

CARDIOVASCULAR

Arteriosclerosis

Def: Is the hardening and thickening of the walls of the smaller arteries. Peripheral chronic arterial occlusive disease involves progressive narrowing and eventual obstruction of the arteries to the extremities occurring predominantly on the legs. Other areas where it may occur may affect the aorta: iliac, femoral artery; popliteal; tibial or any combination of these areas.

Incidence: Usually occurs in the 6th and 8th decades of life. Affects primarily men and has a family tendency (genetic).

Predisposing Factors

There are three most significant factors which are risk for peripheral arterial disease i.e.

- i) Cigarette smoking
- ii) Hyperlipidemia
- iii) Hypertension

Other factors are;

- i) Diabetes mellitus
- ii) Positive family history
- iii) Obesity
- iv) Sedentary life style

Pathophysiology

Chronic arterial obstruction leads to progressively inadequate oxygenation of the tissues supplied by the obstructed arteries. The pain attributable to Ischaemia is produced by the end products of anaerobic cellular metabolism such as lactic acid. This usually occurs in the larger muscle groups of the legs during exercise. Once the patient stops exercising, the metabolites are cleared and the pain subsides. But as the disease advances in progress, the pain develops even at rest. This finding indicates insufficient blood flow to the skin and subcutaneous tissues. The patient may notice resting foot pain more often at night and a partial relief by lowering the limb below heart level.

Clinical Manifestations

The severity of clinical manifestations depends on the site and extent of occlusion as well as the adequacy of collateral circulation. The classic symptom of peripheral arterial disease is intermittent claudication defined as Ischaemic muscle pain that is precipitated by a predictable amount of exercise and relieved by resting. For example;

- a) Occlusive disease of the aorta and iliac arteries may cause claudication in the buttocks and upper part of the thighs.
- b) Disease involving the femoral or popliteal arteries may cause claudication in the calf.
- c) If disease extends into the external iliac arteries, impotence may result.
- d) If the disease becomes more severe, the limb will progress to ulceration and gangrenous necrosis (tissue dies and rot).

Paresthesia: Manifested as numbness or tingling occurring in the toes or feet as a result of nerve tissue ischaemia.

True Peripheral Neuropathy: As a result of progressive long standing arterial obstruction seen mainly in diabetics. It produces excruciating shooting (burning pain the extremities). This gradually if ignored progresses to loss of both sensation and deep pain as a result of diminished perfusion to nerve tissue cells.

On physical examination the limb appears pallor or blanching due to inadequate blood flow. When the limb is allowed to hang in a dependent position, there is hyperemia (redness of the skin) and a bluish or dusky appearance.

The skin: Becomes shiny and taut and there is loss of hair on lower legs.

On palpation: There may be diminished or absent pedal, popliteal or femoral pulses.

Diagnosis

- i) History and physical examination including palpation of peripheral pulses.
- ii) Oscillometry will be done to determine pulse volume to the amplitude of pulsation.
- iii) Doppler ultrasound will be done to measure the velocity of the blood flow through the vessel.
- iv) Angiography is used to delineate the location and extent of the disease process.

Management

There are two forms of management i.e.

- a) Therapeutic
- b) Surgical

a) Therapeutic Management/Conservative Management

Its objectives include:

- i) Protecting the extremity from trauma.
- ii) Slowing the progression of arterial sclerosis.
- iii) Decreasing vaso spasm.
- iv) Preventing and controlling infection.
- v) Improving collateral circulation.

The client's risk factors should be assessed and proper intervention should begin regarding cessation of smoking; weight reduction if indicated and control of lipid disorders. Hypertension also needs to be properly managed. Slow progressive and physical activity should be encouraged to help develop collateral circulation. For example, the p+ should be out of bed at least four times a day; walk for 30 minutes twice a day as tolerated and stop if pain occurs but after a rest-break, the client should continue walking. Keep the foot of the bed in the reverse trendleburg position at 10°.

Soaking of the affected part and application of a topical antibiotic may be advised to treat or prevent infection. If ulceration is present, the affected foot should be kept clean and dry. Covering the foot with a dry sterile dressing helps to protect the extremity from trauma and avoid infection.

b) Surgical Management

Indications to this;

- i) When the symptoms of intermitted claudication becomes incapacitating.
- ii) When ulceration or gangrene is severe enough to threaten the viability of the limb. The latter problem will likely progress unless arterial circulation can be restored.

Various surgical approaches can be used to improve arterial blood flow beyond stenotic or occluded artery i.e.

- i) Bypass operation (most common): with autogenous vein or synthetic graft material to carry/bypass blood around the lesion.
- ii) The other surgical option include: Endarterectomy which means opening the artery and removing the obstructing plug.

And patch graft angioplasty: Means opening the artery; removing the plug; gawing a patch to the artery opening to widen the lumen.

- iii) For clients who are not suitable candidates for extensive surgery or in whom the surgical option are impossible, the blood flow to the peripherally may be increased through surgical interruption of the sympathetic nerves supplying the blood vessel of the affected limb. **Rationale:** Stimulation of the sympathetic nervous system results in constriction of the blood vessel. So sympatectomy prevents this vaso constriction hence causing permanent dilatation.
- iv) **Amputation:** Is the last surgical intervention, but it may be required if gangrene is extensive; infection is present in the bone (osteomyelitis); and or major arteries in the limb are occluded precluding the possibility of bypass surgery.

NB: Every effort is made to preserve as much of the limb as possible so that the potential for rehabilitation with orthotic shoes or prosthesis is optimized.

Pharmacological Management

- i) Although various drugs are commonly prescribed to treat peripheral arteriosclerosis disease, no specific agent is known to be effective especially pentoxifylline (Trental) which increase crythrocyte flexibility and reduces blood viscosity thus improving the supply of oxygenatal blood to ischaemic muscle.
- ii) Although it is not conclusive that anticoagulants and antiplatelet aggregating agents i.e. aspirin improves circulation through diseased arteries, they are sometimes used after arterial bypass surgery to promote graft patency.

Nutritional Management Consideration

If the client has evidence of arteriosclerosis, the following should be encouraged:

- a) Caloric adjustment so that optimum weight can be achieved.
- b) Decrease of dietary cholesterol to less than 300mg per day.
- c) Substantial reduction in saturated dietary fat.
- d) Restriction of sodium to 2g per day if oedema is present.

Nursing Management

The client needs to be assessed and nursing diagnosis made. The nursing diagnoses are determined when the problems and aetiological factors are supported by clinical data. Nursing diagnosis related to chronic arteriosclerosis occlusive disease may include:

- i) Altered tissue perfusion related to decreased arterial blood flow.
- ii) Impaired skin integrity related to decreased peripheral circulation; altered sensation and increased susceptibility to infection.
- iii) Pain related to ischaemia and exercise.
- iv) Activity intolerance related to imbalance to oxygen supply and demand.
- v) High risk for injury related to decreased sensation and tissue hypoxia.
- vi) Anticipatory grieving related potential loss of the disease part.

Nursing Intervention

- i) **Health Promotion and Maintenance:** The client should be assessed for risk factors and be taught how to control them. The nursing role in client education in the p+ care facility is important in identifying high risk clients. For example, the nurse should also be involved at the community level such as in screening clinic for hypertension; youth education on hazards of cigarette smoking; assisting families in diet modification to reduce the intake of animal fat and refined sugars; proper care of the feet and the avoidance of injuries to the extremities.

Clients with positive family history of cardiac; diabetes or vascular disease need to be encouraged to obtain regular follow-up care.

Acute Intervention: This is after surgical intervention where the p+ is placed in ICU or recovery area for close observation to be taken.

Observations taken

- a) The operative extremity should be checked every 15 minutes. Initially and then hourly for colour; temperature; capillary refill and palpate for the presence of peripheral pulses distal to the operative site. These findings should be compared with the events pre-operative baseline and with findings in the opposite limb.
- b) Other vital observations are taken. When the client is transferred from ICU or recovery room, nursing care should focus on continued circulatory assessment and monitoring for the development of potential complications.

Symptoms such as recurrent of severe ischaemic pain; loss of pulse or pulses; numbness or tingling and or cold temperature change may indicate occlusion on the bypass graft and should be reported to the surgeon immediately. On the 2nd or 3rd post operative day the p+ should be out off bed 3 to 4 times daily; sitting for prolonged period of time may be discouraged because leg dependency promotes oedema and depending on the location of the bypass may impede flow through the graft. Sitting is only limited to meal time and bathroom privileges only other time the pts should be lying. If oedema develops the leg should be elevated above heart level. If no complications are present discharge from the hospital can be anticipated 7-10 days post operatively.

Chronic/Subsequent Management

Arteriosclerosis is not localized to the lower extremities but is a systemic disease process. The overall approach in the control of arteriosclerosis involves: management of risk factors. This is by:

- a) Tobacco in any form is totally contra-indicated not only because of the vaso constrictive effects of ricotine but also because tobacco smoke impaires transport and cellular utilization of oxygen and increases blood viscosity.

- b) The client be given health education on the right diet; programmed exercises meticulous foot care; to improve collateral circulation through the exercises. The client should be taught to avoid injuries to extremities for skin colour; scabs; mottling or treshions in the texture of the skin or subcutaneous fat or reduction of skin or hair growth. Any ulceration or inflammation be reported to the health care provider. Skin temperature be noted and capillary refill of the fingers and toes be tested. They should be advised to avoid prolonged standing or sitting. Measures to avoid compromising circulation should be implemented including tight bands or socks; tight nylon gathers; tight waist bands; shoes should not be taled tightly and new shoes should be worn gradually.

Complications of Arteriosclerosis

- a) Chronic peripheral arterio disease progresses slowly and prolonged ischaemia leads to citrophy on the skin and underlying structures.
- b) Because of decreased ability to heal infections and necrosis may result from trauma to the feet.
- c) The ischaemic ulcers caused by arterio-insufficiency most commonly occurs over bony prominences on the toes and feet.
- d) If severe ischaemia persist gangrene will develop.

ANEURISMS

Def: These are out pouching or dilatation of the arterial wall and are common problems involving the aorta.

Incidence: They occur in men more often than women and the incidence increases with age. They are seen more often in patients who are 70 to 80 years old and are considered uncommon before age of 50 years.

Pathophysiology

- i) The most common cause of aortic aneurisms is arteriosclerosis; plaques composed of lipids, cholestral and fibrin and other debris adhere along and beneath the intima.
- ii) Plaque formation cause degenerative changes in the media leading to the loss of elasticity, weakening and eventual dilatation of the aorta. As this condition.

- iii) Occur the pulsatile flow of the blood places added stress on the already weakened vessel and causes it to increase in the size. The growth rate of;
- iv) Aneurisms is unpredictable but the larger the aneurism the greater
- v) The risk of rupture. Arteriosclerosis can affect the entire length of the aorta, however most aneurisms related to arteriosclerosis are found in the abdominal aorta below the level of the renal arteries.

Causes of Aneurism

- i) Arteriosclerosis (the major cause)
- ii) Syphilis
- iii) Infections like TB bacterial endocarditis
- iv) Conginetal disorders such as coarctation of the aorta and trauma

Classification of Aneurisms

They are classified into 2 basic classifications;

- i) True
- ii) False

I. True Aneurism: Is one in which the wall of the artery forms the aneurism with at least one vessel layer still intact. This type is most commonly caused by arteriosclerosis. $\frac{3}{4}$ occurs in the abdomen and $\frac{1}{4}$ in the thoralic aorta. Popliteal artery aneurisms rank 3rd in frequency. True aneurism is further sub-divided into:

- a) Fusiform dilatation
- b) Saccular dilatation

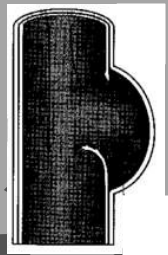
A. Fusiform Aneurism: It is circumferential and relatively uniform in shape.



B. Saccular Aneurism: It is potch like and has a narrow neck connecting the bulge to one side of the arterial wall.



II. False/Pseudo Aneurism: It is not an aneurism but a disruption of all layers of the arterial wall resulting in a leak of blood that is contained or temponaded by surrounding structures. False aneurisms may result from trauma infection or a disruption of an arterial suture line after a bypass surgery.



The false aneurism may also result from arterial leakage after removal of arterial cannula such as upper streimity arterial catheters and intra-aortic balloon pump devides.

Clinical Manifestations

These are varied depending on the site of the aneurism. Thoracic aneurisms are usually asymptomatic.

- i) Deep diffuse chest pain.
- ii) Aneurism located in the ascending aorta at the arch can produce hoarseness in the client as a result of pressure on the recurrent laryngeal nerve.
- iii) Pressure on the oesophagus can cause dysphagia (difficult in swallowing).
- iv) If the aneurism presses on the superior vena cava it can cause distended neck vein and oedema of the head and arm.
- v) Pressure of aneurism on pulmonary structures can lead to coughing; dysponea and airway destruction.

Management

i) Diagnostic studies

Most aneurisms are found on routine physical or x-ray examination i.e.

- a) Chest x-ray films are useful in demonstrating the mediasternal silhouette and any abnormal widening of the thoracic aorta.
- b) A plane abdominal film may show calcification within the wall of an abdominal aortic aneurism.
- c) When ECG is performed, it is used to rule out evidence of myocardial infarction.
- d) Echo cardiograph assists in the diagnosis of aortic insufficiency related to the ascending dilation.

Other examinations are: Computerized tomography, aortograph and ultrasonography.

Therapeutic Management (Medical Management)

The goal of therapeutic management is:

To prevent rupture of the aneurism: Therefore early detection and prompt reaction of the client are imperative. Once an aneurism is suspected studies are performed to determine its exact size and location. A careful review of all body systems is necessary to identify any co-existing disorders. The carotid and coronary arteries should be assessed for an arteriosclerosis disease indication if obstructions in these vessels are present: they may need to be corrected before the aneurism is repaired.

The only effective reaction for an aortic aneurism is surgery. Surgical repair of fusiform aneurism is known as Endoaneurysmorrhaphy. The technique involves incising the diseased segment of the aorta; removing an intra luminal plug inserting a synthetic graft like Dacron or polytetrafluoroethylene) which is sutured to the normal aorta proximal and distal to the aneurism and then suturing the native aortic wall around the graft. If the iliac arteries are also aneurismal, the entire diseased segment is replaced by a bifurcation graft.

Nursing Management

Health Promotion and Maintenance

The client with aneurism may have a variety of manifestations or may be totally free of symptoms. Therefore, the nurse must use assessment skills to focus on early detection and reaction (proper history taking and physical examination). Client should be urged to receive regular routine physical examination and should be reminded that any symptom no matter how minor must be investigated if it persists. Nurses need to encourage persons with venereal diseases and their contacts to obtain appropriate therapeutic intervention in the course of disease and their contacts to obtain appropriate therapeutic intervention in the course of disease progress. Nurses must be aware of cardiovascular disease factors which are risk and be alert for opportunities to teach health measures to clients in the hospital and the community. Trauma victims should be urged to seek medical attention even in the absence of symptoms.

Pre-Operative Care

The nursing role during the pre-operative period should include:

- ii) Teaching.
- iii) Providing support for their clients and families.
- iv) And careful assessing all body systems.

A thorough nursing history and assessment should be performed because most aneurisms are arterosclerotic and arterosclerosis is a systematic disease process it is likely that the disease process is present throughout the body. Therefore, it is important the nurse to watch for signs:

- Of cardiac, pulmonary; cerebral and peripheral vascular problems.
- Clients should also be monitored for signs of ruptured aneurisms.
- Establishing a data baseline is important for later post-operative assessment and intervention. In addition to gathering data: the nurse should;
- Observe the client for subtle abnormality. Special attention should be paid to the quality and character of peripheral pulses; the voice and neurological status. Arterial pulse sites in the lower extremities should be checked and marked before surgery.

