COURSE GUIDE

NSC 306
MEDICALSURGICAL NURSING II

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INTRODUCTION

Welcome to the first course in Medical Surgical Nursing. This is the first of the four courses in this specialty area of Nursing. It focuses on updating your knowledge and improving your competency in the care of patients with medical and or surgical conditions. The nurse plays a core and significant role in providing care for patients who have medical and or surgical conditions in the hospital. This course builds on your previous knowledge and experiences and hopes to see you improve the quality of care given to your patients one-on-one on a daily basis as you apply new knowledge to provide evidence based care in your place of work as well as engage in intellectual presentations in patient care as professionals. The course has theoretical and practical components. This course guide provides you with basic information about how to navigate through the course. It is importnant that you read the guide and seek further information as you may need to get the best out of this course. Best wishes.

COURSE OVERVIEW

Medical Surgical Nursing (I)
Medical Surgical Nursing (I) is the first of the four Medical Surgical Nursing courses in your degree programme. It is registrable at the first semester of the third year. The course shall improve on your previous knowledge to enhance better understanding of principles, concepts and theories of Medical Surgical Nursing. It also briefly presents the models and theories of nursing that are used to inform current nursing care planning and implementation. The care of patients with diverse medical-surgical conditions are discussed with activities expected of you to be done to aid application of new knowledge to your current practice. The course has the theory, laboratory components as well as clinical practice that spread over 15 weeks. The course is presented in Modules with small units. Each unit is presented to follow the same pattern that guides your learning. Each module and unit have the learning objectives that helps you track what to learn and what you should be able to do after completion. Small units of contents will be presented every week with guidelines of what you should do to enhance knowledge retention as had been laid out in the course materials. Practical sessions will be negotiated online with you as desirable with information about venue, date and title of practical session.

COURSE OBJECTIVE

At the completion of this course, you should be able to:

- Discuss the concepts and theories of nursing care
- Apply new knowledge in providing care for patients with alterations in fluid and electrolyte balance, shock, stress, pain temperature control and skin care
- Discuss physical and psychosocial needs of clients/patients with special medical/surgical conditions with adequate nursing care.
- Discuss the cause, and the management of inflammation.

COURSE IMPLEMENTATION
DOING THE COURSE

The course will be delivered adopting the blended learning mode; 70% of online interactive sessions and 30% of face-to-face laboratory sessions. You are expected to register for this course online in order to gain access to all the materials and class sessions online. You will have access to both hard and soft copies of course materials as well as online interactive sessions and face-to-face interaction with instructors during practical sessions in the laboratory. The interactive online activities will be available to you on the course link on the Website of NOUN. There are activities and assignments online for every unit every week. It is important that you visit the course sites weekly and do all assignments to meet deadlines and to contribute to the topical issues that would be raised for everyone's contribution.

You will be expected to read every module along with all assigned readings to prepare you for meaningful contributions to all sessions and completion of all activities. It is important that you attempt all the Self Assessment Questions (SAQ) at the end of every unit to help your understanding of the contents and to help you prepare for the in-course tests and the final examination. You will also be expected to keep a portfolio where you keep all your completed assignments.

COURSE REQUIREMENTS AND EXPECTATIONS OF YOU

Attendance of 95% of all interactive sessions, submission of all assignments to meet deadlines; participation in all CMA, attendance of all laboratory sessions with evidence as provided in the log book, submission of reports from all laboratory practical sessions and attendance of the final course examination. You are also expected to:

1. Be versatile in basic computer skills
2. Participate in all laboratory practical up to 90% of the time
3. Submit personal reports from laboratory practical sessions on schedule

4. Log in to the class online discussion board at least once a week and contribute to ongoing discussions.
5. Contribute actively to group seminar presentations.

EQUIPMENT AND SOFTWARE NEEDED TO ACCESS COURSE

You will be expected to have the following tools:
1. A computer (laptop or desktop or a tablet)
2. Internet access, preferably broadband rather than dial-up access
3. MS Office software – Word PROCESSOR, Powerpoint, Spreadsheet
4. Browser – Preferably Internet Explorer, Moxilla Firefox
5. Adobe Acrobat Reader

NUMBER AND PLACES OF MEETING (ONLINE, FACE-TOFACE, LABORATORY PRACTICALS)

The details of these will be provided to you at the time of commencement of this course

DISCUSSION FORUM

There will be an online discussion forum and topics for discussion will be available for your contributions. It is mandatory that you participate in every discussion every week. You participation link you, your face, your ideas and views to that of every member of the class and earns you some mark.

COURSE EVALUATION

There are two forms of evaluation of the progress you are making in this course. The first are the series of activities, assignments and end of unit, computer or tutor marked assignments, and laboratory practical sessions and report that constitute the continuous assessment that all carry 30% of the total mark. The second is a written examination with multiple choice, short answers and essay questions that take 70% of the total mark that you will do on completion of the course.
Students evaluation: The students will be assessed and evaluated based on the following criteria

- **In-Course Examination:**

In line with the university's regulation, in-course examination will come up in the middle of the semester these would come in form of Computer Marked Assignment. This will be in addition to 1compulsory Tutor Marked Assignment (TMA's) and three Computer marked Assignment that comes after every module…..

- **Laboratory practical:** Attendance, record of participation and other assignments will be graded and added to the other scores form other forms of examinations.
- **Final Examination:** The final written examination will come up at the end of the semester comprising essay and objective questions covering all the contents covered in the course. The final examination will amount to 60% of the total grade for the course.

Learner-Facilitator evaluation of the course
This will be done through group review, written assessment of learning (theory and laboratory practical) by you and the facilitators.

GRADING CRITERIA

Grades will be based on the following Percentages

Tutor Marked Individual Assignments			10%
Computer marked Assignment		10%	
Group assignment	5%		40%
Discussion Topic participation		5%	
Laboratory practical		10%	
End of Course examination	60%		

GRADING SCALE
A = 70-100
B = 60 - 69
C= 50 - 59
F = ≤49

SCHEDULE OF ASSIGNMENTS WITH DATES

To be provided for each module by the facilitator in addition to the ones already spelt out in the course materials.

Specific Reading Assignments
To be provided by each module.

REFERENCE TEXTBOOKS

Daniel, R., Nicoll, L.H. [2012] Contemporary Medical-Surgical Nursing, [2nded]. New York: Delmar.

Kluwer, W. [2012] Medical-Surgical Nursing made incredibly easy [3rded], Philadelphia PA: Lippincott Williams and Wilkins.

Smeltzer, S.,et al. [2010] Brunner and Suddarth's Textbook of Medical-Surgical Nursing, [12thed]. Philadelphia, PA: Lippincott Williams and Wilkins.

MAIN COURSE

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MODULE 1 DISEASES OF CARDIOVASCULAR AND CIRCULATORY SYSTEM

UNIT 1 ASSESSMENT OF CARDIOVASCULAR FUNCTION

CONTENTS

1.0 INTRODUCTION

Throughout the continuum of care, whether at home, in the hospital, or community setting, all patients with cardiovascular disorders (involving the heart and major blood vessels; CVD) require similar assessments and monitoring. Key components of the cardiovascular assessment include obtaining a health history, performing a physical assessment, and monitoring a variety of laboratory and diagnostic test results. A correct and timely assessment of cardiovascular function provides the necessary data for identification of nursing diagnoses, formulation of a plan of care, and evaluation of patient's responses to the care provided. Essential to the development of these assessment skills is an understanding of the structure and function of the heart in health and in illness.

MODULE 2 DISEASES OF THE BLOOD AND LYMPH

Unit 1 Hematologic and Lymphatic Assessment & Diagnostic Evaluation
Unit 2 Review of Anatomy of Blood and the Lymphatic System
Unit 3 Caring for Patient With red Blood Disorder; Anaemia, Polycythemia
Unit 4 Caring for Patients with White Blood cell Disorder - Leukamia, Multiple Myeloma, Agranulocytosis
Unit 5 Caring for Patients with Platelet and Coagulation Disorders; Thrombocytopenia, Haemophilia & Disseminated Intravascular Coagulation
Unit 6 Caring for Patients with Lymphatic Disorder; Lymphagitis & Lymphadenitis, Lymphedema & Elephantiasis; Infectious Mononucleosis & Malignant Lymphoma.

UNIT 1 HEMATOLOGIC & LYMPHATIC ASSESSMENT & DIAGNOSTIC EVALUATION

OBJECTIVES

At the end of this unit, you should be able to:

- describe the blood
- describe the lymph
- list the signs and symptoms suggestive of hematological disorders.
- enumerate and describe the various indices of Complete Blood Count
- describe the investigations that can be used to assess the lymphatic system.

The hematologic system which involves the movement of blood and the lymphatic system which involves the movement of lymph are two major systems in the human body which encompasses the entire human body.

Blood is a connective tissue that provides one of the means of communication between the cells of different the cells of different parts of the body and the external environment. Blood makes about 7% of the body weight and the blood is continuously being pumped around the body to maintain a fairly constant environment through homeostatic mechanism for body cells.

Lymph is derived from interstitial fluid that flows in the lymphatic system. The lymphatic system is an accessory route through which fluid

MODULE 3 CARING FOR PATIENTS WITH DISORDER OF GASTROINTESTIMAL TRACT

UNIT 1 GASTROINTESTINAL SYSTEM, LIVER AND BILIARY TRACT ASSESSMENT AND EVALUATION

CONTENTS

MODULE 4 CARING FOR PATIENTS WITH HEPATOBILIARY TRACT AND PANCREATIC DISORDER

UNIT 1 ASSESSMENT AND DIAGNOSTIC EVALUATION

CONTENTS

1.0 INTRODUCTION

We regularly ingest chemicals inform of food stuffs and drugs. These are digested, used up by body tissues and leads to release of toxic metabolic products some of which are cannot be directly eliminated, hence the need for conversion to a less toxic substance to facilitate their easy and rapid excretion. This is part of the main function of the hepatobiliary system.

The focus of this unit will be to explore comprehensively the assessment of the hepatobiliary system with a view to helping you to further understand this concept and improve your clinical competence in caring for patients with these conditions.

This system thus plays an important role in maintaining balanced chemical environment and by extension health and wellbeing.

You will be expected to consolidate on your knowledge base as well as sharpen your skills in caring for patients with hepatobiliary disorders.

2.0 OBJECTIVE

At the end of this unit, you should be able to:

- assess patients with hepatobiliary disorders and
- manage patients with disorders of the hepatobiliary system

- discuss the subjective assessment of the hepatobiliary system
- discuss the objective assessment of the hepatobiliary system
- discuss various liver function test
- discuss the responsibilities of the nurse for a patient undergoing liver biopsy

3.0 MAIN CONTENT

3.1 Subjective Assessment (Health History);

This will focus on;

- Exposure of the patient to hepatotoxic substances or infectious agents.
- Occupational, recreational and travel history; may assist in identifying exposure to hepatotoxins (e.g., industrial chemicals, other toxins) responsible for illness.
- History of alcohol and drug use, including but not limited to the use of injectable drugs
- Medications history; should include all over-the counter medications, herbal remedies and dietary supplements used by the patient currently and in the past.
- Lifestyle behaviors that increase the risk for exposure to infectious agents such as injection drug use and sexual practices
- Foreign travel are all potential risk factors for liver disease.
- Amount and type of alcohol consumption
- Past medical history to identify risk factors for the development of liver disease.
- Current and past medical conditions, including psychological or psychiatric illnesses
- Family history about familial liver disorders as well as other familial or genetic diseases, such as hemochromatosis, Wilson's disease, or alpha-1 antitrypsin disease
- Reviewing symptoms that suggest liver disease such as jaundice, malaise, weakness, fatigue, pruritus, abdominal pain, fever, anorexia, weight gain, edema, increasing abdominal girth, hematemesis, malena, hematochezia (passage of bloody stools), easy bruising, decreased libido in men and secondary amenorrhea in women, changes in mental acuity, personality changes and sleep disturbances.

3.2 Objective Assessment (Physical Examination)

- Assesses patient for physical signs that may occur with liver dysfunction, including pallor (chronic illness) and jaundice
- Inspect the skin, mucosa and sclerae for jaundice,

- Assess the extremities for muscle atrophy, edema and skin excoriation secondary to scratching.
- Observe the skin for petechiae or ecchymotic areas (bruises), spider angiomas and palmar erythema.
- Assess male patient for unilateral or bilateral gynecomastia and testicular atrophy due to endocrine changes.
- Assess patient's cognitive status (recall, memory, abstract thinking) and neurologic status
- Observe for general tremor, asterixis, weakness and slurred speech.
- Abdominal assessment;
- Assess for dilated abdominal wall veins, ascites and a fluid wave
- Palpated to assess liver size and to detect any tenderness over the liver; the liver may be palpable in the right upper quadrant as a firm mass with sharp ridge and smooth surface
- Estimates liver size by percussing its upper and lower borders.
- Notes and records the size, consistency and outline (regular or irregular) of a palpable liver.
- Discern the size of an enlarged liver by estimating the degree to which it descends below the right costal margin
- A cirrhotic liver is small and hard
- Acute hepatitis presents a soft liver with smooth surface
- Tenderness of the liver implies recent acute enlargement with consequent stretching of the liver capsule.
- The absence of tenderness may imply a long-standing enlargement
- Enlargement of the liver is an abnormal finding requiring evaluation.

3.3 Diagnostic Evaluation

Liver Function Tests

Function is generally measured in terms of;

- Serum enzyme activity (i.e., alkaline phosphatase, lactic dehydrogenase, serum aminotransferases)
- Serum concentrations of proteins (albumin and globulins), bilirubin, ammonia, clotting factors, and lipids

Several of these tests may help establish liver disease but inadequate to determine the nature and extent of hepatic dysfunction

Serum aminotransferases (also called transaminases);

- Sensitive indicators of injury to the liver cells
- They are useful in detecting acute liver disease such as hepatitis.

Alanine aminotransferase (ALT) (formerly called serum glutamic-pyruvic transaminase [SGPT])

- Its level increase primarily in liver disorders
- It therefore may be used to monitor the course of hepatitis or cirrhosis or the effects of treatments that may be toxic to the liver.

Aspartate aminotransferase (AST) (formerly called serum glutamic-oxaloacetic transaminase [SGOT]);

- It is present in tissues that have high metabolic activity
- Its serum level may be increased if there is damage to or death of tissues of organs such as the heart, liver, skeletal muscle and kidney.
- Although not specific to liver disease, levels of AST may be increased in cirrhosis, hepatitis, and liver cancer.

Gamma glutamyltransferase (GGT) (also called G-glutamyltranspeptidase);

- Increased GGT levels are associated with cholestasis but can also be due to alcoholic liver disease.
- Although the kidney has the highest level of the enzyme, the liver is considered the source of normal serum activity.
- Its main value in liver disease is confirming the hepatic origin of an elevated alkaline phosphatase level.

Serum Alkaline Phosphatase:

- Serum alkaline phosphatase is manufactured in bones, liver, kidneys, and intestine and excreted through biliary tract.
- In absence of bone disease, it is a sensitive measure of biliary tract obstruction.

Pigment Studies;

- This include
  - Direct Serum bilirubin
  - Total Serum bilirubin
  - Urine bilirubin
  - Urine urobilinogen
  - Fecal urobilinogen (infrequently used);
- These studies measure the ability of the liver to conjugate and excrete bilirubin. Results are abnormal in liver and biliary tract disease and are associated with jaundice clinically.

Protein Studies;

- This includes;
  - Total serum protein (Serum albumin, Serum globulin)

- Serum protein electrophoresis (Albumin, α1-Globulin, α2-Globulin. β-Globulin, γ-Globulin)
- Albumin/globulin (A/G) ratio:

- Proteins are manufactured by the liver. Their levels may be affected in a variety of liver impairments.
  - Albumin: Cirrhosis, Chronic hepatitis, Edema, ascites
  - Globulin: Cirrhosis, Liver disease, Chronic obstructive jaundice, Viral hepatitis
  - A/G ratio is reversed in chronic liver disease (decreased albumin and increased globulin).

Prothrombin Time:

- 100% or 12–16 seconds
- Prothrombin time may be prolonged in liver disease.
- It will not return to normal with vitamin K in severe liver cell damage.

Cholesterol;

- This includes;
  - Ester
  - HDL (high-density lipoprotein)
  - LDL (low-density lipoprotein)
- Cholesterol levels are elevated in biliary obstruction and decreased in parenchymal liver disease.

Serum Ammonia:

- Liver converts ammonia to urea.
- Ammonia level rises in liver failure.

3.4 Liver Biopsy

Liver biopsy is the removal of a small amount of liver tissue
It is usually done through needle aspiration.
It permits examination of liver cells.

Purposes;

- To evaluate diffuse disorders of the parenchyma
- To diagnose space-occupying lesions.

Liver biopsy is especially useful when clinical findings and laboratory tests are not diagnostic.

Approaches to liver biopsy;

- Percutaneous approach using ultrasound guidance

- Transvenous approach through the right internal jugular vein to right hepatic vein under fluoroscopic control
- Laparoscopic approach.

Nursing responsibilities

Before the Procedure

- Ascertain results of coagulation tests (prothrombin time, partial thromboplastin time, and platelet count) are available and that compatible donor blood is available.
- Check for signed consent; confirm that informed consent has been provided.
- Measure and record the patient's pulse, respirations, and blood pressure immediately before biopsy.
- Describe to the patient in advance: steps of the procedure; sensations expected; after-effects anticipated; restrictions of activity and monitoring procedures to follow.

During the Procedure

- Support the patient during the procedure.
- Expose the right side of the patient's upper abdomen (right hypochondriac).
- Instruct the patient to inhale and exhale deeply several times, finally to exhale, and to hold breath at the end of expiration.
- The physician promptly introduces the biopsy needle by way of the transthoracic (intercostal) or transabdominal (subcostal) route, penetrates the liver, aspirates, and withdraws.
- Instruct the patient to resume breathing.

After the Procedure

- Immediately after the biopsy, assist the patient to turn onto the right side; place a pillow under the costal margin, and caution the patient to remain in this position, recumbent and immobile, for several hours. Instruct the patient to avoid coughing or straining.
- Measure and record the patient's pulse, respiratory rate, and blood pressure at 10- to 15-minute intervals for the first hour, then every 30 minutes for the next 1 to 2 hours or until the patient's condition stabilizes.
- If the patient is discharged after the procedure, instruct the patient to avoid heavy lifting and strenuous activity for 1 week.

Complications

- Bleeding
- Bile peritonitis

4.0 SUMMARY

At this juncture, you are expected to have learnt about:

- subjective assessment of the hepatobiliary system
- objective assessment of the hepatobiliary system
- liver function tests
- the responsibilities of the nurse for a patient undergoing liver biopsy.

Activities

Now that you have learnt this much, visit a hospital of choice within your vicinity and manage a patient undergoing any form of the diagnostic modality discussed within the content of this discuss. Share your answers with your colleague in the discussion forum.

SELF-ASSESSMENT EXERCISE

Mr. Akinade is a 50 year old with history of 30years of binge drinking and smoking. He presents within your facility with complaints of 3 weeks of jaundice and intense pruritus. Examination revealed an enlarged liver and pale, greasy, bulky stool with marked offensive odour. He was scheduled for liver biopsy;

- what are the indications of liver biopsy?
- discuss liver function test
- discuss the responsibilities of the nurse for a Mr. Akinade
- mention the possible complications that may occur.

You can compare your answers with the content of the course and reference materials. Endeavour to share your answer with your colleagues on the discussion platform.

5.0 REFERENCES/FURTHER READING

Levey, R., Williams-Wilson, B. (2002). Anorexia Nervosa: eMedicine Journal, April 2 2002, Volume 3, Number 4.

Smeltzer, S.C., Brenda, B. (2006). Brunner and Suddhart's Textbook of Medical-Surgical Nursing, 10th edition. Lippincott-William & Wilkins.

Templer, J. W. (2001). Parotitis; eMedicine journal; July, 2001, Vol. 2, Number, 7.

UNIT 3 CARING FOR PATIENT WITH LIVER DISORDERS

CONTENTS

1.0 INTRODUCTION

The focus of this unit is to explore various disorders of the liver with a view to improving your knowledge base and sharpen your clinical competence in caring for patients with these conditions.

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- describe various forms of hepatitis
- discuss the disturbances of the structures and functions of the oral cavity
- identify the etiologic factors of nutritional and the Oral Cavity disorders
- explain the Pathophysiology of nutritional and the Oral Cavity disorders
- adopt the nursing process approach to provide care for patients with disorders of nutrition and oral cavity

3.0 MAIN CONTENT

3.1 Viral Hepatitis

Viral hepatitis involves inflammation and necrosis of liver cells producing a characteristic cluster of clinical, biochemical and cellular changes.

- Types of viral hepatitis

To date, five definitive types of viral hepatitis have been identified: hepatitis A, B, C, D and E.

All are caused by viruses named by the corresponding alphabet
The increasing incidence of viral hepatitis is a public health concern: this is based on:

- Easy transmission
- High morbidity and
- High economic implications

It is estimated that 60% to 90% of cases are subclinical, mild cases and often misdiagnosed

Hepatitis a Virus (HAV)

- HAV accounts for 20% to 25% of cases of clinical hepatitis in the developed world.
- Hepatitis A is formerly called infectious hepatitis
- It is caused by an RNA virus of the Enterovirus family.
- The mode of transmission is the fecal–oral, i.e. ingestion of contaminated food or liquids
- An infected food handler can spread the disease
- It is rarely, if ever, transmitted by blood transfusions.
- Hepatitis A can be transmitted during sexual activity; more likely with oral–anal contact, anal intercourse especially with sex partners
- Infected patients can shed the virus in stool before the onset of symptoms and during the first few days of illness.
- There is only transient viremia; evidenced by transient jaundice

Predisposing factors:

- Poor hygiene
- Hand-to-mouth contact or close contact with infected individuals
- Overcrowding and
- Poor sanitation (sewage-contaminated waters).

The illness spectrum:

- The incubation period is estimated to be 15 to 50 days, with an average of 30 days
- The illness may be prolonged, lasting 4 to 8 weeks.
- It generally lasts longer and is more severe in those older than 40 years of age.

Clinical Manifestations

- Most cases anicteric (without jaundice) and asymptomatic
- Symptoms may be mild and include;
- Acute flu-like upper respiratory tract infection
- Low-grade fever
- Anorexia: an early symptom, often severe.
- Jaundice and dark urine may become apparent: often peaks 10 days after its initial appearance
- Varying degrees of indigestion marked by vague epigastric distress, nausea, heartburn and flatulence.
- Hepatosplenomegaly for few days after onset of the disease
- Strong aversion to strong odors and taste e.g. that of cigarettes.

Assessment and Diagnostic Findings

- Hepatitis A antigen may be found in the stool a week to 10 days before illness and for 2 to 3 weeks after symptoms appear.
- HAV antibodies; detectable in the serum but not usually until symptoms appear.
- Immunoglobulins subclasses analysis; determine antibody against acute or past infections.

Medical Management

- Stable patient is usually managed on outpatient basis
- Bed rest during the acute stage
- This should be guided by patient's sense of well-being as well as laboratory test
- Balanced diet
- Small frequent feedings during the period of anorexia
- Supplementations with IV glucose
- Gentle persistence and creativity to stimulate appetite
- Optimal food and fluid levels to counteract weight loss and slow recovery
- Restriction of physical activity
- Gradual but progressive ambulation
- Adequate rest after activity to avoid fatigue.

- Instruct to seek additional health care if the symptoms persist or worsen
- Avoid alcohol
- Specific family guidance about diet, rest, follow-up blood work, environmental sanitation (safe food and water supply, as well as effective sewage disposal) and hygiene measures (particularly hand washing (after bowel movements and before eating).

Prevention

A number of strategies exist to prevent transmission of HAV.

- HAV vaccines include Havrix and Vagta:
  - recommended two-dose vaccine for adults above 18years, with the second dose 6 to 12 months after the first: Children and adolescents 2 to 18 years requires three doses, with the second dose 1 month after the first and the third dose 6 to 12 months later. It will protect against hepatitis A for at least 20 years
  - Indications include:
- Travelers to locations where sanitation and hygiene are unsatisfactory.
- High-risk groups (homosexual men, injection/intravenous drug users, staff of day care centers, and health care personnel)
- Intramuscular Immune Globulin booster: required during the incubation period (within 2 weeks of exposure) boost the person's antibody production and provides 6 to 8 weeks of passive immunity.
- Pre-exposure prophylaxis: with hepatitis A vaccine for high risk people who do not have sufficient time to acquire immune protection.

Prognosis:

- Recovery is almost certain
- Hepatitis A rarely progresses to acute liver necrosis, fulminant hepatitis and terminating in cirrhosis of the liver or death.
- Hepatitis A confers immunity against itself
- No carrier state exists and no chronic condition.

Mortality

- The mortality rate of hepatitis A is approximately 0.5% for those under 40 years of age
- This increases to 1% to 2% for those over 40.

Hepatitis B Virus (HBV)

- HBV is a DNA virus composed of the following antigenic particles:

- HBcAg—hepatitis B core antigen (antigenic material in an inner core)
- HBsAg—hepatitis B surface antigen (antigenic material on surface of HBV)
- HBeAg—an independent protein circulating in the blood
- HBxAg—gene product of X gene of HBV/DNA.

Modes of transmission

- Primarily through blood (percutaneous and permucosal routes)
- Through other body fluids such as saliva, semen, and vaginal secretions (through breaks in the skin and mucous membranes)
- From carrier mothers to their babies, especially in areas with high incidence (usually not via the umbilical vein, but at the time of birth and during close contact afterward).

Pathophysiology

- HBV has a long incubation period.
- It replicates in the liver and remains in the serum for relatively long periods, allowing transmission of the virus.
- High risk group:
  - Clinicians and other health care workers (surgeons, clinical laboratory workers, dentists, nurses, respiratory therapists)
  - Staff and patients in hemodialysis and oncology units
  - Sexually active homosexual and bisexual men
  - Injection drug users
- Most cases will recover spontaneously in 6 months and develop antibodies.
- Mortality rate is as high as 10%.
- About 10% of cases will progress to a carrier state or chronicity with persistent infections, and hepatocellular injury.
- It remains a major cause of cirrhosis and hepatocellular carcinoma worldwide.

Clinical Manifestations

Clinically, the disease closely resembles hepatitis A.
The incubation period is 1 to 6 months.
Subclinical episodes also occur frequently.
Manifestations may be insidious and variable.

- Fever and respiratory symptoms are rare;
- Arthralgias and rashes in some cases
- Loss of appetite, dyspepsia, abdominal pain, generalized aching, malaise and weakness may occur.
- Jaundice may be evident: associated with light-colored stools and dark urine.
- Tender hepatomegaly (up to 12 to 14 cm vertically)

- Splenomegaly in a few patients
- Posterior cervical lymphadenopathy.

Assessment and Diagnostic Findings

Antigen specific antibody markers for different stages of the disease process:

- anti-HBc—antibody to core antigen or HBV; persists during the acute phase of illness; may indicate continuing HBV in the liver
- anti-HBs—antibody to surface determinants on HBV; detected during late convalescence; usually indicates recovery and development of immunity
- anti-HBe—antibody to hepatitis B e-antigen; usually signifies reduced infectivity
- anti-HBxAg—antibody to the hepatitis B x-antigen; may indicate ongoing replication of HBV

HBsAg appears in the circulation in 80% to 90% of infected patients within 1 to 10 weeks after exposure to HBV and 2 to 8 weeks before the onset of symptoms.

Medical Management

The goals of treatment are;

- Minimize infectivity
- Normalize liver inflammation and
- Manage symptoms.

Treatment modalities:

- Alpha interferon; administered by injection
  - Significant side effects, includes fever, chills, anorexia, nausea, myalgias and fatigue.
  - Late side effects such as bone marrow suppression, thyroid dysfunction, alopecia and bacterial infections, are more serious and may necessitate dosage reduction or discontinuation.
- Antiviral agents: (lamivudine [Epvir] and adefovir [Hepsera])
  - Oral nucleoside analogs
  - Viral resistance may be an issue with these agents
- Bed rest until the symptoms have subsided
- Activities are restricted until the hepatic enlargement, elevated levels of serum bilirubin and liver enzymes have disappeared
- Allow gradual increase of activity afterwards
- Adequate nutrition should be maintained;
- Proteins are restricted due to the liver's impaired ability to metabolize protein byproducts
- Measures to control the dyspeptic symptoms and general malaise include antacids and antiemetics.

- Severe and persistent vomiting may require hospitalization and fluid therapy.
- Evaluate for other blood borne diseases (e.g., HIV infection).

Nursing Management

- During convalescence provide adequate rest and encourage gradual resumption of physical activity after resolution of jaundice.
- Adequate nutrition must be ensured.
- Encourage the patient to avoid drinking alcohol.
- Identify and manage psychosocial issues and concerns, particularly effects of separation from family and friends in hospitalized patients during the acute and infective stages.
- Encourage to avoid sexual contact.
- Support the family to decrease their fears and anxieties about the spread of the disease.
- Follow-up visits at home to assess the patient's progress and answer family members' questions about disease transmission.
- Inform intimate family members and friends about the risks of contracting hepatitis B.
- Intimate them about ways to reduce risk of transmission and early signs of hepatitis B.
- Make arrangements for them to receive hepatitis B vaccine for hepatitis B immune globulin as required.
- Emphasize the importance of keeping follow-up appointments and participating in other health promotion activities and recommended health screenings.

Prevention

- Interrupt the chain of transmission
  - Continued screening of blood donors will decrease the risk of transmission by blood transfusion. The use of disposable syringes, needles and needleless IV administration systems reduce the risk of spread during par-enteral therapy.
  - Good personal hygiene.
  - Use of standard precautions when handling all blood and body fluid specimens
  - Adequate sterilization and disinfection.
  - Patient education regarding the nature of the disease, its infectiousness and prognosis.
- Active immunization: with hepatitis B vaccine, to protect high risk population.
  - Recommended for high risk individuals e.g., health care personnel and hemodialysis patients.
  - Administration of HBV to provide active immunity

- Immunologic memory remains intact for at least 5 to 10 years after which booster doses are required. This is not generally required in normal immune systems
- There are two forms of HBV
  - Yeast-recombinant hepatitis B vaccine (Recombivax HB)
  - Human plasma of chronic infections
- Both forms are administered intramuscularly in three doses: the second and third doses 1 and
- 6 months respectively after the first dose. The third dose is very important in producing prolonged immunity.
- The vaccine produces active immunity to HBV in 90% of healthy people
- Side effects of immunization are infrequent; soreness and redness at the injection site are the most common complaints.

- Passive Immunity: Hepatitis B Immune Globulin
  - It is indicated for new exposure to HBV who has never received hepatitis B vaccine i.e. post-exposure prophylaxis
  - HBIG provides passive immunity
  - It is prepared from plasma selected for high titers of anti-HBs.
  - Specific indications for post-exposure vaccine with HBIG include:
    - Inadvertent exposure to HBAg-positive blood through percutaneous (needle stick) or transmucosal (splashes in contact with mucous membrane) routes
    - Sexual contact with people positive for HBAg and
    - Perinatal exposure (babies born to HBV-infected mothers should receive HBIG within 12 hours of delivery).

Hepatitis C Virus (HCV)

HCV is a common chronic blood-borne infection

It was formerly referred to as non-A, non-B hepatitis (NANB hepatitis).

Transmission means;

- Blood transfusions
- Sexual contact
- Par-enteral means, such as sharing contaminated needles by IV/injection drug users and unintentional needle-sticks and other injuries in health care workers

HCV is the underlying cause of about one-third of cases of hepatocellular carcinoma and the most common reason for liver transplantation

Individuals at special risk for hepatitis C include;

- IV/injection drug users
- Sexually active people with multiple partners

- Patients receiving frequent transfusions or who require large volumes of blood
- Health care personnel

Clinical course:

- The incubation period is variable and may range from 15 to 160 days.
- The clinical course is similar to that of hepatitis B
- Symptoms are usually mild.
- A chronic carrier state occurs frequently
- Present an increased risk of chronic liver disease, including cirrhosis or liver cancer
- Regular intake of small amounts of alcohol appears to encourage disease progression.

Management:

- Avoid alcohol
- Rest
- Diet or vitamin supplements
- Combination of two antiviral agents: Interferon (Intron-A) and Ribavirin (Rebetol): effective to treat active infection and even relapse.

Prophylaxis:

- Regular blood screening to reduce incidence associated with blood transfusions.
- Public health programs to reduce the number of cases associated with shared needles in illicit drug use.

Hepatitis D Virus (HDV)

Hepatitis D is a delta agent
It occurs in some cases of hepatitis B
It is also common among:

- IV/injection drug users
- Hemodialysis patients
- Recipients of multiple blood transfusions
- Sexual contact with hepatitis B cases.

The incubation period varies between 21 and 140 days
Hepatitis D produces symptoms similar to those of hepatitis B
It produces more fulminant hepatitis
Patients are more likely to develop and to progress to chronic active hepatitis and cirrhosis.

The diagnosis is confirmed by presence of anti-delta antibodies in presence of HBAg

Treatment is similar to that of other forms of hepatitis;
Interferon as a specific treatment for hepatitis D is under investigation.

Hepatitis E Virus (HEV)

Hepatitis E is believed to be transmitted by the fecal–oral route principally through contaminated water in areas with poor sanitation.
The incubation period is variable; estimated to be between 15 and 65 days.
Hepatitis E resembles hepatitis A.
It has an abrupt onset with a self-limiting course
Jaundice is nearly always present.
Chronic forms do not develop.
Prophylaxis requires avoiding contact with the virus through good hygiene including hand washing
The effectiveness of immune globulin in protecting against hepatitis E virus is uncertain.

Hepatitis G (HGV) and GB Virus-C

It is a non-A, non-B, non-C agent
They are two different isolates of the same virus.
The incubation period for post-transfusion hepatitis is 14 to 145 days (too long for hepatitis B or C).

Clinical picture include;

- Cryptogenic chronic liver disease (not caused by autoimmune or viral factors) in patients who have previously received transfusions.
- Absent auto-antibodies

The clinical significance of this virus remains uncertain.
Risk factors similar to those for hepatitis C
There is no clear relationship between GBV-C/HGV infection and progressive liver disease.

Persistent infection does occur but does not affect the clinical course.

3.2 Non-Viral Hepatic Disorders

Certain chemicals are hepatotoxic thus can produce acute liver cell necrosis or toxic hepatitis.

Toxic Hepatitis
Toxic hepatitis resembles viral hepatitis in onset.
Preceded with a history of exposure to hepatotoxic chemicals, medications or other agents.

Most common true hepatotoxic chemicals include;

- Carbon tetrachloride
- Phosphorus
- Chloroform
- Gold compounds.

Clinical manifestations:

- Symptoms severity is directly proportional to the toxicity of the offending agent
- Fever
- Patient becomes toxic and prostrated.
- Anorexia, nausea and vomiting (usual symptoms)
- Vomiting may be persistent with hematemesis; may lead to vascular collapse
- Jaundice and hepatomegaly
- Severe clotting abnormalities and skin hemorrhages
- Delirium, coma and seizures may develop
- Patient may die of fulminant hepatic failure within a few days unless a liver transplant is procured.

Management:

- Few treatment options are available
- There are no effective antidotes.
- Restoring and maintaining fluid and electrolyte balance
- Blood replacement and
- Comfort and supportive measures
- Liver transplant could be a life saving measure.

Prognosis:

- A few patients recover from acute toxic hepatitis
- Recovery is rapid if the hepatotoxin is identified early and removed or if exposure to the agent has been limited.
- Recovery is unlikely in cases of associated with a prolonged period between exposure and onset of symptoms.
- A few will progress to develop chronic liver disease
- The liver may heal with scarring followed by post-necrotic cirrhosis.

Drug-Induced Hepatitis

Drug-induced hepatitis is responsible for some cases of acute hepatic failure

Clinical picture:

- It may occur on the first day of exposure to the offending drug (as with acute sensitivity) or several months later depending on the medication.
- Usually the onset is abrupt
- Initial manifestations include chills, fever, rash, pruritus, arthralgia, anorexia and nausea.
- Later manifestations include jaundice, dark urine and an enlarged, tender liver.
- Symptoms may gradually subside with withdrawal of the offending medication.
- Reactions may be severe and even fatal enough to produce an irreversible reaction even after the medication is stopped.
- Drug-induced hepatitis is similar to acute viral hepatitis but with a more extensive parenchymal destruction.

Offending medications include:

- Acetaminophen (Paracetamol) the leading cause of acute liver failure
- Anesthetic agents
- Anti rheumatic agents
- Antimetabolites
- Drugs used to treat musculoskeletal disease
- Antidepressants
- Psychotropic medications
- Anticonvulsants and
- Anti-tuberculosis agents
- Inhalational agents of the halothane family (halokanes) can cause halothane hepatitis.

Treatment;

- A short course of high-dose corticosteroids for cases related to severe hypersensitivity; although with uncertain efficacy
- Liver transplantation is an option for drug-induced hepatitis.

Jaundice

This is due to abnormally elevated serum bilirubin concentration

It presents yellow-tinged or greenish-yellow discoloration of all the body tissues including the sclerae and the skin.

Jaundice becomes clinically evident when the serum bilirubin level exceeds 2.5mg/dL (43fmol/L).

Increased serum bilirubin levels and jaundice may result from impairment of hepatic uptake, metabolism (conjugation) or excretion of bilirubin into the biliary system.

Types of jaundice:

- Hemolytic jaundice
- Hepatocellular jaundice
- Obstructive jaundice
- Jaundice due to hereditary hyperbilirubinemia.

Hepatocellular and obstructive jaundice are commonly associated with liver disease.

Hemolytic Jaundice

Hemolytic jaundice is the result of an increased destruction of the red blood cells.

It is a common phenomenon in hemolytic transfusion reactions and other hemolytic disorders.

Rapid hepatic hemolysis leads to high production of bilirubin thus overwhelming the normally functioning and thus cannot excrete the bilirubin as quickly as it is formed.

Biochemical features

- Un-conjugated bilirubinaemia
- High levels of fecal and urine urobilinogen
- Absent urine bilirubin.

Clinical manifestations:

- Asymptomatic unless extreme hyperbilirubinemia.
- Prolonged mild jaundice may lead to formation of gallbladder stones.
- Extremely severe jaundice (levels of free bilirubin exceeding 20 to 25mg/dL) predisposes to brain stem damage (Kernicterus).

Hepatocellular Jaundice

This case is due to inability of the failing liver to clear normal amounts of bilirubin from the blood

The hepatocellular damage may due to:

- Viral hepatitis (e.g., hepatitis A, B, C, D, or E); other viruses (e.g., yellow fever virus, Epstein-Barr virus).
- Medication or chemical toxicity (e.g., carbon tetrachloride, chloroform, phosphorus, arsenicals, certain medications), or from alcohol.

Cirrhosis of the liver is a form of hepatocellular disease that may produce jaundice.

Clinical manifestations:

- Patients with hepatocellular jaundice may present with
- Mild or severe illness with lack of appetite, nausea, malaise, fatigue, weakness and possible weight loss
- Jaundice may not be obvious in some cases
- Elevated serum bilirubin and urine urobilinogen level
- High levels of AST and ALT (indicating cellular necrosis)
- Headaches, chills and fever (if the cause is infectious in origin)
- Hepatocellular jaundice may or may not be completely reversible depending on the cause and extent of the liver cell damage.

Obstructive Jaundice

Types:

- Extra-hepatic obstructive jaundice may be caused by occlusion of the bile duct by;
  - Gallstone
  - An inflammatory process
  - Tumor
  - Pressure from an enlarged organ
- Intra-hepatic obstruction involve the small bile ducts within the liver, due to pressure on these channels by;
  - Inflammatory (swelling) of the liver
  - Inflammatory exudate within the ducts
  - Cholestatic agents; results in stasis and inspissation (thickening) of bile within the canaliculi. These include phenothiazines, anti-thyroid medications, sulfonylureas, tricyclic anti-depressant agents, nitrofurantoin, androgens and estrogens.

Pathophysiology:

Intra-hepatic or extra-hepatic obstruction impedes bile flow into the intestine but cause reflux up into the liver substance. It is then reabsorbed into the blood and carried throughout the entire body, building up and staining the skin, mucous membranes and sclerae.

Clinical manifestations:

- Urine becomes deep orange and foamy.
- Because of the decreased
- Stools become light or clay-colored
- Intense pruiritus
- Dyspepsia and intolerance to fatty foods due to impaired fat digestion in the absence of intestinal bile
- AST, ALT, and GGT levels generally rise only moderately
- Bilirubin and alkaline phosphatase levels are elevated.

3.3 Hereditary Hyperbilirubinemia

Increased serum bilirubin levels (hyperbilirubinemia) resulting from several inherited disorders such as;

- Gilbert's syndrome; a familial disorder characterized by an increased level of un-conjugated bilirubin. This syndrome affects 2% to 5% of the population.

o Clinical features:

- Raised serum bilirubin levels
- Normal liver histology and liver function test
- No evidence of hemolysis.

Other conditions are caused by inborn errors of biliary metabolism include;

- Dubin–Johnson syndrome (chronic idiopathic jaundice, with pigment in the liver)
- Rotor's syndrome (chronic familial conjugated hyperbilirubinemia without pigment in the liver)
- Benign cholestatic jaundice of pregnancy; with retention of conjugated bilirubin, probably secondary to unusual sensitivity to the hormones of pregnancy and
- Benign recurrent intrahepatic cholestasis.

3.3.1 Portal Hypertension

This is due to obstructed blood flow through the damaged liver with resultant increased blood pressure throughout the portal venous system
Portal hypertension is commonly associated with hepatic cirrhosis
It can also occur with non-cirrhotic liver disease.

Clinical presentations

- Splenomegaly (enlarged spleen) with possible hypersplenism (a common manifestation)
- Ascites: fluid accumulates in the abdominal cavity
- Varices: elevated pressures transmitted to all of the veins that drain into the portal system: They are prone to rupture and often massive upper GI tract and rectal hemorrhages
- Blood clotting abnormalities, often in severe liver disease: increase the likelihood of bleeding.

3.3.1 Ascites

Pathophysiology
The mechanisms of development are not completely understood.
It is thought to be due to;

Increased hydrostatic pressure within the portal system due to;
Hepatic venous obstruction.

Increased intravascular fluid volume due to renal sodium and water retention; a response to liver's failure to metabolize aldosterone;

Reduced plasma oncotic pressure due to low serum albumin following reduced hepatic synthesis of albumins;

All these favor fluid shift from the portal veins into the peritoneal sac as ascitis: i.e. fluid extravasation.

Liver damage may cause large amounts of albumin-rich fluid, (15L or more) to accumulate in the peritoneal cavity.

This further depletes plasma and plasma albumin and worsens systemic hypovolemia.

In response further renal sodium and water retention is triggered to maintain intra-vascular fluid volume.

This initiates and maintains a vicious cycle.

Clinical Manifestations

- Increased abdominal girth
- Rapid weight gain
- Dyspnoeas due to the displacement of the diaphragm
- Loss of comfort from the enlarged abdomen
- Abdominal striae and distended veins may
- Positive fluid trill
- Fluid and electrolyte imbalances.

Assessment and Diagnostic Evaluation

- Abdominal percussion: by eliciting for shifting dullness and fluid wave helps determine the presence and extent of Ascites respectively
- Daily measurement and recording of abdominal girth and body weight to assess the progression of ascites and response to treatment.

Abdominal palpation to demonstrate fluid wave

Management

- Dietary Modification
  - Ascites treatment requires a negative sodium balance to reduce fluid retention.
  - Salt should be restricted in diet
  - Avoid table salt, salty foods, salted butter and margarine and all ordinary canned and frozen foods that are not specifically prepared for low-sodium diets
  - Use salt substitutes such as lemon juice, oregano, and thyme
  - Avoid commercial salt substitutes that contain ammonia as it could precipitate hepatic coma
  - Avoid potassium containing salts and diets in case of associated impaired renal function
  - Dietary control of ascites with strict sodium restriction is difficult to achieve at home
  - Approximately 10% of ascites will resolve with these measures alone.
- Diuretic Therapy
  - Use of diuretics along with sodium restriction is successful in 90% cases
  - Spironolactone (Aldactone), an aldosterone-blocking agent is often the first-line therapy in ascites due to cirrhosis; helps spare potassium loss.
  - Furosemide (Lasix) may be added per oral but cautiously used as long-term use may also induce severe sodium depletion (hyponatremia).
  - Ammonium chloride and acetazolamide (Diamox) are contraindicated because of the possibility of precipitating hepatic coma.

- Fluid restriction is not attempted unless the serum sodium concentration is very low.
- Possible complications of diuretic therapy include;
  - Fluid and electrolyte disturbances (including hypovolemia, hypokalemia, hyponatremia & hypochloremic alkalosis)
  - Encephalopathy due to dehydration and hypovolemia and hyperkalemia-induced increased endogenous ammonia synthesis
- Bed Rest
- Bed rest may is useful especially for conditions refractory to diuretics
- Paracentesis
- Paracentesis is the removal of fluid (ascites) from the peritoneal cavity through a small surgical incision or puncture made through the abdominal wall under sterile conditions.
- This may be done with ultrasound guidance in cases with high risk for bleeding due to abnormal coagulation profile or previous history of abdominal surgery with high possibilities of adhesions
- A sample of the ascitic fluid may be sent to the laboratory for analysis. Cell count, albumin and total protein levels, culture, and occasionally other tests are performed.
- Severe ascitis may require large-volume (5 to 6 liters) draining per time
- This technique should be combined with intravenous infusion of salt-depleted albumin or other colloid
- Insertion of a Peritoneovenous Shunt
- This is to redirect ascitic fluid from the peritoneal cavity into the systemic circulation: seldom used because of high complication rate and high incidence of shunt failure.

Nursing Management

Hospitalized patient will require the following nursing measures;
Assessment and documentation of the following;

- Fluid intake and output
- Abdominal girth and
- Daily weight to assess fluid status
- Serum ammonia level as an indicator for encephalopathy.
- Serum electrolyte levels to assess response to therapy.

Teaching Patients Self-Care

Teach the patient and family about the treatment plan
Instruct them on the need to;

- Avoid all alcohol intake
- Adhere to a low-sodium diet: may require storing appropriate foods
- Take medications as prescribed

- Check with the physician before taking any new medications
- Meticulously care for the skin to avoid pressure ulcers
- Weigh the patient daily
- Watch for and report signs and symptoms of complications like changes in cognitive and emotional status
- Keep scheduled health care appointments.

3.3.3 Esophageal Varices

Bleeding or hemorrhage from esophageal varices occurs in approximately one third of patients with cirrhosis and varices.

The mortality rate resulting from the first bleeding episode is 45% to 50% and increases with each subsequent bleeding episode

It is one of the major causes of death in patients with cirrhosis.

Pathophysiology

- Varices are dilated, tortuous veins
- Esophageal varices are usually found in the sub-mucus layer of the lower esophagus but may develop higher in the esophagus or extend into the stomach.
- This main cause is portal hypertension due to obstruction of the portal venous system within the damaged liver.
- This leads to formation of alternate collateral circulation to drain mesenteric and splenic venous blood directly to the right atrium, shunting the portal venous system.
- The net effect is a pressure increase in the vessels of the sub-mucosal layer of the lower esophagus and upper stomach.
- These collateral vessels are tortuous and fragile and thus bleed easily.
- Less common causes of varices are abnormalities of the circulation in the splenic vein; superior vena cava or hepatic venothrombosis.
- Bleeding esophageal varices are life-threatening and can result in hemorrhagic shock.
- The result is decreased cerebral, hepatic and renal perfusion.
- Bleeding into the GI becomes degraded into urea and ammonia by the enteric flora thus increasing serum ammonia level and the risk for encephalopathy.
- Usually the dilated veins lie latent unless sharp portal pressure increase rupture them and massive hemorrhage ensues.
- Factors that contribute to hemorrhage are:
  - Muscular exertion from lifting heavy objects
  - Straining during defecation.

- Sneezing, coughing or vomiting.
- Esophagitis.
- Irritation of vessels by poorly chewed, irritating fluids.
- Erosion by reflux of stomach contents (especially alcohol); salicylates or anti-neoplastic drugs.

Clinical Manifestations

The patient with bleeding esophageal varices may present with;

- Hematemesis
- Melena
- General deterioration in mental or physical status
- Signs and symptoms of shock (cool clammy skin, hypotension, tachycardia).

Assessment and Diagnostic Findings

- Endoscopy; Aimed to identify the cause and the site of bleeding.

Nursing support include;

- Relieving anxiety
- Careful monitoring to detect early signs of cardiac dysrhythmias, perforation and hemorrhage
- Restrict fluids until the gag reflex returns
- Lozenges and gargles to relieve throat discomfort
- Restrict oral intake if the patient is actively bleeding
- Portal Hypertension Measurements
- Indices of portal hypertension include;
  - Dilated abdominal veins and hemorrhoids
  - Palpable enlarged spleen (splenomegaly)
  - Ascites
- Indirect measurement of the hepatic vein pressure gradient;
  - It is the most common procedure
  - It requires insertion of a fluid-filled balloon catheter into the antecubital or femoral vein.
  - The catheter is advanced under fluoroscopy to a hepatic vein.
  - A “wedged” pressure (similar to pulmonary artery wedge pressure) is obtained by occluding the blood flow in the blood vessel.
  - Pressure in the un-occluded vessel is also measured.
- Direct measurement of portal vein pressure; can be obtained by several methods.
  - During laparatomy; a needle may be introduced into the spleen; a manometer reading of more than 20 mL saline is abnormal.
  - Insertion of a catheter into the portal vein or one of its branches
  - Endoscopic measurement of pressure within varices is used only in conjunction with endoscopic sclerotherapy.

- Laboratory Tests; may include; Liver function tests such as serum aminotransferase, bilirubin, alkaline phosphatase and serum proteins.
- Splenoportography; Serial or segmental x-rays to detect extensive collateral circulation in esophageal vessels which would indicate varices.
- Hepatoportography.
- Celiac angiography.

These are usually performed in the operating room or radiology department.

Medical Management

- Requires aggressive medical care usually in the intensive care unit for close monitoring.
- The extent of bleeding is evaluated.
- Vital signs are monitored continuously when hematemesis and malena are present.
- Evidences of potential hypovolemia should be noted, such as cold clammy skin, tachycardia, a drop in blood pressure, decreased urine output, restlessness and weak peripheral pulses.
- Blood volume is monitored by a central venous pressure or arterial catheter.
- Oxygen is administered to prevent hypoxia and to maintain adequate blood oxygenation.
- Intravenous fluids with electrolytes and volume expanders are provided to restore fluid volume and replace electrolytes.
- Transfusion of blood components also may be required.
- An indwelling urinary catheter is usually inserted to permit frequent monitoring of urine output.
- Pharmacologic Therapy; Active bleeding requires medications such as;
    - Vasopressin (Pitressin): to produces constriction of the splanchnic arterial bed and a resulting decrease in portal pressure.
    - The combination of vasopressin and nitroglycerin (administered by the intravenous, sublingual, or transdermal route) has been effective in reducing or preventing the side effects (constriction of coronary vessels and angina) caused by vasopressin alone.
    - Somatostatin and octreotide (Sandostatin); more effective than vasopressin in decreasing bleeding from esophageal varices without the vasoconstrictive effects of vasopressin: These medications cause selective splanchnic vasoconstriction.

- Propranolol (Inderal) and nadolol (Corgard): beta-blocking agents that decrease portal pressure, have been shown to prevent bleeding from esophageal varices in some patients; in combination with other treatment modalities such as sclerotherapy, variceal banding, or balloon tamponade.
- Nitrates; such as isosorbide (Isordil) lower portal pressure by venodilation and decreased cardiac output.

- Balloon Tamponade
  - This is used to control hemorrhage in certain patients
  - In this procedure, pressure is exerted on the cardia (upper orifice of the stomach) and against the bleeding varices by a double-balloon tamponade (Sengstaken-Blakemore tube)
  - The tube has four openings, each with a specific purpose such as;
  - Gastric and esophageal aspiration
  - Inflation of the gastric and esophageal balloon
  - The balloon in the stomach is inflated with 100 to 200 mL of air.
  - An x-ray confirms proper positioning of the gastric balloon.
  - Then the tube is pulled gently to exert a force against the gastric wall
  - The desired pressure in the esophageal and gastric balloons is 25 to 40 mm Hg, as measured by the manometer.
  - There is a possibility of injury or rupture of the esophagus with inflation of the esophageal balloon, so constant nursing surveillance is necessary.
  - Gastric suction is provided by connecting the gastric catheter outlet to suction. The tubing is irrigated hourly, and drainage will indicate whether bleeding has been controlled.
  - Room-temperature lavage or irrigation may be used in the gastric balloon.
  - The pressure within the esophageal balloon is measured and recorded every 2 to 4 hours via the manometer to detect under inflation or over inflation with potential for esophageal injury.
  - After several hours without bleeding, the gastric balloon may be deflated safely.
  - If there is still no bleeding, the tamponade tube is removed.
  - Displacement of the tube and the inflated balloon into the oropharynx can cause life-threatening obstruction of the airway and asphyxiation.

- Nursing measures include frequent mouth and nasal care.
- For secretions that accumulate in the mouth, tissues should be within easy reach of the patient.
- Oral suction may be necessary to remove oral secretions.
- Once the balloons are deflated or the tube is removed, the patient must be assessed frequently because of the high risk for recurrent bleeding.

- Endoscopic Sclerotherapy: is done by injecting a sclerosing agent through a fiberoptic endoscope into the bleeding esophageal varices to promote thrombosis and eventual sclerosis.
  - Endoscopic varicealsclero therapy has been used in the primary prophylaxis of variceal bleeding
  - After treatment, the patient must be observed for bleeding, perforation of the esophagus, aspiration pneumonia, and esophageal stricture.
  - Antacids may be administered after the procedure to counteract the effects of peptic reflux.
- Esophageal Banding Therapy (Variceal Band Ligation); involves variceal litigation through an elastic rubber band threaded through an overtube directly to band the varix (or varices) through a modified endoscope with a view to inducing necrosis, ulceration and eventual sloughing of the varix.
  - Complications include superficial ulceration and dysphagia, transient chest discomfort and rarely esophageal strictures
- Transjugular Intrahepatic Portosystemic Shunting (TIPS): a cannula is threaded into the portal vein by the transjugular route.
  - An expandable stent is inserted and serves as an intrahepatic shunt between the portal circulation and the hepatic vein thus reducing portal hypertension. The therapy has been indicated for;
  - Recurrent variceal bleeding refractory to pharmacologic or endoscopic therapy
  - Control of refractory ascites.
  - This technique is also used as a bridge to liver transplantation.
  - Complications may include bleeding, sepsis, heart failure, organ perforation, shunt thrombosis, and progressive liver failure.

Surgical Management

Aims;

- To treat esophageal varices and
- To minimize re-bleeding.

It is often accompanied by significant risk.

Surgical Bypass Procedures

Surgical decompression of the portal circulation with shunt

- Selective Shunts:

- Distal splenorenal shunt; made between the splenic vein and the left renal vein after splenectomy.
- Mesocaval shunt; by anastomosing the superior mesenteric vein to the proximal end of the vena cava or to the side of the vena cava using grafting material to drain only a portion of venous blood from the portal bed to decrease portal pressure
- Non-selective shunts:
- Portacaval shunts; divert all portal flow to the vena cava via end-to-side or side-to-side approaches.
- Partial portacaval shunts; with interposition grafts are as effective as other shunts but are associated with a lower rate of encephalopathy.
- Mesoatrial shunt; is required when the infra-hepatic vena cava is thrombosed and must be bypassed.

Devascularization and Transection

Devascularization and staplegun transection procedures to separate the bleeding site from the high-pressure portal system; have been used in the emergency management of variceal bleeding

The lower end of the esophagus is reached through a small gastrostomy incision; a staple gun permits anastomosis of the transected ends of the esophagus. Recurrent bleeding is a risk.

Nursing Management

Overall nursing management includes;

- Monitoring and recording vital signs.
- Assessing the patient's nutritional and neurologic status to identify hepatic encephalopathy due to the breakdown of blood in the GI tract and a rising serum ammonia level.
- Ensuring complete rest of the esophagus if active bleeding is evident.
- Administer par-enteral nutrition.
- Initiate gastric suction to keep the stomach as empty as possible and prevent straining and vomiting.
- Perform frequent oral hygiene and apply moist sponges to the lips to relieve severe thirst.
- Close monitoring of the blood pressure.
- Administer Vitamin K therapy and multiple blood transfusions because of blood loss.
- Maintain a quiet environment and calm reassurance may help to relieve the patient's anxiety .
- Monitoring for alcohol withdrawal manifestations if the patient has been a heavy user of alcohol.
- Providing support and explanations regarding medical and nursing interventions.

- Monitoring the patient closely will help in detecting and managing complications.

3.3.4 Hepatic Encephalopathy and Coma

Hepatic encephalopathy is a life-threatening complication of liver disease. It occurs with profound liver failure.

It may result from the accumulation of ammonia and other toxic metabolites in the blood.

Hepatic coma represents the most advanced stage of hepatic encephalopathy.

Theories/assumptions:

- Synthesis of a false or weak neurotransmitter from an intestinal source that may precipitate encephalopathy.
- Excess tryptophan and its metabolites.
- Endogenous benzodiazepines or opiates; Benzodiazepine-like compounds have been detected in the plasma and cerebrospinal fluid of patients with hepatic encephalopathy due to cirrhosis.
- Portal-systemic encephalopathy; the most common type of hepatic encephalopathy, occurs primarily in patients with cirrhosis with portal hypertension and portal-systemic shunting.

Pathophysiology

Ammonia accumulates because damaged liver cells fail to detoxify and convert to urea.

Sources of ammonia include the GIT, kidney and muscle cells.

- Enzymatic and bacterial synthesis from dietary and blood proteins in the GI tract represent the largest source of ammonia: due to;
  - GI bleeding (i.e., bleeding esophageal varices or chronic GI bleeding)
  - High-protein diet
  - Bacterial infections
  - Uremia.
- The ingestion of ammonium salts.
- Alkalosis or hypokalemia; increases amounts of ammonia absorbed from the GI tract and renal tubular fluid.

Other factors unrelated to increased serum ammonia levels that may cause hepatic encephalopathy in susceptible patients include;

- Excessive diuresis

- Dehydration
- Infections and fever
- Surgery
- Medications (sedative agents, tranquilizers, analgesics and Potassium wasting diuretics).

Increased ammonia concentration in the blood causes brain dysfunction and damage, resulting in hepatic encephalopathy.

Serum ammonia can be decreased by elimination of protein from the diet and administration of antibiotic agents, such as neomycin sulfate, that reduce the intestinal bacterial load, capable of converting urea to ammonia.

Clinical Manifestations

- The earliest symptoms of hepatic encephalopathy include;
  - Minor mental changes and
  - Motor disturbances such as hyperactive deep tendon reflexes
  - Slightly confusion and alterations in mood
  - Hygiene neglect and
  - Altered sleep patterns; patient tends to sleep during the day and have restlessness and insomnia at night.
- As hepatic encephalopathy progresses;
  - Difficulty in patient arousal
  - Mentation generally worsens
  - Motor disturbances such as Asterixis (flapping tremor of the hands) may occur with.
  - Constructional Apraxia; Inability to reproduce a simple figure
  - Reflexes may disappear and
  - The extremities may become flaccid
  - **Fetor hepaticus;** a sweet, slightly fecal odor to the breath presumed to be of intestinal origin may be noticed especially in cases with extensive collateral portal circulation in chronic liver disease.
- In a more advanced stage, there are;
  - Gross disturbances of consciousness, with complete disorientation to time and place
  - Frank coma
  - Seizures.
- Approximately 35% of all patients with cirrhosis of the liver die in hepatic coma.
- The electroencephalogram (EEG); shows generalized slowing, an increase in the amplitude of brain waves, and characteristic triphasic waves.

Medical Management

- Lactulose (Cephulac); to reduce serum ammonia levels.
  - It acts by several mechanisms that promote the excretion of ammonia in the stool:
- Keeping ammonia in its ionized state results in a fall in colon pH and thus blocks absorption into the blood.
- Induce diarrhea and thus decreases the ammonia absorbed from the colon and
- Mediate fecal flora mutation to organisms that do not produce ammonia from urea.
  - Two or three soft stools per day are desirable;
  - Possible side effects include intestinal bloating and cramps; which usually disappear within a week.
  - Lactulose can be diluted with fruit juice to mask its sweet taste.
  - Avoid other laxatives during lactulose administration
  - Consider administration by nasogastric tube or enema for comatose cases or in cases oral administration is contraindicated or impossible.
- Intravenous administration of glucose to minimize protein breakdown.
- Administration of vitamins to correct deficiencies.
- Correction of electrolyte imbalances (especially potassium).

Principles of management of hepatic encephalopathy include the following:

- Therapy is directed toward treating or removing the cause.
- Neurologic status is assessed frequently. Keep daily record of handwriting and performance in arithmetic to monitor mental status.
- Fluid intake and output and body weight are recorded each day.
- Vital signs are measured and recorded every 4 hours.
- Potential sites of infection (peritoneum, lungs) are assessed frequently, and abnormal findings are reported promptly.
- Serum ammonia level is monitored daily.
- Protein intake is restricted in patients who are comatose or who have encephalopathy that is refractory to lactulose and antibiotic therapy: may be up to 0.8 to 1.0 g/kg per day. Vegetable protein intake may improve nitrogen balance without precipitating or advancing hepatic encephalopathy.
- Reduction in the absorption of ammonia from the GI tract is accomplished by the use of gastric suction, enemas, or oral antibiotics.
- Electrolyte status is monitored and corrected if abnormal.

- Sedatives, tranquilizers, and analgesic medications are discontinued.
- Benzodiazepine antagonists (flumazenil [Romazicon]) may be administered to improve encephalopathy whether or not the patient has previously taken benzodiazepines.
- Maintaining a safe environment to prevent injury, bleeding and infection.
- Monitoring the patient for the many potential complications.
- Communicating with the patient's family to keep them informed about the patient's status.
- Supporting them by explaining the procedures and treatments that are part of the patient's care.
- Discussing signs of subtle signs of recurrent encephalopathy so as to recognize them when they recur.

