral valvotomy
- ✓ Mitral valvectomy and valve replacement
- ✓ Coronary bypass surgery and anastomosis with a segment of saphenous vein, to bypass an arteriosclerotic lesion
- ✓ Aneurysmectomy
- √\*\*Transplant

# **Post-Operative care**

✓ Monitor cardiac functioning

- ✓ Observation of vital signs, especially peripheral pulses
- ✓ Neurological signs
- ✓ Maintain airway through care and suctioning of endotracheal tube in mechanical ventilation
- ✓ Monitor input and output
- ✓ Assess state of hydration through
  - o CVP
  - o Electrolytes
  - o Specific gravity of urine
  - o Observation of the patient
- ✓ Care of chest tubes by
  - o Milking
  - o Avoiding kinking
  - o Checking drainage should not be more than 200 ml / hr
- ✓ Maintain Foley catheter in place / catheter care
- ✓ Assess pain
  - o Nature, site, duration, type
  - o Provide relief with prescribed analgesics

- ✓ Encourage coughing and deep breathing
- ✓ Change patient`s position (turning) frequently as prescribed / and condition of the patient
- ✓ Evaluation of arterial blood gases
- ✓ Administer parenteral therapy (electrolytes / blood ) as ordered
- ✓ Plan with client and family for meeting both short-term and longterm goals
- ✓ Provide emotional support, relief of anxiety and fear by staying with the patient and explaining the procedures. Encourage verbalization of feelings

# **Complications**

- ➤ Haemorrhage leading to hypovolaemic shock with
  - o Decreasing blood pressure
  - o Increasing pulse rate
  - o Pallor
  - o Restlessness / apprehension

- o Lowered CVP readings
- > Cardiac tamponade causing
  - o Decreased arterial pressure
  - o Increased CVP
  - o Rapid thready pulse
  - o Diminished output
- **➤ Congestive cardiac failure (CCF)**
- > Myocardial infarction
- > Renal failure
- > Embolism
- ➤ Psychosis resulting from inability to cope with anxiety for cardiac surgery

### **VARICOSE VEINS**

- Varicosity is a non-occlusive venous disorder resulting from structural alterations in the venous walls or incompetent valves, usually of the superficial veins of the lower extremities, especially the saphenous vein and its tributaries
- They can also develop elsewhere e.g. in

- o The oesophagus (oesophageal varices) and
- o Rectum (haemorrhoids)

## **Incidence**

- > 10 % of the population
- > More common in women

#### **Risk Factors**

- > Family history
- >Advanced age
- >Any condition which impedes venous return from lower extremities e.g.
  - o Prolonged standing / sitting
  - o Obesity
  - o Pregnancy
  - o Constricting garments

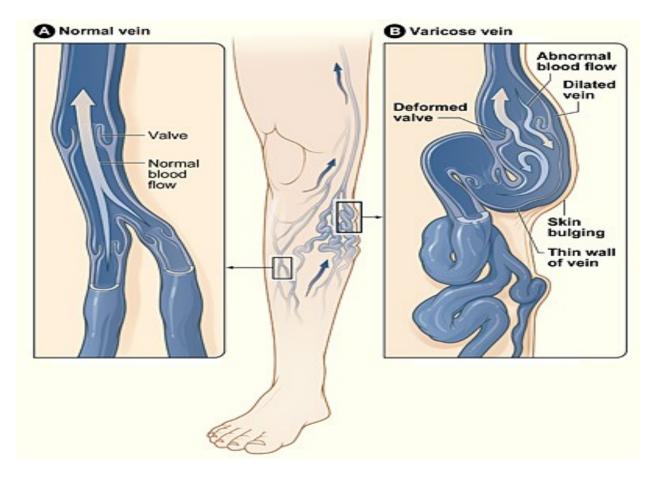
## **Aeteology**

- Structural weaknesses in either walls or valves of the veins affect their distensibility.
- Primary varicosities have an insidious onset but secondary

# varicosities may result from obstruction of deep veins

# **Pathophysiology**

- > Impeded venous return
- ➤ Pooling of blood in the lower extremities
- ➤ Dilatation and stretching of the venous walls which prevent valve leaflets from approximating and closing
- > Retrograde (back) flow
- ➤ Back pressure causes further dilatation and stretching
- ➤ The vein walls stiffen and do not recoil effectively
- ➤ Eventually they become chronically enlarged and tortuous (twisted)



## **Clinical Manifestations**

- ➤ Visible, purplish, prominent, enlarged, twisted veins in the legs which are hard and rope-like on palpation
- > Sensation of heaviness in the legs
- ➤ Cramping can be sharp and stabbing due to ischaemia from venous stasis
- Long-standing varicosities and impaired circulation lead to skin

# colour changes and venous stasis ulcers

# **Diagnosis**

- >Trendelenburg test
  - The patient lies down with legs elevated to 65 ° to facilitate venous emptying
  - A tourniquet is tied on the upper thigh to occlude superficial veins
  - The patient then stands and both direction and speed of filling of vein is recorded
  - o \*\* Fill rapidly from below \*\*
  - The tourniquet is then removed and both refill and speed are recorded
  - o \*\* There is rapid flow of blood downward \*\*
- > Plethysmography
  - o This is use of an instrument which measures accurately the blood flow in a limb
- **➤ Doppler Ultrasound Technique**