Post-Operative Care

In addition to maintaining adequate respiratory function; fluid and electrolyte balance and pain control, the nurse needs to monitor graft patency; renal perfusion and circulation. The nurse can also assist in preventing ventricular arrhythmias; infections and neurological complications.

A. Graft Patency

Patency of an aortic graft can be assured with maintenance of adequate;

- i) Systematic blood pressure. Prolonged hypotension may result to thrombosis of the graft as a result of decreased blood flow. Hypovolemia can be avoided by administration of IV fluids and blood components as indicated.
- ii) Central venous pressure readings should be monitored hourly to help.
- iii) Assess the client's state of hydration. Marked hypertension may cause undue stress on the proximal and distal arterial anastomosis resulting in leakage of blood or rupture at the suture line. Pharmacological interventions with diuretics or antihypertensive agents may be indicated if severe hypertension persists.

B. Ventricular Dysrhythmias

Are usually caused by hypoxia; hypothermia or unrecognized electrolyte imbalance clients with coexisting coronary artery arteriosclerosis are prone to dysrhythmias.

Nursing Interventions

- i) Do cardiac monitoring and the result of electrolyte studies and arterial.
- ii) Blood gases determinations. Persons who return from surgery with
- iii) Hypothermia should be placed with hyperthermia blankets urinary output should be monitored carefully.

C. Infection

The development of a prosthetic vascular graft infection can be a life threatening infection. Nursing intervention to prevent infection should include;

- i) Ensuring that the patient receives a broad spectrum antibiotic as prescribed to maintain adequate blood levels of the drug. It is important;
- ii) To assess body temperature regularly and reporting any elevation. Laboratory data;
- iii) Should also be monitored because arising wide blood cell count may be the first indication of an infection.
- iv) The nurse should ensure adequate nutrition; monitoring serum albumin levels to ensure proper wound healing; signs of infection or any unusual drainage.
- v) Aseptic technique should be maintained when handling any catheters or drainages because they are frequently a portal of entry for bacteria.
- vi) Meticulous perineal care for clients with indwelling catheter for urine is also essential to minimize incidences of urinary tract infections.
- vii) Operation site should be kept clean and dry.

D. Gastro Intestinal Status

After abdominal aneurism resection paralytic ideals may develop as a result of the incinual manipulation and displacement of the bowel for long periods of the surgery. The intestines may become swollen and bruised and peristalsis ceases for variable intervals.

An NGT is inserted and connected to allow intermittent suction. This decompresses the stomach and duodenum and prevents aspiration of the stomach contents and decreases pressure on the suture lines.

The nurse's Responsibility

- i) The NGT should be irrigated with N/saline solution as needed and the amount.
- ii) Character of the drainage should be recorded.
- iii) The nurse should ausculted for the return of bowel sounds.
- iv) The passing of fluters can also be a sign of returning bowel functioning and should be reported.
- v) If the arterial blood supply to the bowel is disrupted during the surgery, ischaemia or death of the intestinal tissue may result. This is evidenced by lack of bowel sounds; fever; abdominal distension and diarrhea stools.

E. Neurological Status

The neurological complications may arise after procedures of surgical or the aorta especially when the ascending aorta and the arch are involved.

Nursing Interventions

- i) Hourly assessment of neurological signs i.e. level of consciousness pupil size and response to light; ability to move all extremities and quality of the hand grasp. These information should be recorded in detail;
- ii) With careful description of the patient's response. Any function decreased from the baseline assessment should be reported to the doctor immediately.

F. Circulatory Status

The anatomical location of the aneurism indicates the areas of major concern related to the circulatory status. For the 1st 24 hours, peripheral pulses should be checked regularly every hour. Depending on where surgery was performed, pulses to be assessed includes the dorsals pedis; posterior tibial; popliteal; removal as well as the brachial; radial; carotid and temporal pulses. It is also important to note the temperature, colour and movement of the extremities.

G. Renal Perfusion

- i) One of the causes of decreased renal perfusion is the possible dislodgement of a fragment of debris from the aorta that subsequently lodges in one or more renal arteries. This causes obstruction and ischaemia of one or both kidneys.
- ii) Hypotension, poor hydration, prolonged aortic clamping or blood loss can also lead to decreased renal perfusion. Therefore, an accurate record of fluid intake and urine output should be kept until the patient resumes the pre-operative diet. If hourly urine output drops below 30mls per hour for 2 consecutive hours, the doctor should be notified immediately. Central venous pressure readings also give important information regarding hydration.

There should be daily blood urea nitrogen (BUN) and serum creatinine studies to evaluate renal function.

Chronic/Subsequent Management

Psychological Support

Clients may be apprehensive about returning home after major surgery involving the aorta. They should be encouraged to express their concerns and be reassured that they can return to activities of normal living. They are taught to observe changes in color or warmth of the extremities. They can also be taught to take peripheral pulses.

Complications of Aneurism

- i) **Rupture of the aneurism:** If rupture occurs posteriorly into the lateral peritoneal space bleeding may be tamponaded by surrounding structures preventing leakage. In this case, the patient has severe back pain and may have back or flank ecchymosis (Turner's sign).

If rupture occurs anteriorly into the abdominal cavity death from massive haemorrhage is likely. If the client does reach the hospital presenting signs are manifestations of shock such as tachycardia, hypotension, pale clammy skin, decreased urinary output, altered sensorium as well as abdominal tenderness.

- ii) **Paraplegia:** Occurs if the blood supply to the spinal cord is severely compromised as a result of rupture, prolonged hypertension or prolonged clamp time during surgery.
- iii) Pressure
- iv) Thrombosis
- v) Stroke
- vi) Lower extremity ischaemia.

THROMBOPHLEBITIS

Is a disorder of a vein. The formation of a thrombus (clot) is associated with inflammation of the vein. Is the formation of a clot in an inflamed vein.

Classification of Thrombophlebitis

- i) Superficial
- ii) Deep

In about 65% of all parts receiving I.V therapy superficial thrombophlebitis develops because of immobility.

NOTE: Of great significance is that embolisation of thrombi from deep veins to the lungs may be fatal and at the least result in prolonged hospitalized.

Causes of Thrombophlebitis

Three important of aetiology causes are:

- i) Stasis low of venous flow.
- ii) Damage of the endothelial/inner layer/lining of the vein.
- iii) Hyper coagulability of the blood.

Therefore, clients who are at high risk for development of thrombophlebitis are those who have conditions predisposing them to any of these three disorders.

i) How Venous Stasis comes about.

- a) Normal blood flow in the venous system depends on the action of muscles in;
- b) The extremities and on the functional adequacy of venous valves allowing flow in one direction only. Venous stasis occurs if the valves are dysfunctional or if the muscles of the extremities are inactive. It usually occurs in people who are •obese; those with Congestive Heart Failure (CHF); those who are immobile for long periods; •also pregnant women and women in the post partum period and •Age.

Clients with arterial fibrillation are also at high risk because of stagnation of blood and the adding of blood flow caused by irregular ventricular contractions in response to fibrillations.

Drugs: Like steroids and quinine also predispose patients to stasis and clot formation.

ii) Endothelial Damage

- a) Damage in the lining of the vein is caused by trauma or external pressure and occurs any time a vein- puncture is performed.

Damaged endothelium has decreased fibrinolytic properties predisposing to development of thrombus.

b) Increased endothelial damage can also be sustained when clients receiving I.V. therapy are receiving high dose antibiotics; potassium; chemotherapeutic agents or hypertonic solutions such as contrast media.

Other Factors Predisposing to Endothelial Inflammation or Damage are:

- a) Presence of I.V. catheter in the same site for longer than 48 hours.
- b) Use of contaminated I.V. equipment.
- c) A fracture that also causes damage to the blood vessels.
- e) Diabetes.
- e) Burns.

iii) Hyper Coagulation of Blood

- a) This occurs in many haemolytic disorders particularly, polycythemia, severe anaemia and anti thrombin (III) deficiency.
- b) Clients with systematic infection in which endotoxins are released also have hypercoagulability.
- c) Clients who take oral contraceptives especially those containing oestrogen are at great risk of thrombo-embolic disease.
- d) Smoking may cause hyper coagulability.

Pathophysiology

- a. Red blood cells, white blood cells, platelets and fibrin adhere to form a thrombosis.
- b. A frequent site of a thrombus formation is the valve of cusps of veins where venous stasis allows accumulation of blood products. As the thrombus;
- c. Enlarges increased amounts of blood cells and fibrin collect around it producing a large tail with a 'tail' that eventually occludes the;
- d. Lumen of the vein. If a thrombus only partially occludes the vein of the blood flow continuous, the thrombus becomes covered by;
- e. Endothelial cells and the thrombotic process stops. If the thrombus does not become detached, it undergoes lysis or becomes firmly organized and adherent within 24 to 48 hours. The organized thrombi may detach giving rise to embolic. This embolic flow

through the venous circulation back to the heart and into the pulmonary circulation resulting to pulmonary embolism.

Clinical Manifestations

They vary according to the size and location of the thrombus and the adequacy of collateral circulation around the obstructive process.

- i) Clients with superficial thrombophlebitis may have a palpable firm sub-cutaneous cord-like vein. The area surrounding the vein may be tender to touch, reddened and warm. There may be raised systemic temperature and leukocytosis. There may be also oedema of the affected part.
 - ii) Clients with deep thrombophlebitis may have no symptoms or have unilateral (1) leg oedema, pain, warm skin and a temperature of greater than 38°C.
- a) If the calf is involved, tenderness may be present on palpation with Homan's sign or dorsiflexion of the foot.
 - b) If the inferior vena cava is involved both lower extremities may be;
 - c) Oedematous and cyanotic. If the superior vena cava is involved both upper extremities as well as the neck and back become oedematous and cyanotic.

Management

- i) **Diagnostic Studies:** Various diagnostic studies are used to determine the site and extent of the thrombus.
- a) **Coagulation Studies:** Platelet count, bleeding time, prothrombin time and partial thromboplastin time is checked.
 - b) **Venous Doppler Evaluation:** Is determination of venous flow in deep femoral, popliteal and posterior tibial veins.
 - c) **There is also Venogram:** Is a radiographic definition of location and site of clot. It can also have filling defect in vein lumen and development of collateral circulation.
 - d) **Lung Scan (Ventilation and Perfusion):** Is a means of determining presence of pulmonary embolism and extent of resulting lung damage.
 - e) **Pulmonary Arteriogram:** Used to define the location and size of pulmonary embolism.

Therapeutic mx (Conservative mx)

- i. The patient is usually kept in bed after diagnosis with elevation of the affected extremity until the tenderness has subsided usually 5-7 days.
- ii. Warm moist heat may be used to relieve the pain and treat the inflammation.
- iii. Mild oral analgesics such as aspirin and codeine are used to relieve pain for more severe pain non-steroidal anti-inflammatory agents i.e. Ibuprofen may be used to treat inflammatory causes and to relieve pain.
- iv. Anti-coagulant therapy is usually used for deep vein thrombophlebitis and not superficial. The patient gets heparin I.V. for 10 days followed by Warfarin which is an anticoagulant for 3 days.
- v. If oedema is present when the client becomes ambulant, elastic stockings are recommended. If oedema persists the use of elastic stockings should be continued after discharge.

Surgical Intervention

Most clients are treated conservatively but a small percentage requires surgical intervention. The primary indication for surgical intervention is to prevent pulmonary emboli.

Surgical Procedures Involved

- i) **Venous Thromboectomy:** Removal of an occluding clot through an incision in the vein.
- ii) **Interior vena cava interruption:** To prevent pulmonary emboli and the surgeon uses teflon clips.
- iii) **Intracaval filter insertion:** Done to patients who are very ill to undergo major surgery and it involves a caval filter device threaded into the right internal jugular vein and into the interior vena cava below the level of the renal veins through local anaesthesia.

Pharmacological Management

The goals of anticoagulant therapy in the treatment of thrombophlebitis are to prevent:

- i) Progression of the clot.
- ii) Development of a new thrombus.
- iii) Embolisation

NOTE: Anticoagulant therapy does not dissolve the clot. Lysis of the clot begins simultaneously through the body's intrinsic fibrinolytic system.

Precautions when using Anticoagulants

- i) Intramuscular injection of any drug is contra-indicated for patients receiving anticoagulants because of the risk of haematoma complications.
- ii) Patients on I.V. heparin shouldn't be given antibiotics and hydrocortisone co-currently because drug interaction may occur. The patient receiving anticoagulant therapy should be advised to seek medical advice before starting to take other medications.

Other drugs used apart from anticoagulants include indocine; Aspirin, Buta zelodine etc. These drugs alter platelet function.

Nursing Management

i) Nursing Diagnosis

- a) Pain related to oedema:** Secondary to impaired circulation in the extremities (complaints of pain in extremities; presence of oedema in the extremities, redness, tenderness and warmth around affected vein.
- b) Potential complication:** Which is pulmonary embolism related to dehydration, immobility and embolization of thrombus signs i.e.
 - (i) Sudden onset of dyspnoea, tachycardia, chest pain, change in mental status, tachpnoea and haemoptysis.
 - (ii) Haemorrhage related to anticoagulant medication because there is bright blood from any body orifice, decreased BP, increase pulse and respiration and restlessness.
 - (iii) High risk for impaired skin integrity related to alteration in peripheral tissue perfusion and possible valvulae destruction (altered skin pigmentation in lower extremities, pain, open ulcer and oedema of lower extremity.
 - (iv) Altered health maintenance related to lack of knowledge about disorder and its reaction (many questions or no questions from the patient).

Health Promotion and Maintenance

Thrombus formation can be prevented in many situations. Prophylactic measures include:

- Early ambulation and leg exercise, post-operative, use of elastic stocking, avoidance of dehydration and;
- Low dose anticoagulant therapy to those at risk.
- The patients should be taught to avoid prolonged standing or sitting in a motionless position which is dependent.
- Patients also be taught on importance of not smoking and the way to perform deep breathing and range of motion exercises.

In addition, the nurse should be able to recognize the patients at high risk with deep venous thrombophlebitis. Avoid use of contraceptives.

Acute Intervention

This nursing intervention is directed towards reduction of inflammation and prevention of emboli formation.

Acute Intervention in Superficial Thrombophlebitis

- i) Use of warm moist packs/socks.
- ii) Elevation of the affected extremity.
- iii) Removal of an I.V. catheter if present.
- iv) Provision of analgesics to minimize pain and inflammation.
- v) Surgical intervention if the greater saphenous of the lower extremity system is involved.

Acute Intervention in Deep Vein Thrombophlebitis

- i) I.V. and oral anticoagulant.
- ii) 5 to 7 days of bed rest with elevation of the affected extremity.
- iii) Use of elastic support (bandages or stockings) to promote venous return in the affected extremity.

- iv) While the patient is receiving anticoagulant therapy the nurse should observe closely for any indications of bleeding. Urine should be assessed for gross of hygroscopic haematuria.
- v) Particular attention be paid to the protection of skin areas that may been traumatized.
- vi) Surgical incisions be closely observed for evidence of bleeding.
- vii) Stools be tested to determine the presence of occult blood from the gastro intestinal tract GIT.
- viii) The Hb and haematocrit levels be monitored when the patient is receiving anticoagulant drugs.

Chronic/Subsequent Management

- i) The patient be encouraged to use elastic stockings, properly measured and fitted and evenly applied.
- ii) The nurse should take care to prevent any pressure under the knee by use of pillows or bed gadgets.
- iii) Patient taught to avoid crossing their legs at the knees to prevent pressure on the popliteal space hence decrease in venous return to the heart.
- iv) Psychological support be given to the client and family.
- v) Discharge teaching should stress the avoidance of contraceptives, hazards of smoking and the need to avoid constricting gathers/girdles.
- vi) Exercise programme be developed with an emphasize of swimming or webbing which are particularly beneficial due to general given pressure of water.
- vii) Dietary consideration for overweight/obese clients are aimed to limit caloric intake to attain desired weight.
- viii) Sodium limitation is necessary if oedema is present.
- ix) Proper fluid balance is required to prevent addition hypercoagulability of the blood.
- x) A well balanced diet is important because calcium, Vitamin E and Vitamin K, all play active roles in clotting mechanisms.
- xi) The client should be instructed on the dosage, action and side effects of anticoagulant.