3.3.5 Fulminant Hepatic Failure

Fulminant hepatic failure is the clinical syndrome characterized by sudden and severe impairment of liver function in a previously healthy person often within 8 weeks of the first appearance of jaundice.

Clinical categories;
Patterns of progression from jaundice to encephalopathy have been classified into three categories:

- Hyper-acute liver failure; encephalopathy ensues within 0 to 7 days after the first appearance of jaundice;
- Acute liver failure: it is 8 to 28 days;
- Sub-acute liver failure; it is 28 to 72 days.

Common causes of fulminant hepatic failure;

- Viral hepatitis (commonest cause).
- Toxic medications (e.g., acetaminophen).
- Toxic chemicals (e.g., carbon tetrachloride).
- Metabolic disturbances (Wilson's disease, a hereditary syndrome with deposition of copper in the liver) and
- Structural changes (Budd-Chiari syndrome, an obstruction to outflow in major hepatic veins).

Clinical manifestations;

- Jaundice and Profound anorexia may be the initial reasons the patient seeks health care.

It is often accompanied by;

- Coagulation defects
- Electrolyte disturbances

- Renal failure
- Infection
- Hypoglycemia
- Encephalopathy and cerebral edema.

Management

The keys are rapid recognition of acute liver failure and intensive interventions.

Treatment modalities may include;

- Plasma exchanges (plasmapheresis)
- Charcoal hemoperfusion for the removal (theoretically) of potentially harmful metabolites.
- Liver assist devices; Extracorporeal Liver Assist Devices (ELAD) and Bioartificial liver (BAL) i.e. Hepatocytes within synthetic fiber columns have been tested as liver support systems to promote survive until transplantation is possible.
    - This approach has been associated with a high risk for cerebral edemas hence these patients will require intracranial pressure monitoring. The following adjuvant measures to promote adequate cerebral perfusion thus suffice;
- Careful fluid balance and hemodynamic assessments.
- A quiet environment.
- Diuresis with mannitol (an osmotic diuretic)
- Barbiturate anesthesia or pharmacologic paralysis and sedation to prevent agitation.
- Monitoring for and treating hypoglycemia, coagulopathies and infection.
- Liver transplantation: is the treatment of choice for fulminant hepatic failure.

Prognosis;

- Much worse than chronic liver failure
- Associated hepatic injury is potentially reversible
- Survival rates are approximately 50% to 85%
- Survival depend greatly on the etiology of liver failure
- Death is often due to massive hepatocellular injury and necrosis.

3.3.6 Hepatic Cirrhosis

Cirrhosis is a chronic disease characterized by extensive diffuse liver fibrosis that disrupts the structure and function of the liver.

There are three types of cirrhosis or scarring of the liver:

- ***Alcoholic cirrhosis***, in which the scar tissue characteristically surrounds the portal areas.

- This is the most common type of cirrhosis.
- It is most frequently due to chronic alcoholism.
- However, contributory effects of nutritional deficiency (reduced protein intake).
- The disease usually has an insidious onset and a protracted course, occasionally proceeding over a period of 30 or more years.

- ***Post-necrotic cirrhosis***, in which there are broad bands of scar tissue as a late result of a previous bout of acute viral hepatitis. It may be due to chemicals (carbon tetrachloride, chlorinated naphthalene, arsenic, or phosphorus) or infectious schistosomiasis.
- ***Biliary cirrhosis***, in which scarring occurs in the liver around the bile ducts.
  - This type usually is the result of chronic biliary obstruction and infection (cholangitis);
  - It is much less common than the other two types.
  - The portion of the liver chiefly involved consists of the portal and the periportal spaces (where bile canaliculi of each lobule communicate to form the liver bile ducts).
  - These areas become the sites of inflammation with resultant biliary duct occlusion by inspissated bile and pus.
  - The liver attempts to form new bile channels; hence an overgrowth of tissue made up largely of disconnected, newly formed bile ducts and surrounded by scar tissue.

Pathophysiology:

The following events are evident;

- Repeated episodes of inflammation with massive hepatic cell injury.
- Liver cell necrosis follows.
- Gradual organization and fibrosis of dead liver cells
- Concurrent regeneration of the surviving liver cells
- Eventually the amount of scar tissue exceeds that of the functioning liver tissue.
- Islands of residual normal tissue and regenerating liver tissue may project from the constricted areas, giving the cirrhotic liver its characteristic hobnail appearance.

Epidemiology:

- Men are twice affected as women.
- Women are at greater risk of developing alcohol-induced liver disease for an as yet undiscovered reason.
- Most patients are between 40 and 60 years of age.

Clinical Manifestations
Signs and symptoms of cirrhosis increase in severity as the disease progresses.

The severity of the manifestations helps to categorize the disorder into two main presentations;

- ***Compensated cirrhosis;*** presents with less severe often vague symptoms and thus often discovered accidentally at a routine physical examination.
    - Common presentations include;
- Intermittent mild fever
- Vascular spiders
- Palmar erythema (reddened palms)
- Unexplained epistaxis
- Ankle edema
- Vague morning indigestion
- Flatulent dyspepsia
- Abdominal pain
- Firm, enlarged liver
- Splenomegaly.
- ***Decompensated manifestations cirrhosis;*** Result from failure of the liver to synthesize proteins, clotting factors and other substances and manifestations of portal hypertension (ascites, varices and hepatic encephalopathy)

o Common presentations include;

- Ascites
- Jaundice
- Weakness
- Muscle wasting
- Weight loss
- Continuous mild fever
- Clubbing of fingers
- Purpura (due to decreased platelet count)
- Spontaneous bruising
- Epistaxis
- Hypotension
- Sparse body hair
- White nails
- Gonadal atrophy

Common manifestations are;

- Intermittent jaundice and
- Fever
- Liver Enlargement;
    - Early in the course of cirrhosis the liver pathology include;

- Hepatomegaly; may cause abdominal pain due to tension on its fibrous covering (Glisson's capsule)
- Fatty infiltration of the liver
- Liver architecture; firm with a sharp edge noticeable on palpation

o Later in the disease;

- The liver decreases in size as scar tissue contracts the liver tissue.
- The liver edge, if palpable, is nodular.

- Portal Obstruction and Ascites are manifestations due to obstruction of the portal circulation.
- Infection and Peritonitis: Spontaneous bacterial peritonitis i.e. bacterial peritonitis even in the absence of an intra-abdominal source of infection or an abscess.
- Gastrointestinal Varices; presents with visibly distended abdominal blood vessels (caput medusae), and esophageal, gastric and lower rectal (hemorrhoids) varices
- Edema: often affects lower extremities, upper extremities, and the presacral area. Facial edema is not typical. It is attributable to chronic liver failure, reduced plasma albumin concentration and aldosterone excess (causing sodium and water retention)
- Vitamin Deficiency and Anemia: due to inadequate formation, use, and storage of certain vitamins (notably vitamins A, C, and K)
- Mental Deterioration: indicates an impending hepatic encephalopathy and hepatic coma.

Assessment and Diagnostic Findings

- Liver biopsy; confirms the diagnosis
- Serum albumin levels tends to decrease
- Serum globulin level rises.
- Enzyme tests indicate liver cell damage: serum alkaline phosphatase, AST, ALT, and GGT levels increase
- Serum cholinesterase level may decrease.
- Bilirubin tests; to measure bile excretion or bile retention; elevated levels can occur with cirrhosis and other liver disorders.
- Prothrombin time is prolonged.
- Ultrasound scanning; to measure the difference in density of parenchymal cells and scar tissue.
- CT, MRI and radioisotope liver scans give information about liver size and hepatic blood flow and obstruction.
- Arterial blood gas analysis may reveal a ventilation–perfusion imbalance and hypoxia.

Medical Management

The management of the patient with cirrhosis is usually based on the presenting symptoms.

Although the fibrosis of the cirrhotic liver cannot be reversed, its progression may be halted or slowed by such measures.

- Antacids to decrease gastric distress and minimize the possibility of GI bleeding.
- Vitamins and nutritional supplements promote healing of damaged liver cells and improve the general nutritional status.
- Potassium-sparing diuretics (spironolactone [Aldactone], triamterene [Dyrenium]) may be indicated to decrease ascites, if present; these diuretics are preferable to other diuretic agents because they minimize the fluid and electrolyte changes common with other agents.
- An adequate diet
- Avoidance of alcohol is essential.
- Colchicine, an anti-inflammatory agent used to treat the symptoms of gout, may increase the length of survival in patients with mild to moderate cirrhosis. Colchicine is believed to reverse the fibrotic processes in cirrhosis and thus improve survival

3.4 Liver Abscesses

Two categories of liver abscess have been identified:

- Amebic liver abscesses are most commonly caused by *Entamoebahistolytica.* Most cases occur in the developing countries of the tropics and subtropics due to poor sanitation and hygiene.
- Pyogenic liver abscesses are much less common but are more common in developed countries than the amebic type.

Pathophysiology

- Infection along the biliary or GI tract may spread to affect the liver through the biliary system, portal venous system or hepatic arterial or lymphatic system.
- Most bacteria are destroyed promptly
- Established infection produce an initial liver cell necrosis which eventually wall off the pathogens.
- Resultant inflammation and leukocytosis may produce an abscess cavity full of exudates (a product of life and dead leukocytes, liquefied liver cells and bacteria).
- Abscesses may be either single or multiple.

Clinical Manifestations

- Sepsis with few or no localizing signs
- Fever with chills and diaphoresis, malaise, anorexia, nausea and vomiting

- Weight loss may occur.
- Dull abdominal pain
- Tenderness in the right upper quadrant of the abdomen
- Hepatomegaly
- Jaundice
- Anemia and
- Pleural effusion may develop.
- Sepsis and shock may be severe and life-threatening.

Assessment and Diagnostic Findings
- Blood cultures; may not identify the organism.
- Ultrasonic guided aspiration of the liver abscess;
- Cultures of aspirates to identify the organism.

Medical Management
- IV antibiotic therapy; depends on the organism identified.
- Continuous percutaneous drainage of pyogenic abscesses with catheter to evacuate the abscess material and promote healing
- Open surgical drainage; if antibiotic therapy and percutaneous drainage are ineffective.

Nursing Management
- Management of the drainage system
- Skin care
- Infection control strategies (safe handling of drained exudates)
- Vital signs monitoring
- Pain evaluation; increase pain may indicate rupture or extension of the abscess; it must be reported promptly.
- Administration of IV antibiotic therapy as prescribed.
- Monitored closely white blood cell count and other laboratory test for changes consistent with worsening of infection.

Prognosis:
- In the past, mortality rate was 100%
- Diagnostic sophistication (ultrasound, CT and MRI scans and liver scans), antibiotics and advancement in surgical techniques of drainage the abscess have greatly reduced the mortality rate.

3.4.1 Cancer of the Liver

Hepatic tumors may be malignant or benign.
Benign liver tumors were uncommon until the widespread use of oral contraceptives, occurring most frequently in women in their reproductive years.

Few cancers originate in the liver.
Primary liver tumors usually are associated with;

- Chronic liver disease
- Chronic hepatitis B and C infections and
- Exposure to certain chemical toxins (e.g., vinyl chloride, arsenic)
- Cirrhosis
- Cigarette smoking and alcohol use
- Aflatoxin, a metabolite of the fungus *Aspergillusflavus:* Aflatoxin and other similar toxic molds can contaminate food such as ground nuts and grains and may act as a co-carcinogen with hepatitis B. The risk of contamination is greatest when these foods are stored unrefrigerated in tropical or subtropical climates.

Types of primary liver cancer

- Hepatocellular carcinoma (HCC): by far the most common type of primary liver cancer. HCC is usually non-resectable because of rapid growth and metastasis.
- Cholangiocellular carcinoma.
- Combined hepatocellular and cholangiocellular carcinoma.

If found early, resection may be possible
Early detection is unlikely.
Many patients have metastases from the primary liver tumor to other sites by the time diagnosis; occurring primarily to the lung, regional lymph nodes, adrenals, bone, kidneys, heart, pancreas, and stomach.

3.4.2 Liver Metastases

Metastases from other primary sites may be found in the liver
Malignant tumors are likely to reach the liver eventually

- Through the portal system or lymphatic channels
- By direct extension from an abdominal tumor.

The liver apparently is an ideal place for these malignant cells to thrive.
Often the first evidence of cancer in an abdominal organ is the appearance of liver metastases;

Primary tumor may never be identified unless exploratory surgery or an autopsy is performed.

Clinical Manifestations

- Pain; continuous, dull ache in the right upper quadrant, epigastrium or back
- Weight loss
- Loss of strength

- Anorexia and
- Anemia
- The liver may be enlarged and irregular on palpation.
- Jaundice is present only if the larger bile ducts are occluded by the pressure of malignant nodules in the hilum of the liver.
- Ascites develops if such nodules obstruct the portal veins or if tumor tissue is seeded in the peritoneal cavity.

Assessment and Diagnostic Findings

Diagnosis is based on clinical signs and symptoms, history and physical examination and results of laboratory and x-ray studies.

- Possible laboratory findings include:
  - Increased liver enzymes: serum levels of bilirubin, alkaline phosphatase, AST, GGT, and lactic dehydrogenase
  - Leukocytosis (increased white blood cells)
  - Erythrocytosis (increased red blood cells)
  - Hypercalcemia
  - Hypoglycemia and
  - Hypocholesterolemia.
- The serum level of alpha-fetoprotein (AFP): a tumor marker is elevated in 30% to 40% of patients with primary liver cancer, thus indicating primary liver cancer.
- Levels of carcinoembryonic antigen (CEA); a marker of advanced cancer of the digestive tract, may be elevated; thus specific for metastatic liver cancer.
- X-rays, liver scans, CT scans, ultrasound studies, MRI, arteriography and laparoscopy may help determine the extent of the cancer.
- Positive emission tomograms (PET scans) are used to evaluate a wide range of metastatic tumors of the liver.
- Confirmation of a tumor's histology can be made by biopsy under imaging guidance (CT scan or ultrasound) or laparoscopically.

Medical Management

Radiation therapy:

- The use of external beam radiation is limited by the radiosensitivity of normal hepatocytes as doses over 2,500 to 3,000 cGy may result in radiation hepatitis.
- More effective methods of delivering radiation to tumors of the liver include;
  - Intravenous or intra-arterial injection of antibodies tagged with radioactive isotopes and specifically attack tumor-associated antigens and

- Percutaneous placement of a high-intensity source for interstitial radiation therapy (delivering radiation directly to the tumor cells).

Chemotherapy;

- It also may be used as adjuvant therapy after surgical resection of tumors.
- It may be delivered as systemic or regional infusion chemotherapy.
- Both methods describes administration of anti-neoplastic agents to case with primary and metastatic hepatic tumors.
- These therapies may prolong survival and improve quality of life by reducing pain and discomfort, their major effect is palliative.

Non-Surgical Palliative Treatments

Percutaneous Biliary Drainage

It is also called trans-hepatic drainage

The aim of the procedure is to bypass the obstructed biliary ducts;

The duct is often obstructed by liver, pancreatic or bile duct tumors

- Indications include;
  - Patients with inoperable tumors or
  - Patients with poor surgical risks.

The procedure requires inserting a catheter through the abdominal wall and advanced it past the obstruction into the duodenum under fluoroscopy

- The purposes of the procedure include;
  - To re-establish biliary drainage
  - To relieve pressure and pain from the buildup of bile behind the obstruction
  - To decrease pruritus and jaundice.

The procedure seek to make the patient more comfortable and improve the quality of life.

The bile is observed closely for amount, colour and presence of blood and debris.

- Complications of percutaneous biliary drainage include
  - Sepsis
  - Leakage of bile
  - Hemorrhage and
  - Re-obstruction of the biliary system by debris in the catheter or from encroaching tumor.
- Signs of complications include;
  - Fever and chills
  - Changes in vital signs

- Evidence of biliary obstruction, including increased pain or pressure, pruritus and recurrence of jaundice.

Laser hyperthermia:

- It has been used to treat hepatic metastases.
- It requires direct application of heat to tumors to cause necrosis of the tumor cells while sparing normal tissue.

Immunotherapy:

- Another treatment modality under investigation
- This therapy uses lymphocytes with antitumor reactivity to cause regression of the tumor; it is indicated for metastatic cancer with no response to standard treatment.

Trans-catheter arterial embolization:

- The treatment induces tumor ischemia by interrupting arterial blood flow to small tumors.
- It is achieved by injecting small particulate embolic or chemotherapeutic agents into the artery supplying the tumor.

Ultrasound-guided injection of alcohol into multiple small lesions;

- Promotes dehydration of tumor cells and tumor necrosis

Surgical Management

Surgical resection is the treatment of choice when HCC is confined to one lobe of the liver.

This allows for recovery of the remaining liver tissue.
Capitalizing on the regenerative capacity of the liver cells, some surgeons have successfully removed 90% of the liver.

The presence of cirrhosis limits the ability of the liver to regenerate.

- Preparation for surgery include;
  - Assessment of the patient's nutritional and fluid status.
  - Support, explanation, and encouragement are provided to help the patient prepare psychologically for the surgery.
  - Extensive diagnostic studies may be performed.
  - Specific studies may include liver scan, liver biopsy, cholangiography, selective hepatic angiography, percutaneous needle biopsy, peritoneoscopy, laparoscopy, ultrasound, CT scans, MRI, and blood tests, particularly determinations of serum alkaline phosphatase, AST and GGT and its isoenzymes.

Surgical procedures include

- ***Lobectomy:***Removal of a lobe of the liver: the most common surgical procedure for excising a liver tumor. The right liver lobectomy or an extended right lobectomy (including the medial left lobe), indicate the use of a thoracoabdominal incision. An extensive abdominal incision is made for a left lobectomy.
- ***Cryosurgery (cryoablation);*** tumors are destroyed by liquid nitrogen at −196°C. The treatment requires delivery of two or three freeze-and-thaw cycles via probes during open laparotomy. This technique has been used alone or as an adjunct to hepatic resection in HCC and colorectal metastases not amenable to radical surgical excision.
- ***Liver Transplantation;***Removing the liver and replacing it with a healthy donor organ is another way to treat liver cancer. Recurrence of the primary liver malignancy after transplantation, however has been reported in 70% to 85% of cases.

Nursing Management

- Precipitous hypoglycemia from decreased gluconeogenesis
- Metabolic abnormalities require careful attention.
- A constant infusion of 10% glucose within the first 48 hours of surgery.
- Infusions of blood and IV fluids due to extensive blood loss during surgery.
- Constant, close monitoring and care for the first 2 or 3 days.
- The patient undergoing cryosurgery is monitored closely for hypothermia, hemorrhage or bile leak.

Potential complications include;

- Cardiopulmonary and vascular complications
- Respiratory compromise and
- Liver dysfunction.
- Myoglobinuria: due to tissue necrosis: can be managed with hydration, diuresis and medications (allopurinol) to bind to and aid in the excretion of toxic products.

3.4.3 Liver Transplantation

Liver transplantation is used to treat life-threatening, end-stage liver disease for which no other form of treatment is available.

It is now recognized as an established therapeutic modality rather than an experimental procedure to treat these disorders.

The transplantation procedure involves total removal of the diseased liver and its replacement with a healthy liver in the same anatomic location ***(orthotopic liver transplantation [OLT])*** with anatomic reconstruction of the hepatic vasculature and biliary tract as close to normal as possible.

The success of liver transplantation depends on successful immunosuppression which can be induced with immunosuppressants such as cyclosporine (Neoral), corticosteroids, azathioprine (Imuran), mycophenolatemofetil (CellCept), OKT3 (a monoclonal antibody), tacrolimus (FK506, Prograf), sirolimus (formerly known as rapamycin [Rapamune]) and anti-thymocyte globulin.

General indications for liver transplantation include;

- Irreversible advanced chronic liver disease
- Fulminant hepatic failure
- Metabolic liver diseases and
- Some hepatic malignancies
- Hepatocellular liver disease (e.g., viral hepatitis, drug- and alcohol-induced liver disease, and Wilson's disease)
- Cholestatic diseases (primary biliary cirrhosis, sclerosing cholangitis and biliary atresia)
- Contraindications include systemic problems that influence preoperative and postoperative care like severe GI bleeding and hepatic coma.

Determinants of successful transplant;

- Long-standing systemic problems resulting from the primary liver disease.
- Previous surgery of the abdomen, including procedures to treat complications of advanced liver disease (i.e., shunt procedures used to treat portal hypertension and esophageal varices).

Surgical Procedure

- Preparation of the donor liver; include
  - Freeing it from other structures
  - Flushing bile from the gallbladder to prevent damage to the walls of the biliary tract
  - Perfusion of the liver with a preservative
  - Cooling of the liver.
- The procedure:
  - Flush the donor liver with cold lactated Ringer's solution to remove potassium and air bubbles before placing it in the recipient
  - Anastomose the blood vessels and bile duct of the donor liver with the recipient liver.

- Perform biliary reconstruction with an end-to-end or end-to-side anastomosis (between the common bile duct of the graft and a loop (Roux-en-Y portion) of jejunum) of the donor and recipient common bile ducts
- Insert a stented T-tube for external drainage of bile.
- Reestablish portal circulation to the new liver by;

- Ligating as many venous collateral vessels as possible
- Lysing adhesions from previous abdominal surgery
  - Reversing a shunt procedure if it was performed previously.

Liver transplantation is a long surgical procedure and blood loss during the procedure may be extensive.

Complications

The post-operative complication

- Immediate postoperative complications may include;
- Bleeding; due to coagulopathy, portal hypertension and fibrinolysis caused by ischemic injury to the donor liver.
- Infection; is the leading cause of death after liver transplantation; the chances of pulmonary and fungal infections are increased by the immunosuppression needed to prevent rejection.
- Rejection; A transplanted liver is perceived by the immune system as a foreign antigen and triggers an immune response, activation of T lymphocytes that attack and destroy the transplanted liver. Long term treatment with immunosuppressive agents to prevent this.
- Disruption, infection or obstruction of the biliary anastomosis and impaired biliary drainage.
- Vascular thrombosis and stenosis.

Nursing Management

Emotional and psychological care of the patient and family

Referral of the patient and family to a psychiatric liaison nurse, psychologist, psychiatrist, or spiritual advisor may help to them as they deal with the stressors associated with end-stage liver disease and liver transplantation.

Preoperative Nursing Interventions

- An extensive diagnostic evaluation.
- The nurse and other health care team members provide the patient and family with full explanations about the procedure, the chances of success, and the risks, including the side effects of long-term immunosuppression.
- The need for close follow-up and lifelong compliance with the therapeutic regimen, including immunosuppression, is explained to the patient and family.

- Obtain a written consent for surgery.
- Seek for matched donor organ as they become available.
- Support the patient and family through the stressful waiting period of searching for appropriate organ.
- The patient must be accessible at all times in case an appropriate liver becomes available.
- Treat malnutrition, massive Ascites, fluid and electrolyte disturbances to increase the chance of a successful outcome
- The nurse serves as a patient and family advocate and assumes the important role of link between the patient and the other members of the transplant team.
- The nurse also serves as a resource to other nurses and health care team members involved in evaluating and caring for the patient.

Post-Operative Nursing Interventions

Strict infection prophylaxis because of immunosuppressive medications
Monitor closely the following;

- Cardiovascular functions: Mean arterial pressure, cardiac output, Pulmonary artery pressures (central venous pressure and pulmonary capillary wedge pressure), heart rate and blood pressure.
- Pulmonary functions through mixed venous blood gases, oxygen saturation, oxygen demand and delivery
- Renal functions through urine output, electrolyte levels
- Neurologic and metabolic functions; blood glucose levels
- Liver function tests.
- Coagulation profile
- Chest x-ray
- Electrocardiogram
- Drainage from Jackson-Pratt tubes
- Respiratory management
    - Endotracheal intubation tube in place and will require
    - Mechanical ventilation
    - Suctioning is performed as required
    - Sterile humidification is provided.
- Vital signs monitoring
- After weaning from the endotracheal tube, use an incentive spirometer to decrease the risk for atelectasis.
- Maintain arterial lines and the urinary catheter are removed, the patient is.
- Assist patient to get out of bed and ambulate as tolerated.
- Promote patients participation in self-care to prevent the complications associated with immobility.

- Close monitoring for signs and symptoms of liver dysfunction and signs of rejection should continue throughout the hospital stay.
- Plans for close follow-up after discharge
- Teaching, initiated during the preoperative period, continues after surgery.

4.0 SUMMARY

At this juncture, you are expected to have learnt about:

- Discuss the various forms of hepatitis
- Discuss jaundice
- discuss comprehensively, cirrhosis of the liver
- discuss cancer of the liver
- explain nursing responsibilities associated with liver transplant.

Activities

Now that you have learnt this much, visit a hospital of choice within your vicinity and manage a patient with any of the liver pathology described within this unit. Share your answers with your colleague in the discussion forum.

SELF-ASSESSMENT EXERCISE

A 59-year-old white woman received an orthotopic liver transplant 6 months ago. She did well postoperatively and was discharged on immunosuppressive therapy. She now presents with a low-grade fever (99.8° F), 5-lb weight gain, tenderness over her transplanted liver, and mild jaundice. She also reports that her urine has become dark and has decreased in amount over the past 2 weeks. Her liver function test results are abnormal. Additional laboratory test results reveal that the serum level of tacrolimus (one of her immunosuppressant medications) is sub-therapeutic.

i. Describe the relationship of each of her current symptoms to liver dysfunction.
ii. What treatment would you anticipate, and what are the nursing measures for her at this time?
iii. What explanation would you give to her for her symptoms?

You can compare your answers with the content of the course and reference materials. Endeavour to share your answer with your colleagues on the discussion platform.

5.0 REFERENCES/FURTHER READING

Levey, R., Williams-Wilson, B. (2002). Anorexia Nervosa: eMedicine Journal, April 2 2002, Volume 3, Number 4.

Smeltzer, S.C., Brenda, B. (2006). Brunner and Suddhart's Textbook of Medical-Surgical Nursing, 10th edition. Lippincott-William & Wilkins.

Templer, J. W. (2001). Parotitis; eMedicine journal; July, 2001, Vol. 2, Number, 7.

UNIT 4 CARING FOR PATIENT WITH GALLBLADDER DISORDERS

CONTENTS

1.0 INTRODUCTION

The focus of this unit is on exploring various disorders of the gall bladder, with a view to improve your knowledge base and sharpen your clinical competence in caring for patients with these conditions.

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- discuss the various disorders of the gall bladder
- explain the Pathophysiology of gall bladder disorders
- discuss the management of disorders of the gall bladder.

3.0 MAIN CONTENT

3.1 Cholecystitis

Definition of Terms:

- Cholecystitis: inflammation of the gallbladder
- Cholelithiasis: the presence of calculi in the gallbladder
- Cholecystectomy: removal of the gallbladder
- Cholecystostomy: opening and drainage of the gallbladder
- Choledochotomy: opening into the common duct
- Choledocholithiasis: stones in the common duct
- Choledocholithotomy: incision of common bile duct for removal of stones
- Choledochoduodenostomy: anastomosis of common duct to duodenum
- Choledochojejunostomy: anastomosis of common duct to jejunum
- Lithotripsy: disintegration of gallstones by shock waves

- **Laparoscopic cholecystectomy:** removal of gallbladder through endoscopic procedure
- **Laser cholecystectomy:** removal of gallbladder using laser rather than scalpel and traditional surgical instruments

Cholecystitis is an acute inflammation of the gallbladder.

Clinical manifestations:

- Pain, tenderness and rigidity of the upper right abdomen that may radiate to the mid-sternal area or right shoulder
- Nausea
- Vomiting.

Clinical forms;

- Calculouscholecystitis; occurs in more than 90% of cases of acute cholecystitis. It is due to gallbladder obstruction by gall stone. Resultant bile stagnation initiates a chemical reaction (inflammation); autolysis and edema. Compression of gall bladder vasculatures cause ischemia, gangrene and subsequent perforation.
- Bacterial cholecystitis: rarely caused by primary of the gall bladder. May be due to secondary infection of bile with *Escherichia coli, Klebsiella* species and other enteric organisms
- Acalculouscholecystitis; is an acute inflammation of the gallbladder in the absence of a cholelith (gall stone).

o Etiologic factors include;

- Post major surgical complication
- Severe trauma or burns
- Torsion of the biliary duct
- Cystic duct obstruction
- Primary bacterial infections of the gallbladder
- Multiple blood transfusions
- Alterations in fluids and electrolytes
- Alteration in regional blood flow in the visceral circulation.
- Bile stasis due to lack of gallbladder contraction and
- Increased viscosity of the bile.

Complications;

- An empyema of the gallbladder (purulent fluid in the gallbladder).

3.2 Cholelithiasis

Calculi, or gallstones, usually form in the gallbladder from the solid constituents of bile;
They vary greatly in size, shape, and composition.
They are uncommon in children and young adults

They become increasingly prevalent after 40 years of age up to 50% over the age of 70 and over 50% above 80years.

Common risk factors

- Obesity
- History of multiple pregnancies
- Multiparity
- Frequent changes in weight
- Rapid weight loss
- Treatment with high-dose estrogen (i.e., in prostate cancer)
- Low-dose estrogen therapy (estrogens and clofibrate)
- Ileal resection or disease
- Cystic fibrosis
- Diabetes mellitus.

Pathophysiology

There are two major types of gallstone composition;

- Predominantly bile pigment stones:
  - Probably form precipitation of un-conjugated bilirubin.
  - Often due to hepatic cirrhosis, hemolysis and biliary tract infections.
  - Pigment stones cannot be dissolved and must be removed surgically.
- Primarily cholesterol stones:
  - Derived from solidified cholesterol due to formation of bile supersaturated with cholesterol (i.e. bile secretion with low bile acid: cholesterol content ratio):
  - This stone is primarily insoluble in water;
  - Its solubility depends on bile acids and lecithin (phospholipids) in bile.
  - These stones can irritate the gallbladder and produce inflammation: cholesterol stones affect women four times more than men.

Clinical Manifestations

Gallstones may be silent producing no pain and only mild gastrointestinal symptoms.

Such stones may be detected incidentally during surgery or evaluation for unrelated problems.

The symptoms may be acute or chronic.

- Epigastric distress, such as fullness, abdominal distention and vague pain in the right upper quadrant of the abdomen, may occur. This distress may follow a meal rich in fried or fatty foods.

- Fever
- Palpable abdominal mass.
- Biliary colic; excruciating upper right abdominal pain that radiates to the back or right shoulder usually associated with nausea and vomiting, and is noticeable several hours after a heavy meal. It is caused by contraction of the gallbladder against an obstructed duct.
- Marked tenderness in the right upper quadrant on deep inspiration (prevents full inspiratory excursion).
- Jaundice; usually due to obstruction of the common bile duct with subsequent reflux and absorption in the liver; associated with itching.
- Dark colored urine due to renal excretion of bile pigments.
- Pale, greasy, bulky and clay colored feces often with exaggerated offensive odour due to loss of colored pigments;
- Vitamin Deficiency; especially fat soluble vitamins A, D, E, and K. Therefore, the patient may exhibit bleeding and clotting disorders caused by vitamin K deficiency.

Assessment and Diagnostic Findings

An abdominal x-ray

- It is useful to exclude other causes of symptoms.
- Calcified sufficiently to be visible on plain x-ray studies.

Ultrasonography

- This has replaced oral cholecystography
- It can detect calculi in the gallbladder with 95% accuracy or a dilated common bile duct.

Radionuclide Imaging or Cholescintigraphy; It has been used successfully to diagnose acute cholecystitis.

- Procedure;
  - It involves administering a radioactive agent intravenously, which is expected to be taken up by the hepatocytes and excreted rapidly through the biliary tract.
  - This will be followed by biliary tract scanning and imaging of the gallbladder and biliary tract to outline its patency;
- The main disadvantages are;
  - Longer procedure time
  - Exposure to radiation and
  - Inability to detect gallstones.

Cholecystography
It is required if ultrasound equipment is not available or the ultrasound results are inconclusive.

It can be used as part of the evaluation following gallstone dissolution therapy or lithotripsy.

- Oral cholangiography; may be performed to detect gallstones and assess the filling, concentration and emptying ability of the gallbladder.
    - It requires administration of an iodide-containing contrast agent that will be excreted by the liver and concentrated in the gallbladder followed by series of x-ray film.

Contrast agents include iopanoic acid (Telepaque), iodipamidemeglumine (Cholografin), and sodium ipodate (Oragrafin). These agents are administered orally 10 to 12 hours before the x-ray study

- Nursing responsibilities;
    - Maintain patient on NPO to prevent contraction and emptying of the gallbladder
    - Determine allergies to iodine or seafood before administration
- Contra indications;
    - Jaundice prevents excretion of the radiopaque dye into the gallbladder.

Endoscopic retrograde cholangiopancreatography(ERCP);
This permits direct visualization of structures of the hepatobiliary system via a side-viewing flexible fiberoptic endoscope, inserted into the esophagus to the descending duodenum.

This is followed by a careful advancement of a catheter through the endoscope into the common bile duct (through the sphinter of Oddi).
Fluoroscopy and multiple x-rays are used during ERCP to evaluate the presence and location of ductal stones.

The procedure requires multiple position changes during the procedure, beginning in the left semi-prone position to pass the endoscope. During this, gallstone can be extracted

- Nursing Implications;
    - Before the procedure
- Explain the procedure to the patient
- Explain her expected role during the procedure
- Place patient on nothing by mouth for several hours before the procedure.

- Use moderate sedation with combination of an opioid and a benzodiazepine
- Closely monitor the sedated patient
- Administer glucagon or anticholinergics to eliminate duodenal peristalsis to make cannulation easier.
- Observes closely for signs of respiratory and central nervous system depression, hypotension, over-sedation and vomiting (if glucagon is given).

o During ERCP;

- Monitors intravenous fluids
- Administers medications as prescribed
- Position the patient appropriately

o After the procedure;

- Monitor the patient's condition
- Observe vital signs
- Monitor for signs of perforation or infection.
- Monitor the patient for side effects of any medications received during the procedure
- Observe for return of gag and cough reflexes after the use of local anesthetics.

Percutaneous Trans-hepatic Cholangiography

This involves the injection of dye directly into the biliary tract to clearly outline all components of the biliary system, including the intra hepatic bile ducts, the entire length of the common bile duct, the cystic duct and the gallbladder.

- Purpose:
  - To distinguish hepatocellular jaundice from that caused by biliary obstruction
  - To investigate the gastrointestinal symptoms of a patient whose gallbladder has been removed
  - To locate stones within the bile ducts and
  - To diagnose cancer involving the biliary system.
- Procedure;
  - It is a sterile procedure
  - It is performed under moderate sedation
  - Place the patient on fasting
  - Give the patient receives local anesthesia and intravenous sedation.
  - Obtain coagulation parameters and platelet count to minimize the risk for bleeding.
  - Give broad-spectrum antibiotics during the procedure due to the high prevalence of bacterial colonization from obstructed biliary systems
  - Perform local infiltration with a local anesthetic agent

- Insert a flexible needle into the liver from the right side in the mid-clavicular line immediately beneath the right costal margin.
- Note bile aspiration to mark a successful entry of a duct
- Ultrasound guidance can be used for duct puncture.
- Send bile aspirate and samples for bacteriology and cytology
- Inject a water-soluble contrast agent to fill the biliary system.
- Tilt the fluoroscopy table and reposition the patient to allow x-rays to be taken in multiple projections.
- Take a delayed x-ray views to identify abnormalities of more distant ducts and determine the length of a stricture or multiple strictures.
- Aspirate as much dye and bile as possible before removing the needle to forestall subsequent leakage into the needle tract and eventually into the peritoneal cavity, thus minimizing the risk of bile peritonitis

- Contraindication:
  - Liver dysfunction
  - Jaundice.

Medical Management

Supportive Therapy

Approximately 80% of the cases will achieve remission with;

- Rest
- Intravenous fluids
- Nasogastric suction
- Analgesia and
- Antibiotic agents
- Delay surgical intervention unless the patient's condition deteriorates
- Dietary management; usually limited to low-fat liquids, supplements high in protein and carbohydrate in form of skim milk. The patient should avoid eggs, cream, pork, fried foods, cheese and rich dressings, gas-forming vegetables, and alcohol.

Pharmacologic Therapy

Ursodeoxycholic acid (UDCA) and chenodeoxycholic acid (chenodiol or CDCA); to dissolve small, radiolucent gallstones composed primarily of cholesterol. It acts by inhibiting the synthesis and secretion of cholesterol, thereby desaturating bile. It can reduce the size of an existing stones, dissolve small ones and prevent new stones from forming.

- Contra indicated in cases with;
  - Significant, frequent symptoms

- Cystic duct occlusion or
- Pigment stones.

Nonsurgical Removal of Gallstones

Dissolving Gallstones

Infusion of a solvent (mono-octanoin or methyl tertiary butyl ether [MTBE]) into the gallbladder through the following routes:

- Percutaneously through a tube or catheter inserted directly into the gallbladder
- A tube or drain inserted through a T-tube tract
- An ERCP endoscope
- A trans-nasal biliary catheter; the catheter is introduced through the mouth into the common bile duct. The upper end of the tube is then rerouted from the mouth to the nose and left in place. This enables the patient to eat and drink normally while passage of stones is monitored or chemical solvents are infused to dissolve the stones. This method of dissolution of stones is not widely used in patients with gallstone disease.

Stone Removal by Instrumentation

This can be done using the following methods;

Threading a catheter and instrument with a basket attached through the T-tube tract or fistula formed at the time of T-tube insertion to retrieve and remove the stones lodged in the common bile duct with the basket.

Using the ERCP endoscope, a cutting instrument may be passed into the ampulla of Vater of the common bile duct to cut the submucosal fibers or papilla of the sphincter of Oddi and enlarge its opening to allow the stones pass spontaneously into the duodenum. The stone may be retrieved with another instrument with a small basket or balloon at its tip may be inserted through the endoscope to retrieve the stones.

Complications after this procedure are rare, but may include;

- Bleeding
- Perforation
- Pancreatitis
- Sepsis.

Extracorporeal Shock-Wave Lithotripsy (ESWL)

The word **lithotripsy** is derived from *lithos,* meaning stone, and *tripsis,* meaning rubbing or friction.

This is a non-invasive procedure

It uses repeated shock waves directed at the gallstones to fragment the stones.

The energy is transmitted to the body through a fluid-filled bag or while the patient is immersed in a water bath.

The converging shock waves are directed to the stones to be fragmented and fragmented stones can be removed;

- Spontaneously from the gallbladder or common bile duct
- Manually by endoscopy
- Through oral bile acid or
- Dissolution in solvents.

The procedure is often done on an outpatient basis because it requires no incision and no hospitalization.

Intracorporeal Lithotripsy

The procedure fragments stones in the gallbladder or common bile using laser pulse technology.

The laser pulse is directed under fluoroscopic guidance with devices that can distinguish between stones and tissue. The laser pulse produces rapid expansion and disintegration of plasma on the stone surface, resulting in a mechanical shock wave.

- ***Electrohydraulic lithotripsy;*** the procedure uses a probe with two electrodes to deliver electric sparks in rapid pulses. This thus creates expansion of the liquid environment surrounding the gallstones and the resultant pressure waves fragments the stones. This technique can be done percutaneously with a basket or balloon catheter system or direct visualization through an endoscope. Repeated procedures may be necessary because of stone size, local anatomy, bleeding or technical difficulty.
- ***A nasobiliary tube*** can be inserted to allow for biliary decompression and prevent stone impaction in the common bile duct. This approach allows time for improvement in the patient's clinical condition until gallstones are cleared endoscopically, percutaneously, or surgically.

Surgical Management

Surgical treatment is aimed at;

- Relieving persistent symptoms
- Removing the cause of biliary colic and
- Treating acute cholecystitis.

Surgery may be delayed until the patient's symptoms have subsided
It may be performed as an emergency procedure if the patient's condition necessitates it.

Pre-Operative Measures

- A chest x-ray
- Electrocardiogram

- Liver function tests
- X-ray studies of the gallbladder
- Vitamin K administration if the prothrombin level is low.
- Blood component therapy before surgery
- Nutritional requirements; Suboptimal status may necessitate;
- Intravenous glucose
- Protein hydrolysate supplements to aid wound healing and help prevent liver damage
- Pre- Operative instructions and explanations regarding turning and deep breathing to prevent pneumonia and atelectasis
- Inform the patient that drainage tubes and a nasogastric tube and suction may be required during the immediate postoperative period.

Surgical Procedures

Laparoscopic Cholecystectomy

The procedure has become the new standard for therapy of symptomatic gallstones.

It means surgical removal of the gallbladder

Common bile duct obstruction by a gallstone will require an ERCP with sphincterotomy to explore the duct before laparoscopy.

Before the procedure, inform the patient that an open abdominal procedure may be necessary and general anesthesia is administered.

Procedure;

- Laparoscopic cholecystectomy is performed through a small incision or puncture made through the abdominal wall in the umbilicus.
- The abdominal cavity is insufflated with carbon dioxide (pneumoperitoneum) to assist in inserting the laparoscope and to aid the surgeon in visualizing the abdominal structures.
- The fiberoptic scope is inserted through the small umbilical incision.
- Several additional punctures or small incisions are made in the abdominal wall to introduce other surgical instruments into the operative field.
- The surgeon visualizes the biliary system through the laparoscope;
- A camera attached to the scope permits a view of the intra-abdominal field to be transmitted to a television monitor.
- After the cystic duct is dissected, the common bile duct is imaged by ultrasound or cholangiography to evaluate the anatomy and identify stones.
- The cystic artery is dissected free and clipped.

- The gallbladder is separated away from the hepatic bed and dissected.
- The gallbladder is then removed from the abdominal cavity after bile and small stones are aspirated.
- Stone forceps also can be used to remove or crush larger stones.

Advantages of the procedure:

- Avoids paralytic ileus that occurs with open abdominal surgery
- There is less postoperative abdominal pain.
- The patient is often discharged from the hospital on the day of surgery.
- The patient can resume full activity within a day or two and job within a week of the surgery.

Post operative management;

- Obtain written as well as verbal instructions about managing postoperative pain.
- Report signs and symptoms of intra-abdominal complications, including loss of appetite, vomiting, pain, distention of the abdomen and temperature elevation
- Ensure the patient has assistance at home during the first 24 to 48 hours.
- Explain that pain in the right shoulder or scapular area is from migration of the CO_2 used to insufflate the abdominal cavity during the procedure.
- Recommend use of a heating pad for 15 to 20 minutes hourly, walking, and sitting up when in bed to manage this.

The most serious complication is a bile duct injury.

Cholecystectomy

This is the removal of the gallbladder through an abdominal incision (usually right subcostal).

The procedure requires ligation of the cystic duct and artery.

The procedure is performed for acute and chronic cholecystitis.

Some patients may require a drain placed close to the gallbladder which should be closely observed for bile leak: A small leak should close spontaneously in a few days.

The drain prevents accumulation of bile.

Usually only a small amount of serosanguinous fluid will drain in the initial 24 hours after surgery.

The drain is usually maintained if there is excess oozing or bile leakage.

The bile duct may be injured by the drain but occurs less frequently.

Mini-cholecystectomy
This is removal of the gallbladder through a small incision
The surgical incision may be extended to remove large gallbladder stones.
Drains may or may not be used.
The procedure is cost saving due to shorter hospital stay.

Choledochostomy
Removal of stonesthrough an incision into the common duct
After the stones have been evacuated, a tube usually is inserted into the duct for drainage of bile until edema subsides.
This tube is connected to gravity drainage tubing.
It is done along side with cholecystectomy.

Surgical Cholecystostomy
Indications;
- When patient's condition prevents more extensive surgery.
- When an acute inflammatory reaction is severe.

It involves surgical opening into the gallbladder to remove the stones and drain the bile or purulent drainage followed by securing a drainage tube with a purse-string suture.

The drainage tube is connected to a drainage system to prevent bile from leaking around the tube or escaping into the peritoneal cavity.

After recovery from the acute episode, the patient may return for cholecystectomy.

Surgical cholecystostomy has a high mortality rate (reported as high as 20% to 30%) due to underlying disease process.

Percutaneous Cholecystostomy
It has been used to treat and diagnose acute cholecystitis
Indicated for patients with poor tolerance for general anesthesia and any surgical procedure: These may include patients with;
- Sepsis

- Severe cardiac failure
- Renal failure
- Pulmonary failure or
- Liver failure.

The procedure;

- Induce a local anesthesia
- Insert a fine needle through the abdominal wall under liver edge into the gallbladder under the guidance of ultrasound or computed tomography.
- Aspirate bile to ensure adequate placement of the needle.
- Insert a catheter into the gallbladder to decompress the biliary tract.
- The procedure affords almost immediate relief of pain and resolution of signs and symptoms of sepsis and cholecystitis
- Administer antibiotic agents before, during and after the procedure.

4.0 SUMMARY

At this juncture, you are expected to have learnt about:

i. discuss the various disorders of the gall bladder
ii. explain the Pathophysiology of gall bladder disorders
iii. discuss the management of disorders of the gall bladder.

Activities

Now that you have learnt this much, visit a hospital of choice within your vicinity and report the management of a patient with any of the disorders with particular reference to the diagnostic modalities used. Share your answers with your colleague in the discussion forum.

SELF-ASSESSMENT EXERCISE

A 30-year-old, obese woman has been diagnosed with cholelithiasis. She reported the history of oral contraceptive medications for 3 years now.

i. Discuss the clinical manifestations of this condition.
ii. What is the nursing management of this patient?
iii. Discuss medical and surgical treatment of this condition
iv. What instructions about the condition will be necessary during convalescence?

You can compare your answers with the content of the course and reference materials. Endeavour to share your answer with your colleagues on the discussion platform.

5.0 REFERENCES/FURTHER READING

Levey, R., Williams-Wilson, B. (2002). Anorexia Nervosa: eMedicine Journal, April 2 2002, Volume 3, Number 4.

Smeltzer, S.C., Brenda, B. (2006). Brunner and Suddhart's Textbook of Medical-Surgical Nursing, 10th edition. Lippincott-William & Wilkins.

Templer, J. W. (2001). Parotitis; eMedicine journal; July, 2001, Vol. 2, Number, 7.

UNIT 5 CARING FOR PATIENT WITH EXOCRINE PANCREATIC DISORDERS

CONTENTS

1.0 INTRODUCTION

The focuses of this unit is on exploring the disorders of the pancreas with a view to improving your knowledge base and sharpening your clinical competence in caring for patients with these conditions.

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- describe the course pancreatitis
- discuss various malignancies of the pancreas
- list various predisposing factors to chronic pancreatitis
- explain the Pathophysiology of chronic pancreatitis
- discuss surgical lines of managing pancreatic malignancy
- discuss nursing management of patients with pancreatic malignancy.

3.0 MAIN CONTENT

3.1 Pancreatitis

This is inflammation of the pancreas
It is a serious disorder.
The most basic classification system divides the disorder into acute or chronic forms.

- Acute pancreatitis;
    - It can be a medical emergency
    - It is associated with a high risk for life-threatening complications and mortality

- Acute pancreatitis does not usually lead to chronic pancreatitis unless complications develop.
- Chronic pancreatitis;
- It often goes undetected until 80% to 90% of the exocrine and endocrine tissue is destroyed.
- Chronic pancreatitis can present with acute exercerbations
- Age related incidence; Typically affects
- Men aged 40 to 45years with a history of alcoholism
- Women aged 50 to 55years with a history of biliary disease.

Mechanism of pancreatitis

- Pancreatitis is commonly described as auto-digestion of the pancreas.
- This is generally triggered by;
- Pancreatic duct obstruction
- Associated hypersecretion of the exocrine enzymes of the pancreas
- Redirected enzymes into the bile duct, where they are activated
- Reflux of activated enzymes together with bile into the pancreatic duct
- Pancreatitis thus ensue.

3.2 Acute Pancreatitis

Acute pancreatitis can presents as a mild, self-limiting disorder to a severe, rapidly fatal disease resistant to any treatment.

- Mild acute pancreatitis;
  - This is characterized by localized pancreatic edema and inflammation
  - It is due to minimal organ dysfunction
  - It resolves completely usually within 6 months
- Severe acute pancreatitis; characterized by;
  - Extensive auto-digestion of the pancreas
  - Pancreatic tissue necrosis and
  - Retroperitoneal tissue damage
  - Local complications include;
  - Pancreatic cysts or abscesses
  - Acute fluid collections in or near the pancreas.

Mortality may be as high as 50% or higher

Predisposing factors

- Biliary tract disease
- Gallstones
- Long-term use of alcohol.

Pathophysiology

The main common cause of acute pancreatitis is due to auto-digestion of the pancreas by its own proteolytic enzymes principally trypsin

This process is heralded by;

- Obstruction of the common bile duct with gallstones at the ampulla of Vater results in;
- Obstruction of the flow of pancreatic juice
- Reflux of bile from the common bile duct into the pancreatic duct.

This leads to activation of the powerful pancreatic enzymes within the substance of the pancreas.

Normally pancreatic enzymes remain inactive until the pancreatic juice reaches the lumen of the duodenum.

Activation of these enzymes leads to vasodilation, increased vascular permeability, necrosis, erosion and hemorrhage.

Other less common causes of pancreatitis include;

- Bacterial or viral infection
- Pancreatitis following mumps virus infection
- Duodenitis with spasm and edema of the ampulla of Vater
- Blunt abdominal trauma
- Peptic ulcer disease
- Ischemic vascular disease
- Hyperlipidemia
- Hypercalcemia
- Medications like corticosteroids, thiazide, diuretics and oral contraceptives
- Surgery on or near the pancreas
- After instrumentation of the pancreatic duct
- Idiopathy in up to 20% of the cases of acute pancreatitis
- Hereditary pancreatitis.

Prognosis;

- Mortality is as high as 10% due to co morbidities such as shock, anoxia, hypotension or fluid and electrolyte imbalances.
- Acute attacks may resolve spontaneously and the patient completely recovers.
- Reoccurrence is possible without permanent damage.
- Some cases may however progress to chronic pancreatitis.

Clinical Manifestations

- The patient appears acutely ill.

- Severe abdominal pain; the major symptom that causes the patient to seek medical care.
  - The pain may be localized in the mid-epigastrium
  - Pain may be diffuse and difficult to localize in some cases
  - Abdominal pain and tenderness due to increased tension on the pancreatic capsule and pancreatic ducts obstruction
  - Pain is frequently acute in onset, occurring 24 to 48 hours after a very heavy meal or alcohol ingestion and is unrelieved by antacids.
- Back pain result from irritation and edema of the inflamed pancreas
- Abdominal distention;
- Poorly defined, palpable abdominal mass; and
- Decreased peristalsis
- Abdominal guarding is present
- A rigid or board-like abdomen may develop (generally an ominous sign)
- Ecchymosis (bruising) in the flank or around the umbilicus (may indicate severe pancreatitis) Nausea and vomiting: contained gastric juice and occasionally bile-stained
- Fever
- Jaundice
- Mental confusion and agitation
- Hypotension (reflects hypovolemia and shock) caused by the loss of large amounts of protein-rich fluid into the tissues and peritoneal cavity.
- Respiratory distress associated with diffuse pulmonary infiltrates, dyspnea, tachypnea and abnormal blood gas values.

Systemic complications include;

- Acute respiratory distress syndrome
- Shock
- Disseminated intravascular coagulopathy
- Pleural effusion
- Acute renal failure
- Myocardial depression
- Hypocalcemia
- Hyperglycemia
- Disseminated intravascular coagulopathy (DIC)
- Sepsis.

Assessment and Diagnostic Findings

- The diagnosis is based on;
  - History of abdominal pain
  - Presence of known risk factors

- Physical examination findings and
- Diagnostic findings
- Serum amylase and lipase levels; usually rise in excess of three times their normal upper limit within 24 hours and may remain elevated for 7 to 14 days in 90% of cases
- Urinary amylase levels; also become and remain elevated longer than serum amylase levels.
- White blood cell count; usually elevated
- Hypocalcemia; correlates well with the severity of pancreatitis.
- Blood and urine glucose level; transiently high
- Serum bilirubin levels; elevated
- Chest and Abdominal X-ray studies; to differentiate pancreatitis from other similar disorders.
- Ultrasound and contrast-enhanced computed tomography scans; to identify an increase in the diameter of the pancreas and to detect pancreatic cysts, abscesses or pseudocysts.
- Hematocrit and hemoglobin levels; to monitor for bleeding
- Peritoneal fluid analysis may contain increased pancreatic enzymes levels.
- Stool analysis; stools are often bulky, pale and foul-smelling; Fat content of stools varies between 50% and 90% in pancreatic disease.
- ERCP; rarely used in the diagnostic evaluation of acute pancreatitis because the patient is acutely ill; however, it may be valuable in the treatment of gallstone pancreatitis.

Medical Management

Management is directed toward;

- Relieving symptoms and
- Preventing or treating complications

Management strategies;

- Withhold all oral intake to inhibit pancreatic stimulation and secretion of pancreatic enzymes
- Parenteral nutrition particularly in debilitated patients
- Nasogastric suction to;
- Relieve nausea and vomiting
- Decrease painful abdominal distention and paralytic ileus
- Remove hydrochloric acid and subsequent pancreatic stimulation
- Histamine-2 (H2) antagonists (e.g., cimetidine [Tagamet] and ranitidine [Zantac]); to decrease pancreatic activity by inhibiting HCl secretion.
- Pain Management; pain medication for sufficient pain relief and minimize restlessness;
- Avoid morphine and morphine derivatives because it cause spasm of the sphincter of Oddi;

- Meperidine (Demerol); often used because it is less likely to cause spasm of the sphincter
- Antiemetic agents; to prevent vomiting
- Correction of fluid and blood loss and low albumin levels to maintain fluid volume and prevent renal failure.
- Hemodynamic monitoring
- Arterial blood gas monitoring
- Antibiotic agents; if infection is present
- Insulin; to treat significant hyperglycemia
- Respiratory Care
  - Close monitoring of arterial blood gases
  - Humidified oxygen
  - Intubation and mechanical ventilation
- Biliary Drainage; include placement of;
  - Biliary drains for external drainage
  - Stents (indwelling tubes) in the pancreatic duct through endoscopy to reestablish pancreatic drainage.

Surgical Intervention

- Risky because acutely ill patient has a poor surgical risk
- The aims of the surgery may include;
  - To assist in diagnosis (diagnostic laparotomy)
  - To establish pancreatic drainage
  - To resect or debride a necrotic pancreas.

Post-Acute Management

- Antacids; when acute pancreatitis begins to resolve.
- Gradually initiate low fat and protein diets.
- Eliminate caffeine and alcohol from the diet.
- Excercerbation will require discontinuation of thiazide diuretics, corticosteroids or oral contraceptives.
- Follow-up ultrasound, x-ray studies, or ERCP to determine extent of resolution and development of abscesses and pseudocysts.

3.3 Chronic Pancreatitis

Pathophysiology

- Chronic pancreatitis is an inflammatory disorder characterized by progressive anatomic and functional destruction of the pancreas.
- Evident changes in the gland include;
  - Fibrosis due to increase pressure within the pancreas
  - Mechanical obstruction of the pancreatic and common bile ducts and the duodenum.
  - Atrophy of the epithelium of the ducts.
  - Inflammation and destruction of the secreting cells of the pancreas.

- Calcification of the gland (deposition of calcium stones within the ducts).

Major causes of chronic pancreatitis

- Excessive and prolonged consumption of alcohol accounts for approximately 70% of the cases. The impact of alcohol on the pancreas include;
  - Hypersecretion of protein in pancreatic secretions with resultant formation of protein plugs and calculi within the pancreatic ducts.
  - Direct toxic effect on the cells of the pancreas.
- Malnutrition; Diets poor in protein content and either very high or very low in fat.

Clinical Manifestations

Chronic pancreatitis is characterized by:

- Recurring attacks of severe upper abdominal and back pain.
- Associated vomiting
- As the disease progresses, recurring attacks of pain are more severe, more frequent, and of longer duration.
- Weight loss in more than 75% of patients usually caused by decreased dietary intake secondary to anorexia or fear that eating will precipitate another attack.
- Malabsorption occurs late in the disease, when as little as 10% of pancreatic function remains. Manifests as steatorrhea (frequent, frothy and foul-smelling stool) because of high fat content in stool.

Assessment and Diagnostic Findings

- ERCP; outlines details about the anatomy of the pancreas and the pancreatic and biliary ducts. It is also helpful in obtaining tissue for analysis and differentiating pancreatitis from other conditions, such as carcinoma.
- Imaging procedures; Computed tomography scanning or ultrasound is helpful to detect pancreatic cysts.
- Glucose tolerance test; evaluates pancreatic islet cell function
- Serum amylase levels and White blood cell count; may not be elevated significantly.

Medical Management

Management depends on its probable cause in each patient.

- Goals of treatment;
  - Preventing and managing acute attacks.
  - Relieving pain and discomfort.

- Managing exocrine and endocrine insufficiency of pancreatitis.

Non-Surgical Management

- Indications;
  - Patient who refuses surgery.
  - Patients with poor surgical risk.
  - Patients whose disease and symptoms do not warrant surgical intervention.

Management strategies:

- Endoscopy to remove pancreatic duct stones and stent strictures; may be effective in selected patients to manage pain and relieve obstruction.
- Management of abdominal pain and discomfort usually with non-opioid analgesics
- Dietary management; avoiding alcohol and other foods that tend to produce abdominal pain and discomfort.
- Insulin or Oral anti-diabetic agents; to manage diabetes mellitus resulting from dysfunction of the pancreatic islet cells
- Pancreatic enzyme replacement; indicated in the patient with malabsorption and steatorrhea.

Surgical Management

- Goals of surgical management;
  - To relieve abdominal pain and discomfort
  - To restore drainage of pancreatic secretions
  - To reduce the frequency of acute attacks of pancreatitis.
- The choice of surgical procedure depends on
  - Location of disease within the pancreas
  - Co-morbidities like; diabetes, exocrine insufficiency, biliary stenosis and pseudocysts of the pancreas.

Surgical options;

- ***Pancreaticojejunostomy(also referred to as Roux-en-Y)*** with a side-to-side anastomosis or joining of the pancreatic duct to the jejunum allows drainage of the pancreatic secretions into the jejunum. Pain relief only lasts for 6 months in more than 80% of cases.
- Revision of the sphincter of the ampulla of Vater, to internal drainage of a pancreatic cyst into the stomach.
- Insertion of a stent
- ***Pancreatic resection;*** Whipple resection (pancreaticoduodenectomy); to relieve the pain of chronic pancreatitis.

- ***Pancreatic removal;*** Total removal (pancreatectomy) will require auto-transplantation or implantation of the patient's pancreatic islet cells to preserve the endocrine function of the pancreas
- ***Cholecystectomy;*** the gallbladder is removed when chronic pancreatitis is associated with gallbladder disease.
- ***A sphincterotomy;*** dividing the sphincter of Oddi, (a muscle that is located at the ampulla of Vater) to improve the drainage of the common bile duct and the pancreatic duct.

Post operative expectations;

- A T-tube usually is placed in the common bile duct, requiring a drainage system to collect the bile postoperatively.
- Weight gain and improved nutritional status; post operation may result from reduction in pain associated with eating rather than from correction of malabsorption.
- Post operative morbidity and mortality are high because of;
  - Poor pre-operative physical condition and
  - Concomitant occurrence of cirrhosis.
- Pain and impaired digestion may continue after surgical procedures unless alcohol is avoided completely.

3.4 Cancer of the Pancreas

Primary tumors of the pancreas can arise in any portion of the pancreas (the head, body or tail).

The pancreas can also be the site of metastasis from other tumors i.e. secondary tumors.

Approximately 75% of pancreatic cancers originate in the head of the pancreas.

Tumors can be a functioning one e.g. those involving the insulin-secreting pancreatic islet cells.

Patients usually do not seek medical attention until late in the disease
Pancreatic carcinoma has only a 2% to 5% survival rate at 5 years regardless of the stage of disease at diagnosis or treatment.

Predisposing factors;

- Cigarette smoking.
- Exposure to industrial chemicals or environmental toxins.
- Diet high in fat, meat; their role is not completely clear.
- Diabetes mellitus.
- Chronic pancreatitis.
- Hereditary pancreatitis.

Clinical Manifestations

- These vary depending on the;
  - Location of the lesion e.g. cancer of the head of the pancreas presents with classical obstructive symptoms of the biliary system.
  - Whether functioning e.g. Functioning islet cell tumors, whether benign (adenoma) or malignant (carcinoma) are responsible for the syndrome of hyper-insulinism.
- Symptoms are generally non-specific
  - Classical signs are Pain, Jaundice and (rapid, profound and progressive) weight loss. However, they often do not appear until in advanced disease.
  - Other signs include;
  - Vague upper or mid-abdominal pain or discomfort; with the following attributes;
- It is unrelated to any gastrointestinal function.n
- It is often difficult to describe
- It radiates as a boring pain in the mid-back and is unrelated to posture or activity.
- It is often progressive and severe, requiring the use of opioids.
- It is often more severe at night.
- Relief may be obtained by sitting up and leaning forward
- It is aggravated when lying supine or meals
  - Ascites; due to shedding of malignant pancreatic cells into the peritoneal cavity
  - Symptoms of insulin deficiency: glucosuria, hyperglycemia and abnormal glucose tolerance; diabetes may be an early sign of carcinoma of the pancreas.

Assessment and Diagnostic Findings

- Magnetic resonance imaging and computed tomography; to identify pancreatic tumors.
- ERCP; to diagnose pancreatic carcinoma Cells obtained during ERCP are sent to the laboratory for examination.
- Gastrointestinal x-ray findings may demonstrate deformities in adjacent viscera caused by the impinging pancreatic mass.
- Percutaneous fine-needle aspiration biopsy of the pancreas;
  - Is useful to diagnose pancreatic tumors
  - It requires inserting a needle through the anterior abdominal wall into the pancreatic mass, guided by computed tomography, ultrasound, ERCP, or other imaging techniques to aspirate tissue sample which can be analyzed for malignant cells;
  - Disadvantages of this procedure include;
- A false negative result if small tumors are missed

- Seeding of cancer cells along the needle track; this will require a low-dose radiation to the site before the biopsy to reduce the risk of seeding.
- Percutaneous trans-hepatic cholangiography; to identify obstructions of the biliary tract by a pancreatic tumor
- Several tumor markers (e.g., CA 19-9, CEA, DU-PAN-2); they are nonspecific for pancreatic carcinoma. These are useful as indicators of disease progression.
- Angiography, computed tomography, and laparoscopy; to determine whether the tumor can be removed surgically.
- Intra-operative ultrasonography; to determine metastatic sites on other organs.

