



NATIONAL OPEN UNIVERSITY OF NIGERIA

SCHOOL OF SCIENCE AND TECHNOLOGY

COURSE CODE: NSS 403

COURSE TITLE: MEDICAL-SURGICAL NURSING 111

COURSE GUIDE

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1.0 Introduction

The course you are about to study is Medical-Surgical Nursing IV. It deals with disorders of the digestive, musculoskeletal, renal, central nervous and endocrine systems. Fluid and electrolyte distribution and acid-base imbalances are also discussed. Medical-Surgical IV, is designed in such a way that you will learn to identify clients/patients suffering from specific medical or surgical conditions, formulate and implement nursing care plans based on the needs of individuals. Manage patients before, during and after specific diagnostic and nursing procedures. A nursing process approach is used in the discussion of the disorders.

2.0 The Course

This course, is divided into three modules.

Module I deals with fluid and electrolyte distribution and imbalances, and shock.

Module 2 deals with the challenges of medical and surgical nursing of patients with disorders of the digestive, hepatic, biliary and musculo-skeletal systems. The diagnostic measures in these medical-surgical conditions are also discussed. The nursing process is used in the care of the patient.

Module 3 deals with the challenges of medical, surgical nursing of patients with disorders of the renal, central nervous and endocrine systems. The diagnostic measures in these medical-surgical conditions are also discussed. The nursing process is used in the care of the patient.

3.0 Course Aim

The goal of this course (medical surgical nursing IV) is to provide you with the necessary knowledge of the art and science of adult medico-surgical nursing and the therapeutic skills needed for effective management of systemic disorders in the body.

4.0 Course Objectives

In addition to the aims above, this course set to achieve some objectives. After going through this course, you should be able to:

- Compare the etiology, clinical manifestations, and management of acute gastritis, chronic gastritis, and peptic ulcer.
- Describe the dietary, pharmacologic, and surgical treatment of peptic ulcer.
- Describe the various types of intestinal obstructions and their management.
- Use the nursing process as framework for care of the patients with an anorectal condition.
- Compare the various types of hepatitis and their causes, prevention, clinical manifestations and management
- Use the nursing process in the care of the patient with cirrhosis of the liver.
- Differentiate between Type 1 and Type 2 diabetes.
- Describe etiologic factors associated with diabetes.
- Develop a plan for teaching insulin self-administration.
- Identify the role of oral antidiabetic agents in diabetic therapy.
- Differentiate between hypoglycemia and diabetic ketoacidosis, and hyperosmolar nonketotic syndrome.
- Compare hypothyroidism and hyperthyroidism: their causes, clinical manifestations, management, and nursing interventions.

5.0 Working through the Course

This course involves that you would be required to spend lot of time to read. The contents of this material are very dense and require you spending great time to study it. This account for the great effort put into its development in the attempt to make it very readable and comprehensible. Nevertheless, the effort required of you is still tremendous. I would advice that you avail yourself the opportunity of attending the tutorial sessions where you would have the opportunity of comparing knowledge with your peers.

The Course Material

You will be provided with the following materials;

Course guide

Study units.

The Study units covered on this course are:

MODULE 1

Unit 1 General introduction to the course

Unit II Fluid and electrolyte imbalances

Unit III Acid–base imbalances

MODULE 1I

Unit 1 Care of clients with digestive disorders

Unit II Hepato-billiary system

Unit III Care of clients with musculo-skeletal disorders

MODULE 1II

Unit 1 Care of clients with renal system disorders

Unit II Care of clients with neurologic disorders

Unit III Care of clients with endocrine disorders

APPENDIX I Normal Values for Laboratory Test

Text Books

In addition, the course comes with a list of recommended textbooks, which though are not compulsory for you to acquire or indeed read, are necessary as supplements to the course material.

1. Timby K. Barbara, Jeanne C. Scherer, & Nancy E. Smith (1999). Introductory Medical-Surgical Nursing. (7th Edition) Lippincott.
2. Kozier Barbara, Glenora Erb. Fundamentals of Nursing. Concepts and procedures. (2nd ed.)
3. Brunner & Suddarth's (2004) Medical Surgical Nursing. (10th ed) Lippincott Wilkins
4. Walsh M., Watson's (1997). Clinical Nursing and Related Sciences. (5th Edition)
5. Suzanne C. Smeltzer, Brenda Bare (2004). Medical Surgical Nursing. Lippincott Williams & Wilkins
6. Ethelwynn L. Stellenbery, Juditt C. Bruce (2007). Nursing Practice: Medical-Surgical Nursing for Hospital and Community. Elsevier Edinburgh.

Assessment

There are two components of assessment for this course. The Tutor Marked Assignment (TMA), and the end of course examination.

Tutor Marked Assignment

The TMA is the continuous assessment component of your course. It accounts for 30% of the total score. You will be given 4 TMA's to answer. Three of these must be answered before you are allowed to sit for the End of the course examination. The TMA's would be given to you by your facilitator and returned after you have done the assignment.

Module 1

Unit I

1. Define the following:
2. Acid – base balance
3. Buffer:
4. Acidosis:
5. Alkalosis:
6. Explain how respiratory mechanism and the kidney helps to maintain acid – base balance.

Unit II

What are the main buffers pairs present in body fluids?

Module II

Unit I

Identify the causes of peptic ulcer diseases, and describe the nursing care of a client with this condition.

Unit II

1. List common findings manifested by clients with cirrhosis.

2. Discuss the nursing management of clients undergoing medical or surgical treatment of a gall bladder disorder.
3. Compare the types of Viral Hepatitis

Unit III

1. Identify the causes of osteomyelitis.
2. Describe methods to prevent or reduce low back pain due to poor posture and body mechanics.
3. Discuss the nursing management for a client with a sprain, dislocation, or cast; who is in traction or undergoing orthopedic surgery; or with an amputation.

Module III

Unit I

1. Explain the difference between acute and chronic renal failure.
2. Explain the purpose of dialysis and name two methods for performing the procedure.

Unit II

1. State the pathophysiology of Alzheimers disease
2. Identify the causes of epilepsy.
3. Discuss the nursing management for a client with epilepsy

Unit III

1. Differentiate between the symptoms of hypoglycemia and hyperglycemia.
2. Discuss the nursing management of the client with diabetes mellitus.

End of Course Examination

This examination concludes the assessment for the course. It constitutes 70% of the whole course. You will be informed of the time for the examination. It may or not coincide with the university semester examination.

Summary

This course intends to provide you with the necessary knowledge of the art and science of adult medico-surgical nursing and the therapeutic skills needed for effective management of systemic disorders in the body.

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MODULE ONE

UNIT I: FLUID AND ELECTROLYTE IMBALANCES

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1.0 Introduction

Almost all medical-surgical conditions threaten fluid and electrolyte balance. There may be deficits or excesses of water or of any electrolyte. The assessment and maintenance of a patient's fluid and electrolyte balance is a major nursing responsibility. This unit describes some basic information about water and electrolytes in the body and the causes and effects of common fluid and electrolyte imbalances. Nursing process approach is used in discussing the condition.

2.0 Objective

By the end of this unit you will be able to:

- Distinguish between electrolyte balance and imbalance.
- Explain all the clinical manifestation of Fluid electrolyte imbalance.
- Apply nurisng process in the assessment and management of conditions due to Fluid electrolyte imbalance

3.0 Main Content

3.1 Definition

Hypervolemia: This is a condition in which the ECF compartment becomes expanded, and there is a surplus of circulating fluid with normal or near normal proportions of electrolytes.

3.1.1 Causes

1. Inability of the kidneys to excrete excess water and electrolytes as seen in chronic renal disease, chronic liver disease congestive heart failure, or administration of oral or parenteral fluids at a rate beyond renal capacity for excretion.
2. Administration of intravenous fluids at a rate beyond renal capacity for excretion especially in patients with impaired kidney function, in infants or elderly people.
3. Fluid retention following administration of large doses of corticosteroids resulting from the increased level of aldosterone.

3.1.2 Clinical manifestations:

These are due to expanded extracellular volume

1. If excess fluids are in the vascular space there will be elevated BP, bounding pulse, distended neck veins, weight gain, dyspnea, crackles (rales), and pretibial and sacral edema. If overload becomes sufficiently severe to exceed the pumping capacity of the left ventricle, pulmonary edema will result.
2. Laboratory findings are variable. Serum osmolality usually remains unchanged. Serum sodium values are not often affected, although they may be low. Hematocrit may be decreased.

3.1.3 Medical management:

The treatment is according to severity but the goal is to obtain a definitive diagnosis of the underlying cause to determine appropriate treatment.

1. Restrict fluids and sodium intake.
2. Administer diuretics e.g. Lasix to eliminate excess fluids.
3. Replace potassium losses secondary to diuretic therapy.
4. Administer dialysis for patients with renal failure or life-threatening hypervolemia.

3.1.4 Nursing diagnosis: Fluid volume excess, edema related to surplus of circulating fluid.

Nursing Objective: Patient's vital signs, physical findings, and laboratory values are within acceptable limits.

Interventions:

1. Assess vital signs and monitor input and output; measure weight daily. Watch out for an irregular pulse, which can be indicative of dangerous hypokalemia.
2. Observe for and report edema, which may not be clinically evident until 5 – 10 pounds of fluid have been retained. Check sacral areas in patients on bed rest. Look for edema in the ankles and pretibial areas of ambulatory patients.
3. Maintain fluid and sodium restrictions as prescribed.
4. Administer diuretics as prescribed.
5. Monitor lab values; be especially alert to decreased potassium in patients on diuretics.
6. Monitor for clinical indicators of potassium depletion during diuretic therapy. These include muscle weakness, cramping, nausea, anorexia, and cardiac dysrhythmias.
7. Replace potassium losses by administering potassium supplements as prescribed.
8. Teach patient about foods high in potassium, including oranges, tomatoes, and bananas.

Nursing diagnoses: Impaired gas exchange related to tissue hypoxia secondary to pulmonary oedema.

Nursing Objective: Patient does not exhibit signs of respiratory dysfunction.

Interventions

1. Monitor character, rate, and depth of respirations; auscultate lung fields for adventitious breath sounds.
2. Keep patient in semi-Fowler's position to facilitate respirations.
3. Teach patient deep-breathing exercises to enhance gas exchange.

3.2 Extracellular volume deficit-Hypovolemia

Hypovolemia is a condition in which depletion of ECF occurs as a result of water and sodium loss in varying proportions from the body, depending on underlying pathology there. One third of the lost is from vascular space and two third from the interstitial space. There is no water shift between intra and extracellular fluid compartment because there is no change in osmolarity.