CORONARY HEART DISEASES

Terminologies

Arteriosclerotic heart disease, cardiovascular heart disease, ischaemic heart disease, coronary heart disease, coronary artery disease, dysrhythmias. The term is used to describe disease mechanisms involved in the coronary heart disease are plug formation, atheromatous deposits and coronary deposits.

Aetiology and Pathophysiology

The major cause is arteriosclerosis.

Pathophysiology

It is characterized by focal deposits of cholesterol and lipids primarily within the intima wall of the artery. The genesis of plug formation is the result of complex interventions between the components of the blood and the elements forming the vascular wall. Intact normal endothelial cell is non-reactive to platelets and leukocytes as well as coagulation fibrinolytic and complement factors. However, the endothelial lining can be altered as a result of chemical injuries such as hyperlipidemia or high shear stress such as hypertension with either type of endothelial alteration. Platelets are activated and release a growth factor that stimulates smooth muscle proliferation. The smooth muscle cell proliferation entraps which are calcified over time and form an irritant to the endothelium upon which the platelets adhere and aggregate. In the process, thrombin is generated and fibrin formation and thrombi occur. This process takes many years to develop that when it becomes symptomatic; the disease process is usually well advanced.

Risk Factors of C.H.D

They are divided into two:

- i) Unmodifiable risk factors
- ii) Modifiable risk factors

I) Unmodifiable Risk Factors

a) Age, Gender and Race

The incidence of coronary heart disease is greater for white middle-aged men. After the age of 60 years, the incidence on men and women equalizes although it may be more increased in

women due to increased stress, increased cigarette smoking, presence of hypertension and use of birth control pills.

b) Family History and Heredity

Genetic predisposition is an important factor in the occurrence of coronary heart disease although the exact mechanism of inheritance isn't fully understood. Some congenital defects in coronary artery walls predispose to the formation of plugs.

c) Diabetes mellitus

This is because persons with diabetes have increased tendency toward connective tissue degeneration.

II) Modifiable Risk Factors

a) Elevated Serum Lipids

A person is at risk of developing coronary heart disease if serum cholesterol level is more than 200mg/dl.

NOTE: The liver is capable of producing cholesterol from saturated fats even when the dietary intake of fats is severely limited.

b) Hypertension

It is related to the shearing stress causing injury to the endothelial lining. Arteriosclerosis in turn causes narrowed, thickened arterial walls and decreases the distensibility and extensibility of vessels.

c) Smoking

The risk of developing coronary heart diseases is 2-6 higher to cigarette smokers than non-smokers. Nicotine in the cigarette smoke cause catecholamine release that causes an increased heart rate, increased blood pressure and peripheral vaso constriction. These changes increase the cardiac work load necessity greater myo-cardial oxygen consumption.

CO₂ a by-product of combustion affects the oxygen carrying capacity of Hb by reducing the sites available for oxygen transport. Thus the effects of an increased cardiac workload combined with oxygen depleting effect of CO₂ from smoking significantly decrease the oxygen available to the myo-cardium.

Other factors are:

- i) Obesity
- ii) Sedentary life style
- iii) Stress and behavior patterns

(I) Type A Behaviour includes: Perfection, hardworking, driving personality.

They suppress anger and hostility.

(II) Have a sense of time urgency. Are impatient and cause stress and within themselves often when a situation does not warrant it.

(III) Type B Behaviour: Easier going, take upsets in stride, know their limitations. They take time to relax, are not over achievers and are able to keep priorities in perspectives.

Clinical Manifestations

Major three ones for coronary heart disease are:

- i) Sudden cardiac death (major).
- ii) Angina pectoris.
- iii) Acute myo-cardial infarction.

A. Sudden Cardiac Death

This is unexpected collapse and cardio-pulmonary arrest of a previously well appearing person within minutes to one hour after the onset of acute symptoms. It occurs secondary to natural cause. The affected person may or may not have prior history of a cardiovascular disease.

Significance

This is the leading cause of death in industrialized nations.

Aetiology: In most instances, sudden cardiac death occurs as a primary manifestation of coronary heart disease and victims usually have multi-vessels coronary arteriosclerosis. Persons who experience sudden cardiac death as a result of coronary heart disease fall into two groups.

- i) Those who had acute myocardial infarction account for 25%.
- ii) Those who didn't have an acute myo-cardial infarction 75%.

In this group of 75%, victims usually offer no warning signs and have no known symptoms. Death is as a result of dysrhythmia formation usually ventricular, tachycardia, ventricular fibrillation or both. These clients are at risk of recurrent sudden death probably because of continued electrical instability of the myocardial which cause the initial event to occur.

The 25% cases usually do have prodromal symptoms such as chest pain and dyspnoea and they have less chances of recurrence.

Risk Factors to Development of Sudden Cardiac Death

- i) Male gender.
- ii) Family history of premature arteriosclerosis.
- iii) Cigarette smoking.

Therapeutic Management for Services

- i) Survivors of sudden cardiac death generally require a diagnostic work up to determine whether they have had an acute myocardial infarction. Thus serum cardiac ISO enzymes and ECG's must be obtained and the client treated accordingly.
- ii) In addition since most persons with sudden cardiac death have coronary heart disease secondary to multi vessel coronary arteriosclerosis cardiac catheterization is indicated to determine the possible location and extent of coronary artery occlusion.
- iii) Coronary artery by-pass graft surgery may be indicated. Drugs to correct dysrhythmias are used such as procainamide, quinidine etc.
- iv) The nurse caring for a survivor of sudden cardiac death needs to be attuned to client psychosocial adaption to this sudden 'brush' with death as these clients develop a time bomb mentality. They fear the recurrence of cardio pulmonary arrest and may become

anxious, angry and depressed. Their families are likely to experience the same feelings. The nurse should provide emotional support.

B. Angina Pectoris

Def: Is a transient chest pain due to myocardial ischaemia. It is literally translated as pain/angina in the chest/pectoris.

Characteristics of this Pain

It usually lasts for a few minutes 3-5 minutes and subsides when the precipitating factor usually exertion is relieved. Typical exertional angina should not persist longer than 20 minutes after a rest or administration of nitro glycerine.

Pathophysiology

- i) Myocardial ischaemia develops when the demand for myocardial oxygen exceeds.
- ii) The ability of the coronary arteries to supply it. The primary reason for insufficient flow is narrowing of the coronary arteries by arteriosclerosis.
- iii) Although skeletal muscles extract only 20% of available oxygen and maintain a reserve, the myocardium at rest extracts 75-85% of the available oxygen. If myocardial needs of oxygen are not met from this near maximum extraction, coronary blood flow is increased through vaso dilation and increased rate of flow.
- iv) In a person with coronary artery disease, coronary arteries are unable to dilate to meet increased metabolic needs because they are already chronically dilated beyond the obstructed area. For ischaemia due to arteriosclerosis to occur,
- v) The artery is usually 75% or more stenosed. In addition, the diseased heart has difficulty increasing blood flow, this creates an oxygen deficit and in arteriosclerotic stenosis oxygen deficit is also caused by coronary artery spasms and thrombosis.
- vi) The left ventricle is more susceptible to ischaemia and injury because of its higher myocardial oxygen demand, larger mass, higher wall tension and higher systemic pressures usually.

- vii) Ischaemia causes transient left ventricular dysfunction resulting in an increased left ventricular diastolic pressure. On the cellular level, the myocardium becomes cyanotic within the 1st 10 seconds of coronary ceases after several minutes depriving the myocardial cells off glucose for aerobic metabolism.
- viii) Anaerobic metabolism begins and lactic acid accumulates myocardial nerve fibres are irritated by increased lactic acid and transmits a pain message to the cardiac nerves and upper thoracic posterior roots (the reason for referred cardiac pain to the left shoulder and arm).

Precipitating Factors to Anginal Pain

- i) **Physical Exertion** which increases the heart rate. Increasing the heart rate decreases the time the heart spends in diastole which is the time of greatest coronary blood flow. Examples of exertions are walking outdoor, wrecking leaves, painting or lifting heavy object.
- ii) **Strong Emotions** which stimulate the sympathetic nervous system and increase the work of the heart. This results in an increase in heart rate; blood pressure and myocardial contractivity.
- iii) **Consumption of a Heavy Meal:** These increases the workload of the heart and during the digestion process, blood is diverted to the GI system causing a low flow rate in the coronary arteries.
- iv) **Temperature Extremes:** Either hot or cold which increase the workload of the heart. This is because the blood vessels constrict in response to a cold stimulus and dilate and pools in the skin in response to a hot stimulus. Cold weather also causes increased metabolism to maintain internal temperature regulation.
- v) **Cigarette Smoking:** Causes vaso constriction, increased heart rate and reduces oxygen carrying capacity of the blood.
- vi) **Sexual activity:** Increases the cardiac workload and sympathetic stimulation.
- vii) **Stimulants:** Such as cocaine or caffeine cause increased heart rate and subsequent myocardial oxygen demand.

Types of Angina

- i) **Stable Angina:** Refers to chest pain occurring intermittently over a long period of time with the same pattern of onset, duration and intensity of symptoms.
- ii) **Unstable Angina:** It is unpredictable, easily provoked by minimal or no exercise occurs during sleep or at total rest.
- iii) **Prinzmetal's Angina:** Often occurs at rest and is as a result of spasm of a major coronary artery.
- iv) **Nocturnal Angina:** Occurs at night but not necessarily when the person is in the recumbent position or during sleep.
- v) **Angina Decubitus:** Is chest pain that occurs only while the person is lying down and is usually relieved by standing or sitting.

Clinical Manifestation of Angina

- i) Most common initial symptom is chest pain or discomfort.

Description of this pain: It is an unpleasant feeling often described as a constrictive squeezing, heavy choking/suffocating sensation. It appears substernally. The sensation may occur in the neck or radiate to the shoulders and down in the arms. Often the patient will compare the pain between the shoulder blades. It often occurs in the left side.

- ii) Severe indigestion or burning.
- iii) Depending on the severity of the Anginal attack the patient may remain motionless or may clench a fist over the sternal area.
- iv) The patient often refers to a feeling of anxiety and impending doom.

Causes of Angina Pains

- i) Severe anaemia
- ii) Arteriosclerosis
- iii) Hypertension
- iv) Aortic stenosis
- v) Aortic aneurism

Management of Angina Pains

A. Diagnostic Management

- i) History and physical examination
- ii) Chest x-ray film
- iii) ECG
- iv) Serum enzyme level tests
- v) Serum lipid level tests
- vi) Exercise stress test
- vii) Angiography studies

B. Therapeutic Management

The drug of choice is glyceryl trinitrate 0.5g sublingually up to 6mg per day. Other drugs are: Nifedine, Isobide, Dinitrite.

Anti-platelet agglutinating therapy i.e. Aspirin. Also drugs acting on the heart can be given i.e. propranolol or oxprenolol.

C. Nursing Management

a) Emergency (During the Attack)

Signs/Presentation/Attacks

- i) Pain located in neck breast bone, left arm & shoulder.
- ii) Cold clammy skin.
- iii) Profuse diaphoresis.
- iv) Nausea and vomiting.
- v) Difficult in breathing.
- vi) Weakness.
- vii) Anxiety CO₂ or feeling impending doom.
- viii) Tachycardia.
- ix) Irregular heart rate and palpitations.
- x) Decreased blood pressure.
- xi) Fainting and loss of consciousness.

Management

- i) Place in semi fowl position.
- ii) Assess severity and location of pain.
- iii) Medication for pain as ordered.
- iv) Monitor cardiac rhythm and vital signs.
- v) Administer oxygen by face mask or nasal cannula at 4 to 6 litres per minute.
- vi) Loosen any constricting clothing.
- vii) Comfort and reassure the patient.
- viii) Transfer the patient to the hospital as soon as possible where cardiac pulmonary resuscitation or defibrillation can be carried out.
- ix) Start the patient on I.V. infusion fluid.
- x) Determine the patient cardiac history.
- xi) Maintain calm environment.

b) Surgical Management of Angina

Two managements are given here i.e.

- i) Coronary artery bypass surgery – Laser Angioplast
- ii) Extra percutaneous transluminal coronary angioplasty.

c) Nursing Management

A. Nursing Diagnosis

- I)
 - (i) Chest pain or discomfort related to ischaemic myocardium.
 - (ii) Anxiety related to diagnosis and awareness of being a victim of heart disease.
 - (iii) Pain and limited activity tolerance.
 - (iv) Uncertainties about the future.
 - (v) Advanced diagnostic test carried on your and pending surgery.
- II) Altered pulmonary tissue perfusion related to decreased cardiac output and abnormality of left heart function.

Management

- i) The patient is nursed on bed rest in the most comfortable position.
- ii) The meals should be small light diet with plenty fluids.

- iii) Close monitoring of vital signs 1 to 2 hourly or depending on the condition.
- iv) The nurse takes part in the facilitations of the investigations.
- v) Drugs are administered as ordered by the doctor and its effects monitored and even the side effects i.e. increased heart rate, pounding headache, dizziness and flushing.
- vi) Patient should be cautioned against rising to a standing position quickly because postural hypotension may occur after nitral glycerol ingestion.
- vii) The patient and relatives are constantly reassured to allay anxiety.

d) Subsequent Management

i) Client/Patient's Education

- a. The patient needs to be reassured that a long productive life is possible even with angina.
- b. Patient needs to know precipitating factors to avoid the attacks and medications.
- c. Relatives needs to be encouraged to give the patient support and have to play a major part in assisting the patient to avoid the precipitating factors by providing small frequent light diet and the importance of continuing with medication.

Complications of Angina

- i) Disrhythymias
- ii) Myocardial infarction

Myocardial Infarction

This occurs when ischaemic intracellular changes become irreversible and necrosis results. Angina due to ischaemia causes reversible cellular injury and infarction is the result of the sustained ischaemia causing irreversible cellular death.

Incidence

Pre-hospital mortality in clients with acute myocardial infarction is approximately 30% to 50%. A substantial number of these deaths occur before hospitalization mortality among clients who reach hospital is about 5%. Most of these deaths occur within the first 3 to 4 days.

Pathophysiology

- i) Cardiac cells can withstand ischaemic conditions for about 20 minutes before cellular death begins.
- ii) Contractile function of the heart stops in the areas;
- iii) Of myocardial necrosis.
- iv) The degree of altered function depends on the area of the heart involved and the size of the infarct.

Lost infarcts involve the left ventricle. This can be on the anterior lateral or posterior wall. An inferior myocardial infarction is called diaphragmatic.

- v) The degree of pre-established collateral circulation also determines the severity of infarction.
- vi) The body's response to cell death is inflammatory process within 24 hours leukocytes infiltrate the area. Enzymes are released from the dead cardiac cells and are important diagnostic indicators.
- vii) The proteolytic enzymes of the neutrophils remove all the necrotic tissue by the 2nd or 3rd day.
- viii) During this time the necrotic muscle wall is thin.
- ix) The development of the collateral circulation improves areas of poor perfusion and may limit the areas of injury and infarction.
- x) At 10 to 14 days a week scar tissue forms the myocardium is considered to be especially vulnerable to increased stress because of the unstable;
- xi) State of the healing heart wall. The scarred tissue is often less compliant;
- xii) Than the surrounding fibres. This condition may be manifested by uncoordinated wall motion, ventricular dysfunction or pump failure.

Clinical Manifestations of MI

NOTE: During the attack, they are similar to those of Angina.