Medical Management

Definitive surgical treatment;

- Total excision of the lesion of a localized and resectable tumors (typically tumors in the head of the pancreas)
  - This is often not possible because of probable widespread metastases (especially to the liver, lungs and bones) at the time of diagnosis.

Palliative measures;

- Radiation therapy; although pancreatic tumors may be resistant to standard radiation therapy
- This therapy can be delivered using the following means;
- Intra-operative radiation therapy (IORT) may deliver a high dose of radiation to the tumor with minimal injury to other tissues thus helpful in pain relief.
- Interstitial implantation of radioactive sources; has also been used, although with a high rate of complications.
- Chemotherapy (with fluorouracil and gemcitabine).
- A large biliary stent may be inserted percutaneously or by endoscopy may be used to relieve jaundice.

Nursing Management

- Assessment of the patient's physical status, fluid and nutritional status, and skin integrity and the adequacy of pain management
- Pain management; may require liberal use of opioids with Patient-controlled analgesia especially for the patient with severe, escalating pain
- Improve the level of comfort; using specialty mattresses to protect bony prominences from pressure
- Attention to nutritional requirements
- Skin care to prevent pressure ulcers
- Pain associated with pancreatic cancer

- End-of-life preferences are discussed and honored because of the poor prognosis and likelihood of short survival
- Managing side effects and complications of chemotherapy
- Management of the drainage system if surgery has been performed.
- Monitoring for complications
- A referral for home care may be necessary to help the patient and family deal with the physical problems and discomforts associated with pancreatic cancer and the psychological impact of the disease.

4.0 SUMMARY

At this juncture, you are expected to have learnt about:

- the course pancreatitis
- malignancy of the pancreas
- predisposing factors to chronic pancreatitis
- the Pathophysiology of chronic pancreatitis
- surgical lines of managing pancreatic malignancy
- nursing management of patients with pancreatic malignancy.

Activities
Now that you have learnt this much, draw a hypothetical nursing care plan for a case of chronic pancreatitis.

SELF-ASSESSMENT EXERCISE

A 57-year-old man presents in the accident and emergency with complaints of severe epigastric pain associated with vomiting and diarrhea: he has a positive history of alcoholism and cirrhosis. He was diagnosed to be having acute pancreatitis.

i. Discuss the possible laboratory tests and expected outcomes that will be prescribed for this patient?
ii. What physical assessment findings that will be evident in this patient?
iii. Describe nursing care for this patient.

Note that, you are to grade yourself based on the information provided within the content of the unit. If you have graded yourself poor, you can re-work.

5.0 REFERENCES/FURTHER READING

Levey, R., Williams-Wilson, B. (2002). Anorexia Nervosa: eMedicine Journal, April 2 2002, Volume 3, Number 4.

Smeltzer, S.C., Brenda, B. (2006). Brunner and Suddhart's Textbook of Medical-Surgical Nursing, 10th edition. Lippincott-William & Wilkins.

Templer, J. W. (2001). Parotitis; eMedicine journal; July, 2001, Vol. 2, Number, 7.

1.0 INTRODUCTION

The body has been likened to an efficient machine that runs on metabolic fuels. These fuels mainly include nutrients gotten from foods. Foods are ingested, digested and absorbed into the into the body systems by the gastrointestinal system. This system represents the gateway of food nutrients into the body and thus vital for the running of the body as a whole. You will be expected to consolidate on your knowledge base as well as sharpen your skills in caring for patients with gastrointestinal tract (GIT) disorders.

The focus of this unit will be to explore comprehensively, gastrointestinal (GI) assessment by discussing various modalities of the assessment with a view to help you to further understand this concept and improve your clinical competence in caring for patients with these conditions.

2.0 OBJECTIVE

At the end of this unit, you should be able to:

- assess patients with GIT disorders and
- manage patients with disorders of the gastrointestinal tract highlight the changes in the GIT due to ageing
- mention various modalities of GI assessment
- discuss the comprehensive subjective assessment of a patient with GI disturbances
- discuss the objective modalities of GI assessment
- explain nursing responsibilities associated with all the GI assessment modalities

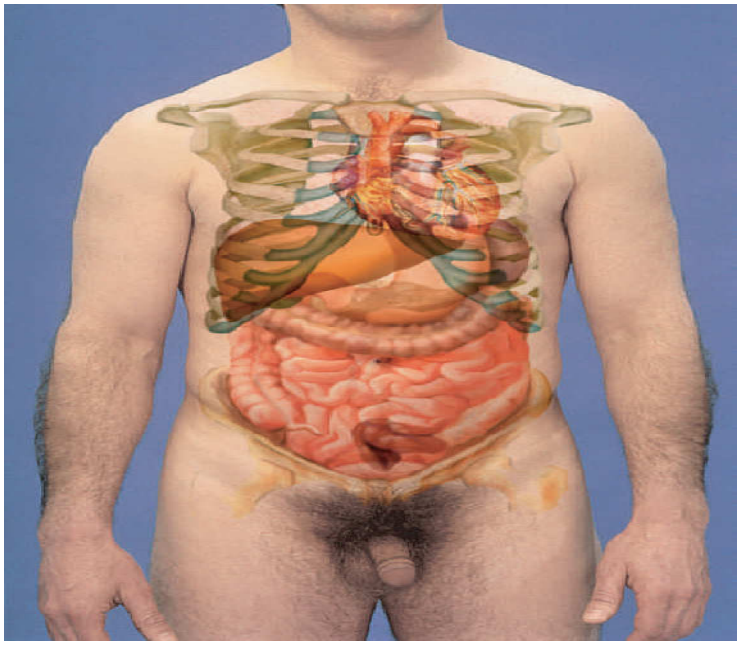


Image 1: the gastrointestinal system

3.1 Pathophysiologic and Psychological Considerations of Gastrointestinal (GI) disorders

- GI abnormalities are numerous and may include;
  - Bleeding
  - Perforation
  - Obstruction
  - Inflammation and
  - Neoplastic (cancers).
- These conditions may include;
  - Congenital
  - Inflammatory
  - Infectious
  - Traumatic and
  - Neoplastic lesions
  - Stress and anxiety
  - Circulatory disturbances
  - Faulty nervous system control
  - Aging.
- Common manifestations associated include:
  - Indigestion
  - Anorexia
  - Motor disturbances of the intestines (constipation or diarrhea)
  - Pains.

3.2 Gerontologic Considerations: Age-Related Changes of the Gastrointestinal System

Oral Cavity and Pharynx

- Injury/loss or decay of teeth
- Atrophy of taste buds
- Decreased saliva production
- Reduced ptyalin and amylase in saliva
- The results are difficulty in mastication and dysphagia.

Esophagus

- Decreased motility and emptying
- Weakened gag reflex
- Decreased resting pressure of lower esophageal sphincter
- Results in reflux and heartburn.

Stomach

- Degeneration and atrophy of gastric mucosal surfaces with decreased production of HCl
- Decreased secretion of gastric acids and most digestive enzymes
- Decreased motility and emptying
- Results include:
  - Decreased gastric motility
  - Delayed gastric emptying
  - Decrease in HCl production
  - Food intolerances and malabsorption
  - Decrease in vitamin B12 absorption (pernicious anemia).

Small Intestine

- Atrophy of muscle and mucosal surfaces
- Thinning of villi and epithelial cells
- Results in food intolerances and malabsorption.

Large Intestine

- Decrease in mucus secretion
- Decrease in elasticity of rectal wall
- Decreased tone of internal anal sphincter
- Slower and duller nerve impulses in rectal area
- Results in
  - Complaints of indigestion, constipation or fecal incontinence
  - Decreased absorption of nutrients (dextrose, fats, calcium, and iron).

3.3 Assessment

3.3.1 Health History and Clinical Manifestations

The initial and vital duty of the nurse is taking a comprehensive health history, with particular emphasis on complaints associated with GI dysfunction for instance;

- Pain; should include its character, duration, pattern, frequency, location, distribution of referred pain and any aggravating or relieving factors, such as meals, rest, medications. Note that, the time of the pain vary based on the underlying cause
- Indigestion (pyrosis); This is upper abdominal discomfort or distress associated with eating which often is due to gastric peristaltic movements. Indigestion can result from;

- Disturbed nervous system control of the stomach or
- GI tract disorders
- Systemic disorders

- Fatty foods, coarse vegetables and highly seasoned foods
- Intestinal gas accumulation may cause belching (the expulsion of gas from the stomach through the mouth) or flatulence (the expulsion of gas from the rectum). It often manifests as bloating and distention. Excessive flatulence may indicate gallbladder disease or food intolerance.

- Nausea and vomiting; Vomiting is another major symptom of GI disease. Vomiting is usually preceded by nausea, and may be triggered by odors, activity or food intake. The vomitus (emesis) may vary in color and content and may contain undigested food particles or blood (hematemesis). Fresh hemorrhage gives a bright red-coloured emesis while stalled blood takes on a coffee-ground appearance
- Alteration in bowel habits; may signal colon disease. It includes two extremes;
  - Diarrhea (an abnormal increase in the frequency and liquidity of the stool or in daily stool weight or volume) due to incomplete digestion and absorption of food particles from the gut. It is associated with abdominal pain or cramping and nausea or vomiting.
  - Constipation; a decrease in the frequency of stool; presents hard, dry stools which are smaller in volume and may be associated with anal discomfort and rectal bleeding.
- Alterations in stool characteristics; Normal stool is light to dark brown. However, conditions such as ingestion of certain foods, medications, blood (upper GI bleeding presents a tarry-black color – ***malena*** while lower GI bleeding presents a bright or dark red stool ***(hematochexia)***. Blood streaked stool is a hallmark of lower rectal or anal bleeding

Other common abnormalities in stool characteristics may include the following:

- Bulky, greasy, foamy stools that are foul in odor; stool color is gray, with a silvery sheen often due to fat indigestion
- Light gray or clay-colored stool, caused by the absence of urobilin
- Stool may contain mucus threads or pus
- Small, dry, rock-hard masses called scybala; sometimes streaked with blood due to rectal trauma during defecation
- Loose, watery stool that may or may not be streaked with blood

Moreover, History of any previous GI disease is vital.
Also, worthy of note are past and current medication use and any previous treatment or surgery.

Furthermore, a dietary history is needed to assess nutritional status: such as the use of tobacco and alcohol (facts about the type and amount is often necessary).
Other important issues will include:

- Specific changes in appetite or eating patterns
- Unexplained weight gain or loss in the recent times.
- Issues about psychosocial, spiritual, or cultural factors that may be affecting the patient.

3.3.2 Physical Assessment

The physical examination includes assessment of the;

- Mouth, tongue, buccal mucosa, teeth, and gums are inspected, and ulcers, nodules, swelling, discoloration, inflammation and dentures (remove during examination to allow good visualization.
- Abdominal examination: The patient assumes supine position with knees flexed slightly. Abdominal examination include inspection, auscultation, palpation, and percussion
  - ***Inspection*** is done first, noting skin changes, previous surgical scars, contour and symmetry of the abdomen, to identify any localized bulging, distention, or peristaltic waves.
  - ***Auscultation*** should be done before percussion and palpation; it will require listening to the character, location, and frequency of bowel sounds. Bowel sounds should be assessed in all the four quadrants of the abdomen with the diaphragm of the stethoscope; it is described as a high-pitched and gurgling sounds. The nurse should document its frequency as *normal* (1 sound per every 5 to 20 seconds), *hypoactive* (one or two sounds in 2 minutes), *hyperactive* (5 to 6 sounds heard in less than 30 seconds), or *absent* (no sounds in 3 to 5 minutes).
  - ***Percussion*** affords the nurse to observe sound notes such as tympany or dullness.
  - ***Palpation***; areas of tenderness or swelling (with light palpation); masses (with deep palpation), area of discomfort or rebound tenderness
- Rectal examination:
  - Inspection of the anal and perineal area; for areas of excoriation, rash, fissures or fistula openings, or external hemorrhoids
  - A digital rectal examination; for any areas of tenderness or mass
- Diagnostic Evaluation
  - Hematological work-up: may include;
    - Complete blood count (CBC),
    - Carcinoembryonic antigen (CEA),
    - Liver function tests,
    - Serum cholesterol and triglycerides.

The aim is to reveal alterations in basal metabolic function and perhaps, indicate the severity of a disorder.
Many other GI diagnostic modalities are available and can be done on an outpatient basis in specialized units (e.g., endoscopy or GI laboratory).

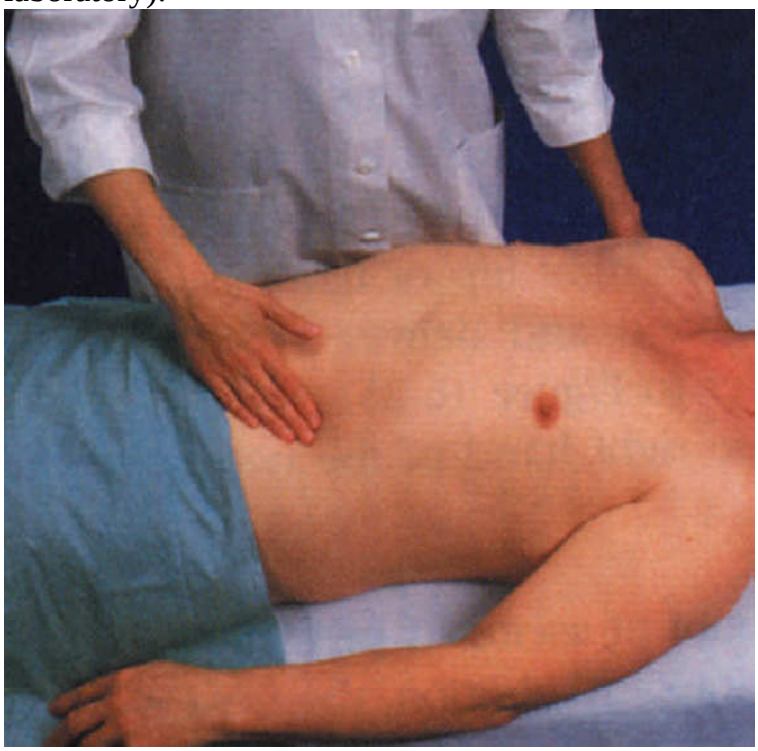


Photo 2: Abdominal palpation

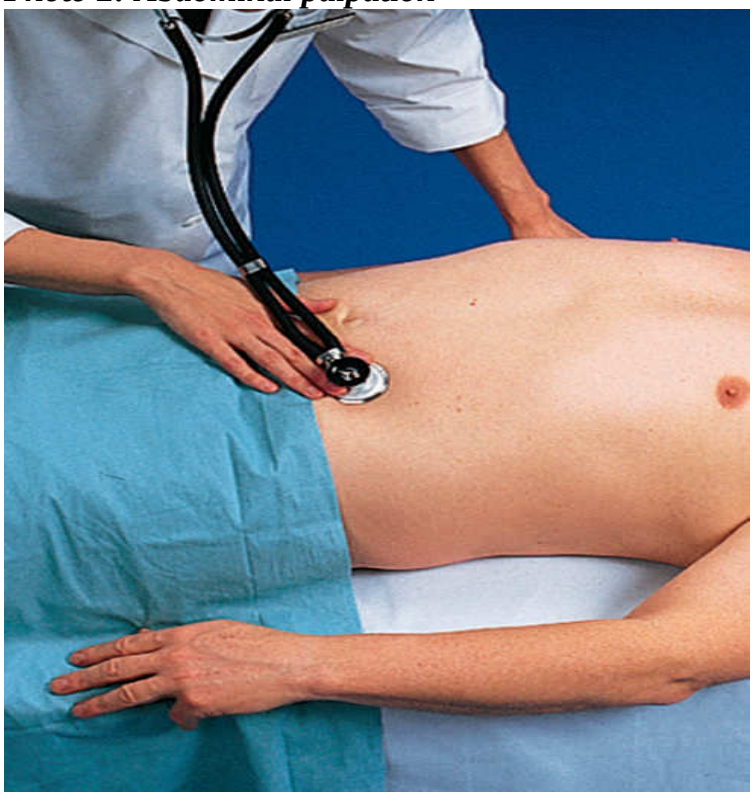


Photo 3: Abdominal Auscultation

3.3.3 General nursing responsibilities for a patient undergoing GI assessment

- The nurse supports and educates patients who are undergoing diagnostic evaluation
- Preparation for many of these studies may include fasting, the use of laxatives or enemas, and ingestion or injection of a contrast agent or a radiopaque dye.
- Further assessment (or possible treatment) for electrolyte imbalances may be needed after the procedure
- Providing general information about a healthy diet and the nutritional factors that can cause GI disturbances; after a diagnosis has been confirmed, the nurse provides information about specific nutrients that should be included in the diet
- Providing needed information about the test and the activities required of the patient
- Providing instructions about post-procedure care and activity restrictions
- Alleviating anxiety
- Helping the patient cope with discomfort
- Encouraging family members or others to offer emotional support to the patient during the diagnostic testing
- Assessing for adequate hydration before, during, and immediately after the procedure, and providing education about maintenance of hydration

3.3.4 Objective assessment of GI function

Stool Tests

Basic examination of the stool; includes;

- Inspecting the specimen for consistency and color and
- Testing for occult (not visible) blood; useful in initial screening for several disorders and cancer screening programs. Occult blood tests may be done using Hematest II SENSA and Hemo Quant. Immunologic tests such as Hemoporphyrin assays, Immunochemical tests using anti-human antibodies.
- Special tests, including tests for fecal urobilinogen, fat, nitrogen, parasites, pathogens, food residues, and other substances done in the laboratory.

3.3.5 Breath Tests

- The *hydrogen breath test;* to evaluate carbohydrate absorption, to aid the diagnosis of bacterial overgrowth in the intestine, and short bowel syndrome. The test determines the amount of hydrogen expelled in the breath after it has been produced in the

colon (by galactose fermenting bacteria) and absorbed into the blood.

- *Urea breath tests* detect *Helicobacter pylori.* The bacterium has been implicated in peptic ulcer disease. The patient takes a capsule of carbon-labeled urea and then provides a breath sample 10 to 20 minutes later. *H. pylori* metabolizes urea rapidly, the labeled carbon is absorbed quickly; it can then be measured as carbon dioxide in the expired breath to determine whether *H. pylori* is present.

3.3.6 D.N.A. Testing

This enables genetic risk assessment, pre-clinical diagnosis and pre-natal diagnosis of certain GI disorders such as gastric cancer, lactose deficiency, inflammatory bowel disease, colon cancer.
Thus leaving room to prevent (or minimize) disease, intervening before the onset of the active disease and/or improve therapy.

3.3.7 Imaging Studies

Imaging studies include x-ray and contrast studies, computed tomography (CT) scans, magnetic resonance imaging (MRI), and scintigraphy (radionuclide imaging).

Abdominal Ultrasonography

- Ultrasonography is a noninvasive diagnostic technique that uses a high-frequency sound wave and ultrasonic echoes to outline tissues of different densities.
- Abdominal Ultrasonography can highlight an image of the abdominal organs and structures on the oscilloscope. This procedure generally can indicate the size and configuration of abdominal structures and diagnosing inflammations such as cholelithiasis, cholecystitis, appendicitis, acute colonic diverticulitis.
- It requires no ionizing radiation, hence no noticeable side effects, and it is relatively inexpensive.
- Ultrasonographic waves cannot penetrate bone tissue, Gas and fluid collections in the abdomen or pleural structures
- Endoscopic ultrasonography (EUS); an adapted technique for better GI tissue definition, hence can be used in staging of a tumor, (based on size & extent of spread) and even evaluating trans-mural bowel wall changes in ulcerative colitis
- This uses a specialized enteroscopic procedure to directly visualize a target GI area with the aid of a small high-frequency ultrasonic transducer mounted on a fiber-optic scope even in

presence of intestinal gas, bone, and thick layers of adipose tissue.

Nursing Interventions

- Fast the patient for 8 to 12 hours before the procedure to decrease the amount of intestinal gas.
- Avoiding a fatty diet 24 hours before gallbladder studies.
- Schedule ultra sonographic investigations before barium studies as barium will interfere with the transmission of the sound waves.

Upper Gastrointestinal Tract Study

X-rays best outlines the entire GI tract when used with a contrast agent (a radiopaque liquid) such as barium sulfate, which the patient ingests before the X ray: Barium sulfate is tasteless, odorless, non-granular and completely insoluble in water hence, not absorbable

The upper GI series reveals facts about anatomic or functional integrity of upper GI structures (organs or sphincters) hence useful in diagnosing ulcers, varices, tumors, regional enteritis and malabsorption syndromes.

Upper GI investigation involves barium ingestion under direct fluoroscopic examination to visualize the position, patency and caliber of the esophagus; stomach's motility, wall thickness, mucosal pattern, pyloric valve's patency and anatomy of the duodenum; gastric emptying rate through multiple x-ray films at intervals for up to 24 hours; small bowel's motility, obstructions, ileitis and diverticula.

Nursing Interventions

- Maintain a low-residue diet for several days before the test.
- Nothing by mouth after midnight before the test.
- Laxative to clean out the intestinal tract.
- Avoid smoking on the morning before the examination to avoid stimulating gastric motility
- Withholds all medications.
- Follow-up care is needed after any of the upper GI procedures to ensure complete elimination of the ingested barium.
- Increase fluids to facilitate evacuation of stool and barium.
- Monitor stools until they return to their normal color (the barium will look like clay).
- Use a laxative or enema prn.

Lower Gastrointestinal Tract Study

In this case, a barium enema is required to visualize the lower GI tract. This will detect polyps, tumors and other large bowel lesions and to demonstrate any bowel malfunction.

Barium enema is instilled in the radiology department during fluoroscopy.
The procedure usually takes about 15 to 30 minutes, during which time x-ray images are taken.
Nursing Interventions

- Empty and cleanse the lower bowel with a low-residue diet 1 to 2 days before the test; a clear liquid diet and a laxative the evening before;
- Nothing by mouth after midnight;
- Cleansing enemas until returns are clear the following morning.
- Schedule barium enemas before any upper GI studies.
- Avoid enema in active inflammatory disease of the colon, evidences of perforation or obstruction, active GI bleeding
- Administers an enema or laxative after the procedure to facilitate barium removal.
- Increase fluid intake to assist eliminates the barium.
- Monitors the patient for complete elimination of the barium

Computed Tomography
CT provides cross-sectional images of abdominal organs and structures. It involves taking multiple x-ray images from many different angles, digitize them in the computer, and re-construct them so that they can be viewed on a computer monitor.

Indications: for abdominal CT scanning are diseases of the liver, spleen, kidney, pancreas, and pelvic organs with a view to detecting and localizing many inflammatory conditions in the colon, such as appendicitis, diverticulitis, regional enteritis, and ulcerative colitis.
Nursing Interventions

- Nothing per oral for 6 to 8 hours before the test.
- Give intravenous or oral contrast agent.
- Question about contrast dye allergies.
- Schedule barium studies after CT scanning, so as not to interfere with imaging.

Magnetic Resonance Imaging
MRI is used in gastroenterology to supplement Ultrasonography and CT scanning. It is a noninvasive technique that uses magnetic fields and radio waves to produce an image of the area being studied. Physiologic artifacts of heartbeat, respiration, and peristalsis may create a less-than-clear image.

It is useful to evaluate abdominal soft tissues as well as blood vessels, abscesses, fistulas, neoplasms, and other sources of bleeding.

Contraindications; MRI is contraindicated for patients with;

- Permanent pacemakers
- Artificial heart valves
- Defibrillators
- Implanted insulin pumps
- Implanted transcutaneous electrical nerve stimulation devices
- Internal metal devices (e.g., aneurysm clips)
- Intraocular metallic fragments.

This is because the magnetic field could cause malfunction.
Nursing Interventions

- Nothing per oral for 6 to 8 hours before the test.
- Remove all jewelry and other metals before the test.
- Warn patients about feelings of claustrophobia
- Prepare patient that the machine will make a knocking sound during the procedure.

Scintigraphy
Scintigraphy is a radionuclide testing

It relies on the use of radioactive isotopes (i.e., technetium, iodine, and indium)
Purposes of Scintigraphic scanning:

- It can be used to outline anatomic disruptions in tissue structures, as with neoplasms or other focal lesions, such as cysts or abscesses and detect changes in organ size.
- It can also define areas of inflammatory conditions by measuring the degree of uptake of stained erythrocytes and leukocytes with a view to diagnosing abscess, blood loss, or neoplasm. This is by staining a withdrawn sample of blood with a radioactive substance and subsequently re-injected into the patient so that abnormal concentrations of blood cells can then be detected at sites of inflammation within 24 and 48 hours.
- Radionuclide testing also is used to assess gastric emptying and colonic transit time. This involves staining meal components with radionuclide markers and their movements in the GIT can be tracked with a scintiscanner following ingestion by the patient. This works essentially for gastric emptying studies. This procedure is thus helpful for evaluating any functional derangement associated with gastric emptying, such as common clinical conditions as diabetic gastroparesis and dumping syndrome which delay or increase gastric transit time respectively.

- This can also evaluate colonic motility to rule out functional derangement such as obstruction. This study involves administration of a capsule containing 20 radionuclide markers and followed by a regular diet and normal daily activities. The sequel is a series of abdominal x-rays taken every 24 hours until all markers are passed (usually over the next 4 to 5 days). Severe constipation may present as long as 10 days while diarrhea presents a short transit time. In essence, the amount of time it takes for the radioactive material to move through the colon indicates colonic motility.

Endoscopic Procedures

Endoscopic procedures available for GIT assessment include;

- Fibroscopy/esophagogastroduodenoscopy
- Anoscopy
- Proctoscopy
- Sigmoidoscopy
- Colonoscopy
- Small-bowel enteroscopy and
- Endoscopy through ostomy.

Upper Gastrointestinal Fibroscopy/Esophagogastroduodenoscopy

This procedure uses a fiberscope, which are flexible tubes, equipped with fiberoptic lenses. With which a direct visualization of the upper GIT structures is possible.

- ***Gastroscopy or esophagogastroduodenoscopy (EGD);*** describes direct visualization of the esophageal, gastric, or duodenal abnormalities such as inflammatory, neoplastic, or infectious processes. It can also evaluate esophageal and gastric motility and to collect secretions and tissue specimens for further analysis.
- ***Electronic video endoscopes;*** has a video processor, that will convert the electronic signals into pictures on a television screen thus allowing a larger and continuous viewing capabilities, as well as the simultaneous recording of the procedure.
- ***Endoscopic retrograde cholangiopancreatography (ERCP);*** uses a side-viewing flexible scope to visualize the common bile duct and the pancreatic and hepatic ducts through the ampulla of Vater in the duodenum. It is helpful in evaluating jaundice, pancreatitis, pancreatic tumors, common duct stones, and biliary tract disease.

Indications;

- Diagnostic purpose

- Therapeutic dimension when combined with other procedures and thus can be used to;
  - Remove common bile duct stones,
  - Dilate strictures, and
  - Treat gastric bleeding and esophageal varices by introducing a sclerosing solution into the leisions.
  - Treating upper GI neoplasms with laser therapy.

Procedure;

- It is done by the gastroenterologist
- Sedate patient
- Lubricate the endoscope with a water-soluble lubricant
- Pass the lubricated endoscope smoothly and slowly along the back of the mouth and down into the esophagus and view the gastric wall and the sphincters
- Then advance into the duodenum for further examination.
- Obtain tissue specimens (cells) with biopsy forceps or cytology brushes which can be passed through the scope.
- The procedure usually takes about 30 minutes.

Nursing Interventions

- Before the procedure;
  - Place patient on NPO 6 to 12 hours before the examination
  - Administer a local anesthetic spray or gargle
  - Administer intravenous sedative e.g. midazolam just before the scope is introduced.
  - Administer atropine to reduce secretions
  - Also glucagon may be necessary to relax smooth muscle.
  - Position the patient on the left side to facilitate saliva drainage and to provide easy access for the endoscope.
  - Support patient as he may experience nausea, gagging or choking.
- After the procedure;
  - Maintain NPO until the gag reflex returns (usually, 1 to 2 hours) to prevent aspiration of food or fluids into the lungs.
  - Places the patient in the Simms position until full consciousness is regained, after which he can be placed in the semi-Fowler's position until ready for discharge.
  - Assess for signs of perforation, such as pain, bleeding, unusual difficulty swallowing, and an elevated temperature
  - Monitor the pulse and blood pressure for changes that can occur with sedation.
  - Monitor and maintain the oral airway during and after the procedure.
  - Monitor oxygen saturation with oximeters

- Administer supplemental oxygen as required
- Make ready emergency equipment.
- Test the gag reflex, by placing a tongue blade onto the back of the throat to see whether gagging occurs.
- As soon as the gag reflex has returned, offer lozenges, saline gargle, and oral analgesics to relieve minor throat discomfort.
- Sedated patients need to stay on bed rest until fully alert.
- Protect the scope, as the fiberoptic bundles can break if the scope is bent at an acute angle.
- Ensure the patient wears a mouth guard to keep from biting the scope.

Anoscopy, Proctoscopy, and Sigmoidoscopy

These procedures provide direct visualization of the lower portion of the colon.

They can be used to evaluate rectal bleeding, acute or chronic diarrhea, or change in bowel patterns and to observe for ulceration, fissures, abscesses, tumors, polyps, or other pathologic processes.
Types of scopes;

It uses a rigid or flexible fiberoptic scopes.

- ***Rigid scopes:***
- ***Anoscope:*** used to examine the anus and lower rectum.
- ***Proctoscope:*** inspect the rectum and
- ***Sigmoidoscope:*** inspect the sigmoid colon.

These procedures requires the patient to assume a knee-chest position at the edge of the examining table, with the back inclined at about a 45-degree angle

- ***Flexible scopes:*** have largely replaced the rigid scopes for routine examinations.
- *Flexible fiberopticsigmoidoscope:* permits examination of the colon up to 40 to 50 cm from the anus up into the descending colon. It can also use a still or video images to document findings. The procedure is called *flexible fiberopticsigmoidoscopy*

The procedure presents an urge to have a bowel movement.
It is better done in the left lateral position with the right leg bent and placed anteriorly.

The procedure also affords for biopsies and polypectomies (i.e. removal of polyps for laboratory analysis)
Nursing Interventions
The procedure requires;

- Limited bowel preparation, such as bowel clearing enema
- Dietary restrictions may be used
- Sedation usually is not required.

During the procedure;
- Monitor the following;
  - Vital signs
  - Skin color and temperature
  - Pain tolerance and
  - Vagal response

After the procedure;
- Monitor the following;
  - Evidences of rectal bleeding
  - Signs of intestinal perforation (i.e., fever, rectal drainage, abdominal distention, and pain).
- Encourage patient to resume regular activities and dietary practices as soon as possible.

Fiber optic Colonoscopy

Direct visual inspection of the colon to the cecum
This is through a flexible fiberopticcolonoscope
The procedure produces a larger diameter and longer still and video recordings *Purposes:*

Commonly used for both diagnostic and screening means such as for;
- Cancer screening
- Early detection and prevention of colorectal cancer
- Obtaining tissue biopsies
- Removal of polyps
- Evaluation of diarrhea of unknown cause, occult bleeding, or anemia
- Further study of abnormalities detected on barium enema
- Diagnosis, clarification, and determination of the extent of inflammatory or other bowel disease
- Treat areas of bleeding or stricture through the use of bipolar and unipolar coagulators, heater probes, and injections of sclerosing agents or vasoconstrictors
- Bowel decompression

Procedure;
- Position for the procedure is left lateral with the legs drawn up toward the chest with positional changes to facilitate advancement of the scope.
- The procedure usually takes about 1 hour.
- bowel preparation

- Instill air to expand the colon
- Insert the scope.

Potential complications include;

- Cardiac dysrhythmias and
- Respiratory depression
- These complications are due to;
- The medications administered
- Vasovagal reactions and
- Circulatory overload or hypotension resulting from over-hydration or under-hydration during

Nursing Interventions

- Bowel preparation: for optimal visualization; Cleansing may be by means of;
  - Laxatives for two nights before the examination
  - Saline enema until clear return
  - Intestinal lavages with polyethylene glycol electrolyte lavage solutions; with side effects such as nausea, bloating, cramps or abdominal fullness, fluid and electrolyte imbalance and hypothermia; its use is contraindicated in intestinal obstruction or inflammatory bowel disease.
- Routine medications; debilitated patients, elderly and diabetics will require medication adjustments
- Maintain adequate fluid, electrolyte, and caloric intake
- Special considerations; the procedure is contraindicated in cases, such as those with;
  - Implantable defibrillators and pacemakers; due to high risk for malfunction associated with electrosurgical procedures (i.e., polypectomy)
  - Colon perforation
  - Acute severe diverticulitis
  - Fulminant colitis
  - Coagulopathies and Anti-coagulation therapy (due to a high risk for excessive bleeding, associated with therapeutic procedures such as polypectomy)
- Discontinue non-steroidal anti-inflammatory agents (NSAIDs) such as aspirin, before the test and 2 weeks after the procedure.
- Informed consent is obtained before the test.
- Ensure nothing by mouth (NPO) after midnight before the test
- Intravenous opioid analgesic or a sedative (e.g., midazolam) may be required before the procedure
- Glucagon may be needed to relax the colonic musculature and to reduce spasm during the test.
- Monitor changes in;

- Oxygen saturation
- Vital signs
- Skin color and temperature
- Level of consciousness
- Abdominal distention
- Vagal response and
- Pain intensity.

After the procedure;

- Maintain sedated cases on bed rest until fully alert.
- Observe for signs and symptoms of bowel perforation (e.g., rectal bleeding, abdominal pain or distention, fever, focal peritoneal signs)
- Explain the amnesic effect of midazolam and reassure the patient
- During this period provide written instructions for easy recall.
- After a therapeutic procedure, instruct the patient to report any bleeding to the physician.

Small-Bowel Enteroscopy

The procedure requires the small-caliber trans-nasal endoscope
It allow direct inspection of the wall of the small intestine
The procedure is done using the "push" and the "pull" endoscope methods.

- The "pull" endoscope is very long and flexible and has a balloon at its tip which can be inflated and advanced through the small intestine. Actualizing this may take up to 10 hours, during which the patient may be in the recovery area or sent home. The balloon can then be deflated once the scope has entered the distal ileum and the tube retracted slowly while the endoscopist examines the intestinal wall.
- The "Push" endoscopes are smaller in caliber and longer.

Indications; These two methods have been reserved for;

- Evaluation of intractable bleeding
- Diagnosis of malabsorption syndromes.

Endoscopy through Ostomy

Endoscopy is possible through percutaneous ostomies with the aid of a flexible endoscope. It affords the visualization of segments of the small or large bowels to evaluate and/ or treat;

- An anastomosis
- Recurrent disease
- A bleeding segment

Nursing responsibilities for other endoscopic procedures suffice.

Laparoscopy (Peritoneoscopy)

- This is percutaneous visualization of the abdominal organs and structures.
- This procedure is done through a small incision in the abdominal wall.
- Then, a special fiberoptic laparoscope is thread through the opening to allow direct visualization and identification of growths, anomalies, and inflammatory processes, taking of tissue specimens and thus evaluate peritoneal disease, chronic abdominal pain, abdominal masses, and gallbladder and liver disease.
- Its use has been limited by less invasive modalities such as CT and MRI.
- The procedure requires;
  - General anesthesia
  - Sometimes stomach and bowel decompression
  - Gas (usually carbon dioxide) insufflations into the peritoneal cavity for easy visualization.
- The procedure affords for excision of diseased portions of an organ.

Manometry and Electrophysiologic Studies

These are methods for evaluating GI motility.

The manometry test measures changes in intra-luminal pressures and smooth muscle coordination.

The pressures can be recorded through various means such as;

- Manual recordings
- A physiograph or
- A computer.

Forms of manometry studies:

Esophageal manometry;

This evaluates disorders of the esophagus and the lower esophageal sphincter.

Preparations may include;

- Restricting eating or drinking for 8 to 12 hours before the test
- Withhold medications directly affect motility such as calcium channel blockers, anticholinergics and sedatives for 24 to 48 hours.

Procedure:

- Insert a pressure-sensitive catheter through the nose

- Connect the catheter to a transducer and a video recorder.
- Allow the patient swallow small amounts of water
- Record the resultant pressure changes are recorded.

Gastroduodenal, small-intestine, and colonic manometry;

- Evaluate gastric emptying and gastric and intestinal motility
- It thus can diagnose disorders such as irritable bowel syndrome or atonic colon.
- This is often an ambulatory outpatient procedure and may last up to 24 to 72 hours.

Anorectalmanometry;

- Measures the resting tone of the internal anal sphincter and the contractibility of the external anal sphincter
- It thus can help evaluating chronic constipation or fecal incontinence and treatment of fecal incontinence.
- It can be done together with rectal sensory functioning tests.
- Preparation will require;
  - Phospho-Soda or saline cleansing enema 1 hour before the test
  - Positioning; either prone or lateral position.

A rectal sensory function test;

Evaluates rectal sensory function and neuropathy
Procedure;

- Pass a catheter and balloon into the rectum
- Inflate the balloon until the patient feels distention.
- Measure the tone and pressure of the rectum
- Also measure anal sphincteric tone.

Clinical implications; help evaluate

- Chronic constipation
- Chronic diarrhea or
- Incontinence.

Electrogastrography and electrophysiologic study;

- Assess gastric motility disturbances.
- It involves placing electrodes over the abdomen and record the gastric electrical activity for up to 24 hours.
- Possible findings may range from rapid to slow or irregular waveform activity.
- Clinical implications; detecting gastric motor or nerve dysfunction.

Defecography;

- Defecography measures anorectal function.
- Procedure:
  - It involves instilling a very thick barium paste into the rectum
  - Encourage patient to expel the barium
  - Then perform a fluoroscopy to assess the rectal and anal sphincter function
- The test requires no specific preparation.

Gastric Analysis, Gastric Acid Stimulation Test, and pH Monitoring

Analysis of the gastric juice provides insight about;

- The secretory activity of the gastric mucosa
- Presence and degree of gastric retention

May be useful for diagnosing diseases such as Zollinger-Ellison syndrome

Preparation:

- NPO for 8 to 12 hours before the procedure
- Discontinue any medications that affect gastric secretions 24 to 48 hours before the test. Discourage smoking on the morning before the test as it will increases gastric secretions

Procedure:

- Insert a small nasogastric tube (with a catheter tip marked at various points) through the nose
- Insert the tube to a point slightly less than 50 cm (it should be lying along the greater curvature within the stomach at this point)
- Secure the tube to the patient's cheek
- Place the patient in a semi-reclining position.
- Aspirate the entire stomach contents by gentle suction through a syringe
- Collect gastric samples every 15 minutes for the next hour.
- Some patients may require gastric acid stimulation, with subcutaneous injection of histamine or pentagastrin (Prepare the patient for associated flushed feeling).
- Monitor blood pressure and pulse frequently to detect hypotension.
- Collect gastric specimens after the injection every 15 minutes for 1 hour
- Label specimens to indicate the time of specimen collection after histamine injection.
- Measure the volume and pH of collected specimen.

Analysis indicated may include;

- Cytologic study by the Papanicolaou technique, to determine the presence of malignant cells
- Enzyme analysis of the gastric juice

Clinical implications;
- Important diagnostic information includes the secretory ability of the gastric mucosa, which often is altered in various disease states such as;
- Pernicious anemia; associated with no acid secretion under basal conditions or after stimulation
- Severe chronic atrophic gastritis or gastric cancer, with little or no acid secretion
- Peptic ulcer, with some acid secretions
- Duodenal ulcers, usually secrete an excess amount of acid

Ambulatory pH monitoring:
- Often diagnose esophageal reflux of gastric acid

Preparations:
- NPO for 6 hours before the test
- Stop all medications affecting gastric secretions 24 to 36 hours before the test.

Procedure;
- The procedure uses a probe that measures pH
- Insert a probe through the nose into position about 5 inches above the lower esophageal sphincter
- Connect the probe to an external recording device
- The patient wears this apparatus for 24 hours
- The patient should continue the usual daily activities
- The end result is a computer analysis and graphic display of the results

4.0 SUMMARY

At this juncture, you are expected to have learnt about:
- highlight the effects of ageing on the GIT
- mention various modalities of GI assessment
- discuss the comprehensive subjective assessment of a patient with GI disturbances
- discuss the objective modalities of GI assessment
- explain nursing responsibilities associated with all the GI assessment modalities

Activities

Now that you have learnt this much, visit a hospital of choice within your vicinity and manage a patient undergoing any form of the diagnostic modality discussed within the content of this discuss. Share your answers with your colleague in the discussion forum

SELF-ASSESSMENT EXERCISE

i. Mention the influence of ageing on the structures of the GIT
ii. Discuss the content of a comprehensive history required of a patient with GI complaints
iii. Write short note on the following, with particular emphasis on the associated nursing responsibilities;
    a. Endoscopy
    b. Imaging studies
    c. Gastric juice analysis
    d. Manometry and Electrophysiologic Studies

You can compare your answers with the content of the course and reference materials. Endeavour to share your answer with your colleagues on the discussion platform.

5.0 REFERENCES/FURTHER READING

Levey, R., Williams-Wilson, B. (2002).Anorexia Nervosa: eMedicine Journal, April 2 2002, Volume 3, Number 4.

Smeltzer, S.C., Brenda, B. (2006). Brunner and Suddhart's Textbook of Medical-Surgical Nursing, 10th edition. Lippincott-William & Wilkins.

Templer, J. W. (2001). Parotitis; eMedicine journal; July, 2001, Vol. 2, Number, 7.

UNIT 2 CARING FOR PATIENT WITH NUTRITIONAL AND ORAL CAVITY DISORDERS

CONTENTS

1.0 INTRODUCTION

The focus of this unit is on exploring various disorders of nutrition and the diseases of the oral cavity (the teeth and the salivary glands), with a view to improve your knowledge base and sharpen your clinical competence in caring for patients with these conditions.

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- describe various disorders of nutrition
- discuss the disturbances of the structures and functions of the oral cavity
- identify the etiologic factors of nutritional and the Oral Cavity disorders
- explain the Pathophysiology of nutritional and the Oral Cavity disorders
- adopt the nursing process approach to provide care for patients with disorders of nutrition and oral cavity.

3.0 MAIN CONTENT

3.1 Over-nutrition (Obesity)

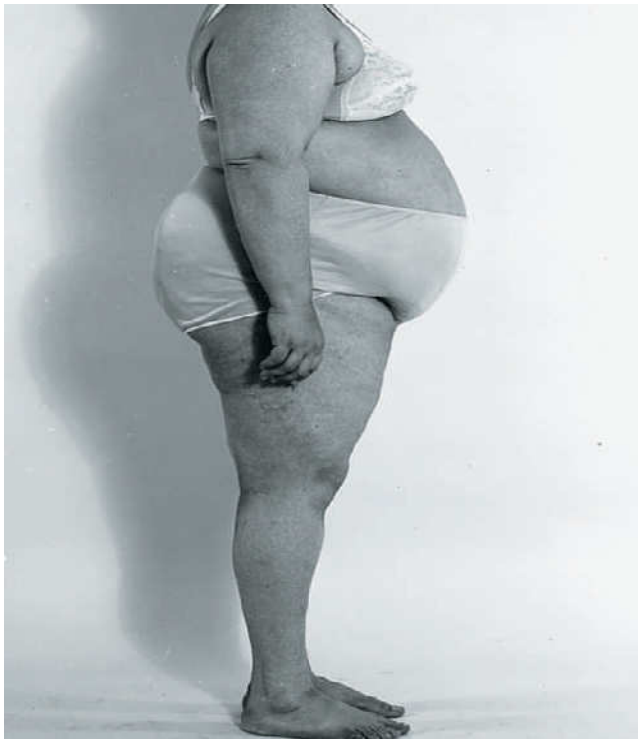


Photo 4: Obesity

Malnutrition simply is abnormal nutrition, which means, intake of food nutrients in abnormal concentration.

This concept refers to both over-nutrition and under-nutrition.
Over-nutrition refers to intake of nutrients far above the body required concentration while under-nutrition defines intake of nutrients far below what the body requires.

Mal-nutrition affects all classes of food nutrients most importantly the macro-nutrients- amino-acids (proteins), glucose (carbohydrates) and lipids (fats).

Obesity represents a state of excess storage of body fat, greater than 25% in men and over 33% for women.

Types of obesity

- Android type; parades a high abdominal (visceral) fat and lesser subcutaneous abdominal fat.
- Gynecoid type; parades abundant peripheral distribution especially in the gluteal region.

Classification of obesity

For adults, the World Health Organization (WHO) classification is based on Body Mass Index (BMI). It includes:

- Grade 1 overweight (overweight): BMI of 25-29.9 kg/m^2
- Grade 2 overweight (obesity): BMI of 30-39.9 kg/m^2
- Grade 3 overweight (severe or morbid obesity): BMI greater than or equal to 40 kg/m^2

The body mass index (BMI) = Weight (kg)/Height^2 (meters) or
Weight (pounds) X 0.703/height (inches^2)

The surgical literature classification; particularly deals with severe obesity; the categories include:

- Severe obesity - BMI greater than 40 kg/m^2
- Morbid obesity - BMI of 40-50 kg/m^2
- Super obese - BMI greater than 50 kg/m^2

In children;

- BMIs greater than the 85th: Overweight percentile or
- BMIs greater than 95th (Obesity) percentile.

Associated risk includes:

- Cardio-metabolic syndrome
- Type 2 diabetes
- Hypertension
- Dyslipidemia
- Coronary heart disease
- Osteoarthritis
- Stroke
- Gall bladder disease
- Obstructive sleep apnea
- Gastro-esophageal reflux disease (GERD)
- Some cancers (endometrial, breast, and colon).

Causes;

Factors to be considered in the development of obesity include the following:

- Metabolic factors
- Genetic factors (single gene or multiple genes defects)
- level of activity
- Behavior
- Endocrine factors (Cushing's syndrome; hypothyroidism; hunger and satiety modulators- Melanocortin; gut hormones (glucagon-like peptide-1, neuropeptide YY, & cholecystokinin); Leptin; Pancreatic amylin; Ghrelin,)

- Race, sex, and age factors
- Ethnic and cultural factors
- Socioeconomic status
- Dietary habits
- Smoking cessation
- Pregnancy and menopause
- Psychologic factors
- History of gestational diabetes
- Lactation history in mothers.

Prevalence

The prevalence of obesity worldwide is increasing

Estimates suggest as many as 250 million people (approximately 7% of the estimated current world population) are obese while 2- 3 times more people are probably overweight (Hamdy et al, 2006).

Obesity affects all races and socioeconomic classes worldwide.

Age predilection; Children and particularly adolescents are becoming recently more affected.

Factors that modulate associated morbidity and mortality:

- Age of onset and duration of obesity
- Severity of obesity
- Amount of central adiposity
- Other co-morbidities
- Gender
- level of cardiorespiratory fitness
- Race.

Associated co-morbidities include:

- Respiratory: Obstructive sleep apnea, higher chances of respiratory infections, bronchial asthma and Pickwickian syndrome (obesity hypoventilation syndrome)
- Malignant: Endometrial, prostate, colon, breast, gall bladder, and lung cancers
- Psychologic: Social stigmatization and depression
- Cardiovascular: Coronary artery disease, essential hypertension, left ventricular hypertrophy, Corpulmonale, obesity-associated cardiomyopathy, accelerated atherosclerosis, and pulmonary hypertension of obesity
- Central nervous system (CNS): Stroke, idiopathic intracranial hypertension, and meralgiaparesthetica
- Obstetric and perinatal problems: Pregnancy-related hypertension, fetal macrosomia, and pelvic dystocia.

- Surgical: Increased surgical risk and postoperative complications, including wound infection, postoperative pneumonia, deep venous thrombosis, and pulmonary embolism
- Pelvic disturbances: Stress incontinence
- Gastrointestinal (GI) disturbances: Gall bladder disease (cholecystitis, cholelithiasis), non-alcoholic steatohepatitis (NASH), fatty liver infiltration, and reflux esophagitis.
- Orthopaedic problems: Osteoarthritis, coxavera, slipped capital femoral epiphyses, Blount disease and Legg-Calvé-Perthes disease, and chronic lumbago.
- Metabolic disturbances: Type 2 DM, insulin resistance, hyper-insulinemia and dyslipidemia (high total cholesterol, high triglycerides, low high-density lipoprotein, normal or elevated low-density lipoprotein).
- Reproductive problems: Anovulation, early puberty, infertility, hyper-androgenism and polycystic ovaries (in women), and hypogonadotropichypogonadism (in men).
- Cutaneous: Intertrigo (bacterial and/or fungal), acanthosisnigricans, hirsutism, and increased risk for cellulitis and carbuncles.
- Peripheral disorders: Venous varicosities, lower extremity venous and/or lymphatic edema.
- Musculoskeletal: Reduced mobility; ostheoathritis.

Management of obesity:
Weight Loss therapy; intended weight loss at approximately 1-2pounds/week. The ultimate aim of therapy
This can be attained by:

- Diet modification with Very–low-calorie diets (VLCDs); Balanced, low-calorie diets (or reduced portion sizes); Diets with different macronutrient compositions e.g. Low-fat diets (e.g., Ornish diet), low-carbohydrate diets (e.g., Atkins diet), and Fad diets (e.g., soup, single-food).
- Activities modification; proportionate increase of appropriate physical activity and non-strenuous exercise e.g. Aerobic isotonic exercise (of greater value) rather than the anaerobic isometric exercise (including resistance training).
- Reducing time spent in sedentary activities (e.g., watching television).
- Behavioral modification; involves use of inventory to identify and manage cues, circumstances, and practices that favor non-meal eating and snacking; Sufficient amount of sleep.

Medical management;
Sibutramine (Meridia); a central acting appetite suppressant: It acts by inhibiting noradrenalin, serotonin, and dopamine reuptake in the CNS.

Ephedrine and caffeine; they increase energy expenditure but are associated with the potential for tachycardia, hypertension, and palpitations.

Under-nutrition

Definition:

World Health Organization defines malnutrition as "the cellular imbalance between supply of nutrients and energy and the body's demand for them to ensure growth, maintenance, and specific functions."

Otherwise called Protein-energy malnutrition (PEM)

Observed most frequently in developing countries especially among hospitalized and chronically ill children in developed world

PEM has 2 forms;

- Kwashiorkor; fair-to-normal calorie with grossly inadequate protein intake; represents a maladaptation to starvation
- Marasmus; inadequate intake of protein and calories; an adaptation to starvation.

Pathophysiology:

Its effect is multi-systemic. Lack of dietary protein, energy and micronutrients is essential for tissue and enzyme synthesis and repairs necessary for all biochemical and physiologic functions.

Lack of these nutrients will result in impairment of physical, cognitive and physiological development and functions.

Notable physiological derangement affects changes within the immune system, which often indicate poor outcomes. These changes mimic those in acquired immune deficiency syndrome (AIDS).

These changes include; Loss of delayed hypersensitivity, fewer T lymphocytes, impaired lymphocyte response, impaired phagocytosis secondary to decreased complement and certain cytokines, and decreased secretory immunoglobulin A (IgA).

Frequency:

Affects mainly children under five years old: common in the developing countries notably, children in rural areas. It also affects hospitalized children more.

Mortality/Morbidity:

This is affected by;

- Impaired physical, cognitive and physiological developmental
- Impaired immunologic functions (immunosuppression)
- Predisposition to opportunistic and other typical childhood infections

- Behavioral changes such as irritability, apathy and decreased social responsiveness, anxiety and attention deficits
- Developmental delay or permanent cognitive deficits
- Death from malnutrition; more in the developing than in the developed countries.

Predisposing factors;

- Children in infancy and early childhood stage of development.
- Premature infants (due to their special nutritional needs and challenges with feeding)
- Self-imposed dietary restrictions (among adolescent)
- Socioeconomic factors like poverty, ignorance
- Maternal diseases like TB, HIV
- Childhood conditions like failure to thrive, congenital deformities
- Conflicts and social unrest
- Natural food scarcity like famine.

Clinical signs and symptoms: include:

- Poor weight gain
- Slowing of linear growth
- Behavioral changes - Irritability, apathy, decreased social responsiveness, anxiety, and attention deficits
- Micronutrient deficiencies:

o Iron deficiency; Fatigue, anemia, decreased cognitive function, headache, glossitis, and nail changes
o Iodine deficiency; Goiter, developmental delay and mental retardation
o Vitamin D deficiency; Poor growth, rickets, and hypocalcemia
o Vitamin A; Night blindness, xerophthalmia, poor growth, and hair changes
o Folate deficiency; Glossitis, megaloblastic anemia and neural tube defects

- **Physical findings;**

o Decreased subcutaneous tissue: especially in the legs, arms, buttocks, and face
o Edema: in distal extremities and anasarca [generalized edema].
o Oral changes; Cheilosis, Angular stomatitis, Papillar atrophy,
o Abdominal findings; Abdominal distension (poor abdominal musculature tone), Hepatomegaly (fatty infiltration),
o Skin changes; Dry peeling skin with raw exposed areas, Hyper-pigmented plaques over areas of trauma
o Nail changes: Nails become fissured or ridged.
o Hair changes: Hair is thin, sparse, brittle, easily pulled out, and turns a dull brown or reddish color.

Causes:

- Inadequate food intake; the most common cause; due to insufficient or inappropriate food supplies
- Inadequate sanitation; increases risk for infectious diseases
- Chronic illnesses; due to anorexia, increased inflammatory burden and high metabolic demands, impaired digestion and/or absorption from the GIT,

o Chronic illnesses that commonly are associated with nutritional deficiencies include:

- Cystic fibrosis
- Chronic renal failure
- Childhood malignancies
- Congenital heart disease
- Neuromuscular diseases
- Multiple food allergies (due to severe dietary restrictions).

Laboratory Studies:

- Hematological studies; complete blood count (CBC), Erythrocyte sedimentation rate (ESR)
- Protein status; serum albumin, retinol-binding protein, pre-albumin, transferrin, creatinine, and BUN
- Serum electrolytes
- Urinalysis and culture
- Stool analysis
- Thyroid functions tests
- Sweat chloride tests (to exclude cystic fibrosis)

Practical nutritional assessment; should include;

- Complete history and detailed dietary history
- Growth measurements including;

o Weight and height

o Head circumference in children younger than 3 years

- Complete physical examination
- Sensitive measures of nutritional status

o Height-for-age; Less than 5 cm/year of growth in children older than 2 years

o Weight-for-height measurements; less than 90% of expected value

o Body mass index (BMI).

Management:

- Immediate assessment of nutritional status
- Identification of the underlying etiology
- Dietary intervention planning with a dietitian or other nutritional professional

- Dietary planning should be done with careful consideration of patients economic status
- Caloric (Protein and energy) intakes more than 120-150 kcal/kg/day may be required
- Micronutrient deficiencies must be corrected at once
- Food source should utilize locally available and affordable food stuffs
- Hospitalization may be required based on severity and instability of the clinical situation.
- Detailed documentation of actual intake and feeding difficulties.
- Monitor patients closely for growth and resolution of clinical signs and symptoms of malnutrition.

Prevention:
- Promotion of breastfeeding
- Mothers should be thought appropriate weaning of their infant from breast-milk
- Appropriate and adequate food supplementation.
- Health care providers should continue to provide age-appropriate nutritional counseling at every opportunity.
- Programs addressing micronutrient supplementation and fortification such as (iodine, vitamin D, vitamin A) should be emphasized.
- The issue of immunization and vaccination is crucial
- Careful neonatal examination and care by qualified medical practitioner
- Ante-natal and delivery in a hospital, supervised by a qualified midwife is crucial
- Improvement in hygiene practices and sanitation.

3.3 Eating Disorders (Anorexia Nervosa)

Definition
Anorexia nervosa refers to an intense fear of gaining weight characterized by the individual's refusal to maintain minimally normal body weight. It is associated with a significant disturbance in body image perception (body shape or size).

It has been divided into bulimic and non-bulimic subtypes based on purging behaviors associated with binge eating.

Types
- The bulimic type; is associated with binge-eating followed by purging behaviors (like self-induced vomiting, or misuse of laxatives, diuretics, or enemas) to maintain weight. This is often

associated with problems with impulse control (e.g. substance use disorder, emotional lability, sexual activity).

- The non-bulimic type; also called "the restrictive type", parades weight loss primarily through dieting, fasting, or excessive exercise; but no regular binge-eating or purging behavior: patients are more likely to be more obsessional, socially awkward and more isolated.

Associated Features and Disorders May Include;

- Affective disorders; depressed mood, social withdrawal, irritability
- Insomnia
- Diminished interest in sex
- Obsessive-compulsive features
- Concerns about eating in public
- Feelings of ineffectiveness or a strong need to control one's environment
- Inflexible thinking
- Limited social spontaneity and
- Overly restrained initiative and emotional expression.

Epidemiology

- More in females in late adolescence and early adulthood
- Incidence rates have increased in recent years
- The disorder is far more prevalent in industrialized societies where food is abundant and thinness is a measure of feminine attractiveness.
- Significantly higher in white populations than in nonwhite populations
- More common among competitive athletes: Female athletes include those in sports like gymnastics and distance running and Males in sports such as bodybuilding and wrestling.

Mortality/Morbidity:

- Illness and death often is secondary to starvation or suicide.

Clinical Manifestations;

- Weakness and lassitude
- Malnutrition; Low BMI, low body fat percentage
- Central nervous system; Apathy, poor concentration
- Cognitive impairment; depressed, irritable mood
- CT scan: Ventricular enlargement; MRI: Decreased gray and white matter
- Cardiovascular and peripheral vascular; Palpitations, weakness, dizziness, shortness of breath, chest pain, coldness of extremities

- Clinical History:
  - Interviews are necessary for establishing the diagnosis.
  - Note that denial is common and Patients are notoriously unreliable informants. Hence a sense of acceptance and compassion from the interviewer is important.
  - Screen for comorbid psychiatric factors like; substance abuse/dependence, mood disorders, social phobia, obsessive-compulsive disorder, and personality disorders

Gather data from collateral sources with a view to;

- Screening for the presence/severity of symptomatology
- Guiding treatment planning
- Revealing other psychiatric symptomatology
- Assessing the progress of treatment.

It is worthy of note to expect some initial resistance.

Physical Examination

- Appearance; significantly reduced weight (in some cases, emaciation).
- Skin; lanugo (downy soft body hair on the face, volar forearms, and other surfaces of the body); scalp hair loss; Brittle nails and dry skin with yellowish discoloration (may be due to carotenemia).
- Bone: osteoporosis (due to decreased calcium, phosphorus and magnesium).
- Teeth; decalcification of the lingual, palatal and posterior occlusal surfaces of the teeth with projecting of the acid-resistant amalgams above the teeth surface (due to corrosive effects of acidic gastric contents of vomit, especially in the purging type)
- Cardiovascular; bradycardia and low blood pressure due to chronic starvation.
- Conduction abnormalities occur occasionally because of hypokalemia and less frequently because of hypomagnesemia, hypocalcemia, and hypophosphatemia).
- Cardiomyopathy can result from excessive, aggressive re-feeding
- Weight loss may follow mitral valve prolapse.
- ECG abnormalities; Prolongation of QT interval (predispose to life-threatening arrhythmias), decreased QRS amplitude, non-specific ST segment changes and T-wave changes.
- Pulmonary: Spontaneous pneumothorax and pneumomediastinum
- Fluid and electrolyte abnormalities may include dehydration, rebound peripheral edema, hyponatremia, hypokalemia, low chloride, metabolic alkalosis, hypomagnesemia and hypophosphatemia.

- Gastrointestinal complications; may include reduced taste, parotid enlargement, esophageal trauma and/or dysfunction, delayed gastric emptying, peptic ulcers, gastric dilatation, superior mesenteric artery syndrome, malabsorption, duodenal dilation, jejunal dilation, damage to the mesenteric plexus due to laxative use, pancreatitis, and hepatitis.
- Metabolic complications; may include impaired glucose tolerance, increased cholesterol levels, beta-hydroxybutyric acid increase, protein deficiency (rare), increased carotene, zinc deficiency (rare), impaired temperature regulation, and sleep deficiencies (less deep sleep, more disrupted sleep).
- Renal complications; include pre-renal failure (dehydration), dyscontrol of anti-diuretic hormone secretion, hypokalemic nephropathy, and renal stones.
- Endocrine system complications; may include abnormal hypothalamic-pituitary-gonadal axis, abnormal hypothalamic-pituitary-adrenal axis, abnormal menses, and delayed puberty.
- Hematological complications; may include anemia, leukopenia, thrombocytopenia, and abnormal cytokines.
- Neurological system: Electroencephalogram, CT scan, and MRI generally demonstrate enlarged ventricles and increased ventricle-brain ratios. These abnormalities generally normalize with weight gain.

Mental Status Evaluation

- Orientation: always well oriented to time, person, and place
- Affect often is depressed.
- Appearance; usually well-groomed and appropriately dressed; Females often wear loose clothing to cover their emaciation.
- Hallucinations and formal delusions; not commonly present; body image disturbance in some cases may be a product of delusion.

Causes:

Combination of genetic, neuro-endocrine, physiological and psychosociological influences.

- Genetic factors; based on its familial tendency
- Neuroendocrine: based on various hypotheses;
  - Hypothalamic abnormalities (e.g., neurotransmitter disturbances) primarily cause dysfunctional eating and neuro-endocrine dys-regulation in anorexia.
  - The starvation theory; abnormal hormone and neurotransmitter regulation result from reduced caloric intake.
- Physiological: Several hypotheses have been proposed.