3.2.1 Causes:

1. Gastro Intestinal (GI) losses such as vomiting, diarrhea, fistulous drainage, ileostomy, gastric suction.
2. Urinary losses from diuretic administration, renal or adrenal disease, diabetes insipidus.
3. Sequestration of fluid that is (plasma-to-interstitial fluid shift). This is with burns, peritonitis, ileus, ascites, acute pancreatitis.
4. Profuse diaphoresis, hyperventilation, fever.
5. Decreased intake of water and electrolytes.
6. Blood loss

3.2.2 Clinical manifestation:

1. Postural hypotension, weak pulse with tachycardia, flattened neck veins, increased respirations, poor skin turgor, longitudinal furrows in the tongue, absence of moisture in the groin and axillae, decreased tearing and salivation, anorexia, nausea, vomiting, weakness, apathy, weight loss, subnormal temperature, and decreased urine output. Shock and coma can ensue if volume depletion is severe.
2. Laboratory findings: BUN is elevated to serum creatinine, and an elevated hematocrit and protein count, all reflective of hemoconcentration. Urinary sodium is decreased, and urine specific gravity is elevated.

3.2.3 Medical management:

The goal is to restore ECF volume and correct the underlying cause.

1. Administer oral or Intravenous fluids to replace water and electrolyte losses while definitive diagnosis is being made. Isotonic fluid such as Lactate Ringer solution and 0.9% saline are given. Blood transfusion may be given in severe shock.

3.2.4 Nursing care

Nursing diagnosis: Fluid volume deficit related to abnormal loss and/or decreased intake.

Nursing objective: Patient's vital signs, physical findings, and lab values are within acceptable limits.

Interventions:

1. Monitor vital signs, laboratory values, and input and output for evidence of dehydration; measure weight daily. Check specific gravity of urine.
2. As appropriate, encourage oral intake or administer prescribed replacement solutions. Observe for indications of fluid overload during rapid IV replacement.
3. Provide oral hygiene at frequent intervals.
4. Obtain accurate measurements of “third space” (interstitial) fluid accumulation areas such as the abdomen and limbs. Measure abdomen or limb(s) at the same place with each assessment. To ensure accuracy, mark the measurement site with indelible ink, and use the same tape measure for all assessments.
5. Position patient in supine with foot slightly elevated to allow blood flow to the brain and the heart.
6. Teach patients on diuretic therapy, leg exercises and to rise up slowly from bed.

3.3 Electrolyte (Osmolality) Disturbances

This disturbance affects both intracellular and extracellular compartments. When osmolality in one compartment is altered water shifts to balance the osmolality. Therefore both compartments become equally increased or decreased.

Sodium: Sodium is the major cation of extracellular fluid and is primarily responsible for osmotic pressure in that compartment. Normal serum sodium concentration is approximately 137 – 144 mEq/L. Body water and electrolyte regulation by the kidneys is based in part on sodium concentration in the ECF. When ECF sodium concentration rises, the kidneys attempt to maintain normal sodium concentration by retaining water. When ECF water increases, sodium is retained. An elevated serum sodium level (hypernatremia) usually reflects a relative ECF water deficit rather than an increase in total body sodium. Hyponatremia exists when the serum sodium concentration in a given amount of plasma water falls below normal. Symptoms might not occur until the serum sodium level is <120–125 mEq/L. Sodium is also important in cellular functioning normal acid–base balance. Aldosterone, which is secreted by the adrenal cortex, is essential in sodium regulation through its effect on renal tubular resorption of sodium.

3.4 Hypernatremia: Increased serum osmolality

3.4.1 Causes:

1. Decreased intake of water due to inability to respond to thirst, such as in an unconscious state; less efficient functioning of the thirst center in the base of the brain, as is commonly seen with the elderly.
2. Increased output of water from severe hypotonic fluid losses through the GI and respiratory tracts,
3. increased urinary water loss through osmotic diuresis, and diabetes insipidus.
4. Increased intake of sodium from excessive administration of concentrated electrolyte mixtures.

3.4.2 Clinical manifestation:

- (1) Intense thirst
- (2) Flushed skin; dry
- (3) Sticky mucous membranes
- (4) Rough, reddened, dry tongue

- (5) Elevated temperature
- (6) Lost of skin turgor
- (7) Agitate behavior such as restlessness, excitement, convulsions; decreased reflexes; oliguria or anuria.

Laboratory findings: Serum sodium >147 mEq/L, increased serum osmolality, urine specific gravity >1.030 (except in diabetes insipidus).

3.4.3 Medical management: The goal is to restore normal sodium concentration.

1. Replace water: Plain water given by mouth may be sufficient in the early stages of sodium excess or if serum sodium <160 mEq/L; IV infusion of hypotonic solution of water and electrolytes (5%Dextrose water intravenously) in advanced stages or if serum sodium is >160 mEq/L. **Note:** Rapid reduction of serum sodium (serum osmolality) may lead to cerebral edema, seizures, or death.
2. Administer diuretics by mouth with plain water.
3. Draw serum sodium levels q6h.

3.4.4 Nursing care

Nursing diagnosis

Fluid volume deficit related to abnormal (hypotonic) loss or decreased intake.

Nursing objective: Patient's vital signs, physical findings, and lab values are within acceptable limits.

Interventions:

1. Monitor vital sign and input and output, and assess skin turgor and mucous membranes for evidence of dehydration. Check urine specific gravity and monitor serum sodium levels.
2. As appropriate, encourage oral fluids or administer prescribed fluid replacement.
3. Administer diuretics, if prescribed.
4. Assess patient's sensorium; institute seizure precautions and notify doctor if significant findings are noted.

3.5 Hyponatremia:

3.5.1 Causes:

1. Loss of sodium-containing fluids from vomiting, diarrhea, profuse diaphoresis, salt-losing nephropathy, adrenal insufficiency,
2. excessive diuretic use together with reduced sodium intake, and
3. Plasma-to-interstitial fluid shift in massive burns and trauma.
4. Impaired renal excretion of water as seen in renal failure, nephrotic syndrome, CHF, and hepatic cirrhosis.
5. Increased intake of water, which dilutes serum sodium:
6. Excessive administration of electrolyte-free IV solutions, fresh-water drowning, or compulsive polydipsia.
7. Secretion of inappropriate antidiuretic hormone (SIADH).
8. Loss from skin such as diaphoresis, large open lesion and burns

3.5.2 Clinical manifestation:

1. Include:
 - (1) anorexia, nausea, vomiting
 - (2) cold and clammy skin,
 - (3) postural hypotension
 - (4) apprehension
 - (5) Seizures.
 - (6) Headache
 - (7) Abdominal cramps.
2. Laboratory findings:
 - (1) serum sodium below normal
 - (2) Urine specific gravity <1.010.

3.5.3 Medical management:

The goal is to restore normal serum sodium levels as quickly as possible without volume overload and to establish a definitive diagnosis to determine appropriate therapy.

1. Replace salt and water orally in cases of mild deficit.
2. Provide parenteral replacement with 3–5% sodium chloride in water if the deficit is severe.
3. Restrict water if the hyponatremia is dilutional.

3.5.4 Nursing care

Nursing diagnosis

Fluid volume deficit or excess related to abnormal fluid loss, increased intake, or interstitial spacing of fluids.

Nursing Objective: Patient's physical findings and lab values are within acceptable limits.

Interventions:

1. Monitor input and output and weigh patient daily.
2. Monitor serial sodium levels.
3. Maintain fluid restrictions, or administer oral or parenteral fluids as prescribed.
4. Provide safety measures as indicated for patients with altered LOC.

3.6 Potassium:

Potassium is the major cation of intracellular fluid, and it plays a leading role in cellular metabolic activities. It is essential for neuromuscular function and is instrumental in maintaining normal cellular water content. Potassium is not stored in the body, nor do the kidneys conserve it. Most of the daily potassium intake is excreted in the urine, with only small amounts lost through perspiration and feces. Potassium excess does not usually develop in the presence of normal renal function. Although only 2% of body potassium are extracellular, serum potassium concentration generally reflects total body potassium and is affected by the pH of ECF. In acidosis, extracellular hydrogen is exchanged for intracellular potassium. An opposite reaction occurs in alkalotic states. The body is intolerant of fluctuations from normal serum potassium concentration, which is 3.5–5.5 mEq/L; excess or deficit can cause a medical crisis.

3.6.1 Hyperkalemia (potassium is >5.0mEq)

3.6.2 Causes:

1. Decreased potassium excretion as seen in renal failure; adrenal insufficiency (Addison's disease).
2. Increased tissue breakdown, as in crush injuries, burns, major surgery, rhabdomyolysis, severe hemolysis, or GI bleeding;
3. Excessive administration of potassium-containing IV solutions or potassium supplements; potassium-sparing diuretics or
4. High doses of penicillin in patients with renal failure;
5. Massive transfusions of stored blood.
6. Redistribution of intracellular potassium resulting from metabolic acidosis.

3.6.3 Clinical manifestation

1. Neuromuscular: Irritability, weakness, paresthesia, muscular or respiratory paralysis.
2. Nausea, diarrhoea, intestinal.
3. Weak heart muscle: Bradycardia, ventricular fibrillation.
4. Laboratory findings: Repeated serum potassium values >5.6 mEq/L.
5. EKG: Tall, peaked T-waves, development of wide, bizarre QRS complexes culminating in ventricular fibrillation or asystole.

3.6.4 Medical management:

The goal is the rapid restoration of normal serum potassium levels.

1. Administer IV calcium gluconate or calcium chloride, 5–10 mL of a 10% solution, to quickly antagonize the toxic neuromuscular and cardiac effects of hyperkalemia, particularly if hypocalcemia is present.
2. Redistribute ECF potassium: IV injection of one ampule of sodium bicarbonate, which causes rapid movement of potassium into the cells; IV administration of hypertonic solutions of glucose and regular insulin, which causes intracellular potassium shift. These are temporary measures for the immediate reduction of serum potassium until potassium removal can be effected by other means.
3. Perform dialysis to remove potassium from the blood.
4. Treat the underlying disease.

3.6.5 Nursing care

Nursing diagnosis.

Ineffective breathing patterns related to restricted chest movement secondary to impairment/paralysis of respiratory muscles.

Nursing Objective: Patient's respiratory rate and depth are within acceptable limits

Interventions:

1. Assess character, rate, and depth of respirations.
2. Reposition patient to enhance aeration. Elevate head of bed to facilitate respirations; ensure that patient deep breathes and coughs at frequent intervals.
3. Suction airway if patient is unable to expectorate secretions.

Risk to alterations in cardiac output: Decreased: Risk of dysrhythmias and cardiac arrest secondary to hyperkalemia.

Desired outcome: Patient's VS and lab and physical findings are within acceptable limits.

1. Monitor EKG, cardiac rate and rhythm, and serial serum potassium values. Notify MD if potassium levels exceed 6.0 – 6.6 mEq/L.
2. Administer IV calcium gluconate or calcium chloride as prescribed.
3. Administer prescribed IV or oral fluids and/or ion-exchange resins.

