CARDIOVASCULAR NURSING

CONGESTIVE CARDIAC FAILURE /HEART FAILURE

Definition

This is an abnormal clinical syndrome involving impaired cardiac pumping or filling.
CCF is associated with many types of cardiovascular diseases such as hypertension, coronary artery disease and myocardial infection.

AETIOLOGY /CAUSES

- Coronary artery disease is myocardial infarction.
- Advancing age – primarily a disease of older adults
- Hypertension
- Diabetes mellitus
- Cigarette smoking
- Obesity and high serum cholesterol
- Rheumatic heart disease
- Hyperthyroidism
- Valvular disorders i.e. mitral stenosis
- Myocarditis

Precipitating factors of heart failure

Anaemia – reduced oxygen carrying capacity of the blood stimulating increase in cardiac output to meet tissue demands.

Infections – causes increased oxygen demand of tissues, thereby stimulating increased cardiac output

Thyrotoxicosis – changes the tissue metabolic rate, increases heart rate and workload of the heart

Dysrhythmias – causes decreased cardiac output and increases workload and oxygen requirements of myocardial tissues.

Interference with the normal mechanism regulating cardiac output may cause heart failure.
Cardiac output depends on pre-load, after load, myocardial contractility and heart rate.

Pre-load is the volume of blood in the ventricles at diastole before the next contraction.
Preload determines the amount of stretch placed on myocardial fibers.
Afterload is the peripheral resistance against which the left ventricle must pump.
Afterload is affected by the size of the ventricle, wall tension and arterial blood pressure.
Cardiac output is the amount of the blood pumped by each ventricle in 1 minute
It is calculated by multiplying the amount of blood ejected from the ventricle with each heartbeat, the stroke volume (sv), by the heart rate (HR) per minute.

CO = SV x HR

CLASSIFICATION OF HEART FAILURE

Heart failure is classified as follows:

a) Systolic failure

- Results from an inability of the heart to pump blood effectively
- It is caused by impaired contractile function e.g. myocardial infarction, valvular heart disease, hypertension etc.
- The left ventricle in systolic dysfunction loses its ability to generate enough pressure to eject blood forward through the aorta.

b) Diastolic failure

This is the inability of the ventricles to relate and fill during diastole

c) Mixed systolic and diastolic failure

Systolic & diastolic failure of mixed origin is seen in diseases states such as dilated cardio myopathy

Dilated cardiomyopathy is a condition in which poor systolic function is further compromised by dilated left ventricular walls that are unable to relax

PATHOPYSIOLOGY OF HEART FAILURE

Heart failure results from an inability of the heart to pump blood effectively

The left ventricle loses its ability to generate enough pressure to eject blood forward through the aorta.

Overtime, the left ventricle becomes dilated and hypertrophied

This leads to a decrease in the left ventricular ejection fraction

The right and the left ventricles have the inability to relax and fill during diastole

Decreased filling of the ventricles will result in decreased stroke volume and cardiac output

Compensatory mechanisms

In heart failure, the overloaded heart resorts to compensatory mechanisms to try to maintain adequate cardiac output.

The main compensatory mechanisms include:

a) Sympathetic nervous system activation

In response to an inadequate stroke volume and cardiac output, there is increased sympathetic nervous system activation, resulting in the increased release of catecholamines (epinephrine and norepinephrine)

This results in increased heart rate, increased myocardial contractility and peripheral vasoconstriction

b) Neuro hormonal response

As the cardiac output falls, blood flow to the kidney decreases
This is sensed by the juxtaglomerular apparatus in the kidneys as decreased volume
In response, the kidneys release renin which converts angiotensinogen to angiotensin I
Angiotensin I is subsequently converted to angiotensin II by a converting enzyme made in the lungs.
Angiotensin II causes the following:

- The adrenal cortex to release aldosterone, which results in sodium and water retention
- Increased peripheral vasoconstriction, which increases blood pressure.
- This response is known as the renin-angiotensin –aldosterone system cascade.
- Low cardiac output causes a decrease in cerebral perfusion pressure.
- The posterior pituitary gland then secretes antidiuretic hormone (ADH), also called vasopressin
- ADH increases water reabsorption in the renal tubules, causing water retention and therefore increased blood volume
- As a result, blood volume is increased in a person who is already volume overloaded.

c) Dilation

Dilation is an enlargement of the chambers of the heart
It occurs when pressure in the heart chambers is elevated overtime
The muscle fibers of the heart stretch in response to the volume of blood in the heart at the end of diastole
The degree of stretch is directly related to the force of the contraction (systole)
This increased contraction initially leads to increased cardiac output and maintenance of arterial blood pressure and perfusion.
Dilation starts as an adaptive mechanism to cope with increasing blood volume
Eventually this mechanism becomes inadequate because the elastic elements of the muscle fibers are overstretched and can no longer contract effectively, thereby decreasing the cardiac output.

d) Hypertrophy

Hypertrophy is an increase in the muscle mass and cardiac wall thickness in response to overwork and strain.
It occurs slowly because it takes time for this increased muscle tissue to develop.
Hypertrophy follows persistent/chronic dilation and thus further increases the contractile power of the muscle fibers.
This will lead to an increase in cardiac output and maintenance of tissue perfusion.

NB: Cardiac compensation occurs when compensatory mechanisms succeed in maintaining an adequate cardiac output that is needed for tissue perfusion.
Cardiac decompensation occurs when these mechanisms can no longer maintain adequate cardiac output and inadequate tissue perfusion results.

TYPES OF HEART FAILURE

1. Left – Sided Failure

Results from left ventricular dysfunction.

Left ventricular dysfunction prevents normal blood flow and causes blood to back up into the left atrium and into the pulmonary veins

The increased pulmonary pressure causes fluid movement from the pulmonary capillary bed into the interstitium and then the alveoli which manifests as pulmonary congestion and edema.

2. Right Sided Failure

Right sided failure causes a backup of blood into the right atrium and venous circulation.

Venous congestion in the systemic circulation results in jugular venous distention, hepatomegaly, splenomegaly and peripheral edema.

CLINICAL MANIFESTATIONS OF CCF

a) Fatigue

Fatigue is caused by decreased cardiac output, impaired perfusion to vital organs, decreased oxygenation of the tissues and anemia.

Anaemia can result from poor nutrition, renal disease or drug therapy i.e. (Angiotensin – converting enzyme inhibitors (ACE)

b) Dyspnea

- Dyspnea is shortness of breath
- It is caused by increased pulmonary pressures due to interstitial and alveolar edema.
- Dyspnea can occur with mind exertion or at rest
- Orthopnea is shortness of breath that occurs when the patient is in a recumbent position
- Paroxysmal nocturnal dyspnea is shortness of breath that occurs when the patient is a sleep
- It is caused by the reabsorption of fluid from dependent body areas when the patient is recumbent. The patient awakens in a panic, has feelings of suffocation, and has a strong desire to seek relief by sitting up
- The patient develops adaptive behavior such as sleeping with two or more pillows to aid breathing

c) **Tachycardia**

This is increased heart rate

Because of diminished cardiac output, there is increased sympathetic nervous system stimulation, which increases heart rate.

d) Edema

This is accumulation of fluid in the tissues.

It may occur in dependent body areas (peripheral edema), liver (Hepatomegaly), abdominal cavity (ascites) and lungs (pulmonary edema and pleural effusion)

If the patient is in bed, sacral and scrotal edema may develop

Pressing the edematous skin with the finger may leave a transient indentation (pitting edema)

e) **Nocturia**

Heart failure with decreased cardiac output causes impaired renal perfusion and decreased urinary output during the day. However, when the patient lies down at night, fluid movement from interstitial spaces back into the circulatory system is enhanced. This causes increased renal blood flow and diuresis.