- Delayed gastric emptying especially among anorectic and who are restricting their diet.
- Altered levels of cholecystokinin (CCK); with resultant altered gastric emptying induce a set of pathophysiologic reaction which helps sustain a psychosocially induced commitment to dieting.
- Psychological: various hypotheses support psychological, sociological and family influences.
- Psychoanalytic theorists have implicated failure to separate and develop autonomy from the primary caregivers.
- Self-psychology theorists blamed inability to express any thoughts or feelings different from her own.
- Family theories view eating disorders as a method used by the female patient as a cry for help for a conflicted and dysfunctional family.
- Cognitive-behavioral theories: believed that anorexia nervosa is conceptualized as a learned behavior maintained by positive reinforcement.
- Media influences: a product of multitude of social pressures have been promoting dietary restraint in medias like books and magazines e.g. fashion industry promote slimness, television and film industry associate thinness with "being sexy" and professional success and emphasis on physical fitness and athleticism.

Workup

- Lab Studies:
- Complete blood count
- Urinalysis
- Fecal occult blood
- Ethanol and dangerous drug screen
- Serum erythrocyte sedimentation rate (ESR) and thyroid function tests
- Imaging Studies:
- Brain imaging - Increase in ventricular-brain ratio secondary to starvation often observed
- Chest and abdominal x-rays - May be indicated
- Electrocardiogram.

Treatment

Medical Care:

- Often challenging and complicated.
- Long-term follow-up may be necessary.
- Hospitalization may be necessary:
  - To restore weight and interrupt steady weight
  - To interrupt medical risks or complications due to binging, vomiting and/or laxative use

- To evaluate and treat other potential serious physical complications
- To manage associated conditions (e.g., severe depression, suicide risk, substance use disorders)
- To ensure structure of mealtimes
- To Offer intensive therapy without breaking off outside supports and challenges
- Advantages;
- ✓ More economical than full hospitalization
- ✓ Provides a useful bridge between inpatient and outpatient care
- o Outpatient medical management;
- Indicated for patients with chronic conditions often for careful evaluation especially in resistant cases
- Also to maintain body weight, electrolytes, and vital signs
- To make medical referral to specialists as needed
- Education-based interventions include;
- o Diet, meal planning
- o Nutritional management
- o Self-help interventions
- Psychotherapy: essential for effective utilization of other treatment modalities. The following are types of psychotherapy that clinicians have found useful:
- o Individual include Psychodynamic; Self; Interpersonal and Cognitive-behavioral variants
- o Group: psychodynamic-oriented group psychotherapy to address underlying personality disorders.
- o Family therapy and couples therapy: These frequently useful to reduce symptom and solve family related problems
- Managing medical complications: requires continuous and repeated monitoring of:
- o Electrolyte status and dehydration (e.g., potassium, calcium, magnesium, phosphate levels)
- o Hypo-estrogenemia, amenorrhea, and osteoporosis
- o Frequent dental evaluations
- o Input into nutritional support
- o Medical emergencies (e.g., cardiac arrhythmias, symptomatic electrolyte disturbances, significant GI bleeding)
- o Infertility
- o Pregnancy
- Treatment of patients with personality disorders: Especially cluster B personality pathology (particularly borderline or histrionic personality features) or cluster C has been associated with anorexia nervosa (specifically avoidant, obsessive-compulsive, or dependent personality disorders).
- Addressing treatment refusal issues;
- o Seek to engage in a strong voluntary therapeutic alliance.

- Reasons for treatment refusal should be identified.
- Make sure the patient understands treatment recommendations.
- Expect the patient to want to negotiate aspects of the treatment plan.
- Promote autonomy to the greatest extent possible.
- Realistically assess the risks and benefits of imposed treatment.
- Avoid power struggles.
- Allow patients as much control as possible without endangering the recovery process.
- Assure treatment interventions are not punitive.
- The family should be involved in the treatment.
- Obtain ethical and legal clarification and support when considering imposed treatment.
- Only consider legal means of imposing treatment when refusal is judged to have serious risk.
- Consider alternative approaches when treating chronic cases.

Medication

- Antidepressant medications; fluoxetine (average 40 mg/day) ; Selective serotonin reuptake inhibitors (SSRIs)
- Low doses of neuroleptics to treat marked obsessionality, anxiety, and psychotic-like thinking
- Anti-anxiety agents used selectively before meals to reduce anticipatory anxiety concerning eating.
- Estrogen replacement; to reduce calcium loss (thereby reducing risks of osteoporosis)
- Pro-motility agents such as metoclopramide to treat bloating and abdominal pains (due to gastroparesis and premature satiety).

Prevention:

- Primary prevention targets the following:
- Societal concerns with thinness
- Enlightening the public about the dangers of dieting and anorexia nervosa
- Emotional problems of female adolescents and young adults
- Secondary prevention;
- Early recognition and early intervention
- Patient Education: about the dangers of dieting and anorexia nervosa.

3.4 Disorders of the Oral Cavity (Dental Plaque and Caries)

Dental plaque is a dull-white, sticky substance that adheres on the teeth. Its presence sets in motion, the process of tooth decay, which is an erosive process due to the action of bacteria on these plaques to

produces powerful acids that can dissolve dental enamel. The result in the breakdown of the enamel due to formation of a fissure (hole):
If this process remains un-halted, the erosion proceed through the hard, avascular dental tissues (enamel & dentine) to involve the soft, vascular and innervated dental tissue (pulp)
The extent and pace of this process will depend on;

- The extent of deposition of plaque
- The extent of activities of acid-producing bacteria
- The presence of saliva to inactivate the bacteria and reduce the rate of acid production
- The presence of exogenous acids in the mouth
- The structural integrity of the teeth.

Predisposing factors include;

- Xerostomia associated with Sjȍgren's syndrome, drug-induced, post-irradiation of the head and neck)
- Diabetes mellitus
- Alcohol consumption
- Tobacco use
- Down's syndrome
- Gingival hyperplasia
- Dehydration.

Signs and symptoms

- Pain is the initial complaint
- Presence of fissure (a break in the tooth's enamel)
- Facial swelling
- Enlarged regional lymph nodes
- Dental x-ray studies may show the extent of damage.

Treatment; may include;

- Conservative treatment;
  - During acute painful stage, the patient will require strong analgesics and antibiotics
  - A lot of rest
  - Hospitalization may be required to control severe pain
- Definitive management; often done after acute symptoms have subsided and may include;
  - Dental fillings
  - Dental implants
  - Dental extractions.

Prevention

- Effective mouth care with correct mouth wash

- Reducing the intake of starches and sugars (refined carbohydrates)
- Intake of adequate fluoride; may be through;
  - Direct application to the teeth
  - Drinking fluoridated water
- Stop smoking and excessive alcohol intake
- Controlling diabetes mellitus
- Using pit and fissure sealants
- Rinsing mouth with water after an acidic meal or before bed time

Complications
- Dental abscess
- Sepsis
- Chronicity
- Oral malignancy
- Ludwigs angina (deep neck spaces infection and abscesses).

Parotitis
Background:
The parotid glands unobtrusively shape themselves to fit into the available spaces of the dental occlusal planes. Perfect functioning often goes unnoticed

They are situated on either side to irrigate and saturate a food bolus with saliva.

The parotid glands are mechanically squeezed into the buccal cavity by the action of the muscles of mastication (masseter and medial pterygoid) during chewing.

Saliva synthesis and secretion is governed by the parasympathetic nervous system

Saliva assists in moistening, lubricating, and protecting the teeth and oropharyngeal mucosa linning.

Parotitis refers to inflammation of the salivary gland. This may be viral or bacterial, autoimmune in origin or even a combination.
Classification and etiology
- Infectious Parotitis; caused by known infectious agents.
  - Acute bacterial parotitis; commonly in terminally ill dehydrated or the elderly due to saliva stasis; Often cause parotid abscesses. Rare in neonates and infants. Also called acute suppurative parotitis.

- Chronic bacterial parotitis; may co-exist with calculi or stenosis of the ducts (secondary to injury); a sequela of acute bacterial infection
- Acute viral parotitis (mumps); a classic childhood infection spread by droplets or by direct contact with infected oro-pharyngeal secretions. It is caused by the *Paramyxovirus*. May be complicated by autoimmune meningoencephalitis, pancreatitis, orchitis or deafness: Treatment requires a symptomatic and supportive management
- HIV parotitis; due to lymphocyte infiltration; the gland is firm, non-tender and chronically enlarged (unilateral or bilateral) and usually causes few symptoms. No specific treatment is required
- Tuberculous Parotitis; uncommon associated with AIDS

- Chronic Punctate Parotitis (Chronic Autoimmune Parotitis);
  - Associated with Mikulicz disease, Sjögren syndrome, benign lymphoepithelial lesion of Godwin, chronic punctate sialectasis and recurrent parotitis of childhood. The basic features include massive lymphoid infiltration, acini cell atrophy, proliferation of the cells of the small ducts and obliteration of their lumen.
- Idiopathic diseases.
  - Recurrent parotitis of childhood; uncommon syndrome clinically resemble mumps. Usually begin by age 5 and become asymptomatic by age 10-15 years. Duration of attacks averages 3-7 days. The disease is often unilateral. Bacterial cultures of the saliva produce low virulent *Streptococcus viridians.*

Other causes of parotitis include;

- Neoplasms
- Drugs such as iodides, guanethidine, phenylbutazone, thiouracil, isoproterenol, heavy metals, sulfisoxazole, phenothiazines and antihistamines.
- Systemic conditions such as uremia and kwashiorkor.
- Autoimmune diseases such as rheumatoid arthritis
- Dehydration (an infrequent cause)
- Diseases such as Wegener granuloma or Kimura Disease (rare cause).

Epidemiology;

- Death is extremely unusual.
- The degree of morbidity has equal proportion with the original disease
- Sex predilection: affects both sexes equally; Parotitis due to Sjögren syndrome occur with male-to-female ratio of 1:9.
- Viral parotitis (mumps) is very common among children.

Clinical Manifestations;
These vary based on the etiology and classifications of the disease;

- Infectious parotitis:
  - Acute bacterial parotitis; presents with complains of progressive painful swelling of the gland; chewing aggravates the pain.
  - Acute viral parotitis (mumps); Pain and swelling of the gland lasting 5-9 days; moderate malaise, anorexia, and fever; bilateral involvement present in the majority of instances
  - HIV parotitis; Non-painful swelling of the gland; otherwise, patient is asymptomatic.
  - Parotitis in tuberculosis; Chronic non-tender swelling or a lump within the gland; often ipsilateral;
- Chronic punctate parotitis (chronic autoimmune parotitis)
  - Lymphoepithelial lesion of Godwin and Mikulicz disease; represents an historical disease which should not be diagnosed today.
  - Sjögren syndrome; Recurrent or chronic swelling of unilateral or bilateral parotid glands with no apparent cause; frequently associated with autoimmune disease; discomfort modest in most cases; dry mouth and eyes
- Diseases of uncertain etiology;
  - Recurrent parotitis of childhood; Repetitious episodes of unilateral or bilateral mumps-like episodes in a young child
  - Sarcoidosis - Chronic non-tender swelling of parotid gland

Physical findings;

- Inspection: Swelling and erythema of the overlying skin
- Palpation:
  - Tenderness in acute inflamation: Chronic autoimmune parotitis usually is non-tender.
  - Massage postero-anteriorly expresses;
    - Saliva (if parotid duct is patent).
    - Purulent saliva in cases of bacterial parotitis
    - Clear saliva with small yellow curds in chronic punctate (autoimmune) parotitis.

Investigations;
Lab Studies:
Chemical analysis of saliva
Saliva culture (rarely is helpful)

Imaging Studies:
CT scans and MRI scans; determine the size, shape, presence of neoplasms or swelling within the gland (as well as differentiates between solid masses, cystic lesions) and diffuse involvement of the gland.

Sialography; to demonstrate the anatomy of the drainage system and to rule out stones or calcification within the gland

Surgical Treatment:

- Incisional biopsy; should be done by an experienced surgeon under local anesthesia to avoid facial nerve injury.
- Fine needle aspiration biopsy; preferred to diagnose tumors, identify cell types and to obtain material for cultures (when the clinical picture suggests infection)
- Excisional biopsy; may help in diagnosis when clinical picture suggests Sjögren syndrome.
- Incision and drainage; in case of abscess due to acute bacterial parotitis that does not respond to intravenous antibiotics and hydration
- Parotidectomy; in chronic parotitis especially due to frequent episodes of severe bacterial infection

Management

- Conservative treatment:
- Most episodes require symptomatic treatment with;
  - Local heat
  - Gentle massage of the gland from posterior to anterior and
  - Hydration
  - Culture and sensitivity of the saliva is required by pussy saliva to guide antibiotic selection.
  - Treatment of the primary disease is essential
- Hospitalization for parotitis is extremely unusual
- Discontinue medicines with atropine-like effects, and substitute other medications when feasible
- Avoid mechanically hard food stuffs but rather soft liquid diet
- Oral toileting with soothing antiseptic mouthwash

Antibiotic treatment;

- Empirical treatment; Iv. Vancomycin, 500 mg q 6 hours is begun empirically.
- Definitive treatment; guided by results of cultures

Prevention:

- Adequate hydration
- Oral hygiene
- Discontinuing use of medications with atropine effects in case of evident xerostomia may be helpful
- Immunization avoids epidemic parotitis (mumps).

Complications:

- Dental infections and caries due to xerostomia

- Lymphoma often may follow autoimmune parotitis.

3.5 Dentoalveolar Abscess or Peri-apical Abscess

Peri-apical abscess is the collection of pus in the apical dental periosteum
Apical dental periosteum is the fibrous membrane that supports the apical tooth structure and its surrounding tissue. The apex of the tooth is where it is suspended in the jaw bone.

Classifications;
It can be an acute or a chronic process.
Acute peri-apical abscess; often a sequela of a suppurative pulpitis due to an infected dental caries
Chronic abscess formation is often a slow and almost silent process that occurs following a long-standing infectious process. The end result may be a peri-apical granuloma formation which often can be discovered on x-ray films.

Clinical Manifestations
- Pain which can be described as dull, gnawing and continuous
- Evidences of adjacent facial tissue inflammation (cellulitis and edema)
- Loose and mobility of the involved tooth
- Adjacent gingival swelling is also possible
- Difficulty in opening the mouth due to inflamed facial structures
- Fever and malaise.

Definitive Management
- Abcess drainage which may be done through different approaches such as through;
  - A fine needle aspiration
  - An opening into the pulp chamber through the enamel
  - An incision through the gingiva down to the jawbone
- Tooth extraction; often indicated in a chronic process. Done after complete resolution of an acute inflammation
- Antibiotics therapy.

Conservative Management
- Monitor and record bleeding following an invasive tooth procedure
- Encourage patient to use a warm saline for oral toileting
- Ensure patient comply with antibiotics and analgesics regimen
- Advice on liquid to a soft diet as tolerated
- Encourage patient to keep follow-up appointments.

3.6 Malocclusion

Malocclusion describes a phenomenon characterized by a mis-alignment of the (upper and lower) dental arcs in a closed jaw
Causes;
This may be due to an inherited or acquired condition

- Acquired causes may follow;
  - Mechanical displacement of the dental arc such with factors as thumb-sucking or trauma
  - Acquired prognathism due to *acromegaly*
  - Maxillary or mandibular deformity due to *Paget's disease* of the bone.

It is a very common phenomenon.
Its main implications include;

- Aesthetic concern to the patient
- Predisposition to;
  - Teeth decay
  - Gingival disease and
  - Excess wear on supporting bone and gum tissues.

Management
Silent condition may be left alone.
Obvious condition may be of major concern to the patient and will seek help.

Management is done in two phases;

- Phase 1; involves re-alignment of the teeth; that is, gradually forcing the teeth into alignment and supporting it with braces inform of wires or plastic bands.
- Phase 2; wearing a retaining device for several hours each day to support the tissues as they adjust to the new alignment of the teeth.

Treatment is best done as soon as the last primary tooth has been shed and the last permanent successor has erupted
Meanwhile, encourage;

- Meticulous oral hygiene
- Continuous wearing the retainer (supporting braces).

3.7 Temporomandibular Disorders

Temporomandibular disorders mat include;
Has been categorized as follows;

- Myofascial pain—a discomfort in the muscles controlling jaw function and in neck and shoulder muscles.

- Internal derangement of the joint—a dislocated jaw, a displaced disc, or an injured condyle.
- Degenerative joint disease—rheumatoid arthritis or osteoarthritis in the jaw joint.

Causes include;
- Arthritis of the jaw
- Head injury
- Trauma (jaw or joint)
- Stress
- Malocclusion (uncertain).

Clinical Manifestations
- Pain; described as dull ache or throbbing, can radiate to the ears, teeth, neck muscles, and facial sinuses.
- Often restricted jaw motion
- Locked jaw
- Clicking and grating noises with jaw movement
- Difficulty of chewing and swallowing
- Speech difficulty
- Depression (a response to these symptoms).

Assessment and Diagnostic Findings
Patient may require radiographic investigations like MRI, X-ray and an arthrogram.

Management
- Conservative management:
  - Stress management (may be help reduce grinding and clenching of teeth)
  - Range of motion (ROM) exercises
  - Pain management with non-steroidal anti-inflammatory drugs (NSAIDs), opioids, muscle relaxants or mild antidepressants.
  - TMJ support/ immobilization with a bite plate or splint (plastic guard worn over the upper and lower teeth).

Surgical Management
Surgical correction: Useful in irreversible
Surgical options may include jaw repositioning or reconstruction
- Jaw repositioning; indicated for simple mandibular fractures (without displacement). Fractured jaw can be reconstructed with internal plate fixation to approximate and stabilize the bone
- Jaw reconstruction; may be indicated by severe mandibular injury with a significant amount of tissue loss following trauma or cancer. Usually, a bone graft is required.

Nursing Management

- Patient with a rigid internal fixation will do well without chewing hard food within the first 1 to 4 weeks post-operation.
- A liquid diet will
- Dietary caloric and protein intake should be optimal
- Mouth care is essential
- Report any form oral irritated as soon as possible
- Keeping scheduled appointments to assess the stability of the fixation appliance is of essence.

3.9 Sialadenitis

Refers to inflammation of the salivary glands

Causes/ predisposing factors; may include;

- Dehydration
- Radiation therapy
- Stress
- Malnutrition
- Salivary gland calculi (stones)
- Improper oral hygiene
- Inflammation
- Microbial agency like;

o *Staphyloccocusaureus, Streptococcus viridans,* or pneumococcus.
o Methicillin-resistant *S. aureus*(MRSA) in hospitalized patients.

Signs and Symptoms;

- Pain
- Swelling
- Purulent discharge.

Management;

- Antibiotics
- Massage and analgesics to manage pain
- Hydration and
- Corticosteroids
- Chronic, recurrent cases will require;

o Surgical incision and drainage
o Surgical excision of the gland and its duct.

3.9.1 Salivary Calculus (Sialolithiasis)

Refers to deposition of calculi (stones) within the salivary gland
It often affects the sub-mandibular gland
Chemically, the stone is commonly calcium phosphate

It is often formed within the substance of the gland, however few cases may be located within the ductal system.
Stones are irregularly shaped and vary in diameter from 3 to 30mm.

Predisposing factors include;
- Impaired drainage of saliva as with salivary duct stenosis
- Dehydration
- Excessive production and stasis
- Hypercalcemia
- Infection.

Stones may be demonstrated by;
- Ultrasonography or
- Sialography (specialized x-ray following injection of a radiopaque substance into the duct).

Signs and symptoms;
May be asymptomatic unless infected
Sudden, local, and often colicky pain; due to salivary duct obstruction; relieved transiently by a gush of saliva
On palpation, the gland is swollen and tender
Management;
- Stones can be spontaneously passed via the enlarged ductal orifice
- Calculus extraction through the duct in the mouth
- Lithotripsy; disintegration of the stone with shock waves to disintegrate the stone: This may be used as an alternative to surgery.
- Surgical extraction of the stones done under anesthesia, sedation or analgesia: surgical complications include hemorrhage and swelling.

3.9.2 Oral Cancers

Malignant changes can occur in any part of the mouth or throat
This type of malignancy is curable if discovered early.

Epidemiology;
There is a strong association between oral malignancies and;
- Use of tobacco
- Alcohol intake
- Age above 40 years
- Male gender
- Dietary deficiency like Vitamin B-complex
- Ingestion of smoked meats (carbon dusts).

Clinical Manifestations

- Early manifestations:
  - Asymptomatic
  - Typical painless sore or mass
  - A non-healing ulcer (more than 2 weeks)
  - Typical lesion is painless and indurated with raised edges.
- Late manifestations;
  - Pain and tenderness
  - Dysphagia
  - Difficulty in chewing or speaking
  - Coughing productive of blood-tinged sputum
  - Enlarged cervical lymph nodes.

Assessment and Diagnostic Findings

- Physical assessment;
  - Oral examination for chronic ulcers
  - Neck assessment for evidence of local metastases (enlarged, fixed and rough masses) Biopsies; of suspicious lesions
  - High-risk areas include;
    - The buccal mucosa and gingival; (for snuffers or smokers)
    - The buccal floor, ventrolateral tongue and soft palate complex (soft palate, anterior and posterior tonsillar area, uvula, and the area behind the molar and tongue junction) for those who smoke cigarettes and drink alcohol.

Medical Management

- Management choice is based on;
  - Nature of the lesion: Tumors larger than 4 cm requires a more aggressive treatment as they often recur while Cancer of the tongue will require radiation and chemotherapy due to high chances of metastasis
  - Physicians preference and
  - Patient choice (aesthetics); large lip tumors may be treated with radiation rather than surgical resection.
- The choices include;
  - Surgery: small, localized tumors will require resection of the lesion; evidence of regional metastasis will require a more radical approach with dissection of regional lymphatics
  - Radiation therapy: can be form implanted internal radiation sources or external radiation source. Internal radiation sources is a new and better way of curbing systemic and regional metastasis
  - Chemotherapy or
  - Combination of these therapies may be effective.

Nursing Management

- Assessment of patient's nutritional status
- Nutritional supplementation; through enteral (through the intestine) or par-enteral (intravenous) feedings to maintain adequate nutrition
- Post-operation;
  - Assess the airway for patency
  - Oropharyngeal suctioning
  - If grafting was done;
    - Careful suctioning to prevent damage to the graft
    - Assess for viability (white patch indicate arterial occlusion while a blue mottling may indicate venous congestion)
    - A radial pulse at the graft site may be located with a doppler ultrasound device to assess graft perfusion.

Relevant Nursing Diagnoses for patients with Oral cavity and nutritional disorders

i. Altered nutrition: less than body requirement
ii. Altered nutrition: more than body requirement
iii. Impaired oral mucosa
iv. Pain
v. Body image disturbance related to swelling of the cheek
vi. Risk for infection related to saliva stasis or saliva lack.

Relevant nursing outcomes:

i. Enhancement of weight
ii. Weight management
iii. Oral care promotion
iv. Infection management
v. Esteem enhancement.

4.0 SUMMARY

At this juncture, you are expected to have learnt about:

i. various disorders of nutrition
ii. the etiologic factors and Pathophysiology of nutritional and the Oral Cavity disorders
iii. caring for patients with disorders of nutrition and oral cavity using the nursing process approach.

Activities

Now that you have learnt this much, visit a hospital of choice within your vicinity and manage a patient with any disorder nutrition or the oral cavity using the nursing process approach.

SELF-ASSESSMENT EXERCISE

Now, you will be required to evaluate yourself with the following questions:
Mr. O.J. is a 45 year old, who presented in the clinic with severe pain in the mouth, worse on mastication associated with dry mouth, furred tongue and halitosis. He was diagnosed to be having parotid stones.

i. What are the types of nutritional disorders
ii. Enumerate the social, psychological and physical factors that affects eating
iii. Using the nursing care plan, identify and solve in order of priority, the two nursing diagnoses of Mr. O.J.

Note that, you are to grade yourself based on the information provided within the content of the unit. If you have graded yourself poor, you can re-work.

5.0 REFERENCES/FURTHER READING

Levey, R., Williams-Wilson, B. (2002).Anorexia Nervosa: eMedicine Journal, April 2 2002, Volume 3, Number 4.

Smeltzer, S.C., Brenda, B. (2006). Brunner and Suddhart's Textbook of Medical-Surgical Nursing, 10th edition. Lippincott-William & Wilkins.

Templer, J. W. (2001). Parotitis; eMedicine journal; July, 2001, Vol. 2, Number, 7.

UNIT 3 CARING FOR PATIENT WITH NECK AND ESOPHAGUS

CONTENTS

1.0 INTRODUCTION

This unit will focus on exploring care of patients with neck and esophageal disorders, with the intent of improving your knowledge base and requisite clinical skills.

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- describe various disorders of the neck and esophagus
- identify the etiologic factors of esophageal disorders
- explain the Pathophysiology of esophageal disorders
- care for patients with disorders of the esophagus using the nursing process approach.

3.0 MAIN CONTENT

3.1 Dysphagia

This means difficulty in swallowing
This represents the most common esophageal disorder

This manifestation may vary from mere uncomfortable sensation to overt painful sensation especially one that accompany swallowing (**odynophagia**).

The problem can be within the upper, middle, or lower segments of the esophagus.

Causes of dysphagia include;

- Achalasia
- Diffuse spasm
- Gastro-esophageal reflux disease (GERD)
- Hiatal hernia
- Esophageal diverticula
- Esophageal perforation
- Presence of foreign bodies
- Chemical burns
- Benign tumors and carcinoma.

3.2 Achalasia

This is the most common cause of dysphagia. It refers to absent or ineffective peristalsis, often within the distal esophagus, with subsequent lower esophageal sphincter failure. This cause arrest of a bolus during swallowing: The result is gradual dialation of the proximal esophageal portion, which presents as a chest mass.
The condition often develops slowly over a long period.
It is more evident in ages above 40 years of age.

Clinical Manifestations

- Difficulty in swallowing
- Regurgitation of food (either spontaneous or induced), often to relieve pressure symptoms
- Chest pain and heartburn (pyrosis)
- Cough and acute breathlessness due to aspiration of retained esophageal contents.

Diagnostic Findings
Diagnosis is through imaging techniques:

- Plain chest X-ray: show esophageal dilation above a narrowing at the lower gastro-esophageal sphincter.
- Specialized barium x-ray
- Computed tomography (CT)
- Endoscopy: to confirm diagnosis
- Manometry: measuring the esophageal pressure.

Management

- Fluid diet is necessary
- Instruct patient to eat slowly and take liberal fluids post-prandial
- Conservative management includes;
  - Smooth muscle relaxants such as calcium channel blockers and nitrates can be helpful
  - Botulinum toxin (Botox) can be injected directly into the proximal esophagus (via endoscopy) to inhibits smooth muscle contraction
- A more aggressive approach may be employed in case the conservative approaches fail; these include;
  - Pneumatic (forceful) dilation to open up the narrowed esophageal area; the procedure is quite painful and associated with high chances of perforation.
  - Surgical separation of the muscle fibers (Esophagomyotomy) with(out) anti-reflux procedure; could be another therapy approach. It is often done through a laparascopic guidance.

3.3 Diffuse Spasm

A motor disorder of the esophagus
The etiology is unknown; may follow a stressful event.
It commonly manifests among women within their middle age

Clinical Manifestations

- Dysphagia
- Odynophagia.

Diagnostic Findings

- Esophageal manometry
- Diagnostic x-ray (plain or specialized barium x-ray).

Management

- Sedatives (to reduce anxiety)
- Long-acting nitrates (to relieve pain)
- Calcium channel blockers (to manage spasms)
- Small, frequent soft diet to reduce esophageal pressure and irritation
- Bougienage; to dilation the esophagus
- Pneumatic dilation
- Esophagomyotomy (if pain is intolerable).

3.4 Hiatal Hernia

Protrussion of the upper stomach (cardia) into the lower portion of the thoraxic cavity via the weakened esophageal hiatus on the diaphragm, hence the name, hiatal hernia.
Hiatal hernia occurs more often in women than men.

Types:
Sliding (type I hiatal hernia): displacement and the sliding of the upper stomach (cardia) and the Gastro-esophageal junction (GEJ) upward and into the thorax. Represents 90% of the case and Para-esophageal hernia: describes the pushing of the stomach through the diaphragm beside the esophagus. Para-esophageal hernias may be further classified as types II, III, or IV, depending on the extent of herniation.

Clinical Manifestations

- Sliding hernia may present as;
  - Asymptomatic in 50% of cases
  - Heartburn
  - Regurgitation
  - Dysphagia
  - Reflux
- Para-esophageal hernia presents as;
  - Asymptomatic in some cases
  - A sense of fullness post-prandial
  - Reflux usually does not occur due to intact gastro-esophageal sphincter.

Diagnostic Findings

- Plain or specialized barium (swallow) x-ray
- Fluoroscopy.

Management

- Frequent, small feed
- Avoid recline for 1 hour post-prandial to prevent reflux of food or worsening of the hernia
- Patient should eat in head-up position
- Surgery to correct torsion (twisting) of the stomach especially in para-esophageal hernia.

Complications;

- Hemorrhage
- Obstruction and
- Strangulation.

3.5 Esophageal Diverticulum

A diverticulum is an out-pouching of mucosa and sub-mucosa due to weakness of the underlying musculature.

Diverticula may affect the proximal, middle or distal (epi-phrenic) esophageal portions

Types:

- Zenker's diverticulum (Pharyngo-esophageal pulsion diverticulum or a pharyngeal pouch) is the most common type, found three times more frequently in men especially those well above their 6^{th} decade of life. It occurs posteriorly through the crico-pharyngeal muscle in the nunchal midline.
- Mid-esophageal diverticula; an uncommon variant with less acute symptoms that rarely require surgery
- Epi-phrenic diverticula; usually larger variant, located in the lower esophagus just above the diaphragm: supposedly due to improper functioning of the lower esophageal sphincter
- Intramural diverticula: a numerous small diverticula associated with a upper esophageal stricture

Clinical Manifestations

- Pharyngo-esophageal pulsion diverticulum presents with;
  - Dysphagia
  - Nunchal fullness
  - Belching
  - Regurgitation of undigested food and
  - Gurgling noises after eating
  - Coughing (due to aspiration)
  - Halitosis
- Mid-esophageal diverticula presents with less acute.
- Epi-phrenic diverticula;
  - Asymptomatic in one-third of the cases
  - Dysphagia
  - Chest pain
- Intramural diverticulosis; presents commonly with chest pain

Diagnostic Findings

A barium swallow; to determine the nature and location of a diverticulum

Manometric studies; often for epi-phrenic diverticula cases to exclude a motor disorder

Esophagoscopy and blind insertion of a nasogastric tube is often contraindicated due to the danger of perforation

Management
Surgical measures: Diverticulectomy preferred in pharyngo-esophageal pulsion diverticulum. The possibility of common carotid artery and internal jugular veins damage should be skillfully avoided during surgery.
This can be accompanied by a myotomy of the crico-pharyngeal muscle to relieve a spastic muscle.
Postoperative management include;

- Nasogastric tube insertion.
- Careful assessment of the surgical incision for evidence of esophageal leakage and development of fistula
- Avoid food and fluids per-oral until x-ray studies confirms no leakage
- Begin diet with liquids and progresses as tolerated.

3.6 Esophageal Perforation

Traumatic esophagus injury and perforation is not a common phenomenon.

Causes; may result from;

- Traumatic causes;
  - Penetrating chest wound by a stab or bullet
  - Motor vehicle crash
- Non-traumatic esophageal injury and perforation may result from chemical burns
- Iatrogenic (often accidental) puncture may follow;
  - Surgical procedure
  - Instrumentation as during examination or dilation.

Clinical Manifestations

- Persistent pain
- Dysphagia
- Fever and leukocytosis (evidence of infection)
- Severe hypotension
- Signs of pneumothorax (sudden onset labored breathing).

Diagnostic Findings
Imaging studies are useful to identify the site of the injury

- Diagnostic x-ray studies
- Fluoroscopy

Management

- Broad-spectrum antibiotic therapy (to prevent or treat infection)

- A nasogastric tube; to aspirate and reduce the tendency of aspiration of gastric juice maintain patient on NPO
- Par-enteral nutrition is essential and preferred to gastrostomy (to reduce the chances of reflux and aspiration)
- Surgery repair of the perforation may be indicated.

3.7 Foreign Bodies in the Esophagus

Foreign bodies can interfere, injure or obstructs the esophageal lumen
Examples of these foreign bodies include;

- Dentures
- Fish bones
- Pins
- Small batteries
- Items containing mercury or lead.

As a rule, all foreign bodies must be removed as soon as possible
Complaints include;
Pain and dysphagia
Dyspnea (due to pressure on the trachea)

Investigations;

- Plain X-ray will identify the foreign object and reveal evidence of perforation.

Management;

- Intramuscular injection of Glucagon; to relax the esophageal muscle.
- An endoscopic remove the trapped foreign object.
- A mixture of sodium bicarbonate and tartaric acid; a gas forming compound that will increase intra-luminal and help expel any trapped object: This measure risks perforation and should be done with caution.

3.8 Chemical Burns of the Esophagus

Chemical burns refer to coagulative necrosis of the esophageal mucosa following ingestion of corrosive chemicals.

Ingestion can be intentional (as with suicidal ideation) or non-intentional (accidental).
Predisposing factors;

- Extreme of age

- Esophageal dysmotility
- Mental illness e.g. depressive illness with suicidal ideation
- Emotional disturbance in acute physical pain.

Causes;
- Swallowing of a battery which may release caustic alkaline
- Strong acid or base (e.g., lye).

Clinical manifestations;
- Acute chemical burn of the esophagus may be associated by;
- Severe burns of the lips, mouth and pharynx
- Dysphagia
- Dysponoea (due to pharyngolaryngeal edema)
- Toxic appearance, fever and shock.

Diagnosis;
- Esophagoscopy
- Barium swallow (to determine the extent and severity of damage).

Management;
- Nothing by mouth
- Intravenous fluids administration
- A nasogastric tube for feeding and prevention of aspiration
- Avoid inducing vomiting and gastric lavage (to avoid further exposure erosion)
- Corticosteroids; to reduce inflammation and minimize subsequent scarring and stricture
- Prophylactic antibiotics use
- Nutritional support should be via enteral or par-enteral feedings; enteral feeding should be withheld until the acute phase has subsided
- Management of strictures of the esophagus if it occurs should be by dilation (bougienage)
- Surgical reconstruction (esophagectomy) relieve of the stricture may be necessary in case bougienage fails.

3.9 Gastro-esophageal Reflux Disease

Gastro-esophageal refluxrefers to back-flow of gastric or duodenal contents into the esophagus. It is normal in both adults and children.
Excessive reflux may occur because of an incompetent lower esophageal sphincter, pyloric stenosis, or a motility disorder.

Clinical Manifestations
Symptoms of gastro-esophageal reflux disease (GERD) may include;

- Pyrosis (burning sensation in the esophagus)
- Dyspepsia (indigestion)
- Regurgitation
- Dysphagia or odynophagia (difficulty swallowing, pain on swallowing)
- Hyper salivation
- Esophagitis.

Diagnostic Findings
Diagnostic testing may include an endoscopy or barium swallow to evaluate damage to the esophageal mucosa.

Management

- Avoid situations that decrease lower esophageal sphincter pressure or cause esophageal irritation.
- The patient is instructed to eat a low-fat diet; to avoid caffeine, tobacco, beer, milk, foods containing peppermint or spearmint, and carbonated beverages;
- Avoid eating or drinking 2 hours before bedtime; to maintain normal body weight;
- Avoid tight-fitting clothes
- Elevate the head of the bed on bed blocks and elevate the upper body on pillows.
- Medications such as;
  - Antacids
  - Histamine receptor blockers.
  - Proton pump inhibitors
  - Prokinetic agents which accelerate gastric emptying
- Surgical intervention involves a fundoplication (wrapping of a portion of the gastric fundus around the sphincter area of the esophagus). Fundoplication may be performed by laparoscopy.

3.10 Barrett's Esophagus

This is due to a long-standing untreated GERD with consequent metaplastic changes within the epithelial lining of the lower esophageal mucosa

This has been identified as a precancerous condition that, if left untreated, can result in adenocarcinoma of the esophagus, which has a poor prognosis.

It is more common among middle-aged white men; however, the incidence is increasing among women and among African Americans.

It is due to chronic irritation from reflux acid, alcohol, tobacco due to the following predisposing conditions such as chronic GERD or cardiac sphincter incompetence

Clinical Manifestations

- Notably frequent heartburn
- Symptoms related to peptic ulcers or esophageal stricture, or both.

Diagnostic Findings

- An esophagogastroduodenoscopy (EGD) is performed. This usually reveals an esophageal lining that is red rather than pink.
- Biopsies are taken, and the cells resemble those of the intestine.

Management

Monitoring varies depending on the amount of cell changes. Some physicians may recommend a repeat EGD in 6 to 12 months if there are minor cell changes. Medical and surgical management is similar to that for GERD.

3.11 Benign Tumors of the Esophagus

Benign tumors can arise anywhere along the esophagus.
The most common lesion is a leiomyoma (tumor of the smooth muscle), which can occlude the lumen of the esophagus.
Most benign tumors are asymptomatic
They are distinguished from cancerous lesions by a biopsy.
Small lesions are excised during esophagoscopy
Lesions that occur within the wall of the esophagus may require treatment via a thoracotomy.

Cancer of the Esophagus

It is seen more frequently in African Americans than in Caucasians and usually occurs in the fifth decade of life.
Predisposing factors;

- Chronic irritation
- Ingestion of alcohol
- Use of tobacco
- GERD
- Barrett's esophagus.

Clinical Manifestations

Many patients have an advanced ulcerated lesion of the esophagus before symptoms are manifested.
Symptoms include;

- Dysphagia, initially with solid foods and eventually with liquids;
- A sensation of a mass in the throat;

- Painful swallowing;
- Substernal pain or fullness;
- Regurgitation of undigested food with foul breath and hiccups.
- Hemorrhage
- Progressive loss of weight and strength
- Substernal pain
- Persistent hiccup
- Respiratory difficulty
- Foul breath.

Diagnostic Findings

- Endoscopy with biopsy and brushings.
- Bronchoscopy for in tumors of the middle and the upper third of the esophagus
- Endoscopic ultrasound or mediastinoscopy.

Medical Management

Treatment is based on type of cell, tumor spread, and patient condition.
Early stage, treatment goals may be directed toward cure; however, it is often found in late stages, making relief of symptoms the only reasonable goal of therapy.
Treatment may include;

- Surgery: total resection of the esophagus (esophagectomy) with removal of the tumor plus a wide tumor-free margin of the esophagus and the lymph nodes in the area. Surgical resection of the esophagus has a relatively high mortality rate because of infection, pulmonary complications, or leakage through the anastomosis.
- Radiation
- Chemotherapy
- Combination of these modalities, depending on the extent of the disease.

Palliative treatment is required in advanced disease and necessary to keep the esophagus open and sustain nutrition and to control saliva.
Palliation treatment modalities include:

- Dilation of the esophagus
- Laser therapy
- Placement of an endoprosthesis (stent)
- Radiation or
- Chemotherapy.

Nursing Management

Promote weight gain based on a high-calorie and high-protein diet, in liquid or soft form, is provided if adequate food can be taken by mouth or par-enteral or enteral nutrition.
Nutritional status is monitored throughout treatment.
Inform patient about the nature of the postoperative equipment that will be used, including that required for closed chest drainage, nasogastric suction, par-enteral fluid therapy, and gastric intubation.
Immediate postoperative care;

- After recovering from the effects of anesthesia, place patient in a low Fowler's position to assist in preventing reflux of gastric secretions.
- The patient is observed carefully for regurgitation and dyspnea.
- The patient's temperature is monitored to detect any elevation that may indicate aspiration or seepage of fluid through the operative site into the mediastinum.
- If jejunal grafting has been performed, the nurse checks for graft viability hourly for at least the first 12 hours.
- Immediately after surgery, the nasogastric tube should be marked for position, and the physician is notified if displacement occurs. The nurse does not attempt to reinsert a displaced nasogastric tube, because damage to the anastomosis may occur. The nasogastric tube is removed 5 to 7 days after surgery, and a barium swallow is performed to assess for any anastomotic leak before the patient is allowed to eat.
- Once feeding begins, the nurse encourages the patient to swallow small sips of water and, later, small amounts of pureed food.
- When the patient is able to increase food intake to an adequate amount, par-enteral fluids are discontinued.
- Family involvement and home-cooked favorite foods may help the patient to eat.
- Antacids may help those with gastric distress.
- If radiation is part of the therapy, the patient's appetite is further depressed and esophagitis may occur, causing pain when food is eaten.
- Liquid supplements may be more easily tolerated.
- Often, in either the preoperative or the postoperative period, drooling is a problem.
- Oral suction may be used
- On discharge, when the patient is ready to go home, the family is instructed about how to promote nutrition, what observations to make, what measures to take if complications occur, how to keep the patient comfortable, and how to obtain needed physical and emotional support.

Relevant nursing diagnoses

i. Altered Nutrition: less than body requirements related to dysphagia and odynophagia
ii. Risk for aspiration related to esophageal constriction, esophageal reflux and presence of feeding tube
iii. Deficient knowledge about the disease process and management
iv. Risk for infection related to esophageal stasis, Gastric acid reflux.
v. Acute pain (odynophagia).

Relevant nursing outcomes

i. Nutritional enhancement
ii. Aspiration risk management
iii. Knowledge enhancement
iv. Infection prevention
v. Pain relief

4.0 SUMMARY

At this time, you should be acquainted with knowledge on disorders affecting the neck and the esophagus and how to manage a patient such disorders using the nursing process.

Activities

You may now proceed to your clinical area and manage a patient with esophageal disorder using the nursing process model.

SELF-ASSESSMENT EXERCISE

When you are through, you are free to grade yourself with information from the course content and the reference given. You are then required to share your findings with your colleagues in the discussion platform.

i. What is dysphagia?
ii. Briefly distinguish between odynophagia and dysphagia.
iii. Identify and briefly discuss, 4 causes of dysphagia.
iv. Using the nursing process, develop at least 2 nursing diagnoses with concurrent nursing outcomes from any of the above listed relevant nursing diagnoses and outcomes.

5.0 REFERENCES/FURTHER READING

Levey, R., Williams-Wilson, B. (2002). Anorexia Nervosa: eMedicine Journal, April 2 2002, Volume 3, Number 4

Smeltzer, S.C., Brenda, B. (2006). Brunner and Suddhart's Textbook of Medical-Surgical Nursing, 10th edition. Lippincott-William & Wilkins.

Templer, J. W. (2001). Parotitis; eMedicine journal; July, 2001, Vol. 2, Number, 7.

UNIT 4 CARING FOR PATIENT WITH GASTROINTESTINAL INTUBATION AND SPECIAL

CONTENTS

1.0 INTRODUCTION

This unit will involve discussions about various types of gastrointestinal intubations and special nutrition requirements so as to widen your knowledge base and improve your skills and clinical competencies in this regard

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- identify various gastrointestinal intubation devices
- describe various approaches to gastrointestinal intubations
- discuss Parenteral nutrition.

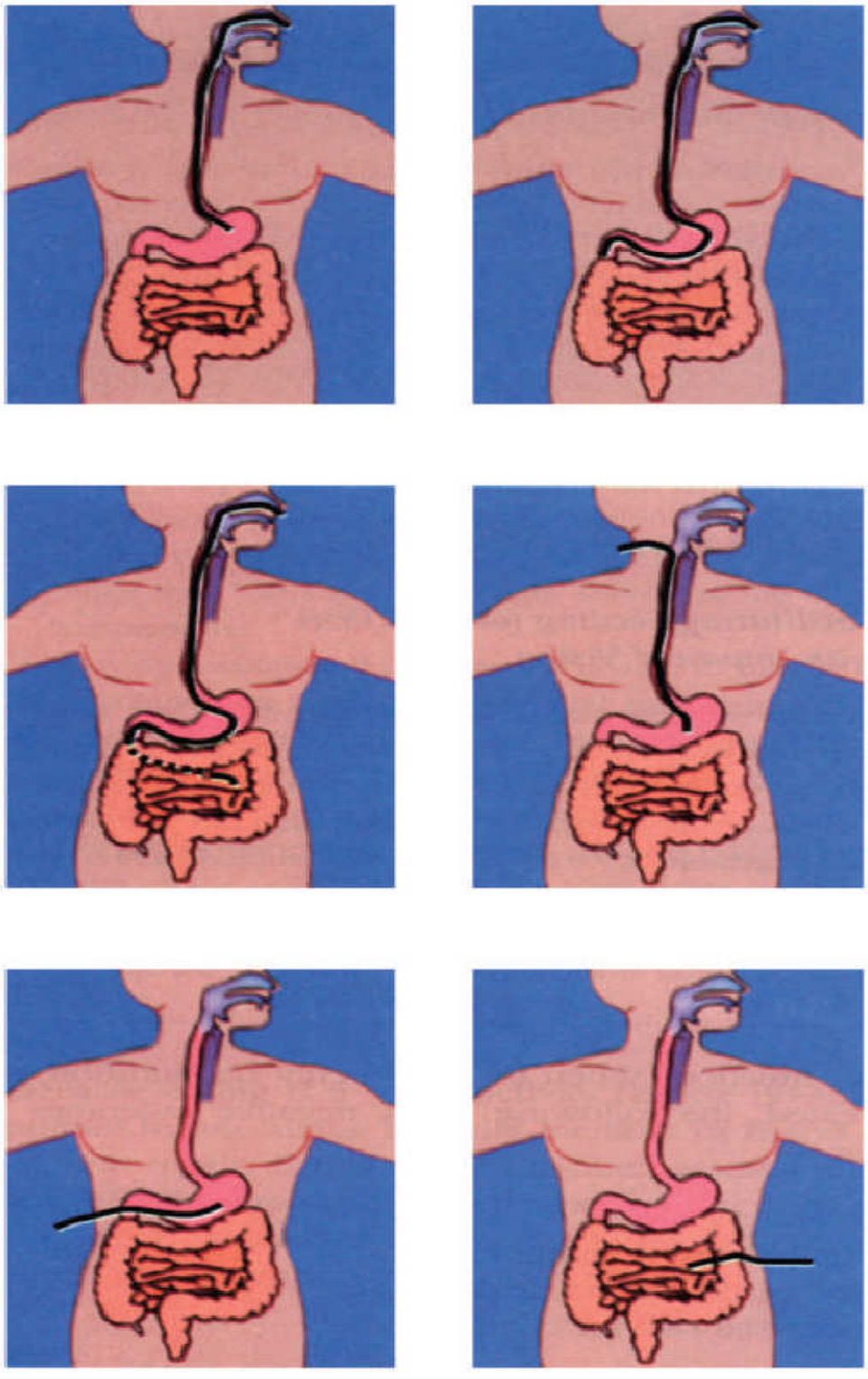


Photo 6: Forms of tube feeding

i. Nasogastric tube feeding (upper row, left)
ii. Nasoduodenal tube feeding (upper row, right)
iii. Nasojejunal tube feeding (middle row, left)
iv. Esophagostomy tube feeding (middle row, right)
v. Gastrostomy tube feeding (lower row, left)
vi. Jejunostomy tube feeding (lower row, right)

3.1 Gastrointestinal Intubation

The insertion of a rubber or plastic tube into the stomach, duodenumor the intestine

The tube may be inserted through the mouth, nose or anterior abdominal wall.

Tube feedings are essential when oral intake is inadequate or not possible

Types of gastrointestinal tubes

- Short; nasogastric (NG) tubes
- Medium; nasoduodenal tubes
- Long; nasoenteric tubes.

Indications and purposes;

- To decompress the stomach and remove gas and fluid
- To lavage the stomach and remove ingested toxins
- To diagnose disorders of GI motility and other disorders
- To administer medications and feedings
- To treat an obstruction
- To compress a bleeding site
- To aspirate gastric contents for analysis.

Varieties of GI tubes;

- The Sengstaken-Blakemore tube; type of NG tube used to treat bleeding esophageal varices.
- Orogastric tubes; large-bore tubes with wide proximal outlets for removal of particles of ingested substances (e.g., pills); they are primarily used in emergency departments.

Advantages of tube feedings over par-enteral nutrition:

- Low in cost
- Safe
- Well tolerated by the patient
- Easy to use both in extended care facilities and in the patient's home
- Preserve GI integrity by delivery of nutrients and medications (antacids, simethicone, and metoclopramide) intra-luminally
- Preserve the normal sequence of intestinal and hepatic metabolism
- Maintain fat metabolism and lipoprotein synthesis
- Maintain normal insulin/glucagon ratios.

Tube feedings can be;

- NG intubation or gastrostomy; into the stomach
- Nasoduodenal or **nasojejunal tube** feeding; into the distal duodenum or proximal jejunum.

Feeds should contain;

- Individual amino acids and carbohydrates
- Fats
- Electrolytes, such as sodium and potassium.

Complications:

- Dumping syndrome; results from giving a concentrated solution of high osmolality in large amounts with subsequent water movement from fluid surrounding the organs and the vascular compartment into the stomach and intestines. This results in feeling of fullness, nausea, and diarrhea, dehydration, hypotension, and tachycardia. Starting with a more dilute solution and increasing the concentration over several days can generally alleviate this problem.

Tube Feeding Administration Methods

- Long-term tube feeding therapy: requires a gastrostomy or jejunostomy tube feeding.
- Intermittent bolus feedings: are administered into the stomach (usually by gastrostomy tube) in large amounts at designated intervals and may be given 4 to 8 times per day.
- The continuous infusion method: is used when feedings are administered into the small intestine. This method is preferred for patients who are at risk for aspiration or who tolerate the tube feedings poorly. The feedings are given continuously at a constant rate by means of a pump. The continuous tube feeding method, which requires a pump device, decreases abdominal distention, gastric residuals, and the risk of aspiration.
- Cyclic feeding: an alternative to the continuous infusion, given at a faster rate over a shorter time (usually 8 to 12 hours): Cyclic continuous infusions may be appropriate for patients who are being weaned from tube feedings to an oral diet, as a supplement for a patient who cannot eat enough, and for patients at home who need daytime hours free from the pump.

3.2 Gastrostomy

A surgical opening into the stomach to administer foods and fluids
Preferences:

- Prolonged nutrition (greater than 3 to 4 weeks).
- Comatose patient because intact Gastro-esophageal sphincter.

Types of feeding gastrostomies;

- A **percutaneous endoscopic gastrostomy (PEG):** requires insertion of a cannula into the stomach through an abdominal incision under a local anesthetic and then secured with a thread of a non-absorbable suture through the cannula. This is done with the endoscopic guidiance. The initial PEG device can be removed and replaced once the tract is well established (10 to 14 days after insertion). The PEG replacement device should be fitted securely to the stoma to prevent leakage of gastric acid and is maintained in place through traction between the internal and anchoring devices.
- L**ow-profile gastrostomy device (LPGD):** An alternative to the PEG device. It may be inserted 3 to 6 months after initial gastrostomy tube placement. These devices are inserted flush with the skin; they eliminate the possibility of tube migration and obstruction and have anti-reflux valves to prevent gastric reflux. Two types of devices may be used.
  - ***The obturated devices (G-button)*** have a dome tip that acts as an internal stabilizer. A major drawback is the need for a physician to obturate (insert a tube that is larger than the actual stoma).
  - ***The non-obturated device (MIC-KEY)*** has an external skin disk and is inserted into the stoma without force; a balloon is inflated to secure placement. A nurse in the home setting can insert these devices easily. The drawbacks of both types of LPGDs are the inability to check residual volumes (one-way valve) and the need for a special adaptor to connect the device to the feeding container.

3.3 Parenteral Nutrition

Refers to a method of providing nutrients to the body by an IV route
It is a very complex admixture of individual chemicals combined in a single container.

The components of a par-enteral nutrition admixture are proteins, carbohydrates, fats, electrolytes, vitamins, trace minerals, and sterile water.
The goals of par-enteral nutrition;

- Improve nutritional status

- Establish a positive nitrogen balance
- Maintain muscle mass
- Promote weight gain
- Enhance the healing process.

Traditional IV fluids do not provide sufficient calories or nitrogen to meet the body's daily requirements.

Parenteral nutrition solutions will supply nutrients such as dextrose, amino acids, electrolytes, vitamins, minerals, and fat emulsions which will provide enough calories and nitrogen to meet the patient's daily nutritional needs.

Clinical Indications

- A 10% deficit in body weight (to pre-illness weight)
- Inability to take oral food or fluids within 7 days after surgery
- Hyper-catabolic situations such as major infection with fever, severe burns, malnutrition, short bowel syndrome, AIDS, sepsis, cancer)
- Impaired ingestion of food orally or by tube e.g., paralytic ileus, Crohn's disease with obstruction, post-radiation enteritis, severe hyper-emesis gravidarum in pregnancy).
- Lack of interested in ingesting adequate nutrients (e.g., anorexia nervosa, postoperative elderly patients).
- Underlying medical contraindicating oral feeding orally or by tube (e.g., acute pancreatitis, high enterocutaneous fistula)
- Pre-operative and postoperative nutritional needs are prolonged (e.g., extensive bowel surgery).

Initiating Therapy

- Parenteral solutions are initiated slowly and advanced gradually each day to the desired rate, as the patient's tolerance permits.
- The patient's assessment includes patient's weight; fluid intake-output and blood glucose.
- Baseline and periodic monitoring of complete blood count, platelet count, and chemistry panel, including serum carbon dioxide, magnesium, phosphorus, triglycerides, and pre-albumin.
- A 24-hour urine nitrogen determination (for analysis of nitrogen balance).

Administration Methods

This is through various vascular access devices; which may be either peripheral or central IV lines, depending on the patient's condition and the anticipated length of therapy.

Peripheral Method: This is through a peripheral vein; requires a less hypertonic solution.
Peripheral Parenteral Nutrition (PPN) formulas are not nutritionally complete as protein and dextrose are limited because they irritate the intima (innermost walls) of small veins, causing chemical phlebitis. The usual length of therapy using PPN is 5 to 7 days
Central Method: This is through a catheter inserted into a high-flow, large blood vessel (the subclavian vein).

Four types of central venous access devices (CVAD) are available:

- Non-tunneled (or percutaneous) central catheters
- Peripherally inserted central catheters
- Tunneled catheters
- Implanted ports.

Catheter tip placement should be confirmed by x-ray studies before parenteral nutrition therapy is initiated.
The optimal position is the mid-proximal third of the superior vena cava.

Discontinuing Parenteral Nutrition

- The solution is discontinued gradually to allow the patient to adjust to decreased levels of glucose.
- After termination, isotonic glucose is administered for several hours to protect against rebound hypoglycemia.
- Oral carbohydrates will shorten the tapering time.
- The venous catheter should be removed while an occlusive dressing is applied to the exit site.

4.0 SUMMARY

At this juncture, you should have understood gastrointestinal intubation and consequently should be able to:

i. Identify various gastrointestinal intubation devices
ii. Describe various approaches to gastrointestinal intubations
iii. Discuss Parenteral nutrition.

Activities

Now, find a patient that is presently on nasogastric tube feeding; interact with them and report the following:

i. His indication(s) for this choice of nutrition
ii. The procedure of insertion of the nasogastric tube
iii. The maintenance of the tube
iv. The procedure of feeding the patient
v. Your assessment of the procedure as a whole

You are required to share this with other colleagues on the discussion forum platform.

SELF-ASSESSMENT EXERCISE

i. Discuss various approaches to gastrointestinal tube feedings.
ii. Identify the indications of gastrointestinal tube feedings.
iii. Enumerate the complications of gastrointestinal tube feedings.
iv. Discuss the procedure of feeding a patient on Parenteral nutrition.

After you are through with these questions, you may assess your extent of learning by referring to the unit content and the reference resources. Good luck.

5.0 REFERENCES/FURTHER READING

Levey, R., Williams-Wilson, B. (2002). Anorexia Nervosa: eMedicine Journal, April 2 2002, Volume 3, Number 4.

Smeltzer, S.C., Brenda, B. (2006). Brunner and Suddhart's Textbook of Medical-Surgical Nursing, 10th edition. Lippincott-William & Wilkins.

Templer, J. W. (2001). Parotitis; eMedicine journal; July, 2001, Vol. 2, Number, 7.

UNIT 5 CARING FOR PATIENT WITH GASTRIC AND DUODENAL

CONTENTS

1.0 INTRODUCTION

This unit will focus on detailed discussion of diseases affecting the stomach and duodenum, with the intent of further expanding your knowledge base and consequently improving your competence in caring for patients with these disorders

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- identify diseases affecting the stomach and the duodenum
- discuss the Pathophysiology of these conditions
- using the nursing process, diagnose the patients and manage them accordingly.

3.0 MAIN CONTENT

3.1 Gastritis

This is the inflammation of the gastric mucosa
It is a common GI problem.

Classifications;

Acute gastritis; symptoms last several hours to a few days
Chronic gastritis; refers to a repeated attacks due to continuous exposure to the offending agents.

Causes;

- Micro-organisms e.g. *Helicobacter pylori*
- Mechanical irritants like highly seasoned food
- Non-steroidal anti-inflammatory drugs (NSAIDs)
- Alcohol intake

- Bile reflux
- Radiation therapy
- A more severe
- Chemical irritants; strong acid or alkali
- Tumors; benign or malignant
- Stimulants such as caffeine.

Pathophysiology

Gastritis parades gastric mucosa edema and hyperemia with subsequent superficial erosion. Associated scanty secretion of gastric juice (achlorhydria or hypochlorhydria) results in reduced absorption of vitamin B12 and folic acid resulting in megaloblastic anemia
Superficial ulceration may result in hemorrhage.

Clinical Manifestations

- Abdominal discomfort
- Headache
- Lassitude
- Nausea, anorexia or vomiting
- Hiccupping
- Heartburn after eating
- Belching
- Sour taste in the mouth (signifies regurgitation).

Diagnostic Findings

- Quantitative analysis of gastric juice to exclude achlorhydria or hypochlorhydria
- Endoscopy
- Upper GI radiographic studies
- Histologic examination of a tissue specimen obtained by biopsy
- Serologic testing for antibodies against the *H. pylori* antigen

Management

- Avoid alcohol and food until symptoms subside
- Diet modification
- Promoting rest and reducing stress
- Parenteral fluid administration
- Diluting and neutralizing the offending agent if known; strong acids can be diluted with common antacids (e.g., aluminum hydroxide) while strong alkali with diluted lemon juice or
- Avoid emetics and lavage if evidence of ingestion of corrosive poison
- Nasogastric (NG) intubation
- Analgesics

- *A*ntibiotics; anti-*H. pylori* e.g., tetracycline or amoxicillin, combined with clarithromycin)
- Proton pump inhibitor (e.g., Omeprazole
- Sedatives
- Fiber optic endoscopy
- Surgical management;
- Intended for complicated cases to remove gangrenous or perforated tissue
- Gastrojejunostomy or gastric resection may be necessary to correct pyloric obstruction.

3.2 Peptic Ulcers (Gastric & Duodenal Ulcers)

A peptic ulcer results from digestion and ulceration of area of GI mucosal wall prone to actions of peptic acid such as the gastric **pylorus**, **duodenum** or distal esophagus.

Erosion may extend as deeply to affect the muscle layers or through the muscle to the peritoneal layer of the gut.
A peptic ulcer is frequently referred to as a gastric, duodenal, or esophageal ulcer, depending on its location; they may occur alone or in multiples.

Chronic gastric ulcers tend to occur in the lesser curvature of the stomach, near the pylorus.

Peptic ulcer disease occurs with the greatest frequency in people between the ages of 40 and 60 years and more common in women.
Causes/ predisposing factors;

- Stress
- Anxiety
- Infection with the gram-negative bacteria *H. pylori*
- Smoking
- Alcohol intake
- Familial tendency
- Blood type O
- Chronic use of NSAIDs
- Ingestion of milk and caffeinated beverages
- Excessive amounts gastrin due to tumors.
- They result in excessive secretion of HCl.

Other rare variants of PUD

- Zollinger-Ellison syndrome (ZES); consists of severe peptic ulcers, extreme gastric hyperacidity, and gastrin-secreting benign

or malignant tumors (gastrinomas) of the pancreas. Diarrhea and steatorrhea (unabsorbed fat in the stool) may be evident.

- Stress ulcers; ulcerations in the mucosa that can occur in the gastroduodenal area especially in patients exposed to stressful conditions.
- Esophageal ulcers; occur as a chronic gastro-esophageal reflux disease [GERD].
- Cushing's ulcers are common in patients with head injury. They may occur in the esophagus, stomach, or duodenum.
- Curling's ulcer is frequently observed about 72 hours after extensive burns and involves the antrum of the stomach or the duodenum.

Clinical Manifestations

May be asymptomatic in some cases

Many people may present with perforation or hemorrhage without any fore warning

Some common complaints include;

- Pain: the principal complaint. Has been described as a dull, gnawing pain or a burning sensation in the mid-epigastrium radiating to the back; usually relieved by eating or by taking alkali
- Localized epigastric tenderness on palpation
- P**yrosis**(heartburn)
- Vomiting (rare in duodenal ulcer); often follow severe pain or bloating
- Constipation or diarrhea
- Bleeding manifested as hematemesis or malena stool
- Vomiting may or may not be preceded by nausea;

Photo 7: Deep peptic ulcer: From Porth, C. (2002). In Smeltzer, S.C., Brenda, B. (2006). Brunner and Suddhart's Textbook of Medical-Surgical Nursing, 10th edition. Lippincott-William & Wilkins

Diagnostic Findings

A physical examination may reveal pain, epigastric tenderness, or abdominal distention.

Upper GI endoscopy; the diagnostic test: allows direct visualization of inflammatory changes, ulcers, and lesions.

Biopsy of the gastric mucosa for histology to exclude malignancy
Stool examination for blood.

Gastric secretory studies; to diagnose achlorhydria and ZES
Gastric juice culture and serologic tests to identify H. pylori infection.

Management

Pharmacologic Therapy

- Antibiotics to eradicate *H. pylori*
- Proton pump inhibitors to stop acid secretion from the parietal and oxynthic cells of the gastric mucosa linning.
- Bismuth salts
- Histamine 2 (H2) receptor antagonists; Maintained usually for 1 year
- Adherence to the medication regimen is vital to ensure complete healing of the ulcer.
- Rest should be advocated for
- Sedatives and tranquilizers to promote rest
- Octreotide (Sandostatin) may be used in ZES to suppresses gastrin levels
- Cytoprotective agents like misoprostol and sucralfate to prevent upper GI hemorrhage. Frequent gastric aspiration to monitor gastric secretion pH

Stress Reduction and Rest

- Reducing environmental stress requires physical and psychological modifications
- Help patient identify and manage stressful situations
- Encourage regular rest periods during the day and during the acute phase of the disease. Biofeedback, hypnosis, or behavior modification may be helpful.
- Strongly encouraged to stop smoking.

