BLOOD DISORDERS

- Blood biology
- Anaemia
- Bleeding disorders
- WBC disorders
- Leukaemia
- Blood transfusion.
- Plasma cell disorders
- Lymphomas

BLOOD BIOLOGY.

DEF: Blood is a combination of liquid, cells and cell like particles that goes through the arteries, capillaries and veins – delivering oxygen and other essential nutrients to tissues and carrying away carbon dioxide and other wastes from tissues.

Blood production.

(i) In the bone marrow.

(ii) Reticuloendothelia system.
Liver and spleen during embryological development.

(iii) And during sickness.

Functions of blood.

Primary function.

(i) Transportation of various substances and

(ii) Exchange of materials in and out of tissues.

Secondary functions.

- Cellular metabolism.
- Homeostasis of fluid volume.
- Homeostasis of PH.

- Homestasis of body temperature and depends against micro-organisms.

Blood volume.

The average volume in a 70kg in an adult male is around 5 litres.

Blood is composed of plasma and cells.

Plasma is the fluid portion of blood.

When plasma is allowed to clot the fluid part is called Serum.

It has the same components as plasma except the clotting factors.

Cells contain 45% of the total blood volume.

Plasma contains 55% of the total blood volume.

Blood cells.

- Red blood cells are erythrocytes.
- White blood cells are leucocytes.
- Platelets are the thrombocytes.

1. **ERYTHROCYTES. (RED BLOOD CELLS)**

- Are the most numerous.
- The mature RBC have no nucleus.
- They are biconcave in shape.
- Do not contain organelles like ribosomes and mitochondria.
- In males they are 5.5 million mm/cube.
- In females they are 4.8 million mm/cube.
- In diameter 8 UM (Micrometer).

Is so flexible that can pass easily through the capillaries as small as 2.8um in diameter.

Anaemia can be classified according to their sizes, microcytic, normocytic and microcytic.

Production.

They are produced in the bone marrow i.e. ribs, sterum, pelvis.

The adult bone marrow produces 175 billion encythrocytes 70 billion leukocytes and 175 billion thrombocytes daily.

Destruction.

Fragmentation occurs in the capillaries of the liver, spleen and bone marrow
Life span is 120 days.

2. LEUKOCYTES. (WHITE BLOOD CELLS).

Are of five types either granular or non-granular.

Granular.

(a) Neutrophilis.
(b) Eosinophilis
(c) Basophilis

Non-granular.

(a) Lymphocytes.
(b) Monocytes.

Neutrophilis are most prevalent and defends the body against fungus and bacteria. Are 1st to arrive at site of infection within one hour.

Eosinophilis kill parasites e.g. worm and other cancer cells.
Basophilis are elevated during allergic conditions.
Lymphocytes – T-Lymphocytes – cellular immunity – (delayed allergic reactions).
B – Lymphocytes – humoral immunity.
T- Lymphocytes derived from thymus.
B-Lymphocytes from bone marrow.

T-cells - During transplant and during destruction of tumor cells.
B-cells – differentiate into plasma cell, which produces antibodies called the Immunoglobulin's which normally destroy the foreign materials.

Monocytes are used in phagocytes. Produce the macrophages. Are 5% of total leukocytes.
Population of leukocytes 5-10 thousand mm/cube.

3. PLATELETES. (THROMBOCYTES).

Used for blood clotting or homeostasis.

Formed in bone marrow and spleen.

Lifespan is 10 days.

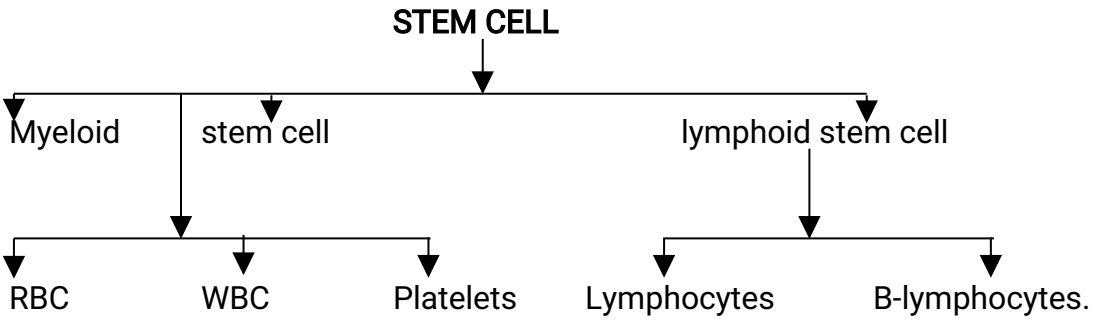
Amount 15 – 450 thousand/mm cube.

BLOOD PLASMA

90% of plasma is water and 10% is plasma.

Proteins Albumins, fibrinogen, globulins.

Blood cells formation (Hematopoiesis)



Precursor of RBC are pro-ecythrocytes which differentiate into reticulocytes (immature RBC) which produces mature RBC.

Precursor of WBC is called Myeloblast which differentiate into myelocytes (Immature WBC) which gives rise to the granular WBC – Neutrophilis, Eosinophilis, and Basophile.
Processor of platelets is megataryplasts which differentiate into megakaryocytes which gives rises to thrombocytes.

ANAEMIA.

Is a condition where the number of RBC or HB levels is below normal.

Causes.