The patient may complain of having to void 6 or 7 times during the night.

f) Skin changes

The skin of people with heart failure may appear dusky.

This is because tissue capillary oxygen extraction is increased.

The skin may also be cool and damp to the touch

The lower extremities are shiny and swollen, with diminished or absent hair growth.

g) Behavioural changes

Cerebral circulation may be impaired with chronic heart failure due to decreased cardiac output and poor gas exchange

The patient may report unusual behavior such as restlessness, confusion, and decreased attention span or memory

h) Chest pain

Chest pain occurs in heart failure due to decreased coronary perfusion from decreased cardiac output, and increased myocardial work.

i) Weight changes

Weight gain is caused by fluid retention. However, abdominal fullness from ascites and hepatomegaly causes anorexia and nausea

This causes muscle and fat loss

The actual weight loss may not be apparent until after edema subsides

COMPLICATIONS OF HEART FAILURE

a) Pleural Effusion

Results from increasing pressure in the pleural capillaries

The movement of fluids occurs from pleural capillaries into the pleural space

b) Dysrhythmias

Chronic heart failure causes enlargement of the chambers of the heart

This enlargement (stretching of the atrial and ventricular tissues) may causes an alteration in the normal electrical pathway, especially in the atria.

Numerous sites in the atria fire spontaneously and rapidly (atrial fibrillation), and the organized spread of atrial depolarization (contraction or systole) no longer occurs

Atrial fibrillation also promotes thrombus formation within the atria which may break loose and form emboli

This puts the patients at risk for developing stroke

c) Left Ventricular Thrombus

With heart failure, the enlarged left ventricle and decreased cardiac output combine to increase the chance of thrombus formation in the left ventricle
The thrombus decreases left ventricular contractility, decreases cardiac output thus further worsening the patient's perfusion
Thrombus formation also puts the patient at risk for stroke

d) Hepatomegaly

With heart failure, the liver lobules become congested with venous blood
The hepatic congestion leads to impaired liver function
Eventually the liver cells die, fibrosis occurs and cirrhosis can develop

e) Renal failure

Decreased cardiac output that occurs in heart failure results in decreased perfusion of the kidneys and can lead to renal insufficiency or failure

MANAGEMENT OF HEART FAILURE (NURSING MANAGEMENT)

Goals of treatment

The main goals in the treatment of chronic heart failure are: -

- To treat the underlying cause and contributing factors
- Maximize cardiac output
- Provide comfort to alleviate symptoms.
- Improve ventricular function
- Improve quality of life
- Preserve target organ function
- Improve the risks of mortality and morbidity

MANAGEMENT INCLUDE THE FOLLOWING

- Place the patient with heart failure in a high fowler's position. This position helps to increase the thoracic capacity, allowing for improved ventilation
- Administer oxygen therapy via nasoprongs or facemask. This is to improve saturation and assists in meeting tissue oxygen needs thereby relieving dyspnea and fatigue
- Monitor viral sights every hour to monitor patients progress.
- Encourage alternate rest and activity periods to reduce cardiac workload and conserve energy
- Monitor for cardiac dysrhythmias to detect cardiac decompensation
- Administer prescribed diuretics as appropriate i.e. furosemide to treat hypervolemia
- Monitor potassium levels after diuresis to detect electrolyte loss
- Monitor intake & output to assess fluid status
- Monitor changes in peripheral edema to assess response to treatment
- Assess patient for depression and anxiety and initiate treatment plans if appropriate

- Administer vasodilators as prescribed e.g. nitroglycerin to increase myocardial oxygen supply
- Monitor blood pressure frequently (every five to 10 minutes) to avoid symptomatic hypotension.
- Give patient a low sodium diet to prevent oedema
- Restrict fluid intake in moderate and severe heart failure with renal insufficiency
- Weigh patient daily to monitor fluid retention.

DRUGS USED IN HEART FAILURE

Drug therapy is essential in treating acute heart failure. They are as follows: -

i. Diuretics

Loop diuretics e.g. furosemide (Lasix) is administered. Mechanism of action of furosemide is that it acts in the kidneys to stimulate excretion of sodium chloride and water.
This decreases venous return (preload) thereby reducing the amount of volume returned to the left ventricle during diastole
By decreasing venous return to the left ventricle and thereby reducing pre-load the left ventricle may contract more efficiently and improve cardiac output.
This increases left ventricular function, decreases pulmonary vascular pressures and improves gas exchange**. side effects** – reduction in serum potassium levels, ototoxicity.

ii. Vasodilators

Vasodilators reduce circulating volume by decreasing pre-load
They also increase coronary artery circulation by dilating the coronary arteries
Thus they reduce pre-load and increases myocardial oxygen supply
They therefore improve myocardial contraction, increases cardiac output and reduces pulmonary congestion. Examples of vasodilators are nitroglycerin (given iv, sodium nitroprusside (Nipride) (given iv). nursing responsibilities when giving vasodilators are:
Assess blood pressure before administration and continuously during administration
Too rapid rate of I.V administration can reduce blood pressure too quickly therefore, it should be administered slowly

Side effects of vasodilators

- Headache
- Nausea
- Dizziness
- Dyspnea
- Blurred vision
- Sweating
- Restlessness
- Hypotension

iii. Angiotensin – converting enzyme inhibitors (ACE)

These drugs block the renin – Angiotensin – Aldosterone system

E.g. of ACE are captopril, benazepril, enalapril
The conversion of angiotensin 1 to angiotensin II requires the presence of angiotensin converting enzyme (ACE)
ACE inhibitors act by blocking this enzyme resulting in decreased levels of angiotensin II
As a result, plasma aldosterone levels are also reduced
The ACE inhibitors are considered neurohormonal blocking agents
They increase cardiac output

Side effects of ACE inhibitors
Hypotension
Hyperkalemia
Angioedema (allergic reaction involving edema of the face and airways)
Renal insufficiency

Angiotensin II Receptor Blockers
These drugs prevent the vasoconstrictor and aldosterone secreting effects of angiotensin II by binding to the angiotensin II receptor sites
E.g. of Angiotensin II receptor blockers are losartan and valsartan

Aldosterone Antagonists
These drugs block the neuro hormonal effects of aldosterone on the heart blood vessels
They are also potassium sparing diuretics that promote sodium and water excretion while retaining potassium
These effects occur because these drugs bind to recepors at the aldosterone dependent sodium – potassium exchange site in the distal renal tubules

Side effects of aldosterone antagonists
Gynecomastia in males (enlargement of breast tissue in males),
Hyperkalemia (high potassium levels in the blood)
Headache, dizziness

Vasodilators
Examples of vasodilators are nitrates (nitroglycerin, hydralazine, isosorbide dinitrate.
Vasodilators cause vasodilation of the coronary arteries
Side effect of nitrates are erectile dysfunction in men

Beta – Adrenergic blockers
Examples of B-blockers are metoprolol, bisoprolol, carvedilol
They directly block the negative effects of the sympathetic nervous system on the failing heart, such as increased heat rate
These drugs should be started slowly, increasing the dosage slowly every two weeks as tolerated by patient
This is because they can reduce myocardial contractility.