Knowledge deficit: Foods relatively high in potassium and diuretics that are potassium-sparing.

Desired outcome: Patient can verbalize knowledge of foods that are relatively high in potassium and diuretics that are potassium-sparing.

1. Teach patient the importance of limiting dietary potassium intake.
2. As appropriate teach patient about diuretics that spare potassium.

3.7 Hypokalemia

3.7.1 Causes:

1. GI losses: Diarrhea, vomiting, NG suctioning, intestinal or biliary fistulas.
2. Urinary losses as occurs in renal tubular disorders, osmotic diuresis, administration of potent diuretics, corticosteroid therapy.
3. Inadequate intake as seen in starvation, inadequate replacement during diuretic therapy, prolonged administration of potassium-free parenteral fluids.

3.7.2 Clinical manifestation:

1. (1) fatigue
(2) muscle weakness,
(3) anorexia
(4) nausea
(5) vomiting
(6) decreased bowel sounds, paralytic ileus.
2. Heart arrhythmias
3. Laboratory findings: Repeated serum potassium <3.5 mEq/L.
4. On EKG: There is prolonged P–R interval, flattened or inverted T waves, S–T segment depression, prominent U wave.

3.7.3 Medical management:

The goal is to replenish potassium without inducing hyperkalemia.

1. Administer oral potassium through dietary intake of potassium-rich foods or give oral potassium supplements in liquid, tablet, or powder form.
2. Administer IV potassium chloride if hypokalemia is severe.

3.7.4 Nursing diagnoses

Knowledge deficit: Foods high in potassium and diuretics that spare potassium.

Nursing Objective: Of patient can verbalize knowledge of foods that are high in potassium and diuretics that spare potassium.

Interventions:

1. Teach patient the importance of eating foods in potassium.

2. Give diuretics that spare potassium.

Risk for alterations in cardiac output: Decreased: Risk of dysrhythmias secondary to hypokalemia.

Desired outcome: Patient's vital sign and laboratory and physical findings are within acceptable limits.

1. Monitor vital sign. Assess cardiac rate and rhythm, noting character and intensity of pulse and heart tones.
2. Monitor serum potassium levels. Especially if below Notify MD if K⁺ is below 3.5 mEq/L.
3. Administer oral potassium supplements with a lot of 4 ounces of water or fruit juice to minimize gastric irritation.
4. Where necessary administer prescribed parenteral potassium supplements.

3.8 Calcium:

Calcium serum level is controlled by hormonal activity of the parathyroid glands and is inversely related to phosphate levels. Calcium is necessary for the formation of bones and teeth, blood clotting, maintenance of the normal transmission of nerve impulses, and muscle contraction. Sufficient vitamin D and protein are required for normal calcium utilization. Approximately half the circulating calcium is bound to albumin; the rest is ionized (free). Hypercalcemia refers to excess calcium. Hypocalcemia refers to calcium deficiency.

3.8.1 Hypercalcemia

3.8.2 Causes:

1. Excessive administration of vitamin D.
2. Prolonged immobility
3. Multiple fractures
4. Osteoporosis
5. Osteomalacia.
6. Ingestion of excessive amounts of dietary calcium and/or calcium-containing antacids.

3.8.3 Clinical manifestation:

1. Anorexia
2. Nausea
3. Vomiting
4. Pathologic fractures
5. Deep bone pain
6. Flank pain (related to kidney stone formation)
7. Relaxed skeletal muscles
8. Personality changes
9. Lethargy
10. Stupor
11. Coma.
12. Laboratory findings: Repeated serum calcium levels >5.8 mEq/L.
13. EKG: Shortening of Q-T interval.
14. Radiographic findings: Generalized osteoporosis, urinary calculi, bone cavitation.

3.8.4 Medical management:

The goal is to restore normal serum calcium levels.

1. Promote renal calcium excretion: Rapidly infuse saline solution to induce calcium diuresis (sodium inhibits tubular reabsorption of calcium) and diuretics to prevent volume excess; replace urinary water, sodium, and potassium losses.
2. Restrict calcium intake.
3. Administer steroids to inhibit intestinal absorption of calcium and reduce inflammation and associated calcium-mobilizing stress response.
4. Administer calcitonin subcutaneously or intramuscular to reduce serum calcium levels temporarily when hypercalcemia is caused by increased parathyroid hormone (PTH).
5. Monitor serial serum calcium values.

3.8.5 Nursing diagnoses:

Alteration in pattern of urinary elimination: Dysuria, urgency, or frequency related to presence of renal calculi.

Nursing Objective: Patient relates the return of a normal voiding pattern.

Interventions:

1. Encourage early mobility to prevent further mobilization of calcium from the bones.
2. If patient is on bed rest, assist with ROM exercises.
3. Turn patient q2h, and encourage gastrocnemius, gluteal, and quadriceps muscle-setting exercises.
4. Administer prescribed fluids and medications, and encourage oral fluid intake to dilute urinary calcium, which can result in kidney stones.
5. Monitor I&O and serum calcium levels
6. Strain all urine to check for renal stones.
7. Teach patient to avoid foods and medications high in calcium (eg, cheese, milk, spinach, eggs, peanuts, oysters, and calcium-containing antacids).

3.9 Hypocalcemia

3.9.1 Causes: Loss of calcium-rich secretions through diarrhoea or wound exudate

3.9.2 Clinical manifestation:

1. Muscle cramps, paresthesia, numbness and tingling of the fingers, tetany.
2. Cardiovascular: Hypotension, bleeding if hypocalcemia is severe.
3. Laboratory findings: Repeated serum calcium values <4.5 mEq/L or 8.5 mg/dL (provided that albumin level is within normal range)
4. EKG: Prolonged Q–T interval.

3.9.3 Medical management: The goal is to restore serum calcium level to normal with minimal hypercalciuria.

1. Administer IV calcium: 100–200 mg calcium (10–20 mL 10% calcium gluconate) over 10–15 minutes in acute symptomatic hypocalcemia, followed by IV administration of 600–800 mg calcium gluconate in 1000 mL D₅W (5% dextrose in water), which is titrated until the need can be met orally.
2. Administer oral calcium supplements in less acute conditions.
3. Administer vitamin D to enhance calcium absorption from the GI tract.
4. Monitor serial serum calcium.

3.9.4 Nursing diagnoses:

Potential for injury related to increased risk of seizure activity secondary to hypocalcemia.

Nursing Objective: Patient's physical findings are within acceptable limits.

Interventions:

1. Administer prescribed calcium, vitamin D, and magnesium supplements. Teach patient about foods containing calcium.
2. Observe patient for (1) numbness and tingling around the mouth, an early indicator of hypocalcemia, (2) signs and symptoms of tetany: muscle twitching, facial spasms, and painful tonic muscle spasms.
3. Monitor serum calcium values.
4. Assess for carpopedal spasm when blood supply to hand is decreased.
5. Assess spasm of lip and cheek when the facial nerve is tapped.
6. If significant findings are noted notify doctor.

4.0 Conclusion

Almost all medico surgical condition threatened fluid and electrolyte balance. There may be deficit or excesses of water or any electrolyte.

5.0 Summary

1. Sodium, Potassium, Calcium and Magnesium are major electrolyte in the body.
2. The Buffer systems in the body are haemoglobin, protein carbonic acid concentration and bicarbonate concentration.
3. The normal PH of the body is slightly alkaline is 7.30 – 7.45.

6.0 Tutor Marked Assignment

1. Explain the influence of the buffer system in maintaining acid balance.

7.0 Further Reading and Other Resources

1. Brunner & Suddarth's (2004) Medical Surgical Nursing. (10th ed) Lippincott Wilkins
2. Barbara C, Long and Wilma J. Phipps (1985). Essentials of Medical-Surgical Nursing. A Nursing Process Approach. The C. V. Mosby Company. St.Louis.

UNIT II: ACID-BASE IMBALANCE

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1.0 Introduction

The human body maintains a relatively constant internal environment, of which the balance between acids and bases is one vital aspect. Optimally, cellular processes occur within a narrow range of pH values (concentration of free hydrogen ions). When an imbalance occurs, compensatory mechanisms engage to bring the pH into normal range. Arterial blood gas (ABG) analysis is a clinical tool that can reveal a variety of acid–base disturbances. Arterial blood is slightly alkaline solution with a normal pH range of 7.35 – 7.45. A decrease in the pH below approximate 6.8 or above 7.8 is incompatible with life. A variety of homeostatic mechanism interact to maintain the pH with normal limit

2.0 Objective

By the end of this unit you will be able to:

1. Explain the role of the kidney, lungs and chemical buffers in maintaining acid-base balance.
2. Compare metabolic acidosis and alkalosis with regard to causes, clinical manifestations, diagnosis, and management.
3. Compare respiratory acidosis and alkalosis with regard to causes, clinical manifestations, diagnosis, and management.
4. Interpret arterial blood gas measurements.
5. Demonstrate a safe and effective procedure of venipuncture.
6. Describe measures used for preventing complications of intravenous therapy.

3.0 Main Content

3.1 Acid

There are two categories of acids found in the body: non-fixed(volatile) and fixed (non-volatile).

1. **Nonfixed acids:** These are acids that can change easily between a liquid and gas state. Carbonic acid (carbon dioxide dissolved in water) is the most prevalent non-fixed acid and is primarily controlled and excreted by the respiratory system.
2. **Fixed acids:** These are produced by metabolic processes within the body and buffered and excreted by the kidneys. The three predominant categories include the following:
 - i. Sulfuric, phosphoric, and other acids that are produced from dietary intake.
 - ii. Lactic acid, produced by RBCs, WBCs, skeletal muscles, and the brain, and during periods of anaerobic metabolism (eg, vigorous exercise, cardiac/respiratory arrest).
 - iii. Ketoacids, produced as by-products of fatty acid oxidation. Fatty acids are an alternate energy source for cell metabolism in glucose-deficient states such as starvation and insulin-deficient states such as diabetes mellitus.

3.1.2 Bases

These are substances that are capable of accepting free hydrogen ions. Bicarbonate is the body's pre-dominant base.

3.2 Acid–Base Imbalance

Body fluids are maintained within a pH range of 7.35 to 7.45. Imbalance in the pH of the body leads to acidosis or alkalosis. There are two categories of acid–base imbalance. These are respiratory and metabolic acidosis.

1A. Respiratory acidosis: This is caused by clinical situations that interferes with pulmonary gas exchange, causing retention of CO₂ and increase in the blood carbonic acid. Two major conditions that can cause respiratory acidosis are central nervous system depression and obstructive pulmonary disease.

1B. Respiratory alkalosis: This is the result of lack of carbonic acid due to hyperventilation as in fever and anxiety.