1. Excessive bleeding.
2. Decreased production caused by iron deficiency, Vit Biz deficiency, Vitamin C deficiency, Follic acid deficiency and chronic illness.
3. Increased destruction of Red Blood Cells caused by spleenomegally, mechanical damage of Red blood cells, autoimmune reactions, haemoglobin abnormalities, hereditary factors, G6PD deficiency, sickle cell diseases.

1. **Anaemia due to excessive bleeding.**

   Is the most common cause of anaemia and there is haemodilution.

   Massive blood loss lead to hypotension and hypo-oxygenation.

Clinical features.

- Thirst.
- Fainting.
- Sweating
- Weak rapid pulse.
- Rapid breathing.
- Orthostatic hypotension.
- Shortness of breath (dyspnoea)
- Fatigue.

NB: Rapid loss of atleast 1/3 of blood is fatal.

Management

- Stop the bleeders.
- Blood transfusion.
- Raise foot of the bed.
- Fluid replacement i.e. saline, ringers lactate etc.
- Give, ion and folates for chronic conditions.
- Give oxygen.

2. **Anaemia caused by decreased Red blood cell production.**

   There is decrease in nutrients needed in ecythropoiesis.

   Nutrients needed are iron, Vit B_{12} and folic acid ,Vit C, riboflavin, copper and erythropoietin.

   (i) Iron Deficiency Anaemia.

Causes.

- Through blood loss.
- In infants and children dietary deficiency.
- Chronic loss e.g. Ca stomach , which cannot be adequately replaced by diet.
- Pregnant women as the fetus needs iron for growth.

Factors leading to decreased absorption of iron.

- Vegetable fibres.
- Foods with phosphates e.g. beans (always soak beans before cooking to remove phytate)
- Antacids reduces absorption of iron e.g. actals.

Clinical features.

- Fatigue.
- Shortness of breath.
- Activity intolerance.
- Pica
- Glossitis – tongue irritation. And very smooth.

- Koilonychias – spoon like deformity on the finger nails.
- Cheillosis – cracks at the sides of the mouth.

Diagnosis.

- Clinical manifestation.
- Lab investigation – (full hemogram)
- Bone marrow studies.(Biopsy)

NB: Vit B_{12} and folic acid deficiency all lead to megaloblastic anaemia.

In both cases the WBC and platelets are abnormally large.

(ii) VITAMIN B_{12} DEFICIENCY.

- Also called Pernicious Anaemia (Vit B_{12} is absorbed in the ileum (last part of small intestines.).
- Vit B_{12} must combine with intrinsic factors which are produced in the stomach for absorption to occur.
- Vit B_{12} is stored in the liver.

Causes of Vit B_{12} Deficiency.

- Lack of intrinsic factors (proteins produced in the stomach for specific functions).
- Certain diseases e.g. chrohns disease, abnormal bacterial growth which is not conducive.
- Surgery of the stomach and the ileum.

Clinical features.

- Night blindness.
- Sore tongue
- Weight loss.
- Darkened skin.

- Decreases intellectual activity.
- Nervous system pathology i.e. tingling, numbness of extremities.

Diagnosis.

- Full haemogram.
- Gastric analysis.

Management. (specific)

Replacement of Vit B_{12} i.e. by injection that is given of life.

Any other management for anaemia.

(iii) Folic acid deficiency anaemia.

It is caused by lack of folic acid. It also called megaloblastic anaemia.

Sources Folic acid

Dark green leafy vegetables, small sweet bananas.

Other causes.

- Epileptic drugs.
- Oval contraceptics.
- Alcohol affects folic acid metabolism thus its absorption is reduced.
- Hemodialysis.

Long term effects.

- Congenital abnormalities in pregnancy i.e congenital abnormality e.g. spinabifida, hydrocephalus.
- In infants it causes neurologic deficiencies.
- In adults it causes anaemia (folic acid anaemia)

Diagnosis

- Full haemogram.
- Clinical presentation/manifestation.

Management.

- Folic supplements.
- Replacement for life.
- Other management for anaemia.

(iv) **Anaemia Caused by Chronic Disease**.

Is due to suppression of RBC production in the bone marrow. It is also called Iron-Re-utilization anaemia because iron stored in the bone marrow cannot be used by the developing red blood cells.
It is gradual and mild.

Clinical features.
Gradual anaemia attacks.

Management.
Blood transfusion.
Erythropoietin hormone – produced in the kidney and stimulates production of Red Blood cells.

3. **INCREASED DESTRUCTION OF RED BLOOD CELLS.**

The scavenger cells from the bone marrow, spleen and liver, detect and destroy the red blood cells and when the destruction exceeds production, hemolytic anaemia results.
NB: It is very uncommon.

Factors leading to hemolytic anaemia.

- Spleenomegally
- Mechanical damage of RBC
- Auto Immune reaction.
- RBC abnormality.

- Hemoglobin abnormalities.

(i) Spleenomegally

It the enlargement of the spleen caused by many condition i.e. malaria, kalaazar etc
The enlarged spleen traps and destroys the RBC and the more they are trapped the more the enlargement.
Its gradual and symptoms are very mild.

Management.

Treat the underlying cause of splenomegally.

(ii) Mechanical damage of RBC.

It leads to microangiopathic hemolytic anaemia.
Normally RBC should migrate without injuries, however some conditions such as aneurism(weakness of blood vessel and dilatation) leads to mechanical damage of the red blood cells.
Also extreme high blood pressure.

Diagnosis

Microscopy of the blood. (Check for damaged red blood cells).