- o The machine sends ultrasounds which pick up the velocity of blood flow through the vein and are transmitted as sound
- o \*\* An occluded vein is silent \*\*

### **▶CT Scan**

- o Computerized imaging during a scanning motion
- > Phlebography / Venogram
  - o Is using dye through the dorsal vein of the foot and X-ray film taken

#### **MANAGEMENT**

#### A. General

- a. Weight reduction if obese
- b. Avoiding situations that impede venous return
  - i. Long standing / sitting)
  - ii. Crossing legs at knees
  - iii. Constricting garments
- c. Elevating foot of bed 6 8 inches (15 20 cm)

d. Wearing of full leg elastic stockings

## **B.** Sclerosing Therapy

- Involves injection of a chemical (sodium tetradecyl sulphate or sodium morrhuate) into the varicose vein to produce local, chemical thrombophlebitis (inflammation)
- The endothelial vein walls adhere to each other, obliterating the vessel (Procedure not common)

# C. Phlebectomy

- a. Surgical Ligation / Stripping or Vein
  - i. Treatment of choice for advanced varicosities
  - ii. After ligating both proximal and distal ends of the vein, it is excised and removed (i.e. stripped)
- b. Multiple Cosmetic Phlebectomy (MCP)

- i. Removal of varicose veins through little incisions (stabs) which heal without scarring
- ii. Complications
  - 1. Stasis ulcers
  - 2. Cellulitis
  - 3. Haemorrhage from rupture

## **Prognosis**

Recurrence can occur due to added strain on remaining veins after removal of primary varicosities

# **Nursing Care**

- ✓ General and surgical
- ✓ Support for legs
- ✓ Education on prevention as treatment is palliative
- ✓ Avoid injury which could lead to infection

TRANSIENT ISCHAEMIC ATTACKS (TIAs)

- A TIA is a temporary episode of neurologic dysfunction manifested by a sudden loss of
  - o Motor
  - o Sensory or
  - o Visual dysfunction,

lasting a few seconds / minutes but less than 24 hours

- It is due to temporary impairment of blood flow to a specific region of the brain resulting from
  - o Atherosclerosis of brain supplying vessels at the
    bifurcation (branching) of the
    common carotid and origin of
    vertebral arteries and cranial
    middle cerebral artery
  - Obstruction of cerebral microcirculation by small embolus
  - o Decreased cerebral perfusion pressure and
  - o Cardiac arrhythmias

\*\* Complete recovery usually occurs between attacks but they may serve as a warning of impeding CVA, especially in the first month after the first attack \*\*

## **Clinical Manifestations**

- **Classic symptom Amaurosis fugax** → Classic symptom Amaurosis fugax
  - Painless, temporary blindness
     without warning with loss of vision
     or
  - o Dimming of field of vision of one eye, from retinal ischaemia
    - due to decreased blood supply to the homolateral ophthalmic or carotid artery
- ➤ If ischaemia is in the vertebral basilar system,
  - o Vertigo
  - o Diplopia
  - o Disturbances of consciousness
  - o Signs of motor / sensory impairment

# **Diagnostic Evaluation**

- ✓ Auscultation
  - o A bruit (abnormal sound) heard over the carotid artery
  - Diminished or absent carotid pulsations in the neck
- ✓ Carotid Phonoangiography
  - For auscultation, direct
     visualization and photographic
     recording of carotid bruits
- ✓ Carotid Angiography
  - o Visualizes abnormalities of intracranial and cervical vessels
- ✓ Digital Subtraction Angiography
  - Defines carotid artery obstruction through patterns of cerebral blood flow

#### **MANAGEMENT**

✓ Platelet aggregation inhibiting drugs to prevent cerebral infatction (CVA) in patients with multiple TIAs o Aspirin

- ✓ Carotid Angioplasty
  - Inserting a ballooned catheter in the artery to break up the plaque and dilate the artery (c.f. coronary angioplasty)
- ✓ Carotid Endarterectomy (disobliteration)
  - Is surgical excision and removal of atherosclerotic plaques (qv thromboangiitis obliterans, also diagram in some nurses` dictionaries), or thrombus from the carotid artery to prevent CVA

#### **Post Procedure care**

- √ General (excision site)
- ✓ Neurological observations e.g.
  - Weakness of one side (hemiparesis) due to formation of thrombus at site of endaterectomy

## **Complications**

- >CVA from disruption of blood flow
- **≻Cranial nerve injuries**

- > Infection
- > Haematoma
- ➤ Haemorrhage due to break at site of reconstruction
- > Excessive hypertension from oedema
- ➤ Difficulty in swallowing and hoarseness due to swelling
- ➤ Obstruction of the airway by a large haematoma (may necessitate tracheostomy)
- > Myocardial Infarction



## CHARLES NYORO DISEASE