2A. Metabolic acidosis: This occurs because of high acid content in extracellular fluid and low base bicarbonate. This is characterised by deep and rapid breathing as the lungs exhale more CO₂ and the kidneys excrete hydrogen and urine becomes acidic. This is seen in diarrhea, vomiting, diabetes mellitus etc.

2B. Metabolic alkalosis: It occurs when the level of base bicarbonate is high. This may be caused by ingestion of large amounts of sodium bicarbonate or by the loss of chloride through vomiting or gastric suction.

3.3 Maintenance of Acid–Base Balance

There are three ways the body maintains acid–base balance: the buffer system response, respiratory response and renal response

Buffer system response: A buffer is a combination of two or more compounds that can combine either with acids or bases to maintain pH. One common combination is carbonic acid and sodium bicarbonate. Others are the plasma proteins, haemoglobin, phosphate, and ammonium complexes. A buffer may be regarded as a chemical sponge.

Respiratory system response: This involve the change in rate and depth of ventilation. Increased respirations will cause CO₂ levels to decrease, and decreased respirations will increase CO₂ levels.

Renal response: Kidney excretes varying amounts of acid or base thus controlling the body's PH. Controlling base bicarbonate does this. Excess hydrogen are excreted in urine. The kidney also excrete more or less bicarbonate to achieve balance. This occurs over a 2–3 day period and is the slowest of the three reponses.

3.4 Components of Arterial Blood Gases

Normal Values for Arterial Blood Gases

pH—7.40	range 7.38–7.42
Paco ₂ —40 mm Hg	range 36–44
HCO ₃ —24 mEq/L	range 22–26
Pao ₂ —90 mm Hg	range 80–100 (room air)

1. **pH:** The concentration of hydrogen and hydroxyl ions in equivalents per liter, or in the commonly known scale of pH. An increase in hydrogen ions will cause a more acidic environment, and a decrease will cause a more alkaline environment. pH is inversely proportional to the number of hydrogen ions. As they increase in number, the pH decreases (acidosis occurs); as they decrease in number, pH increases (alkalosis occurs).

2. **Paco₂**: The partial pressure of dissolved CO₂ in arterial blood, along with water and CO₂ are an end-products of cell metabolism; therefore, Paco₂ can be considered an index of the effectiveness of ventilation in relation to the metabolic rate. Carbon dioxide is highly soluble and can rapidly diffuse into plasma to form carbonic acid which breaks down to form hydrogen and bicarbonate ions. The formation of hydrogen and bicarbonate ions plays an important role in diffusing O₂ and CO₂ in the lungs and in maintaining electrical neutrality within the RBCs.
3. **HCO₃**: The measurement of bicarbonate ion concentration in the blood. The bicarbonate system is the major and most immediate buffer response. Bicarbonate is a base, and is capable of accepting hydrogen ions. Increased amounts of bicarbonate or other bases can cause an alkaline environment. The kidneys regulate bicarbonate excretion and reabsorption.
4. **Pao₂**: The partial pressure of dissolved oxygen in arterial blood. Oxygen is dissolved and carried in the plasma and combined with haemoglobin in the RBCs. Haemoglobin plays a key role in the transport of CO₂ and O₂ from the lungs and tissue. Generally, haemoglobin has a strong affinity for oxygen, but this affinity can be altered by hydrogen ion concentration, CO₂ concentration, and body temperature.

3.5 Respiratory Acidosis.

3.5.1 Causes: Reduced ventilation states found with respiratory arrest, head/brain trauma, pneumonia, hypoventilation caused by sedation or anesthesia, atelectasis, Guillain-Barre syndrome, chronic obstructive pulmonary disease (COPD).

3.5.2 Signs and symptoms: Early signs include weakness, headache, fatigue, anxiety, and tremors. Progressive signs include dehydration and confusion, leading ultimately to coma if untreated.

3.5.3 Medical management:

- a. Find the cause to determine appropriate treatment.
- b. Administer bicarbonate.
- c. Begin antibiotic therapy for patients with pneumonia.
- d. Administer naloxone hydrochloride for patients who are oversedated.
- e. Replace potassium chloride (because acidosis causes potassium ions to leave and hydrogen ions to enter the cells).

3.5.4 Nursing diagnoses and interventions:

Potential alteration in respiratory function related to prolonged inactivity and/or omission of deep breathing,

Ineffective Breathing Pattern related to decreased respiratory depth secondary to anesthesia, immobility, and guarding with painful surgical incision. These are only a few examples of related nursing diagnoses.

3.6 Metabolic Acidosis

3.6.1 Causes: Build up of fixed acids, as in cardiac arrest, renal failure, keto-acidosis, or ingestion of acidic substances; loss of base, as in diarrhoea.

3.6.2 Signs and symptoms: Kussmaul's respirations, dehydration, lethargy, malaise, fatigue, nausea/vomiting, headache, SOB, vasodilation, tremors, coma... etc

3.6.3 Medical management:

- i. Find the cause to determine appropriate treatment.
- ii. If ketoacidosis is the cause, administer glucose, insulin, or IV potassium chloride.
- iii. Replace fluid losses.
- iv. Administer bicarbonate.
- v. If renal failure is the cause, prescribe diet low in protein and high in carbohydrates.
- vi. Replace phosphates.

3.6.4 Nursing diagnoses: Fluid volume deficit related to abnormal losses.

Nursing objective: Patient's vital signs, physical findings, and lab values are within acceptable limits.

Nursing intervention

1. Monitor input and output and vital sign; evaluate laboratory results for abnormal values of glucose and potassium; monitor EKG for evidence of cardiac dysrhythmias.
2. Assess for signs of dehydration and decreased sensorium.
3. Test urine pH and specific gravity.
4. Encourage intake of fluids and/or administer fluids such as IV lactate and NaHCO_3 as prescribed.
5. Institute seizure precautions if patient exhibits signs of decreased sensorium.

3.7.0 Respiratory Alkalosis

3.7.1 Causes: Hyperventilation states, as in mechanical overventilation, pain, anxiety, brain injury, fever, pulmonary oedema, acute asthma.

3.7.2 Signs and symptoms: Dizziness, lethargy, weakness, tingling, spasms, tetany, anxiety.

3.7.3 Medical management:

- a. Find the cause to determine appropriate treatment.
- b. Decrease ventilations, for example, with sedation or rebreathing apparatus.
- c. Replace sodium and/or chloride.
- d. Replace potassium chloride.

3.7.4 Nursing diagnoses: Ineffective-breathing patterns related to hyperventilation.

Nursing objective: Patient's respiratory rate and depth are within acceptable limits.

Nursing intervention

1. Monitor vital signs..
2. Place patient in semi-fowler's position to enhance ventilation.
3. Allay patient's anxieties.
4. Sedate patient as prescribed.

3.8.0 Metabolic Alkalosis

3.8.1 Causes: Buildup of bicarbonate or base by ingestion of bicarbonate in the form of antacids; loss of chloride or hydrogen ions as with long-term NG suctioning, diuretic therapy, and/or vomiting; and corticosteroid treatment.

3.8.2 Signs and symptoms: Dizziness, lethargy, weakness, dysrhythmias, tetany, hypoventilation, convulsions, irritability, disorientation.

3.8.3 Medical management:

- a. Find the cause to determine appropriate treatment.
- b. Replace fluids; administer Intravenous fluids.
- c. Replace potassium, sodium, and/or chloride, if needed.
- d. Administer acetazolamide to increase excretion of HCO_3^- .

3.8.4 Nursing diagnoses : Fluid volume deficit related to abnormal losses.

Nursing Objective: Patient's vital signs, physical findings, and laboratory values are within acceptable limits.

1. Monitor input and output; monitor for indicators of hypokalemia, such as dysrhythmia and tetany.
2. Ensure minimal bicarbonate administration or ingestion.
3. Administer potassium chloride, sodium chloride, and fluids as prescribed.
4. Use saline rather than water to irrigate NG tube.
5. Institute seizure precautions if indicated.

3.9 Parenteral Fluid Therapy

Intravenous fluid administration is performed in the hospital, out patient diagnostic and surgical settings, clinics, and home to replace fluids, administer medications, and provide nutrients when no other route is available.

3.9.1 Purpose

Generally, Intravenous fluids are administered to achieve one or more of the following goals.

- To provide water, electrolytes, and nutrients to meet daily requirements.
- To replace water and correct electrolyte deficits.
- To administer medications and blood products.

3.9.2 Types of IV Solutions

Solutions are often categorized as isotonic, hypotonic, or hypertonic, according to whether their total osmolality is the same as, less than, or greater than that of blood.

Isotonic Fluids

Fluids that are classified as isotonic have a total osmolality close to that of the ECF and do not cause red blood cells to shrink or swell. The composition of these fluids may or may not approximate that of the ECF.

D₅W

A solution of D₅W has a serum osmolality of 252 mOsm/L. Once administered, the glucose is rapidly metabolized, and this initially isotonic solution then disperses as a hypotonic fluid, one-third extracellular and two-thirds intracellular. During fluid resuscitation, this solution should not be used because it can cause hyperglycemia. Therefore, D₅W is used mainly to supply water and to correct an increase serum osmolality.

Normal Saline Solution

Normal saline (0.9% sodium chloride) solution has a total osmolality of 308 mOsm/L. Because the osmolality is entirely contributed by electrolytes, the solution is often used to correct an extracellular volume deficit. Although referred to as "normal," it contains only sodium and chloride and does not actually simulate the ECF. It is used with administration of blood transfusions and to replace large sodium losses, as in burn injuries. It is not used for heart failure, pulmonary oedema, renal impairment, or sodium retention. Normal saline does not supply calories.

3.9.3 Types of IV Solutions

Several other solutions contain ions in addition to sodium and chloride and are somewhat similar to the ECF in composition. Lactate Ringer's solution contains potassium and calcium in addition to sodium chloride. It is used to correct dehydration and sodium depletion and replaces gastro intestinal losses. Lactated Ringer's solution contains bicarbonate precursors as well.

Hypotonic Fluids

One purpose of hypotonic solutions is to replace cellular fluid, because it is hypotonic as compared with plasma. Another is to provide free water for excretion of body wastes. At times, hypotonic sodium solutions are used to treat hypernatremia and other hyper-osmolar conditions. Excessive infusions of hypotonic solutions can lead to intravascular fluid depletion, decreased blood pressure, cellular oedema, and cell damage. These solutions exert less osmotic pressure than the ECF.

Hypertonic Fluids

Higher concentrations of dextrose, such as 50% dextrose in water, are administered to help meet caloric requirements. These solutions are strongly hypertonic and must be administered into central veins so that they can be diluted by rapid blood flow.