Dietary Modification

- Avoid extremes of temperature in meals

- Avoid stimulants such as meat extracts, alcohol, coffee (including decaffeinated coffee) and other caffeinated beverages as well as diets rich in milk and cream
- Also avoid mechanically irritating food like dry foods, smoked foods and hard food stuffs
- Also, gas forming foods should be avoided especially late into the night
- Encourage patient to eat three regular meals a day
- Small, frequent feedings are not necessary as long as an antacid or a histamine blocker is taken.
- Encourage patient to eat foods that can be tolerated and avoids those that produce pain.

Surgical Management

Usually recommended for patients with;

- Intractable ulcers (those that fail to heal after 12 to 16 weeks of medical treatment)
- Life-threatening hemorrhage
- Perforation
- Obstruction
- ZES not responding to medications

Surgical procedures include

- Vagotomy with or without pyloroplasty
- The Billroth I and Billroth II procedures.

Conditions for relapse;

- Smoking
- Coffee (including decaffeinated coffee) and other caffeinated beverages ingestion
- Alcohol
- Ulcerogenic medications (e.g., NSAIDs).

Complications

- Perforation
- Peritonitis
- Shock
- Acute renal failure
- Bleeding
- Gastric outlet obstruction

3.3 Gastric Cancer

Malignancy of the gastric mucosa

Most of these deaths occur in people older than 40 years of age

Men have a higher incidence of gastric cancers than women do.

Predisposing factors;

- Diet: smoked foods and low in fruits and vegetables may increase the risk.
- Chronic inflammation of the stomach
- Pernicious anemia
- Achlorhydria
- Gastric ulcers
- *Chronic H. pylori* infection
- Genetics predilection

Prognosis is poor, because of late presentation and diagnosis

Pathophysiology features

- Most gastric cancers are adenocarcinomas
- They can occur in any portion of the stomach
- Local infiltration of the adjacent mucosa, stomach wall and adjacent organs and structures such as the liver, pancreas, esophagus, and duodenum is possible (regional metastasis). This is via contiguous spread of malignant cells
- Distant metastasis may occur via the blood and lymphatics in the later disease.

Clinical Manifestations

- Asymptomatic in the early stages of the disease
- Early symptoms are often vague because most tumors hide on the lesser curvature
- These may include;

o Pain resembling those of benign ulcers; relieved with antacids.

- Symptoms of progressive disease may include;

o Anorexia
o Dyspepsia (indigestion)
o Weight loss
o Abdominal pain
o Constipation
o Anemia
o Nausea and vomiting.
o Ascites (due to liver metastasis).

Diagnostic Findings

- Endoscopy for biopsy
- Cytologic washings is the usual diagnostic study
- A barium x-ray examination of the upper GI tract
- A computed tomography (CT) scan

- Bone scan
- Liver scan
  - Valuable to determine the extent of metastasis
- A complete x-ray of the GI tract should be performed for any person older than 40 years old.

Management

- Surgical management: may be used as curative or palliative therapy to relieve discomfort
  - Tumor resection: indicated for localized tumors
  - Regional metastasis to vital organs like the liver will require palliative rather than curative surgical resection. Effective palliation will reduce discomfort
  - Radical subtotal gastrectomy with anastomosed to the jejunum
  - Total gastrectomy; with anastomosis between the ends of the esophagus and the jejunum.
- Chemotherapy; for further control of the disease or palliation: Commonly used chemotherapeutic medications include cisplatin, irinotecan, or a combination of 5-fluorouracil, doxorubicin (Adriamycin), and mitomycin-C.
- Radiation therapy: useful for palliation.
- Assessment of tumor markers (blood analysis for antigens indicative of colon cancer) such as carcinoembryonic antigen, CA 19-9, and CA 50 may help determine the effectiveness of treatment.

Relevant nursing diagnoses:

i. Anxiety related to treatment
ii. Imbalanced nutrition, less than body requirements, related to inadequate intake of nutrients
iii. Risk for imbalanced fluid volume related to insufficient fluid intake and excessive fluid loss subsequent to vomiting
iv. Deficient knowledge about dietary management and disease process
v. Acute pain related to irritated stomach mucosa.

Relevant nursing outcomes:

i. Anxiety reduction
ii. Nutritional enhancement
iii. Fluid enhancement
iv. Knowledge enhancement
v. Pain relief.

4.0 SUMMARY

Now that you have gone through this module, you should be able to:

i. Identify diseases affecting the stomach and the duodenum.
ii. Discuss the Pathophysiology of these conditions.
iii. Care for the patients with disorders of the stomach and duodenum utilizing the nursing process.

Activities
Visit a health facility close to you, identify a patient with a stomach or duodenal disorder, and manage him utilizing the nursing process. You will be expected to share your report on the discussion platform with the other group members.

SELF-ASSESSMENT EXERCISE

i. Identify some of the disorders of the stomach and the duodenum
ii. Discuss the Pathophysiology of peptic ulcer disease.
iii. Discuss the nursing management of a patient with peptic ulcer disease.
iv. Discuss other treatment modalities available for the management of peptic ulcer disease.

5.0 REFERENCES/FURTHER READING

Levey, R., Williams-Wilson, B. (2002).Anorexia Nervosa: eMedicine Journal, April 2 2002, Volume 3, Number 4.

Smeltzer, S.C., Brenda, B. (2006). Brunner and Suddhart's Textbook of Medical-Surgical Nursing, 10th edition. Lippincott-William & Wilkins.

Templer, J. W. (2001). Parotitis; eMedicine journal; July, 2001, Vol. 2, Number, 7.

UNIT 6 CARING FOR PATIENT WITH INTESTINAL MOTILITY AND ABSORPTION

CONTENTS

1.0 INTRODUCTION

This unit will focus on caring for patients with disorders affecting intestinal motility and absorption of ingested nutrients from the gastrointestinal tract.

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- define diarrhoea
- define constipation
- differentiate between diarrhoea and constipation
- discuss all the causes of intestinal motility and absorption
- manage intestinal motility and absorption, using the nursing process approach
- discuss various complications of intestinal motility and absorption.

3.0 MAIN CONTENT

3.1 Diarrhea

Diarrhea can be defined as;

- Increased frequency of bowel movements (more than three per day)
- Increased amount of stool (more than 200g per day)
- Altered consistency (i.e., looseness) of stool

It is usually associated with urgency, perianal discomfort, incontinence or a combination of these factors.

Causes:
It often follow any conditions that

- Increases intestinal secretions
- Decreases mucosal absorption
- Alters GI motility

Actual causes include;

- Medications (e.g., thyroid hormone replacement, stool softeners and laxatives, antibiotics, chemotherapy, antacids)
- Certain tube feeding formulas
- Metabolic and endocrine disorders (e.g., diabetes, Addison's disease, thyrotoxicosis)
- Infectious processes (e.g., dysentery, shigellosis, food poisoning and Acquired immunodeficiency syndrome- AIDS)
- Nutritional and malabsorptive disorders (e.g., celiac disease)
- Anal sphincter incompetence
- Zollinger-Ellison syndrome
- Paralytic ileus
- Intestinal obstruction
- Irritable bowel syndrome (IBS)
- Inflammatory bowel disease (IBD)
- Lactose intolerance.

Classifications;
Diarrhea can be acute or chronic.

- *Acute diarrhea;* often associated with infection and is usually self-limiting.
- *Chronic diarrhea;* persists for a longer period and may return sporadically.

Types of diarrhea;

- *Secretory diarrhoea;* usually high-volume diarrhea and is caused by increased production and secretion of water and electrolytes by the intestinal mucosa into the intestinal lumen.
- *Osmotic diarrhoea;* occurs due to transudation of water into the intestines by the osmotic forces exerted by hypertonic unabsorbed particles
- *Mixed diarrhoea;* is caused by increased peristalsis (usually from IBD) and a combination of increased secretion and decreased absorption in the bowel.

Diarrhea is a physiologic defence to eradicate offensive particles from the GIT and operates with positive feed-back mechanism.

Clinical Manifestations

- Increased frequency and fluid content of stools
- Abdominal cramps
- Abdominal distention
- Intestinal rumbling (i.e., borborygmus)
- Anorexia
- Thirst
- Painful spasmodic contractions of the anus and ineffectual straining (i.e., tenesmus)
- Symptoms related to dehydration and fluid and electrolyte imbalances.

Stool characteristics;

- Watery stools are characteristic of small bowel disease
- Loose, semisolid stools are associated often associated with disorders of the colon.
- Voluminous, greasy stools suggest intestinal malabsorption
- Presence of mucus and pus suggests inflammatory enteritis or colitis
- Oil droplets are almost always diagnostic of pancreatic insufficiency
- Nocturnal diarrhea may be a manifestation of diabetic neuropathy.

Diagnostic Findings

- Complete blood cell count
- Urinalysis
- Stool examination and analysis for blood or fat
- Stool microbiology for infectious or parasitic organisms, bacterial toxins,
- Serum Electrolytes.
- Endoscopy or barium enema may assist in identifying the cause.

3.2 Complications

- Potential for cardiac dysrhythmias (i.e., atrial and ventricular tachycardia, ventricular fibrillation, and premature ventricular contractions) due to significant electrolyte loss (especially potassium)
- Shock
- Acute renal failure.

Management

The goal of management is directed at;

- Controlling symptoms
- Preventing complications
- Eliminating or treating the underlying disease.

Measures of management include:

- *Fluid and electrolyte replacement;* is vital and should be cautiously done in extremes of age and coma: it is electrolyte repletion that is more important, as patient will drink water automatically
- *Anti-motility agents;* such as diphenoxylate, loperamide (Imodium) but contraindicated in mucosal inflammation. Its side effects - abdominal cramps, toxic megacolon
- Absorbents such as kaolin/pectin (Kaopectate), methylcellulose, activatedattapulgite: they act by absorbing intestinal toxins / micro-organsims, or by coating / protecting intestinal mucosa. They are much less effective than anti-motility agents
- *Modifiers of fluid transport;* may be helpful for example, bismuth subsalicylate (Pepto-Bismol)
- *Antibiotics;* rarely indicated unless bacteria agency have been implicated. The attendant risk include;

o Prolonged excretion of enteric pathogen
o Drug side effects (including C. *difficile*)
o Development resistant strains

Nursing Management

- Assess and monitor the characteristics and pattern of diarrhea.
- Abdominal auscultation and palpation for bowel activities and abdominal tenderness respectively
- Mucous membranes and skin evaluation for hydration status
- Stool examination and samples are obtained for testing.
- Strict fluid intake and output
- Bed rest during acute episodes
- Avoid GI stimulants such as caffeine, carbonated beverages and extremes of food temperatures, because they stimulate intestinal motility.
- Restrict milk products, fat, whole-grain products, fresh fruits and vegetables for several days.
- Administers anti-diarrheal medications such as diphenoxylate (Lomotil) and loperamide (Imodium) as prescribed and watch for side effects
- Intravenous fluid therapy may be necessary for rapid rehydration, especially for the elderly and those with preexisting GI conditions (e.g., IBD).

- Closely monitoring of serum electrolyte levels
- Identify and at once report evidence of dysrhythmias or a change in the level of consciousness.
- Perianal area hygiene with skin sealants and moisture barriers especially in older folks to avoid excoriation

3.3 Irritable Bowel Syndrome (IBS)

Definition; a form of functional bowel disease

Epidemiology

Affects30% of North Americans with onset of symptoms usually in young adulthood

Affects more Females than the Male

Causes;

IBS results from a functional disorder of intestinal motility.

- Neurologic regulatory system dysfunction
- Infection or irritation
- Vascular disturbance
- Metabolic disturbance.

Diagnosis

"Rome Criteria"

- At least three months of continuous or recurrent symptoms of
  - Abdominal pain or discomfort which is relieved by defecation and /or
  - Associated with a change in stool frequency and/or
  - Associated with a change in stool consistency plus two or more of the following, at least 25% of the time which may be inform of;
    - Altered stool frequency
    - Altered stool form (lumpy/hard or loose/watery)
    - Altered stool passage (straining, urgency, or feeling of incomplete evacuation)
    - Passage of mucus
    - Bloating or feeling of abdominal distention
- Absence of negative features such as;
  - Weight loss
  - Nocturnal defecation
  - Blood or pus in stool
  - Fever
  - Anemia
  - Abnormal gross findings on flexible sigmoidoscopy.

Normal physical exam

Investigations; may include;

- Complete Blood Count
- ESR
- Stool for Culture & Sensitivity and fat excretion
- Sigmoidoscopy

Management
No therapeutic agent effective
Over 50% of cases will improve with time
The patient will require reassurance
Administer bran or psyllium for constipation
Loperamide may be required to manage diarrhea
Symptom - guided treatment; should include;

- Pain predominant variant will require;
  - Change diet (anticholinergic diet)
  - Tricyclic compounds
  - Visceral antinociceptive agent
  - Selective serotonin reuptake inhibitors (SSRI)
  - NSAID
- Diarrhea predominant variant will require;
  - Change diet
  - Loperamide
  - Diphenoxylate
  - Cholestyramine
- Constipation predominant variant;
  - High fibre diet
  - Osmotic or other laxatives.

3.4 Fecal Incontinence

The term *fecal incontinence* describes the involuntary passage of stool from the rectum.
Several factors influence fecal continence;

- The ability of the rectum to sense and accommodate stool
- The amount and consistency of stool
- The integrity of the anal sphincters and musculature, and rectal motility

Causes
Fecal incontinence can result from;

- Trauma (e.g., after surgical procedures involving the rectum)
- Neurologic disorder (e.g., stroke, multiple sclerosis, diabetic neuropathy, dementia), inflammation, infection, radiation treatment, fecal impaction, pelvic floor relaxation, laxative abuse, medications, or advancing age (i.e., weakness or loss of anal or rectal muscle tone)
- Structural abnormalities such as fistula in ano

- Tumors

It is an embarrassing problem that requires a multi-disciplinary approach to treatment.

Clinical Manifestations

- Minor soiling of clothes with fecal matter
- Occasional urgency
- Loss of control of defecation (complete incontinence).
- Poor control of flatus
- Diarrhea or constipation.

Diagnostic Findings

A rectal examination

Endoscopic examinations such as a flexible sigmoidoscopy to rule out tumors, inflammation, or fissures

X-ray studies such as barium enema, computed tomography (CT) scans, anorectalmanometry, and transit studies; may help identify alterations in intestinal mucosa and muscle tone or in detecting other structural or functional problems.

Management

- No known cause or cure for fecal incontinence
- Conservative care;
  - Treat underlying causes such as diarrhea, fecal impaction
  - Biofeedback therapy; to help increase anal sphincter tone
  - Bowel training programs can also be effective.
  - Surgical procedures include surgical reconstruction, sphincter repair, or fecal diversion.

Nursing Management

- A thorough health history, including information about previous surgical procedures, chronic illnesses, bowel habits and problems, and current medication regimen
- Complete examination of the rectal area.
- Initiate a bowel-training program (setting a schedule to establish bowel regularity)
- Use suppositories to stimulate the anal reflex
- Use biofeedback to help improve sphincter contractility and rectal sensitivity.
- Maintaining peri-anal skin integrity especially in the debilitated or elderly patient through meticulous skin hygiene
- Assist the patient and family in accepting and coping with this chronic situation in case continence cannot be re-established.

- Advise patients to use fecal incontinence devices such as external collection devices (special drainable pouches) and internal drainage systems (a large catheter system inserted into the rectum and connected to a drainage system) which is better in eliminating fecal skin contact and preventing excoriation or skin breakdown.

3.5 Conditions of Malabsorption

Malabsorptionis the inability of the digestive system to absorb one or more of the major vitamins (especially vitamin B12), minerals (i.e., iron and calcium), and nutrients (i.e., carbohydrates, fats, and proteins)
The conditions that cause malabsorption can be grouped into the following categories:

- Mucosal (transport) disorders causing generalized malabsorption (e.g., celiac sprue, regional enteritis, radiation enteritis)
- Infectious diseases causing generalized malabsorption (e.g., small bowel bacterial overgrowth, tropical sprue, Whipple's disease)
- Luminal problems causing malabsorption (e.g., bile acid deficiency, Zollinger-Ellison syndrome, pancreatic insufficiency)
- Postoperative malabsorption (e.g., after gastric or intestinal resection)
- Disorders that cause malabsorption of specific nutrients (e.g., disaccharidase deficiency leading to lactose intolerance)

Clinical Manifestations

- Diarrhea or frequent, loose, bulky, foul-smelling stools (due to high fat content)
- Abdominal distention
- Abdominal pain
- Increased flatus
- Weakness,
- Weight loss.

Diagnostic Findings

- Stool analysis for quantitative and qualitative fat analysis, lactose tolerance tests, D-xylose absorption tests, and Schilling tests
- Hydrogen breath test to evaluate carbohydrate absorption
- Endoscopy with biopsy of the mucosa; to identify infection or destruction of mucosa
- Small intestine enzymatic assay
- Ultrasound studies
- CT scans
- Plain X-ray findings
- A complete blood cell count is used to detect anemia.

- Pancreatic function tests can assist in the diagnosis of specific disorders.

Management

Goal of management is aimed at;

Avoiding dietary substances that aggravate malabsorption and

- Supplementing nutrients that have been lost

Common supplements are;

- Water-soluble vitamins (e.g., B12, folic acid)
- Fat-soluble vitamins (i.e., A, D, and K)
- Folic acid supplements for tropical sprue
- Minerals (e.g., calcium, iron)
- Primary disease states may require non-surgical management;
- Reducing gluten intake for celiac sprue
- Antibiotics (e.g., tetracycline, ampicillin) are sometimes needed to treat bacterial overgrowth syndromes.
- Antidiarrheal agents; to decrease intestinal spasms
- Parenteral fluids may be necessary to treat dehydration
- Patient and family education on diet and the use of nutritional supplements
- Monitor patients with diarrhea for fluid and electrolyte imbalances.

Complications

- Malnutrition evidenced by weight loss and other signs of vitamin and mineral deficiency (e.g., easy bruising, osteoporosis, anemia)
- Dehydration
- Avitaminlosis due to failure to absorb the fat-soluble vitamins A, D, E and K

Relevant nursing diagnoses

i. Acute diarrhoea
ii. Constipation
iii. Fluid volume deficit related fluid loss (Diarrhoea)
iv. Acute pain related to inflammatory bowel condition
v. Risk for impaired (peri-anal) skin integrity related to diarrhoea
vi. Risk for infection related to skin breakdown
vii. Situational low self esteem related to diarrhoea.

Relevant nursing outcomes

i. Fluid and electrolyte conservation
ii. Pain relief
iii. Skin management
iv. Infection control
v. Self esteem enhancement

4.0 SUMMARY

At the end of this unit, you should be able
i. Define diarrhoea
ii. Define constipation
iii. Differentiate between diarrhoea and constipation
iv. Discuss all the causes of intestinal motility and absorption
v. Manage intestinal motility and absorption, using the nursing process approach
vi. Discuss various complications of intestinal motility and absorption.

Activities
Before now in the event of clinical practice, you would have come across or even managed a case of diarrhoea, compare your knowledge of diarrhoeal management then and now that you have completed this unit. Share your report with others in the discussion forum.

SELF-ASSESSMENT EXERCISES

i. Distinguish between diarrhoea and constipation
ii. Discuss the causes of diarrhoea
iii. Using the nursing process, manage a patient with diarrhoea considering the actual and potential consequences of the condition.

Note that, you are free to evaluate yourself with the content of the unit and other reference materials.

5.0 REFERENCES/FURTHER READING

Levey, R., Williams-Wilson, B. (2002). Anorexia Nervosa: eMedicine Journal, April 2 2002, Volume 3, Number 4.

Smeltzer, S.C., Brenda, B. (2006). Brunner and Suddhart's Textbook of Medical-Surgical Nursing, 10th edition. Lippincott-William & Wilkins.

Templer, J. W. (2001). Parotitis; eMedicine journal; July, 2001, Vol. 2, Number, 7.

UNIT 7 CARING FOR PATIENT WITH ACUTE INFLAMMATORY INTESTINAL DISORDERS

CONTENTS

1.0 INTRODUCTION

This unit will focus on caring for patients with acute inflammation of the intestines.

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- identify various types of inflammatory intestinal disorders
- discuss the Pathophysiology of the different types of acute inflammatory intestinal disorders
- care for patients with acute inflammatory intestinal disorders utilizing the nursing process approach.

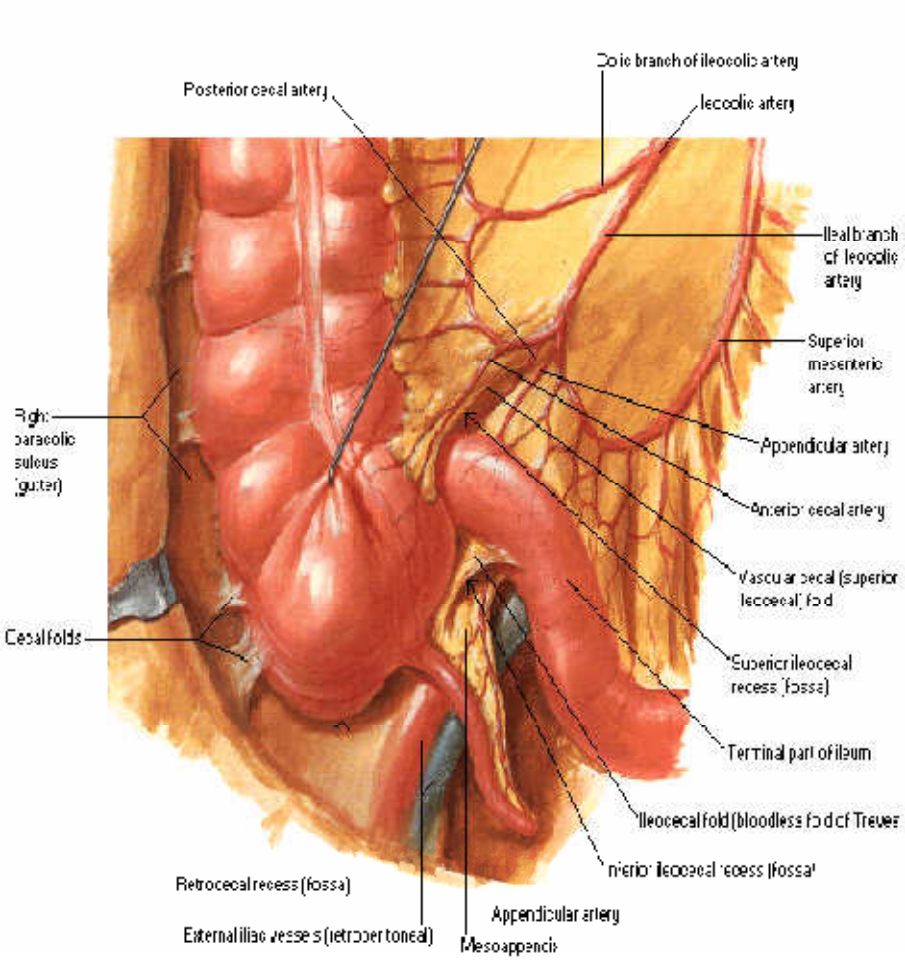


Photo 8: Diagram showing the Vermiform appendix

The appendix is a small, finger-like appendage that is attached to the cecum just below the ileocecal valve. It regularly fills with food and empties inefficiently but regularly into the cecum, thus prone to obstruction and subsequent infection (i.e., appendicitis).

Appendicitis, is the inflammation of the vermiform appendix
It is a common disease and also a common indication for laparatomy
Males are affected more than females and teenagers more than adults.

Pathophysiology
Inflamed and edematous appendix will become kinked or occluded by a fecalith (i.e., hardened mass of stool), tumor, or foreign body, with subsequent increases in the intra-luminal pressure. The inflamed appendix may start suppurating

Clinical Manifestations

- Vague epigastric or peri-umbilical pain progresses to right lower quadrant pain
- Low-grade fever
- Nausea and sometimes by vomiting

- Loss of appetite
- Local tenderness at McBurney's point
- Rebound tenderness (i.e., production or intensification of pain when pressure is released)
- Rovsing's sign; palpating the left lower quadrant elicits pain at the right lower quadrant
- Constipation

Diagnostic Findings

- Complete physical examination
- Complete blood cell count; demonstrates leucocytosis (elevated white blood cell count) more than 10,000cells/mm_3 and neutrophil count may exceed 75%
- Abdominal x-ray films
- Ultrasound studies
- CT scans; all may reveal a right lower quadrant density or localized distention of the bowel.

Complications

- Perforation of the appendix: A fairly common phenomenon which occurs more readily among children and the elderly. It occurs 24 hours after the onset of pain. Symptoms include a fever of 37.7°C (100°F) or higher, a toxic appearance, and continued abdominal pain or tenderness.
- Peritonitis or an abscess
- Acute appendicitis seldom occur in the elderly; when it does, the symptoms are vague. Pain is absent or minimal, thus diagnosis and treatment is often delayed, causing potential complications and mortality. Some are asymptomatic until the appendix ruptures.

Management

- Correct or prevent fluid and electrolyte imbalance and dehydration
- Antibiotics
- Intravenous fluids
- Analgesics
- Surgery is the definitive management: Appendectomy (i.e., surgical removal of the appendix) performed as soon as possible to decrease the risk of perforation. It may be performed under a general or spinal anesthetic with a low abdominal incision or by laparoscopy.

Nursing Management

Goals include;

- Analgesia (relieving pain)
- Preventing fluid volume deficit
- Reducing anxiety
- Eliminating infection from the potential or actual disruption of the GI tract
- Maintaining skin integrity
- Attaining optimal nutrition
- Surgical preparations: includes;
- Intravenous infusion to replace fluid loss and promote adequate renal function
- Antibiotic therapy to prevent infection.
- NG tube in case of evidence or likelihood of paralytic ileus
- Avoid an enema as it can lead to perforation.

Post-operative care;

- Place the patient in a semi-Fowler position; to reduce tension on the incision
- An opioid, usually morphine sulfate, is prescribed to relieve pain.
- Oral fluids are administered when it can be tolerated.
- Intravenous fluids dehydrated individuals
- Food is provided as desired and tolerated on the day of surgery.
- The patient may be discharged on the day of surgery if fever and pains have been well controlled
- Discharge teaching; should include;
  - Keeping to an appointment with the surgeon remove the sutures between the fifth and seventh days after surgery
  - Incision care and activity guidelines
  - Normal activity can usually be resumed within 2 to 4 weeks.

3.2 Peritonitis

Peritonitis is inflammation of the peritoneum
The peritoneum is the serous membrane lining the abdominal cavity and covering the viscera.

Causes/ predisposing factors

- Bacterial infection;
  - Endogenously from;
    - The GI tract
    - Internal reproductive organs in women
  - Exogenous infection may follow;
    - Trauma (e.g., gunshot wound, stab wound)
    - Contiguous spread from an inflammation at the adjacent organ outside the peritoneal area, such as the kidney.

The most common bacteria implicated are *Escherichia coli*, *Klebsiella, Proteus,* and *Pseudomonas.*

- Appendicitis
- Perforated ulcer
- Diverticulitis
- Bowel perforation
- Abdominal surgical procedures
- Peritoneal dialysis.

Pathophysiology

Peritonitis often is due to leakage of visceral contents into the abdominal cavity and consequent inflammation, infection, ischemia, trauma, or tumor perforation.

This is followed by bacterial proliferation which worsens the inflammation. Tissue edema cause exudation thus fluid accumulates within n the peritoneal cavity. This fluid is turbid with high protein, white blood cells, cellular debris and blood elements. This resultant fluid is hypertonic and cause more transudation into the peritoneal cavity.
Immediate response of the intestinal tract is hypermotility and soon paralytic ileus with an accumulation of air and fluid in the bowel.

Clinical Manifestations

- Pain: constant, localized, and more intense near the site of the inflammation. Aggravated by movement
- Generalized abdominal tenderness
- Abdominal distension
- Abdominal rigidity
- Rebound tenderness
- Paralytic ileus
- Nausea and vomiting
- Diminished peristalsis and bowel sounds
- Fever and tachycardia
- Leucocytosis

Diagnostic Findings

- CBC with differential leukocyte count
- The hemoglobin and hematocrit levels may be low if bleeding has occurred.
- Serum electrolyte studies may reveal altered levels of potassium, sodium, and chloride.
- An abdominal x-ray; may show air and fluid levels as well as distended bowel loops.

- A CT scan of the abdomen may show abscess formation.
- Peritoneal aspiration and culture and sensitivity studies to isolate infectious organisms

Medical Management

- Fluid, colloid, and electrolyte replacement; the core focus of management
- Analgesics: for pain
- Anti-emetics for nausea and vomiting
- Intestinal intubation and suction to relieve abdominal distention and promote intestinal function
- Oxygen therapy by nasal cannula or mask can promote adequate oxygenation, but airway intubation and ventilator assistance occasionally are required.
- Massive antibiotic therapy; with large doses of a broad-spectrum antibiotic
- Surgical management: aims to remove the infected material and correcting the cause. Approaches include;
  - Surgical excision (i.e., appendix)
  - Surgical resection with or without anastomosis (i.e., intestine)
  - Surgical repair (i.e., perforation)
  - Surgical drainage (i.e., abscess)
  - A fecal diversion may be required in presence of extensive sepsis

Nursing Management

- Ongoing assessment of pain, vital signs, GI function, and fluid and electrolyte balance
- Administration of analgesic medication
- Positioning the patient for comfort; place patient on the side with knees flexed; this position decreases tension on the abdominal organs.
- Accurate recording of all intake and output and central venous pressure assists in calculating fluid replacement.
- The nurse administers and monitors closely intravenous fluids.
- Encourage oral feeds only when active disease subsides: signs of subsidence include;
  - Resolution of fever and tachycardia
  - Softening of the abdomen
  - Evident peristaltic sounds/ bowel movement (passing of flatus)
- Increases fluid and food intake gradually and reduce par-enteral fluids as prescribed.
- Prepare the patient for emergency surgery with evidence of worsening of clinical condition (which may indicate a complication)
- Drains in the surgical wound must be observed

- Record the character of the drainage postoperatively
- Avoid dislodgement of drains while moving and turning the patient.

Complications
- Generalized sepsis: the major cause of death from peritonitis
- Shock may result from septicemia or hypovolemia
- Intestinal obstruction primarily due to bowel adhesions
- Post- operative wound evisceration
- Post- operative abscess formation.

3.3 Inflammatory Bowel Disease (IBD)

This refers to Crohn's disease and Ulcerative Colitis (UC)

Etiology
This is less understood than most other diseases
Perhaps due to chronic infection by undetectable organism with subsequent inappropriate immune attack on normal mucosal bowel flora.

3.4 Crohn's Disease

Definition
This is a chronic inflammatory disorder that affects the small intestine and/or large intestine
Epidemiology
- It is bimodal: onset before 30 years and second peak age 60years
- Its incidence has been increasing (relative to UC) especially in young females and more common in Caucasians, Ashkenazi Jews but less reported in Africa.

Pathology
It may affect any part of GI tract from mouth to anus with evidence of trans-mural inflammation with “skip" lesions associated with granulomas and deep fissuring/aphthous ulcerations, strictures. It may also present linear ulcers leading to mucosal islands and “cobble-stoning"
Also deep fissures with risk of perforation into contiguous viscera (leads to fistulae and abscesses). Resultant enteric fistulae may communicate with skin, bladder, vagina, and other parts of bowel.

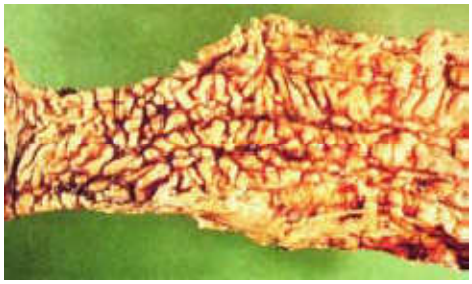


Photo 9: Crohn's disease: The mucosal surface of the colon displays a "cobblestone" appearance owing to the presence of linear ulcerations and edema and inflammation of the intervening tissue. (From Rubin E., Farber J.L. [1999]. Pathology [3rd ed., p. 728]. Philadelphia: Lippincott Williams & Wilkins)

Signs and Symptoms

- Most common presentation include recurrent episodes of mild diarrhea (more common with involvement of colon), abdominal pain, and fever
- Ileitis may present with post-prandial pain, vomiting, Right Lower

Quadrant mass, acute appendicitis

- Fistulas, fissures, abscesses are common
- Extra-intestinal manifestations are more common with colonic involvement

Investigations

- Endoscopy with biopsy to diagnose
- Barium studies
- Bacterial cultures, C. *difficile* toxin to exclude other causes of inflammatory diarrhea.

Management

Most uncomplicated cases can be managed medically with drugs such as;

- Sulfasalazine or metronidazole to treat mild disease
- Steroids; prednisone 20-40 mg OD for acute exacerbations (but use only if symptoms are severe). However, no proven role for steroids in maintaining remissions and may masks intra-abdominal sepsis. Complications of steroid therapy include insomnia, emotional lability, weight gain/enhanced appetite, hypertension, diabetes, PUD, acne, Cushing's habitus, impaired wound healing, adrenal suppression, infection diathesis, osteonecrosis, myopathy, osteoporosis, skin atrophy, cataracts, atherosclerosis, growth retardation, fatty liver, glaucoma, pseudotumourcerebri
- Immunosuppressives (6-mercaptopurine, azathioprine); are used chiefly as steroid-sparing agents and requires > 3 months to have

beneficial effect which may include helping to heal fistulae, decreased disease activity. Side effects include pancreatitis, bone marrow suppression, increased risk of cancer

- Diet: elemental diets help remit acute Crohn's disease but are not palatable: Extensive small bowel involvement will need electrolyte, mineral and vitamin supplements
- Antidiarrheal agents:
  - Loperamide (Imodium); Diphenoxylate (Lomotil); Codeine (cheap but addictive); all work by decreasing small bowel motility but should be used with caution
  - Cholestyramine; is a bile salt binding resin, indicated for watery diarrhea with less than 100 cm of terminal ileum diseased or resected
- Immunomodulators; e.g. Infliximab (antibody to TNF α); proven effective for treatment of fistula and current trials are favourable for patients with Crohn's disease.

Surgical treatment:ileal resection;

- Generally reserved for complications such as fistulae, obstruction, abscess, perforation, bleeding and rarely for medically refractory disease.
- Complications of ileal resection will include.
- Watery diarrhea (due to impaired bile salt absorption). Treatment is with cholestyramine.
- Steatorrhea (due to bile salt deficiency). Treatment is by fat restriction.

Complications

- Intestinal obstruction due to edema, fibrosis.
- Fistula formation.
- Intestinal perforation (uncommon in Crohn's).
- Malignancy - increased risk, but not as high as ulcerative colitis.

Ulcerative Colitis (UC)

Definition

It is a chronic inflammatory disease affecting colonic mucosa from rectum to cecum, characterized by rectal bleeding and diarrhea and prone to remissions and exacerbations.

Epidemiology

Majority of cases starts by age 30 (with second peak after 50).
Both genders are equally affected
There is a small hereditary contribution.

Pathology

The disease can affect any portion of the lower bowel ranging from rectum only (proctitis) to entire colon (pancolitis). More commonly affects the rectum. Inflammation often is diffuse and confined to mucosa

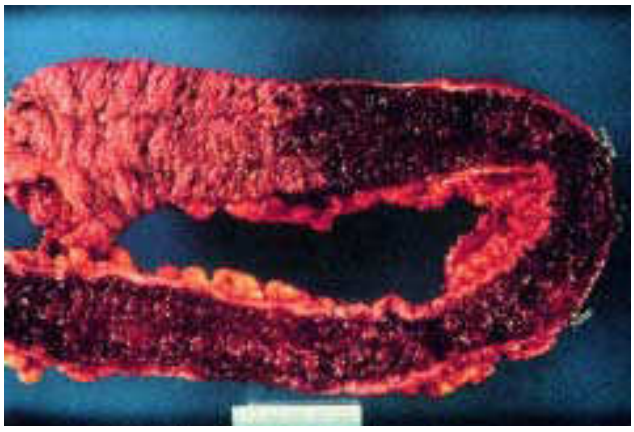


Photo 10: Ulcerative colitis: Prominent erythema and ulceration of the colon begin in the ascending colon and are most severe in the rectosigmoid area. (From Rubin E., Farber J.L. [1999]. Pathology [3rd ed., p. 731]. Philadelphia: Lippincott Williams & Wilkins).

Signs and Symptoms

There is a direct relationship between the extent of the disease and severity of symptoms. They include;

- Diarrhea
- Rectal bleeding most frequent
- Abdominal cramps/pain (especially with defecation).
- Tenesmus
- Urgency
- Incontinence
- Systemic symptoms include:

o Fever
o Anorexia
o Weight loss
o Fatigue

Investigations

- Sigmoidoscopy without bowel prep to diagnose
- Colonoscopy: contraindicated in severe exacerbation
- Barium enema: not during acute phase or relapse.

Both determine length of bowel involved

- Stool cultures to exclude infection
- Mucosal biopsy: to exclude acute self-limited colitis.

Management

Mainstays of treatment:

- 5-ASA drugs; e.g. sulfasalazine (Salazopyrin). They act by blocking arachidonic acid metabolism to prostaglandins and leukotrienes.
- It can be used as a topical preparation (in form of enema, suppository) and Oral preparations (in form of a capsule to delay absorption).
- Steroids: (e.g. prednisone 40 mg daily); best drugs to remit acute disease, especially if severe or first attack. It can be used as suppositories for proctitis or enemas for proctosigmoiditis.
- Immunosuppressants (steroid sparing): e.g. Azathioprine: often used in severe Ulcerative Colitis resistant to steroid therapy.

Surgical treatment:

- Indicated for fulminant cases and toxic megacolon
- The surgical procedure performed is Colectomy;
- Indications include;
  - Failure of adequate medical therapy
  - Toxic megacolon
  - Bleeding
  - Pre-cancerous changes picked up with screening endoscopic biopsies (dysplasia).

Complications

- Liver problems (especially primary sclerosing cholangitis in men).
- Increased risk of colorectal cancer: risk increases with duration and extent of disease and presence of sclerosing cholangitis, sialosye-Tn antigen in mucosal biopsy.
- Toxic megacolon (transverse colon diameter > 6 cm on abdominal x-ray) with immediate danger of perforation.

Relevant Nursing Diagnoses

Based on the assessment data, the nursing diagnoses may include the following:

i. Constipation related to narrowing of the colon from thickened muscular segments and strictures
ii. Acute pain related to inflammation and infection.

Expected Patient Outcomes

Expected patient outcomes may include the following:

i. Attains a normal pattern of elimination
   - Reports less abdominal cramping and pain
   - Reports the passage of soft, formed stool without pain
   - Adds unprocessed bran to foods

- Drinks at least 10 glasses of fluid each day (if fluid intake is tolerated)
- Exercises daily

ii. Reports decreased pain
- Requests analgesics as needed
- Adheres to a low-fiber diet during acute episodes

iii. Recovers without complications
- Is a-febrile
- Has normal blood pressure
- Has a soft, non-tender abdomen with normal bowel sounds
- Maintains adequate urine output
- Has no blood in the stool.

4.0 SUMMARY

At the end of the unit, you should be able to:

- identify various types of inflammatory intestinal disorders
- discuss the Pathophysiology of the different types of acute inflammatory intestinal disorders
- care for patients with acute inflammatory intestinal disorders utilizing the nursing process approach.

Activities in your place of practice, identify a patient with any type of inflammatory bowel disease and develop an outline of comprehensive assessment and management of the patient

SELF-ASSESSMENT EXERCISES

Now that you have studied this unit, you may attempt the following questions: you may grade yourself using the course content and reference materials.

i. outline the types of inflammatory intestinal disorders
ii. discuss the Pathophysiology of regional illitis (Crohn's disease)
iii. using the nursing process, identify and solve in order of priority, three (3) possible nursing diagnoses of a patient with inflammatory intestinal disease.

5.0 REFERENCES/FURTHER READING

Levey, R., Williams-Wilson, B. (2002).Anorexia Nervosa: eMedicine Journal, April 2 2002, Volume 3, Number 4

Smeltzer, S.C., Brenda, B. (2006). Brunner and Suddhart's Textbook of Medical-Surgical Nursing, 10th edition. Lippincott-William & Wilkins

Templer, J. W. (2001). Parotitis; eMedicine journal; July, 2001, Vol. 2, Number, 7

UNIT 8 STRUCTURAL AND OBSTRUCTIVE DISORDERS

CONTENTS

1.0 INTRODUCTION

This unit will focus on caring for patients with structural and obstructive disorders of the intestines.

2.0 OBJECTIVES

At the end of the unit, you should be able to:

- identify various types of structural and obstructive disorders of the intestines
- discuss the Pathophysiology of the different types of structural and obstructive disorders of the intestines
- care for patients with structural and obstructive disorders of the intestines utilizing the nursing process approach.

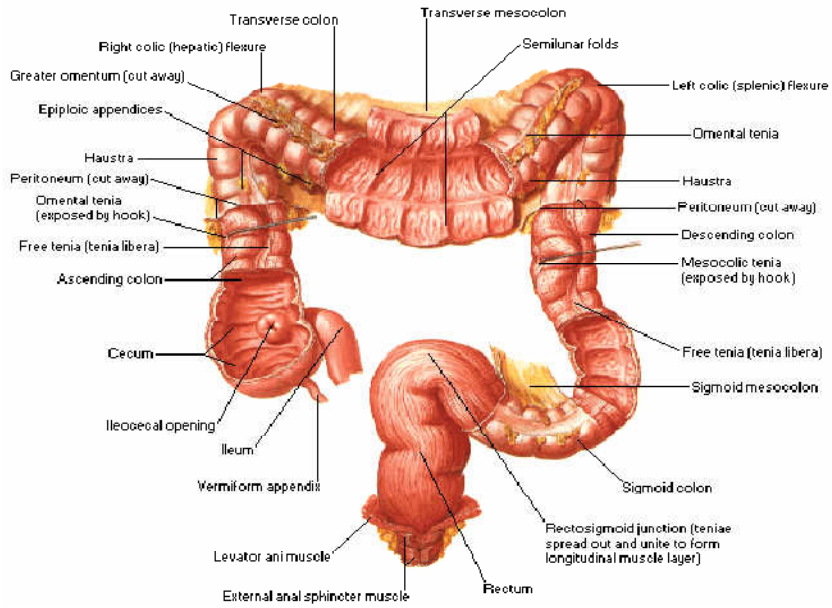


Photo 11: Pictoral presentation of the Large Intestine

3.1 Colon Cancer

Etiology/Epidemiology
Environmental influences include;
High dietary fat consumption
Low dietary fiber consumption
Genetic influences
Inherited or acquired:

Familial syndromes; individuals inherit genetic alterations that make these individuals susceptible to environmental factors in the development of colon cancer

People acquire multiple "step-wise" somatic mutations such as activation of proto-oncogenes (K-ras); loss of tumour-supressor gene activity (APC, DCC, p53) and abnormalities in DNA repair genes (hMSH2, hMLH1) - especially HNPCC syndromes **Pathophysiology**

Changes follow hyperproliferative epithelium which will result in adenoma and will eventually undergo malignant transformation (carcinoma)

Risk Factors

- Age: majority of cases occur in people > 50 years old
- Adenomatous polyps; large, villous, and moderate to severe dysplasia more likely to be cancer.
- Family history.
  - Sporadic cancer: risk increases 1.8 times for those with one affected relative, 2-6 times with two affected relatives. The risk is greater if relative has cancer diagnosed < 45 years old.
  - Familial adenomatous polyposis and Gardner's syndrome: an autosomal dominant variant due to inactivated APC gene on 5q. Over 100 of adenomatous polyps develop in colon and rectum, starting at age 15-20 years old and pose 100% risk of cancerous changes.
- Gardner's syndrome is a variant, with polyposis plus extracolonic manifestations (osteomas, soft tissue tumours, congenital hypertrophy of retinal pigmented epithelium).
- Hereditary nonpolyposis colorectal cancer (Lynch syndrome, or HNPCC): are autosomal dominant (hMSH2, hMLH1), manifested as discrete adenomas (polyposis does not occur), which occurs earlier, age 40 -50 years, often proximal in location and multiple, more commonly mucinous or poorly differentiated because no preceding polyp stage; cancer are often diagnosed late in disease.

- Inflammatory Bowel Disease (IBD): Ulcerative colitis (UC) for 10 years.

Medical Management

Intravenous fluids
Nasogastric suction
Blood component therapy may be required if bleeding has occurred
Treatment depends on the stage of the disease and consists of;
Surgery:

To remove the tumor. It may be curative or palliative: The type of surgery recommended depends on the location and size of the tumor.

- Local cancers can be removed through;
  - Colonoscopy with attendant advantages of minimizing the extent of surgery as it is a guided invasion into the colon
  - Laparoscopic colotomy with polypectomy
  - Use of the neodymium/yttriumaluminum-garnet (Nd:YAG)

Bowel resection is indicated for most class A lesions and all class B and C lesions but sometimes for class D colon cancer (as palliation in this case).

- Local metastasis to the surrounding vital structures will indicate more complex surgeries like:
  - Segmental resection with anastomosis (i.e., removal of the tumor and portions of the bowel on either side of the growth, as well as the blood vessels and lymphatic nodes).
  - Abdominoperineal resection with permanent sigmoid colostomy (i.e., removal of the tumor and a portion of the sigmoid and all of the rectum and anal sphincter).
  - Temporary colostomy followed by segmental resection and anastomosis and subsequent re-anastomosis of the colostomy, allowing initial bowel decompression and bowel preparation before resection
  - Permanent colostomy or ileostomy for palliation of un-resectable obstructing lesions.
  - Construction of a coloanal reservoir called a colonic J pouch is performed in two steps. A temporary loop ileostomy is constructed to divert intestinal flow, and the newly constructed J pouch (made from 6 to 10 cm of colon) is reattached to the anal stump. About 3 months after the initial stage, the ileostomy is reversed, and intestinal continuity is restored. The anal sphincter and therefore continence are preserved.

Supportive therapy
Adjuvant therapy such as;

- Chemotherapy; e.g. 5-fluorouracil; Mitomycin;
- Radiation therapy: can be used before, during, and after surgery to shrink the tumor

- Immunotherapy
- Multimodality therapy.

3.2 Intestinal Obstruction

Intestinal obstruction refers to blockage to the normal flow of intestinal contents through the intestinal tract.
Two types of processes can impede this flow.

Mechanical obstruction: may be;
An intra-luminal obstruction or a mural obstruction from pressure on the intestinal walls occurs. Examples are intussusception, polypoid tumors and neoplasms, stenosis, strictures, adhesions, hernias, and abscesses.
Functional obstruction: The intestinal musculature cannot propel the contents along the bowel. Examples are amyloidosis, muscular dystrophy, endocrine disorders such as diabetes mellitus, or neurologic disorders such as Parkinson's disease.

The obstruction can be partial or complete
Etiology:

- Common causes of small bowel obstruction include;
  - Adhesions
  - Hernias
  - Neoplasms
  - Intussusception
  - Volvulus (i.e., twisting of the bowel)
  - Paralytic ileus
- Common causes of large bowel obstruction are;
  - Carcinoma
  - Diverticulitis
  - Inflammatory bowel disorders
  - Benign tumors.

Pathophysiology
Intestinal contents, fluid, and gas accumulate above the obstruction. Thus reduce absorption of fluids and stimulate more gastric secretion. More distention cause more intra-luminal pressure with subsequent decrease in venous and arteriolar capillary pressure. This causes edema, congestion, necrosis, and eventual rupture or perforation of the intestinal wall, with resultant peritonitis.

Clinical Manifestations

- Crampy colicky pain
- Blood and mucus without fecal matter and no flatus
- Vomiting
- Visible peristaltic waves on the anterior abdominal wall in case of complete obstruction

- Vomitus will contain fecal matter, bile and stomach contents if obstruction is in the ileum
- Signs of dehydration such as intense thirst, drowsiness, generalized malaise, aching, and a parched tongue and mucous membranes
- Abdominal distension
- Constipation
- Loops of large bowel become visibly outlined through the abdominal wall
- Hypovolemic shock occurs from dehydration and loss of plasma volume.

Diagnostic Findings
X-ray findings: Abdominal x-ray studies show abnormal quantities of gas, fluid, or both in the bowel.

Laboratory studies (i.e., electrolyte studies and a complete blood cell count) reveal a picture of dehydration, loss of plasma volume, and possible infection.

Colonoscopy
Medical Management
- Decompression of the bowel through a nasogastric or small bowel tube
- Intravenous therapy is necessary to replace the depleted water, sodium, chloride, and potassium.
- Surgical correction in case of complete obstruction with possibility of strangulation: via colonoscopy in large bowel obstruction.
- A temporary or permanent colostomy may be necessary.
- An ileoanal anastomosis may be performed if it is necessary to remove the entire large colon.

Nursing Management
- Maintaining the function of the nasogastric tube
- Assessing and measuring the nasogastric output
- Assessing for fluid and electrolyte imbalance
- Monitoring nutritional status
- Assessing improvement (e.g., return of normal bowel sounds, decreased abdominal distention, subjective improvement in abdominal pain and tenderness, passage of flatus or stool)
- Prepare patient for surgery

3.3 Diverticular Disease

A diverticulum is a saclike out-pouching of the lining of the bowel that extends through a defect in the muscle layer.

Diverticulosis exists when multiple diverticula are present without inflammation or symptoms.
Diverticular disease of the colon is very common in developed countries, and its prevalence increases with age.

Diverticulitis results when food and bacteria retained in a diverticulum produce infection and inflammation that can impede drainage and lead to perforation or abscess formation.
Diverticulitis is most common in the sigmoid colon.
Predisposing factors:
The exact cause is unknown
A congenital predisposition
Low intake of dietary fiber.

Pathophysiology
A diverticulum forms when the mucosa and submucosal layers of the colon herniate through the muscular wall because of high intra-luminal pressure, low volume in the colon (i.e., fiber-deficient contents), and decreased muscle strength in the colon wall (i.e., muscular hypertrophy from hardened fecal masses). Bowel contents can accumulate in the diverticulum and decompose, causing inflammation and infection. A diverticulum can become obstructed and then inflamed if the obstruction continues. The inflammation tends to spread to the surrounding bowel wall, giving rise to irritability and spasticity of the colon (i.e., diverticulitis). Abscesses develop and may eventually perforate, leading to peritonitis and erosion of the blood vessels (arterial) with bleeding.

Clinical Manifestations
- Chronic constipation

Signs of acute diverticulosis are;
- Bowel irregularity
- Intervals of diarrhea
- Abrupt onset of crampy pain in the left lower quadrant of the abdomen.
- Low-grade fever
- Nausea and anorexia.
- Bloating or abdominal distention.

Subsequent narrowing of the large bowel may narrow (fibrotic strictures) due to repeated local inflammation of the diverticula will lead to;
- Cramps

- Narrow stools
- Increased constipation.

Diagnostic Findings

- A CT scan is the procedure of choice and can reveal abscesses
- Abdominal x-ray findings may demonstrate free air under the diaphragm if a perforation has occurred from the diverticulitis.
- Barium enema: may diagnose diverticulosis. This is contraindicated if peritoneal irritation is suspected
- A colonoscopy: deferred until acute phase resolve
- Laboratory tests that assist in diagnosis include a complete blood cell count (revealing an elevated leukocyte count)
- Elevated sedimentation rate.

Complications

- Peritonitis
- Abscess formation
- Bleeding
- Obstruction

Management

- Diverticulitis is usually treated on an outpatient basis with diet and medicine therapy.
- Hospitalization is required in acute disease; often indicated for;
  - The elderly
  - Immunocompromised
  - Corticosteroid therapy
- Rest
- Analgesics: An opioid is prescribed for pain relief; avoid morphine because it increases segmentation and intra-luminal pressures.
- Antispasmodics: such as propantheline bromide (Pro-Banthine) and oxyphencyclimine (Daricon) may be prescribed.
- Withhold oral intake in the acute phase
- Initial diet should be a clear liquid until the inflammation subsides:

Then high-fiber, low-fat diet is recommended.

- Antibiotics are prescribed for 7 to 10 days.
- A bulk forming laxative e.g. Metamucil
- Administer intravenous fluids
- Institute nasogastric suctioning if vomiting or distention occurs

Antispasmodics Normal stools

Surgical Management

Used as an immediate surgical intervention in case of complications or to prevent repeated episodes of diverticulitis.
Two types of surgery are considered:

- One-stage resection in which the inflamed area is removed and a primary end-to-end anastomosis is completed
- Multiple-staged procedures for complications such as obstruction or perforation.

These can be performed through traditional laparatomy or laparoscopically assisted colectomy.

Possible Nursing Diagnoses

i. Imbalanced nutrition, less than body requirements, related to nausea and anorexia.
ii. Risk for deficient fluid volume related to vomiting and dehydration.
iii. Anxiety related to impending surgery and the diagnosis of cancer
iv. Impaired skin integrity related to the surgical incisions (abdominal and perianal), the formation of a stoma, and frequent fecal contamination of peristomal skin.
v. Disturbed body image related to colostomy.
vi. Ineffective sexuality patterns related to presence of ostomy and changes in body image and self-concept.

4.0 SUMMARY

At the end of the unit, you should be able to:

- identify various types of structural and obstructive disorders of the intestines.
- discuss the Pathophysiology of the different types of structural and obstructive disorders of the intestines.
- care for patients with structural and obstructive disorders of the intestines utilizing the nursing process approach.

Activities in your place of practice, identify a patient with any type of structural and obstructive disorders of the intestines and develop an outline of comprehensive assessment and management of the patient

SELF-ASSESSMENT EXERCISE

Now that you have studied this unit, you may attempt the following questions: you may grade yourself using the course content and reference materials.

i. outline the types of structural and obstructive disorders of the intestines

ii. discuss the Pathophysiology of intestinal obstruction
iii. using the nursing process, identify and solve in order of priority, three (3) possible nursing diagnoses of a patient with structural and obstructive disorders of the intestines.

5.0 REFERENCES/FURTHER READING

Levey, R., Williams-Wilson, B. (2002).Anorexia Nervosa: eMedicine Journal, April 2 2002, Volume 3, Number 4

Smeltzer, S.C., Brenda, B. (2006). Brunner and Suddhart's Textbook of Medical-Surgical Nursing, 10th edition. Lippincott-William & Wilkins

Templer, J. W. (2001). Parotitis; eMedicine journal; July, 2001, Vol. 2, Number, 7

UNIT 9 CARING FOR PATIENT WITH ANORECTAL DISORDER

CONTENTS

1.0 INTRODUCTION

This unit will focus on caring for patients with structural and obstructive disorders of the intestines.

2.0 OBJECTIVES

At the end of the unit, you will be able to:

- identify various types of anorectal disorders of the intestines
- discuss the Pathophysiology of the different types of anorectal disorders
- care for patients with anorectal disorders utilizing the nursing process approach.

3.0 MAIN CONTENT

3.1 Diseases of the Anorectum

Anorectal disorders are common
Common disorders include;

- Pain; the major complaint
- Rectal bleeding
- Change in bowel habits
- Protrusion of hemorrhoids
- Anal discharge
- Perianal itching and swelling
- Anal tenderness, stenosis, and ulceration

Constipation; results from delaying defecation due to anorectal pain

3.2 Anorectal Abscess

An anorectal abscess is caused by obstruction of an anal gland, resulting in retrograde infection.
Predisposing factors;

- Regional enteritis
- Immunosuppressive conditions such as AIDS

Many of these abscesses result in fistulas.
An abscess may occur in a variety of spaces in and around the rectum.
It often contains foul-smelling pus and is painful.

- Superficial abscess presents as;
  - Swelling
  - Redness and
  - Tenderness
- Deeper abscess may result in toxic symptoms such as
  - Lower abdominal pain
  - Fever.

Management:

- Palliative therapy consists of;
  - Sitz baths
  - Analgesics
- Surgical incision and drainage is the treatment of choice with possible fistulectomy
- The wound may be packed with gauze and allowed to heal by granulation.

3.3 Anal Fistula

An anal fistula is a tiny, tubular, fibrous tract that extends into the anal canal from an opening located beside the anus.
Causes;

- Infection
- Trauma
- Fissures
- Regional enteritis

Clinical manifestations:

- Pus or stool may leak constantly through the cutaneous opening
- Passage of flatus or feces from the vagina or bladder
- Systemic infection with untreated cases

Management

- Few fistulas may heal spontaneously

- Surgery is always recommended: Fistulectomy (i.e., excision of the fistulous tract)
- Enemas to evacuate the lower bowel

3.4 Anal Fissure

An anal fissure is a longitudinal tear or ulceration in the lining of the anal canal
Causes
- Trauma due to
  - Passing a large, firm stool
  - Persistent tightening of the anal canal because of stress and anxiety
- Childbirth
- Trauma
- Overuse of laxatives.
- Clinical manifestations;
- Extremely painful defecation
- Burning anus
- Bleeding on defecation

Management
- Most fissures heal with conservative measures such as;
  - Stool softeners
  - Bulk agents
  - Increased water intake
  - Sitz baths
  - Emollient suppositories
  - Anal dilation under anesthesia may be required.
- Surgery; indicated when conservative treatment fails
  - The procedure of choice is the lateral internal sphincterotomy with excision of the fissure

3.5 Hemorrhoids

Hemorrhoids are dilated portions of veins in the anal canal.
They are very common
Causes;
- Shearing of the mucosa during defecation
- Pregnancy due to increased pressure in the hemorrhoidal tissue

Classifications;
Hemorrhoids are classified as one of two types.
- Internal hemorrhoids; Those above the internal sphincter

- External hemorrhoids; those appearing outside the external sphincter

Clinical manifestations;

- Itching
- Excessive pain especially with external hemorrhoids due to inflammation and edema caused by thrombosis (i.e., clotting of blood within the hemorrhoid)
- Bright red bleeding with defecation
- Prolapse

Management;

- Conservative management:
  - Good personal hygiene
  - Avoiding excessive straining during defecation
  - A high-residue diet with an increased fluid intake
  - Hydrophilic bulk-forming agents such as psyllium and mucilloid may help.
  - Warm compresses
  - Sitz baths
  - Analgesic ointments
  - Suppositories
  - Astringents (e.g., witch hazel)
  - Bed rest
- Nonsurgical treatments;
  - Infrared photocoagulation
  - Bipolar diathermy
  - Laser therapy
  - Injecting sclerosing solutions for small, bleeding hemorrhoids
- A conservative surgical treatment:
  - Rubber-band ligation procedure; hemorrhoid is visualized through the anoscope, and its proximal portion above the mucocutaneous lines is grasped with an instrument. A small rubber band is then slipped over the hemorrhoid. Tissue distal to the rubber band becomes necrotic after several days and sloughs off.
  - Cryosurgical hemorrhoidectomy; involves freezing the hemorrhoid for a sufficient time to cause necrosis. Although relatively painless, this procedure is not widely used because the discharge is very foul smelling and wound healing is prolonged.
  - The Nd:YAG laser;
- Surgery: Hemorrhoidectomy, or surgical excision it involves digital dilatation of rectal sphincter and ligagtion and excision of the hemorrhoids

Possible Nursing Diagnoses

- Constipation related to ignoring the urge to defecate because of pain during elimination
- Anxiety related to impending surgery and embarrassment
- Acute pain related to irritation, pressure, and sensitivity in the anorectal area from anorectal disease and sphincter spasms after surgery
- Urinary retention related to postoperative reflex spasm and fear of pain
- Risk for ineffective therapeutic regimen management

4.0 SUMMARY

At the end of the unit, you should be able to:

- identify various types of anorectal disorders of the intestines
- discuss the Pathophysiology of the different types of anorectal disorders
- care for patients with anorectal disorders utilizing the nursing process approach.

Activities

In your place of practice, identify a patient with any of the types of anorectal disorders and develop an outline of comprehensive assessment and management of the patient

SELF-ASSESSMENT EXERCISES

Now that you have studied this unit, you may attempt the following questions: you may grade yourself using the course content and reference materials.

i. outline the types of anorectal disorders
ii. discuss the Pathophysiology of hemorrhoids
iii. using the nursing process, identify and solve in order of priority, three (3) possible nursing diagnoses of a patient with anorectal disorders.

5.0 REFERENCES/FURTHER READING

Levey, R., Williams-Wilson, B. (2002). Anorexia Nervosa: eMedicine Journal, April 2 2002, Volume 3, Number 4.

Smeltzer, S.C., Brenda, B. (2006). Brunner and Suddhart's Textbook of Medical-Surgical Nursing, 10th edition. Lippincott-William & Wilkins.

Templer, J. W. (2001). Parotitis; eMedicine Journal; July, 2001, Vol. 2, Number, 7.

can flow from the interstitial spaces into the blood. The lymphatics can carry proteins and large particulate matter away from the tissue spaces, neither of which can be removed by absorption directly into the blood capillaries. One of the main function is the returning of protein to the blood from the interstitial spaces.

i. the various assessments that can be done on the various body system to detect hematological functions

Assessment and diagnostic evaluation of the hematologic and lymphatic system involves a basic understanding of the various normal indices of these systems as this would serve as a guide for conducting most assessments, making diagnosis and giving evaluative reports.

Most hematologic disease conditions indicates an anomaly in the hematopoetic, homeostatic or the recticulo-endothelial system. This anomaly may be quantitative (increase or decrease in the production of cells) or qualitative (cells produced are defective in the normal functional capacity) or both.