- i) Pain: Severe immobilizing chest pain not relieved by rest or nitrite administration. This pain is due to inadequate oxygen supply to the myocardium. It is described as heaviness, tightness or constriction. It lasts for about 20 minutes or more and described

as more severe than Anginal pain. On the other hand, some clients may not experience pain but may have discomfort, weakness or shortness of breath.

ii) Nausea and vomiting: Results from;

- A. Reflex stimulation of the vomiting center by the severe pain.
- B. It can also result from vaso vago reflexes from the area of the infarcted myocardium that affect the GIT.

iii) Sympathetic Stimulation: During the initial phases of M.I., increased catecholamines i.e. nore and epinephrine are released. The increased sympathetic response results in diaphoresis and vaso constriction of the peripheral blood vessels.

iv) On physical examination, clients will be clammy; ashen and cool condition referred to as a cold sweat.

v) Temperature may increase within the 1st 24 hours up to 38°C and may last as long as 1 week. This is a systematic manifestation of the inflammatory process caused by the infarcted myocardium.

vi) Cardiovascular manifestations i.e.

- a) BP and pulse rate may be elevated initially later the BP may drop due to decrease in cardiac output.
- b) Urine output may decrease.
- c) Rales may be noted in the lungs persisting for several hours to several days.
- d) Hepatic engorgement and peripheral oedema may indicate overt cardiac failure.
- e) Jugular veins may be distended and have obvious pulsations indicating early right heart dysfunction and pulmonary congestion.
- f) The presence of murmurs may indicate valve incompetence.
- g) There may be splitting of heart sounds indicating left ventricular dysfunctions.

Diagnostic Management/Investigations

In addition to those done on angina patients;

- a) Complete blood count.
- b) Thyroid profile.
- c) ECG is performed.

Therapeutic/Medical Management

The doctor will order a number of medications which must be given promptly and side effects monitored. Examples;

- I. Diamorphine strong analgesic given for severe pain dose: 5 to 10 mgs I.V. during emergency and later I.M. Its absence gives morphine sulphate.
- II. Antiemetic to relieve nausea and vomiting i.e. hargactil; scopotamine, plasil/metrocheopramide 10mg I.M., largactil I.V. 25-50mg , stellazine 2 to 3mg I.M. or stemetil 12.5 mgs I.M. These drugs can be continued orally as the patients condition improves.
- III. Aspirin 300mgs Nocte (Antiplatelet).
- IV. Streptokinase 1.5 mu I.V.
- V. Laxatives i.e. Sena group.
- VI. Tranquilizers 5 to 40 mgs for anxiety relieve.
- VII. Start patient on I.V. infusion therapy.
- VIII. Give oxygen therapy through nasal canula also.

Management during Acute State

Acute nursing interventions for the patient/client with acute M.I. are best done in a specialized care unit such as I.C.U. The patient has to be here for 2 to 3 days and the rest hospitalization in the ward priorities for the client care in the initial stage/phase of recovery after M.I. include:

- (i) Pain assessment and relieve-give morphine.
- (ii) Physical monitoring.
- (iii) Promotion of rest and comfort.
- (iv) Alleviation of stress and anxiety.
- (v) Emotional support.

I) Give morphine to **relieve pain** and monitor its efficiency and patient's response.

II) **Monitoring:** The patient has continued ECG monitoring and this should;

- a) Continue in the ward. The nurse should be able to identify disrhythmias and eliminate them from EGG.

- b) Vital signs are taken $\frac{1}{2}$ to 1 hour depending on patient's condition.
- c) Intake and output be evaluated at least once a shift and physical assessment be carried out to detect deviation from the client's parameters.
- d) Check on the patient's neck distention, heart and lung sounds and inspection of evidence of fluid retention.
- e) Assessment of patient's oxygenation is required and the nares should be checked for crepitations and dryness.

III) **Rest and Comfort:** With the severe insult to the myocardium as in the case of infarction, it is important for the nurse to promote rest and comfort. Bed rest be promoted during 1st and 2nd days in a quiet room.

This is because when the patient is resting, the body requires less work from the heart than when it is active. Therefore it is important to plan nursing and therapeutic actions to ensure adequate rest periods free from interruptions:

- a) The environment be very quiet,
- b) Bed clothes be smooth.
- c) Frequent oral care be given.
- d) Adequate warmth is required.
- e) Assurance that personnel are nearby and responsible for client's needs.

It is important that the client understands the reasons why the activities are limited.

NOTE: In spite of these limitations, however, the patient is not immobilized.

IV) **Anxiety** is present in all patients/clients in various degrees, the nurse's role is to identify the source of anxiety and assist the client in reducing it i.e. if the patient is afraid of being alone, a family member is allowed to sit quietly by the bed side or to check in for the patient/client frequently.

V) **Emotional and Behavioural Reactions:** These are varied and frequently follow a predictable response pattern i.e. denial, anger, anxiety and fear. Dependency, depression, realistic acceptance. The role of the nurse in intervention is to understand

what the client is currently experiencing. To assist the client in testing reality and to support the use of constructive coping styles.

Subsequent Management

- (i) Maintain fluid intake and output chart to detect renal impairment.
- (ii) Provide light easily digestible food/diet low in salt and cholesterol.
- (iii) Prevent constipation.
- (iv) Arrange for rehabilitation of the patient (cardiac rehabilitation) which is the restoration of a person to an optimal state of function in 6 areas i.e. physiological, psychological, mental, economic, vocational and spiritual. Client's teaching is a continuous process. There should be close follow-up even after discharge at the special cardiac clinics.

Complications of M.I

- (i) Dysrhythmias
- (ii) Congestive cardiac failure (CCF)
- (iii) Cardiogenic shock
- (iv) Papillary muscle dysfunction
- (v) Ventricular aneurism
- (vi) Pericarditis
- (vii) Dressler syndrome
- (viii) Pulmonary embolism

HYPERTENSION

It is a sustained high mean arterial pressure and is diagnosed when the average of 2 or more blood measurements on at least 2 recordings reveals a diastolic pressure of 90mm/Hg or higher or a systolic pressure of 140 to 150 mm/Hg or higher.

Significance

It is often asymptomatic and many a times there are no symptoms to motivate a person to seek treatment. When symptoms do occur they are often ignored by the person who believes that they are probably insignificant.

Incidence: This increases with age. It is higher in blacks than in whites and is more prevalent in men than in women until the age of 55.

A. Classification of Hypertension

- (i) **High Normal (Mild) Hypertension:** Diastolic range of 85 to 95 mm/Hg and the patient requires more frequent BP check. Patient can be advised on rest.
- (ii) **Severe Hypertension:** Diastolic reading (15mm/Hg) or greater.
- (iii) **Accelerated Hypertension:** The client has a diastolic pressure of 120mg/Hg and a grade 3 retinopathy.

B. Aetiology of hypertension can be classified either as primary or secondary.

- (i) **Primary (Essential) Hypertension:** Accounts for 90% of all cases of hypertension usually occurs between ages of 30 and 50 years. The cause is unknown though it has a familiar tendency.
- (ii) **Secondary Hypertension:** Is elevated BP with an identified cause that can be often be corrected by surgery or medication. Accounts for less than 5% of hypertensions in adults. And 75 to 30°C of hypertensions in you children.

Causes of Secondary Hypertension

- (i) **Renal Disorders:** i.e. acute and chronic glomerulonephritis, pylonephritis, kidney tumour, diabetic nephropathy.
- (ii) **Endocrine Diseases**
 - a) Thyrotoxicosis in which the risk of oversecretion of the thyroid hormone.
 - b) **Cushing syndrome:** Over activity of the adrenal cortex.
 - c) **Pheocsromocytoma:** Due to over secretion of adrenaline as a result of tumour of the adrenal medulla.
 - d) Primary hyperaldorestonism
 - e) Adrenal genital syndrome: Due to over secretion of sex hormones.
- (iii) **Neurogenic conditions that may contribute to hypertension.**

Head injury following an accident; meningitis, cerebral vascular accident/stroke, cerebral, cerebral tumors, acute increase intra cranial pressure.

(iv) **Cardiovascular Disease** i.e. severe anaemia, patent ductus arteriosus, coarctation of the aorta, hyperxalcemia, aortic valvular insufficiency, arteriosclerosis.

(v) **Psychogenic Factors:** Anxiety, stress and emotional disturbance.

(vi) **Other causes are;**

(a) Toxemia of pregnancy i.e. pre-eclampsia.

(b) High fever.

(c) Hormonal therapy i.e. oral contraceptives in steroids.

(d) Drugs esp. antidepressants.

Risk Factors Predisposing One to Essential Hypertension

- I. **Excess Sodium Intake:** This is because sodium causes water retention which causes the walls of the blood vessels to swell with resulting constricting of the lumen and increased systemic vascular resistance.
- II. **Altered Renin Angiotension Mechanism:** Excess quantities of rennin re secreted by the kidneys in some people. This results in the conversion of the angiotensinogen to angiotensin. The angiotensin causes direct arterial constriction and a secondary increase in aldoresterone. This is followed by retention of water and electrolytes with the result of hypertension.
- III. **Excessive Mineral Corticoids:** This is especially an increase in aldoresterone.
- IV. **Stress and Increased Sympathetic Activity:** Factors such us anger, fear and exercise have physiological responses which are normally protective but if they persist, this result in an increased nervous activity of the sympathetic. Increased sympathetic nervous stimulation results in increased vascular vaso constriction and increased heart rate. This also results in rennin release. This result in activation of rennin angiotensin mechanism and increased aldoresterone secretion both leading to elevated blood pressure.

Pathophysiology of Hypertension

For an arterial pressure to rise, there must be an increase either in cardiac output or systemic vascular resistance (arterial blood pressure) $BP = \text{Cardiac Output (CO)} \times \text{Systemic Vascular Resistance (SVR)}$ ($B = CO \times SVR$)

Cardiac Output: Is the stroke volume (volume of blood pumped from one ventricle per beat approximately 70mls) multiplied by the heart rate for 1 minute.

Systemic Vascular Resistance: Refers primarily to the vaso motor tone of the blood vessels in the peripheral vascular system. It is the force opposing the movement of blood. This force is created primarily in the small arteries and arterial and a small change in the diameter of the arterial creates a major cause (change in the systemic vascular resistance). It follows therefore that when SVR is increased a greater amount of pressure is required to pump blood throughout the body. The concept of SVR is so important in understanding arterial blood pressures that some clinicians have defined hypertension as increased SVR usually most people with hypertension have a normal cardiac output.

Clinical Manifestations of Hypertension

Hypertension is often called the “silent killer” because symptoms don’t usually develop until the disease is advanced. If symptoms develop as a late manifestation they are usually secondary to effects on blood vessels in the various organs to tissues or to the increased work load of the heart.

- I) Headache (Major Complaint):** Which occurs frequently in the morning and disappears as the day goes on. It is usually in the occipital region and may be no more than a feeling of stiffness/tightness. It is thought that this type of headache is due to changes in cerebral spinal fluid (C.S.F) in the supine position, C.S.F pressure increases resulting in headache. When the person stands upright, the C.S.F pressure decreases and the headache disappears.

Other symptoms

- II)** Easy fatigueability, dizziness, palpitations, blurred vision, epistaxis (nose bleeding), haematuria, oedema, angina pectoris (overload on the heart), blood pressure constantly higher than 90mm/Hg diastolic, vomiting and retinal haemorrhage, loss of vision due to

papillarity oedema (optic disc of the eye oedema), on the x-ray film, enlarge left ventricle is seen, proteinuria due to renal impairment.

Investigations

- (i) The patient/client's BP measurements in both arms should be carefully evaluated to detect hypertension.
- (ii) History taking which should include the time and duration of headache, family and social history.
- (iii) Routine urinalysis: For blood urea and nitrogen and serum creatine are carried out to screen for renal involvement.
- (iv) Measurements of serum electrolytes particularly potassium level is important to detect aldosterolism.
Blood glucose levels are very important to exclude diabetes mellitus and Cushing's syndromes.
- (v) The patient care have ECG done to provide cardiac status and CXR provides baseline information regarding the heart size as well as aortic dilatation rib notching which occur in coarctation of the aorta.
- (vi) Intravenous pyelogram (IVP) can be carried out to check on kidney deficiency.

Management

The goal in treating hypertensive patients/clients is:

- (i) **To prevent the morbidity and mortality associated with high blood pressure.**

The decision to initiate therapy depends on two major factors i.e.

- a) The severity of the blood pressure elevation.
- b) Presence of other complications.

Non-drug therapeutic interventions in all persons either border line or sustained hypertension.

These measures include:

- (i) Diet management
- (ii) Regular exercise
- (iii) Smoking cessation
- (iv) Stress avoidance and management

(I) Regular Exercise Advises: Regular isotonic exercises such as walking, jogging and swimming can help control blood pressure, promote relaxation and control body weight.

(II) Stress Reduction and Management: The person should learn to identify events and agents that act as stressors in life and develop and implement methods to cope with them. This will help reduce BP and even control it. The patient needs to be taught relaxation techniques and also psychotherapy.

(III) Avoidance of cigarette smoking:

Pharmacological Management

The goals of these are:

- (i) To reduce and maintain the diastolic at less than 90mm/Hg.
- (ii) Keep uncomfortable or disabling side effects to a minimum.

The drugs used are:

- (i) Beta Blockers** i.e. propranolol 40mgs daily.
- (ii) Alpha Blockers** i.e. labetalol 30mgs daily.
- (iii) Vaso dilators** i.e. hydralazine (apresoline) 50 to 200mgs od orally.

In very acute stage, give 20 to 40mgs I.V. Others or aldomet (methyldopa) 250 to 500mgs orally.

(iv) Diuretics i.e. furosemide 40 to 80mgs I.V., I.M., orally conditionally or aldactone A; a slow acting diuretic and blocks the aldosterone hormone production dose 50 to 200 mgs daily.

(v) Nutritional Considerations: Dietary management of hypertension consist of;

- a) Sodium restriction
- b) Caloric restriction if the patient is overweight.
- c) Restriction of cholesterol, fat and alcohol intake.

A. Sodium Restriction: A high sodium intake may limit the effectiveness of certain anti hypertensive drugs.

Restriction reduces the circulatory volume thus reducing the work of the heart. A common recommended restriction is 2gm of sodium per day. This involves not adding salt in the preparation of foods or at meals and avoiding food known to be high in sodium.

B. Caloric Restriction

Obesity has a high correlation with hypertension thus weight reduction has a significant effect on lowering BP. The amount of caloric restriction depends on the degree of obesity. The restriction of cholesterol and fats will retard the progress of arteriosclerosis.

Nursing Management

- (i) Usually patients with severe hypertension are admitted in the ward on complete bed rest. Therefore, the nurse should ensure comfort and rest.
- (ii) Thorough investigations are undertaken to identify the cause of hypertension.
- (iii) Observations of vital signs are taken, temperature and respirations are taken 4 hourly but the BP and the pulse are taken 4 hourly; 2 to 4 hourly when the patient is standing or sleeping lying to detect postural hypotension.

NOTE: The frequency of BP monitoring depends on how elevated the BP is.

- (iv) The patient is weighed twice a week; the nurse should monitor nervous system complication i.e. headache, vomiting problems with vomiting etc.
- (v) Fluid intake and output chart be maintained with particular attention to urinary output. Daily urinalysis to detect proteinuria and blood in urine be done.
- (vi) Drugs are given as per the doctor's prescription, following by careful monitoring of BP to check on effectiveness of the drugs and client's response to therapy.
- (vii) Since the patient is on complete bed rest, the nurse provides for other patients' needs i.e. hygiene, elimination, feeding etc.
- (viii) The patient requires reassurance/psychotherapy to reduce anxiety.

Subsequent Management

The primary nursing responsibilities for the long term management of the client with hypertension are:

- a) To assist in reducing blood pressure.
- b) To begin or continue client education i.e.
 - (i) Diet therapy
 - (ii) Stress modification
 - (iii) Drug therapy taken as prescribed.
 - (iv) Exercise
 - (v) Home monitoring BP if appropriate
 - (vi) Smoking cessation if applicable

The client/patient should be educated to get out of bed in the morning gradually to prevent postural hypotension. Therefore, get up and sit upon the edge of the bed, then stand up and hold the bed frame before walking, walk slowly to the toilet or any other place you wish.