Side effects

- Edema
- Hypotension
- Fatigue
- Bradycardia

Positive Inotropes

- Examples are digitalis preparations such as digoxin
- Digitalis increase the force of cardiac contraction (inotropic action)
- They also decrease the conduction speed within the myocardium and slow the heart rate (chronotropic action)
- These actions allow for more complete emptying of the ventricles during diastole
- Cardiac output increases because of an increased stroke volume from improved contractility
- Patients taking digitalis preparations are prone to developing digitalis toxicity
- Early symptoms of toxicity include:
- Anorexia
- Nausea
- Vomiting
- Visual disturbances such as yellow vision
- Dysrhythmias
- Causes of digitalis toxicity include hypokalemia due to the use of potassium – depleting diuretics such as loop diuretics (Lasix)
- Low serum potassium enhances the action of digitalis, causing a therapeutic dose to achieve toxic levels
- Hyperkalemia, inhibits the action of digitalis causing a therapeutic dose to become sub therapeutic
- Thus monitoring serum potassium levels of patients receiving digitalis preparations and potassium depleting and potassium sparing diuretics is essential

RHEUMATIC FEVER AND RHEUMATIC HEART DISEASE

Rheumatic fever is an acute, inflammatory disease of the heart involving all layers (endocardium, myocardium and pericardium)

Rheumatic heart disease is a chronic condition resulting from rheumatic fever that is characterized by scarring and deformity of the heart valves

Etiology/Causes

Rheumatic fever is caused by group A streptococcal bacterium which causes pharyngitis

Pathophysiology

- Following infection of the pharyngeal and tonsils with streptococcal bacteria, the body forms immunologic response to group A streptococcal cell membrane antigen
- Rheumatic fever affects the heart, skin, joints and central nervous system
- Rheumatic endocarditis occurs in the valves, with swelling and erosion of the valve leaflets
- Deposits of fibrin and blood cells in areas of erosion occurs in the valves
- Valve leaflets may fuse and become thickened or calcified, resulting in stenosis
- Reduction in the mobility of valve leaflets may occur with failure of the leaflets to close properly, resulting in regurgitation
- The mitral and aortic valves are most commonly affected
- Myocardial involvement is characterized by nodules formed by a reaction to inflammation with swelling and destruction of collagen fibers
- With time, the nodules become more fibrous, and scar tissue forms in the myocardium
- Rheumatic pericarditis develops and affects both layers of the pericardium. The layers become thickened
- The lesions of rheumatic fever are systemic and involve the skin, joints and central nervous system
- Painless subcutaneous nodules, arthritis may develop

Clinical manifestations

Carditis (inflammation of endocardium, myocardium & pericardium) This is manifested by a heart murmur of mitral or aortic regurgitation.
Arthritis – Inflammation of joints whereby synovial membranes of joints are affected causing swelling, redness, tenderness, & limitation of motion
Chorea is a central nervous system manifestation which is characterized by involuntary movements of the face and limbs, muscle weakness and disturbance of speech and gait
Erythema marginatum which are lesions occurring on the trunk and proximal extremities
Subcutaneous nodules – are firm small hard painless swellings located over extensor surfaces of joints such as the knees, wrists and elbows.
Fever
-Polyarthralgia (painful joints).

Complications of Rheumatic fever

Chronic rheumatic carditis – results from changes in valvular structure
Rheumatic endocarditis result in fibrous tissue growth in valve leaflets with scarring and contractures
Valves most affected are the mitral, aortic and tricuspid

MANAGEMENT

Drug therapy

Antibiotic therapy eliminates residual group A streptococcal bacteria remaining in the tonsils and pharynx

They also prevent the spread of organisms to close contacts. Examples of antibiotics is Amoxicillin.
Non-steroidal anti-inflammatory drugs and corticosteroids are the anti-inflammatory agents most widely used in the management of rheumatic fever

Nursing Management

- Health educate patient about preventive measures such as early detection and immediate treatment of group A streptococcal pharyngitis
- Administrative medications i.e. antibiotics such as penicillin
- Teach patient the importance of drug compliance
- Administer NSAIDs & corticosteroids as prescribed
- Monitor fluid intake & output as appropriate
- Promote rest periods between activities to reduce the cardiac workload and diminish the metabolic needs of the body
- Position painful joints for comfort and proper alignment
- Teach about a well-balanced diet and hygiene practices to promote patient's well being
- Teach patient to seek medical attention if symptoms such as excessive fatigue, dizziness, palpitations or dyspnea develop

Acquired valve disease / valvular disease

- The heart contains two atrio ventricular valves (mitral and tricuspid) and two semilunar valves (aortic and pulmonic); which control unidirectional blood flow
- In stenotic valve, the valve opening is smaller. The forward flow of blood is impaired, creating a difference in pressure on the two sides of the open valve.
- Stenosis is constriction or narrowing of the valve.
- In regurgitation, incomplete closure of the valve leaflets results in the backward flow of blood.
- Valve disorders occur in children and adolescents mainly from congenital disorders
- However, they also occur in adults due to cardiovascular disease, Aids and the use of antiparkinsonian drugs
- Examples of valvular disorders are as follows:-

Mitral valve stenosis

Causes

- Rheumatic heart disease (Rheumatic mitral stenosis)
- Congenital mitral stenosis
- Rheumatoid arthritis
- Systemic lupus erythematosus

Pathophysiology

Rheumatic endocarditis causes scarring of the valve leaflets
Contractures and adhesions develop between the junctional areas

The stenotic mitral valve takes on a “fish mouth” shape because of the thickening and shortening of the mitral valve structures
These structural deformities cause obstruction of blood flow and create a pressure difference between the left atrium of the left ventricle during diastole
Left atria pressure and volume increase
This causes higher pulmonary vasculature pressure and then hypertrophy of the pulmonary vessels

Clinical manifestations
Dyspnea on exertion due to reduced lung compliance
Fatigue and palpitations due to atrial fibrillation
Heart murmur
Hoarseness from atrial enlargement pressing on the laryngeal nerve
Hemoptysis from pulmonary hypertension
Chest pain from decreased cardiac output and coronary perfusion
Seizures or stroke from emboli which can arise from blood stasis in the left atrium due to atrial fibrillation

Mitral valve regurgitation
Etiology
Myocardial infarction
Rheumatic heart disease
NB/ Mitral valve is situated between the left atrium and the left ventricle. It has two cusps. Mitral valve allows blood to flow from the left atrium into the left ventricle but prevents blood from flowing back into the atrium.

Pathophysiology
Mitral valve regurgitation allows blood to flow backward from the left ventricle to the left atrium due to incomplete valve closure during systole
The left ventricle and left atrium both work harder to preserve an adequate cardiac output. This additional volume results to left atria enlargement, left ventricular dilation and hypertrophy & decrease in cardiac output

Clinical manifestations
- Thready peripheral pulse
- Cool crummy extremities
- Fatigue
- Weakness
- Palpitations
- Dyspnea
- Peripheral edema
- Murmur

AORTIC VALVE STENOSIS

Etiology/Causes

Congenital
Rheumatic fever

Pathophysiology

- In Rheumatic valve disease, fusion of the joints and secondary calcification cause the valve & leaflets to stiffen and retract resulting in stenosis
- Aortic valve stenosis causes obstruction of flow from the left ventricle to the aorta during systole
- The effect is left ventricular hypertrophy and increased myocardial oxygen consumption because of the increased myocardial mass.
- As the disease progresses and compensatory mechanisms fail, reduced cardiac output leads to decreased tissue perfusion, pulmonary hypertension and heart failure.

Clinical manifestations

Angina
Exertional dysphonia

AORTIC VALVE REGURGITATION

Etiology/causes

Rheumatic heart disease
Congenital bicuspid aortic valve
Syphilis
Arthritis

Pathophysiology

Aortic valve regurgitation causes retrograde blood flow from the ascending aorta into the left ventricle during diastole, resulting in volume overload
The left ventricle initially compensates for chronic aortic valve regurgitation by dilation and hypertrophy
Myocardial contractility eventually declines and blood volumes increase in the left atrium and pulmonary bed
This results in pulmonary hypertension and right ventricular failure

Clinical manifestation

Dyspnea
Chest pain
Hypotension

TRICUSPID AND PULMONIC VALVE DISEASE

Tricuspid stenosis occurs in patients with Rheumatic fever, IV drug users.
Tricuspid stenosis results in right atrial enlargement and elevated systematic venous pressure.
Pulmonary stenosis is almost always congenital and results in right ventricular hypertension and hypertrophy

MANAGEMENT

Conservative Therapy

- Use of prophylactic antibiotic therapy to prevent bacterial infections
- Heart failure is treated with vasodilators, Beta -adrenergic blockers, diuretics and a low sodium diet.
- Anticoagulant therapy prevents and treats systemic or pulmonary emboli. It is used prophylactically in patients ₩ith atrial fibrillation
- Dysrhythmias are treated with digoxin and Beta blockers.