There are a number of features which can be revealed by history and clinical examination. This can provide clues for diagnosis and help in the interpretation of complete blood count (CBC) results in context. Signs and symptoms relevant to CBC are; pallor, jaundice, fever, lymphadenopathy, bleeding or bruising, hepatomegaly, splenomegaly, frequency and severity of infections, mouth ulcers, recent viral infections, exposure to herbal drugs and toxins, fatigue/ weight loss to mention a few.
The use of symptoms might not be a very reliable index to make diagnosis. Hence, there is a need to conduct extensive and thorough laboratory tests and other investigations. Investigations that can be conducted are;

A. Complete blood count (CBC); blood for CBC are typically obtained by venipuncture. The various indices in the CBC are

i. Hemoglobin (Hb) & Hematocrit (Hct); this measures the amount of packed cells of hemoglobin. The values could be decreased in some conditions such as aneamia and it can be increased in conditions such as polycythermia.

ii. Erythrocyte count (RBC); the values vary in various conditions. It can be decreased in pernicious aneamia, it can be microcytic with hypochromic erythrocytes in iron deficiency aneamia.

iii. Stained RBC examination; it detects changes in colour and shape.

iv. Reticulocyte count; it assesses bone marrow functions. When the values are decreased, it could indicate folic acid deficiency, pernicious anaemia, bone marrow failure etc. it is elevated in cases of heamolysis, leukemia, etc.

v. White blood cell count; this value may be increased or decreased to indicate different conditions
vi. Platelet count; a decreased value of platelet count could indicate aplastic anaemia, an elevated level could be a pointer to iron deficiency anemia or hemolytic anaemia
vii. Erythrocyte sedimentation rate (ESR); elevated levels indicates the presence of an inflammatory reaction in conditions such as increased RBC destruction or malignant disease.
viii. RBC survival time; this can be used in differential diagnosis of anaemia because RBCs have shortened lifespan in pernicious and hemolytic aneamia.
ix. Erythrocyte fragility test; the values are decreased in iron deficiency anemia and an increased value confirms hemolytic or autoimmune anaemia.
x. Heamoglobin electrophoresis; it identifies the types of heamoglobin structure and this aids in determining the source of heamolyticanaemia.
xi. Serum folate and vitamin B12; it aids in diagnosing anaemia related to deficiencies in dietary intake or malabsorption.
xii. Serum iron; iron is absent in iron deficiency anaemia and the values are elevated in hemolytic and a plastic anaemia.
xiii. Serum total iron binding capacity (TIBC); this values are increased in iron deficiency and it is normal or slightly reduced in aplastic aneamia.
xiv. Serum ferritin; the values are decreased in iron deficiency aneamia.
xv. Serum bilirubin (unconjugated); the values are elevated in pernicious anemia or heamolytic anaemia.
xvi. Serum lactate dehydrogenase; the value may be elevated in pernicious anemia.
xvii. Bleeding time; the time is further prolonged in aplastic anemia
xviii. Schillings test; decreased urinary excretion of vitamin B12 is indicative of aplastic anemia.
xix. Guaiac test; it may be positive or occult blood in urine, stool or gastric content which could be an indication of acute or chronic bleeding iron deficiency anemia
xx. Gastric analysis; results of this shows decreased secretions with elevated pH and absence of free HCl in pernicious anaemia.
xxi. Bone marrow aspiration/ biopsy evaluation; cells may show changes in number, size and shape aiding in differentiation of the type of aneamia e.g. increased megaloblasts in pernicious anaemia and fatty marrow with diminished or absence of blood cells at several sites in aplastic anaemia.
xxii. Endoscopic & radiographic studies; this aids in detecting bleeding site as in acute/ chronic gastrointestinal GI bleeding.

Client Assessment

Client assessment should use questions that can be used to explore the client's

- ACTIVITY AND REST: if tachypnea, tachycardia, orthopnea, dyspnea or exertion at rest is noticed, it is indicative of a hematological disorder.
- CIRCULATORY FUNCTION: a positive history of blood loss, palpitation, increased blood pressure, dysrhythmia, systolic murmur, throbbing carotid pulsations, pallor of skin, mucous membrane and nail beds , greyish casts in black clients, waxy pale skin as can be seen in aplastic anaemia etc. all these are indicative of hematological or lymphatic disorder.

The sclera could be blue or pearl white, capillary refill time is delayed, the nails are brittle.

- ELIMINATION: on assessing the elimination pattern, a positive history of pyelonephritis, renal failure, flatulence, malabsorption syndrome, hematemesis, fresh blood or in stool or maleana could all be suggestive of hematological imbalances.
- FLUID AND ELECTROLYTE IMBALANCES: there is decreased dietary intake of animal protein, high intake of cereal product, there could be difficulties in swallowing due to ulcerations in the pharynx, recent weight loss, poor skin tugor. There could also be cheilitis (inflammation of the lips with cracking at the corners of the mouth) in heamatological and lymphatic system disorders.
- On general physical examination, the client may look unkempt with dry, brittle, thinning out hair. They could also be premature greying of the hair.
- On examination of the sensorineural organs, the client could report headaches, fainting, dizziness, vertigo, tinutus, insomnia, weakness, paresthesia of hands/ feet claudication, irritability, apathy, retinal heamorrhages, epistaxis, etc.
- The client could have low grade fever, chills, night sweat, generalized lymphadenopathy, petechiae, ecchymosis which occurs in aplastic aneamia.
- On assessment of the reproductive system, the client could have menorrhagia or amenorrhea in women in iron deficiency aneamia.

Investigations to Assess the Functioning the Lymphatic System Include;

- Lymphangiography; this provides a means of detecting lymph node involvement resulting from metastatic carcinoma,

lymphoma or infection in sites that are otherwise inaccessible to the examiner except by surgery. The test is conducted, lymphatic vessels in each of foot or hand is injected with a contrast agent. A series of x-rays are taken at the end of the injection, at 24 hours after and periodically after that. The failure to identify subcutaneous lymphatic collection contrast agent in the persistence of contrast agent in the tissue for days afterwards is indicative of lymphedema.

- Lymphoscintography; it is a reliable alternative to lymphangiography. A radioactively labelled colloid is injected subcutaneously into the second interdigital space. The extremities are the exercised to facilitate the uptake of colloid by the lymphatic system and then serial images are obtained at preset interval.

Nursing care during these assessments include

i. An explicit explanation of the procedure to the client and antianxiety drugs could be administered to very anxious clients if not contraindicated to the procedure.
ii. Informed consent should be obtained
iii. Aseptic technique from be maintained for all the procedures
iv. All nursing care required during the procedure should be accorded.
v. Results should be interpreted to the client adequately in accordance with organization protocol.

REFERENCES/FURTHER READING

Fuad, I. & Welday, M. (2008); Pathophysiology. Ethiopia Public Health Training Initiative. Ethiopia; Hirmaya University.

Famakinwa, O.T. Synopsis of medicosurgical Nursing. Ibadan; Bayosore

Heyman, P. ; Anatomy of the immune system supplemental reading.

Smettzer, C.S., Bare, B.G, Hinkle, L.J. & Cheever, H.K., (2008). Brunner & Suddarth's textbook of Medical-Surgical Nursing. (11th Ed). India; Wolters Kluwer.

Waugh, A., Grant, A. (2006). Ross & Wilson Anatomy and Physiology in Health and Illness (10th Ed). Philadelphia; Elsevier.

UNIT 2 REVIEW OF ANATOMY OF BLOOD AND THE LYMPHATIC SYSTEM

OBJECTIVE

At the end of this unit, you should be able to:

- describe the functions of the blood.
- list and describe the components of blood
- discuss the lymph and functions of the lymphatic system.

Blood is a connective tissue that provides a means of communication between the cells and different parts of the body. This form of communication involves;

- Carrying oxygen from the lungs to tissue and carbon dioxide from the tissues to the lungs for oxygenation.
- Carrying of nutrient from the alimentary tract to tissues and cell waste to the excretory organs, principally the kidneys.
- Hormones secreted by the endocrine gland to their target glands and tissues.
- Protective substances to areas of infection or attack
- Transport of clotting factors that coagulate blood

Blood makes up about seven percent of body weight and the values are lesser in women than in men. The values also seems higher in children. The continual flow of blood maintains a fairly constant homeostatic environment for the body. Blood is composed of straw coloured transparent fluid which is called plasma. Within the plasma, different types of cells are suspended. Plasma is about 55% and the blood cells is about 45%.

Components of Blood

The various component of blood are;

i. Plasma
ii. Erythrocytes (Red cells)
iii. Platelets (Thrombocytes)
iv. Leucocytes (White cells)

i) PLASMA

The constituents of plasma are water and dissolved substance including plasma protein, inorganic salt, nutrient especially from digested food, waste materials, hormones and gases.

Plasma proteins

They make up about seven percent of plasma are normally retained in the blood because they are too big to escape through the capillary wall. They are responsible for maintaining osmotic pressure of blood which keeps plasma fluid within circulation. These plasma proteins include albumin, globulins and clotting factors. These proteins function in various capacities, ranging from maintenance of plasma osmotic pressure, carrier molecule for lipids and steroid hormones, serving as antibodies, transportation of some hormones and mineral salt and blood coagulation.

Inorganic salt

They are involved in a variety of activities such as muscle contractions, transmission of nerve impulses, formation of secretion and maintenance of acid-base balance. In health, the blood is slightly alkaline. The pH blood is maintained between 7.35 and 7.5 by an ongoing complicated series of chemical activities involving buffering system.

Nutrients

Food is broken down into small molecules to aid absorption. These molecules together with mineral synthesis of other blood component and secretions.

Waste products

Urea, creatinine and uric acid are waste products of protein metabolism. They are formed in the liver and transported to the kidney for excretion

Hormones

These are substances synthesized by endocrine glands. Hormones pass directly from the endocrine cells into the blood where they are transported to the target organs or cells.

Gases

Oxygen, carbondioxide and nitrogen are transported around the body dissolved in plasma. Oxygen and carbondioxide are also transported in combination with the haemoglobin of red blood cells. Most oxygen are transported in combination with haemoglobin and most carbondioxide as bicarbonate ions dissolved in plasma.

ii) RED BLOOD CELLS

The red blood cells, also known as erythrocytes, functions in to transporting hemoglobin, which in turn carries oxygen from the lungs to the tissues. When it is free in the plasma of the human being, about 3 per cent of it leaks through the capillary membrane into the tissue spaces or through the glomerular membrane of the kidney into the glomerular filtrate each time the blood passes through the capillaries. For

hemoglobin to remain in the human bloodstream, it must exist inside red blood cells. The hemoglobin in the cells is an excellent acid-base buffer (as is true of most proteins), so that the red blood cells are responsible for most of the acid-base buffering power of whole blood.

Normal red blood cells are biconcave discs having a mean diameter of about 7.8 micrometers and a thickness of 2.5 micrometers at the thickest point and 1 micrometer or less in the center. The average volume of the red blood cell is 90 to 95 cubic micrometers. The shapes of red blood cells can change remarkably as the cells squeeze through capillaries. Actually, the red blood cell is a "bag" that can be deformed into almost any shape. Furthermore, because the normal cell has a great excess of cell membrane for the quantity of material inside, deformation does not stretch the membrane greatly and, consequently, does not rupture the cell, as would be the case with many other cells.

In normal men, the average number of red blood cells per cubic millimeter is 5,200,000 (±300,000);in normal women, it is 4,700,000 (±300,000).Persons living at high altitudes have greater numbers of red blood cells. Furthermore, in normal people, the percentage of hemoglobin is almost always near the maximum in each cell.

Production of Red Blood Cells

In the early weeks of embryonic life, primitive, nucleated red blood cells are produced in the yolk sac. During the middle trimester of gestation, the liver is the main organ for production of red blood cells, but reasonable numbers are also produced in the spleen and lymph nodes. Then, during the last month or so of gestation and after birth, red blood cells are produced exclusively in the bone marrow, the bone marrow of essentially all bones produces red blood cells until a person is 5 years old.The marrow of the long bones, except for the proximal portions of the humeri and tibiae, becomes quite fatty and produces no more red blood cells after about age 20 years.

Genesis of Blood Cells

The blood cells begin their lives in the bone marrow from a single type of cell called the pluripotential hematopoietic stem cell, from which all the cells of the circulating blood are eventually derived. There is successive divisions of the pluripotential cells to form the different circulating blood cells. As these cells reproduce, a small portion of them remains exactly like the original pluripotential cells and is retained in the bone marrow to maintain a supply of these, although their numbers diminish with age. The intermediate- stage cells are very much like the pluripotential stem cells, even though they have already become committed to a particular line of cells and are called committed stem cells. The different committed stem cells, when grown in culture, will

produce colonies of specific types of blood cells. A committed stem cell that produces erythrocytes is called a colony-forming unit–erythrocyte, and the abbreviation CFU-E is used to designate this type of stem cell. Growth and reproduction of the different stem cells are controlled by multiple proteins called growth inducers. Four major growth inducers are described, each having different characteristics. One of these, interleukin-3, promotes growth and reproduction of virtually all the different types of committed stem cells, whereas the others induce growth of only specific types of cells. The growth inducers promote growth but not differentiation of the cells. This is the function of another set of proteins called differentiation inducers. Each of these causes one type of committed stem cell to differentiate one or more steps toward a final adult blood cell. Formation of the growth inducers and differentiation inducers is itself controlled by factors outside the bone marrow. For instance, in the case of erythrocytes (red blood cells), exposure of the blood to low oxygen for a long time results in growth induction, differentiation, and production of greatly increased numbers of erythrocytes. In the case of some of the white blood cells, infectious diseases cause growth, differentiation, and eventual formation of specific types of white blood cells that are needed to combat each infection.

Stages of Differentiation of Red Blood Cells

The first cell that can be identified as belonging to the red blood cell series is the proerythroblast. Once the proerythroblast has been formed, it divides multiple times, eventually forming many mature red blood cells. The first-generation cells are called basophil erythroblasts because they stain with basic dyes; the cell at this time has accumulated very little hemoglobin. In the succeeding generations, the cells become filled with hemoglobin to a concentration of about 34 per cent, the nucleus condenses to a small size, and its final remnant is absorbed or extruded from the cell. At the same time, the endoplasmic reticulum is also reabsorbed. The cell at this stage is called a reticulocyte because it still contains a small amount of basophilic material, consisting of remnants of the Golgi apparatus, mitochondria, and a few other cytoplasmic organelles. During this reticulocyte stage, the cells pass from the bone marrow into the blood capillaries by diapedesis (squeezing through the pores of the capillary membrane). The remaining basophilic material in the reticulocyte normally disappears within 1 to 2 days, and the cell is then a mature erythrocyte. Because of the short life of the reticulocytes, their concentration among all the red cells of the blood is normally slightly less than 1 per cent.

Regulation of Red Blood Cell Production

The total mass of red blood cells in the circulatory system is regulated within narrow limits, so that an adequate number of red cells is always

available to provide sufficient transport of oxygen from the lungs to the tissues and that the cells do not become so numerous that they impede blood flow.

Tissue Oxygenation Is the Most Essential Regulator of Red Blood Cell Production. Any condition that causes the quantity of oxygen transported to the tissues to decrease ordinarily increases the rate of red blood cell production. Thus, when a person becomes extremely anemic as a result of hemorrhage or any other condition, the bone marrow immediately begins to produce large quantities of red blood cells. Also, destruction of major portions of the bone marrow by any means, especially by x-ray therapy, causes hyperplasia of the remaining bone marrow, thereby attempting to supply the demand for red blood cells in the body. At very high altitudes, where the quantity of oxygen in the air is greatly decreased, insufficient oxygen is transported to the tissues, and red cell production is greatly increased. In this case, it is not the concentration of red blood cells in the blood that controls red cell production but the amount of oxygen transported to the tissues in relation to tissue demand for oxygen. Various diseases of the circulation that cause decreased blood flow through the peripheral vessels, and particularly those that cause failure of oxygen absorption by the blood as it passes through the lungs can also increase the rate of red cell production. This is especially apparent in prolonged cardiac failure and in many lung diseases, because the tissue hypoxia resulting from these conditions increases red cell production, with a resultant increase in hematocrit and usually total blood volume as well.
Erythropoietin Stimulates Red Cell Production, and Its Formation Increases in Response to Hypoxia. The principal stimulus for red blood cell production in low oxygen states is a circulating hormone called erythropoietin. In the absence of erythropoietin, hypoxia has little or no effect in stimulating red blood cell production. But when the erythropoietin system is functional, hypoxia causes a marked increase in erythropoietin production, and the erythropoietin in turn enhances red blood cell production until the hypoxia is relieved.

Life Span and Destruction of Red Blood Cells
When red blood cells are delivered from the bone marrow into the circulatory system, they normally circulate an average of 120 days before being destroyed. Even though mature red cells do not have a nucleus, mitochondria, or endoplasmic reticulum, they do have cytoplasmic enzymes that are capable of metabolizing glucose and forming small amounts of adenosine triphosphate. These enzymes also maintain pliability of the cell membrane, maintain membrane transport of ions, keep the iron of the cells' hemoglobin in the ferrous form rather than ferric form and prevent oxidation of the proteins in the red cells. Even so, the metabolic systems of old red cells become progressively

less active, and the cells become more and more fragile, presumably because their life processes wear out. Once the red cell membrane becomes fragile, the cell ruptures during passage through some tight spot of the circulation. Many of the red cells self-destruct in the spleen, where they squeeze through the red pulp of the spleen. When red blood cells burst and release their hemoglobin, the hemoglobin is phagocytized almost immediately by macrophages in many parts of the body, but especially by the Kupffer cells of the liver and macrophages of the spleen and bone marrow. During the next few hours to days, the macrophages release iron from the hemoglobin and pass it back into the blood, to be carried by transferrin either to the bone marrow for the production of new red blood cells or to the liver and other tissues for storage in the form of ferritin. The porphyrin portion of the hemoglobin molecule is converted by the macrophages, through a series of stages, into the bile pigment bilirubin, which is released into the blood and later removed from the body by secretion through the liver into the bile.

iii) PLATELETS (THROMBOCYTES)

These are small non-nucleated discs derived from the cytoplasm of megakaryotes in the red bone marrow. They contain a number of substances which aid blood clotting and hemostasis. The normal blood platelet count is between 200 x 10^9/litre and 350 x 10^9 / litre. The control of platelet production is not very clear but the kidneys release thrombopoetin which stimulates platelet synthesis and other cytokines are involved .

iv) LEUKOCYTES (WHITE BLOOD CELLS)

The leukocytes, also called white blood cells, are the mobile units of the body's protective system. They are formed partially in the bone marrow (granulocytes and monocytes and a few lymphocytes) and partially in the lymph tissue (lymphocytes and plasma cells). After formation, they are transported in the blood to different parts of the body where they are needed. The real value of the white blood cells is that most of them are specifically transported to areas of serious infection and inflammation, thereby providing a rapid and potent defense against infectious agents. Granulocytes and monocytes have a special ability to "seek out and destroy" a foreign invader.

Six types of white blood cells are normally present in the blood. They are;

- Polymorphonuclear neutrophils
- Polymorphonucleareosinophils
- Polymorphonuclear basophils
- Monocytes
- Lymphocytes and, occasionally, Plasma cells.

The first three types of cells, the polymorphonuclear cells, all have a granular appearance. The granulocytes and monocytes protect the body against invading organisms mainly by ingesting through phagocytosis.

The lymphocytes and plasma cells function mainly in connection with the immune system. The adult human being has about 7000 white blood cells per microliter of blood (in comparison with 5 million red blood cells).Of the total white blood cells, the normal percentages of the different types are approximately the following:

a. Polymorphonuclear neutrophils 62.0%
b. Polymorphonucleareosinophils 2.3%
c. Polymorphonuclear basophils 0.4%
d. Monocytes 5.3%
e. Lymphocytes 30.0%.

In the early differentiation of the pluripotential hematopoietic stem cell into the different types of committed stem cells two major lineages of white blood cells are formed, the myelocytic and the lymphocytic lineages. The granulocytes and monocytes are formed only in the bone marrow. Lymphocytes and plasma cells are produced mainly in the various lymphogenous tissues—especially the lymph glands, spleen, thymus, tonsils, and various pockets of lymphoid tissue elsewhere in the body, such as in the bone marrow and in Peyer's patches underneath the epithelium in the gut wall. The white blood cells formed in the bone marrow are stored within the marrow until they are needed in the circulatory system. Normally, about three times as many white blood cells are stored in the marrow as circulate in the entire blood. This represents about a 6-day supply of these cells. The lymphocytes are mostly stored in the various lymphoid tissues, except for a small number that are temporarily being transported in the blood.

Life Span of the White Blood Cells

The life of the granulocytes after being released from the bone marrow is normally 4 to 8 hours circulating in the blood and another 4 to 5 days in tissues where they are needed. In times of serious tissue infection, this total life span is often shortened to only a few hours because the granulocytes proceed even more rapidly to the infected area, perform their functions, and, in the process, are themselves destroyed. The monocytes also have a short transit time, 10 to 20 hours in the blood, before wandering through the capillary membranes into the tissues. Once in the tissues, they swell to a much larger size to become tissue macrophages, and, in this form, can live for months unless destroyed while performing phagocytic functions. These tissue macrophages are the basis of the tissue macrophage system, which provides continuing defense against infection. Lymphocytes enter the circulatory system continually, along with drainage of lymph from the lymph nodes and other lymphoid tissue. After a few hours, they pass out of the blood back into the tissues by diapedesis. Then, still later, they re-enter the lymph and return to the blood again and again; thus, there is continual circulation of lymphocytes through the body. The lymphocytes have life

spans of weeks or months; this life span depends on the body's need for these cells. The platelets in the blood are replaced about once every 10 days; in other words, about 30,000 platelets are formed each day for each microliter of blood.
It is mainly the neutrophils and tissue macrophages that attack and destroy invading bacteria, viruses, and other injurious agents. The neutrophils are mature cells that can attack and destroy bacteria even in the circulating blood. Conversely, the tissue macrophages begin life as blood monocytes, which are immature cells while still in the blood and have little ability to fight infectious agents at that time. However, once they enter the tissues, they begin to swell—sometimes increasing their diameters as much as five fold—to as great as 60 to 80 micrometers, a size that can barely be seen with the naked eye. These cells are now called macrophages, and they are extremely capable of combating intra-tissue disease agents.

LYMPH AND THE LYMPHATIC SYSTEM
Lymph is a clear watery fluid, similar in composition to plasma, with the important exceptions of plasma proteins and identical in composition to interstitial fluid. Lymph transports the plasma protein that seeps out of the capillary beds back to the blood stream. It also carries larger particles, e.g bacteria and cell debris from damaged tissues which can be filtered out and destroyed by the lymph nodes. Lymph contains various lymphocytes which circulate in the lymphatic system allowing them to move freely around the body.

The lymphatic system represents an accessory route through which fluid can flow from the interstitial spaces into the blood. Most importantly, the lymphatics can carry proteins and large particulate matter away from the tissue spaces, neither of which can be removed by absorption directly into the blood capillaries. This return of proteins to the blood from the interstitial spaces is an essential function without which we would die within about 24 hours.

Almost all tissues of the body have special lymph channels that drain excess fluid directly from the interstitial spaces. The exceptions include the superficial portions of the skin, the central nervous system, the endomysium of muscles, and the bones. But, even these tissues have minute interstitial channels called prelymphatics through which interstitial fluid can flow; this fluid eventually empties either into lymphatic vessels or, in the case of the brain, into the cerebrospinal fluid and then directly back into the blood. Essentially all the lymph vessels from the lower part of the body eventually empty into the thoracic duct, which in turn empties into the blood venous system at the juncture of the left internal jugular vein and left subclavian vein. Lymph from the left side of the head, the left arm, and parts of the chest region also enters the

thoracic duct before it empties into the veins. Lymph from the right side of the neck and head, the right arm, and parts of the right thorax enters the right lymph duct(much smaller than the thoracic duct),which empties into the blood venous system at the juncture of the right subclavian vein and internal jugular vein. Most of the fluid filtering from the arterial ends of blood capillaries flows among the cells and finally is reabsorbed back into the venous ends of the blood capillaries; but on the average, about 1/10 of the fluid enters the lymphatic capillaries and returns to the blood through the lymphatic system rather than through the venous capillaries. The total quantity of all this lymph is normally only 2 to 3 liters each day.

The fluid that returns to the circulation by way of the lymphatics is extremely important because substances of high molecular weight, such as proteins, cannot be absorbed from the tissues in any other way, although they can enter the lymphatic capillaries almost unimpeded. The lymphatics have valves at the very tips of the terminal lymphatic capillaries as well as valves along their larger vessels up to the point where they empty into the blood circulation.

Formation of Lymph

Lymph is derived from interstitial fluid that flows into the lymphatics. Therefore, lymph as it first enters the terminal lymphatics has almost the same composition as the interstitial fluid. The protein concentration in the interstitial fluid of most tissues averages about 2 g/dl, and the protein concentration of lymph flowing from these tissues is near this value. Conversely, lymph formed in the liver has a protein concentration as high as 6 g/dl, and lymph formed in the intestines has a protein concentration as high as 3 to 4 g/dl. Because about two thirds of all lymph normally is derived from the liver and intestines, the thoracic duct lymph, which is a mixture of lymph from all areas of the body, usually has a protein concentration of 3 to 5 g/dl. The lymphatic system is also one of the major routes for absorption of nutrients from the gastrointestinal tract, especially for absorption of virtually all fats in food. Indeed, after a fatty meal, thoracic duct lymph sometimes contains as much as 1 to 2 per cent fat. Even large particles, such as bacteria, can push their way between the endothelial cells of the lymphatic capillaries and in this way enter the lymph. As the lymph passes through the lymph nodes, these particles are almost entirely removed and destroyed.

The primary factors that determine lymph flow are; interstitial fluid pressure and the activity of the lymphatic pump. Therefore, one can state that, roughly, the rate of lymph flow is determined by the product of interstitial fluid pressure times the activity of the lymphatic pump.

Lymph nodes in the body

Lymph nodes channels through which the lymph drains through. They are oval or bean-shaped organs that lie often in groups along the length of the lymphatic vessels. The lymph could drain into a number of lymph nodes of between 8-10 before returning to venous circulation. The nodes vary in sizes, some are minutely small and the largest is about an almond size.

Lymph from the head and neck passes through the deep and superficial cervical nodes.

Lymph from the upper limbs passes through nodes situated in the elbow region, then through the deep and superficial axillary nodes.

Lymph from organs and tissues in the thoracic cavity drains through group of nodes situated close to the mediastinum, large airways, oesophagus and chest wall. Most of the lymph from the breast passes through the axillary nodes.

Lymph from the pelvic and abdominal cavities passes through many lymph nodes before entering the cisterna chyli. The abdominal and pelvic nodes are situated mainly in association with blood vessels supplying the organs and close to the main arteries i.e. the aorta and the external and internal iliac arteries.

The lymph from the lower limbs drains through deep and superficial nodes including groups of nodes behind the knee and in the groin i.e. the inguinal nodes

UNIT 3 CARING FOR PATIENT WITH RED BLOOD DISORDER; ANAEMIA, POLYCYTHEMIA

OBJECTIVE

At the end of thisunit, you should be able to:

- define anaemia
- describe the types anaemia
- describe the management of patients with anaemia and list the complications of anaemia
- define polycthermia and list the signs and symptoms
- dDescribe the management of patients with polycythermia.

ANAEMIAS

Anaemia is a condition in which the concentration of oxygen-carrying pigment, haemoglobin, in the blood is below normal level it also means deficiency of hemoglobin in the blood, which can be caused by either too few red blood cells or too little hemoglobin in the cells. It occurs when the rate of production of mature cells entering the blood from the red bone marrow does not keep pace with the rate of haemolysis.Red cells may appear abnormal when viewed microscopically. Anaemia is not a disease condition but a feature of many different disorders. The severity of symptoms depend on how low the haemoglobin concentration has become as the body cell require adequate oxygen for aerobic respiration and other body activities. Haemoglobin level value is considered in relation to age and sex. Adult males have heamoglobin levels on average 2g/dl.

Values of Red Blood cells

Diagnostic indices	Male	Female
Red blood count	4.5-6.5 x 10^{12}/litre	3.8-5.8 x 10^{12}/ litre
Haemoglobin content estimation	13-18g/dl	12g/dl
PCV	40-50%	37-47%

Types of anemia

The types of anaemia are broadly categorized into;

i) Impaired erythrocyte production; Iron deficiency, megaloblasticanaemia, aplastic anaemia.

ii) Increased erythrocyte loss; haemolyticanaemia, normocytic anaemia

Impaired Erythrocyte Production
This occurs as a result of distortion in the formation of red blood cells causing abnormal red blood cells which cannot function optimally. Types of anaemia of the impaired erythrocyte origin include;

(a) Iron deficiency Anaemia

This is a very common form of anaemia. The normal daily requirement of iron intake in men is about 1-2mg, derived from meat and green leafy vegetables. The normal requirement for women is 3mg because of the blood loss during menstruation and the iron needed to nurture pregnancy.Anaemia is confirmed when the haemoglobin level is lower than the normal. Erythrocytes in this type of anaemia is microcytic and hypochromic because the haemoglobin level is low. Causes of iron deficiency include; Malabsorption of iron which can occur in increased pH of the stomach or in surgical removal of the stomach, pregnancy when iron requirements are both from the maternal and fetal ends, chronic blood loss, restricted diets in vegetarians e.t.c

(b) Megaloblastic Anemia

Vitamin B12, folic acid, and intrinsic factor from the stomach mucosa inproduction of erythroblasts in the bone marrow. As a result, the red cells grow too large, with odd shapes, and are called megaloblasts. Thus, atrophy of the stomach mucosa, as occurs in pernicious anemia, or loss of the entire stomach after surgical total gastrectomy can lead to megaloblastic anemia. Also, patients who have intestinal sprue, in which folic acid, vitamin B12, and other vitamin B compounds are poorly absorbed, often develop megaloblasticanemia.Because in these states the erythroblasts cannot proliferate rapidly enough to form normal numbers of red blood cells, those red cells that are formed are mostly oversized, have bizarre shapes, and have fragile membranes. These cells rupture easily, leaving the person in dire need of an adequate number of red cells. Megaloblasticanaemia can be diagnosed by blood tests and bone marrow biopsy.

(c) Aplastic Anaemia

Bone marrow aplasia means lack of functioning bone marrow. It is a condition in which the red blood cells are reduced due to failure of the bone marrow to produce stem cells which is the initial form of all blood cells. A person exposed to gamma ray radiation from a nuclear bomb blast can sustain complete destruction of bone marrow, followed in a few weeks by lethal anemia. Likewise, excessive x-ray treatment, certain industrial chemicals like insecticides used over a prolonged

period of time, and even drugs like anticancer drugs to which the person might be sensitive can cause the same effect. It can also be idiopathic.

Increased Erythrocyte Loss

(a) Hemolytic Anemia

Abnormalities of the red blood cells are mostly hereditarily acquired, making the cells fragile, so that they rupture easily as they go through the capillaries, especially through the spleen. Even though the number of red blood cells formed may be normal, or even much greater than normal in some hemolytic diseases, the life span of the fragile red cell is so short that the cells are destroyed faster than they can be formed, and serious anemia results.

In hereditary spherocytosis, the red cells are very small and spherical rather than being biconcave discs. These cells cannot withstand compression forces because they do not have the normal loose, baglike cell membrane structure of the biconcave discs. On passing through the splenic pulp and some other tight vascular beds, they are easily ruptured by even slight compression. In sickle cell anemia, the cells have an abnormal type of hemoglobin called hemoglobin S, containing faulty beta chains in the hemoglobin molecule, as explained earlier in the chapter. When this hemoglobin is exposed to low concentrations of oxygen, it precipitates into long crystals inside the red blood cell. These crystals elongate the cell and give it the appearance of a sickle rather than a biconcave disc. The precipitated hemoglobin also damages the cell membrane, so that the cells become highly fragile, leading to serious anemia. Such patients frequently experience a vicious circle of events called a sickle cell disease “crisis,” in which low oxygen tension in the tissues causes sickling, which leads to ruptured red cells, which causes a further decrease in oxygen tension and still more sickling and red cell destruction. Once the process starts, it progresses rapidly, eventuating in a serious decrease in red blood cells within a few hours and, often, death.

In erythroblastosisfetalis, Rh-positive red blood cells in the fetus are attacked by antibodies from an Rh-negative mother. These antibodies make theRh-positive cells fragile, leading to rapid rupture and causing the child to be born with serious anemia.

(b) **Normocytic Anaemia**

This red blood cells in this type of anaemia are normal but their numbers are significantly reduced. And the proportion of the reticulocytes in the blood may be increased as the body tries to restore erythrocyte numbers to normal. This occurs in chronic inflammation, severe haemorrhage or any other haemolytic disease.

Effects of Anemia on Function of the Circulatory System

The viscosity of the blood depends almost entirely on the blood concentration of red blood cells. In severe anemia, the blood viscosity may fall to as low as 1.5 times that of water rather than the normal value of about 3. This decreases the resistance to blood flow in the peripheral blood vessels, so that far greater than normal quantities of blood flow through the tissues and return to the heart,thereby greatly increasing cardiac output. Moreover, hypoxia resulting from diminished transport of oxygen by the blood causes the peripheral tissue blood vessels to dilate, allowing a further increase in the return of blood to the heart and increasing the cardiac output to a still higher level sometimes three to four times normal. Thus, one of the major effects of anemia is greatly increased cardiac output, as well as increased pumping workload on the heart. The increased cardiac output in anemia partially offsets the reduced oxygen-carrying effect of the anemia, because even though each unit quantity of blood carries only small quantities of oxygen, the rate of blood flow may be increased enough so that almost normal quantities of oxygen are actually delivered to the tissues.

However, when a person with anemia begins to exercise, the heart is not capable of pumping much greater quantities of blood than it is already pumping. Consequently, during exercise, which greatly increases tissue demand for oxygen, extreme tissue hypoxia results, and acute cardiac failure ensues.

Signs and symptoms; pallor, brittle nails, and dry hair, shortness of breath on exertion, increased respiratory rate, fluid collection in the base of the lung, increased pulse rate, cardiac palpitations, angina pectoris, increased stoke volume, dizziness, general fatigue, tingling sensation in the extremities, syncope, anorexia, flatulence, irregular menstruation, decreased renal function etc.

Complications; they include, heart failure, paresthesia, confusion, angina pectoris,brain damage etc.

Management of Patients With Anaemia

i. A comprehensive assessment is necessary as Identification of the cause is key to appropriate management.
ii. Adequate nutritional and fluid supply, the food should be highly rich in protein, vitamin, folic acid and vitamin B12 in appropriate proportions. There should also be a generous intake of fluid as increased fluid reduces blood viscosity and prevents circulatory stagnation. Fluid can be given either orally or intravenously or

both ways and the fluid should be monitored closely to prevent cardiac overload.

iii. Prevention of infection; opportunistic infections should be prevented through aseptic techniques, nursing the patient in reverse isolation and proper screening of infected visitors.

iv. Encouraging adequate rest and prevention of exertion. Activities that increase oxygen demand should be avoided, activities can also be planned to prevent undue exertion of energy.

v. Oxygen may be administered in cases of dyspnea and the patient may be nursed in Fowlers position. In severe anaemic cases, packed cells of blood may be transfused to increase the amount of haemoglobin in the blood and in turn the oxygen carrying capacity of the blood.

vi. General care include; prompt oral hygiene with mild alkaline mouth washes should be encouraged to prevent ulceration of the oral mucosa and tongue, roughages and other hot spicy food should be avoided, adequate treatment of pressure areas, use of mild warm water with sodium bicarbonate to ease itching of the skin in cases of jaundice. Warmth can also be provided to prevent heat loss.

vii. Provision of adequate information about the disease process, prognosis and therapeutic regimen to the client through health education, to encourage patient's participation in their own care.

viii. Nursing diagnosis can also be made based on the individual patient's condition and a care plan can be made drawn.

Polycythemia

This is an abnormality of the red blood cell in which there is an abnormally large number of erythrocyte in the blood increasing the viscosity and slowing the rate of flow and also increasing the risk of intravascular clotting, ischaemia and infarction. In cases where the tissues become hypoxic because of too little oxygen in the breathed air, such as at high altitudes, or because of failure of oxygen delivery to the tissues, such as in cardiac failure, the blood-forming organs automatically produce large quantities of extra red blood cells causing polycythemia, and the red cell rises to 6 to 7 million/mm^3, about 30 per cent above normal. Heamatocrit of more than 55% in males and more than 50% in females is indicative of polycythermia.

A common type of polycythemia, called physiologic polycythemia, occurs in natives who live at altitudes of 14,000 to 17,000 feet, where the atmospheric oxygen is very low.

Polycythemia Vera (Erythremia) is a type of pathological condition, in which the red blood cell count may be 7 to 8 million/mm^3 and the hematocrit may be 60 to 70 per cent instead of the normal 40 to 45 per

cent. Polycythemia vera is caused by a genetic aberration in the hemocytoblastic cells that produce the blood cells. The blast cells no longer stop producing red cells when too many cells are already present. This cause excess production of red blood cells in the same manner that a breast tumor causes excess production of a specific type of breast cell. It usually causes excess production of white blood cells and platelets as well. In polycythemia vera, not only does the hematocrit increase,but the total blood volume also increases, on some occasions to almost twice normal. As a result, the entire vascular system becomes intensely engorged. In addition, many blood capillaries become plugged by the viscous blood; the viscosity of the blood in polycythemia vera sometimes increases from the normal of 3 times the viscosity of water to 10 times that of water.

Causes and effects of Polycythemia on Function of the Circulatory System

Causes: It can be caused secondarily to other conditions like cigarette smoking, pulmonary disease, bone marrow cancer.

Effects of Polycythemia on Function of the Circulatory System
Because of the greatly increased viscosity of the blood in polycythemia, blood flow through the peripheral blood vessels is often very sluggish. Increasing blood viscosity decreases the rate of venous return to the heart. Conversely, the blood volume is greatly increased in polycythemia, which tends to increase venous return. Actually, the cardiac output in polycythemia is not far from normal, because these two factors more or less neutralize each other. The arterial pressure is also normal in most people with polycythemia, although in about one third of them, the arterial pressure is elevated. This means that the blood pressure–regulating mechanisms can usually offset the tendency for increased blood viscosity to increase peripheral resistance and, thereby, increase arterial pressure. Beyond certain limits, however, these regulations fail, and hypertension develops. The color of the skin depends to a great extent on the quantity of blood in the skin subpapillary venous plexus. In polycythemia vera, the quantity of blood in this plexus is greatly increased. Further, because the blood passes sluggishly through the skin capillaries before entering the venous plexus, a larger than normal quantity of hemoglobin is deoxygenated. The blue color of all this deoxygenated hemoglobin masks the red color of the oxygenated hemoglobin. Therefore, a person with polycythemia vera ordinarily has a ruddy complexion with a bluish (cyanotic) tint to the skin.

Signs and symptoms; ruddy complexion of the skin, splenomegaly, headache, dizziness, tinnitus, fatigue, paresthesia, blurred vision, angina

pectoris, claudication, dyspnea, thrombophlebitis, elevated uric acid level, generalized pruritus, erythromelegia, etc.

Complications; brain attack, stroke, myocardial infarction, bleeding, intracranial haemorrhage, death.

Management

i. A comprehensive assessment is required and consultation with other members of the health team is required.
ii. Routine phlebotomy is conducted on the patient to remove some blood from the patient and thereby reducing the amount of iron thereby causing a reduction in the production of red blood cells.
iii. Suppression of the bone marrow function by the use of radioactive phosphorous or chemotherapeutic agentse ghydroxyurea. But this can in turn increase the risk for leukemia.
iv. Aspirin may be given to reduce the risk of embolism if it is not contraindicated in the patient.
v. Provision of adequate information about the disease process, prognosis and therapeutic regimen to the client through health education, to encourage patient's participation in their own care.
vi. All other patient specific nursing care may be accorded to each client as the need may be.

UNIT 4 CARING FOR PATIENTS WITH WHITE BLOOD CELL DISORDER - leukamia, multiple myeloma, agranulocytosis

OBJECTIVES

At the end of this unit, you should be able to:

- describe leukemia and explain the types
- discuss the effects of leukemia on the body, listing the signs and symptoms and the complications
- describe the management of a leukemic patient'
- describe multiple myeloma, list the signs and symptoms and complications
- discuss the management of a patient with multiple myeloma.
- explain agranulocytosis, list its causes
- describe the care of a patient with agranulocytosis.

LEUKAMIA

Leukemia is a malignant proliferation of white blood cell precursors by the bone marrow. It results in the uncontrolled increase in the production of leukocytes and/or their precursors. The leucocytes produced are immature and not useful to the body as they are unable to perform their normal functions. The uncontrolled proliferation of white blood cells is seen in the liver, spleen and lymph nodes. There is an invasion of the non-hematological organs such as kidney, skin, spleen and the normal marrow elements are replaced by accumulation of white blood cells in the bone marrow.Blood examination reveals increasing aneamia, a decrease in platelet count and bone marrow sample shows a replacement of normal myeloblasts with leukemic cells.

Types of Leukemia

Leukemias are divided into two general types:
- Lymphocytic leukemias
- Myelogenousleukemias.

The lymphocytic leukemias are caused by cancerous production of lymphoid cells, usually beginning in a lymph node or other lymphocytic tissue and spreading to other areas of the body.

Myelogenous leukemia, begins by cancerous production of young myelogenous cells in the bone marrow and then spreads throughout the body so that white blood cells are produced in many extramedullary tissues—especially in the lymph nodes, spleen, and liver. In

myelogenousleukemia, the cancerous process occasionally produces partially differentiated cells, resulting in what might be called neutrophilic leukemia, eosinophilic leukemia, basophilic leukemia, or monocytic leukemia. However, the leukemia cells are bizarre and undifferentiated and not identical to any of the normal white blood cells.

Usually, the more undifferentiated the cell, the more acute is the leukemia, often leading to death within a few months if untreated. With some of the more differentiated cells, the process can be chronic, sometimes developing slowly over 10 to 20 years. Leukemic cells, especially the undifferentiated cells, are usually nonfunctional for providing the normal protection against infection.

Effects of Leukemia on the Body

The first effect of leukemia is metastatic growth of leukemic cells in abnormal areas of the body. Leukemic cells from the bone marrow may reproduce so greatly that they invade the surrounding bone, causing pain and, eventually, a tendency for bones to fracture easily. Almost all leukemias eventually spread to the spleen, lymph nodes, liver, and other vascular regions, regardless of whether the origin of the leukemia is in the bone marrow or the lymph nodes. Common effects in leukemia are the development of infection, severe anemia, and a bleeding tendency caused by thrombocytopenia (lack of platelets). These effects result mainly from displacement of the normal bone marrow and lymphoid cells by the nonfunctional leukemic cells.

The most important effect of leukemia on the body is excessive use of metabolic substrates by the growing cancerous cells. The leukemic tissues reproduce new cells so rapidly that tremendous demands are made on the body reserves for foodstuffs, specific amino acids, and vitamins. Consequently, the energy of the patient is greatly depleted, and excessive utilization of amino acids by the leukemic cells causes especially rapid deterioration of the normal protein tissues of the body. Thus, while the leukemic tissues grow, other tissues become debilitated. After metabolic starvation has continued long enough, this alone is sufficient to cause death.

Signs and symptoms; fever ,malaise, weight loss, tachycardia, fatigue, pallor, shortness of breath, bleeding gums, epistaxis, hepatomegaly, pain in the bones, prolonged clotting time, etc.

Complications; anaemia, renal insufficiency, anaemia, septicaemia, increased susceptibility to infection, haemorrhage, thrombocytopaenia.

Management

i. A comprehensive history should be taken assessing the various body systems and identifying the various symptoms of anaemia.
ii. Diagnostic investigations should be conducted and interpreted as appropriate. E.g. haemoglobin level, reticulocytes, platelet count, white blood count, prothrombin time, etc.
iii. Prevention of infection through maintenance of aseptic technique and the use of all relevant infection preventive measures.
iv. Maintenance of adequate amount of circulating fluid.
v. Alleviation of pain through adequate rest and analgesia use.
vi. Promoting optimal functioning by restriction of patients from performing life threatening activities and prevention of bleeding and infection.
vii. Use of chemotherapy to eliminate leukemic tissues.
viii. Promoting optimal physical care
ix. Provision of adequate information about the disease process, prognosis and therapeutic regimen to the client through health education, to encourage patient's participation in their own care.

Multiple Myeloma

This is also called myelomatosis, it is a rare cancerous condition in which plasma cells in the bone marrow proliferate uncontrollably and function incorrectly. It is a malignant disease of the most mature form of B lymphocyte, the plasma cells. The proliferating plasma cells produce excessive amounts of a particular type of immunoglobulin, while the production of the other types are impaired. This makes infection more likely. The proliferation of these abnormal cells causes pain and destroys bone tissue. The affected vertebrae may collapse and compress nerves, causing numbness or paralysis. The blood calcium levels increase as bone is destroyed.

The specific immunoglobulin, secreted by the myeloma cells is detectable in the blood urine and is referred to as monoclonal protein or M protein which serves as a useful marker to monitor the extent of disease and the patient's response to therapy. The malignant plasma cells also secrete certain substances to stimulate the creation of new blood vessels to enhance the growth of these cluster of plasma cells, this process is called angiogenesis. The plasma cells can also infilterate other tissues and these are called plasmacytomas.

Signs and symptoms; Bone pain (backs or ribs) that increases with movement and decreases with rest. Presence of lytic lesions and osteoporosis in bone xrays, hypercalcaemia, renal failure, anaemia, increased susceptibility to infections, bleeding, etc.

Diagnostic indices

- Elevated monoclonal protein spike in serum, urine or light chain in the urine.
- Xrays show presence of lytic lesions.
- Complete blood count shows presence of anaemia or hypercalcaemia.
- Bone biopsy shows the presence of sheets of plasma cells.

Management

i. Adequate fluid intake to allow for optimal hydration to diminish effects of hypercalcaemia.
ii. Chemotherapy with the use of corticosteroid, cyclophosphamide, vincristine and other cytotoxic drugs.
iii. Radiotherapy in combination with other systemic treatment
iv. Vertebroplasty is usually conducted in cases of veterbral compression fracture.
v. Use of bisphosphonate to diminish the secretion of osteoclasts activating factors.
vi. Pain management with the use of NSAIDs in combination with opiod analgesics.
vii. Various infection prevention measures should be used to prevent infection.
viii. Vaccination against all opportunistic infectious conditions
ix. Provision of adequate information about the disease process, prognosis and therapeutic regimen to the client through health education, to encourage patient's participation in their own care

Agranulocytosis

This is a condition in which there is extreme shortage or absence of granulocytes. This occurs when the number of granulocytes produced does not keep pace with the normal removal of cells or when the life span of the cells is reduced. A temporary reduction occurs in response to inflammation but the numbers are usually quickly restored. Inadequate level of granulopoiesis may be caused by

- Use of drugs like cytotoxics, sulphonamides and antibiotics.
- Disease of the bone marrow e.g leukamias, anaemia.
- Irradiation damage to the granulocyte precursors in the bone marrow e.g. X-rays, radioactive isotopes
- Severe microbial infections.

Agranulocytosis predisposes to severe infections that can lead to septicaemia and death.

Clinical Manifestation; A complete blood count is the major investigation that reveals the absence of granulocytes.

Management

i. Identification of the cause is very important for effective management. If it is caused by a medication, the medication may be stopped and if it is due to an immunologic disorder, corticosteroids may be administered.
ii. The severity of the condition should be assessed
iii. The patient should be assessed comprehensively, checking the skin, oral mucosa, respiratory tract and all body systems for any deviation from normal functioning
iv. Measures to prevent infection should be employed.
v. Provide clients with highly nutritious low microbial diet.
vi. Encourage patient to ambulate wearing the high efficiency particulate air filter mask.
vii. Prevent skin dryness with the use of water soluble lubricants.
viii. Inspect sites of intravenous infusion for any discomfort or abnormalities and maintain meticulous IV site care.
ix. Vaccination against all opportunistic infectious conditions
x. Provision of adequate information about the disease process, prognosis and therapeutic regimen to the client through health education, to encourage patient's participation in their own care

UNIT 5 CARING FOR PATIENTS WITH PLATELET AND COAGULATIONDISORDERS; THROMBOCYTOPENIA, HAEMOPHILIA & DISSEMINATEDINTRAVASCULAR COAGULATION

OBJECTIVE

At the end of this unit, you should be able to:

- describe thrombocytopenia, list the causes, signs and symptoms
- explain the management of thrombocytopenia
- describe haemoplilia and its management
- discuss DIC under causes, diagnostic tests and management.

Thrombocytopenia

Thrombocytopenia means the presence of very low numbers of platelets in the circulating blood. People with thrombocytopenia have a tendency to bleed, the bleeding is usually from many small venules or capillaries, rather than from larger vessels as in hemophilia. As a result, small punctuate hemorrhages occur throughout all the body tissues. The skin of such a person displays many small, purplish blotches, giving the disease the name thrombocytopenic purpura. Platelets are especially important for repair of minute breaks in capillaries and other small vessels. Ordinarily, bleeding will not occur until the number of platelets in the blood falls below 50,000/ml, rather than the normal 150,000 to 300,000. Levels as low as 10,000/ml are frequently lethal. Even without making specific platelet counts in the blood, sometimes one can suspect the existence of thrombocytopenia if the person's blood fails to retract, because, as pointed out earlier, clot retraction is normally dependent on release of multiple coagulation factors from the large numbers of platelets entrapped in the fibrin mesh of the clot. Most people with thrombocytopenia have the disease known as idiopathic thrombocytopenia, meaning the thrombocytopenia of unknown cause. In most of these people, it has been discovered that for unknown reasons, specific antibodies have formed and react against the platelets themselves to destroy them. Relief from bleeding for 1 to 4 days can often be effected in a patient with thrombocytopenia by giving fresh whole blood transfusions that contain large numbers of platelets. Also, splenectomy is often helpful, sometimes effecting almost complete cure because the spleen normally removes large numbers of platelets from the blood.

Causes: hematological malignancy, myelodysplastic syndromes, metastatic involvement of bone marrow, aplastic anaemia, infections,

alcohol use, chemotherapy, malignant lymphoma, lupus erythrommatous, disseminated intravascular coagulation.

Signs and symptoms; bleeding and petechiae, excessive menstrual bleeding, excessive bleeding after surgery etc

Management

i. Identify cause and underlying conditions that precipitate this condition. Plan care based of the identified cause.
ii. Platelet may be administered to the patient if platelet production is impaired.
iii. Splenectomy may be performed if not contraindicated.
iv. Conduct a comprehensive general body assessment to identify deviation from normal.
v. Avoid the use of aspirin and other drugs known to inhibit platelet function.
vi. Avoid giving intramuscular injections.
vii. Do not insert indwelling catheters and do not take rectal temperature and avoid the use of suppositories or enemas. Instead use stool softners, oral laxatives to prevent complications.
viii. Use the smallest possible needles during venipuncture and apply pressure at venipuncture site until bleeding has stopped.
ix. Avoid any activity that could cause bleeding.
x. Inform other members of the health team to provide holistic care.
xi. Vaccination against all opportunistic infectious conditions
xii. Provision of adequate information about the disease process, prognosis and therapeutic regimen to the client through health education, to encourage patient's participation in their own care

Heamophilia

Hemophilia is an X-linked congenital bleeding disorder with a frequency of about one in 10,000 births. It is caused by a deficiency of coagulation factor VIII (hemophilia A) or factor IX (hemophilia B) related to mutations of the clotting factor gene. The number of affected persons worldwide is estimated to be about 400,000. Hemophilia A is more common than hemophilia B, representing 80-85% of the total. The life expectancy of persons born with hemophilia, who have access to adequate treatment, should approach normal with currently available treatment.

Diagnosis of Hemophilia

Accurate diagnosis is important and essential for effective management. Hemophilia should be suspected in patients presenting with a history of:

i. Easy bruising in early childhood
ii. Spontaneous bleeding (particularly into the joints and soft tissue)

iii. Excessive bleeding following trauma or surgery. While the history of bleeding is usually lifelong, some severe hemophilic children may not have bleeding symptoms until after the age of one or later when they begin walking and exploring their world. Patients with mild hemophilia may not have excessive bleeding unless they experience trauma or surgery.
iv. A family history of bleeding is commonly obtained. Hemophilia generally affects males on the maternal side. However, both FVIII and FIX genes are prone to new mutations, and as many as 1/3 of all patients may not have a family history of these disorders.
v. Screening tests will show a prolonged activated partial thromboplastin time (aPTT) in severe and moderate cases but may not show prolongation in mild hemophilia. A definitive diagnosis depends on factor assay to demonstrate deficiency of FVIII or FIX. It is a very rare disease that is inherited as X-linked traits thus affecting males, females are mostly carriers and almost always asymptomatic, the disease is always identified in early childhood.
vi. Heamorrhage into various part of the body, hemarthroses, hematomas, chronic pain and ankylosis if the haemorrhage is into the joint, compression of the peripheral nerves, hematuria and Gastrointestinal bleeding.

Management

Keys to improvement of health and quality of life include; Prevention of bleeding, long-term management of joint and muscle damage and other sequelae of bleeding, management of complications from treatment including: - Inhibitor development; and - Viral infection(s) transmitted through blood products requiring long-term management. Other methods of management are;

i. Transfusion of fresh frozen plasma.
ii. Infusion of the identified blood clotting factor responsible for the disorder.
iii. RICE (rest, ice, compression, and elevation) is an important adjunctive management for bleeding in muscles and joints in addition to increasing factor level with clotting factor concentrates or desmopressin in mild hemophilia A. Bleeding muscles and joints can be kept at rest by splinting, casting, or using crutches or a wheelchair. Application of cold/ice packs is useful to decrease inflammation, but ice should be wrapped in a towel and not be applied directly to the skin. It is recommended that ice be applied for 20 minutes, every four to six hours, until swelling and pain decrease.

iv. Antifibrinolytic drugs (e.g., tranexamic acid, epsilon amino caproic acid) for 5-10 days is effective as adjunctive treatment for mucosal bleeds (e.g., epistaxis, mouth bleed) and is used to decrease the use of coagulation products in dental extractions. These drugs should be avoided in renal bleeding as unlysed clots in the renal pelvis and ureter can behave like stones resulting in ureteric colic and obstructive nephropathy. Antifibrinolytic drugs should not be given concurrently with non-activated or activated prothrombin complex concentrates because of potential thrombotic complications.
v. Supporting children to cope with activities of daily living and identifying the positive aspects of their lives.
vi. Avoidance of all factors that can cause bleeding and agents that can interfere with platelet aggregation like aspirins, NSAIDs, herbs, nutritional supplements and alcohol.
vii. Analgesics can be administered to reduce pain associated with hematoma.
viii. Provision of adequate information about the disease process, prognosis and therapeutic regimen to the client through health education, to encourage patient's participation in their own care
ix. Genetic counselling is an important part of hemophilia care to help people with hemophilia, carriers, and their families make more informed choices about having children where there is a possibility of having a child with hemophilia. It includes a wide range of tests for diagnostic and carrier detection, as well as individual counselling.
x. Maintenance of oral hygiene, integrity of the mucosa and skin integrity.

Complications - Chronic hemophilic arthropathy; Chronic synovitis, Deforming arthropathy, Pseudotumour formation (soft tissue and bone), Fracture, Inhibitors against clotting factors VIII and IX, Transfusion-related infections of concern in people with hemophilia like Human immunodeficiency virus, Hepatitis B virus, Hepatitis C virus, Hepatitis A virus, Parvovirus B19, etc.

Disseminated Intravascular Coagulation (DIC)

This is majorly not a disease condition but a sign of an underlying disease condition. It is a clinicopathological syndrome which complicates a range of illnesses. It is characterised by systemic activation of pathways leading to and regulating coagulation, which can result in the generation of fibrin clots that may cause organ failure with concomitant consumption of platelets and coagulation factors that may result in clinical bleeding.

Pathogenesis

DIC never occurs in isolation and recognition that a patient has a clinical disorder which may result in the development of DIC is the key to appropriate investigation and management. DIC may arise in patients with a wide spectrum of disorders including sepsis, malignancy, trauma, liver disease and vascular anomalies. It is also seen when pregnancy is complicated by placental abruption or amniotic fluid embolism and may complicate poisoning, envenomation and major transfusion reactions. All of these conditions share the ability to induce systemic activation of coagulation either by activating cytokines as part of a systemic inflammatory response or by causing the release of, or exposure to, procoagulant substances.

The pathogenesis of DIC is complex and centres on the enhanced generation of thrombin in vivo. The contributing components include increased tissue factor expression, sub- optimal function of natural anticoagulant systems, dysregulation of fibrinolysis and increased anionic phospholipid availability.

DIC alters the normal hemostatic mechanism through the formation of massive amounts of tiny clots form in the microcirculation causing an exhaustion of the platelet and clotting factors, thus causing coagulation to fail. The excessive blood clotting triggers the fibrinolytic system to release fibrin degradation products which are potent anticoagulants, furthering the bleeding.

Diagnosis of DIC

There is no single laboratory test that can establish or rule out the diagnosis of DIC. Thus, it is of utmost importance to assess the whole clinical picture, taking into account the clinical condition of the patient, the diagnosis, and all available laboratory results. As such, a diagnosis of DIC should be made based on an appropriate clinical suspicion supported by relevant laboratory tests. Also, DIC is an extremely dynamic situation and the tests are a snapshot of this dynamic state. In addition, the underlying clinical condition can have an influence on the laboratory tests. However, a combination of tests when repeated in a patient with a clinical condition known to be associated with DIC can be used to diagnose the disorder with reasonable certainty in most cases. Possible diagnostic tests are;

- Platelet count which shows a decrease in the number of platelet
- Prothrombin time pathway shows an increase in the time.
- Partial prothrombin time, the values are higher in DIC
- Thrombin time shows an increase in the time
- Fibrinogen test, the values are reduced in DIC
- D-dimer test, this shows the local fibrinolysis and the values are increased in DIC

- Fibrin degradation products. The values are increased in DIC

Management
Key to the treatment of DIC is the specific and vigorous treatment of the underlying disorder. In many cases the DIC will spontaneously resolve when the underlying disorder is properly managed. Examples are the administration of antibiotics and/or surgical drainage in patients with DIC due to severe infection and sepsis.

Additional supportive treatment, specifically aimed at the coagulation abnormalities, may be required.

Blood component therapy should not be instituted on the basis of laboratory results alone, but is indicated in patients with active bleeding, in those requiring an invasive procedure and those who are otherwise at risk for bleeding complications. The threshold for transfusing platelets depends on the clinical state of the patient. In general, platelet transfusion is administered to patients who bleed and who have a platelet count of <50 · 109/l. In non-bleeding patients, a much lower threshold of 10–20 · 109/l is adopted based on randomised controlled trials in patients with thrombocytopenia following chemotherapy, although in patients perceived to be at high risk of bleeding based on other clinical and laboratory features.

Heparin infusion might also be given if not contraindicated

Complications: Renal failure, gangrene, pulmonary embolism, acute respiratory distress syndrome, stroke, etc.

UNIT 6 CARING FOR PATIENTS WITH LYMPHATIC DISORDER; LYMPHAGITIS & LYMPHADENITIS, LYMPHEDEMA & ELEPHANTIASIS; infectious mononucleosis & malignant lymphoma

OBJECTIVE

At the end of this unit, you should be able to:

- describe lymphagenitis and lymphadenitis
- define lymphedema and elephantiasis
- describe infectious mononucleosis and malignant lymphoma
- discuss the management of patients with lymphatic system disorders.

The lymphatic system is an accessory circulatory system as it transports a connective tissue called lymph. The flow of lymph depends on the intrinsic contractions of the lymphatic vessels which make up the lymphatic system.

Lymphagenitis and Lymphadenitis
This is an acute inflammation of the Lymphatic channel. It occurs mostly from a focus of infection on the extremities. The infectious agent is mostly haemolytic streptococcus. The associated red streaks that extends up the arm or leg from an infected wound outline the lymphatic vessels as they drain. The associated lymph nodes become enlarged, red and tender (acute lymphadenitis). They can also become necrotic, forming abscess and suppurating (suppurative lymphadenitis). The lymph nodes mostly affected are those in the cervical region, axilla and groin. Recurrent episodes of lymphangitis are often associated with progressive lymphedema.

Lymphedema and Elephantiasis
Lymphedema and its more advanced form, elephantiasis, occur primarily in the lower limbs and are commoner in women. Several factors have been implicated in the progression of lymphoedema, including repeated episodes of lymphangitis. Lymphedema may be due to filariasis infestation, heart failure, malnutrition, venous disease, podoconiosis and HIV/AIDS-associated Kaposi sarcoma, there is no agreement on its classification. In its most advanced form, elephantiasis may prevent people from carrying out their normal daily activities.

Lymphedema are classified as either primary or secondary. Tissue swelling occurs in the extremities because of an increased quantity of lymph that results from the obstruction of lymphatic vessels causing

edema which may be pitting. As the condition progresses, the edema becomes firm, non-pitting and unresponsive to treatment. The obstruction may be in the lymph nodes and the lymphatic vessels. When chronic swelling is present, there may be frequent bouts of acute infection characterized by high fever and chills and increased residual edema after the inflammation is resolved. These leads to chronic fibrosis, thickening of the subcutaneous tissues and hypertrophy of the skin. This condition in which chronic swelling of the extremities recedes only on slight elevation is called **Elephantiasis.**

Infectious Mononucleosis (glandular fever)
This is a highly contagious viral infection that spreads by direct contact. It occurs most in young adults, its incubation period is between 7-10days. The viruses multiply during this period within the epithelial cells of the pharynx and spreads to the cervical lymph node and then to other lymphoid tissues in the body. Clinical features include tonsillitis, lymphadenopathy and splenomegaly.

MalignantLymphoma
This includes **hodgkin's disease or non-hodgkins lymphoma.**

Hodgkin's Disease
There is a painless enlargement of lymph nodes throughout the body as the lymphoid tissues within then proliferates. The superficial tissues in the neck are the first to be noticed. The cause of this condition is unknown and the prognosis varies considerably, the swelling occurs across adjacent lymph nodes and to other tissues in a consistent way. The disease leads to reduced immunity because lymphocyte production is depressed and recurrent infections is common. Compression on other nearby organs may cause anaemia and other changes in leukocytes production.

Non Hodgkin's Lymphomas
This tumor may occur in any lymphoid tissue and bone marrow. They are classified according to the type of cells involved and the degree of malignancy i.e. low, intermediate or high grade.

Low-grade tumors consists of well-differentiated cells and slow progress of the disease.