Complications of Hypertension

- (i) Hypertensive encephalopathy.
- (ii) Severe persistent headache.
- (iii) Intra cranial haemorrhage.
- (iv) Acute left ventricular failure with pulmonary oedema.
- (v) Dissecting aortic aneurism.
- (vi) Head haema.
- (vii) Unstable angina.
- (viii) Myocardial infarction.
- (ix) Retinal damage.
- (x) Congestive heart failure.
- (xi) Severe hypertension related with pregnancy.
- (xii) Renal failure.
- (xiii) Severe persistent headache.

PULMONARY EMBOLISM

It is the most common pulmonary complication in hospitalized patients. Most pulmonary emboli arise from the thrombi in the deep veins in the legs. Other sites of origin include the right side of the heart especially with atrial fibrillation, upper extremities and the pelvic veins especially after surgery or child birth. Fatal pulmonary emboli originate most commonly in the removal and iliac veins.

Pathophysiology

- (i) Emboli: Are mobile clots that generally don't stop moving until they;
- (ii) Lodge at a narrowed part of the circulating system. The lungs are an ideal location for the emboli to lodge due to its extensive arterial and capillary network.
- (iii) The lower lobes are most frequently affected because they have a higher blood flow than other lobes.
- (iv) Thrombus in the deep veins can dislodge spontaneously but a common.....is jarring of the thrombus by mechanical forces such as a sudden standing and changes in the rate of blood flow. In addition to dislodged thrombi, other causes of primary thrombi include cur emboli from improperly administered I.V. therapy, pulmonary emboli from fractured bone, amniotic fluid and tumours.

Clinical Manifestations

The severity of the clinical manifestations depends on the size of the emboli and the number of blood vessels occluded.

- (i) Sudden onset of unexplained dyspnea, tachypnea and tachycardia.
- (ii) Cough, chest pain, haemoptysis, rales, fever, a cessation of the pulmonary heart sound.
- (iii) Sudden change in mental status as a result of hypoxia.
- (iv) Massive emboli may cause sudden collapse of patient with shock, pallor, severe dyspnea and crushing chest pain.
- (v) The pulse is rapid and weak.
- (vi) Blood pressure is low and ECG indicates right ventricular strain.
- (vii) When rapid obstruction of 50% or more of the pulmonary vascular bed occurs, acute pulmonary failure may result because the ventricles can no longer pump blood into the lungs.

- (viii) Death occurs in more than 60% of people with massive emboli.
- (ix) Medium sized emboli often cause chlorotic chest pain accompanied by dyspnea, slight fever and a productive cough with blood stained sputum.
- (x) Physical examination may indicate tachycardia and a pleural friction rub.

CVD

Diagnosis

- (i) History and physical examination.
- (ii) Lung scan.
- (iii) Chest x-ray film.
- (iv) Continuous ECG monitoring.
- (v) Pulmonary angiography.
- (vi) Venogram.

Treatment

When the diagnosis of thrombo embolic disease has been made, treatment must be instituted immediately as this is a medical emergency.

Emergency Therapy

- (i) Patient be put in bed in a sitting up position.
- (ii) Resuscitative measures carried out.
- (iii) Oxygen is administered by mask or canula.
- (iv) Establishment of I.V. route for drugs and I.V. infusions.
- (v) Patient is given continuous I.V. heparin 100mgs, morphine 10-15mg I.V., Stat and PRN.
- (vi) Pulmonary embolectomy in life threatening situations may be done.

Objectives of Therapeutic Management (Treatment)

- (i) To prevent further growth/multiplication of thrombi in the lower extremities.
- (ii) To prevent embolization from the lower extremities to the pulmonary arteries.
- (iii) To provide cardio pulmonary support as indicated.

Conservative Therapy

- (i) The patient requires cardio pulmonary support where there is administration of oxygen by mask and cannula and in some conditions endotracheal intubation and mechanical ventilation may be needed to maintain respiration.
- (ii) Respiration measures such as turning, coughing and deep breathing are necessary to prevent/treat atelectasis (lung collapse).
- (iii) If shock is present, vaso pressor agents may be necessary to support circulation.

- (iv) If heart failure is present, digitalis and diuretics are used.
- (v) Pain resulting from pleural irritation or reduced coronary blood flow is treated with narcotics usually morphine.
- (vi) Properly managed anticoagulant therapy is very effective in patients/clients with pulmonary emboli.
- (vii) Heparin and warfarin may be given. Start with heparin and continue with warfarin.

Surgical Intervention

If the degree of pulmonary arterial obstruction is severe, usually greater than 50% and the patient does not respond to conservative therapy, immediate embolectomy may be indicated. This is possible with the use of temporary cardio pulmonary bypass.

NOTE: Thrombolytic agent i.e. Urokinase and streptokinase have been shown to dissolve pulmonary emboli within 24 to 48 hours. Both agents have been suggested for use in patients with massive emboli or in whom surgery is contra-indicated.

Nursing Management

The prognosis/outcome of a client with pulmonary emboli is good if therapy is promptly precipitated/instituted.

- (i) The patient is kept in bed in a semi Fowler's position to facilitate breathing.
- (ii) Patent I.V. line maintained for medication and I.V. line therapy.
- (iii) Oxygen therapy be administered as per arterial blood gases results.
- (iv) Careful monitoring of vital signs; ECG, blood gases and lung sounds is critical to assess the patient's status.
- (v) The client is usually anxious because of pain, inability to breathe and fear of death. The nurse should carefully explain the situation and provide emotional support and reassurance and help thus to relieve the patient's anxiety.
- (vi) During the acute time, someone must be with the patient as much as possible.

Subsequent Management

Clients affected by thrombo embolic processes require much psychological and emotional support. Because in addition with thrombo embolic process, they may be underlying another

(underlying illness requiring lung treatment). Discharge planning is aimed at limiting progress of the condition and preventing complication thus the nurse therefore must reinforce the patient need to return to the health care facilities and follow up.

Complications

- (i) Pulmonary infarction (death of the lung tissue).
- (ii) Pulmonary hypertension.

MYOCARDITIS

It is the inflammation of the myocardium. It may be focal or diffuse.

Aetiology/Cause

- (i) Micro organisms, viral, bacterial, rickettsial, parasitic etc.
- (ii) Radiation, pharmacological and chemical factors.
- (iii) Certain medical conditions such as metabolic disorders and collagen vascular disease i.e. systemic lupus erythematosus.
- (iv) Pericarditis caused by coxsackie virus B. strains or echoviruses.

Pathophysiology

Its mechanisms are poorly understood because there is usually a period of several weeks after the initial infection before the development of manifestations of myocarditis. Immunological mechanisms may play a role in the development of myocarditis.

The majority of infections are benign, self-limiting and sub-clinical, although viral myocarditis in infants and pregnant women may be virulent.

Clinical Manifestations

These are variable, ranging from a benign course without any overt manifestation to severe heart involvement or sudden death i.e.

- | | | |
|---------------|-----------------|-----------------------|
| (i) Fever | (iv) Myalgia | (vii) Liver pathology |
| (ii) Fatigue | (v) Pharyngitis | (viii) GIT complaints |
| (iii) Malaise | (vi) Dyspnoea | |

Investigations

- (i) ECG to check on changes i.e. disrhythmias and conduction disturbances.
- (ii) WBC.
- (iii) Serum enzymes.

Management

- (i) There is no specific management.
- (ii) Patient is nursed in bed.
- (iii) Vital signs are taken 2 to 4 hourly.
- (iv) Investigations are done to find out the underlying course and state of the heart.
- (v) Patient may require oxygen therapy and maintenance of stand by emergency equipment and general supportive measures used for management of myocarditis.
- (vi) Drugs used.
 - (a) **Digoxin:** Small doses are given with myocarditis patients because patients are more sensitive to adverse effects of digoxine.
 - (b) **Immune suppressive therapy** with drugs like prednisone, cyclosporine. These drugs reduce myocardial inflammation and prevent irreversible myocardial damage.
 - (c) **Antibiotics:** Given until the temperature subsides.
 - (d) **Analgesics:** For relieving pain.

Prognosis

The majority of individuals with myocarditis recover spontaneously. Occasionally acute myocarditis progresses to chronic dilated cardiomyopathy.

PERICARDITIS

It is made up of two layers i.e.

- (i) **Inner serous membrane:** That closely adheres to the epicardial surface of the heart.
- (ii) **Outer fibrous layer** called parietal. Inner visceral layer.

The pericardial space is the cavity between these two layers and in the normal state contains less than 50mls of serous fluid. Although the pericardium may be congenitally absent or surgically

removed the structure serous of useful anchoring function. Provides lubrication from the serous pericardial fluid to decrease friction during systole and diastole and assist in preventing excessive dilatation of the heart during diastole.

Pericarditis: Is a syndrome caused by inflammation of the pericardial sac which may occur on an acute basis.

Causes of Pericarditis

(i) Infectious: Causes

- a) Viral causes including coxsackie virus B and A, Echo virus, adeno virus, mump virus, infectious mono nucleosis, varicella and hepatitis B.
- b) Bacterial causes including pneumococci, staphy lococci, strepto cocci.
- c) Tuberculosis.
- d) Fungal causes including histoplasmosis, candida species.
- e) Infections like toxo plasmosis and lyme disease.

(ii) Non-Infectious Causes

- a) Anaemia.
- b) Acute myocardial infarction.
- c) Rheumatic fever.
- d) Neoplasms i.e. lung cancer, breast cancer, leukaemia, hodykin's disease and lymphoma.
- e) Trauma after thoracic surgery, pace maker insertion, cardiac diagnostic procedures.
- f) Radiation.
- g) Dissecting cortic aneurism.
- h) Myxoedema.

(iii) Hypersensitive or Auto Immune Conditions

- a) Delayed post myocardial pericardial injury.
- b) Dressler syndrome (post myocardial infarction).
- c) Post pericardiotomy syndrome.
- d) Drug reactions i.e. from procanimide or hydrallazine.
- e) Rheumatological diseases i.e. rheumatoid arthritis, systemic lupus, erythomatus etc.

Pathophysiology

The pathological changes related to acute pericarditis is acute inflammation. Findings include:

- a) Presence of polymorph nuclea leucocytes.
- b) Increased pericardial vascularity.
- c) Fibrin deposition on the visceral pericardium which often results to the acute injury by exudation of fluid.

Clinical Manifestation

- (i) **Chest Pain:** The intense preriotic chest pain is generally sharpest over the left precoridum or retrosternally but may radiate to trapezius ridge and next mimicking angina or sometimes to the epigastrium or abdomen mimicking abdominal or other non-cardial pathological conditions. The pain is aggravated by lying supine, deep breathings, coughing, swallowing and moving the trunk and is eased by sitting up and leaning forward.
- (ii) **Dyspnea:** The dyspnea accompanying acute pericarditis is related to the client's need to breath in rapidly, shallow breaths to avoid chest pain and may be aggravated by fever.
- (iii) **Pericardial Friction Rub:** The hallmark finding in the acute pericarditis is the pericardial friction rub. The rub is a scratching, grating, high pitchest sound, believed to arise from friction between the roughened pericardial and epicardial surfaces. It is best heard with stethoscope placed firmly at the lower left sternal border of the chest.
- (iv) **On Chest X-Ray:** Pericardial effusion can be seen on chest x-ray film.
- (v) **Fever**

Diagnosis

- (i) History and physical examination: Cynosis.
- (ii) Auscultation of the chest.
- (iii) ECG
- (iv) CT scan (brain scan)
- (v) Chesdt x-ray film
- (vi) Echocardiograph
- (vii) Pericardiocynthesis (removal of fluid stain)

- (viii) Pericardial biopsy
- (ix) Nuclea scan of the heart

Management

The management of acute pericarditis is directed forward identification and treatment of the underlying problem.

Antibiotics may be used to treat purulent pericarditis. The pain and inflammation of acute pericarditis is usually reaction by non-steroidal and anti-inflammatory agents i.e. high doses of salicylates i.e. 300mg to 900mg Qid or of indomethacin 25 to 500mg orally Qid.

Nursing Care

- (i) Patient is nursed in a complete bed rest in a sitting up position.
- (ii) Assessment of the amount of quality and location of pain is important particularly in distinguishing the pain of pericarditis versus acute myocardial infarction. Also exclude ischaemic chest pains. Other vital signs are taken 2 to 4 hourly.
- (iii) Pain relieve measures i.e. maintaining the client on bed rest with head of the bed elevated to 45° and providing a padded over table for the client for leaning to relieve pain.
- (iv) Monitor the side effects of prescribed anti-inflammatory medications i.e. administration of the drugs with food or milk and instructing the patient to avoid alcoholic beverages while taking this drugs.
- (v) Patient is given high protein diet, light and attractive with plenty of fluids (to treat the fevers). Maintain the fluid balance chart to ensure the patient is getting enough within 24 hours.
- (vi) Anxiety reducing measures including simple explanations completely of all procedures done.

The real potential for decreased cardiac output also exists for the client of acute pericarditis because of the possibility of the cardiac tamponade.

Complications of Pericarditis

- (i) **Pericardial Effusion:** This can cause compression of its adjoining structures if it is large.
- (ii) **Cardiac Tamponade:** Which develops as the pericardial effusion increases in size with failure of compensatory mechanisms to adjust to the continuing altered cardiac compliance.

The patient with pericardium tamponade is often confused, agitated and restless and has tachycardia and tachpnoea with a low output state.

The neck veins are usually markedly distended because of jugular vein pressure elevation and a significant pulses paradoxus is present.

Rheumatic Heart Disease

Rheumatic Fever: Is an inflammatory disease of the heart potentially involving all layers (endocardium, myocardium and pericardium), the resulting damage to the heart.

From rheumatic fever is called rheumatic heart disease a chronic condition characterized by scarring and deformity of the heart valves.

Significance: Acute rheumatic fever is a complication of up to 3% of sporadic upper respiratory infections called by group; A Beta haemolytic streptococci species. Initial and reccurant episodes of acute respiratory infection are most common from ages 6m through 15 years.

Most recurrences occur within 2 years of the initial episodes. Recurrent attacks of rheumatic fever are 2's as common between ages of 11 and 22 years as after the age of 22. The frequency of rheumatic recurrence after streptococcal infection is greater in those with rheumatic heart disease than in those who haven't had cardiac injuries during frequent attacks.

Aetiology/Cause

Rheumatic fever almost always occur as a delayed sequence usually 2 to 3 weeks to a group; A Beta haemolytic streptococcal infections of the upper respiratory system usually a pharyrangeal infections. In addition to the infecting organism, other factors play a predisposing role in the development of rheumatic fever. They include;

- a) Social economic factors i.e. low social income groups, crowded living conditions, neglected inadequate treatment, poor nutrition and a lowered state of health.
- b) There seems to be a familial tendency toward rheumatic fever which may be genetically related possibly due to an altered immune response.

Pathophysiology

The coloration of streptococcal pharyngitis with rheumatic fever is conclusive but the pathogenic mechanisms by which streptococcal infection causes inflammation of the heart and other tissues are not well defined. The organisms is not demonstrated in the lesions when rheumatic fever appears several days or weeks after acute pharyngitis manifestations of acute rheumatic fever appear to be related (in susceptible patients) to abnormal immunological response to an upper respiratory infection with a beta haemolytic streptococcal. Acute rheumatic fever probably affect joints, CNS and the skin because of an abnormal humoral and cell related immune response to group A beta haemolytic streptococcal cell membrane antigens. It is possible that these antigens cross react with other tissues and bind to receptors on heart, muscle, joint and brain cells triggering immune and inflammatory responses.