Surgical Therapy

- The type of surgery can be valve repair or valve replacement
- Valvulotomy is a procedure for valve repair
- Valve replacement may be required for mitral, aortic, tricuspid and pulmonic valve disease
- Prosthetic valves are used in valve replacement

NURSING MANAGEMENT

- Administrator medications as prescribed e.g. Beta blockers, diuretics.
- Design an appropriate exercise plan with the patient that increases cardiac tolerance
- Restrict activities that regularly produce fatigue & dyspnea
- Discourage to tobacco use
- Advice patient to avoid strenuous physical exercise because damaged valves may not handle the increased demand in cardiac output
- Plan patient's activities of daily living with an emphasis on conserving energy, setting priorities and taking planned rest periods
- Refer patient & fly members to a counselor if the patient has a physically or emotionally demanding job
- Perform ongoing cardiac assessments to monitor the effectiveness of cardiac medications
- Teach patient about the actions and side effects of drugs to achieve compliance
- Teach patient about when to seek medical care i.e. notify the health care provider about any planned invasive or dental procedures, signs of bleeding any manifestations of infection, heart failure etc.
- Encourage patient to wear a medic alert bracelet.

CORONARY HEART DISEASE

- Also called coronary artery disease
- It is a type of blood vessel disorder (coronary artery)

Etiology/Causes

A atherosclerosis is the major cause of coronary artery disease

Atherosclerosis is the major cause of coronary artery disease. Atherosclerosis comes from two Greek wards adhere meaning fatty mush and skleros meaning hard. Thus atherosclerosis begins as soft deposits of fat that harden with age.

The atheroma (fatty deposits) has a high affinity for the coronary arteries.

B tobacco use

C Hypertension

D Diabetes mellitus

E Infection i.e. herpes virus, chlamydia, pneumonia that cause a local inflammatory response

Pathophysiology

Coronary heart disease is a progressive disease that takes many years to develop

The stages of development in atherosclerosis are: -

a) Fatty streak

Are the earliest lesions of atherosclerosis.

Are characterized by lipid- filled smooth muscle cells

As streaks of fat develop within the smooth muscle cells, a yellow tinge appears

b) Fibrous Plague

This is the beginning of the progressive changes in the endothelium of the arterial wall.

Low density lipoproteins and growth factors from platelets stimulate smooth muscle proliferation and thickening of the arterial wall.

Lipoproteins (carrier proteins within the blood stream) transport cholesterol and other lipids into the arterial wall

Collagen covers the fatty streak and forms a fibrous plague with a grayish or whitish appearance.

The result is a narrowing of the vessel lumen and a reduction in blood flow to the distal tissues

c) Complicated lesion

Is the final stage in the development of the atherosclerotic lesion

As the fibrous plague grows, continued inflammation can result in plague instability, ulceration and rupture

Platelets accumulate in large numbers, leading to a thrombus.

The thrombus may adhere to the wall of the artery, leading to further narrowing or total occlusion of the artery.

RISK FACTORS FOR CORONARY ARTERY DISEASE

Risk factors are characteristics or conditions that are associated with a high incidence of coronary artery disease

They are classified as non-modifiable & modifiable as follows:

NON-MODIFIABLE RISK FACTORS

a) Age – The incidence of coronary artery disease is highest among the elderly.
b) Family history and genetics - Genetic predisposition plays a role in the occurrence of coronary artery disease

Modifiable Risk Factors

a) **Elevated Serum Lipids** – Elevated serum lipid level is a risk factor for coronary artery disease

For lipids to be used and transported by the body they must become soluble in blood by combining with proteins.

Lipids combine with proteins to form lipoproteins.

Lipoproteins are vehicles for fat mobilization and transport, and vary in composition.

They are classified as high density lipoproteins (HDLs, LDLs, and very low density lipoproteins VLDLs

High density lipoproteins contain more protein by weight and fewer lipids than any other lipoprotein.

HDLs carry lipids away from the arteries and to the liver for metabolism.

This process of HDL transport prevents lipid accumulation within the arterial walls.

Therefore, the higher the HDL levels, in the blood, the lower the risk of coronary artery disease.

HDL levels are higher in children and women, decrease with age and are low in persons with coronary artery disease. Physical activity, moderate alcohol consumption and estrogen administration increase HDL levels.

Low density lipoproteins contain more cholesterol than any other lipoproteins and have an affinity for arterial walls.

Thus they may deposit cholesterol directly on the walls of arteries.

Elevated LDL levels are associated with an increased incidence of coronary artery disease.

Therefore, low LDL, levels are not desirable

Life style factors that can contribute to elevated triglycerides include high alcohol consumption, high intake of refined carbohydrates and simple sugars and physical inactivity.

b) Hypertension

Hypertension is a blood pressure greater than I40/ 90 mmHg

The stress of elevated blood pressure increases the rate of atherosclerotic development.

Atherosclerosis cause narrowed, thickened arterial walls and decreases the elasticity of blood vessels.

More force is required to pump blood through diseased arteries, and this increased force is reflected in a higher blood pressure.

This increased work load results in left ventricular hypertrophy & decreased stroke volume with each contraction.

c) Tobacco use

Tobacco use is a risk factor in coronary artery disease

Tobacco use decreases estrogen levels, placing premenopausal women at greater risk for coronary artery disease.

Nicotine in tobacco smoke causes catecholamine i.e. epinephrine, norepinephrine release.

These neurohormones cause an increased heart rate, peripheral vasoconstriction and increased blood pressure.

These changes increase the cardiac workload necessitating greater myocardial oxygen consumpatiention.

Nicotine also increases platelet adhesion, which increases the risk of emboli formation.

Carbon monoxide, a by-product of combustion found in tobacco smoke, affects oxygen, carrying capacity of hemoglobin by reducing the sites available for oxygen transport.

Thus the effects of an increased cardiac workload, combined with the oxygen depleting effect of carbon monoxide, decreases the oxygen available to the myocardium.

Carbon monoxide is also a chemical irritant thus causing injury to the endothelium.

d) Physical inactivity

Physical inactivity implies a lack of adequate physical exercise on a regular basis.

Examples of physical activity is brisk walking.

Physically active people have increased HDL levels

Exercise enhances fibrinolytic activity thus reducing the risk of clot formation.

Exercise training for those who are physically inactive decreases the risk of CAD through more efficient lipid metabolism, increased HDL production, and more efficient oxygen extraction by the working muscle groups thereby decreasing the cardiac workload.

For those individuals with CAD, regular physical activity reduces symptoms, improves functional capacity and improves other risk factors such as insulin resistance and glucose intolerance.

e) Obesity

Obesity is defined as a body mass index (MBI) of greater than 30kg/m2.

BMI is a calculation of body fat based on weight & height.

Obese persons may produce increased levels of LDLs & triglycerides which are strongly implicated in atherosclerosis.

As obesity increases, the heart size grows causing increased myocardial oxygen consumption.

There is an increase in insulin resistance in obese individuals.

MANAGEMENT OF CORONARY ARTERY DISEASE

Health educate patient on the importance of maintaining an ideal body weight to decrease the symptoms of CAD.