3.9.4 Other IV Substances

When the patient's Gastro Intestinal Tract is unable to tolerate food, nutritional requirements are often met using the IV route. Parental solutions may include high concentrations of glucose, protein, or fat to meet nutritional requirements. The parenteral route may also be used to administer colloids, plasma expanders, and blood products. Examples of blood products include whole blood, packed red blood cells, albumin, and cryoprecipitate. The IV route also delivers many medications, either by infusion or directly into the vein. Because IV medications enter the circulation rapidly, administration by this route is potentially very hazardous. All medications can produce adverse reactions; however, medications given by the IV route can cause these reactions within 15 minutes after administration because the medications are delivered directly into the bloodstream. Administration rates and recommended dilutions for individual medications are available in specialized texts pertaining to IV medications and in manufacturers' package inserts; these should be consulted to ensure safe IV administration of medications.

3.9.5 Nursing Management

Choosing an Intravenous Site

Many sites can be used for Intravenous therapy. Because they are relatively safe and easy to enter, arm veins are most commonly used. The metacarpal, cephalic, basilic, and median veins as well as their branches are recommended sites because of their size and ease of access. Ideally, both arms and hands are carefully inspected before choosing a specific venipuncture site that does not interfere with mobility. For this reason, the antecubital fossa is avoided, except as a last resort. The following are factors to consider when selecting a site for venipuncture:

- Condition of the vein
- Type of fluid or medication to be infused
- Duration of therapy
- Patient's age and size
- Whether the patient is right- or left-handed
- Patient's medical history and current health status
- Skill of the person performing the venipuncture.

After applying a tourniquet, the nurse palpates and inspects the vein. The vein should feel firm, elastic, engorged, and round not hard, flat, or bumpy. Because arteries lie close to veins in the antecubital fossa, the vessel should be palpated for arterial pulsation (even with a tourniquet on), and cannulation of pulsating vessels should be avoided. General guidelines for selecting a cannula include:

- Length: $\frac{3}{4}$ to 1.25 inches long
- Diameter: narrow diameter of the cannula to occupy minimal space within the vein
- Gauge: 20 to 22 gauge for most Intravenous fluids; a larger gauge caustic or viscous solutions; 14 to 18 gauge for blood administration and for trauma patients and those undergoing surgery

Hand veins are easiest to cannulate. The tips should not rest in a flexion area (eg, the antecubital fossa) as this could inhibit the intravenous flow.

3.9.6 Venipuncture Devices

Equipment used to gain access to the vasculitis includes cannulas, needleless intavenous delivery systems, and peripherally inserted central catheter or midline catheter access lines.

Cannulas. Most peripheral access devices are cannulas. They have an obturator inside a tube that is later removed. “Catheter” and “cannula” are terms that are used interchangeably. The main types of cannula devices available are those referred to as winged infusion sets (butterfly) with a steel needle or as an over-the-needle catheter with wings indwelling plastic cannulas inserted through a steel needle. Scalp vein or butterfly needles are short steel needles with plastic wing handles. These are easy to insert, but they are small and nonpliable.

Preparing the IV site

Before preparing the skin, the nurse should ask the patient if he or she is allergic to latex or iodine, products commonly used in preparing for IV therapy. Excessive hair at the selected site may be removed by clipping to increase the visibility of the veins and to facilitate insertion of the cannula and adherence of dressings to the IV insertion site. Because infection can be a major complication of IV therapy, the IV device, the fluid, the container, and the tubing must be sterile. The insertion site is scrubbed with a sterile pad soaked in 10% povidone-iodine (Betadine) or chlorhexidine gluconate solution for 2 to 3 minutes, working from the center of the area to the periphery and allowing the area to air dry. The site should not be wiped with 70% alcohol because the alcohol negates the effect of the disinfecting solution. (Alcohol gadgets are used for 30 seconds instead, only if the patient is allergic to iodine.) The nurse must perform hand hygiene and put on gloves. Nonsterile disposable gloves must be worn during the venipuncture procedure because of the likelihood of coming into contact with the patient’s blood.

3.9.7 Factors Affecting Flow

The flow of an IV infusion is governed by the same principles that govern fluid move in general.

- Flow is directly proportional to the height of the liquid column. Raising the height of the infusion container may improve a sluggish flow.
- Flow is directly proportional to the diameter of the tubing. The clamp on IV tubing regulates the flow by changing the tubing diameter. In addition, the flow is faster through large-gauge rather than small-gauge cannulas.
- Flow is inversely proportional to the length of the tubing. Adding extension tubing to an IV line will decrease the flow.
- Flow is inversely proportional to the viscosity of a fluid. Viscous IV solutions, such as blood, require a larger cannula than do water or saline solutions.

3.9.8 Complications

IV therapy predisposes the patient to numerous hazards, including both local and systemic complications. Systemic complications occur less frequently but are usually more serious than local complications. They include circulatory overload, air embolism, febrile reaction, and infection.

Fluid Overload: Overloading the circulatory system with excessive Intravenous fluids causes increased blood pressure and central venous pressure. Signs and symptoms of fluid overload include moist crackles on auscultation of the lungs, oedema, weight gain, dyspnea, and respirations that are shallow and have an increased rate. Possible causes include rapid infusion of an IV solution or hepatic, cardiac, or renal disease. The risk for fluid overload and subsequent pulmonary oedema is especially increased in elderly patients with cardiac disease; this is referred to as circulatory overload. The treatment for circulatory overload is decreasing the IV rate, monitoring vital signs frequently, assessing breath sounds, and placing the patient in a high Fowler's position. The physician is contacted immediately. This complication can be avoided by using an infusion pump for infusions and by carefully monitoring all infusions. Complications of circulatory overload include heart failure and pulmonary oedema.

Air Embolism: The risk of air embolism is rare but ever-present. It is most often associated with cannulation of central veins. Manifestations of air embolism include dyspnea and cyanosis; hypotension; weak, rapid pulse; loss of consciousness; and chest, shoulder, and low back pain. Treatment calls for immediately clamping the cannula, placing the patient on the left side in the Trendelenburg position, assessing vital signs and breath sounds, and administering oxygen. Air embolism can be prevented by using a Luer-Lok adapter on all lines, filling all tubing completely with solution, and using an air detection alarm on an IV pump. Complications of air embolism include shock and death. The amount of air necessary to induce death in humans is not known; however, the rate of entry is probably as important as the actual volume of air.

Septicaemia and Other Infection: Pyrogenic substances in either the infusion solution or the IV administration set can induce a febrile reaction and septicemia. Signs and symptoms include an abrupt temperature elevation shortly after the infusion is started, backache, headache, increased pulse and respiratory rate, nausea and vomiting, diarrhea, chills and shaking, and general malaise. In severe septicemia, vascular collapse and septic shock may occur. Cause of septicemia include contamination of the IV product or a break in aseptic technique, especially in immunocompromised patients. Treatment is symptomatic and includes culturing of the IV cannula, tubing, or solution if suspected and establishing a new IV site for medication or fluid administration.

Infiltration and Extravasation: Infiltration is the unintentional administration of a nonvesicant solution or medication into surrounding tissue. This can occur when the IV cannula dislodges or perforates the wall of the vein. Infiltration is characterized oedema around the insertion site, leakage of Intravenous fluid from insertion site, discomfort and coolness in the area of infiltration, and a significant decrease in the flow rate. When the solution is particularly irritating, sloughing of tissue may result. Closely monitoring the insertion site is necessary to detect infiltration before it becomes severe.

Phlebitis: Phlebitis is defined as inflammation of a vein related to a chemical or mechanical irritation, or both. It is characterized by a reddened, warm area around the insertion site or along the vein, and swelling. Treatment consists of discontinuing the IV and restarting it in another site, and applying a warm, moist compress to the affected site. Phlebitis can be prevented by using aseptic technique during insertion, using the appropriate-size cannula or

needle for the vein, considering the composition of fluids and medications when selecting a site, observing the site hourly for any complications, anchoring the cannula or needle well, and changing the IV site according to agency policy and procedures.

Haematoma: Haematoma results when blood leaks into tissues surrounding the IV insertion site. Leakage can result from perforation of the opposite vein wall during venipuncture, the needle slipping out of the vein, and insufficient pressure applied to the site after removing the needle or cannula. The signs of a haematoma include ecchymosis, immediate swelling at the site, and leakage of blood at the site.

Treatment includes removing the needle or cannula and applying pressure with a sterile dressing; applying ice for 24 hours to the site to avoid extension of the haematoma and then a warm compress to increase absorption of blood; assessing the sites; and restarting the line in the other extremity if indicated. A haematoma can be prevented by carefully inserting the needle and using diligent care when a patient has a bleeding disorder, takes anticoagulant medication, or has advanced liver disease.

4.0 Conclusion

The balance between acids and bases is one vital mechanism that the body uses to maintain internal homeostasis. When an imbalance occurs, compensatory mechanisms engage to bring the pH into normal range. Arterial blood gas (ABG) analysis is a clinical tool that can reveal a variety of acid–base disturbances.

5.0 Summary

1. Intravenous fluids are administered to provide water, electrolytes, and nutrients to meet daily requirements and to administer medications and blood products.
2. **IV Solutions** are often categorized as isotonic, hypotonic, or hypertonic, according to whether their total osmolality is the same as, less than, or greater than that of blood.
3. There are three ways the body maintains acid–base balance: the buffer system response, respiratory response, and renal response

6.0 Tutor Marked Assignment

1. Define:
 - Acid – base balance
 - Buffer:
 - Acidosis
 - Alkalosis
2. Explain how respiratory mechanism and the kidney helps to maintain acid – base balance.

7.0 Further Reading and Other Resources

Barbara C, Long and Wilma J. Phipps (1985). Essentials of Medical-Surgical Nursing. A Nursing Process Approach. The C. V. Mosby Company. St.Louis.

MODULE TWO

UNIT I: CARE OF CLIENTS WITH DIGESTIVE DISORDERS

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1.0 Introduction

This unit deals with common medical-surgical conditions of the digestive systems from the mouth to the anus

2.0 Objective

By the end of this unit you will be able to:

- List common disorders of the mouth and oesophagus.
- Identify the causes of peptic ulcer diseases
- Identify the causes of intestinal obstruction
- Describe the medical and nursing care of a client with acute abdomen disorders

3.0 Main Content

3.1 Disorders of the Mouth and Oesophagus

3.1.1 Stomatitis

Inflammatory and infectious diseases of the mouth are commonly overlooked in the debilitated hospitalized patient. Typically, they occur secondary to systemic disease and infection, nutritional and fluid deficiencies, poorly fitting dentures, and neglect of oral hygiene, and as side effects of irritants and drugs. Stomatitis (inflammation of the mouth and mucous membrane) is the term generally applied to a variety of mouth disorders characterized by mucosal cell destruction and disruption of the mucosal lining. It is one of major side effects of cancer chemotherapy, occurring in over 30% of the population.

A. Assessment

Clinical manifestations: Oral pain; sensitivity to hot, spicy foods; foul taste; oral bleeding or drainage; fever; xerostomia (dry mouth); difficulty chewing or swallowing; poorly fitting dentures.