Management.

Identify the cause and treat it.

(iii) Auto Immune Reactions.

This is the destruction of own red blood cells because it (body) identifies them as foreign body.
When it is directed at red blood cells it is called Auto Immune Hemolytic anaemia.

Classification of Auto Immune Hemolytic Anaemia.

Are of two types.

- Warm, antibody hemolytic anaemia
- Cold antibody hemolytic anaemia.

- In warm antibody hemolytic anaemia the body develops auto antibody that reacts against red blood cells in high body temperatures.

- Antibodies will coat the red blood cells and the red blood cells are destroyed by scavenger cells in spleen and bone marrow.

- It's common in women than men and could lead to lymphoma, leukemia, connective tissue disease e.g. SLE or exposure to certain drugs like antihypertensive.

Symptoms of warm antibody hemolytic anaemia.

- Spleenomegally.
- Upper left abdominal tenderness.
- Other symptoms of severe anaemia.

- Cold antibody hemolytic anaemia body reacts against red blood cells due to low temperatures.

Symptoms are mild but when exposed to cold they worsen.

Diagnosis.

Titre test – identification of antibodies.

MANAGEMENT

1. Identity and treat the cause
2. Use of corticosteroids and cytotoxic drugs
3. Splenectomy
4. Blood transfusion

5. Other general management of anaemia

(iv) Hemoglobin abnormalities

Sickle cell disease

It's an inherited condition characterized by
Sickle shaped red blood cells and chronic hemolytic anemia
In sickle cell the red blood cell contain an abnormal form of hemoglobin that reduces the amount of oxygen in the cells causing them to become Cresent shaped or sickle shaped.
The sickle shaped cell block and damage the spleen, kidney, bones and other organs.
The deformed cells are fragile i.e. delicate and easy to break as they travel through the vessels leading to:-

- Severe anemia
- Mild jaundice
- Blocked blood flow
- Organ damage and death.

Clinical Features

- Severe pain
- Anaemia
- Jaundice (destruction of RBC and the liver cannot conjugate bilirubin)

Sickle cell crisis includes:-

- Severe pain
- Fever.
- Shortness of breath after exhaustion.
- Enlarged liver
- Cardiomegally of heart murmurs
- Poor circulation of blood especially to the ankle area thus poor healing of

wounds

- Painful erection (priapism) especially for young men.

DIAGNOSIS

- Blood microscopy
- Physical presentation especially in times of crisis
- Electrophoresis (abnormal detection of HB).

Complications

- Severe anaemia
- Impotence
- Pulmonary hypertension
- Heart failure
- Renal failure

Prevention of sickle cell crisis

- Avoid strenuous exercise e.g high attitudes
- Avoid dehydration
- Early treatment of infections.

Management

- Blood transfusion
- I.V Rehydration –(dilution of clumpy blood).
- Strong analgesics like morphine
- Oxygen therapy
- Prophylactic antibiotics
- Lower body temperatures
- Bone marrow transplant
- Hydroxyurea reduces production of abnormal RBC

- Psychological counseling.

NURSING DIAGNOSIS IN ANEMIA

1. Altered tissue perfusion related to inadequate oxygen capacity as evidenced by decreased HB and red cells.
2. Activity intolerance related to impaired oxygenation as evidenced by patients' verbalization.
3. Altered nutrition less than body requirements related to increased need for substances necessary for normal ecythropoiesis as evidenced by decreased iron in blood, decreased vit B_{12} etc
4. Knowledge deficit related to health status of the patient, regarding treatment plan as evidence by patient verbalization.
5. Risk of infection related to impaired immunity.
6. Risks to injury related to compromised haematological status.

BLEEDING DISORDERS

1. THROMBOCYTOPAENIA

It is the deficiency of platelets by ≤ 30,000/mm^3

(Normal 150-450/mm^3)

4 MAIN CAUSES OF THROMBOCYTOPENIA

1. Bone marrow in effectiveness e.g. in leukemia and anaemia.
2. Spleenomegally i.e. the enlarged spleen entraps the platelets thus causing thrombocytopenia.
3. Increased use or destruction of platelets e.g. in chronic illness.
4. Platelets dilution e.g. in exchanged blood transfusion.

Clinical features

- Bleeding under skin (peupura)
- Bleeding gums
- Heavy, menstrual
- Blood in stool and urine
- Difficulty in controlling bleeding

Diagnosis

- Through clinical features and history taking
- Full haemogram (shows low platelets count)
- Clotting time test
- Bone marrow analysis.

Management

1. Blood transfusion (fresh blood)
2. Administer haematencs
3. Assess the need of oxygen if dysphoeic
4. Rx the cause of the thrombocytopoerua

5. Avoid injuries especially giving i.m injection.
6. If bone marrow ineffectiveness give erythropoietin.

2. HAEMOPHILIA.

Is a bleeding disorder caused by deficiency of one blood clotting factors eg. fibrogen.

Types of haemophilia

Haemophilia A also called **classic haemophilia**

Haemophilia B also called **christmass disease**

a) Haemophilia A (Classic)

It accounts for 80% of all the cases

There is deficiency of clotting factor Viii (8) while

b) Haemophilia B (christmass disease)

It accounts for 20% of all the cases. There is deficiency of clotting factor 1X (9)

A Bleding pattern is similar in both cases.

Both are inherited from the mother, but affects men most i.e (the mother is the carrier)

Symptoms

There is severe bleeding which may occur without any apparent reason e.g from tooth extraction.