Cardiac lesions and Valvular Deformities

About 40% of acute rheumatic fever episodes are marked by carditis and all layers of the heart may be involved. This generalized involvement gives rise to the term rheumatic. Rheumatic endocarditis is found primarily in the valves with swelling and erosion of valves reflexes. Vegetations form in areas of erosion from deposits of blood cells and fibrin. These lesions initially create fibrous thickening of the valve commissures chordate tendinealand fibrosis of the papillary muscle. The valves may become stenotic and insufficient. The initial and cortic values are most commonly affected. Less commonly involved are the tricuspid value and rarely pulmonary values. Value leaflets may fuse and become thickened or even calcified resulting in stenosis. Reduction in the mobility of value leaflets may occur with failure of the leaflets to oppose, regurgitation and result afterwards. Myocardial involvement is characterized by aschoff bodies which are nodules formed by a reaction to inflammation and accompanying swelling, fragmentation of collagen fibres. As aschoff bodies age, they become more fibrous and scarred tissue is formed in the myocardium. In addition to the aschoffs, a defused cellular infiltration is

present interstitial tissues of the myocardial. Rheumatic pericarditis affects both layers of the pericardium which becomes thickened and covers with fibrous exudates a serous effusion. Pericardial fluid may be present.

When healing occurs, fibrosis and adhesions develop partially or completely obliterate the pericardial sac extra cardiac lesions.

Extra Cardiac Lesions

Lesions of rheumatic fever are systematic involving especially the connective tissues i.e.

- (i) **Joints:** Polyarthritis
- (ii) **Skin:** Subcutaneous nodules
- (iii) **CNS:** Chorea
- (iv) **Antigen:** (Erythema marginatum)
- (v) **Lungs:** Fibrinous pleuritis and rheumatic pneumonitis

Clinical Manifestations

Diagnosis of acute rheumatic fever is suggested by clustering of signs and symptoms as well as lab findings. The presence of 2 major criteria or 1 major and 2 minor criteria, indicate a high possibility of acute rheumatic fever. Either combination must have evidence of an existing streptococcal infection.

Jones Criteria of A.R.F (Acute Rheumatic Fever)

Major

- Carditis
- Polyarthritis
- Chorea
- Erythema marginatum
- Subcutaneous nodules

Minor

- Fever
- Previous occurrence of rheumatic heart disease
- Arthralgia
- Prolonged PR interval
- Laboratory findings

A. MAJOR CRITERIA

Because the five major manifestations of rheumatic fever (carditis, migratory polyarthrititis, chorea, erythema marginatum and subcutaneous nodules) can appear alone or in combination. Rheumatic fever has many clinical patterns fever is a prominent syndrome but is not specific.

a) Migratory Polyarthrititis

Joints generally the large ones such as the wrists, elbows, shoulders, knees and hips show limited range of motion, extreme tenderness swelling and inflammation.

The arthritis is migratory and reversible in nature of progression polyarthrititis. Others during the 1st to 2 weeks during the febrile period and lasts for a few days in certain affected joints before moving to other joints.

b) Carditis

It is seen in approximately 30% of children who have acute rheumatic fever. Inflammatory, haemorrhagic, bullous known aschoffs bodies/nodules form in the interstitial tissues of the heart in response to rheumatic myocarditis. An organic heart murmurs not usually present usually from mitral or aortic regurgitation or mitral stenosis are heard.

c) Chorea (Sydenham's chorea: St. Vitus Dance)

It is the major CNS manifestation. It is characterized by involuntary random muscular movements of the extremities, involuntary facial grimaces, weakness, ataxia which tends to intensify with voluntary activities. The child has emotional lability, speech disturbance and severe muscle weakness.

NOTE: Children with chorea may be considered irritable or restless and judged to be behavior problem.

Attempts to control fine motor activities and intensifies involuntary movements, whereas sleep and rest relieve them. These clinical manifestations may last for months before disappearing.

d) Erythema Marginatum

These are less common features of rheumatic fever. They are bright pink like map like macular lesions which eventually join together in a connective pattern giving a chicken wire decoration on the skin (appearance). Mainly found on the trunk or inner aspects of the upper arm and thigh but never on the face. Characteristically these lesions are evanescent and can be encouraged to appear with application of heat. They are neither puritic nor itching and also not raised. The rashes are usually transitory that is lasting for a few hours and may reoccur intermittently for months.

e) Subcutaneous Nodules

Are firm small hard painless swellings and most commonly over bony prominences. For example, knees, elbows, spine, scapula, knuckles, ankles, scalp and spinous processes of the vertebra. They frequently aren't noticed by the person because the skin overlying nodules moves freely and is not inflamed. They gradually resolve over a period of time with no residue.

B. MINOR MANIFESTATIONS

Are two non-specifics to be able to be used solely for diagnosis and therefore are used as assisting criteria when evaluating the patient. These include;

- (i) A low grade fever highest in the afternoon.
- (ii) Weakness.
- (iii) Fatigue.
- (iv) Abdominal pain sometimes thought to be symptomatic of appendicitis.
- (v) Weight loss.
- (vi) Epistaxis with no known cause.

Diagnosis

- (i) History and physical examination.
- (ii) ASO Title (Anti streptolysin o) – 15 the test used to confirm a recent group A streptococcal infection.
- (iii) Throat culture for group A streptococcus

(iv) WBC

(v) ESR

(vi) Chest x-ray film may show an enlarged heart if C.C.F is present.

(vii) ECG

(viii) Echocardiogram: Will show valvular insufficiency and pericardial fluid or thickening.

Summary of Therapeutic Management

(i) Bed rest.

(ii) Benzathine penicilline 1.2 mg I.M. or procain penicilline 600,000 units for 10 days.

Aspirin, corticosteroids

No specific treatment will cure rheumatic fever. Reaction consists of drug therapy and supportive measures.

I) Antibiotic Therapy does not modify the course of the acute disease or the development of carditis.

Penicillin eliminates residue group A beta haemolytic streptococcal remaining in the tonsils and pharynx and prevents the spread of organisms to close contact.

II) Aspirins and Steroids: Are the two anti-inflammatory agents most widely used in the management of acute rheumatic fever. Both are effective in controlling fever and joint manifestations. Salicylates are used when arthritis is the main manifestation and steroids if severe carditis is present.

III) Bed rest: Prolonged periods of bed rest have previously been recommended but the patient without carditis may be ambulant as soon as the symptoms have subsided and may return to normal activities when the anti-inflammatory therapy has been discontinued. When carditis is present, ambulation is postponed until CCF has been controlled with reaction.

Nursing Management of Patients with Rheumatic Fever

- (i) The patient should be assessed for risk and aetiological factors such as a previous infection by A Beta haemolytic streptococcal, social economic status and living conditions. Body systems should also be assessed.

(ii) Nursing Diagnosis

- a) Activity intolerance related to athralgia, CCF, malaise.
- b) Decreased cardiac output related to dyspnoea, tachycardia, orthopnoea secondary to rheumatic carditis or CCF.
- c) Impaired physical mobility related to polyarthritis, malaise, fatigue and athralgia.
- d) High risk for injury related to involuntary movements, secondary to chorea.
- e) Pain related to polyarthritis.
- f) High risk for infection transmission related to group A Beta haemolytic streptococcal infection.
- g) High risk for altered health maintenance related to lack of knowledge concerning the need for long term prophylactic antibiotic therapy and possible disease process sequelae.

Nursing Interventions

(i) Prevention/health promotion and maintenance.

Rheumatic fever is one of the cardiovascular diseases that is preventable. Preventions is frequently classified as primary or secondary.

- a) Primary Prevention:** Involves early detection and immediate reaction of group A Beta haemolytic streptococcal pharyngitis. The nurse's role in educating the community to seek medical attention for symptoms of streptococcal pharyngitis and emphasizing the need for adequate treatment of streptococcal infection cannot be over emphasized.
- b) Secondary Prevention:** Focuses on the use of prophylactic antibiotics to prevent recurrent rheumatic fever that is monthly injection.
- c) Benzathine Penicilline G. (Panada)** for life in individual who had rheumatic carditis as children but rheumatic fever without carditis after the age of 18 years may need only 5

years of prophylactic antibiotic therapy.

(ii) Acute Intervention

Primary goals of managing a patient with acute rheumatic fever are;

- a) To control and alleviate the infecting organisms.
 - b) To prevent cardiac complications.
 - c) Relieve joint pain; fever and other symptoms.
 - d) Support the patient psychologically and emotionally.
-
- A. The nurse must administer antibiotics as ordered to treat the streptococcal infections and teach the patient that oral antibiotic therapy requires faithful adherence to the full 10 day course of therapy.
 - B. Tepid sponge bath should be given to relieve fever and antipyretics should be administered as prescribed.
 - C. Oral fluids should be encouraged if the patient is able to swallow or I.V. fluids be administered as prescribed.
 - D. Promotion of optimal rest is essential to reduce cardiac workload and diminish the metabolic need of the body.
The major role of the nurse to ensure bed rest is to assist the patient and relatives to understand the need for bed rest (amount) ordered by the physician.
 - E. The nurse should ensure the patient doesn't develop constipation, decreased muscle tone which will lead to foot drop, skin break down, excessive weight gain and boredom.
 - F. Arranging for light activities which can be done in bed is encouraged.
 - G. Relieve of joint pain or bronchaditis is an important nursing goal. Painful joints should be positioned for relieving of painful;
 - H. Proper alignment. Removal of covers from painful joints can be done with a bed cradle. Heat may be applied and salicylates may be administered to relieve joint pains.
 - I. Other measures to reduce discomfort during acute laze include:
 - a) Planning the care such that the patient is not disturbed frequently.

- b) The only reaction of chorea is prevention of injury during the period when involuntary jerking and possibly violent movements occur. Protective measures include bed rails, padding on the sides of the bed/chair to prevent injury, rest rings, thick clothing and mittens to prevent bruising of the skin, assistance during walking to prevent falling and also feeding the patients to prevent injuries from use of feeding implements/equipment.
- c) Psychological and emotional care can be more important than physical care because children and young clients are the primary clients and the heart is often viewed as the center of life. Any alteration in cardiac function may be perceived as a threat to the person's body image.
- d) To evaluate the progression or regression of the disease process the nurse should assess the patient for remission of symptoms. This includes;
- Monitoring intake and output.
 - Daily weight.
 - Resting and sleeping heart rates. This is because an accelerated sleeping heart rate in the presence of a febrile state is an indication of cardiac involvement.
 - The lab tests be monitored for return to normal ranges.

(iii) Chronic Management

Secondary preventions aim at preventing recurrence of rheumatic fever.

- a) The patient should be taught about the disease process, possible sequence and need for prophylactic antibiotics.
- b) The ongoing client education and reinforcement encourages good health practices. The patient should also be cautioned about the possibility of development of valvular heart disease.
- c) Nurse should teach the patient to seek medical attention i.e. excessive fatigue, dizziness, palpitations or dyspnea on exertion develops.
- d) The child with cardiac manifestations of the disease i.e. aortic regurgitation or mitral stenosis or insufficiency must be followed regularly with additional testing to determine the progression or regression of the lesions.

The nurse should encourage the patient and relatives to it with follow-up care even though the condition has improved.

Outcome of Rheumatic Fever

Prognosis of this disease is generally good. The degree of carditis and subsequent damage to cardiac structures are of primary concerns since they will be reflected in the levels of activity restrictions in the future with each recurrence of streptococcal infection the likelihood for potential need for valve replacement increases therefore prevention of recurrences is a high priority.

Complications of Rheumatic Fever

NOTE: Once or evidence of rheumatic fever has abated rheumatic fever doesn't occur in the absence of a new streptococcal infection. If the initial episode isn't associated with carditis there is little likelihood of subsequent damage if repeated attacks don't occur.

- (i) Chronic rheumatic carditis which results from changes in valvular structures that may occur months to years after an attack of acute rheumatic fever.
- (ii) Rheumatic endocarditis can result in fibrous tissue growth to valve leaflets chord tendinae will scarring and contractures.
- (iii) Other valves that may also be affected are the aortic and the tricuspid valves.

Rheumatic Heart Disease (Chronic Valvular Disease)

Mainly involves the mitral and aortic valves.

Chronic Valvular Disease

This condition usually occurs as a result of the inflammation of the valve cusps/flaps leading to their distortion fibrosis and calcification. The valves therefore become ineffective in performing their normal functions.

Terminologies used in Connection to Chronic Valvular Disease

Stenosis: Narrowing of the valves.

Incompetence: Inadequate closure of the valves.

Regurgitation: Back flow of blood as a result of incompetence.

Types of Chronic Valvular Disease

- (i) Mitral valve disease.
- (ii) Aortic valve disease.
- (iii) Pulmonary and tricuspid valve disease.

- a) Mitral Value Disease:** In half of the people suffering from this disease, more than 50% of them have a history of rheumatic fever. Mitral stenosis is more common than mitral incompetence although in some cases, they may occur simultaneously. The lungs become very congested causing shortness of breath and tiredness. Acute pulmonary oedema may occur. Atrial fibrillation is usually present and left heart failure gradually appears.
- b) Aortic Valvular Disease:** In this condition, aortic valve stenosis or incompetence may occur. The main result is the enlargement of the heart called cardiac hypertrophy. The left ventricular failure eventually occurs followed by right heart failure. Main symptoms are; dyspnoea, tiredness and angina.
- c) Pulmonary and Tricuspid Value Disease.** Tricuspid valve disease is usually due to rheumatic heart disease and is associated with mitral and aortic valve disease. In both cases, right heart failure occurs and back pressure is transmitted via inferior vena cava into the systemic circulation.

Management of Chronic Valvular Disease

- (i) Same as for congestive heart failure.
- (ii) Cardiac surgery may be performed i.e. valvotomy – dilatation of the valves.

Valve Replacement: There are two types i.e.

- a) Homo Graft:** Using human tissue.
- b) Hetero Graft:** Non-human tissues are used.
- c) Mechanical valves** which are plastic valves.

NOTE: There is usually risk of infection associated with the type of surgery and therefore the need for antibiotics and analgesics.

In case mechanical valve is inserted, anticoagulant therapy is indicated to prevent thrombosis around the valve. Cardiac surgery is usually a very major operation and the patient needs to be nursed and treated in the ICU for a few days and on I.V. fluids and blood transfusion as indicated.

Congestive Cardiac/Heart Failure (C.C.F)

This is a cardiovascular state in which the heart is unable to pump an adequate amount of blood to meet the metabolic needs of the tissues. C.C.F is not a disease, but a syndrome caused by a variety of pathophysiological processes.

Common Causes of C.C.F

Chronic Causes

- (i) Coronary heart/artery disease.
- (ii) Hypertensive heart disease.
- (iii) Cardio myopathy.
- (iv) Bacterial endocarditis
- (v) Rheumatic heart disease.
- (vi) Cor-pulmonare
- (vii) Anaemia

Acute Causes

- (i) Acute myocardial infarction.
- (ii) Pulmonary emboli.
- (iii) Hypertensive crisis.
- (iv) Ventricular septal defect
- (v) Disyrrhythmias.
- (vi) Thyrotoxicosis.
- (vii) Anaemia

Pathophysiology of C.C.F

- (i) C.C.F is usually manifested by biventricular failure although one ventricle.
- (ii) May precede the other in dysfunction. Normally the pumping actions of the left and right sides of the heart complement each other producing a;
- (iii) Continuous flow of blood, however, as a result of pathological conditions, one side may fail while the other side continues to function normally;
- (iv) For a period of time. Because of the prolonged strain, the functioning side of the heart will eventually fail resulting in biventricular;

Left Sided Heart Failure

This results from left ventricular dysfunction which causes blood to backup through left atrium and into the pulmonary veins. The increased pressure causes fluid extravasation from the pulmonary capillary bed into the interstitium and then the alveoli which is manifested as pulmonary congestion and oedema.