Physical examinations: The oral mucosa will appear swollen, red, and ulcerated; the lymph glands may be swollen; and the breath is often foul-smelling. The lips may have cracks, fissures, blisters, ulcers, and lesions; the tongue may appear dry, cracked, and contain masses, lesions, or exudates.

B. Diagnostic Tests

In most incidences, diagnosis of the offending organism is made by physical examination. However, the following may be used in selected patients:

1. Culture: May be taken of the lesion or drainage to identify the offending organism, if appropriate.
2. Platelet count: Taken if bleeding is present in the immunosuppressed patient.

C. Medical Management

The treatment varies, depending on the type of impairment and its cause.

1. **Identify and/or attempt to control or remove causative factor(s)**
2. **Oral hygiene/mouth irrigations.**
3. **Pharmacotherapy**
 - Local/systemic analgesics for relief of pain.
 - Topical/systemic steroids to reduce inflammation and promote healing.

- Antibiotics, antifungals, and antiviral agents to combat infection.
 - Vitamins: to correct deficiencies (eg, vitamin C to strengthen connective tissue in the gums, and niacin and riboflavin to promote efficient cellular growth).
 - Artificial saliva products: To maintain a normal fluid and electrolyte environment in the mouth.
4. **Dietary management:** Food high in protein, high in calories, and high in vitamins is given to promote wound healing, correct the specific deficiency. Hot and spicy foods are restricted, and the consistency of the food can range from liquid to regular, as tolerated. Fluids are encouraged.
 5. **Cauterization of ulcerations:** If necessary.
 6. **Dental restoration and repair:** If indicated.
 7. **Adequate rest** for optimal tissue repair.

D. Nursing Interventions

1. Inspect the mouth for inflammation, lesions, and bleeding. Record observations and reports appropriately.
2. Administer analgesics, corticosteroids, anesthetics, and mouthwashes as prescribed.
3. Provide mouth care q2h or even hourly if indicated.
4. Instruct the patient to brush teeth after meals and at night, using a soft-bristled toothbrush and nonabrasive toothpaste.
5. Dietary alterations may be necessary, for example, changing to a full liquid or pureed diet.
6. Keep the lips moist with emollients such as lanolin or petroleum jelly.
7. Advise patient to avoid irritants, including smoking and foods that are hot, spicy, and rough in texture.
8. Explain the importance of meticulous, frequent oral hygiene and periodic dental exams.

3.1.2 Hiatal Hernia and Reflux Oesophagitis

A hiatal hernia occurs when there is a weakening of the muscular collar around the oesophageal and diaphragmatic junction, permitting a portion of the lower oesophagus and stomach to rise up into the chest during an increase in intra-abdominal pressure. Causative factors include degenerative changes (aging), trauma, oesophageal neoplasms, kyphoscoliosis (a curvature of the spine), and surgery. Increased intra-abdominal pressure can occur with coughing, straining, bending, vomiting, obesity, pregnancy, trauma, constricting clothing, ascites, and severe physical exertion. The incidence of hiatal hernia increases with age, and women and obese individuals are more often affected. Complications of hiatal hernia include pulmonary aspiration of reflux contents, ulceration, hemorrhage, stenosis, obstruction, and gastritis. The most common complication of hiatal hernia is gastroesophageal reflux, the result of an incompetent lower cardiac sphincter that allows regurgitation of acidic gastric contents into the oesophagus.

The most common type of hiatal hernia is the sliding hernia, which accounts for 90% of adult hiatal hernias. It is characterized by the upper portion of the stomach and oesophageal junction sliding up into the chest when the individual assumes a supine position, and sliding back into the abdominal cavity when sitting or standing.

A. Assessment

Many patients are asymptomatic unless oesophageal reflux is also present.

Clinical manifestations: Reflux oesophagitis 1–4 hours after eating, possibly aggravated by reclining, stress, and increased intra-abdominal pressure. Heartburn, belching, regurgitation,

vomiting, retrosternal or substernal chest pain (dull, full, heavy), hiccups, mild or occult bleeding found in vomitus or stools, mild anaemia, and dysphagia also can occur.

Physical examination: Auscultation of peristaltic sounds in the chest, presence of palpitations, abdominal distention.

B. Diagnostic Tests

1. Chest x-ray: May reveal a large hernia
2. Barium swallow: Will reveal gastroesophageal and diaphragmatic abnormalities.
3. Oesophagoscopy and biopsy: Aid in differentiating between hiatal hernia, varices, and gastroesophageal lesions; determine the extent of oesophagitis or ulceration; detect organic stenosis; and rule out malignancies.
4. Esophageal function studies (EFS): Identify primary and secondary motor dysfunction before surgical repair of the hernia is performed.
5. Gastric analysis: To assess for bleeding, this can occur if ulceration is present.
6. CBC: May reveal anaemic conditions if bleeding ulcers are present.
7. Stool guaiac test: Will be positive if bleeding has occurred.
8. EKG: To rule out cardiac origin of pain.

C. Medical Management and Surgical Interventions

Conservative management, is successful in 90% of the cases. The goals are to prevent or reduce gastric reflux caused by increased intraabdominal pressure and increased gastric acid production.

1. **Encourage limitation of activities that increase intra-abdominal pressure:** For example, coughing, bending, straining, and physical exertion.
2. **Restrict or limit gastric acids stimulants:** For example, caffeine and nicotine.
3. **Dietary management:** Small, frequent meals; bland foods; weight reduction for obese patients; food restriction 2–3 hours before reclining.
4. **Elevate head of bed using 6–10 inch blocks** to prevent postural reflux at night.
5. **Restrict tight, waist-constricting clothing.**
6. **Pharmacotherapy**
 - Antiemetics, cough suppressants, and stool softeners to prevent increased intra-abdominal pressure from vomiting, coughing, and straining with bowel movements; antacids to neutralize gastric acid.
 - Cholinergics to promote motility and prevent reflux.
 - Histamine H₂ receptor blockers to suppress acid secretion.

D. Nursing Diagnoses and Interventions

1. Assess and document the amount and character of the discomfort.
2. Administer medications as prescribed.
3. Encourage the patient to follow dietary and activity restrictions.
4. Assess the patient's knowledge of the disorder, its treatment, and the methods use to prevent symptoms and their complications. Provide instructions as appropriate.
5. Explain the need to follow dietary management: eating a low-fat, high-protein diet; eating small, frequent meals; eating slowly; chewing well to avoid reflux; avoiding extremely hot or cold foods; limiting stimulants of gastric acid such as alcohol, caffeine, chocolate, spices, fruit juices, nicotine, and stress; and losing weight, if appropriate.
6. Advise the patient to drink water after eating to cleanse the esophagus of residual food, which can be irritating to the esophageal lining.
7. Explain the effect of alternations in body positions and activities: avoiding the supine position 2–3 hours after eating; sleeping on the right side with the **head of bed** elevated on 6–10 inch blocks to promote gastric emptying; and avoiding bending, coughing,

lifting heavy objects, straining with bowel movements, strenuous exercise, and clothing that is too tight around the waist.

3.1.3 Achalasia

Achalasia (cardiospasm) is a chronic, progressive motor disorder that affects the lower two-thirds of the oesophagus. It is characterized by ineffective peristalsis, a hypertonic lower esophageal sphincter (LES) that does not relax in response to swallowing, and oesophageal dilation. The exact cause of achalasia is unknown, but evidence indicates there is an impairment in the innervative response of the esophagus or parasympathetic activity. Complications of achalasia include esophagitis with oedema and hemorrhage, respiratory complications caused by aspiration of esophageal contents, malnutrition, and a predisposition for esophageal carcinoma. Symptoms can be potentiated by pregnancy, emotional stress, and URIs.

A. Assessment

As the disease progresses, symptoms increase in severity and frequency.

Clinical manifestations: Dysphagia, especially with cold liquids; halitosis; feeling of fullness in the chest; weight loss; and retrosternal pain during or after meals that radiates to the back, neck, and arms. In addition, regurgitation of esophageal contents can occur when the patient is horizontal, and nocturnal choking can occur during the later stages of the disorder.

B. Diagnostic Tests

Barium swallow, esophageal function studies, esophagoscopy, and biopsy may be performed.

C. Medical Management and Surgical Interventions:

Medical management strives toward relieving symptoms caused by the LES obstruction and emptying esophageal contents.

- 1. Activity/positional alterations:** Patient is instructed to remain upright after meals, wait 2–4 hours after a meal before lying down, and sleep with the head of bed elevated or raised on 6–10 in. blocks. In addition, to help increase hydrostatic pressure and thereby facilitate swallowing, patients are taught to arch their back, flex the chin toward the chest, and strain (Valsalva maneuver) while swallowing.
- 2. Dietary management:** Small, frequent meals. The patient is taught to eat and drink slowly in a relaxed environment; avoid rough foods and foods that can cause discomfort, such as spices, stimulants, and cold fluids; and drink fluids with meals to enhance movement of food into the stomach.
- 3. Pharmacotherapy:** Steroids and non-steroidal anti-inflammatory agents are contraindicated because they can cause ulceration.
 - Vitamins and iron supplements: To treat malnutrition and anemia.
 - Antacids: To reduce the amount of gastric acid and relieve pain.
 - Local anesthetics/analgesics: Before meals to minimize discomfort and promote esophageal relaxation.
- 4. Mechanical esophageal dilation with the use of** inflatable tube into the esophagus. This treatment provides temporary symptomatic relief of dysphagia and facilitates emptying of the esophagus.
- 5. Surgical interventions:** The most common procedure is an esophagomyotomy. This enables the the mucosa under the muscular layers to expand and allow food to pass into the stomach unobstructed.

D. Nursing Interventions

1. Monitor I&O;
2. Document weight daily.
3. Administer local anesthetics/analgesics before meals as prescribed to relax the esophagus and aid ingestion.
4. Monitor and document substances patient can and cannot swallow.
5. Provide oral hygiene before and after meals and at bedtime.
6. Restrict or limit (as prescribed) foods and substances that decrease LES pressure, such as fats and refined carbohydrates, as well as stimulants such as chocolate, peppermint, alcohol, and tobacco.
7. Advise the patient to avoid smoking and constrictive clothing.
8. Emphasize the importance of increased nutritional intake and the precautions to take while eating. Teach the patient to eat small, frequent meals; chew thoroughly; eat slowly; and dine in a relaxed atmosphere.
9. Instruct the patient to remain upright after meals, wait 2–4 hours after meals before reclining, and sleep with the HOB elevated.
10. Provide information about how stress can precipitate the symptoms and measures that can be taken to reduce stress.
11. Restrict or limit (as prescribed) foods that can irritate the esophageal lining, for example, coffee, citrus juices, and tomato juice, as well as all other foods known to cause patient distress.
12. Administer vitamin and iron supplements if prescribed.