Diagnosis

Blood analysis through full haemogram

The condition is diagnosed by 18months of age.

Management

- Blood transfusion
- Haematenics
- Avoid injuries and injections especially intramuscular.
- Follow up care.

3. DISSEMINATED INTRAVASCULAR COAGULATION (DIC)

Also called consumption coagulopathy.

It is a condition whereby small blood clots disseminated / spread through stream thereby blocking the small bood vessels thereby depleting the clotting factors which are needed to contrl bleeding so there is no hemostatis.

It begins with excessive clotting that is stimulated by a toxic substance.

People at risk

- Complicated obstetric surgery or during child birth.
- People with leukemia,CA stomach CA pancreases CA prostate.
- Severe bacterial infectons because of toxins leading to blood clotting.

Clinical features.

- Severe bleeding from incision site or from torn tissues as it follows surgery or child birth.
- Bleeding may persist in the site of iv injection or puncture.
- Massive bleeding in brain,hit,skin and other organs.

Note: clots may damage the kidneys.

Diagnosis

- Through clinical feartures and history taking.
- Blood analysis.

Management

- Transfusion platelets and other clotting factors.
- May be given heparin to slow clotting.
- Manage the cause e.g bacterial infection eg if during is can do hysterecto

4. WHITE BLOOD CELLS DISORDERS.

Normal WBC levels are 4,000 to 10,000/mm^3 these disorder may be less or more WBC.

a) Neutropaenia

This is an abnormal number of Neutrophilis in blood.

Causes of neutropaenia

Ineffective production in bone marrow.

I. Can be caused by hereditary factors,cancer,certain medications e.g anticonvuls ants and chlorophenicol which depresses bone marrow.
II. Vitamin B_{12} of folates deficiency

2. Large number being destroyed in condition like autoimmune disease eg SLED

chemotherapy.

Signs and symptoms

- Fever
- Severe infection (sepsis)
- Moth ulcers
- Diarrhoea
- Chills
- sore throat.

Diagnosis

Full blood count

Bone marrow biopsy.

Management

- Treat the cause
- Granulocytes colony stimulating factor (hcsf)
- Other general management eg giving antibiotics.

Lymphocytes

Abnormally number of lymphocytes.

Levels of lymphocytes

In children is ≤3,000/mm^3 } in lymphocytopaenia

In adults is ≤ 15,000/mm^3 }

Causes

- Severe stress
- Corticosteroids
- Chemotherapy
- Radiotherapy
- Hereditary factors
- Aids
- Cancer e.g leukemia
- Chronic infections e.g TB.

Clinical Features

- Frequent infections from fungi, bacteria and parasites.

Diagnosis

- Bone marrow biopsy
- Full blood count

Management

- Treat and manage the cause.
- Live hammaglobulin i.e substances rich in antibiotics.
- Treat the infections, the patient has presented with
- Bone marrow or stem transplantation.
- Other general management.

(C) EOSINOPHILIA

- Abnormally number of easinophils in blood
- It could be a respond to parasites, allergies or abnormal cells.

Signs and symptoms

- Inflammation
- Rashes

Management.

Treat the cause's e.g. allergies of parasites, fungi.

5 **LEUKAEMIA**

Leukaemia is the cancer of blood cells or bone marrow.

It is characterized by abnormal proliferation (multiplication) of blood cells usually leukocytes (white blood cells).

Causes

- Radioactive exposure
- Viruses
- Chemicals e.g benzene
- Anticancer drugs (chemotherapy)

NB:

The leukemia cells invade the bone borrow and eventually other organs.

Classification

1. It's classified according to how quickly they progress i.e. (a) acute or (b) Chronic.
2. Which kind of blood cells they affect? They affect the **lymphocytes** and **myelocytes.**

(a) Acute lymphocytic leukemia

- It's a life threatening disease in which the cells that normally develops into lymphocytes i.e lymphoblast become cancerous and rapidly replace the normal cells in the bone marrow.
- It is the most common cancer in children and often affects children of age 3 to 5 years and in adolescents.

Clinical features

- General body weakness
- Dyspnoea
- Frequent infections
- Fever
- Bone and joints pains.

Leukaemia cells in brain - present signs of increased intracranial pressure.

Diagnosis

1. Complete blood cell count (show normal increased or decreased blood cells)
- Decreased platelets and white blood cells.
- Immature white blood cells in circulation
2. Bone marrow biopsy.

Management

- Bone marrow transplant

- Chemotherapy/radiotherapy
- Transfusion to blood
- Erythropoietin to stimulate the bone marrow
- Antibiotics
- Prognosis- good for children between 3 to 5 years and poor for those over 20 years.
- Analgesics.

Complications

- Anaemia
- Meningitis

(b) Chronic Lympholytic Leukaemia

- It is characterized by a large number of cancerous mature lymphocytes and enlarged lymph nodes.
- 3/4 of those people are over 60years.
- It is common in men than women
- The number of cancerous mature lymphocytes increases fast in the lymph node, then to spleen and liver which then enlarges.

Clinical features

- Anaemia
- Decreased levels of antibodies
- There is confused immune system such that there is destruction of red blood cells and platlets.
- Inflammation of blood vessels
- Rheumatoid arthritis
- Enlarged lymph nodes.

Management and diagnosis same as acute lymphocytic leukemia.

Acute Myelocytic Leukemia.

- Is a life threatening disease in which the myelocytes becomes cancerous and rapidly replace the normal cells in the bone marrow.