- (i) Diseases of the coronary arteries.
- (ii) Cardio myopathy.
- (iii) Aortic valvular disease.
- (iv) Hypertension.
- (v) Rheumatic heart disease resulting into mitral valve incompetence or stenosis.

When a myocardial infarction occurs, myocardial tissue is damaged and replaced by scar tissue. The scar tissue is less elastic and has poorer contractility than the undamaged myocardium. The loss of myocardial mass increases the workload on the remaining functioning tissue. If the functioning myocardium can't compensate for this loss, the volume of blood ejected from the ventricles decreases and left heart failure results. When hypertension is present, the heart must pump blood against a high arterial pressure eventually this can lead to left ventricular hypertrophy. Hypertrophic muscle has poor contractility and overtime will result in failure.

In aortic valvular heart disease, the left ventricle must contract forcefully to pump blood through the stenotic aortic valve, overtime this results in left ventricular failure. This requires an increased amount of pressure that must be generated by the left ventricle. In addition, the valve often fails to close completely and blood is regurgitated into the left ventricle.

In mitral valve disease, a similar process involving the left atrium occurs.

Right-Sided Heart Failure

Right-sided heart failure from weakened right ventricle causes venous congestion in the systemic circulation and results in peripheral oedema, hepatomegaly and splenomegaly.

The primary cause of right-sided heart failure is left sided heart failure. In this situation, left-sided failure results in pulmonary congestion and increased pressure in blood vessels of the lungs

(pulmonary hypertension).

Eventually pulmonary hypertension results in right-sided heart failure cor-pulmonale (right ventricular dilatation and hypertrophy due to pulmonary pathology) can also cause right-sided heart failure.

Distended neck veins can be seen when a client with right-sided heart failure is in a semi-recumbent position and this is due to increased pressure in the right atrium.

Other Causes of Right-Sided Heart Failure

- (i) Myocardial infarction of the right atrium and ventricle.
- (ii) Chronic pulmonary diseases i.e. pulmonary TB, bronchitis, chronic pneumoconiosis, pneumonia etc.
- (iii) Tricuspid and pulmonary valve stenosis.
- (iv) Pericarditis.

Factors Precipitating to Heart Failure

There are certain factors that can precipitate heart failure in a person with heart disease i.e.

- (i) Dysrhythmias which lead to ineffective mechanical pumping.
- (ii) Reduction or cessation of cardiac therapy either pharmacological or dietary.
- (iii) Infections either viral or bacterial.
- (iv) Emotional or physical stress.
- (v) Second type of heart disease i.e. rheumatic heart disease.
- (vi) Anaemia which causes an increased heart rate as a compensatory mechanism to maintain tissue oxygenation.
- (vii) Thyrotoxicosis which causes an increased heart rate.

Compensatory Mechanisms

C.C.F can have an abrupt onset as with acute myocardial infarction or can be a gradual/slow insidious process and thus the result of slow progressive changes. The overloaded heart results to certain compensatory mechanisms to try to maintain adequate cardiac output.

- i. **Sympathetic nervous system activation:** Because there is inadequate stroke volume and cardiac output, the baro receptor reflexes cause sympathetic nervous system activation which increases the release of epinephrine and norepinephrine. This results in an increased heart rate and myocardial contractility to raise cardiac output. This response also increases myocardial oxygen demand.
- ii. **Dilatation:** Is an enlargement of the chambers of the heart. It occurs when pressure in the left ventricle is elevated over time. The muscle fibres of the heart stretch and thereby increase their contractility force. However, this increased contractility produces greater wall tension and more myocardial oxygen is required for contraction. Therefore, dilatation is a mechanism developed to cope with increasing blood volume. After maximum hypertrophy dilatation becomes primary mechanism. Eventually it becomes inadequate because the elastic elements of the muscle fibres are overstrained. Dilatation can progress to mitral valve incompetence and regurgitation which further increases the cardiac workload.
- iii. **Renal response to heart failure:** As cardiac output falls, blood flow to the kidneys decreases causing decreased glomeruli filtration. A complex reaction begins, the kidneys release rennin which reacts with angiotensinogen to form angiotensin then this causes;
- a) The adrenal cortex to release aldosterone which causes sodium retention.
 - b) Increased vaso constriction which increases the arterial pressure.
 - c) The posterior pituitary senses the increased osmotic pressure due to sodium retention and secretes anti-diuretic hormone (ADH).
 - d) ADH increases water reabsorption in the renal tubules causing water retention.
 - e) The decreased renal blood flow also stimulates the secretion of ADH. The cycle repeats itself creating a downward spiral of a client's condition, since vaso constriction increases after load and causes an increased workload on the heart.
 - f) **Hypertrophy:** In chronic congestive heart failure, hypertrophy is an increase in the muscle mass and the cardiac wall thickens due to overwork and strain. It occurs slowly because it takes time for muscle tissue to develop. As myocardial mass increases, the need for additional blood and oxygen grows. This cannot be met in the client with heart disease.

Clinical Manifestations

The clinical manifestations of chronic C.C.F depend on:

- a) Age
- b) Underlying type
- c) Extent of heart disease
- d) Which ventricle is failing to pump blood effectful.

These manifestations include:

- | | | |
|------------------|--------------------------|------------------|
| i) Fatigue | v) Nocturia | viii) Chest pain |
| ii) Dyspnea | vi) Skin changes | ix) Cough |
| iii) Tachycardia | vii) Behavioural changes | |
| iv) Oedema | | |

Fatigue: Is one of the earliest symptoms of chronic C.C.F. The client notices fatigue after activities that normally aren't tiring. The fatigue is due to impaired circulation and oxygenation of tissues. It is sometimes described as 'sick fatigue' because of the decreased amount of blood reaching the musculo skeletal system.

Dyspnea: Is a common sign of chronic C.C.F. It is caused by increased pulmonary pressures secondary to interstitial and alveolar oedema. This results in fluid poor gaseous exchange because of fluid in the alveolar. The shortness of breath makes the client short of air hunger that prompts rapid shallow respirations. Dyspnea can occur with mild exertion or at rest. Othopnoea is shortness of breath that occurs when a client is in a recumbent position.

Tachycardia: Because cardiac output is diminished there is an increased sympathetic nervous system stimulation to compensate for low output (It is important to remember that cardiac output = stroke volume x heart rate). If the stroke volume decreases, the heart rate increases to maintain the cardiac output.

Oedema: Is a common sign of C.C.F. It may occur in the legs (peripheral oedema), liver (hepatomagaly), abdominal cavity (ascites), lungs (pulmonary oedema and pleural effusion). If the client is bedfast, sacral oedema is mostly likely to develop pressing the oedematous skin with

a finger may leave a transient indelitation (pitting oedema). The development of dependent oedema and or a sudden weight gain 2kg or more is often indicative of exacerbated congestive failure.

Nocturea: A person with chronic C.C.F will have decreased cardiac output, impaired renal perfusion and decreased urinary output during the day. However, when the person lies down at night, fluid movement from the interstitial spaces back into the circulatory system is enhanced. This causes increased renal blood flow and diuresis. The client may complain of having to void 6 or 7 times during the night.

Skin Changes: Because tissue capillary oxygen extractions increased in a person with chronic C.C.F, the skin appears dusky, it is also called cold and diaphoretic to touch. The peripheral vaso constriction that occurs to shunt blood to the vital organs is a minor compensatory mechanism in chronic C.C.F.

Behavioural Changes: Cerebral circulation may be impaired with chronic C.C.F especially in the presence of more widespread arteriosclerosis. The client or family may report unusual behavior including restlessness, confusion and decreased attention span or memory. These behavioural changes occur most often at night possibly because the client is experiencing more stimulation than during the day.

Chest Pain: In the presence of arteriosclerosis, C.C.F can precipitate chest pain because of decreased coronary perfusion from decreased cardiac output and increased myocardial work. Angina type of pain may accompany C.C.F whether it is acute or chronic. Others will be as per the side the failure has occurred.

Summary of the Clinical Manifestation

Right-Sided Heart Failure

- (i) Peripheral oedema weight gain.
- (ii) Oedema of dependent body parts (sacrum, anterior tibia and pedal).
- (iii) Ascites
- (iv) Anasarca (massive generalized body oedema).
- (v) Jugular vein distention.
- (vi) Liver engorgement (hepatomegaly) splenomegaly

Left-Sided Heart Failure

- (i) Increased heart rate.
- (ii) Left ventricular hypertrophy.
- (iii) Poor oxygen exchange.
- (iv) Pulmonary oedema (rales).
- (v) Dyspnoea shallow up to 32 to 40/min.
- (vi) Orthopnoea.
- (vii) Paroxysmal nocturnal dyspnoea.
- (viii) Cough which dry hacking is caused by alveolar irritation from fluid accumulation.
- (ix) Heart sound will be heard from vibrations of the ventricle wall due to resistance to ventricular filling.

Diagnosis of C.C.F

The primary goal in the diagnosis is to determine the underlying cause of heart failure. Diagnostic measures to assess the degree of heart failure include:

- (i) History and physical examination.
- (ii) Chest x-ray film.
- (iii) ECG.
- (iv) Exercise stress testing.
- (v) **Echocardiograph:** To measure the size of cardiac chambers and to assess ventricular function.
- (vi) Cardiac catheterization and angiography are useful in detecting the underlying heart disease.
- (vii) Haemodynamic monitoring via a pulmonary artery catheter which provides for a means of directly assessing cardiac function.

Classifications of Persons with C.C.F Based on the Persons Tolerance to Physical Activity

(By New York Heart Association)

- **There is class one:** There is no limitation on physical activity and ordinary physical activity not resulting in symptoms.
- **Class II:** Slight limitation on physical activity, no symptoms at rest but symptoms possible with ordinary physical activity.
- **Class III:** There are more severe limitations. Client may be comfortable at rest but clinical manifestations will show with slight physical activities.
- **Class IV:** Inability to carry on any activity without producing symptoms and symptoms are also possible at rest.

Therapeutic Management

Treatment of underlying cause is one of the most important goals of reaction for C.C.F. For example;

- (i) Dysrhythmias have precipitated the failure they should be treated accordingly.
- (ii) Hypertension is the cause antihypertensive drugs should help.
- (iii) Valvular defects are the cause surgery is required.
- (iv) **Oxygen therapy:** Oxygen is given to the patient at 2 to 6 litres per minute. This is because in a person with C.C.F, oxygen circulation of the blood is reduced because the blood is not adequately oxygenated at the lungs. Therefore, administration of oxygen improves circulation and assists greatly in meeting tissue oxygen needs. Thus oxygen therapy helps to relieve dyspnea and fatigue.
- (v) **Rest:** Physical and emotional rest allows the client to conserve energy and decreases the need for additional oxygen. The degree of rest recommended depends on the severity of heart failure. For example, a patient with severe C.C.F needs to be on complete bed rest while the one with mild C.C.F can be ambulatory with restriction of strenuous activities.

Pharmacological Therapy

- (i) **Digitalis Preparations**
 - a) This increases the;
 - Force/strength of cardiac contraction.

- b) They also; decrease the conduction speed within the myocardium and show the heart rate. This action provides more complete emptying of the ventricles thus diminishing the volume remaining in the ventricles during diastole cardiac output increases because of an increased stroke volume from improved contractility.

Dosage

Usually 0.25mgs d or od. At times the patient may be given 0.5 to 1mg I.V. or I.M. then followed by 0.25mgs. For children and old patients, the dosage is reduced to 0.025mgs daily.

- (ii) **Diuretics:** These are used in heart failure to mobilize oedematous fluid, reduce pulmonary venous pressure.

To reduce preload

If excess vascular volume is excreted, blood volume returning to the heart can be reduced and thus improved cardiac function. Thiazides are usually the first choice, because they are useful in treating oedema secondary to C.C.F as well as in controlling hypertension.

Example: Lasix (frusemide: Dose 40 to 80mgs depending on the severity of oedema I.V., I.M. or orally.

- (iii) **Vaso Dilator Drugs:** These reduce systemic vascular resistance and pulmonary and peripheral venous pressure. This increases left ventricular preload thus myocardial function is enhanced and myocardial oxygen demand is lessened. These drugs include:

- a) Angiotensin converting enzymes inhibitors.
- b) Calcium channel blockers etc.

- (iv) **For the Cough:** The patient is given cough mixture or mist or expectorant

Nutritional Considerations

- i) The oedema of chronic C.C.F is often treated by dietary restriction of sodium. A diet for a client with C.C.F should have 2g of sodium.
- ii) The diet such as milk, cheese, bread, cereals, vegetables, canned foods must be eliminated from the patient's diet.

Nursing Management

- a) Activity intolerance related to fatigue secondary to cardiac insufficiency, pulmonary congestion and inadequate nutrition (dyspnoea, weakness, fatigue, increase or decrease in pulse on exertion).

Nursing Interventions

- (i) Have the client to rest in bed or chair when tired.
- (ii) Provide emotional and physical rest.
- (iii) If the patient is in bed, teach leg exercises to prevent phlebothrombosis.
- (iv) Assess the patient daily for dyspnoea, fatigue and pulse rate to determine the level of activity that can be performed.
- (v) Provide frequent small feeds instead of 3 large meals per day.
- (vi) Teach client about expenditure of energy on various activities.
- b) Sleep pattern disturbance related to nocturnal dyspnoea and inability to assume favoured sleep position (inability to sleep through the night).

Nursing Interventions

- (i) Explain aetiology of nocturnal dyspnoea.
- (ii) Explore with the patient alternative positions of comfort to relieve dyspnoea.
- c) Fluid volume excess related to heart/pump failure (oedema, dyspnoea on exertion).

Nursing Interventions

- (i) Evaluate degree of peripheral oedema and measure abdominal girth daily.
- (ii) Administer digitalis agents and diuretics as presented daily.
- (iii) Assess intake and output 8 hourly.
- (iv) Weigh the client daily.
- (v) Observe manifestations of hypovolaemia.
- (vi) Provide sodium restricted diet as ordered.

- d) High risk for impaired skin integrity related to oedema or immobility (oedema, taut shiny skin, impaired mobility).

Nursing Interventions

- (i) Handle oedematous skin gently.
 - (ii) Pad bony prominence.
 - (iii) Assess oedematous areas every shift for skin breakdown.
- e) Impaired gas exchange related to excess preload, mechanical failure or immobility (increased respiratory rate, dyspnoea on exertion).

Nursing Interventions

- (i) Elevate head of bed to fallulow's position.
 - (ii) Support client's arms with pillows.
 - (iii) Use food board for legs.
 - (iv) Administer oxygen by naso canula.
 - (v) Auscultate for lung and heart sound 4 hourly.
- f) Anxiety related to dyspnoea or perceived threat of death (restlessness, irritability, expression of feelings of live threat).

Nursing Interventions

- (i) Assess facial expression and behavior for feeling of apprehension.
 - (ii) Allow the patient to ask questions.
 - (iii) Promote sense of security by answering calls promptly from the patient/relative.
 - (iv) Explain all procedures due to the patient.
 - (v) Assess past methods of coping and assist in adapting these methods to present lifestyle limitations.
 - (vi) Demonstrate calm behavior with the patient.
- g) Ineffective individual coping related to alterations in lifestyle possible inability to use past coping methods or perceived loss of control (use of ineffective coping behaviours such as shouting, blaming, anger, withdrawal, social isolation, increased dependency).

Nursing Interventions

- (i) Teach client about disease process and altered physiological function.
 - (ii) Encourage patient to adopt lifestyle compatible with degree of heart impairment.
 - (iii) Assist client and family in planning necessary changes.
 - (iv) Encourage the client who seems discouraged or to plan and participate in own plan of care.
 - (v) Question client regarding concerns.
 - (vi) Support the coping strategies and suggest alternate strategies to replace ineffective ones.
- h) Self care deficit (total related to dyspnoea and fatigue) inability to perform part or all of the activities of daily living.