Advice & assist patient to perform physical exercise to increase myocardial oxygen consumption & increase cardiac output. Give patient diet low in saturated fats & cholesterol, and increase complex carbohydrates to lower LDL cholesterol.

Advice patient on the importance of smoking cessation, and increase physical activity as these life style changes reduce the risk of CAD.

Administer Niacin, a water soluble vitamin B to lower LDL & triglyceride levels & to increase HDL level as prescribed.

ANGINA PECTORIS

- DEF: Chest pain caused most often by myocardial anoxia as a result of artheloscrelosis or spasm of the coronary arteries.
- The pain usually radiates to the neck, jaw and shoulder and down the inner aspect of the left arm.
- It is frequently accompanied by a feeling of suffocation and impending death.
- Attack of angina pectoris are often relate to exertion, emotional stress, eating and exposure to intense cold.
- The pain maybe relieved by rest and vasodilation of the coronary arteries by medications such as nitroglycerin.
- Angina is the clinical manifestation of myocardial ischemia.
- When the demand for myocardia oxygen exceeds the ability of the coronary arteries to supply the heart with oxygen, myocardial ischemia occurs.
- Either an increased demand for oxygen or a decreased supply of oxygen can lead to myocardial ischemia.
- The primary reason for insufficient blood flow is narrowing of coronary arteries by atherosclerosis.
- For Ischemia caused by atherosclerosis to occur, the artery is usually obstructed (stenosed)
- On the cellular level, the myocardium becomes hypoxic within the 1st 10 seconds of coronary occlusion.
- With total occlusion of the coronary artery, contractility ceases after several minutes, depriving the myocardial cells of oxygen & glucose for aerobic metabolism .

- Anaerobic metabolism begins and lactic acid accumulates.
- Lactic acid irritates myocardial nerve fibers & transmits a pain message to the cardiac nerves and upper thoracic posterior nerve roots.
- This accounts for referred cardiac pain to the left shoulder and arm.
- In ischemic conditions, cardiac cells are viable for approximate 20 minutes.
- With restoration of blood flow, aerobic metabolism resumes, contractility is restored and cellular repair begins.

TYPES OF ANGINA

1. Chronic stable angina

Refers to chest pain that occurs intermittently over a long period with the same pattern of onset, duration and intensity of symptoms.

The chest pain is described as constrictive, squeezing, heavy, chocking or suffocating sensation.

The pain radiates to various locations i.e. the jaw, shoulders, & down the arms.

The pain usually lasts for a few minutes (5- 15 minutes) and subsides when the precipitating factor is relieved.

2. Nocturnal Angina

Occurs only at night but not necessarily when the person is in the recumbent position or during sleep.

3. Angina Decubitus

Chest pain that occurs only while the patient is lying down and is usually relieved by standing or sitting.

4. Prinz metals Angina (Variant Angina)

Chest pain that occurs at rest, usually in response to spasm of a major coronary artery.

The spasm may occur in the absence of CAD.

Strong contraction (spam) of smooth muscle in the coronary artery results from increased intracellular calcium.

Factors precipitating coronary artery spasm include increased myocardial oxygen demand and increased levels of certain substances such as tobacco, smoke and histamines.

Calcium channel blockers i.e. nifedipine, amlodipine or nitrates (nitroglycerin) are used to control the angina.

5 Unstable Angina

Chest pain that is new in onset and occurs at rest.

It is unpredictable of represents as an emergency.

MANAGEMENT OF ANGINA PECTORIS

- Give antiplatelet to prevent blood clot formation in the coronary arteries, e.g. heparin.
- Give cholesterol lowering drugs to reduce levels of LDLs, i.e. Niacin (a water soluble vitamin B.
- Give nitrates to dilate coronary arteries as well as peripheral blood vessels.
- Nitrates may be administered sub-lingual or I.V. Examples of nitrates are: Nitroglycerine and isosorbide dinitrate
- Give Beta-adrenergic blockers to decrease myocardial contractility, heart rate, & blood pressure, all of which reduce the myocardial oxygen demand.
- Give opioid analgesia i.e. morphine for pain control.
- Give oxygen to supply the myocardial oxygen demand.
- Give psychological care to reassure the patient & family members to ease anxiety.
- Assist patient in activities of daily living i.e. bathing ambulating etc.
- Provide a well-balanced meal, that is low in sodium & fat.
- Asset patient with exercise as tolerated.
- Advice patient on cessation of smoking as this may precipitate angina attack.
- Assess vital signs four hourly to monitor patients progress.
- Assist patient to identify precipitating factors & how to avoid them.
- Obtain a 12 lead ECG to monitor patients heart activity.

MYOCARDIAL INFARCTION

Myocardial infarction is death or necrosis of a portion of cardiac muscle caused by an obstruction in a coronary artery through atherosclerosis, a thrombus or spasm.

Myocardial infarction occurs because of sustained ischemia, causing irreversible myocardial cell death.

Clinical manifestations of myocardial infarction

a) **Pain**

- This is severe, immobilizing chest pain that is not relieved by rest, position change or nitrate administration.
- Pain is persistent and usually described as a heaviness, pressure, tightness, burning constriction or crushing.
- Common locations are sub-sternal and epigastric areas.
- The pain may radiate to the back, neck, jaw or arms.
- It may occur when the patient is active or at rest, a sleep or awake.
- It commonly occurs in the early morning hours, usually lasts for 20 minutes or longer and is more severe than usual angina pain.
- Patients with diabetes may experience silent (asymptomatic) myocardial infarction due to cardiac neuropathy and present with a typical symptoms of dyspnea.

b) **Sympathetic nervous system stimulation**

During the initial phase of myocardial infarction, the ischemic myocardial cells release catecholamine (norepinephrine & epinephrine) that are normally found in these cells.

This results in release of glycogen, diaphoresis and vasoconstriction of peripheral blood vessels.

On physical examination, the patient's skin may be clammy and cool to touch.

c) **Cardiovascular manifestations**

In response to the release of catecholamine, blood pressure & heart rate may be elevated initially.

Later, blood pressure may drop because of decreased cardiac output.

This may result in decreased renal perfusion & urine output.

Presence of crackles suggest left ventricular dysfunction.

Jugular venous distention, hepatic engorgement and peripheral edema may indicate right ventricular dysfunction.

d) **Nausea & vomiting**

The patient maybe nauseated & vomit.

Nausea & vomiting can result from reflex stimulation of the vomiting center by the severe pain.

These symptoms can also result from vasovagal reflexes initiated from the area of the infarcted myocardium.

e) Fever

Increase in body temperature is a systemic manifestation of the inflammatory process ceased by myocardial cell death.

Pathophysiology of Myocardial infarction

- The body's response to cell death is the inflammatory process.
- Within 24hrs, leukocytes infiltrate the area.
- The dead cardiac cells release enzymes.
- The proteolytic enzymes of the neutrophils and macrophages begin to remove necrotic tissue.
- Catechoramine-meidated lipolysis & glycogenolysis occur.
- This allows the increased plasma glucose & free tatty acids to be used by the oxygen depleted myocardium for anaerobic metabolism.
- The collagen matrix is laid down to form scar tissue.
- The scarred area is often less compliant than the surrounding fibers, and this condition is manifested by ventricular dysfunction or heart failure.
- The normal myocardium hypertrophies and dilates in an attempt to compensate for the infarcted muscle, a process called ventricular remodeling.
- Remodeling of normal myocardium can lead to the development of heart failure.

Complications of Myocardial Infarction

a) Dysrhythmias

The intrinsic rhythm of the heart beat is disrupted, causing a fast heart rate (tachycardia) a slow heart rate (bradycardia), or an irregular beat, affecting the ischemic myocardium

b) Heart failure

Is a complication that occurs when the pumping power of the heart has diminished.