High-grade tumors consists poorly differentiated cells and rapid process of the disease.

The expanding lymph nodes may compress adjacent organs causing anaemia, leucocyte abnormalities and other organ dysfunctions. Immunological deficiency leads to increased incidence of infections.

Management

Basic management for lymphatic disorders which include lymphedema and elephantiasis involves simple measures, which can usually be carried out by the patient. The complete set of measures is more complex but usually cannot be implemented in resource-poor settings. Where there is a comprehensive health system, health workers could promote use of the complete package, including compression or pressure bandages, lymphatic massage and other recognized methods. Traditional health workers should be involved in these activities whenever possible.

The affected parts should be washed twice daily with soap and clean water at room temperature and dried carefully with a clean cotton cloth or gauze. The importance of hygiene in the management of lymphoedema cannot be overstated; diligent washing may reduce the requirement for antibiotics and prevent progression of lymphoedema.

Adequate Skin care should be accorded. An intact skin provides an effective barrier against infection. Elevation and exercise of the affected should be encouraged as the affected limb should be raised at night and when possible during the day and exercised regularly with low-intensity movement of the joints. As immobility significantly worsens the condition, patients should be encouraged to keep moving.

Foot care should be accorded. The nails and spaces between the toes must be kept clean. Comfortable shoes should be worn to protect the skin.

Medicated creams or antibiotics (e.g. antiseptics, antifungal and antibiotic creams) should be used to treat small wounds or abrasions. For patients with elephantiasis, antifungal creams can help prevent fungal infections in deep folds and in the interdigital spaces.

Pharmacologically, an initial dose of diuretic furosemide (lasix) may be given to prevent fluid overload due to immobilization of extracellular fluid as the drug limits capillary filteration and decreases circulating blood volume.

Surgery can be performed if other medical management have failed.
Provision of adequate information about the disease process, prognosis and therapeutic regimen to the client through health education, to encourage patient's participation in their own care.

REFERENCES/FURTHER READING

Fuad, I. & Welday, M. (2008); Pathophysiology. Ethiopia Public Health Training Initiative. Ethiopia; Hirmaya University.

Famakinwa, O.T. Synopsis of medicosurgical Nursing. Ibadan; Bayosore.

Heyman, P. Anatomy of the immune system supplemental reading.

Smettzer, C.S. ,Bare, B.G, Hinkle, L.J. &Cheever, H.K., (2008). Brunner & Suddarth's textbook of Medical-Surgical Nursing. (11th Ed). India; Wolters Kluwer.

Waugh, A., Grant, A. (2006). Ross & Wilson Anatomy and Physiology in Health and Illness (10th Ed). Philadelphia; Elsevier.

2.0 OBJECTIVES

At the end of this unit you should be able to:

- explain cardiac physiology in relation to cardiac anatomy and the conduction system of the heart
- incorporate assessment of functional health patterns and cardiac risk factors into the health history and physical assessment of the patient with cardiac conditions
- identify the clinical significance and related nursing implications of the various tests and procedures used for diagnostic assessment of cardiac function.

3.0 MAIN CONTENT

3.1 Anatomic and Physiologic Overview

The heart is a hollow, muscular organ located in the center of the thorax, where it occupies the space between the lungs (mediastinum) resting on the diaphragm. The heart weighs approximately 300 g (10.6 oz), although heart weight and size are influenced by age, gender, body weight, extent of physical exercise and conditioning, and heart disease. The heart pumps blood to the tissues, supplying them with oxygen and other nutrients.
The pumping action of the heart is accomplished by the rhythmic contraction and relaxation of its muscular wall.

During **systole** (contraction of the muscle), the chambers of the heart become smaller as the blood is ejected. During **diastole** (relaxation of the muscle), the heart chambers fill with blood in preparation for the subsequent ejection. A normal adult heart beats approximately 60 to 80 times per minute at rest. Each ventricle ejects approximately 70 ml of blood per beat and has an output of approximately 5 L per minute.

Anatomy of the Heart
The heart is composed of three layers. The inner layer, or **endocardium,** consists of endothelial tissue and lines the inside of the heart and valves. The middle layer, or **myocardium**, is made up of muscle fibers and is responsible for the pumping action. The exterior layer of the heart is called the **epicardium**. The heart is encased in a thin, fibrous sac called the **pericardium**, which is composed of two layers. Adhering to the epicardium is the visceral pericardium. Enveloping the visceral pericardium is the parietal pericardium, a tough fibrous tissue that attaches to the great vessels, diaphragm, sternum, and vertebral column and supports the heart in the mediastinum. The space between these two

layers (pericardial space) is filled with about 30 ml of fluid, which lubricates the surface of the heart and reduces friction during systole.

Heart Chambers

The four chambers of the heart constitute the right- and left- sided pumping systems. The right side of the heart, made up of the right atrium and right ventricle, distributes venous blood(deoxygenated blood) to the lungs via the pulmonary artery (pulmonary circulation) for oxygenation. The right atrium receives blood returning from the superior vena cava (head, neck, and upper extremities), inferior vena cava (trunk and lower extremities), and coronary sinus (coronary circulation). The left side of the heart, composed of the left atrium and left ventricle, distributes oxygenated blood to the remainder of the body via the aorta (systemic circulation). The left atrium receives oxygenated blood from the pulmonary circulation via the pulmonary veins.

The varying thicknesses of the a trial and ventricular walls relate to the workload required by each chamber. The atria are thin-walled because blood returning to these chambers generates low pressures. In contrast, the ventricular walls are thicker because they generate greater pressures during systole. The right ventricle contracts against low pulmonary vascular pressure and has thinner walls than the left ventricle. The left ventricle, with walls two-and-a-half times more muscular than those of the right ventricle, contracts against high systemic pressure.

Because the heart lies in a rotated position within the chest cavity, the right ventricle lies anteriorly (just beneath the sternum) and the left ventricle is situated posteriorly. The left ventricle is responsible for the apex beat or the point of maximum impulse (PMI), which is normally palpable in the left midclavicular line of the chest wall at the fifth intercostal space.

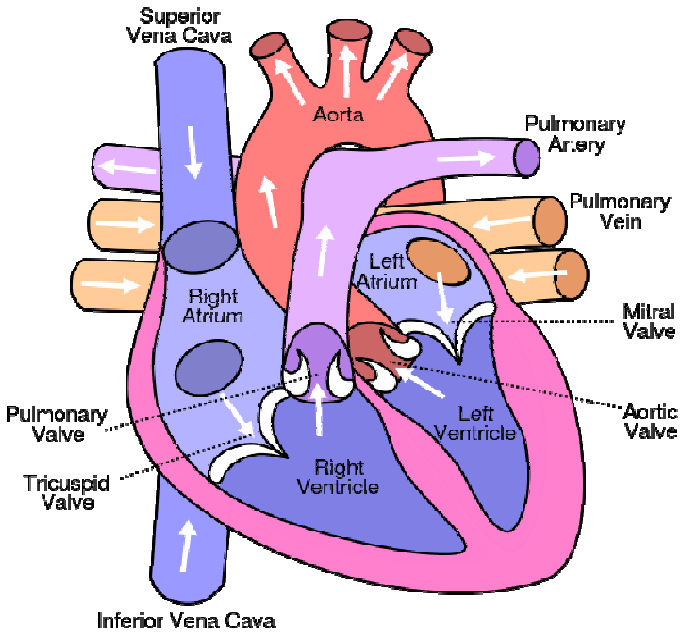


Diagram of the Human Heart

3.2 Heart Valves

The four valves in the heart permit blood to flow in only one direction. The valves, which are composed of thin leaflets of fibrous tissue, open and close in response to the movement of blood and pressure changes within the chambers. There are two types of valves: atrioventricular and semi lunar.

ATRIOVENTRICULAR VALVES

The valves that separate the atria from the ventricles are termed atrioventricular valves. The tricuspid valve, so named because it is composed of three cusps or leaflets, separates the right atrium from the right ventricle. The mitral, or bicuspid (two cusps) valve, lies between the left atrium and the left ventricle.

Normally, when the ventricles contract, ventricular pressure rises, closing the atrioventricular valve leaflets. Two additional structures, the papillary muscles and the chordae tendineae,maintain valve closure. The papillary muscles, located on the sides of the ventricular walls, are connected to the valve leaflets by thin fibrous bands called chordae tendineae. During systole, contraction of the papillary muscles causes the chordae tendineae to become taut, keeping the valve leaflets approximated and closed.

Coronary Arteries

The left and right coronary arteries and their branches supply arterial blood to the heart. These arteries originate from the aorta just above the aortic valve leaflets. The heart has large metabolic requirements,

extracting approximately 70% to 80% of the oxygen delivered (other organs consume, on average, 25%).

Unlike other arteries, the coronary arteries are perfuse during diastole. An increase in heart rate shortens diastole and can decrease myocardial perfusion. Patients, particularly those with coronary artery disease (CAD), can develop **myocardial ischemia** (inadequate oxygen supply) when the heart rate accelerates.

The left coronary artery has three branches. The artery from the point of origin to the first major branch is called the left main coronary artery. Two bifurcations arise off the left main coronary artery. These are the left anterior descending artery, which courses down the anterior wall of the heart, and the circumflex artery, which circles around to the lateral left wall of the heart.

The right side of the heart is supplied by the right coronary artery, which progresses around to the bottom or inferior wall of the heart. The posterior wall of the heart receives its blood supplyby an additional branch from the right coronary artery called the posterior descending artery.
Superficial to the coronary arteries are the coronary veins. Venous blood from these veins returns to the heart primarily through the coronary sinus, which is located posteriorly in the right atrium.

Cardiac Muscle

The myocardium is composed of specialized muscle tissue. Microscopically, myocardial muscle resembles striated (skeletal) muscle, which is under conscious control. Functionally, however,
myocardial muscle resembles smooth muscle because its contraction is involuntary. The myocardial muscle fibers are arranged in an interconnected manner (called a syncytial) that allows for coordinated myocardial contraction and relaxation. The sequential pattern of contraction and relaxation of individual muscle fibers ensures the rhythmic behavior of the myocardium as a whole and enables it to function as an effective pump.

3.3 Function of the Heart

Conduction System

The specialized heart cells of the **cardiac conduction system** methodically generate and coordinate the transmission of electrical impulses to the myocardial cells. The result is sequential atrioventricular contraction, which provides for the most effective flow of blood, thereby

optimizing cardiac output. Three physiologic characteristics of the cardiac conduction cells account for this coordination:

- *Automaticity:* ability to initiate an electrical impulse
- *Excitability:* ability to respond to an electrical impulse
- *Conductivity:* ability to transmit an electrical impulse from one cell to another

The **Sinoatrial (SA) node**, referred to as the primary pacemaker of the heart, is located at the junction of the superior vena and the right atrium. In a normal resting heart the SA node has an inherent firing rate of 60 to 100 impulses per minute, but the rate can change in response to the metabolic demands the body.

The electrical impulses initiated by the SA node are conducted along the myocardial cells of the atria via specialized tracts called internodal pathways. The impulses cause electrical stimulation and subsequent contraction of the atria. The impulses are then conducted to the atrioventricular (AV) node. The AV node (located in the right a trial wall near the tricuspid valve) consists of another group of specialized muscle cells similar to those of the SA node. The AV node coordinates the incoming electrical impulses from the atria and, after a slight delay (allowing the atria time to contract and complete ventricular filling), relays the impulse to the ventricles.

This impulse is then conducted through a bundle of specialized conduction cells (bundle of His) that travel in the septum separating the left and right ventricles.

The bundle of His divides into the right bundle branch (conducting impulses to the right ventricle) and the left bundle branch (conducting impulses to the left ventricle). To transmit impulses to the largest chamber of the heart, the left bundle branch bifurcates into the left anterior and left posterior bundle branches. Impulses travel through the bundle branches to reach the terminal point in the conduction system, called the Purkinje fibers. This is the point at which the myocardial cells are stimulated, causing ventricular contraction.

The heart rate is determined by the myocardial cells with the fastest inherent firing rate. Under normal circumstances, the SA node has the highest inherent rate, the AV node has the second highest inherent rate (40 to 60 impulses per minute), and the ventricular pacemaker sites have the lowest inherent rate (30 to 40 impulses per minute). If the SA node malfunctions, the AV node generally takes over the pacemaker function of the heart at its inherently lower rate. Should both the SA and the AV nodes fail in their pacemaker function, a pacemaker site in the ventricle will fire at its inherent bradycardic rate of 30 to 40 impulses per minute.

Physiology of Cardiac Conduction

Cardiac electrical activity is the result of the movement of ions (charged particles such as sodium, potassium, and calcium) across the cell membrane. The electrical changes recorded within a single cell result in what is known as the *cardiac action potential*. In the resting state, cardiac muscle cells are polarized, which means an electrical difference exists between the negatively charged inside and the positively charged outside of the cell membrane.

As soon as an electrical impulse is initiated, cell membrane permeability changes and sodium moves rapidly into the cell, while potassium exits the cell. This ionic exchange begins **depolarization** (electrical activation of the cell), converting the internal charge of the cell to a positive one.

Contraction of the myocardium follows depolarization. The interaction between changes in membrane voltage and muscle contraction is called electromechanical coupling. As one cardiac muscle cell is depolarized, it acts as a stimulus to its neighboring cell, causing it to depolarize. Sufficient depolarization of a single specialized conduction system cell results in depolarization and contraction of the entire myocardium.

Repolarization (return of the cell to its resting state) occurs as the cell returns to its baseline or resting state; this corresponds to relaxation of myocardial muscle. After the rapid influx of sodium into the cell during depolarization, the permeability of the cell membrane to calcium is changed.

Calcium enters the cell and is released from intracellular calcium stores. The increase in calcium, which occurs during the plateau phase of repolarization, is much slower than that of sodium and continues for a longer period.

Cardiac muscle, unlike skeletal or smooth muscle, has a prolonged refractory period during which it cannot be restimulated to contract. There are two phases of the refractory period, referred to as the absolute refractory period and the relative refractory period. The absolute refractory period is the time during which the heart cannot be restimulated to contract regardless of the strength of the electrical stimulus. This period corresponds with depolarization and the early part of repolarization. During the latter part of repolarization, however, if the electrical stimulus is stronger than normal, the myocardium can be stimulated to contract. This short period at the end of repolarization is called the relative refractory period.

Cardiac Hemodynamic
An important determinant of blood flow in the cardiovascular system is the principle that fluid flows from a region of higher pressure to one of lower pressure. The pressures responsible for blood flow in the normal circulation are generated during systole and diastole, and in the four chambers of the heart during systole and diastole.

Cardiac Cycle
Beginning with systole, the pressure inside the ventricles rapidly rises, forcing the atrioventricular valves to close. As a result, blood ceases to flow from the atria into the ventricles and regurgitation (backflow) of blood into the atria is prevented. The rapid rise of pressure inside the right and left ventricles forces the pulmonic and aortic valves to open, and blood is ejected into the pulmonary artery and aorta, respectively. The exit of blood is at first rapid; then as the pressure in each ventricle and its corresponding artery equalizes, the flow of blood gradually decreases.

At the end of systole, pressure within the right and left ventricles rapidly decreases. This lowers pulmonary artery and aortic pressure, causing closure of the semi lunar valves. These events mark the onset of diastole.

During diastole, when the ventricles are relaxed and the atrioventricular valves are open, blood returning from the veins flows into the atria and then into the ventricles. Toward the end of this diastolic period, the atrial muscles contract in response to an electrical impulse initiated by the SA node (atrial systole). The resultant contraction raises the pressure inside the atria, ejecting blood into the ventricles.

Cardiac Output
This is the amount of blood pumped by each ventricle during a given period. The cardiac output in a resting adult is about 5 L per minute but varies greatly depending on the metabolic needs of the body. Cardiac output is computed by multiplying the stroke volume by the heart rate.

Stroke Volume is the amount of blood ejected per heartbeat. The average resting stroke volume is about 70 ml, and the heart rate is 60 to 80 beats per minute (bpm). Cardiac output can be affected by changes in either stroke volume or heart rate.

3.4 Health History and Clinical Manifestations

For the patient experiencing an acute Myocardial Infarction, the nurse obtains the health history using a few specific questions about the onset and severity of chest discomfort, associated symptoms, current medications, and allergies. At the same time, the nurse observes the

patient's general appearance and evaluates hemodynamic status (heart rate and rhythm, BP). Once the condition of the patient stabilizes, a more extensive history can be obtained.

With stable patients, a complete health history is obtained during the initial contact. Often, it is helpful to have a significant other, like the patient's spouse or partner present during the health history interview. Initially, demographic information regarding age, gender, and ethnic origin is obtained. The family history, as well as the physical examination, should include assessment of height and weight.

The baseline information derived from the history assists in identifying pertinent issues related to the patient's condition, educational and self-care needs. Once these problems are clearly identified, a plan of care is instituted. During subsequent contacts or visits with the patient, a more focused health history is performed to determine whether goals have been met, whether the plan needs to be modified, and whether new problems have developed.

During the interview, the nurse asks questions to evaluate cardiac symptoms and health status.

Cardiac Signs and Symptoms

Patients with cardiovascular disorders commonly have one or more of the following signs and symptoms:

- Chest pain or discomfort (angina pectoris, Myocardis Infaction (MI), valvular heart disease)
- Shortness of breath or dyspnea (MI, left ventricular failure, Heart Failure (HF)
- Edema and weight gain (right ventricular failure, HF)
- Palpitations (dysrhythmias resulting from myocardial ischemia, valvular heart disease, ventricular aneurysm, stress, electrolyte imbalance)
- Fatigue (earliest symptom associated with several cardiovascular disorders)
- Dizziness and syncope or loss of consciousness (postural hypotension, dysrhythmias, vasovagal effect, cerebrovascular disorders)

Not all chest discomfort is related to myocardial ischemia. When a patient has chest discomfort, questions should focus on differentiating a serious, life-threatening condition such as MI from conditions that are less serious or that would be treated differently.

The following points should be remembered when assessing patients with cardiac symptoms:

- Women are more likely to present with atypical symptoms of MI than are men.
- There is little correlation between the severity of the chest discomfort and the gravity of its cause. Elderly people and those with diabetes may not have pain with angina or MI because of neuropathies. Fatigue and shortness of breath may be the predominant symptoms in these patients.
- There is poor correlation between the location of chest discomfort and its source.
- The patient may have more than one clinical condition occurring simultaneously.
- In a patient with a history of Coronary Artery Disease (CAD), the chest discomfort should be assumed to be secondary to ischemia until proven otherwise.

People experiencing myocardial ischemia can have a variety of symptoms. The typical symptom is angina presenting as pressure, fullness, squeezing pain, or discomfort in the center of the chest. This pain may radiate to the shoulders, neck, jaw, or arms. Angina can also have an atypical or uncommon presentation, referred to as *anginal equivalent*. It is characterized by shortness of breath, fatigue, weakness, or pain in other parts of the upper body, including the neck, shoulder, jaw, arm, back, or stomach.

Angina patterns are usually predictable (e.g., with activity). Rest or sublingual Nitroglycerin relieves symptoms within a few minutes. A patient in the midst of an MI, however, can present with angina or its equivalent symptoms, which last longer than 15 minutes. Signs and symptoms associated with an MI include lightheadedness, fainting, diaphoresis, unexplained anxiety, nausea, and shortness of breath. Symptoms are unrelieved by rest or nitroglycerin.

Nursing Assessments

Family History Assessment

- Assess all patients with cardiovascular symptoms for coronary artery disease, regardless of age (early-onset CAD occurs).
- Assess family history of sudden death in persons who may or may not have been diagnosed with coronary disease (especially of early onset).
- Ask about sudden death in a previously asymptomatic child, adolescent, or adult.
- Ask about other family members with biochemical or neuromuscular conditions (e.g., hemochromatosis or muscular dystrophy).
- Assess whether DNA mutation or other genetic testing has been performed on an affected family member

- Assess for signs and symptoms of hyperlipidemias (xanthomas, corneal arcus, abdominal pain of unexplained origin).
- Assess for muscular weakness.

Health Perception and Management

In an effort to determine how patients perceive their current health status. The nurse might ask some of the following questions:

1. Do you have any health problems? If so, what do you think caused them?
2. How has your health been recently? Have you noticed any changes from last year? from 5 years ago?
3. Do you have a cardiologist or primary health care provider?
4. How often do you go for checkups?
5. Do you use tobacco or consume alcohol?
6. What are your risk factors for heart disease? What do you do to stay healthy and take care of your heart?
7. What prescription and over-the-counter medications are you taking? Do you take vitamins or herbal supplements?
8. Some patients may not be aware of their own medical diagnosis. For example, patients may not realize that their heart attack was caused by CAD.
9. The patient's ability to recognize cardiac symptoms and to know what to do when they occur is essential for effective self-care management.
10. All too often, patients' new symptoms or symptoms of progressing cardiac dysfunction go unrecognized. This results in prolonged delays in seeking life-saving treatment. Major barriers to seeking prompt medical care include lack of knowledge about symptoms to expect with heart disease, attribution of symptoms to a benign source, psychological factors such as denial of symptom significance, and social factors, specifically feeling embarrassed about having symptoms (Zerwic,1999).
11. An additional issue to consider is the patient's medication history, dosages, and schedules. Is the patient independent in taking medications? Are the medications taken as prescribed? Does the patient understand why the medication regimen is important?
12. Are doses ever forgotten or skipped, or does the patient ever decide to stop taking a medication? An aspirin a day is a common nonprescription medication that improves patient outcomes after an MI. However, if patients are not aware of this benefit, they may be inclined to stop taking aspirin if they think it is a trivial medication.

13. A careful medication history will often uncover common medication errors and causes for non adherence to the medication regimen.

Nutrition and Metabolism

Dietary modifications, exercise, weight loss, and careful monitoring are important strategies for managing three major cardiovascular risk factors: hyperlipidemia, hypertension, and hyperglycemia (diabetes mellitus).

Diets that are restricted in sodium, fat, cholesterol, and/or calories are commonly prescribed. The nurse should obtain the following information:

- The patient's current height and weight (to determine body mass index), waist measurement (assessment for obesity), BP, and any laboratory test results such as blood glucose, glycosylated hemoglobin (diabetes), total blood cholesterol high-density and low-density lipoprotein levels, and triglyceride levels (hyperlipidemia).
- How often the patient self-monitors BP, blood glucose, and weight as appropriate to the medical diagnoses.
- The patient's level of awareness regarding his or her target goals for each of the risk factors and any problems achieving or maintaining these goals.
- What the patient normally eats and drinks in a typical day and any food preferences (including cultural or ethnic preferences).
- Eating habits (canned or commercially prepared foods versus fresh foods, restaurant cooking versus home cooking, assessing for high sodium foods, dietary intake of fats).

Elimination

Typical bowel and bladder habits need to be determined.

Nocturia (awakening at night to urinate) is common for patients with HF.

Fluid collected in the dependent tissues (extremities) during the day redistributes into the circulatory system once the patient is recumbent at night. The increased circulatory volume is excreted by the kidneys (increased urine production).

Patients need to be aware of their response to diuretic therapy and any changes in urination.

This is vitally important for patients with HF. Patients may be taught to modify (titrate) their dose of diuretics based on urinary pattern, daily weight, and symptoms of dyspnea.

To avoid straining, patients who become easily constipated need to establish a regular bowel regimen. When straining, the patient tends to bear down (the Valsalva maneuver), which momentarily increases pressure on the baroreceptors. This triggers a vagal response, causing the heart rate to slow down and resulting in syncope in some patients. For the same reason, straining during urination should be avoided. Because many cardiac medications can cause gastrointestinal side effects or bleeding, the nurse asks about bloating, diarrhea, constipation, stomach upset, heartburn, loss of appetite, nausea, and vomiting.

Activity and Exercise

As the nurse assesses the patient's activity and exercise history, it is important to note that decreases in activity tolerance are typically gradual and may go unnoticed by the patient. Therefore, the nurse needs to determine whether there has been a change in the activity pattern during the last 6 to 12 months. The patient's subjective response to activity is an essential assessment parameter. New symptoms or a change in the usual angina or angina equivalent during activity is a significant finding.

Fatigue, associated with low ejection fraction and certain medications (e.g., beta-blockers), can result in activity intolerance. Patients with fatigue may benefit from having their medications adjusted and learning energy conservation techniques.

Additional areas to ask about include possible architectural barriers and challenges in the home, and what the patient does for exercise. If the patient exercises, the nurse asks additional questions:

What is the intensity, and how long and how often is exercise performed? Has the patient ever participated in a cardiac rehabilitation program?

Sleep and Rest

Clues to worsening cardiac disease, especially Heart Failure, can be revealed by sleep-related events. Determining where the patient sleeps or rests is important. Recent changes, such as sleeping upright in a chair instead of in bed, increasing the number of pillows used, awakening short of breath at night (paroxysmal nocturnal dyspnea [PND]), or awakening with angina (nocturnal angina), are all indicative of worsening Heart Failure.

Cognition and Perception

Evaluating cognitive ability helps to determine whether the patient has the mental capacity to manage safe and effective self care. Is the patient's short-term memory intact? Is there any history of dementia? Is there evidence of depression or anxiety? Can the patient read? Can the

patient read English? What is the patient's reading level? What is the patient's preferred learning style? What information does the patient perceive as important? Providing the patient with written information can be a valuable part of patient education, but only if the patient can read and comprehend the information. Related assessments include possible hearing or visual impairments. If vision is impaired, patients with HF may not be able to weigh themselves independently nor records of weight, BP, pulse, or other data requested by the health care team.

Self-Perception and Self-Concept

Personality factors are associated with the development of and recovery from CAD. Most commonly cited is "type A behavior," which is characterized by competitive, hard-driving behaviors and a sense of time urgency. Although this behaviour is not an independent risk factor for CAD, anger and hostility (personality traits common in people with "type A behavior") do affect the heart. People with these traits react to frustrating situations with an increase in BP, heart rate, and neuro-endocrine responses. This physiologic activation, called cardiac reactivity, is thought to trigger acute cardiovascular events (Woods et al., 1999).

During the health history, the nurse discovers how patients feel about themselves by asking questions such as: How would you describe yourself? Have you changed the way you feel about yourself since your heart attack or surgery? Do you find that you are easily angered or hostile? How do you feel right now? What helps to manage these feelings? To fully evaluate this health pattern, assistance from a psychiatric clinical nurse specialist, psychologist, or psychiatrist may be necessary.

Roles and Relationships

Determining the patient's social support systems is of vital importance in today's health care environment. Hospital stays for cardiac illnesses have shortened. Many invasive diagnostic cardiac procedures, such as cardiac catheterization and percutaneous transluminal coronary angioplasty (PTCA) are performed as outpatient procedures.

Patients are discharged back into the community with activity limitations, such as driving restrictions, and with greater nursing care and educational needs. These needs have significant implications for people who are independent under normal circumstances, and for people who are at higher risk for problems, such as older adults.

To assess support systems, the nurse needs to ask: Who is the primary caregiver? With whom does the patient live? Are there adequate services in place to provide a safe home environment?

The nurse also assesses for any significant effects the cardiac illness has had on the patient's role in the family. Are there adequate finances and health insurance? The answers to these questions will assist the nurse in developing a plan to meet the patient's home care needs.

Sexuality and Reproduction

Although people recovering from cardiac illnesses or procedures are concerned about sexual activity, they are less likely to ask their nurse or other health care provider for information to help them resume their normal sex life. Lack of correct information and fear lead to reduced frequency and satisfaction with sexual activity.

Therefore, nurses need to initiate this discussion with patients and not wait for them to bring it up in conversation. At first, inform the patient that it is common for people with similar heart problems to worry about resuming sexual activity. Then ask the patient to talk about his or her concerns.

The most commonly cited reasons for changes in sexual activity are fear of another heart attack or sudden death; untoward symptoms such as angina, dyspnea, or palpitations; and problems as a side effect of cardiac medications (beta-adrenergic blocking agents) and may prompt patients to stop taking them. Other medications can be substituted, so patients should be encouraged to discuss this problem with their health care provider. Often, patients and their partners do not have adequate information about the physical demands related to sexual activity and ways in which these demands can be modified. The physiologic demands are greatest during orgasm, reaching 5 or 6 metabolic equivalents (METs).

This level of activity is equivalent to walking 3 to 4 miles per hour on a treadmill. The METs expended before and after orgasm are considerably less, at 3.7 METs (Steinke, 2000). Having this information may make patients and their partners more comfortable with resuming sexual activity.

A reproductive history is necessary for women of childbearing age, particularly those with seriously compromised cardiac function. These women may be advised by their physicians not to become pregnant. The reproductive history includes information about previous pregnancies, plans for future pregnancies, oral contraceptive use (especially in women older than 35 years of age who are smokers), and use of hormone replacement therapy.

Coping and Stress Tolerance

It is important to determine the presence of psychosocial factors that adversely affect cardiac health. Anxiety, depression, and stress are known to influence both the development of and recovery from CAD. High levels of anxiety are associated with an increased incidence of CAD and increased in-hospital complication rates after MI. People with depression have an increased risk of MI and heart disease–related death, compared to people without depression.

It is postulated that people who are depressed feel hopeless and are less motivated to make lifestyle changes and follow treatment plans, explaining the association between mortality and depression (Buselli & Stuart, 1999).

Stress initiates a variety of physiologic responses, including increases in the circulation of catecholamines and cortisol, and has been strongly linked to cardiovascular events. Therefore, patients need to be assessed for presence of negative and positive emotions, as well as sources of stress. This is achieved by asking questions about recent or ongoing stressors, previous coping styles and effectiveness, and the patient's perception of his or her current mood and coping ability. To adequately evaluate this health pattern, consultation with a psychiatric clinical nurse specialist, psychologist, or psychiatrist may be indicated.

Physical Assessment

A physical examination is performed to confirm the data obtained in the health history. In addition to observing the patient's general appearance, a cardiac physical examination should include an evaluation of the following:

- Effectiveness of the heart as a pump
- Filling volumes and pressures
- Cardiac output
- Compensatory mechanisms.

Indications that the heart is not contracting sufficiently or functioning effectively as a pump include reduced pulse pressure, cardiac enlargement, and murmurs and gallop rhythms (abnormal heart sounds). The amount of blood filling the atria and ventricles and the resulting pressures (called filling volumes and pressures) are estimated by the degree of jugular vein distention and the presence or absence of congestion in the lungs, peripheral edema, and postural changes in BP that occur when the individual sits up or stands.

Cardiac output is reflected by cognition, heart rate, pulse pressure, color and texture of the skin, and urine output. Examples of compensatory

mechanisms that help maintain cardiac output are increased filling volumes and elevated heart rate.

Note that the findings on the physical examination are correlated with data obtained from diagnostic procedures, such as hemodynamic monitoring.
The examination, which proceeds logically from head to toe, can be performed in about 10 minutes with practice and covers the following areas: (1) general appearance, (2) cognition, (3) skin, (4) BP, (5) arterial pulses, (6) jugular venous pulsations and pressures, (7) heart, (8) extremities, (9) lungs, and (10) abdomen.

General appearance and cognition
The nurse observes the patient's level of distress, level of consciousness, and thought processes as an indication of the heart's ability to propel oxygen to the brain (cerebral perfusion). The nurse also observes for evidence of anxiety, along with any effects emotional factors may have on cardiovascular status. The nurse attempts to put the anxious patient at ease throughout the examination.

Inspection of the Skin
Examination of the skin begins during the evaluation of the general appearance of the patient and continues throughout the assessment. It includes all body surfaces, starting with the head and finishing with the lower extremities. Skin color, temperature, and texture are assessed. The more common findings associated with cardiovascular disease are as follows.

- Pallor—a decrease in the color of the skin—is caused by lack of oxyhemoglobin. It is a result of anemia or decreased arterial perfusion. Pallor is best observed around the fingernails, lips, and oral mucosa. In patients with dark skin, the nurse observes the palms of the hands and soles of the feet.
- Peripheral cyanosis—a bluish tinge, most often of the nails and skin of the nose, lips, earlobes, and extremities—suggests decreased flow rate of blood to a particular area, which allows more time for the hemoglobin molecule to become desaturated. This may occur normally in peripheral vasoconstriction associated with a cold environment, in patients with anxiety, or in disease states such as HF.
- Central cyanosis—a bluish tinge observed in the tongue and buccal mucosa—denotes serious cardiac disorders (pulmonary edema and congenital heart disease) in which venous blood passes through the pulmonary circulation without being oxygenated.
- Xanthelasma—yellowish, slightly raised plaques in the skin—may be observed along the nasal portion of one or both eyelids

and may indicate elevated cholesterol levels (hypercholesterolemia).

- Reduced skin turgor occurs with dehydration and aging.
- Temperature and moistness are controlled by the autonomic nervous system. Normally the skin is warm and dry. Under stress, the hands may become cool and moist. In cardiogenic shock, sympathetic nervous system stimulation causes vasoconstriction, and the skin becomes cold and clammy. During an acute MI, diaphoresis is common.
- Ecchymosis (bruise)—a purplish-blue color fading to green, yellow, or brown over time—is associated with blood outside of the blood vessels and is usually caused by trauma. Patients who are receiving anticoagulant therapy should be carefully observed for unexplained ecchymosis. In these patients, excessive bruising indicates prolonged clotting times (prothrombin or partial thromboplastin time) caused by an anticoagulant dosage that is too high.
- Wounds, scars, and tissue surrounding implanted devices should also be examined. Wounds are assessed for adequate healing, and any scars from previous surgeries are noted.

Blood Pressure

Systemic arterial BP is the pressure exerted on the walls of the arteries during ventricular systole and diastole. It is affected by factors such as cardiac output, distention of the arteries, and the volume, velocity, and viscosity of the blood. BP usually is expressed as the ratio of the systolic pressure over the diastolic pressure, with normal adult values ranging from 100/60 to 140/90 mm Hg. The average normal BP usually cited is 120/80 mm Hg. An increase in BP above the upper normal range is called hypertension, whereas a decrease below the lower range is called hypotension.

Blood pressure measurement

BP can be measured with the use of invasive arterial monitoring systems (discussed later) or noninvasively by a sphygmomanometer and stethoscope or by an automated BP monitoring device. A detailed description of the procedure for obtaining BP can be found in nursing skills textbooks, and specific manufacturer's instructions review the proper use of the automated monitoring devices. Several important details must be observed to ensure that BP measurements are accurate.

Pulse pressure

The difference between the systolic and the diastolic pressures is called the pulse pressure. It is a reflection of stroke volume, ejection velocity, and systemic vascular resistance. Pulse pressure, which normally is 30 to 40 mm Hg, indicates how well the patient maintains cardiac output.

The pulse pressure increases in conditions that elevate the stroke volume (anxiety, exercise, bradycardia), reduce systemic vascular resistance (fever), or reduce distensibility of the arteries (atherosclerosis, aging, hypertension). Decreased pulse pressure is an abnormal condition reflecting reduced stroke volume and ejection velocity (shock, HF, hypovolemia, mitral regurgitation) or obstruction to blood flow during systole (mitral or aortic stenosis). A pulse pressure of less than 30 mm Hg signifies a serious reduction in cardiac output and requires further cardiovascular assessment

Ensuring accurate blood pressure measurement

- Cuff size must be appropriate for the patient. (The cuff size should have a bladder width at least 40% and length at least 80% of limb circumference.) The average adult cuff is 12 to 14 cm wide and 30 cm long. Using a cuff that is too small will give a high reading, whereas, too large a cuff results in a falsely low reading.
- Calibration of the sphygmomanometer should be performed routinely to ensure accuracy of blood pressure reading.
- Cuff is firmly wrapped around the arm, and cuff bladder is centered over the brachial artery.
- Patient's arm should be at heart level.
- Position of the patient and site of blood pressure measurement (e.g., RA for right arm) are recorded.
- Palpation of the systolic pressure before auscultation helps to detect an auscultatory gap more readily.
- The patient is asked not to talk during blood pressure measurements.

A significant increase in blood pressure and heart rate occurs when subjects are talking.

Heart Inspection and Palpation

The heart is examined indirectly by inspection, palpation, percussion, and auscultation of the chest wall. A systematic approach is the cornerstone of a thorough assessment. Examination of the chest wall is performed in the following six areas:

1. *Aortic area*—second intercostal space to the right of the sternum. To determine the correct intercostal space, start at the angle of Louis by locating the bony ridge near the top of the sternum, at the junction of the body and the manubrium. From this angle, locate the second intercostal space by sliding one finger to the left or right of the sternum. Subsequent intercostal spaces are located from this reference point by palpating down the rib cage.
2. *Pulmonic area*—second intercostal space to the left of the sternum.
3. *Erb's point*—third intercostal space to the left of the sternum.

4. *Right ventricular or tricuspid area*—fourth and fifth intercostal spaces to the left of the sternum.
5. *Left ventricular or apical area*—the PMI, location on the chest where heart contractions can be palpated.
6. *Epigastric area*—below the xiphoid process.

For most of the examination, the patient lies supine, with the head slightly elevated. The right-handed examiner is positioned at the right side of the patient and the left-handed examiner at the left side.

In a systematic fashion, each area of the precordium is inspected and then palpated. Oblique lighting is used to assist the examiner in identifying subtle pulsation. A normal impulse that is distinct and located over the apex of the heart is called the **apical impulse** (PMI). It may be observed in young people and in older people who are thin. The apical impulse is normally located and auscultated in the left fifth intercostal space in the midclavicular line.

In many cases, the apical impulse is palpable and is normally felt as a light pulsation, 1 to 2 cm in diameter. It is felt at the onset of the first heart sound and lasts for only half of systole.

The nurse uses the palm of the hand to locate the apical impulse initially and the finger pads to assess its size and quality. A broad and forceful apical impulse is known as a left ventricular heave or lift. It is so named because it appears to lift the hand from the chest wall during palpation.

An apical impulse below the fifth intercostal space or lateral to the midclavicular line usually denotes left ventricular enlargement from left ventricular failure. Normally, the apical impulse is palpable in only one intercostal space; palpability in two or more adjacent intercostal spaces indicates left ventricular enlargement.

If the apical impulse can be palpated in two distinctly separate areas and the pulsation movements are paradoxical (not simultaneous), a ventricular aneurysm should be suspected.

Abnormal, turbulent blood flow within the heart may be palpated with the palm of the hand as a purring sensation. This phenomenon is called a thrill and is associated with a loud murmur.

A thrill is always indicative of significant pathology within the heart. Thrills also may be palpated over vessels when blood flow is significantly and substantially obstructed and over the carotid arteries if aortic stenosis is present or if the aortic valve is narrowed.

Chest Percussion
Normally, only the left border of the heart can be detected by percussion. It extends from the sternum to the midclavicular line in the third to fifth intercostal spaces. The right border lies under the right margin of the sternum and is not detectable. Enlargement of the heart to either the left or right usually can be noted.

In people with thick chests, obesity, or emphysema, the heart may lie so deep under the thoracic surface that not even its left border can be noted unless the heart is enlarged. In such cases, unless the nurse detects a displaced apical impulse and suspects cardiac enlargement, percussion is omitted.

Cardiac Auscultation
All areas to be auscultated include the aortic area, the pulmonary area, Erb's point, the tricuspid area, and the apical area. The actions of the four valves are uniquely reflected at specific locations on the chest wall. These locations do not correspond to the anatomic locations of the valves within the chest; rather, they reflect the patterns by which heart sounds radiate toward the chest wall. Sound in vessels through which blood is flowing is always reflected downstream. For example, the actions of the mitral valve are usually heard best in the fifth intercostal space at the midclavicular line. This is called the mitral valve area.

Heart sounds
The **normal heart sounds**, S1 and S2, are produced primarily by the closing of the heart valves. The time between S1 and S2 corresponds]to systole. This is normally shorter than the time between S2 and S1 (diastole). As the heart rate increases, diastole shortens.
In normal physiology, the periods of systole and diastole are silent. Ventricular disease, however, can give rise to transient sounds in systole and diastole that are called gallops, snaps, or clicks. Significant narrowing of the valve orifices at times when they should be open, or residual gapping of valves at times when they should be closed, gives rise to prolonged sounds called murmurs.

Auscultation Procedure
During auscultation, the patient remains supine and the examining room is as quiet as possible. A stethoscope with a diaphragm and a bell is necessary for accurate auscultation of the heart.

Using the diaphragm of the stethoscope, the examiner starts at the apical area and progresses upward along the left sternal border to the Pulmonic and aortic areas. If desired, the examiner may choose to begin the examination at the aortic and Pulmonic areas and progress downward to the apex of the heart.

Initially, S1 is identified and evaluated with respect to its intensity and splitting.

Next, S2 is identified, and its intensity and any splitting are noted.
After concentrating on S1 and S2, the examiner listens for extra sounds in systole and then in diastole. The examiner again proceeds to move the stethoscope to all of the designated areas of the precordium, listening carefully for these sounds.

Finally, the patient is turned on the left side and the stethoscope is placed on the apical area, where an S3, an S4, and a mitral murmur are more readily detected.

Once an abnormality is heard, the entire chest surface is reexamined to determine the exact location of the sound and its radiation.
Also, the patient, who may be concerned about the prolonged examination, must be supported and reassured. The auscultatory findings, particularly murmurs, are documented by identifying the following characteristics:

- Location on chest wall.
- Timing of sound as either during systole or during diastole; described as early, middle, or late. (If heard throughout the systole, the sound is often referred to as pansystolic or holosystolic.)
- Intensity of the sound (I, very faint; II, quiet; III, moderately loud; IV, loud; V, very loud; or VI, heard with stethoscope removed from the chest).
- Pitch, described as high, medium, or low.
- Quality of the sound, commonly described as blowing, harsh, or musical

Inspection of the Extremities
The hands, arms, legs, and feet are observed for skin and vascular changes. The most noteworthy changes include the following:

- Decreased capillary refill time indicates a slower peripheral flow rate from sluggish reperfusion and is often observed in patients with hypotension or HF. Capillary refill time provides the basis for estimating the rate of peripheral blood flow. To test capillary refill, briefly compress the nail bed so that it blanches, and then release the pressure. Normally, reperfusion occurs within 3 seconds, as evidenced by the return of color.
- Vascular changes from decreased arterial circulation include decrease in quality or loss of pulse, discomfort or pain, paresthesia, numbness, decrease in temperature, pallor, and loss of movement. During the first few hours after invasive cardiac

procedures (eg, cardiac catheterization), affected extremities should be assessed for vascular changes frequently.

- Hematoma, or a localized collection of clotted blood in the tissue, may be observed in patients who have undergone invasive cardiac procedures such as cardiac catheterization, PTCA, or cardiac electrophysiology testing. Major blood vessels of the arms and legs are selected for catheter insertion.

During these procedures, systemic anticoagulation with heparin is necessary, and minor or small hematomas may occur at the catheter puncture site. However, large hematomas are a serious complication that can compromise circulating blood volume and cardiac output, requiring blood transfusions.

All patients who have undergone these procedures must have their puncture sites frequently observed until homeostasis is adequately achieved.

- Peripheral edema is fluid accumulation in dependent areas of the body (feet and legs, sacrum in the bedridden patient). Assess for pitting edema (a depression over an area of pressure) by pressing firmly for 5 seconds with the thumb overt the dorsum of each foot, behind each medial malleolus, and over the shins. Pitting edema is graded as absent or as present on a scale from slight (1+ = 0 to 2 mm) to very marked (4+ = more than 8 mm). Peripheral edema is observed in patient with HF and in those with peripheral vascular diseases such as deep vein thrombosis or chronic venous insufficiency.
- Clubbing of the fingers and toes implies chronic hemoglobin desaturation, as in congenital heart disease.
- Lower extremity ulcers are observed in patients with arterial or venous insufficiency.

OTHER SYSTEMS

LUNGS

Findings frequently exhibited by cardiac patients include the following:

- *Tachypnea:* Rapid, shallow breathing may be noted in patients who have HF or pain, and in those who are extremely anxious.
- *Cheyne-Stokes respirations:* Patients with severe left ventricular failure may exhibit Cheyne-Stokes breathing, a pattern of rapid respirations alternating with apnea. It is important to note the duration of the apnea.
- *Hemoptysis:* Pink, frothy sputum is indicative of acute pulmonary edema.
- *Cough:* A dry, hacking cough from irritation of small airways is common in patients with pulmonary congestion from HF.

- *Crackles:* HF or atelectasis associated with bed rest, splinting from ischemic pain, or the effects of pain medications and sedatives often results in the development of crackles.

Typically, crackles are first noted at the bases (because of gravity's effect on fluid accumulation and decreased ventilation of basilar tissue), but they may progress to all portions of the lung fields.

- *Wheezes:* Compression of the small airways by interstitial pulmonary edema may cause wheezing. Beta-adrenergic blocking agents (beta-blockers), such as propranolol (Inderal),may precipitate airway narrowing, especially in patients with underlying pulmonary disease.

Abdomen

For the cardiac patient, two components of the abdominal examination are frequently performed.

1. *Hepatojugular reflux:* Liver engorgement occurs because of decreased venous return secondary to right ventricular failure. The liver is enlarged, firm, nontender, and smooth. The hepatojugular reflux may be demonstrated by pressing firmly over the right upper quadrant of the abdomen for 30 to 60 seconds and noting a rise of 1 cm or more in jugular venous pressure. This rise indicates an inability of the right side of the heart to accommodate increased volume.
2. *Bladder distention:* Urine output is an important indicator of cardiac function, especially when urine output is reduced. This may indicate inadequate renal perfusion or a less serious problem such as one caused by urinary retention. When the urine output is decreased, the patient needs to be assessed for a distended bladder or difficulty voiding. The bladder may be assessed with an ultrasound scanner or the suprapubic area palpated for an oval mass and percussed for dullness, indicative of a full bladder.

Laboratory Tests

Laboratory tests may be performed for the following reasons:

- To assist in diagnosing an acute MI. (Angina pectoris, chest pain resulting from an insufficient supply of blood to the heart, cannot be confirmed by either blood or urine studies.)
- To identify abnormalities in the blood that affect the prognosis of a patient with a cardiac condition
- To assess the degree of inflammation
- To screen for risk factors associated with atherosclerotic coronary artery disease
- To determine baseline values before performing therapeutic interventions
- To monitor serum levels of medications

- To assess the effects of medications (e.g., the effects of diuretics on serum potassium levels)
- To screen generally for abnormalities.

Because different laboratories use different equipment and different methods of measurements, normal test values may vary depending on the laboratory and the health care institution.

1. Cardiac Enzyme Analysis

2. Blood Chemistry

- lipid profile/cholesterol levels
- blood urea nitrogen level
- serum electrolyte levels
- serum glucose level
- coagulation studies.

3. Chest x-ray and fluoroscopy

A chest x-ray usually is obtained to determine the size, contour, and position of the heart. It reveals cardiac and pericardial calcifications and demonstrates physiologic alterations in the pulmonary circulation. It does not help diagnose acute MI but can help diagnose some complications (e.g., HF). Correct placement of cardiac catheters, such as pacemakers and pulmonary artery catheters, is also confirmed by chest x-ray.

Fluoroscopy allows visualization of the heart on an x-ray screen. It shows cardiac and vascular pulsations and unusual cardiac contours. Fluoroscopy is useful for positioning intravenous pacing electrodes and for guiding catheter insertion during cardiac catheterization.

4. Electrocardiography

The ECG is a diagnostic tool used in assessing the cardiovascular system. It is a graphic recording of the electrical activity of the heart; an ECG can be recorded with 12, 15, or 18 leads, showing the activity from those different reference points. The ECG is obtained by placing disposable electrodes in standard positions on the skin of the chest wall and extremities. The heart's electrical impulses are recorded as a tracing on special graph paper.

The standard 12-lead ECG is the most commonly used tool to diagnose dysrhythmias, conduction abnormalities, enlarged heart chambers, myocardial ischemia or infarction, high or low calcium and potassium levels, and effects of some medications. A 15-lead ECG adds 3 additional chest leads across the right precordium and is a valuable tool for the early diagnosis of right ventricular and posterior left ventricular infarction. The 18-lead ECG adds 3 posterior leads to the 15-lead ECG

and is very useful for early detection of myocardial ischemia and injury (Wung & Drew, 1999).

To enhance interpretation of the ECG, the patient's age, gender, BP, height, weight, symptoms, and medications (especially digitalis and antiarrhythmic agents) should be noted on the ECG requisition.

5. Continuous Electrocardiographic Monitoring

Continuous ECG monitoring is standard for patients who are at high risk for dysrhythmias. Two continuous ECG monitoring techniques are hardwire monitoring, found in critical care units and specialty step-down units, and telemetry, found in specialty step-down units and general nursing care units. Patients who are receiving continuous ECG monitoring need to be informed of its
purpose and cautioned that this monitoring method will not detect symptoms such as dyspnea or chest pain. Therefore, patients need to be advised to report symptoms to the nurse whenever
they occur.

6. Cardiac Stress Testing

Normally, the coronary arteries dilate to four times their usual diameter in response to increased metabolic demands for oxygen and nutrients. Coronary arteries with atherosclerosis, however, dilate much less, compromising blood flow to the myocardium and causing ischemia. Therefore, abnormalities in cardiovascular function are more likely to be detected during times of increased demand, or "stress."

The **cardiac stress test** procedures—the exercise stress test, the pharmacologic stress test, and, more recently, the mental or emotional stress test—are noninvasive ways to evaluate the response of the cardiovascular system to stress. Contraindications to stress testing include severe aortic stenosis, acute myocarditis or pericarditis, severe hypertension, suspected left mainCAD, HF, and unstable angina. Because complications associated with stress testing can be life-threatening (MI, cardiac arrest, HF, and severe dysrhythmias), testing facilities must have staff and equipment ready to provide advanced cardiac life support.

Mental stress testing uses a mental arithmetic test or simulated public speech to determine whether an ischemic myocardial response occurs, similar to the response evoked by a conventional treadmill exercise test. Although its use for diagnostic purposes in patients with CAD is currently investigational, preliminary results indicate that the ischemic and hemodynamic measures obtained mental stress testing may be useful in assessing the prognosis of patients with CHD who have had a positive exercise test Stress testing is often combined with

echocardiography or radionuclide imaging (discussed later). These techniques are performed during the resting state and immediately after stress.

7. Exercise Stress Testing

In an exercise stress test, the patient walks on a treadmill (most common) or pedals a stationary bicycle or arm crank. Exercise intensity progresses according to established protocols. The Bruce protocol, for example, is a common treadmill protocol in which the speed and grade of the treadmill are increased every 3 minutes.

The goal of the test is to increase the heart rate to the "target heart rate." This is 80% to 90% of the maximum predicted heart rate and is based on the age and gender of the patient.

During the test, the following are monitored:

Two or more ECG leads for heart rate, rhythm, and ischemic changes; BP; skin temperature; physical appearance; perceived exertion; and symptoms including chest pain, dyspnea, dizziness, leg cramping, and fatigue.

The test is terminated when the target heart rate is achieved or when the patient experiences chest pain, extreme fatigue, a decrease in BP or pulse rate, serious dysrhythmias or ST segment changes on ECG, or other complications.

When significant ECG abnormalities occur during the stress test (ST segment depressions), the test result is reported as positive and further diagnostic testing is required.

Nursing interventions

In preparation for the exercise stress test, the patient is instructed to fast for 4 hours before the test and to avoid stimulants such as tobacco and caffeine.

Medications may be taken with sips of water.

The physician may instruct patients not to take certain cardiac medications, such as beta-blockers, before the test.

Clothes and sneakers or rubber-soled shoes suitable for exercising are to be worn.

Women are advised to wear a bra that provides adequate support.

The nurse describes the equipment used and the sensations and experiences that the patient may have during the test.

The nurse explains the monitoring equipment used, the need to have an intravenous line placed, and the symptoms to report.

The type of exercise is reviewed, and patients are asked to put forth their best exercise effort.

If the test is to be performed with echocardiography or radionuclide imaging, this information is reviewed as well.

After the test, patients are monitored for 10 to 15 minutes. Once stable, they may resume their usual activities.

8. Pharmacologic Stress Testing

Physically disabled or deconditioned patients will not be able to achieve their target heart rate by exercising on a treadmill or bicycle.

Two vasodilating agents, dipyridamole (Persantin) and Adenosine (Adenocard), administered intravenously, are used to mimic the effects of exercise by maximally dilating the coronaryarteries. The effects of dipyridamole last about 15 to 30 minutes.

The side effects are related to its vasodilating action and include chest discomfort, dizziness, headache, flushing, and nausea.

Adenosine has similar side effects, although patients report these symptoms as more severe. A unique property of adenosine is that it has an extremely short half-life (less than 10 seconds), so any severe effects rapidly subside.

Dipyridamole and adenosine are the agents of choice used in conjunction with radionuclideimaging techniques. Theophylline and other xanthines, such as caffeine, block the effects of Dipyridamole and Adenosine and must be avoided before either of these pharmacologic stress tests.

Dobutamine (Dobutrex) is another medication that may be used for patients who cannot exercise. Dobutamine, a synthetic sympathomimetic, increases heart rate, myocardial contractility, and BP, thereby increasing the metabolic demands of the heart. It is the agent of choice when echocardiography is used because of its effects on altering myocardial wall motion (due to enhanced contractility). In addition, dobutamine is used for patients who have bronchospasm or pulmonary disease and cannot tolerate having doses of the ophylline withheld.

Nursing interventions
In preparation for the pharmacologic stress test, patients are instructed not to eat or drink for at least 4 hours before the test. This includes chocolate, caffeine, caffeine-free coffee, tea, carbonated beverages, or medications with caffeine (e.g., Anacin, Darvon).

If caffeine is ingested before a Dipyridamole or Adenosine stress test, the test will have to be rescheduled.

Patients taking Aminophylline or Theophylline are instructed to stop taking these medications for 24 to 48 hours before the test (if tolerated). Oral doses of Dipyridamole are to be withheld as well.

Patients are informed about the transient sensations they may experience during infusion of the vasodilating agent, such as flushing or nausea, which will disappear quickly. The patient is instructed to report any other symptoms occurring during the test to the cardiologist or nurse. An explanation of echocardiography or radionuclide imaging is also provided as necessary.

The stress test may take about 1 hour, or up to 3 hours if imaging is performed.

9. Echocardiography
Echocardiography is a noninvasive ultrasound test that is used to examine the size, shape, and motion of cardiac structures. It is a particularly useful tool for diagnosing pericardial effusions, determining the etiology of heart murmurs, evaluating the function of prosthetic heart valves, determining chamber size, and evaluating ventricular wall motion.

It involves transmission of high-frequency sound waves into the heart through the chest wall and recording of the return signals. The ultrasound is generated by a hand-held transducer applied to the front of the chest. The transducer picks up the echoes, converts them to electrical impulses, and transmits them to the echocardiography machine for display on an oscilloscope and recording on a videotape.

An ECG is recorded simultaneously to assist with interpreting the echocardiogram.

M-mode (motion), the unidimensional mode that was first introduced, provides information about the cardiac structures and their motion. Two-dimensional or cross-sectional echocardiography, an enhancement of the technique, creates a sophisticated, spatially correct image of the heart.

Other techniques, such as Doppler and color flow imaging echocardiography, show the direction and velocity of the blood flow through the heart.

As previously mentioned, echocardiography may be performed with an exercise or pharmacologic stress test; resting and stress images are obtained. Myocardial ischemia from decreased perfusion during stress causes abnormalities in ventricular wall motion and is easily detected by echocardiography.

A stress test using echocardiography is considered positive if abnormalities in ventricular wall motion are detected during stress but not during rest.

10. Computed Tomography

Computed tomography (CT), also called computerized axial tomographic (CAT) scanning or electron-beam computed tomography (EBCT), uses x-rays to provide cross-sectional images of the chest, including the heart and great vessels. These techniques are used to evaluate cardiac masses and diseases of the aorta and pericardium.

EBCT, also known as the Ultrafast CT, is an especially fast x-ray scanning technique that results in much faster image acquisition with a higher degree of resolution than traditional x-ray or CT scanning provides (Woods et al., 1999). It is used to evaluate bypass graft patency, congenital heart lesions, left and right ventricular muscle mass, chamber volumes, cardiac output, and ejection fraction. For people without previous MI, PTCA, or coronary artery bypass surgery, the EBCT is used to determine the amount of calcium deposits in the coronary arteries and underlying atherosclerosis. From this scan, a calcium score is derived that predicts the incidence of cardiac events, such as MI or the need for a revascularization procedure within the next 1 to 2 years.

The EBCT is not widely used, but it does show great promise for early detection of CAD that is not yet clinically significant and that would not be identified by traditional testing methods, such as the exercise stress test.

Nursing interventions

Patient preparation is the primary role of the nurse for these tests.
The nurse should instruct the patient that he will be positioned on a table during the scan while the scanner rotates around him.

The procedure is noninvasive and painless. However, to obtain adequate images, the patient must lie perfectly still during the scanning process.

An intravenous access line is necessary if contrast enhancement is to be used.

11. Magnetic Resonance Imaging

Magnetic resonance imaging (MRI) is a noninvasive, painless technique that is used to examine both the physiologic and anatomic properties of the heart. MRI uses a powerful magnetic field and computer-generated pictures to image the heart and great vessels. It is valuable in diagnosing diseases of the aorta, heart muscle, and pericardium, as well as congenital heart lesions. The application of this technique to the evaluation of coronary artery anatomy, cardiac blood flow, and myocardial viability in conjunction with pharmacologic stress testing is being investigated.

Nursing interventions

Because of the strong magnetic field used during MRI, diagnostic centers where these procedures are performed carefully screen patients for contraindications. Standardized questionnaires are commonly used to determine whether the patient has a pacemaker, metal plates, prosthetic joints, or other metallic implants that can become dislodged if exposed to MRI. During an MRI, the patient is positioned supine on a table that is placed into an enclosed imager or tube that contains the magnetic field.

People who are claustrophobic may need to receive a mild sedative before undergoing an MRI. As the MRI is performed, there is an intermittent clanking or thumping sound from the magnetic coils that can be annoying to the patient, so patients are offered headsets to listen to music. The scanner is equipped with a microphone so that the patient can communicate with the staff. During the scanning, the patient is instructed to remain still and not move.

NURSING ALERT

No metal can be in the MRI room because metal objects can become dangerous projectiles; this includes such items as clipboards, paperclips, oxygen tanks, and monitors.

12. Cardiac Catheterization

Cardiac catheterizationis an invasive diagnostic procedure in which radio opaque arterial and venous catheters are introduced into selected blood vessels of the right and left sides of the heart.

Catheter advancement is guided by fluoroscopy. Most commonly, the catheters are inserted percutaneously through the blood vessels, or via a cut-down procedure if the patient has poor vascular access. Pressures and oxygen saturations in the four heart chambers are measured. Cardiac

catheterization is used to diagnose CAD, assess coronary artery patency, and determine the extent of atherosclerosis based on the percentage of coronary artery obstruction.

These results determine whether revascularization procedures including PTCA or coronary artery bypass surgery may be of benefit to the patient.
During cardiac catheterization, the patient has an intravenous line in place for the administration of sedatives, fluids, heparin, and other medications.
Noninvasive hemodynamic monitoring that includes BP and multiple ECG tracings is necessary to continuously observe for dysrhythmias or hemodynamic instability. The myocardium can become ischemic and trigger dysrhythmias as catheters are positioned in the coronary arteries or during injection of contrast agents. Resuscitation equipment must be readily available during the procedure.

Staff must be prepared to provide advanced cardiac life support measures as necessary.

Radio opaque contrast agents are used to visualize the coronary arteries; some contrast agents contain iodine.

The patient is assessed before the procedure for previous reactions to contrast agents or allergies to iodine-containing substances (eg, seafood).
If the patient has a suspected or known allergy to the substance, antihistamines or methylprednisolone (Solu-Medrol) may be administered before the procedure.

In addition, the following blood tests are performed to identify abnormalities that may complicate recovery: BUN and creatinine levels, hematocrit and hemoglobin values, platelet count, and electrolyte levels.
Diagnostic cardiac catheterizations are commonly performed on an outpatient basis and require 2 to 6 hours of bed rest before ambulation.
However, variations in time of ambulation are most often related to the size of the catheter used during the procedure, the anticoagulation status of the patient, other patient variables (e.g., advanced age, obesity, bleeding disorder), the method used for hemostasis of the arterial puncture site after the procedure, and institutional policies.
The use of smaller catheters, which are more amenable to shorter recovery times, is common in diagnostic cardiac catheterizations.

There are several methods available to achieve arterial hemostasis after catheter removal, including manual pressure, mechanical compression devices (placed over puncture site for 30 minutes), and percutaneously

deployed devices. The latter devices are positioned at the femoral arterial puncture site after completion of the procedure. Major benefits of these devices include reliable, immediate hemostasis and shorter time on bed rest without a significant increase in bleeding or other complications

A number of factors determine which hemostatic methods are used and are based on the physician's preference, the patient's condition, cost, and institutional availability of the equipment.

Patients hospitalized for angina or acute MI may also require cardiac catheterization.

After the procedure, these patients usually return to their hospital rooms for recovery.

In some cardiac catheterization laboratories, an angioplasty may be performed immediately after the catheterization if indicated.

13. Angiography

Cardiac catheterization is usually performed with angiography, a technique of injecting a contrast agent into the vascular system to outline the heart and blood vessels. When a particular heart chamber or blood vessel is singled out for study, the procedure is known as selective angiography.

Angiography makes use of cineangiograms, a series of rapidly changing films on an intensified fluoroscopic screen that record the passage of the contrast agent through the vascular site or sites. The recorded information allows for comparison of data over time.

Common sites for selective angiography are the aorta, the coronary arteries, and the right and left sides of the heart.

14. Aortography

An aortogram is a form of angiography that outlines the lumen of the aorta and the major arteries arising from it. In thoracic aortography, a contrast agent is used to study the aortic arch and its
major branches. The catheter may be introduced into the aorta using the translumbar or retrograde brachial or femoral artery approach.

15. Coronary Arteriography

In coronary arteriography, the catheter is introduced into the right or left brachial or femoral artery, then passed into the ascending aorta and manipulated into the appropriate coronary artery.

Coronary arteriography is used to evaluate the degree of atherosclerosis and to guide the selection of treatment. It is also used to study suspected congenital anomalies of the coronary arteries.