3.2 Disorders of the Stomach and Intestines

3.2.1 Peptic Ulcers

Peptic ulcer is an erosion of the stomach (gastric ulcer) or duodenum (duodenal ulcer). Erosions can penetrate deeply into the mucosal layers and become a chronic problem; or they can be more superficial and manifest as a more acute problem as a result of severe physiologic or psychologic trauma, infection, or shock (stress ulceration of the stomach or duodenum). Both duodenal and gastric ulcers can occur in association with high-stress lifestyle, smoking, use of irritating drugs, and secondary to other disease states. Ulceration commonly occurs as a part of Zollinger-Ellison syndrome, in which gastrinomas (gastrin-secreting tumors) of the pancreas and/or other organs develop. Gastric acid hypersecretion and ulceration subsequently occur.

Up to 25% of individuals afflicted with peptic ulcers develop complications such as hemorrhage, gastrointestinal obstruction, perforation, or intractable ulcer. With treatment, ulcer healing occurs within 4–6 weeks, but there is potential for recurrence

A. Assessment

Clinical manifestations: Postprandial epigastric pain (eg, burning, gnawing, dull ache) often relieved with ingestion of food or fluids; GI bleeding, as evidenced by hematemesis or melena.

Physical exam: Tenderness over the involved area of the abdomen.

History of Chronic or acute stress: use of irritating drugs such as caffeine, alcohol, steroids, salicylates, reserpine, indomethacin, or phenylbutazone; disorders of the endocrine glands, pancreas, or liver; and Zollinger-Ellison syndrome.

B. Diagnostic Tests

1. **Barium swallow** to detect abnormalities. impaction.
2. **Endoscopy**: Allows visualization of the stomach (gastroscopy), duodenum (duodenoscopy), or both (gastroduodenoscopy).
3. **Gastric secretion analysis**: In this the stomach contents are aspirated and analyzed for the presence of blood and free hydrochloric acid. Achlorhydria (absence of free hydrochloric acid) is suggestive of gastric cancer, while mildly elevated levels suggest gastric ulcer.
4. **CBC**: Reveals a decrease in hemoglobin and hematocrit when acute or chronic blood loss accompanies ulceration.
5. **Stool test**: Positive if bleeding is present.

C. Medical Management and Surgical Interventions

Conservative management is preferred over surgical intervention, with the therapy aimed at decreasing hyperacidity, healing the ulcer, relieving symptoms, and preventing complications.

1. **Dietary management**: Well-balanced diet with avoidance of foods that are not tolerated. A bland diet that limits spicy, irritating foods might be prescribed. Smaller, more frequent meals help to prevent symptoms. For acute episodes of upper gastric hemorrhage, the patient will be on nil by mouth (NPO) and given intravenous fluids (IV) fluid and electrolyte replacement, with foods and fluids introduced orally as bleeding subsides.
2. **Pharmacotherapy**
 - Antacids: Administered to provide symptomatic relief, facilitate ulcer healing, and prevent further ulceration; They are administered after meals and at bedtime, or are given periodically (q1–6h).
 - Histamine receptor antagonists: Administered to suppress secretion of gastric acid and facilitate ulcer healing.
 - Anticholinergics: To suppress gastric acid secretion
 - Sucralfate: An antiulcer agent used to treat duodenal ulcers. This drug coats the ulcer with a protective barrier so that healing can occur.
3. **Surgical interventions**: Indicated for hemorrhage, intractable ulcers, gastric obstruction and perforation. This can be pyloroplasty, vagotomy, subtotal gastrectomy, total gastrectomy:
4. **Lifestyle alterations**: Such as smoking cessation, avoidance of irritating drugs, and stress reduction therapies.

D. Nursing Diagnoses and Interventions

1. **Alteration in comfort**: Acute epigastric pain related to ulcerations.

Nursing Objective:

1. Patient verbalizes a reduction in discomfort and does not exhibit signs of uncontrolled pain.
2. Patient can verbalize knowledge of foods to avoid and the importance of eating smaller, more frequent meals.

Nursing Intervention

1. Assess for and document presence of pain, including its severity, character, location, duration, precipitating factors, and methods of relief.
2. Administer antacids, histamine H₂-receptor antagonists, and/or sucralfate as prescribed.

3. Advise patient to avoid irritating foods and drugs, especially those associated with the symptoms.
 4. Advise patient to eat smaller, more frequent meals.
 5. Provide comfort measures such as distraction, verbal interaction to allow expression of feelings and reduction of anxiety, backrubs, and stress reduction techniques.
2. **Potential for injury** related to risk of gastrointestinal complications (bleeding, obstruction, and perforated) secondary to ulcerative process.

Nursing Objectives

1. Patient does not exhibit signs of gastrointestinal complications; or if they occur, they are detected and treated promptly, resulting in absence of injury.
2. Patient can verbalize knowledge of necessary lifestyle alterations and demonstrates compliance with the medical therapy.

Nursing Interventions

1. Teach the patient the rationale for lifestyle alterations and compliance with medical therapy to prevent exacerbation of the condition. Examples include smoking cessation, stress reduction, and avoidance of irritating foods and drugs.
 2. Assess for indicators of bleeding, including hematemesis, melena, and occult blood in stool. Administer iced saline lavage as prescribed, if necessary.
 3. Monitor patient for indicators of obstruction, including abdominal pain, distention, nausea and vomiting, and the inability to pass stool or flatus.
 4. Be alert to indicators of peritonitis such as abdominal pain, distention and abdominal rigidity, anorexia, nausea, and vomiting.
 5. Teach the patient the clinical manifestations of GI complications and the importance of reporting them promptly.
3. **Alteration in comfort:** Abdominal fullness, weakness, and diaphoresis related to postgastrectomy dumping syndrome.

Nursing Objectives

1. Patient relates the relief of discomfort and does not exhibit signs of uncontrolled pain.
2. Patient can verbalize preventive measures for discomfort.

Nursing Interventions

1. Advise the patient to avoid high-carbohydrate meals, which precipitate and osmotic pull of fluids into the GI tract and contribute to symptoms.
2. Advise the patient to avoid taking liquids with meals and to lie supine after meals to discourage rapid gastric emptying.

E. Patient–Family Teaching and Discharge Planning

Provide patient and SOs with verbal and written information for the following:

1. Importance of following the prescribed diet to facilitate ulcer healing, prevents exacerbation or recurrence, or control postsurgical dumping syndrome. If appropriate, arrange a consultation with a dietician.
2. Medications, including drug name, rationale, dosage, schedule, precautions, and potential side effects.
3. Clinical manifestations of exacerbation or recurrence, and of potential complications.

4. Care of the incision line and dressing change technique, as necessary. Teach patient about the signs of wound infection, including persistent redness, swelling, purulent drainage, local warmth, fever, and foul odor.
5. Role of lifestyle alterations in preventing exacerbation or recurrence of ulcer, including smoking cessation, stress reduction, and avoidance of irritating foods and drugs.

3.2.2 Intestinal Obstruction

Obstruction of the GI tract is a condition in which the normal peristaltic transport of GI contents does not take place. Digestion and absorption of foods and fluids and the elimination of wastes are impaired or totally blocked. Subsequently, nutritional and fluid and electrolyte status are compromised and distention occurs. Increased pressure in the GI tract also can result in perforation and peritonitis and/or strangulation with necrosis. Obstruction can occur anywhere along the GI tract, but most commonly it occurs at the pyloric area of the stomach or in the small or large intestine. Obstruction can occur as a result of the inflammation and oedema that accompany GI disease (peptic ulcers, diverticulitis, colitis, gastroenteritis, trauma); GI surgery with subsequent oedema and possibly adhesions (gastrectomy, appendectomy, colon resection); growths (polyps, tumors); adynamic (paralytic) ileus secondary to peritoneal insult such as surgery or peritonitis; volvulus; or incarcerated hernia.

A. Assessment

Clinical manifestations: Abdominal pain and distention, nausea and vomiting, hic-coughs, and inability to pass stool or flatus. With partial obstruction, however, diarrhoea might be present.

Physical examinations: Distention, poor skin turgor, dry skin and mucous membranes; and borborygmus with peristaltic rushes (periodic loud bursts of noise) noted on auscultation of abdomen. With paralytic ileus, bowel sounds will be absent or diminished.

History of: Abdominal hernia, recent or past abdominal surgery, GI inflammation or perforation secondary to various disease processes (see above).

B. Diagnostic Tests

1. WBC count: Usually elevated in the presence of strangulation.
2. X-ray of abdomen: Will reveal distention of bowel loops, with air and fluid proximal to the obstruction.
3. Barium swallow/barium enema: Used with caution in selected cases to facilitate diagnosis.

C. Medical Management and Surgical Interventions

Management is supportive and aimed at identifying specific causes of obstruction so that appropriate treatments can be instituted.

1. **Activity as tolerated:** With paralytic ileus, the patient is encouraged to ambulate to enhance return of peristalsis.
2. **GI decompression:** This is done with NG or intestinal tube, depending on site of obstruction. The tube is usually attached to intermittent, low suction and the patient is NPO.
3. **IV fluid and electrolyte support.**
4. **Pharmacotherapy:** May include the following:
 - Antibiotics: To prevent infection.
 - Analgesics: For pain relief. However, they can mask symptoms and interfere with diagnosis.
 - Antiemetic agents: For relief of nausea and vomiting.

5. **Surgical Intervention:** Indicated for obstruction that does not subside.

D. Nursing Diagnoses and Interventions

Alteration in comfort: Nausea, distention, and pain related to abdominal visceral disorder and gastrointestinal obstructive process.

Desired outcome: Patient relates a reduction in discomfort and does not exhibit signs of uncontrolled pain.

1. Implement comfort measures to provide pain relief: distraction, backrubs, conversation, relaxation therapy.
2. Administer prescribed analgesics and antiemetic agents as indicated.
3. Maintain patency and proper functioning of the gastric tube.

Fluid volume deficit related to abnormal losses secondary to vomiting and/or gastric decompression of large volumes of GI fluids and decreased intake secondary to fluid restrictions.

Desired outcome: Patient does not exhibit signs of dehydration.

1. Ensure precise measurement and assessment of fluid I&O records. Take special note of the amount of GI aspirate.
2. Administer appropriate IV fluids at the prescribed rate.

E. Patient–Family Teaching and Discharge Planning

Provide patient and SOs with verbal and written information for the following:

1. Specific disease process that precipitated the obstruction and methods to prevent recurrence, such as compliance with prescribed therapies.
2. Medications, including drug name, purpose, dosage, schedule, precautions, and potential side effects.

3.2.3 Hernia

A hernia is a protrusion of the intestine through the abdominal wall.

Causes-- hernia can occur secondary to a congenital weakness in the abdominal wall, a consequence of disease, old age, increased abdominal pressure, or disruption of the abdominal wall secondary to trauma or surgery (incisional hernia).