Heart failure occurs with signs such as pulmonary congestion on X-ray, crackles on auscultation of breath sounds and jugular vein distention.

c) Cardiogenic shock

Occurs when oxygen and nutrients supplied to the tissues are inadequate because of severe left ventricular failure.

d) Ventricular aneurysm

This results when the infarcted myocardial wall is thin & bulges out during contraction.

Patient with ventricular aneurysm may experience heart failure, dysrhythmias, & angina.

Ventricular aneurysm may rupture or harbor thrombi that can lead to an embolic stroke.

MANAGEMENT OF MYOCARDIAL INFARCTION

- Give oxygen therapy via nasal cannula to meet myocardial oxygen demand.
- Monitor patient's cardiac activity (ECG)
- Give sub-lingual nitroglycerin & aspirin for pain control.
- Give morphine for pain control.
- Monitor vital signs four hourly to monitor patient's progress.
- Maintain bed rest & limitation of activities to reduce myocardial oxygen demand.
- Provide reassurance & emotional support to patient & caregiver to alleviate anxiety.
- Give psychological and emotional support to alleviate stress and anxiety.
- Teach patient about signs & symptoms of angina and what to do should they occur.

VASCULAR CONDITIONS

ANEURYSM

Is an out pouching or dilation of the vessel wall.

Causes

Atherosclerosis

- Atherosclerotic plague formation causes degenerative changes in the blood vessel affected (mainly artery) leading to loss of elasticity; weakening & dilation.
- Congenital predisposition
- Infections i.e. bacterial or viral
- Trauma

Classification of aneurysm

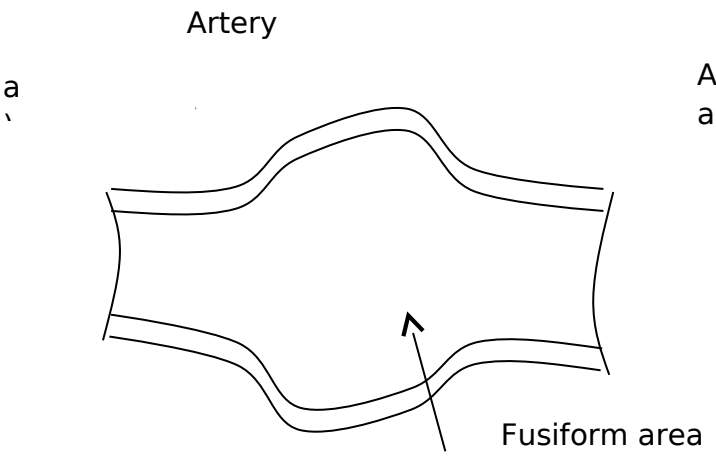
A True

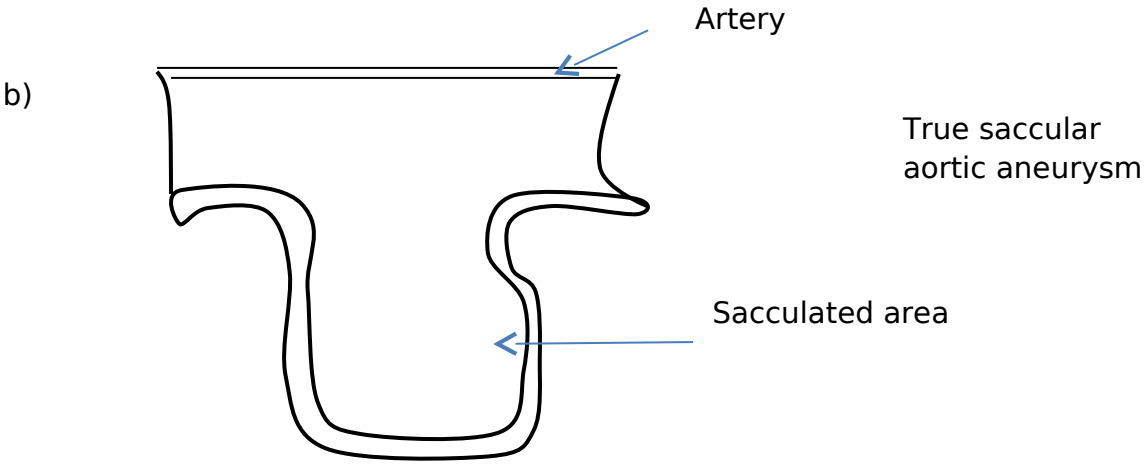
B False (pseudo aneurysm)

A true aneurysm is one in which the wall of the artery forms the aneurysm, with at least one vessel layer still intact.

True aneurysms are further sub-divided into fusiform and saccular types.

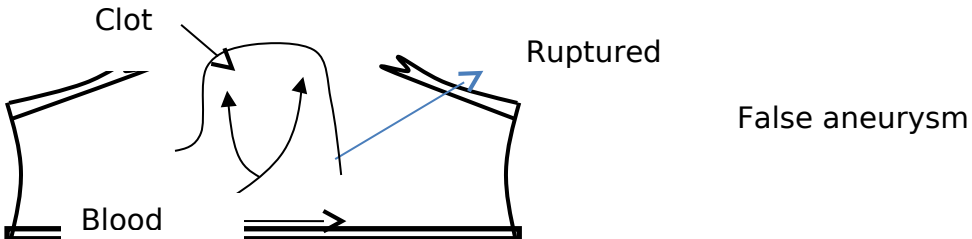
A fusiform aneurysm is circumferential and relatively uniform in shape i.e.





False aneurysm (pseudo aneurysm) is not an aneurysm but a disruption of an arterial wall layers with bleeding that is contained by surrounding anatomic structures.

False aneurysms may result from trauma or infection.



Clinical manifestations

- Chest pain
- Hoarseness from pressure on the laryngeal nerve
- Dysphagia due to pressure on the esophagus
- Jugular nervous distention
- Edema of the face & arms

Complications of aneurysms

Rupture of the aneurysm leading to death

MANAGEMENT OF ANEURYSM

Involves surgical repair

Pre-operative care

- Hydrate patient with IV fluids & correct electrolyte abnormalities
- Psychological care
- Physical preparation
- Starve patient from mid night of the day prior to surgery

Post-operative care

Monitor ECG

Assess level of censoriousness e.g. pupil size response to light, etc.

Monitor vitals. Observe aseptic technique to prevent spread of infection.

Give analgesia as prescribed for pain control.

Check and record peripheral pulses hourly.

Encourage early ambulation. Assess for bowel sounds for return of peristalsis.

Record intake and output to monitor renal perfusion status. Instruct patient & care giver to gradually increase activities after discharge.

Advice patient to avoid heavy lifting for 6 weeks post operatively to avoid excessive pressure.

Advice patient to report signs of redness, swelling, increased pain and fever to the health care provider as these are signs of complications.

VARICOSE VEINS

Also called varicosities.

Are dilated tortuous subcutaneous veins commonly found in the saphenous vein system

Primary varicosities are due to congenital weakness of the veins and are more common in women.

Secondary varicose veins result from a previous venous thromboembolism.

They may occur in the esophagus (esophageal varices, vulva, sparmatic cords (varicoceles) and anorectal area (hemorrhoids).

Causes /Risk Factors

- Chronic cough
- Constipation
- Family history of venous disease
- Female gender
- Use of oral contraceptives
- Age
- Obesity
- Pregnancy
- Occupations that require prolonged standing.

Pathophysiology

- In varicose veins, the vein valve leaflets are stretched & become incompetent (do not fit together properly)
- Incompetent vein valves allow retrograde blood flow especially when the patient is standing, resulting in increased venous pressure & further venous distention .