- It affects adults mostly.

Causes of predisposing factors.

- Exposure to radiation
- Some cytoxic drugs

Symptoms, diagnosis and management.

- Same as the one for chronic myelocttic leukaemia.

Chronic myelocytic leukaemia.

- It is a disease in which the cells in the bone marrow become cancerous and produces a large number of granulocytes.
- It affects any any age and sex.
- The leukaemia cells range from very immature to mature forms.

Clinical features.

- In early stages there are no symptoms.
- Anaemia manifestations in later stages.
- There are sensations of fullness (abdomen i.e spleenomegallly.)
- Features associated to leukocytopenia (reduced W.B.C)
- Skin nodules filled to leukaemic granulocytes.

Diagnosis

- Complete blood cont
- Bone marrow analysis
- Chromosomal analytic of white blood cells.

Management.

- No cure supportive management to slow the progssion like-
- Bone marrow
- Blood transfusion
- Radiotherapy
- Spleenectomy

Drugs given- interferon alpha which normalizes the bone marrow.
Hrdroxurea- it's an antitoxic drug.

PLASMA CELLS DISORDERS.

- These are conditions in which a group of plasma cells multiply excessively and produces large quantities of abnormal antibodies.
- The antibodies normally will produce calories which fight the antigens but in this case the abnormal antibodies which are produced does not protect against infections thereby normal antibody production decreases thus one becomes more vulnerable to infections.

(a) Multiple myeloma

- This is the plasma cells cancer in which abnormal plasma cells multiplies forming tumors in the bone marrow and produces a larger quantity of abnormal antibodies that accumulate in blood or urine.
- Plasma cells tumors are common in the pelvis, spine ribs and the scalp I.e where the bone marrow is found and sometimes outside the bone i.e the lungs and the reproductive organ.
- The abnormal antibodies may end up in the kidneys and other organs leading to organ failure eg kidney failure.
- Abnormal antibodies traces in the urine are called **Bence Jones proteins.**

Clinical features

- Pain in the bones
- Weakness of the bones thus easy to fracture
- Anaemia
- Recurrent infection organs failure e.g. kidney failure
- Thickness of blood viscosity interfering with the flow to the toes and fingers

Diagnosis

- Bence Jones proteins in urine
- Bone marrow biopsy

- X-ray (scalp, pelvis, ribs)
- Full haemoglobin – there is increased ESR
- High calcium levels.

Management

- No cure, give supportive management
- Radiotherapy
- Analgesic
- Drinking alot of fluids.
- Exercise to reduce osteoporosis
- Antibiotic
- Blood transfusion
- Chemotherapy
- Erythropoietin

Special donations procedures.

There are 3 special donations procedures.

1. **Apheresis**

The donor give sonly the specific components needed by the recipient e.g. platelets, red blood cells and the rest of blood is given back to the donor.

2. **Autologous transfusion**

This is self transfusion incase of severe bleeding or when one is undergoing surgery.

3. **Directed or designated donation.**

This is blood donation for a family member or friend.

CARDIOVASCULAR DISORDERS

Symptoms of cardiac disease.

1. Severe chest pains due inadequate blood.

Supply also inadequate oxygen supply causing ishaemia.

- There are metabolic wastes which produces cramping which is painful.

2. Dysphnoe (shortness of breath)

- This is because of fluid sleeping into air spaces of the lungs leading to pulmonary oedema or congestion.

3. Light headedness and fainting due to lack of adequate blood supply to brain.
4. Palpitations – This is forceful irregular or rapid heart beat due to cardiac compensatory activity.
5. Fatigue – This is because of inadequate blood supply to the muscles especially during exercises.
   Its feeling weak and tired.

CONGESTIVE CARDIAC FAILURE (CCF)

It is severe condition in which the quantity of blood pumped by the heart each minute (cardiac output) is insufficient to meet the bodyss normal requirement for oxygen and nutrients.

There are 3 major causes of CCF.

1. **Conditions that affect the heart muscles**

Coronary heart disease which interfere with normal blood supply to the cardiac muscles and myocardilis which is the infection of the heart muscle by bacteria.

2. **Conditions that interfere with flow of blood between the heart chamber.**

e.g stenosis of the semi-lunar valves and stenosis of the atria ventricular valves. Abnormal emptying of the heart or back flow of blood due to insufficiently of the atrioventricular valves.

3. **Conditions i.e systematic disease which cause demand of extra nutrients and oxygen e.g.**

Thyrotoxicosis,anaemia and malnutrition.

PATHOPHYSIOLOGY OF CCF

Cardiac output is below normal range and (cardiac output is equal to heart rate x stroke volume)

stroke volume- is the amount of blood ejected by a ventricle during each systolic contraction because of reduced cardiac output, the heart works hard over a period of time to meet body supply of oxygen and nutrients therefore the heart muscles become enlarged and this enlargement makes the heart to increase the cardiac output but eventually the heart is not able to function well because muscles are overstretched and overstrained and this results in decreased pumping ability and heart failure.

NOTE:compensatory mechanism of CCF

Note: when there is reduced output there is going to be activation of sympathetic nervous system hence increasing heart rate, palpitations mechanism to compensate.

The **rash** (rennin angiotensin stimulating hormone)

Activated due to reduced renal blood flow hence activating (rash) system where rennin converts angiotensin 1 to angiotensin 11 where angiotensin 11.

Later hypertenhon will result.