Nursing Interventions

- (i) Assist clients/patients with all activities of daily living as needed.
 - (ii) Assure patient of your willingness to assist with personal care.
 - (iii) Advise family of patient's fluctuating inability regarding self care activities.
- i) Altered health maintenance related to lack of knowledge regarding signs and symptoms of C.C.F, proper diet and medications (lack of adherence to low sodium diet, questioning of disease, diet and medications).

Nursing Interventions

- (i) Teach the patient manifestations to report including shortness of breath at rest, swelling of ankles feet or abdomen, nausea or vomiting, loss of appetite, weight gain of 1 to 2kgs in a 2 day period, frequent urination, persistent cough, changes in heart rate more than 20 or less than 20 + or – 20 beats.
- (ii) Instruct the client on dietary restriction and medication regime.

NOTE: Many patients with C.C.F do not experience an acute episode, if they do, they are usually initially managed in a critical care unit and later transferred to a general unit when their conditions have stabilized.

Chronic Management

C.C.F may be a chronic illness for most persons and important nursing responsibilities are:

- i) Educating the patient about the physiological changes that have occurred.
- ii) Assisting the patient to adapt to both the physiological and psychological changes. It must be emphasized to the client that it is possible to live productively with his health problem.
- iii) Diet and weight management.
- iv) Drug therapy.
- v) Rest.

Complications of C.C.F

- (i) **Pulmonary Oedema:** Which is a term used to refer to an acute life threatening situation in which the lung alveolar become filled with serous sanguinous fluid.
- (ii) **Pleural Effusion:** Resulting from increasing pressure in the pleural capillaries. A transudation of fluid occurs from these capillaries into the pleural space.
- (iii) **Left Ventricular Thrombus:** With acute or chronic C.C.F, the enlarged ventricle left and poor cardiac output combines to increase the chance of thrombus formation in the left ventricle.
- (iv) **Hepatomegaly:** C.C.F can lead to severe hepatomegaly. The liver lobules become congested with venous blood. The hepatic congestion leads to impaired liver function – functionally live cells die, fibrosis occurs and liver cirrhosis can develop.
- (v) **Weight Changes:** Initially there may be a progressive weight gain due to fluid retention. The client with C.C.F has an increased metabolic rate. Abdominal fullness from ascites and hepatomegaly frequently causes anorexia and nausea. Late the patient decreases in weight, the patient becomes too sick to eat and there is decreased oxygen and nutrients transported to the tissues.
- (vi) **Decompensated Heart Failure:** When the compensatory mechanisms of dilatation, hypertrophy and tachycardia function to provide adequate output to maintain tissue oxygenation. The patient has compensated heart failure when these mechanisms can no longer assist the heart in maintaining cardiac output, the patient has decompensated heart failure and this state is very fatal.

Summary of Nursing Management

- (i) Complete bed rest on sitting up position.
- (ii) Oxygen administration.
- (iii) Administration of medications as prescribed, observe effects and side effects.
- (iv) Frequent total patient assessment depending on the condition (wholly). Any abnormalities be reported immediately (weight, TPRIBP, pulse, urine, skin).
- (v) **Patients comfort:** This include body comfort, linen and the environment, elimination.
- (vi) **Dist:** Should be salt free. Roughage be included to avoid constipation and small frequent fees at intervals and well balanced.
- (vii) Share health messages about the disease and also diet.
- (viii) Physical and emotional support.
- (ix) When patient gets well, discharge home and follow-up.

Infective Endocarditis (Bacterial)

This is an infection of the endocardial surface with micro organisms present in the lesions. The endocardium and the inner layer of the heart is continuous with the heart values therefore, inflammation from infective endocarditis frequently affects the heart values.

Occurrence: The frequency is now seen in older adults, clients after prosthetic value placement and intervenous drug abusers.

Prognosis: The prognosis has changed with introduction of antibiotics because before then, infective endocarditis was almost always fatal. The mortality rate is now 20 to 40%.

Predisposing Conditions

(i) Cardiac conditions;

- | | |
|--|-------------------------------|
| a) Prosthetic heart values (artificial) | f) Rheumatic heart disease |
| b) Congenital heart disease | g) Degenerative heart disease |
| c) Mitral value prolapsed | h) Valvular lesions |
| d) Heart murmurs | i) Cardiac surgery |
| e) Idiopathic hypertrophic sub-aortic stenosis | |

(ii) Immune Suppressive Conditions i.e.

- a) Cancer
- b) Severe burns

(iii) Immune suppressive therapy i.e. radiotherapy.

(iv) Invasive procedures i.e.

- a) Peripheral arterial venous fistula i.e. that used in chronic haemour dialysis.
- b) In dwelling intravenous or intra-arterial catheters.
- c) Intravenous drug abuse.
- d) Invasive diagnostic procedures.
- (v) Alcoholism

Classification of Infective Endocarditis

(i) Sub-acute

(ii) Acute

- a) Sub-Acute:** This form has a longer clinical of more insidious onset with less toxicity and the causative organism is usually of low virulence. Most often streptococcus viridian.
- b) Acute Form:** Has a shorter clinical course with a more rapid onset increased toxicity and a more pathogenic/virulent causative micro organism that is staphylococcus aureous.

Causes

Initially it was being caused by bacteria but of late micro organisms such as fungi, Chlamydia and rickettsial have been identified.

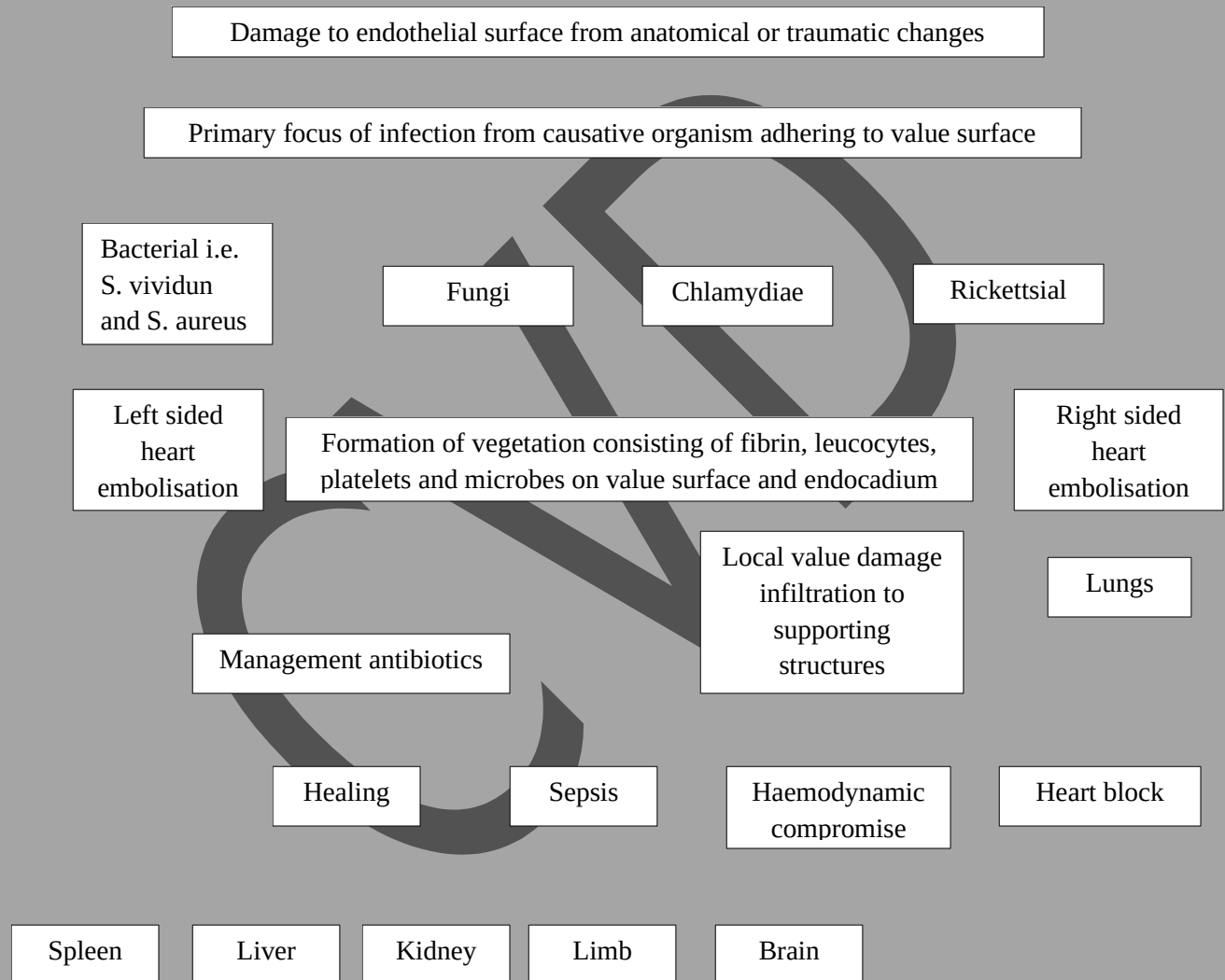
NOTE: These micro organisms get entry into the heart because of surgical interventions, intravenous injections and invasive diagnostic procedures.

Pathophysiology

The vegetation form that is the primary lesions of infective endocarditis consist of fibrin, leucocytes, platelets and microbes that adhere to the valve surface or endocardium. The loss of these fragile vegetations into the circulation results in embolisation. In left sided heart

vegetations systemic embolisation occurs progressing to the organs particularly the kidney, spleen and brain and there may be limb infarction. Right sided heart lesions embolise to the lungs. The infarction may be spread locally to cause damage to the valves or to their supporting structures. The resulting valvular incompetence and eventual invasion of the myocardium in the ongoing disease results in C.C.F, generalized myocardial dysfunction and continued sepsis.

Sequence of Events in Infective Endocarditis



Sources of Infections

Organism

Situation/Origin

- | | |
|-------------------------------------|--|
| (i) Streptococcus viridians | Dental procedures |
| (ii) Streptococcus bovis | GIT malignant disorders |
| (iii) Enterococcus species | GIT and genital urinary procedures |
| (iv) Staphylococcus aureus | I.V. drug use, cardiac surgery, nosocomial infections (URTI), parenteral therapy, prosthetic valves. |
| (v) Pseudomonas aeruginosa | I.V. drug use and surgery |
| (vi) Candida, aspergillus organisms | Immuno compromised persons |

Clinical Manifestations

These aren't quite specific as there can be involvement of multiple organ systems.

- (i) Fever in more than 90% of patients.
- (ii) Chills
- (iii) Weakness
- (iv) Malaise
- (v) Fatigue
- (vi) Anorexia
- (vii) Arthralgia
- (viii) Myalgia
- (ix) Backache
- (x) Abdominal discomfort
- (xi) Weight loss
- (xii) Headache
- (xiii) Clubbing of fingers

The onset of a new murmur is also frequently noted. C.C.F occurs up to 80% of patients with aortic valve endocarditis and in approximation 50% of patients with mitral valve endocarditis. Apart from the above general manifestations there is;

Vascular Manifestations

- (i) Splinter haemorrhages that may occur in the nail beds.
- (ii) Osler nodes – painful, tender, red or purple pea size lesions. They may be found on finger tips or toes.
- (iii) Janeway lesions – Flat, painless, small red spots found on the palms or soles.
- (iv) On fundoscopic examination, there may be retinal haemorrhagic lesions called Roth's spots.

Clinical Manifestations Secondary to Embolisation

- a) Embolisation to the spleen may result into left upper quadrant pain and splenomegaly. There will be local tenderness and abdominal rigidity.
- b) Embolisation to the kidneys may cause pain to the flank, haematuria, asotemia and glomerulonephritis.
- c) Emboli may lodge in small peripheral blood vessels and cause gangrene.
- d) Embolisation to the brain may cause neurological problems i.e. hemiplegia, ataxia, aphasia and change in level of consciousness.
- e) Pulmonary emboli may occur in right sided lesions.

Diagnostic Procedures

- (i) History taking: Obtaining the patient's recent health history is important in assessing infective endocarditis. Queries should be made regarding recent dental, urological, surgical or gynecological procedures, including normal or abnormal obstetrical deliveries. Previous history of heart disease, recent cardiac catheterization, I.V. infusions and catheters as well as skin, respiratory or urinary tract infections should be documented.

- (ii) Laboratory investigations: Blood for culture and sensitivity, WBC, ESR which will be raised, urine for haematuria (microscopic).
- (iii) Chest x-ray performed to check presence of C.C.F.
- (iv) Echocardiogram performed to detect the presence of valvular vegetations or abnormal valve leaflet movement resulting from growth of vegetations.
- (v) Cardiac catheterizations: To detect the degree of valvular insufficiency and to determine the involvement of more than one valve.

Management

A. Medical Management

a) Prophylactic Treatment

Clients with various anatomical abnormalities of the heart or great vessels are at great risk of contracting endocarditis. Prophylactic treatment is recommended for patients with some common cardiac heart conditions before they undergo certain surgical conditions. Specific antibiotic regimens are recommended for dental, respiratory tract, gastro intestinal, genitor urinary procedures.

b) Therapeutic Management

Accurate identification of the infecting organism is the key to successful management. The reaction of infective endocarditis usually requires 4 to 6 weeks of parenteral single or combination of antibiotic therapy.

Drugs given depending on the situation

Clinical Situation

Empirical Situation

Acute

Sub-acute

Antibiotic Regiment

Penicillin, Ampicillin and Gentamycin

Ampicillin and Gentamycin

Other drugs that can be used are penicillin G, streptomycin with fungal infection can be given Amphotericin B. During the reaction periodic monitoring of the patients, antibiotic serum levels

must be performed and subsequent blood cultures may be done to evaluate the effectiveness of the treatment.

B. Nursing Management

a) Prevention/Health Maintenance and Promotion

Assessment of the client's history and understanding of the disease process are crucial for planning and implementing appropriate health maintenance strategies. It is important that these at risk of developing endocarditis are identified and educated to avoid nosocomial infections, fatigue to maintain good oral hygiene, to visit the dentist at regular intervals and to take prophylactic antibiotic therapy before any invasive procedure.

b) Acute Intervention

Patient with infective endocarditis require hospitalization for 2 to 4 weeks. Physical assessment and history taking are done. In particular, there should be frequent monitoring of body temperature as fever is one of the common early signs. Measures to reduce fever are employed. Bed rest is necessary when there is fever or complications such as heart done are present. Otherwise, the patient may be ambulant and perform moderate activities. Heart sounds should be assessed with vital signs to detect a change in the character of the cardiac murmur and presence of extra diastolic sounds. Arthralgia is a common problem and may involve multiple joints and be accompanied by myalgia. The patient should be assessed for joint tenderness, range of motions and muscle tenderness. The oral mucousa, conjunctiva, upper chest and lower extremities should be examined for petechial (bleeding spots of blood under skin). A general assessment of systems should be completed to facilitate recognition of haemodynamic and embolic complications.

Laboratory data should be monitored to determine the effectiveness of long term high dose antibiotic therapy received by the client. The I.V. line should be monitored for patency and antibiotics given as scheduled monitor the side effects of these antibiotic or drugs given.

To prevent consequences from immobility, the patient should wear anti-embolism stockings, perform range of motion exercises and turn, cough and deep breathe every 2 hours. The patient may experience anxieties and fear associated with the illness, provide support during this period and employ measures to reduce fears and anxieties.

c) Chronic Management

This focuses on the education of the patient regarding the nature of the disease and on the reduction on the risk of re-infection. The patient is taught about symptoms that may indicate recurrent infections such as fever, fatigue, malaise, chills and the importance of seeking medical assistance if any of these symptoms occur. The patient needs to be instructed about the need for prophylactic antibiotic therapy before any invasive procedure is performed to prevent reoccurrence. The nurse must explain to the client the relationship of follow-up care, good nutrition and early reaction of common infections to maintain good health.

CVD