Clinical manifestations

- Discomfort
- Disfigurement (cosmetic)
- Pain after prolonged standing
- Swelling
- Noctural leg cramps

NURSING MANAGEMENT

- Advice patient to avoid sitting or standing for long periods of time.
- Advice patient to maintain ideal body weight.
- Advice patient to avoid wearing constrictive clothing and walk daily.
- Advice patient to apply stockings in bed and before rising in the morning.

VENOUS THROMBOSIS

- Venous thrombosis involves the formation of a thrombus in association with inflammation of the vein.
- Classified into: -

a) Superficial vein thrombosis

Is the formation of a thrombus in a superficial vein.

Deep vein thrombosis (DVT) is a disorder involving a thrombus in a deep vein, most commonly the iliac and femoral veins.

Causes

a) Venous stasis

Occurs when the valves are dysfunctional or the muscles of the extremities are inactive.

It occurs most frequently to people who are: -

- Obese
- Pregnant
- Chronic heart failure
- Prolonged surgical procedure e.g. orthopaedic surgery (especially lower extremity)
- Prolonged immobility e.g. spinal cord injury, fractured hip, bed rest, stroke, varicose veins.

b) Endothelial damage

Damage to the endothelium of the vein may be caused by: -

- Abdominal & pelvic surgery
- Fractures of the pelvis, hip or leg
- I.V drug abuse
- Trauma

Damaged endothelium stimulates platelet activation and initiates the coagulation cascade.

This results in decreased fibrinolytic capabilities and predisposes the patient to thrombus development.

c) Hypercoagulability

Hypercoagulability of blood occurs mainly in these conditions: -

- Cigarette smoking
- Dehydration or malnutrition
- Oral hormone replacement therapy
- Malignancies esp. breast, brain, hepatic, pancreatic & gastro intestinal
- Oral contracepatientives especially in women over 35 years of age
- Pregnancy & postpartum period.
- Sepsis
- Severe anaemia

Pathophysiology

Localized platelet aggregation and fibrin entrap red blood cells (RBCs WBCs and move platelets to form a thrombus.

As the thrombus enlarges, increased numbers of blood cells & fibrin collect behind it, producing a larger clot with a “tail” that eventually occludes the lumen of the vein.

If the thrombus does not become detached, it undergoes lysis or become firmly organized and adherent within 5-7 days

The organized thrombi may detach and result in emboli.

Turbulence of blood flow is a major contributing factor to embolization.

The thrombus can become an embolus that flows through the venous circulation to the heart and lodges in the pulmonary circulation, becoming pulmonary emboli.

Clinical manifestations of superficial vein thrombosis

- Tenderness on the area surrounding the vein
- Fever
- Warmth and redness on the area surrounding the vein
- Pain
- Edema

MANAGEMENT

Administer anticoagulants i.e. heparin

Advice patient to wear anti emboli stocking to help reduce edema.

Advice patient to perform mild exercise such as walking to help increase endogenous fibrinolysis.

Deep venous thrombosis

Clinical manifestations

- Leg edema
- Pain on the affected limb
- Fever
- Warmth on touch
- Tender to palpation

Complications

Pulmonary embolism

Chronic venous insufficiency which results from valvular destruction, allowing retrograde venous blood flow.

MANAGEMENT

Preventive measure includes mobilization of patients that can ambulate

Turn patient on bed rest two hourly

Encourage patients to wear anti embolic stockings to increase venous blood flow velocity, prevent venous wall dilation and improve venous valve function.

Give anticoagulants to prevent clot formation or to prevent propagation of the clot and development of any new thrombi.

Examples of anticoagulants are vitamin k antagonists, warfarin etc.

Instruct the patient to avoid constrictive clothing.

Give a well-balanced diet including calcium and vitamin E as these affect coagulations.

Encourage proper hydration to prevent additional hypercoagulability of the blood, which may occur with dehydration.

Encourage exercise as tolerated by the patient.

Phlebitis

This is the inflammation i.e. (redness, tenderness warmth, mild edema) of a superficial vein without the presence of a thrombus (clot).

Embolism

- An abnormal condition in which an embolus travels through the blood stream and become lodged in a blood vessel.
- Symptoms vary with the character of the embolus, the degree of occlusion that results and the size, nature and location of the occluded vessel.

Atherosclerosis

- A common disorder characterized by yellowish plague of cholesterol, other lipids and cellular debris in the inner layers of the walls of arteries.
- It usually occurs with aging & is often associated with tobacco use, obesity, hypertension, elevated low density lipoprotein and depressed high density lipoprotein levels, and diabetes mellitus.
- The condition begins as a fatty streak and gradually builds to a fibrous plague or atheromatous lesion.
- The vessel walls become thick, fibrotic and calcified & the lumen narrows, resulting in reduced blood flow to organs normally supplied by the artery.
- The plague eventually creates a risk for thrombosis & is one of the major causes of coronary heart disease, angina pectoris, Myocardial infarction etc.

Arteriosclerosis

- A disorder characterized by thickening, loss of elasticity and calcification of aerial walls.
- It results in decreased blood supply to the cerebrum, & lower extremities.
- The condition develops with aging & in hypertension, diabetes mellitus etc.

Gangrene

Necrosis or death of tissue, due to ischemia or bacterial invasion.

It occurs as a complication of diabetes mellitus where the lower extremities become gangrenous.

INFECTIVE ENDOCARDITIS

is an infection of the endocardial layer of the heart.

The endocardium, the innermost layer of the heart is contiguous with the heart valves.

Therefore, inflammation from infective endocarditis affects the cardiac valves.

Causes

Bacteria i.e. staphylococcus aureus & streptococcus.

Fungi

Viruses

Contributing factors

Aging, use of prosthetic valves, use of intra vascular devices resulting in hospital acquired infections by staphylococcus bacteria etc.

Pathophysiology

- Infective endocarditis occurs when blood flow turbulence within the heart allows the causative organisms to infect previously damaged valves or other endothelial surfaces.
- The infection may spread locally and damage the valves or their supporting structures

- This causes dysrhythmias, valve incompetence and eventual invasion of the myocardium leading to heart failure and sepsis.

Clinical manifestations

- Fever & chills
- Malaise
- Fatigue
- Anorexia
- Arthralgia
- Back pain
- Abdominal discomfort
- Weight loss
- Headache
- Clubbing of fingers

MANAGEMENT

- Give antibiotics to treat bacterial infection
- Give antipyretics to control fever
- Monitor vital signs four hourly.
- Teach patient to recognize signs & symptoms of complications such as change of mental status etc. & report to health care provider.
- Provide adequate rest to the patient especially when fever is present.
- Perform ROM exercise, cough & deep breathing 2 hourly to prevent problems related to decreased mobility.
- Give psychological support to allay anxiety
- Give a well-balanced diet to promote healing
- Advice the patient about the need for importance of prophylactic antibiotic therapy before certain invasive procedures such as tooth extraction, surgery etc.

CARDIOMYOPATHY

Def.: Is a deceases of the heart muscle that is associated with cardiac dysfunction.

- It is a type of progressive heart disease in which the heart is abnormally enlarged, thickened and stiffened.

- As a results the heart muscles ability to pump blood is less efficient, often causing heart failure and the backup of blood into the lungs or the rest of the body.

TYPE OF CARDIOMYOPATHY

1. **Dilated cardiomyopathy**
    - It is characterized by dilation of the ventricles.
    - It is caused by diseases such as heavy alcohol intake, viral infection, e.g. influenza, chemotherapeutic drugs, thyrotoxicosis, myxedema genetic predisposition.
    - Microscopic examination of the muscle tissue shows diminished contractile elements of the muscles fibers and diffuse necrosis of myocardial cells.
    - The results are poor systolic function.
    - The structural changes decrease the amount of blood ejected from the ventricles with systole, increasing the amount of blood remaining in the ventricle after contraction.
    - Less blood is then able to enter the ventricles during diastole resulting to increased pulmonary and systemic venous pressures.