Factors influencing cardiac output and stroke volume

1. **Heart rate:** the number at time each ventricle contracts per minute.
2. Pre-load volume i.e. of blood at end of diastole i.e. heart relaxation before ventricle contraction (systole).

Frank sterlin law of the heart states that the stretching of the myocardial fibres during diastole increases the force of contract during systole.

3. After volume, the resistance against which the ventricles must pump.

It's affected by systematic vascular resistance.

4. Contractility this is the systematic vascular resistance generated by the myocardium under given conditions.

Impaired cardiac functions results in –

1. Leads to failure to empty the venous reservoir so the systemic and pulmonary venous volumes are elerated.

2. There is reduced delivery of arterial blood into arterial circulation i.e the amount of blood ejected into the aorta and pulmonary arteries are reduced.

Clinical manifestations of CCF

There are four components of the syndrome.

1. **Right sided heart failure (cor-pulmonale)**

Symptoms

- Increased systemic venous pressure
- Jugular venous distance
- Hepatomegally
- Peripheral oedema
- There is ascites
- Shortness of breath (dyspoea)
- Excersice intolerance
- Chest discomfort.

2. **Left sided heart failure**

- Elevated pulmonary venous pressure (i.e to lungs)
- Decreased cardiac output
- Breathless
- Fatigue and weakness
- Confusion
- Pulmonary oedema
- Hypotension.

3. **Caridiogenic shock**

This is circulatory failure due o severe depression of the myocardial contractility in which the cardiac output is markedly depressed.

Symptoms

- Hypotension
- Tachycardia

- Oliguria
- Impaired mentation (pt is not mentally upright)

4. **Acute pulmonary oedema**
   - Marked dysphoea and orthophoea
   - There is expectoration of flothy sputum.
   - Pallor and cyanosis
   - Confusion.

Diagnosis of heart failure

1. Through signs and symptoms (through history taking and examination)
2. Chest x-ray which will show cardiomegally
3. Ech (electro-cardiogram) will show abnormal electric waves.
4. Through blood studies (blood analysis) there is a cardiac enzyme found in blood
5. Urinalysis

In urine there is a cardiac enzyme.

Management of CCF

Once there is heart failure, no complete care.

Aims

- To promote rest and reduce the heart workload
- To increase the heart contractions to increase cardiac output.
- To remove excess fluids from the body.

In treatment of CCF there are 3 approaches

1. To remove the underlying cause.

Through surgical intervention e.g. to repair the heart valves, removal of tumors.

Medical intervention

Gire **inotropics** e.g digoxin (digitalis) to increase the contractility and force of the heart.

Diuretics-e.g lasix which reduces the venous pressure i.e. oedema.

Ace- inhibitors (angiotensin converting enzymes inhibitors) eg captopril.

This reduces peripheral pressure and after load.

Hydralazine – to lower the blood pressure.

2. **Removal of precipitating factors.**
   - Avoid smoking
   - Low salt diet
   - Weight reduction
   - Physical and emotional rest
   - Avoid drinking alcohol.

3. **Control of heart failure.**
   - Early treatment of infection (rheumatic heart disease)
   - Frequent medical check ups
   - Improve quality of life through proper diet and exercise
   - Weight reduction.

Heart valve disorders.

Triscupid valve- opens from the right autrium to the left ventricle.

Pulmonary valve – open from right ventricle into pulmonary arteries.

Bicuspid valve – opens from left atrium into the left ventricle.

Aortic valve – opens from the left ventricle into the aorta

Heart valve malfunctioning

Valve regurgitation – leaking of blood and goes back.

Valve stenosis-failure of the valves to open adequately.

Mitral valve regurgitation.

This is leakage of blood through the mitral valve each time the left ventricle contract. There is leakage of blood back to the left atrium increasing the volume and pressure in the lungs.

Causes of mitral valve regurgitation.

- Heart attack

- Rheumative fevers
- Heart tumors.

Clinical features

- Chest pains heart murmurs
- Dull breath sounds
- Palpitations
- Artial fibrillation.

Management

1. Surgery

Valvaplasty-or replacement

2. Giving antibiotics
3. Hire drugs for atrial fibrillation

Fibrillation- is the rapid contraction of the atrium causing irregular contraction of the atrium causing irregular contraction of the ventricles in both rhythm and force.

Mitral valve stenosis

- Is the narrowing of the mitral valve opening that increases resistance to blood flow from the left atrium to the left ventricle.
- It always results from
    - Rheumatic fever
    - Congenital
    - Clots obstruct the flow of blood through the valves producing the same effects as stenosis.

Signs and symptoms

- Pulmonary oedema
- Heart failure

Prevention and management

- Prevent rheumatic fever
- Give drugs for heart failure
- Valve replacement or repair.

Mitral valve prolapse

The valve leaflets bulge into the left atrium during ventricular contraction, sometimes

allowing leakage of small amount of blood into the left atrium.

Its common in women than men.

Causes

It is inherited, there is connective tissue disorder resulting in enlargement of one or both of the mitral valve leaflets.

Symptoms

- Chest pain
- Palpitations
- Migraine headache
- Dyshoea

Prevention and management

- Patient is advised to eliminate caffeine from diet.
- Stop smoking
- Patient should be given antibiotics before dental surgery,because they will introduce infectious agents systematically into heart valves.
- Surgery= valvo plasty.
- Drugs are calcium channel blockers.