16. Right Heart Catheterization

Right heart catheterization usually precedes left heart catheterization.
It involves the passage of a catheter from an antecubital or femoral vein into the right atrium, right ventricle, pulmonary artery, and pulmonary arterioles. Pressures and oxygen saturations from each of these areas are obtained and recorded.
Although right heart catheterization is considered a relatively safe procedure, potential complications include cardiac dysrhythmias, venous spasm, infection of the insertion site, cardiac perforation, and, rarely, cardiac arrest.

17. Left Heart Catheterization

Left heart catheterization is performed to evaluate the patency of the coronary arteries and the function of the left ventricle and the mitral and aortic valves. Potential complications include dysrhythmias, MI, perforation of the heart or great vessels, and systemic embolization.
Left heart catheterization is performed by retrograde catheterization of the left ventricle. In this approach, the physician usually inserts the catheter into the right brachial artery or a femoral artery and advances it into the aorta and left ventricle.

After the procedure, the catheter is carefully withdrawn and arterial hemostasis is achieved using manual pressure or other techniques previously described.

If the physician performed an arterial or venous cut-down, the site is sutured and a sterile dressing is applied.

Nursing interventions

Nursing responsibilities before cardiac catheterization include the following

- Instruct the patient to fast, usually for 8 to 12 hours, before the procedure.

If catheterization is to be performed as an outpatient procedure, explain that a friend, family member, or other responsible person must accompany the patient home.

- Prepare the patient for the expected duration of the procedure; indicate that it will involve lying on a hard table for less than 2 hours.
- Reassure the patient that mild sedatives or moderate sedation will be given intravenously.

- Prepare the patient to experience certain sensations during the catheterization. Knowing what to expect can help the patient cope with the experience.

Explain that an occasional pounding sensation (palpitation) may be felt in the chest because of extrasystoles that almost always occur, particularly when the catheter tip touches the myocardium.

The patient may be asked to cough and to breathe deeply, especially after the injection of contrast agent. Coughing may help to disrupt a dysrhythmia and to clear the contrast agent from the arteries.

Breathing deeply and holding the breath helps to lower the diaphragm for better visualization of heart structures. The injection of a contrast agent into either side of the heart may produce a flushed feeling throughout the body and a sensation similar to the need to void, which subsides in 1 minute or less.

- Encourage the patient to express fears and anxieties.

Provide teaching and reassurance to reduce apprehension.

Nursing responsibilities after cardiac catheterization may include the following:

1. Observe the catheter access site for bleeding or hematoma formation, and assess the peripheral pulses in the affected extremity (dorsalispedis and posterior tibial pulses in the lower extremity, radial pulse in the upper extremity) every 15 minutes for 1 hour, and then every 1 to 2 hours untilthe pulses are stable.
2. Evaluate temperature and color of the affected extremity and any patient complaints of pain, numbness, or tingling sensations to determine signs of arterial insufficiency. Report changes promptly.
3. Monitor for dysrhythmias by observing the cardiac monitor or by assessing the apical and peripheral pulses for changes in rate and rhythm. A vasovagal reaction, consisting of bradycardia, hypotension, and nausea, can be precipitated by a distended bladder or by discomfort during removal of the arterial catheter, especially if a femoral site has been used. Prompt intervention is critical; this includes raising the feet and legs above the head, administering intravenous fluids, and administering intravenous Atropine.
4. Inform the patient that if the procedure is performed percutaneously through the femoral artery (and without the use of devices such as VasoSeal, Perclose, or Angio-Seal),the patient will remain on bed rest for 2 to 6 hours with the affected leg straight and the head elevated to 30 degrees.

For comfort, the patient may be turned from side to side with the affected extremity straight.

If the cardiologist uses deployed devices, check local nursing care standards, but anticipate that the patient will have less restrictions on elevation of the head of the bed and will be allowed to ambulate in 2 hours or less.

Analgesic medication is administered as prescribed for discomfort.

5. Instruct the patient to report chest pain and bleeding or sudden discomfort from the catheter insertion sites immediately.
6. Encourage fluids to increase urinary output and flush out dye.
7. Ensure safety by instructing the patient to ask for help when getting out of bed the first time after the procedure, because orthostatic hypotension may occur and the patient may feel dizzy and lightheaded.

For patients being discharged from the hospital on the same, day as the procedure, additional instructions are provided.

4.0 REFERENCES/FURTHER READING

Fuad, I. & Welday, M. (2008). Pathophysiology. Ethiopia Public Health Training Initiative. Ethiopia; Hirmaya University.

Famakinwa, O.T. *Synopsis of Medicosurgical Nursing*. Ibadan; Bayosore.

Heyman, P. Anatomy of the immune system supplemental reading.

Smettzer, C.S. Bare, B.G, Hinkle, L.J. & Cheever, H.K., (2008). Brunner & Suddarth's Textbook of Medical-Surgical Nursing. (11th Ed). India; Wolters Kluwer.

Waugh, A., Grant, A. (2006). Ross & Wilson Anatomy and Physiology in Health and Illness (10th Ed). Philadelphia; Elsevier.

UNIT 2 MANAGEMENT OF PATIENTS WITH CORONARY VASCULAR DISORDERS

CONTENTS

1.0 INTRODUCTION

Coronary Artery Disease
Coronary artery disease (CAD) is the most prevalent type of cardiovascular disease. For this reason, it is important for nurses to become familiar with the various types of coronary artery conditions and the methods for assessing, preventing, and treating these disorders medically and surgically.

Coronary Atherosclerosis
The most common heart disease in developed countries is **atherosclerosis**, which is an abnormal accumulation of lipid, or fatty, substances and fibrous tissue in the vessel wall. These substances create blockages or narrow the vessel in a way that reduces blood flow to the myocardium. It was reported that atherosclerosis involves a repetitious inflammatory response to artery wall injury and an alteration in the biophysical and biochemical properties of the arterial walls. An association between an infection (e.g., gingivitis) and the later development of heart disease is being explored, as is the administration of antibiotics to prevent heart disease. Although authorities disagree about how atherosclerosis begins, they agree that atherosclerosis is a progressive disease that can be curtailed and, in some cases, reversed.

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- Describe the pathophysiology, clinical manifestations, and treatment of coronary atherosclerosis.
- Describe the pathophysiology, clinical manifestations, and treatment of angina pectoris.
- Use the nursing process as a framework for care of patients with

- angina pectoris.
- Describe the pathophysiology, clinical manifestations, and treatment of myocardial infarction.
- Describe the nursing care of a patient who has had an invasive interventional procedure for treatment of coronary artery disease.
- Describe the nursing care of the patient treated with cardiac surgery.

3.0 MAIN CONTENT

3.1 Pathophysiology

Atherosclerosis begins as fatty streaks, lipids that are deposited in the intima of the arterial wall. Although they are thought to be the precursors of atherosclerosis, fatty streaks are common, even in childhood. Moreover, not all develop into more advanced lesions. The reason why some fatty streaks continue to develop is unknown, although genetic and environmental factors are involved.

The continued development of atherosclerosis involves an inflammatory response. T lymphocytes and monocytes (that become macrophages) infiltrate the area to ingest the lipids and then die; this causes smooth muscle cells within the vessel to proliferate and form a fibrous cap over the dead fatty core. These deposits, called **atheromas**or plaques, protrude into the lumen of the vessel, narrowing it and obstructing blood flow. If the fibrous cap of the plaque is thick and the lipid pool remains relatively stable, it can resist the stress from blood flow and vessel movement. If the cap is thin, the lipid core may grow, causing it to rupture and hemorrhage into the plaque, allowing a thrombus to develop. The thrombus may obstruct blood flow, leading to sudden cardiac death or an acute **myocardial infarction (MI)**, which is the death of heart tissue.

The anatomic structure of the coronary arteries makes them particularly susceptible to the mechanisms of atherosclerosis. They twist and turn as they supply blood to the heart, creating sites susceptible to atheroma development. Although heart disease is most often caused by atherosclerosis of the coronary arteries, other phenomena decrease blood flow to the heart. Examples include vasospasm (sudden constriction or narrowing) of a coronary artery, myocardial trauma from internal or external forces, structural disease, congenital anomalies, decreased oxygen supply (e.g., from acute blood loss, anemia, or low blood pressure), and increased demand for oxygen (e.g., from rapid heart rate, thyrotoxicosis, or ingestion of cocaine)

Clinical Manifestations

Coronary atherosclerosis produces symptoms and complications according to the location and degree of narrowing of the arterial lumen, thrombus formation, and obstruction of blood flow to the myocardium. This impediment to blood flow is usually progressive, causing an inadequate blood supply that deprives the muscle cells of oxygen needed for their survival. The condition is known as **ischemia**.

Angina pectoris refers to chest pain that is brought about by myocardial ischemia. Angina pectoris usually is caused by significant coronary atherosclerosis. If the decrease in blood supply is great enough, of long enough duration, or both, irreversible damage and death of myocardial cells, or MI, may result.

Over time, irreversibly damaged myocardium undergoes degeneration and is replaced by scar tissue, causing various degrees of myocardial dysfunction. Significant myocardial damage may cause inadequate cardiac output, and the heart cannot support the body's needs for blood, which is called heart failure (HF). A decrease in blood supply from CAD may even cause the heart to stop abruptly, an event that is called **sudden cardiac death**.

The most common manifestation of myocardial ischemia isacute onset of chest pain. However, an epidemiologic study of the people in Framingham, Massachusetts, showed that nearly 15%of men and women who had MIs were totally asymptomatic (Kannel, 1986).

Another study found that 33% of those diagnosed with MI did not present to the emergency room with chest pain (Canto et al., 2000; Ishihara et al., 2000). Those without chest pain tend to be older or women, or to have diabetes or a history of heart failure.

Women have been found to have more atypical symptoms of myocardial ischemia (e.g., shortness of breath, nausea, unusual fatigue) than men (Meischke et al., 1999).

The incidence of prodromal angina (i.e., angina a few hours to days before the MI) was found to be significantly lower in patients older than 70 years of age (Ishihara et al., 2000). Other clinical manifestations of CAD may be abnormalities signaled by changes on the electrocardiogram (ECG), high levels of cardiac enzymes, dysrhythmias, and sudden death.

Risk Factors

Epidemiologic studies point to several factors that increase the probability that heart disease will develop. Major risk factors include use of tobacco, hypertension, elevated blood lipid levels, family history of premature cardiovascular disease (first-degree relative with cardiovascular disease at age 55 or younger for men and at age 65 or younger for women) and age (>45 years for men; >55 years for women). Cholesterol as the primary target of cholesterol-lowering therapy.

Those at highest risk for having a cardiac event within 10 years are those with existing CAD or those with diabetes, peripheral arterial disease, abdominal aortic aneurysm, or carotid artery disease.

The latter diseases are called CAD risk equivalents, because patients with these diseases have the same risk for a cardiac event as patients with CAD (Chart 28-1). The possibility of having acardiac event within 10 years is also determined by points given to several factors, such as age, level of total cholesterol, level of LDL, level of **high-density lipoprotein (HDL)**, systolic blood pressure, and tobacco use. If the total points add up to more than 15 for men or 23 for women, the person has a greater than 20% risk for a cardiac event within 10 years.

A composite of lipid and non lipid risk factors of metabolic origin, called *metabolic syndrome,* is another risk factor for CAD. Metabolic syndrome includes abdominal obesity, an elevated triglyceride level, low HDL level, elevated blood pressure, and impaired function of insulin.

Measurement of other emerging risk factors, such as elevations of Lipoprotein(a) [Lp(a)], remnant lipoproteins, small LDL, fibrinogen, homocysteine, and impaired fasting plasma glucose(110–125 mg/dL), is optional and are not routinely recommended(ATP III, 2001). For example, the Homocysteine Studies Collaboration (2002) found that lower levels of homocysteine, an amino acid, were modestly associated with reduced risk of ischemic heart disease and stroke.

The authors recommend a daily intake of approximately 0.8 mg of folic acid to decrease blood homocysteine levels and reduce the risk of ischemic heart disease and CVA (brain attack, stroke). The American Heart Association has stated that until the results of large-scale randomized trials become available, routine testing of homocysteine concentrations cannot be justified.

Prevention

Four modifiable risk factors—cholesterol abnormalities, cigarette smoking (tobacco use), hypertension, and diabetes mellitus— have been

cited as major risk factors for CAD and its consequent complications. As a result, they receive much attention in health promotion programs.

3.2 Controlling Cholesterol Abnormalities

The association of a high blood cholesterol level with heart disease is well established and accepted. The metabolism of fats is important in understanding the development of heart disease.

Fats, which are insoluble in water, are encased in water-soluble lipoproteins to allow them to be transported within a circulatory system that is water-based. Four elements of fat metabolism-total cholesterol, LDL, HDL, and triglycerides—are primary factors affecting the development of heart disease.

Cholesterol and the lipoproteins are synthesized by the liver or ingested as part of the diet. All adults 20 years of age or older should have a fasting lipid profile (total cholesterol, LDL, HDL, and triglyceride) performed at least once every 5 years and more often if the profile is abnormal. Patients who have had an acute event (MI), percutaneous coronary intervention (PCI), or **coronary artery by pass graft (CABG)** require assessment of the LDL-cholesterol level within 60 to 365 days after the event (LDL levels may be low immediately after the acute event). Subsequently, lipids should be monitored every 6 weeks until the desired level is achieved and then every 4 to 6 months (Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults.

LDL exerts a harmful effect on the arterial wall and accelerates atherosclerosis. In contrast, HDL promotes the use of total cholesterol by transporting LDL to the liver, where it is biodegraded and then excreted. The desired goal is to have low LDL values and high HDL values. The desired level of LDL depends on the patient:

- Less than 160 mg/dL for patients with one or no risk factors
- Less than 130 mg/dL for patients with two or more risk factors
- Less than 100 mg/dL for patients with CAD or a CAD risk equivalent

Individuals at highest risk for a cardiac event within 10 years are those with existing coronary artery disease (CAD) and those with any of the following diseases, which are called CAD risk equivalents:

- Diabetes
- Peripheral arterial disease
- Abdominal aortic aneurysm
- Carotid artery disease.

Risk factors for Coronary Heart Disease

A modifiable risk factor is one over which individuals may exercise control, such as by changing a lifestyle or personal habit or by using medication.

A non modifiable risk factor is a circumstance over which individuals have no control, such as age or heredity. A risk factor may operate independently or in tandem with other risk factors.

The more risk factors individuals have, the greater the likelihood of coronary artery disease. Those at risk are advised to seek regular medical examinations and to engage in "heart-healthy" behavior (a deliberate effort to reduce the number and extent of risks).

Non modifiable Risk Factors

- Family history of coronary heart disease
- Increasing age
- Gender (heart disease occurs three times more often in men than in premenopausal women)
- Race (higher incidence of heart disease in African Americans than in Caucasians).

Modifiable Risk Factors

- High blood cholesterol level
- Cigarette smoking, tobacco use
- Hypertension
- Diabetes mellitus
- Lack of estrogen in women
- Physical inactivity
- Obesity.

Dietary Measures

However, these recommendations may need to be adjusted to match the individual patient who has other nutritional needs, such as the requirements for pregnancy or diabetes.

To assist in following the appropriate TLC diet, the patient should be referred to a registered dietitian. Other TLC recommendations are weight loss, cessation of tobacco use, and increased physical activity.

Soluble dietary fiber may also help lower cholesterol levels. Soluble fibers, which are found in fresh fruit, cereal grains, vegetables, and legumes, enhance the excretion of metabolized cholesterol.

The ability of fiber to reduce serum cholesterol continues to be investigated. Intake of at least 20 to 30 grams of fiber each day is recommended.

Many resources are available to assist people who are attempting to control their cholesterol levels.

Physical Activity

Regular, moderate physical activity increases HDL levels and reduces triglyceride levels. The goal for the average person is a total of 30 minutes of exercise, three to four times per week. The nurse helps patients set realistic goals for physical activity. For example, the inactive patient should start with activity that lasts 3 minutes, such as parking farther from a building to increase the walking time. For sustained activity, patients should begin with a 5-minute warm-up period to stretch and prepare the body for the exercise. They should end the exercise with a 5-minute cool-down period in which they gradually reduce the intensity of the activity to prevent a sudden decrease in cardiac output. Patients should be instructed to engage in an activity or variety of activities that interest them, to maintain motivation. They should also be taught to exercise to an intensity that does not preclude their ability to talk; if they cannot have a conversation, they should slow down or switch to a less intensive activity. When the weather is hot and humid, the patient should be advised to exercise during the early morning or indoors and wear loose-fitting clothing.

When the weather is cold, the patient should be instructed to layer clothing and to wear a hat. The nurse can also advise the patient to avoid adverse weather conditions by participating in local community programs, such as those held at shopping malls. The nurse should inform patients to stop any activity if they develop chest pain, unusual shortness of breath, dizziness, lightheadedness, or nausea.

Medications. Medications are used in some instances to control cholesterol levels. If diet alone cannot normalize serum cholesterol levels, several medications have a synergistic effect with the prescribed diet. Lipid-lowering medications can reduce CAD mortality in patients with elevated lipid levels and in those with normal lipid levels. The lipid-lowering agents affect the different lipid components and are usually grouped into four types:

- 3-Hydroxy-3-methylglutaryl coenzyme A (HMG-CoA) reductase inhibitors or statins (e.g., lovastatin [Mevacor], pravastatin [Pravachol], simvastatin [Zocor]; block cholesterol synthesis, lower LDL and triglyceride levels, and increase HDL levels. These medications are frequently the initial medication therapy for significantly elevated cholesterol and LDL levels. Because of

their effect on the liver, results of hepatic function tests are monitored.

- Nicotinic acids (niacin [Niacor, Niaspan]; decrease lipoprotein synthesis, lower LDL and triglyceride levels, and increase HDL levels. The dose of niacin needs to be titrated weekly to achieve therapeutic dosage. Niacin is the medication most often used for minimally elevated cholesterol and LDL levels or as an adjunct to a statin when the lipid goal has not been achieved and the triglycerides are elevated. Side effects include gastrointestinal upset, gout, and flushing. Because of its effect on the liver, hepatic function is monitored.
- Fibric acid or fibrates (e.g., clofibrate [Atromid-S], fenofibrate [Ticor]; decrease the synthesis of cholesterol, reduce triglyceride levels, and increase HDL levels. Because they have the potential to increase LDLs, fibrates are the medications of choice for patients with triglyceride levels above 400 mg/dL. Because of the risk of myopathy and acute renal failure, fibrates should be used with caution in patients who are also taking a statin.
- Bile acid sequestrants or resins (e.g., cholestyramine [LoCholest, Questran, Prevalite]; bind cholesterol in the intestine, increase its breakdown, and lower LDL levels with minimal effect on HDLs and no effect (or minimal increase) on triglyceride levels. These medications are more often used as adjunct therapy when statins alone have not been effective in controlling lipid levels and the triglyceride levels are less than 200 mg/dL. Significant side effects, such as gastric distention and constipation, can occur from using these medications.

Medication therapy is reserved for at-risk patients and is not regarded as a substitute for dietary modification. All of these medications have been shown to reduce major coronary events (. Some of these may be used in combination to achieve synergistic effects. For example, LDL cholesterol can be lowered more effectively by adding a low dose of resin to a dose of niacin or statins, or both, than a maximum dose of an individual agent.

Patients with elevated cholesterol levels should be monitored for adherence to the medical plan, the effect of cholesterol-lowering medications, and the development of side effects from cholesterol lowering medications. Lipid levels are obtained and adjustments made to the diet and medication every 6 weeks until the lipid goal or maximum dose is achieved and then every 6 months thereafter.

3.3 Promoting Cessation of Tobacco use

Cigarette smoking contributes to the development and severity of CAD in three ways. First, the inhalation of smoke increases the blood carbon monoxide level, causing hemoglobin, the oxygen carrying component of blood, to combine more readily with carbon monoxide than with oxygen. A decreased amount of available oxygen may decrease the heart's ability to pump.

Second, the nicotinic acid in tobacco triggers the release of catecholamines, which raise the heart rate and blood pressure. Nicotinic acid can also cause the coronary arteries to constrict. Smokers
have a tenfold increase in risk for sudden cardiac death. The increase in catecholamines may be a factor in the increased incidence of sudden cardiac death.

Third, use of tobacco causes a detrimental vascular response and increases platelet adhesion, leading to a higher probability of thrombus formation. A person with increased risk for heart disease is encouraged to stop tobacco use through any means possible: counseling, consistent motivation and reinforcement messages, support groups, and medications. Some people have found complementary therapies (eg, acupuncture, guided imagery, hypnosis) to be helpful. People who stop smoking reduce their risk of heart disease by 30% to 50% within the first year, and the risk continues to decline as long as they refrain from smoking.

Exposure to other smokers' smoke (passive or second-hand smoke) is believed to cause heart disease in nonsmokers. Oral contraceptive use by women who smoke is inadvisable because these medications significantly increase the risk of CAD and sudden cardiac death.

Cessation of tobacco use results in a lower rate of cardiac events. Patients should be advised to participate in an educational class, support group, or behavioral program. Use of medications such as the nicotine patch (Nicotrol, Nicoderm CQ, Habitrol) or bupropion (Zyban) may assist with stopping use of tobacco, but do have the same systemic effects: catecholamine release (increasing heart rate and blood pressure) and increased platelet adhesion. These medications should be used for the shortest time and at the lowest effective doses.

3.4 Managing Hypertension

Hypertension is defined as blood pressure measurements that repeatedly exceed 140/90 mm Hg. Long-standing elevated blood pressure may result in increased stiffness of the vessel walls, leading to vessel injury

and a resulting inflammatory response within the intima. Hypertension can also increase the work of the left ventricle, which must pump harder to eject blood into the arteries.

Over time, the increased workload causes the heart to enlarge and thicken (i.e., hypertrophy), a condition that may eventually lead to cardiac failure. Early detection of high blood pressure and adherence to a therapeutic regimen can prevent the serious consequences associated with untreated elevated blood pressure.

3.5 Controlling Diabetes Mellitus

The relationship between diabetes mellitus and heart disease has been substantiated. For 65% to 75% of patients with diabetes, cardiovascular disease is listed as the cause of death. Hyperglycemia fosters dyslipidemia, increased platelet aggregation, and altered red blood cell function, which can lead to thrombus formation. It has been suggested that these metabolic alterations impair endothelial cell–dependentvasodilation and smooth muscle function; treatment with insulin (e.g., Humalog, Humulin, Novolin) and metformin (Glucophage) has demonstrated improvement in endothelial function: improved endothelial-dependent dilation.

Diabetes is considered equivalent to existing CAD in its risk of a cardiac event within 10 years.

Behavior patterns
Most clinicians believe that stress and certain behaviors contribute to the pathogenesis of CAD and a cardiac event, especially in women. Psychological and epidemiologic studies describe behaviors that characterize people who are prone to heart disease: excessive competitiveness, a sense of time urgency or impatience, aggressiveness, and hostility. A person with these behaviors is classified as type A coronary-prone.

The type A coronary-prone classification may not be as significant as was once thought; evidence of its precise role remains inconclusive. To be on the safer side, however, such a person may be wise to alter behaviors and responses to triggering events and to reduce other risk factors. Nurses can assist these people by teaching them cognitive restructuring and relaxation techniques. Because people who are depressed have symptoms of depression and, if diagnosed, appropriately treated.

UNIT 3 THE IMMUNE SYSTEM AND CARE OF PATIENTS WITH INFECTIOUS DISEASES

CONTENTS

1.0 INTRODUCTION

The protective ability of the body to wade off all toxins and invading foreign organisms is called immunity. To perform this vital life process, the immune system has been designed specially to cater for all essential activities involved in performing this function. The immune system function as the body's defense mechanism against invasion and facilitates a rapid reaction to the action of foreign bodies. The immune system is tasked with three distinct and interrelated duties.

1. Defense of the body from external invaders (pathogens and toxins).
2. Surveillance in identifying the body's cells that have mutated and may become or have already become neoplasms (tumors).
3. Maintain homeostasis by removing cellular detritus from the system to ensure uniformity of cells and function.

With so much power over the functioning and viability of the body's cells, it is no coincidence that some of our worst diseases come about as a result of immune dysfunction.

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- describe the immune system and list the major functions of the system
- list and describe the types of immunity
- enumerate and describe the types of cells that performs immune functions
- describe the functions of the lymphoid organs and chemicals.

3.1 Immune

Immunity can be in two forms. These are:

- Innate immunity
- Acquired immunity.

Innate Immunity

This is also called non-specific or natural immunity. This form of immunity results from general processes directed at specific disease organism. It provides some form of rapid non-specific immunity and it is present at birth. Innate immunity can be immediate (occurring within four hours) or delayed (occurring between four to ninety six hours) after exposure. This form of immunity includes the following:

i. Phagocytosis of bacteria and other invaders by the white blood cells and cells of the tissue macrophage system.
ii. Destruction of swallowed organism by the acid secretion of the stomach
iii. Resistance of the skin to invasion by organisms
iv. Presence of certain chemicals in the blood that can attach to foreign organisms or toxins and destroy them. Examples of these compounds are; lysozymes, basic polypeptides, the complement complex and natural killer lymphocytes.

Acquired Immunity

It is also called adaptive or specific immunity. It is the body's response against individual invading organisms. It is caused by a special immune system that forms antibodies and/ or activated lymphocytes that attack and destroy the specific invading organism. This form ofimmunity is not present at birth and develops either as a result of exposure or through an external source such as colostrum or injection of immunoglobulin. Acquired immunity confers great protection as found in the process of immunization against certain infectious diseases. Acquired immunity can be of two types, Humoral or B-cell immunity and Cell mediated immunity

a. <u>Humoral or B-cell immunity</u>

The body develops circulating antibodies also called globulin molecules in the blood plasma. These globulins are capable of attacking the invading agent. These antibodies are produced by the B-lymphocytes in response to specific antibodies. The B-lymphocytes produces the globulin while the macrophages of natural immunity and the T-cell lymphocytes of the cellular immunity are involved in recognizing foreign substances and in producing antibodies.

b. Cell mediated immunity

It is also known as T-cell immunity because the activated Lymphocytes are the T-lymphocytes. The T-cells exists with designated roles in defense against bacteria, viruses, fungi, parasite and malignant cells. The T-cells attack foreign bodies directly by producing antibodies. Cellular reactions emerge by the binding of an antigen to an antigen receptor located on the surface of the T-cell. The T-cell then carries antigenic messages to the lymph node where other T-cells are produced.

NOTE: The adaptive immune system require the innate immune system for initial activation. Once activated, however, much of its effect or mechanisms involve potentiating innate immune responses. Thus the innate system forms part of the adaptive system's response and vice versa. The innate immune system can eliminate some threats by itself, but many invaders either overwhelm it or evade detection by it. In these cases, the adaptive immune system is required. It takes four to ten days for the adaptive immune system to mount its first response. Once developed however, the adaptive immune system will retain some of its effect or cells as memory cells. Upon subsequent exposures, the adaptive immune system can mount a response almost immediately. The key characteristics of both systems are recognition and effect or mechanisms. Recognition mechanisms are the methods by which various immune system cells recognize invading cells and toxins or aberrant host cells. Effect or mechanisms are the methods by which the immune system destroys and eliminates these threats.

3.2 Anatomic and Physiologic Review of the Immune System

A number of body cells are involved in immunity. The main cells of the immune system are white blood cells collectively referred to as leukocytes. Like all blood cells, leukocytes originate from the bone marrow. Stem cells (undifferentiated cells) in the marrow develop into the various white blood cells. In addition to serving as the birthplace for leukocytes, the bone marrow also acts as a reservoir for mature cells that may be needed in the event of infection or blood loss. Although most leukocytes originate in the bone marrow along with red blood cells, most spend very little time in the blood. Leukocytes spend most of their time in storage, in lymphoid tissues, or dispersed throughout the host tissues. Leukocytes use blood mainly as a transport system to travel to areas of the body where they are needed.

There are six families of leukocytes that have distinct roles in the body's defense. They are;

- Monocyte-macrophages
- Dendritic cells
- Mast cells
- Granulocytes

- Lymphocytes
- Natural killer cells.

All the leukocytes except the lymphocytes are considered part of the innate immune system. Lymphocytes are the only leukocytes associated with the adaptive immune system. All the leukocyte families originally come from pluripotent hematopoeitic stem cells in the bone marrow. The pluripotent stem cell differentiates into common lymphoid and common myeloid progenitors. All lymphocytes as well as natural killer cells are descended from the common lymphoid progenitor. The common myeloid progenitor differentiates into monocyte, dendritic cells, granulocyte, erythrocyte, and platelet precursors.

The leukocytes found in the blood and lymph tissues are typically not fully differentiated. As a case study, monocytes descend from the common myeloid progenitor. Monocytes circulate in the blood until summoned to the tissues. At this time, they exit the blood vessels through specialized openings in the vessel wall and enter the tissue. Once in the tissue, monocytes differentiate yet again, maturing into macrophages which usually live in the tissues until their death. Thus the macrophage is the monocyte's final differentiation and the monocyte is simply a relatively inert circulation form of the cell. The exception is the granulocytes which circulate in fully differentiated form. Proliferation is the other concept necessary to understand some white blood cells. Although lymphocytes originate in the bone marrow from stem cells, they are also able to reproduce within lymph tissue. When activated, lymphocytes will proliferate (reproduce) first, then differentiate into their final functioning form. This allows the few cells that are able to respond to a given invader to reproduce quickly without a corresponding increase in lymphocytes that are not needed for the present threat.

TYPES OF CELL

1. Monocyte –Macrophages

The immature stage is referred to as monocyte, while the fully differentiated stage is called a macrophage. Monocytes are continuously migrating to tissue and differentiating into tissue macrophages. Tissue macrophages are called different names, depending on the tissue in which they have differentiated. Tissue macrophages in the nervous system are called microglial cells, while macrophages in the liver are call Kupffer cells. Their functions are to monitor the surrounding tissue for invaders and foreign antigen. They are sometimes referred to as mononuclear phagocytes.

Macrophages are one of three phagocytic cells in the immune system. Having differentiated in tissues, macrophages are relatively immobile, monitoring the nearby tissue for invaders. Upon detecting an invader, macrophages attempt to engulf the invader in an amoeboid-like process called phagocytosis. Macrophages are antigen presenting cells (APCs) and act as one of the first responders in the immune response process. Once activated, a macrophage releases cytokines and chemokines which enables the respective immune function.

2. Dendritic cells

Dendritic cells are star-shaped cells that are so called because they resemble a neuron's dendrites. The immature dendritic cells migrate to tissues, particularly the skin, airway, spleen, and lymph nodes. Tissue dendritic cells that live in the skin are called Langerhans cells. (Skin tissue macrophages are also called Langerhans cells.) Immature tissue dendritic cells are both phagocytic and macropinocytic; that is, they can ingest large amounts of surrounding interstitial fluid. Tissue dendritic cells break down proteins and display the ingested antigens on their cell membranes. At the end of their life cycle, they will migrate to lymph nodes and induce tolerance in lymphoctyes, because they do not have co-stimulatory molecules in their immature stage. The signals for maturation are either direct contact with a pathogen or inflammatory cytokines. Pathogens are ingested when they are recognized by their common features as described above. Macropinocytosis allows the dendritic cell to ingest pathogens that have some mechanism to escape detection by phagocytic receptors. As the products are degraded inside the dendritic cell, it is able to recognize bacterial DNA, bacterial heat shock proteins, and viral double stranded RNA. Once activated, they differentiate into mature dendritic cells, develop co-stimulatory molecules, and migrate to the lymph nodes to activate the lymphocytes that migrate through the nodes.

The dendritic cells are able to activate only the specific T lymphocytes that are needed to respond to a given invader, whether it is a virus, bacteria, or fungus.

The dendritic cell's strength is also a key weakness exploited by several viruses, such as HIV and measles. Instead of activating lymphocytes in lymph nodes against these viruses, the infected dendritic cell acts as a transportation system, allowing the virus to then infect the T lymphocytes.

Much of the extracellular debris that is ingested by dendritic cells is harmless, often byproducts of dead body cells. Dendritic cells are essential in inducing and maintaining tolerance to these antigens, keeping the immune system from reacting to the body's antigens. As T lymphocytes exit the thymus gland, dendritic cells are responsible for

destroying cells that are reactive to self-antigens. This process is referred to as central tolerance and removes the majority of self-reactive T lymphocytes. Dendritic cells also induce peripheral tolerance, suppressing self-reactive lymphocytes that escaped central tolerance or cells that are reactive to antigens not expressed in the thymus.

3. Mast cells

Mast cells live near the skin and connective tissue of small blood vessels and contain granules with stored chemicals. When activated, they release substances within the granules (degranulate) that affect vascular permeability, particularly histamine. Mast cells are thought to play an important part in protecting mucosal surfaces from pathogens and help the inflammatory process to begin the process of healing damaged tissue, although they are primarily known for their role in IgE-mediated allergic reactions.

4. Granulocytes

Also known as polymorphonuclear leukocytes (PMNs). The granules are lysosomes —vesicles filled with destructive enzymes. These enzymes are used to destroy invaders. Neutrophils are the most numerous granulocyte performing phagocytic function in the immune system. Upon engulfing an invader, the granules are fused to the vesicle and the enzymes are released into vesicle, destroying the particle.

- Neutrophils are especially reactive to bacteria, as the number of circulating neutrophils greatly increases during bacterial infections. Neutrophils are the first responders to chemotaxis, and are rarely found in healthy tissue. Neutrophils are relatively fragile compared to macrophages. They can only ingest a few bacteria before dying, while macrophages can ingest a hundred bacteria. Pus is mostly made up of bacteria and dead neutrophils. Because of their expendable nature, they appear in the blood in large numbers, with several times that number in reserve in the bone marrow. They are the most numerous granulocyte and often the most numerous leukocyte. The other two classes of granulocyte cells are exocytic, meaning they produce their effects on outside cells as opposed to phagocytosed cells.
- Eosinophils are found in small quantities in the blood as most of them are distributed in the tissues. Their primary effect or function is to release their highly toxic granules that can kill parasites and other microorganisms. They also produce cytokines, leukotrienes, and prostaglandins. Eosinophils are involved in defense against parasites and increase in numbers when the body has a parasitic infection. They are most well known for their role in IgE mediated allergic reactions and are often present in mucous secretions during allergic reactions.

- Basophils, are the final and most inscrutable granulocyte. Not much is known about them, but they appear to have an effect against fungus and also play a role in inflammation. They behave very similarly to eosinophils and are distributed throughout the tissues.

5. Natural killer cells

Natural killer (NK) cells arise from the common lymphoid progenitor. They appear as large lymphocytes with cytoplasmic granules and circulate in the blood. Although lacking antigen specific receptors, they are able to detect and attack a limited number of abnormal cells such as tumor cells and cells infected with the herpes simplex virus. They are also able to kill cells that are coated in antibody, a process known as antibody-dependent cell-mediated cytotoxicity (ADCC) and is mediated by some receptors. Natural killer cells are also activated by interferons and macrophage-derived cytokines.

6. Lymphocytes

Some lymphocytes mature in the bone marrow, while others migrate to the thymus for maturation. B lymphocytes (also called B cells) are so called because they mature to their intermediate stage in the bone marrow. When activated, B lymphocytes complete their differentiation process and become plasma cells, releasing antibodies. T lymphocytes (T cells) are so called because they mature in the thymus. The main functional characteristic of lymphocytes is the ability to mount specific immune responses against virtually any foreign antigen. All lymphocytes have a prototype receptor that changes during the intermediate maturation process so that taken as a whole, they are able to react with almost any possible antigen. B cells are lymphocytes that develop in the bone marrow and their function upon activation is to produce antibodies.

T lymphocytes progenitors leave the bone marrow and migrate to the thymus gland where they develop into T lymphocytes instead of B lymphocytes. The T cells later develop into CD4 and CD8 T cells.

Lymphoid Organs and chemicals

Anatomically speaking, the immune system is largely identified with the lymphoid portion of the immune system. The primary lymphoid organs are the bone marrow and thymus gland because lymphocytes develop and mature within them. The thymus gland is located superior to the heart. The thymus gland also serves as a reservoir for T lymphocytes. It is believed that the thymus gland's major function is in the development of the immune system. It is larger in children than in adults. Removal of the thymus in children causes a reduction in the number of T lymphocytes and a higher number of granulocytes.

Although lymphocytes are distributed throughout the body, they are concentrated in several tissues. The tissues where they aggregate and function are called secondary lymphoid tissues, and include the spleen, lymph nodes, and epithelial lymphoid tissues. Secondary lymphoid tissues are strategically placed in the body so that invading pathogens will encounter them as early as possible, allowing the immune system to be activated before extensive damage can be done.

Spleen; is a fist-sized organ located on the left side of the body, behind the stomach. It acts as a filter, collecting antigen from the blood and destroying senescent red blood cells. Most of the spleen is made up of tissue called red pulp which primarily serves as the site of red blood cell destruction and also houses macrophages. Interspersed throughout the red pulp, lymphocytes surround artieroles forming pockets called white pulp. The organization of white pulp consists of two layers, the periarteriolar sheath, consisting mainly of T lymphocytes, and the B-cell corona, consisting of mainly B lymphocytes. The white pulp is responsible for generating immune responses to blood borne immunogens and plays an important role in preventing septicemia. Removal of the spleen often results in life-threatening infections known as overwhelming post-splenectomy infections (OPSI).

Lymph Nodes; The lymph nodes are encapsulated lymphoid structures located throughout the lymphatic vascular system and provide the tissues and lymph with the same function that white pulp of the spleen provides for blood. Ranging in size from 1 mm to 20mm, lymph nodes are responsible for generating immune responses to the immunogens in the lymph drainage and interstitial fluid that drains from local tissues into the lymph vessels. Lymph nodes are typically bean shaped with two layers, an outer cortex and an inner medulla. Several afferent lymphatic vessels enter into the cortex which is separated into several compartments called follicles. Each follicle leads to the medulla where the lymph fluid is consolidated and one larger efferent lymphatic vessel exits from the medulla. The medulla is also associated with an artery and vein that is used for incoming naïve lymphocytes. The lymph nodes also act as a pump for lymph fluid, activated by random skeletal muscle contraction.
Lymph nodes are designed so that antigen presenting cells from the tissues will come into the lymph node through the afferent lymphatic vessel and encounter B lymphocytes first, then T lymphocytes, and will then take up residence in the medullary cords.

Cytokines; Cytokines are small proteins that affect the behavior of cells. The cytokines may act in an autocrine manner (affecting the cell that

secreted it), paracrine manner (affecting adjacent cells), or even endocrine manner (affecting distant cells). The ability of a cytokine to act on distant cells depends on its ability to enter the blood and how long it stays in the blood (half-life). Each cytokine has its own set of kinases and kinase inhibitors which are important in the regulation of immune responses. Some diseases may not have anything to do with under or overproduction of cytokines, but rather problems with these regulatory proteins. Too much kinase or too little kinase inhibitor will result in abbreviated immune response, while too little kinase or too much kinase inhibitor will result in prolonged immune response.

Chemokines; Chemokines are a subgroup of cytokines that attract other cells, a process called chemotaxis. They function mainly as chemoattractants, recruiting monocytes, neutrophils, and other leukocytes to the area, however, some chemokines also have roles in lymphocyte development and angiogenesis. Chemokines can be secreted by a wide variety of cells including endothelial cells and keratinocytes (skin cells).

UNIT 4 CARING FOR PATIENTS WITH INFLAMMATION

CONTENTS

1.0 INTRODUCTION

Inflammation is defined as the reaction of vascularized living tissue to local injury. It is a defensive reaction intended to neutralize, control or eliminate the offending agent and to prepare the site for repair. Inflammation can also serve to destroy, dilute or isolate the injurious agent (microbes, toxins) and eliminate the necrotic cells and tissues arising as a consequence to such injury while initiating a series of events which leads as far as possible to the healing and reconstitution of the damaged tissue.
During repair, the injured tissue is replaced by:
i. Regeneration of native parenchyma cells
ii. Filling of the defect by fibroblastic tissue or both

Inflammation and repair are protective response, however they may induce harm e.g. anaphylactic reaction, rheumatoid arthritis, atherosclerosis or pericarditis.

Tissues and cells involved in inflammatory response:
The fluid and proteins of plasma, circulating cells, blood vessels and connective tissue

- The circulating cells: neutrophils, monocytes, eosinophils, lymphocytes, basophils, and platelets.
- The connective tissue cells are the mast cells, the connective tissue fibroblasts, resident macrophage and sand lymphocytes.
- The extra cellular matrix, consists of the structural fibrous proteins (collagen, elastin), adhesive glycoproteins (fibronectin, laminin, nonfibrillar collagen, tenascin, etc), and proteoglycans.
- The basement membrane is a specialized component of the extracellular matrix consisting of adhesive glyco proteins and proteoglycans.

Inflammation can be categorized into:
a. Acute inflammation.
b. Chronic inflammation.

Acute inflammation

It is rapid in onset (seconds or minutes), of relatively short duration, lasting for minutes, several hours, or a few days. Its main characteristics are the exudation of fluid and plasma proteins (edema) and the emigration of leukocytes, predominantly neutrophils. It is the rapid response to an injurious agent that serves to deliver mediators of host defense-leukocytes and plasma proteins-to the site of injury.

Acute inflammatory reactions are triggered by a variety of stimuli:

- Infections (bacterial, viral, parasitic) and microbial toxins
- Trauma (blunt and penetrating)
- Physical and chemical agents (thermal injury, e.g., burns or frostbite; irradiation; some environmental chemicals)
- Tissue necrosis (from any cause)
- Foreign bodies (splinters, dirt, sutures)
- Immune reactions (also called hypersensitivity reactions)

Local clinical signs of acute inflammation are; Heat, Redness, Swelling, Pain and Loss of function

Acute inflammation has three major components:

(1) Alterations in vascular caliber that lead to an increase in blood flow

(2) Structural changes in the microvasculature that permit plasma proteins and leukocytes to leave the circulation (increased vascular permeability)

(3) Emigration of the leukocytes from the microcirculation, their accumulation in the focus of injury, and their activation to eliminate the offending agent

Chronic inflammation

It is of longer duration associated histologically with the presence of lymphocytes and macrophages, the proliferation of blood vessels, fibrosis, and tissue necrosis and it is less uniform. Chronic inflammatory processes are debilitating and can be devastating. The prolongation and chronicity of any inflammation may be the result of an alteration in the immune response.

NOTE: The vascular and cellular reactions of both acute and chronic inflammation are mediated by chemical factors that are derived from plasma proteins or cells/ these chemical factors are produced in response to or activated by the inflammatory stimulus. Such mediators, acting singly, in combinations, or in sequence, then amplify the inflammatory response and influence its evolution. Necrotic cells or tissues themselves can also trigger the elaboration of inflammatory mediators e.g. acute inflammation after myocardial infarction.

Inflammation is terminated when the offending agent is eliminated and the secreted mediators are broken down or dissipated. In addition, there are active anti-inflammatory mechanisms that serve to control the response and prevent it from causing excessive damage to the host.

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- define inflammation
- identify the tissues involved in inflammation
- list and describe the types of inflammation
- describe the pathophysiology of inflammation
- enumerate the systemic manifestations of inflammation.

3.0 MAIN CONTENT

3.1 Pathophysiology of Inflammation

The inflammatory response is a sequential reaction to cell injury. It neutralizes and dilutes the inflammatory agent, removes necrotic materials, and establishes an environment suitable for healing and repair. Inflammation is always present with infection, but infection is not always present with inflammation. However, a person who is neutropenic may not be able to mount an inflammatory response. An infection involves invasion of tissues or cells by microorganisms such as bacteria, fungi, and viruses. In contrast, inflammation can also be caused by nonliving agents such as heat, radiation, trauma, and allergens.

The mechanism of inflammation is basically the same regardless of the injuring agent. The intensity of the response depends on the extent and severity of injury and on the reactive capacity of the injured person. The inflammatory response can be divided into:-

(1) Vascular response
(2) Cellular response
(3) Formation of exudates
(4) Healing.

(1) **Vascular Response**; after cell injury, arterioles in the area briefly undergo transient vasoconstriction. After the release of histamine and other chemicals by the injured cells, the vessels dilate. This vasodilatation results in hyperemia (increased blood flow in the area), which raise filtration pressure. Vasodilatation and chemical mediators cause endothelial cell retraction, which increases capillary permeability. Movement of fluid from capillaries into

tissue spaces is thus facilitated. Initially composed of serous fluid, this inflammatory exudates later contains plasma proteins, mainly albumin. The proteins exert oncotic pressure that further draws fluid from blood vessels. The tissue becomes edematous.

(2) **Cellular response**; this is characterized by extravasation of leucocytes from the lumen into interstitial tissue followed by phagocytosis. Extravasation involves the following sequence of events: -

(a) Margination of leukocytes; It is the adherence of leukocytes to the endothelial cells lining. Mainly to the post Capillary venules.

(b) Transmigration of leukocytes across the endothelium to interstitial tissue (also called diapedesis); it is the movement of leukocytes by extending pseudopodia through the vascular wall by a process called diapedesis. Leukocytes escape from venules and small veins but only occasionally from capillaries.

(c) Migration in the interstitial tissues towards a chemotactic stimulus called Chemotaxis; It is a unidirectional leukocyte attraction within tissue space guided by the presence of bacteria and cellular debris. All granulocytes, monocytes and to a lesser extent lymphocytes respond to chemotactic stimuli.

(d) Phagocytosis; Once the cell has reached the site of injurious agent (in interstitial tissue) phagocytosis ensues. Phagocytic cells include polymorpho nuclear leukocytes (particularly neutrophils), monocytes and tissue macrophages. Phagocytosis involves three distinct but interrelated steps:

- Recognition and attachment of the particle to be ingested by the leukocytes: Phagocytosis is enhanced if the material to be phagocyted is coated with certain plasma proteins called opsonins.
- Engulfment; As a result of fusion between the phagosome and lysosome , a phagolysosome is formed and the engulfed particle is exposed to the degradative lysosomal enzymes
- Killing or degradation; the ultimate step in phagocytosis of bacteria (any foreign body) is killing and degradation.

(3) **Exudates Formation**; Exudates consist of fluid and leukocytes that move from the circulation to the site of injury. The nature and quantity of exudates depend on the type and severity of the injury and the tissues involved .Hyperemia from vasodilatation, Increased metabolism at inflammatory site, Change in PH; Change in ionic concentration; nerve stimulation by chemicals (e.g. histamine, prostaglandins); pressure from fluid exudates,

Fluid shift to interstitial spaces; fluid exudates accumulation, Swelling and pain are some of the effects of exudate formation.

Systemic manifestations of inflammation:- Include leukocytosis, malaise, nausea and anorexia, Increased pulse and respiratory rate, and fever. Leukocytosis results from the increased release of leukocytes from the bone marrow. An increase in the circulating number of one or more types of leukocytes may be found. Inflammatory responses are accompanied by the vaguely defined constitutional symptoms of malaise, nausea, anorexia, and fatigue. The causes of these systemic changes are poorly understood but are probably due to complement activation and the release of cytokines (soluble factors secreted by WBCs that act as intercellular messengers) from stimulated WBCs. Three of these cytokines, interleukin-1 (IL-1), interleukin-6 (IL-6), and tumor necrosis factor (TNF), are important in causing the constitutional manifestations of inflammation, as well as inducing the production of fever. An increase in pulse and respiration follow the rise in metabolism as a result of an increase in body temperature, Fever; the onset of fever is triggered by the release of cytokines. The most potent of these cytokines are IL-1, IL- 6, and TNF (released from mononuclear phagocytic cells). These cytokines cause fever by their ability to initiate metabolic changes in the temperature- regulating center. The synthesis of prostaglandin E2 (PGE2) is the most critical metabolic change. PGE2 acts directly to increase the thermostatic set point. The hypothalamus then activates the sympathetic branch of the autonomic nervous system to stimulate increased muscle tone and shivering and decreased perspiration and blood flow to the periphery. Epinephrine released from the adrenal medulla increases the metabolic rate. The net result is fever. With the physiologic thermostat fixed at a higher- than –normal temperature, the rate of heat production is increased until the body temperature reaches the new set point. As the set point is raised, the hypothalamus signals and increases in heat production and conservation to raise the body temperature to the new level. At this point the individual feels chilled and shivers. The shivering response is the body's method of raising the body's temperature until the new set point is attained. This seeming paradox is dramatic: the body is hot yet an individual piles on blankets and may go to bed to go warm. When the circulating body temperature reaches the set point of the core body temperature, the chills and warmth- seeking behavior cease.

- Nonspecific complaints such as mild headache, fatigue, general malaise, and muscle aches
- Cutaneous vasoconstriction, " goose pimples," pale skin; feeling of being cold; generalized shaking chill; shivering causing body to reach new temperature set by control center in hypothalamus
- Sensation of warmth throughout body; cutaneous vasodilatation; warming and flushing of the skin

- Sweating; decrease in body temperature

The released cytokines and the fever they trigger activate the body's defense mechanisms. Beneficial aspects of fever include increased killing of microorganisms, increased phagocytes by neutrophils, and increased proliferation of T cells. Higher body temperature may also enhance the activity of interferon, body's natural virus- fighting substance.

(4) **Healing Process**: the final phase of the inflammatory response is healing. Healing includes the two major components of regeneration and repair. Regeneration is the replacement of lost cells and tissues with cells of the same type. Repair is healing as a result of lost cells being replaced by connective tissue of different origin. Repair is the more common type of healing and usually results in scar formation.

MANAGEMENT

The inflammation resolves following repair of damaged tissue. This process could be natural, if the body's defense mechanism is adequate to bring about resolution without assistance. In cases where resolution does not occur easily, death of some cells may occur as the area inflamed is healed by replacement of destroyed tissue with living cells. The chain of management involves strengthening of the body's defense mechanism and weakening attack

Methods of strengthening defense and weakening the attack includes;

i. Rest; this can be general body rest or locally resting the affected area via the use of splints, slings and sand bags. This further prevents trauma and reduces pain.
ii. Use of the force of gravity; this is done by elevating the affected part to encourage venous and lymphatic drainage, reducing swelling and increasing the flow of fresh blood to the area.
iii. Thermal applications; hot or cold compress can be used, hot compress would cause relaxation of muscle and facilitate blood flow. While cold compress constricts blood vessels, reduces volume of exudate and degree of exudate causing there to be less pressure of the nerve endings thereby the level of pain.
iv. Nutritional supplements; increased calorie requirement is essential to meet the energy demand of the of the body and tissue catabolism during this period. Among the various vitamins, vitamin c is very essential in the formation of fibrous tissue.
v. Maintaining aseptic technique; this promotes wound healing and reduces further inflammation.
vi. Pharmacological intervention; antibiotics can be used to combat infections which could be further impair healing.

Other nursing care that can be accorded are:

i. A comprehensive history should be obtained about the cause of inflammation, duration of onset and all other associated systemic changes. A typology for assessment can be used to serve as a guide for this assessment e.g. the Gordon's typology. As a head to toe assessment may be needed and a focus assessment may also be needed.
ii. Vital signs are obtained
iii. Other functional or neurological assessment should also be conducted
iv. A microscopic culture and sensitivity may be conducted and this would show elevated levels of white blood cells.
v. A nursing care plan is drawn to guide the care accorded based on the signs and symptoms each patient exhibit. Possible nursing diagnosis are:

Impaired tissue integrity
Impaired skin integrity
Hyperthermia
Acute pain
Excess fluid volume
Risk for infection

Nursing Care Plan Using Selected Diagnosis

NURSING DIAGNOSIS	NURSING OUTCOME	NURSING INTERVENTION
Acute pain(00132)	Pain control	Pain management(1400) -Perform a comprehensive assessment to include location, characteristics, onset, duration, frequency, quality, intensity or severity of pain, precipitating factors. -Assure patients of attentive analgesic care. -Explore patient's knowledge and beliefs about pain -Evaluate with the patient and health care team, the effectiveness of past pain control measures that have been used. -Select and implement a variety of measures (e.g Pharmacological, non-pharmacological measures to facilitate pain relief as appropriate). -Teach principles of pain management

		-Teach the use of non-pharmacological techniques e.g. hot/cold application and massage before and after and if possible during painful activities, before pain increases; along with other pan relief measures. -Encourage patient to use adequate pain medications. -Provide the person optimal pain relief with prescribed analgesics.
Hyperthermia(00007)	Thermoregulation	Infection control (6540) -Allocate the appropriate square feet per patient as indicated by the Centre for Disease Control (CDC) and prevention using CDC guidelines. -Maintain an optimal aseptic environment during bedside insertions. -Ensure aseptic environment while changing tubes, bottles and IV lines. -Ensure appropriate wound care techniques. -Promote appropriate nutritional intake -Encourage fluid intake as appropriate -Administer antibiotics therapy as appropriate. -Promote safe food preservation and preparation

UNIT 5 CARING FOR PATIENTS WITH INFECTIOUS DISEASES

CONTENTS

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- describe what infectious diseases are
- draw and explain the chain of infection
- list the signs and symptoms of infection
- describe the management of a patient who has infection.

An infectious disease is the state in which an infected host displays a decline in wellness. It is also defined as the consequences that results from invasion of the body by microorganism or foreign replicators that can produce harm to the body and potentially death. To explain the infectious diseases, an understanding of the chain of infection is necessary.

3.0 MAIN CONTENT

3.1 The Chain of Infection

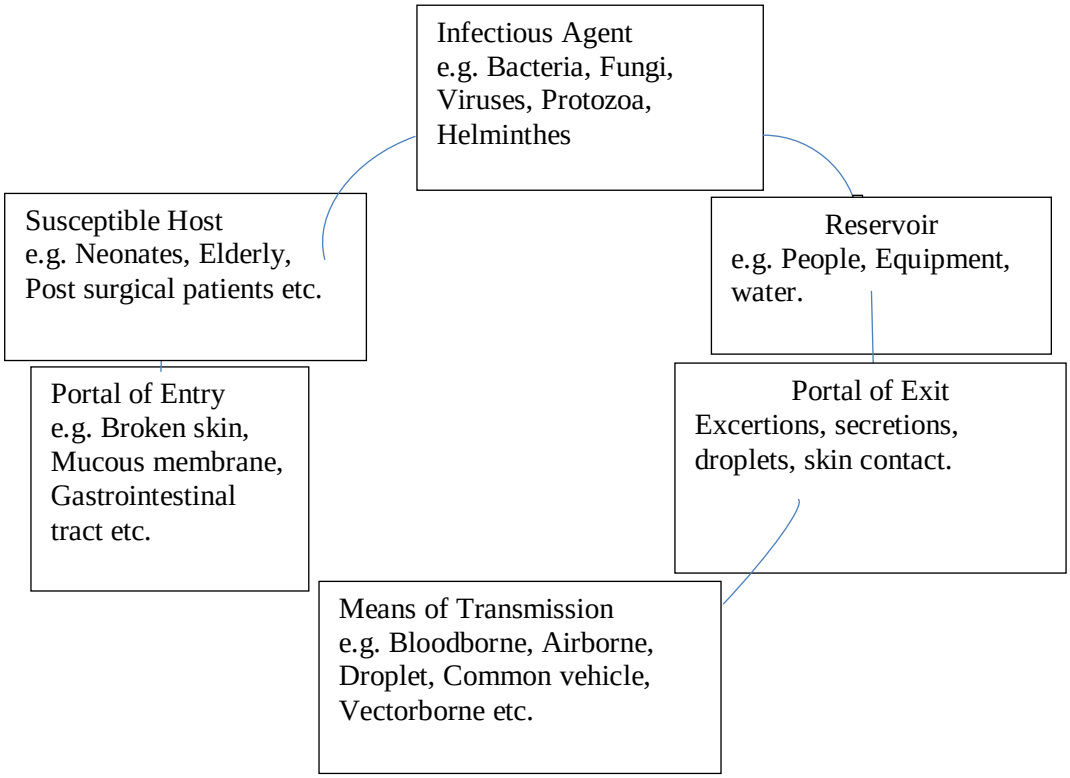
A complete chain is essential for an infection to occur. The elements in the chain are;

i. Infectious agent/ causative organism; these are microorganisms that cause infections. Examples are bacteria, fungi, viruses, protozoa & helminthes.
ii. Reservoir; this can either be a person, equipment, water or any location that provides nourishment for microorganism and allows for further propagation of the microorganisms.
iii. Portal or mode of exit; this is the exit point of the microorganism from the reservoir. For an infectious agent to be propagated, it has to move out from the reservoir. The point at which the microorganisms move out is the mode of exit. Examples are; excretions, secretions, droplets and skin contacts.
iv. Routes/ means of transmission; this is the medium through which the infectious source is connected with a new host. Examples are;

direct contact, ingestion, fomites, airborne, droplet, blood-borne, common vehicle & vector borne.

v. Portal of entry; the intended or new host requires a point of entry for the invading microorganism to come in. this point of entry is called the portal of entry examples are; broken skin, mucous membrane, gastrointestinal tract, respiratory tract, urinary tract etc.

vi. Susceptible host; every organism tries to perform an immune response against an invading agent. The point at which an organism is not able to perform the activity of an immune response on an invading microorganism, the organism becomes a susceptible host. Examples are, neonates, diabetic patients, patients with immune suppression, patients who just had surgery etc.



The Chain of Infection

Examples of microorganisms that can cause infections include, Human immune deficiency virus which causes AIDS, ebola virus which causes ebola, etc.

Student activity; list ten other disease causing microorganisms and the disease they cause

Relevant terms in infectious diseases

i. Disease; illness or diseases or abnormal functioning of body part/s due to specific cause, such as an infection and identifiable by certain signs and symptoms

ii. Communicable disease; this is any disease caused by micro-organism or parasite that can be transmitted from one person to another. E.g. measles
iii. Contagious disease; is a term used to describe a disease that can be transferred from person to person by social ordinary contact. E.g. common cold, chicken pox, typhoid etc.
iv. Cross infection; it is the process by which infective agents are transmitted from their sources to another patient or from paint to nurse. It can be direct or indirect.
v. Vector; an animal that transmits a particular infectious disease. A vector picks up the disease causing organism from a source of infection and carries them in or on its body, and later deposits them where they infect the new host, directly or indirectly. E.g. Mosquitoes, fleas, etc.
vi. Vehicle; this is the carrier of active components of infective agents e.g. water in cholera, food in typhoid, housefly in amoebic dysentery.
vii. Virulence or Pathogenicity; the ability of a microorganism to cause disease. It can also be defined as how rapidly the infection spreads through the body or the mortality from the infection.
viii. Normal flora; these are infective agents that normally inhabit the skin and mucous membrane at specific sites of the body without the tissues being affected or the organisms causing infections. They are also known as commensal organism, even though they are mostly non-essential to life, they are helpful in maintaining the health and normal functioning of the body.
ix. Notifiable diseases; these are medical conditions that must be reported to local health authorities. Notification of certain potentially harmful infectious diseases enable health officers to monitor and control spread of infection. E.g. hepatitis, measles, tuberculosis etc.
x. Nosocomial infection; this refers to hospital acquired infection, the infections usually occurs as a result of hospital admissions.

Factors that predispose to infection

i. Poor nutritional status
ii. Age
iii. Occupation
iv. Exposure to cold
v. Exposure to radiation
vi. Metabolic disturbance
vii. Other diseases such as anaemia, sickle cell disease, diabetes mellitus, immune suppression.

Signs and symptoms

The signs and symptoms of infectious diseases can be multifaceted because various infectious conditions have their own unique signs and symptoms. Common signs and symptoms of all infectious diseases

include: Pyrexia, Weight loss, Pallor, Rashes, Purulent drainage, Pain, Edema, Redness (the last four are common in cases of local infection).

Complications that may arise include: Septicaemia, Septic shock Dehydration, Abscess formation, Endocarditis, Infectious conditions, Congenital abnormalities.

Management

Nursing care encompasses breaking the chain of infection and according due care to clients who have full blown infections.

In preventing the continuity of the chain of infection, the nurse does the following:

- Rapid identification of the organism
- Environmental sanitation to prevent further brooding of the infecting agent
- Disinfectant and sterilization of all items
- Paying prompt attention to the health of employees
- Performing hand hygiene
- Control of excretions and secretions
- Proper trash and waste disposal system
- Isolation and proper quarantine techniques
- Proper food handling techniques
- Air control
- Maintaining standard precautions
- Wound care, catheter care
- Maintenance of aseptic technique
- Recognition of high risk patients
- Treatment of underlying diseases
- Practicing standard precautions
- Vaccination against infectious diseases
- Use of anti-bacterial agents to destroy pathogenic organism and limit their growth.

For clients with infectious diseases, possible diagnosis include;

- Risk for infection
- Deficient knowledge
- Ineffective thermoregulation.

Nursing Care Plan of Some Selected Diagnosis of Patients with Infectious Diseases

Nursing diagnosis	Nursing outcome	Nursing interventions
Risk for infection(00004)	Community risk control	Communicable disease management (8820) -Monitor at risk population for compliance with prevention and treatment . -Monitor adequate continuation of immunization

		in targeted population. -Provide vaccine to targeted population as available. -Monitor sanitation. -Monitor environmental factors that influence the transmission of communicable diseases. -Provide information about adequate control of vector and animal reservoir hosts as needed.
Deficient knowledge (0126)	Knowledge; disease process	Teaching: Disease Process (5602) -Appraise the patient's level of knowledge related to specific disease process. -Explain the pathophysiology of the disease and how it relates to the anatomy and physiology as appropriate -Describe common signs and symptoms of the disease as appropriate. -Identify possible etiologies as appropriate. -Discuss therapy/ treatment options. -Describe rationale behind management/ therapy/treatment recommendations

4.0 REFERENCES/FURTHER READING

Fuad, I. & Welday, M. (2008). Pathophysiology. Ethiopia Public Health Training Initiative. Ethiopia; Hirmaya University.

Famakinwa, O.T. *Synopsis of Medicosurgical Nursing*. Ibadan; Bayosore.

Heyman, P. Anatomy of the immune system supplemental reading.

Smettzer, C.S. Bare, B.G, Hinkle, L.J. & Cheever, H.K., (2008). Brunner & Suddarth'sTtextbook of Medical-Surgical Nursing. (11th Ed). India; Wolters Kluwer.

Waugh, A., Grant, A. (2006). Ross & Wilson Anatomy and Physiology in Health and Illness (10th Ed). Philadelphia; Elsevier.