2. **Restrictive cardiomyopathy.**
    - Is characterized by diastolic dysfunction caused by rigid ventricular walls.
    - This impairs diastolic filling and ventricular stretch.
    - Systolic function is usually normal.
    - Restrictive cardiomyopathy is associated with amyloidosis [amyloid, a protein substance which is deposited within cells:
    - Signs and symptoms include dyspnea, nonproductive cough, and chest pain.

3. **Hypertrophic cardiomyopathy**.
    - Is characterized by asymmetrical increase in the size and mass of the heart muscles.
    - The increased thickness of the heart muscles reduces the size of the ventricular cavities and causes the ventricles to take a longer time to relax after systole.
    - The hypertrophied cardiac muscles cell are disorganized, oblique and perpendicular to each other decreasing the effectiveness of contractions and increasing the risk of dysrhythmias such as ventricular tachycardia.
    - In hypertrophied cardiomyopathy, the coronary arteriole walls are thickened which decreases the internal diameter of the arterioles.
    - The narrow arterioles restrict the blood supply to the myocardium, causing numerous small areas of ischemia and necrosis.

4. **Arrhythmogenic right ventricular cardiomyopathy/dysplasia.**
    - Occurs when the myocardium is progressively infiltrated and replaces by fibrous scar and eclipse tissue.

- Eventually the right ventricle dilates and develops poor contractility and dysrhythmias.

CLINICAL MANIFESTATIONS OF CARDIOMYOPATHY

- ✓ Fatigue
- ✓ Cough on exertion
- ✓ Peripheral edema
- ✓ Nausea
- ✓ Chest pain
- ✓ Palpitations
- ✓ Dizziness
- ✓ Cardiac arrest
- ✓ Severe heart failure
- ✓ Dysrhythmias

Diagnostics findings for patients with cardiovascular diseases i.e. congestive cardiac failure hypertension cardiomyopathy, etc.

1. Physical examination may reveal tachycardia and extra heart sounds [murmurs].
    - Crackles on pulmonary auscultation
    - Jugular vein distention.
    - Hepatomegaly.
    - Pitting edema of dependent body parts
2. Echocardiogram
3. Cardiac MRI, [magnetic resonance imaging]
4. ECG- reveal dysrhythmias
5. Chest x-rays reveals heart enlargement
6. Cardiac catheterization to rule out coronary artery disease.
7. Endo myocardial biopsy is done to analyze myocardial cells.

NURSING NENAGEMENT

-Restrict sodium in the Patients diet to prevent fluid retention.

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- Provide alternate rest periods between activities to conserve energy.
- Give antiarrhythmic drugs to control dysrhythmias as prescribed.
- Give anticoagulants e.g. warfarin to prevent thromboembolism as prescribed.
- Limit fluid intake to two liters per day to ease symptoms of congestion.
- Gives beta-blockers e.g. atenolol, metoprolol to maintain cardiac output. Advice patient and family members on the importance of alcohol intake cessation if he is an alcoholic.
- Offer counseling to patient and family members to alleviate anxiety.
- Give oxygen therapy via nasal prongs or face mask to improve oxygen saturation.
- Take and records patients. Weight dairy to monitor patient's response to treatment.
- Provide a well-balanced meal that is low in sodium in small frequent amounts to meet the patient's nutritional needs.

HYPERTENSION

Def; it is defined as persistent systolic blood pressure greater or equal to 140 mmHg and diastolic blood pressure greater than or equal to 90mmHg.

TYPES OF HYPERTENSION

1. **Primary hypertension (essential/ idiopathic)**

This is elevated blood pressure without an identified cause

Risks Functions for Primary Hypertension

- Age -blood pressure tends to rise with increasing age.
- Alcohol consumption-excessive alcohol intake is strongly associated with hypertension'
- Cigarette smoking- it elevates blood pressure and greatly increases the risks of cardiovascular disease.
- Diabetes mellitus- hypertension is more common in diabetics.
- High cholesterol levels in the blood- contributes to hypertension
- Excess dietary sodium-contributes to hypertension. Also decreases the effectiveness of certain drugs.
- Family history-history of close blood relatives i.e. parents with hypertension are likely to have children who have an increased risk of developing hypertension.
- Obesity-weight gain is associated with increased frequency of hypertension.
- Sedentary life style-regular physical activities can help control weight and reduce cardiovascular risks.

2. **Secondary hypertension.**
   - ✓ This is elevated blood pressure with a specific cause that often can be identified and corrected.

Causes of secondary hypertension

- ❖ Cirrhosis of the liver
- ❖ Endocrine discovers I.e. Cushing syndrome
- ❖ Neurologic disorders such as brain tumors
- ❖ Pregnancy induced hypertension
- ❖ Renal disease i.e. glomerulonephritis.

Clinical manifestations of hypertension

a. Fatigue
b. Reduced activity tolerance
c. Dizziness
d. Palpitations
e. Angina

f. Dyspnea

Complications of high blood pressure

The most common complication of hypertension are target organ diseases such as the following:

(1) Hypertensive heart disease e.g.

a. Coronary artery disease-hypertension disrupts the coronary artery endothelium.
This disruption exposes the intimal layer to activated white blood cells and platelets.
The changes in the artery result in a stiffened arterial wall and a narrowed internal lumen and account for a high incidence of coronary artery disease, angina and M.I

b. Heart failure-occurs when the heart's compensatory adaptations are overwhelmed and the heart can no longer pump enough blood to meet the metabolic needs of the body.
Contractility is depressed, smoke volume and cardiac output is decreased.
The patient may complain of dyspnea, fatigue etc.:

2) Cerebrovascular disease- high blood pressure predisposes one to development of stroke.
3) Nephrosclerosis-hypertension needs to end stage renal disease.
4) Retinal damage- high blood pressure cause damage to the retinal vessels manifested by blurring of vision, retinal hemorrhage and loss of vision.

MANAGEMENT

Teach about lifestyle modification such as the following:

- Weight reduction of obese individuals to reduce the risk of hypertension.
- Consumption of low sodium diet to reduce the risk of hypertension
- Moderation of alcohol consumption-- reduce alcohol consumption to lower the risks of developing hypertension.
- Physical activity- engage in regular exercise several time a week to lower the risk of developing high blood pressure
- Avoidance of tobacco products such as cigarette smoke—it contains nicotine which causes vasoconstriction and high blood pressure
  --Give diet rich in fruits, vegetable, and low fat dairy foods with reduced saturated fats to lower blood pressure.
  Advice the patient to regularly monitor blood pressure to ensure it is well maintained.
  Give antihypertensive drugs such as:
  - Diuretics such as loop diuretics to control blood pressure
  - Beta blockers i.e. atenolol to lower blood pressure, vasodilators i.e. hydralazine to lower blood pressure.
  - Angiotensin converting enzyme inhibitors such as captopril, enalapril
  - Teach patient and family members about compliance to treatment regimen and side effects.

Cardiac catheterization

A diagnostic procedure in which a Cather is introduced through an incision into a large vain, usually of an arm or leg; and threaded through the circulatory system to the heart.

The course of the catheter is followed with fluoroscopy and radiographs may be taken.

An ECG is monitored on an oscilloscope.

As the catheter tip passes through the chambers and vessels of the heart, blood pressure is monitored and blood samples are taken to study the oxygen content.

Many conditions may be accurately identified and assessed including congenital heart disease, tricuspid stenosis and valvular incompetence.

Role of the nurse during cardiac catheterization

- Monitor vital signs
- Reassure the patient and give psychological cane
- Give opioids to control pain and offer comfort
- Hydrate patient via IV and orally
- Measure intake and output to monitor fluid status of the patient.