HYPOTENSION (low blood pressure)

This is low blood pressure o0f less than 80mm/hg i.e systolic or less than 60mm/hg diastolic.

Causes

- Pregnancy
- Medications e.g diuretics, antihypertensives
- Heart problems e.g CCF
- Endocrine problems eg hypothyroidism and hypothyroidism.
    - Addison disease i.e adrenal insufficiency.
- Hypoglycemia
- Dehydration i.e fluid loss
- Blood loss eg during an RTA
- Severe infection (septicemia)

- Allergic reactions (anaphylactic shock)
- Nutritional deficiency (lack of vit B_{12} and Follett leads to anaemia.

Clinical features.

- Dizziness
- Blurred vision
- Fainting
- Nausea
- Cold clummy skin
- Rapid shallow breathing
- Fatigue
- Depression
- Thirst

Types of low blood pressure.

1. Postural (orthostatic hypotension)
2. Multiple system atrophy (shy-dragger syndrome)
3. Postproudal hypotension
4. Neural mediated hypotension.

1. **Postural hypotension (orthostatic)**

This is sudden drop of blood pressure when one stands up after lying down.
This type of hypotension in older adults can also affect young healthy people who stand up suddenly after sitting with their legs crossed for long periods or after doing exercises for sometime in squatting position.

2. **Multiple system atrophy**
   - This is due to nervous system damage
   - Also called shy-dragger syndrome.
   - This is progressive damage to the autonomic nervous system (ans) which control involuntary system heart rate, breathing and digestion

3. **Postpraidal hypotension.**

This is low blood pressure after eating.
A large amount of blood flows to the digestive tract and your body counteracts this by increasing the heart rate and constricting sudden blood vessels to help maintain normal blood pressure but in some people mechanism fails.

4. **Neutrally medicated hypotension.**

This is low blood pressure resulting from faulty brain signals.
Unlike orthostatic hypotension, it causes BP to drop after standing for longer period of time.
This affects young people.
Occurs because of a miscommunication between the heart and the brain.

Risk factors of hypotension.

Age: post praidal and orthostatic hypotension occurs in older adults while neutrally medicated hypotension affects children and younger adults.

Medication; eg diuretics, antihypertensive.
Certain diseases e.g heart disorders and also Parkinson's disease.

Diagnosis of hypotension.

Through monitoring of vital signs i.e BP
Through signs and symptoms.
Through blood tests i.e low HB,hypoglycaemia.
Through EEG shows irregulativities in heart rhythm.

Lifestyles and home remedies.

- Advice people to take more water and less alcohol because alcohol stimulate diuretics to slow, take it easy when you move from a prone or sitting up position from standing position, before getting out of bed in the morning breathe deeply for a few minutes and slowly sit down before standing.
- Sleeping with your head slightly elevated.

NOTES UNDER CCF

Two group indicators of heart diseases ccf.

1. **Hemodynamic indicators.**

There is elevated nventricular and diastolic pressures

Elerated systematic and pulmonary pressure and decreased cardiac output.

Systemic = SVC
LVC

Pulmonary –Lungs.

2. **Clinical indicators**

Activated compensatory mechanism.

In sympathetic system, there is increased heart rte and breath sounds.

In kidneys-there is fluid retention and peripheral vasoconstriction.

- Stretching of cardiac muscles and dilatation to increase the cardiac volume.
- There is myocardial hypertrophy (enlargement)
- There is accumulation of extracellular fluid which leads to oedema and there is impaired organ perfusion.

SHOCK.

This is serious life threatening medical condition where insufficient blood flow reaches the body issues.

As the blood carries nutrients and oxygen around the body, reduced blood flow hinders the delivery of these components to the tissues and can support the tissues from functioning properly.

Stages of shock (ICPR)

1. **Initial stage.**

The hypoperfusional stage causes hypoxia leaching to the, mitochondria being unable to produce ATP and due to lack of oxygen the cell membrane becomes damaged and becomes leaky to extracellular fluid.

Celle will therefore perform anaerobic respiration therefore build up of lactic and phyric acid leading to systemic acidosis. The process of removing the compounds (lactic and pyruvic acid) by the liver requires oxygen which absent.

2. **Compensatory stage.**

This is characterized by the b0ody employing physiological mechanism including hormonal and biochemical mechanisms in an attempt to reverse the condition.
As a result of acidosis there will be hyperventilation in order to get rid of the carbon dioxide. The boro-receptors in the arteries detect the hypotension thus adrehaline and noradrehaline which causes vasoconstriction on with a mild increase in heart rate resulting in an increase in BP antidiuretic hormone (ADH) or vasopressin is thefore released to conserve the fluid in the kidneys.

3. **Progressive or decompensating stage.**

   Here the compasatory mechanisms begin to fail and because or decreased perfusion in cells, sodium ions build up within the cells while K^+ leaks out.
   as anaerobic metabolism continue increasing the body's metabolic acidosis, there will be increased hydrostatic pressure and combined with histamine release there will be leakage of fluid and proteins into the surrounding tissues. As this fluid is lost, the blood concentration and viscosity is going to increase.

4. **Refractory or irreversible stage.**

   At this stage, the vital organs have failed and shock can no longer be reversed. Brain damage and cell death have occurred and death will occur imminently i.e. (death is unavoidable).

Types of shock

1. Hyporolaemic shock
2. Cardiac shock
3. Distributive shock

- Septic shock
- Anaphylactic shock
- Neurogenic shock

4. Obstructive shock.

1. Hyporolaemic shock.

This is the most common type of shock and it's based on insufficient circulatory volume.

Causes

- Bleeding (hemorrhage)
- Severe loss of fluid
- Severe burns.

2. Cardiogenic shock.

It's caused by the failure of the heart to pump effectively.

It can be due to the damage of heart muscle most often from a myocardial infarction.

Other causes

- Arrhythmias
- CCF
- Cardiomyopathy i.e affects muscles of the heart e.g pericarditis and endocarditis.
- Cardiac valve problems.

3. Distributive shock

It is same as hyporoelemic shack but here thre is an insufficient intravascular volume of blood.

TYPE

(a) Septic shock

This is caused by an overwhelming infection leading to vasodilatation e.g gram negative bacteria e.g neiseria gonococci .E. coli,klebsiella.

The micro-organisms releases an endotoxin which causes adverse biochemical immunological and occasionally neurologically effects which are hamful to the body same appliy to the gram positive cocci also produce same exotoxins.

(b) Anaphylactic shock.

Cuased by severe analhylactic reaction to an allergen , antigen,drug or foreign protein causing the release of histamine which causes widespread vasodilation.

(c) Neurogenic shock.

It is the rarest form of shock.

It is caused by trauma to the spinal cord resulting in sudden loss of autonomic and motor reflexes below the injury level.

4. Obstructive shock

Here the flow of blood is obstructed which impedes (prevents0 circulation and can result in circulatory arrest.

(1) Cardiac temponade

In which fluid in the pericardium prevents inflow of blood into the heart.

i.e fluid in the pericardium causes pressure on the blood vessel.

(2). **Constructive pericarditis.**

This is whereby the pericardium shrinks and hardens thus prevents inflow of blood.

3. Tension pneumothorax.

Through increased intrathoracic Pulmonary pressure blood flow to the heart is prevented.

4. **Pulmonary embolism.**

An Emboli, thrombus prevents blood flow from lungs to the heart causing shock.

Signs and symptoms of hyporolamic shock.

- Anxiety
- Restlessness
- Altered mental state due to decreased cerebral perfusion and hypoxia.
- Hypotension because of decreased circulatory volume.
- A rapid weak thread pulse due to decreased flow of blood combined with tachycardia.
- Cool clumpsy skin due to vasoconstriction
- Rapid and shallow respiration due to stimulation of sympathetic nervous system because of acidosis.
- Hypothermia due to decreased perfusion and evaporation of sweat.
- Thirst and dry moth due toi fluid depletion.
- Cold and mottled skin (cutis mormorata) especially in extremities due to insufficient perfusion of the skin.
- Distracted look in the eyes or staring into space often with dilated pupil.

Cardiogenic shock.

Signs and symptoms.

Signs are similar to hyporolemic shock but in addition there is distended jugular vein due to increase jugular venous pressure.

- There is also absent pulse due to tachy arrhythmias.

Obstructive shock.

Signs and symptoms.

- Also similar as hypovolemic shock

- Also there are distended jugular veins.

Distributive shock

Signs and symptoms.

Septic shock- its signs are similar with hyporalemic shock. But there is pyrexia or hyperthermia du to overwhelming bacterial infection.

Neurogenic s/s

Similar to hypovolemic shock expect in skin characteristics.

Anaphylactic shock

Signs and symptoms.

- There are skin eruptions
- Localized oedema especially around the face.
- Weak and rapid pulse
- Breathless and cough due to the narrowing of the airways.

Management of hyporolamic shock.

- A, B, C airway, breathing and circulation, resuscitation
- Hydrate (fluid and electrolytes conservation)
- Blood transfusion
- Inotropic agents' e.g. dopamine, increase force and pressure of the heartbeat.
- Treat the underlying shock.

Management of cardiogenic shock

- Fluid replacement
- Keep pt warm (use blankets)
- Antidiuretics
- Use of antishock trouser –they compress the legs and concentrate blood into vital organs

Management of Distributive shock.

Septic

- Use of strong parental antibiotics to treat the bacterial infection.
- Supportive care e.g. use of oxygen, re-assure.

Anaphylactic mx

- Put the pt in trendel burg position i.e. legs up and head lower than the limbs.
- Use of antidiuretics etc.

Obstructive shock mx

- Remove the obstruction first.

Pneumothorax and haemothorax

In these cases insert an underwater seal drainage.

Pulmonary emboli mx

- Thrombolitics drugs.
- Embolectomy

Cardiac temponade.

Is treated by draining fluid form the pericardial space by use of procedure called periocadiocentesis.

CORONARY ARTERY DISEASE.

- Also called coronary heart disease or coronary atheroschlerosis.
- It is the progressive narrowing of the artery that nourishes the heart muscles.
- Often they are no symptoms but if one or more arteries become severly narrowed Angina pectoris may develop during exercise, stress or other times when the heart muscle is not getting enough blood.

Cause

The narrowing is due to the build up of fatty pledge i.e. atheroschlerosis along the artery

walls.

These deposits are composed of cholestral and othe lipids.

Treatment

- Give beta – blocking drugs e.g. nitrates which act by blocking the effect of sympathetic nervous system on the heart thus slowing the heart rate.
- Give calcium channel blocker by reducing the amount of calcium that enters the muscles in the coronary artery walls spasms can be prevented.
- Surgery called angioplasty and coronary bypass i.e. removing and replacement of the clogged coromy artery.
- **Note**: To include the nursing care management of coronary heart disease.