Risk factors Hernia can be precipitated or aggravated by those factors related to an increased intra-abdominal pressure such as lifting, sneezing, coughing, straining at stool, pregnancy, and obesity.

Sites of hernia--Hernia can develop at the umbilicus, inguinal opening (most common), femoral ring, or at previous surgical or trauma site. Potential complications include strangulation of the protruding bowel, which can result in necrosis and/or infection; and incarceration, with subsequent intestinal obstruction.

A. Assessment

Clinical manifestations: Tenderness and bulging at herniation site; pain with straining.

Physical exam: Bulge with straining will be noted on inspection; palpation of herniation site will reveal a soft and tender mass or bulge. In men, the scrotum should be examined whenever

a hernia is diagnosed or suspected because herniation at the inguinal area can cause herniation at the inguinal area can cause herniation of bowel into the scrotum.

B. Diagnostic Test

X-ray: May reveal presence of a hernia or incarceration. However, diagnosis is made primarily through physical exam.

C. Medical Management and Surgical Interventions

Management is aimed at reduction of the hernia (placement of herniated area back through the abdominal wall) and prevention of strangulation and incarceration.

1. **Activity as tolerated:** With restriction of stretching and straining, and emphasis on proper body mechanics.
2. **Manual reduction:** Placement of the herniated area back to its anatomically correct position. The patient is usually placed in Trendelenburg's position and given sedatives/relaxants to facilitate the procedure.
3. **Truss (firm support):** Might be prescribed for applying pressure to the herniated area to maintain correct anatomic position.
4. **High-residue diet:** To prevent constipation and straining with stools.
5. **Stool softeners and cathartics:** May be prescribed to prevent constipation and straining.
6. **Antibiotics:** Usually prescribed in the presence of strangulation and infection.
7. **With incarceration:** Care of the patient with an obstructive process will apply.
8. **Herniorrhaphy:** Surgery performed when the hernia is irreducible by other means, or when strangulation or incarceration occurs. It is performed under general or regional anesthesia, and often is done on an outpatient basis. Minimal dietary and activity restrictions are necessary.

D. Nursing Diagnoses and Interventions

Alteration in comfort: Pain (especially with straining) related to hernia condition and/or surgical intervention.

Desired outcomes: Patient relates a reduction in discomfort and does not exhibit signs of uncontrolled pain. Patient can verbalize knowledge of activities that can worsen the condition and demonstrates splinting of the incision, use of a truss, and application of scrotal support or ice packs, if appropriate.

1. Assess and document presence of pain: severity, character, location, duration, precipitating factors, and methods of relief. Report presence of severe, persistent pain, which can signal complications.
2. Advise patient to avoid straining, stretching, coughing, and heavy lifting. Teach patient to splint incision manually or with a pillow during coughing episodes. This is especially important during the early postoperative period and for up to six weeks after surgery.
3. Teach patient the use of a truss, if prescribed, and advise its use as much as possible, especially when out of bed.
4. Apply or teach patient application of scrotal support or ice packs, which are often prescribed to limit edema and control pain after inguinal hernia repair.
5. Administer prescribed analgesics as indicated, especially before postoperative activities. Use comfort measures as well: distraction, verbal interaction to enhance expressions of feelings and reduction of anxiety, backrubs, and stress reduction techniques such as relaxation exercises.

Knowledge deficit: Potential for gastrointestinal complications and measures that can prevent their occurrence.

Desired outcome: Patient can verbalize knowledge of the clinical manifestations of gastrointestinal complications and complies with the prescribed measures for prevention.

1. Teach patient to be alert to and report severe and persistent pain, nausea and vomiting, fever, and abdominal distentions, which can herald onset of incarceration or strangulation.
2. Encourage patient to comply with medical regimen: use of a truss or other support and avoidance of straining, stretching, constipation, and heavy lifting.
3. Teach the patient to consume a high-residue diet to prevent constipation

E. Patient–Family Teaching and Discharge Planning

Provide patient and SOs with verbal and written information for the following:

1. Care of incision and dressing change technique, if appropriate.
2. Teach patient the signs of infection at the incision site that require medical intervention: persistent redness, swelling, local warmth, tenderness, purulent drainage, and foul odor.
3. Symptoms of hernia recurrence and postsurgical complications.
4. Postsurgical activity limitations as directed: usually heavy lifting (>5 lb) and straining are contraindicated for about 6 weeks.
5. Importance of proper body mechanics to prevent recurrence, especially when lifting and moving.
6. Prevention of constipation and straining with stools, for example, by following a high-residue diet and using stool softeners and cathartics when needed.
7. Medications, including drug name, purpose, dosage, schedule, precautions, and potential side effects.

3.2.4 Peritonitis

Peritonitis is the inflammatory response of the peritoneum to offending chemical and bacterial agents invading the peritoneal cavity. The inflammatory process can be local or generalized and acute or chronic, depending on the etiology and pathogenesis of inflammation.

Causes ; intraoperative and abdominal trauma; postoperative leakage into the peritoneal cavity; ischemia; ruptured or inflamed organs; poor aseptic techniques, for example, with peritoneal dialysis; and direct contamination of the bloodstream.

Pathophysiology; The peritoneum responds to invasive agents by attempting to localize the infection, which results in tissue edema, the development of fibrinous exudates, and hypermotility of the intestinal tract. As the disease progresses, paralytic ileus occurs, and intestinal fluid, which then cannot be reabsorbed, leaks into the peritoneal cavity. cardiac output and tissue perfusion are reduced as a result of the fluid shift, and this leads to hypoxia and sepsis. If the infection continues, respiratory failure and shock can occur. Peritonitis is frequently progressive and can be fatal. It is the most common cause of death following abdominal surgery.

A. Assessment

Clinical manifestations: Abdominal pain, nausea, vomiting, fever, malaise, weakness, prostration, hiccoughs, diaphoresis.

Physical exam: Presence of tachycardia, hypotension, and shallow and rapid respirations caused by abdominal distention and discomfort. Often, the patient assumes a supine position with the

knees flexed. On abdominal exam, palpation usually reveals distention and abdominal rigidity with general or localized tenderness (often rebound). Auscultation findings include hyperactive bowel sounds during the gradual development of the peritonitis and an absence of bowel sounds during later stages if paralytic ileus occurs.

History of: Abdominal illness, trauma, or surgery,

B. Diagnostic Tests

1. Serum tests: May reveal the presence of leukocytosis, hemoconcentration, and electrolyte imbalance.
2. Arterial blood gases: May reveal the presence of hypoxemia.
3. Urinalysis: Often performed to rule out genitourinary involvement
4. Peritoneal aspiration with culture and sensitivity: May be performed to determine the presence of blood, bacteria, bile, pus, and amylase content and identify the causative organism.
5. Abdominal x-rays: May be performed to determine the presence of abnormal levels of fluid and gas, which usually collect in the large and small bowels.

C. Medical Management and Surgical Interventions

1. **Bed rest** is enforced with patient in semi- or high Fowler's position to enhance fluid shift to the lower abdomen. This which will reduce pressure on the diaphragm and allow for deeper and easier respirations.
2. **NG or intestinal tube:** Inserted to reduce or prevent gastrointestinal distention or ileus and promote intestinal function.
3. **IV fluids, electrolyte therapy, and parenteral feedings:** To correct fluid, electrolytes. Plasma, protein, and blood may be administered to correct hypovolemia, hypoproteinemia, and anemia. Patient is NPO during the acute phase, and oral fluids are not resumed until the patient has passed flatus and the gastric tube has been removed.
4. **CVP catheter:** May be inserted to monitor circulatory status in the critically ill patient.
5. **Parenteral antibiotic therapy Oxygen:** Often prescribed to treat hypoxia and intestinal anoxia.
6. **Narcotics and sedatives:** To relieve severe pain and discomfort once the diagnosis has been confirmed.
7. **Surgical intervention:** May be required to remove the source of infection or drain the abscess and accumulated fluids. This can include the removal of an organ such as the appendix or gallbladder. Drains are usually inserted to remove purulent drainage and excessive fluids. Intestinal decompression may be employed to decrease massive abdominal distention. Intraoperative and postoperative irrigation may be indicated if there has been gross contamination of the the peritoneal cavity with bowel contents.

D. Nursing Diagnoses and Interventions

Alterations in comfort: Pain, abdominal distention, and nausea related to the inflammatory process.

Desired outcome: Patient relates a reduction in discomfort and does not exhibit signs of uncontrolled pain.

1. Keep patient on bed rest to minimize pain, which can be aggravated by activity; provide a restful and quiet environment.
2. Assess and document character and severity of the pain.
3. Administer narcotics, analgesics, and sedatives as prescribed to promote comfort and rest.

4. Explain all procedures to patient to help minimize anxiety, which can augment discomfort.
5. Offer mouth care and lip moisturizers at frequent intervals to help relieve discomfort/nausea from continuous suction, dehydration, and NPO status.

Alteration in respiratory function related to diminished oxygen transport secondary to loss of circulating volume and and/or guarding secondary to severe abdominal pain.

Desired outcome: Patient does not exhibit signs of respiratory dysfunction.

1. Monitor arterial blood gases results and be alert to indicators of hypoxemia, including low PaO₂ and to the following clinical signs: hypotension, tachycardia, hyper ventilation, restlessness, CNS depression, and possibly cyanosis.
2. Auscultate lung fields to assess ventilated and detect pulmonary complications.
3. Keep patient in semi- or high Fowler's position to aid respiratory effort; encourage deep breathing to enhance oxygenation.
4. Administer oxygen as prescribed.

Potential for injury related to risk of GI complications secondary to inflammatory process.

Desired outcome: A worsening condition is detected promptly, resulting in immediate medical treatment and absence of injury to the patient.

1. Assess the abdomen q1–2h during the acute phase and q4h once the patient is stabilized.
2. Monitor for increasing distention by measuring abdominal girth. Auscultate bowel sounds to assess motility.
3. If prescribed, insert NG tube and connect it to suction to prevent or decrease distention.

Potential for injury related to risk of septic shock secondary to infectious process.

Desired outcomes: Patient does not exhibit signs of septic shock; or, if they appear, they are detected promptly, resulting in immediate treatment and absence of injury.

1. Perform comprehensive physical assessments at frequent intervals. Monitor VS (at least q4h, and more frequently if patient's condition is unstable), and be alert to elevated temperature, hypotension, tachycardia, and shallow and rapid respirations, which can occur with sepsis. Also evaluate circulatory status. In the early stages of shock, the skin is usually warm, pink, and dry because of peripheral venous pooling. In later stages, the BP and CVP start to drop and the extremities become cold and pale because of the lack of tissue perfusion.
2. Administer antibiotics promptly as prescribed.
3. Monitor CBC for an increase in WBC count, which occurs with infection; and for hemoconcentration, which occurs with a decrease in plasma volume.
4. If abdominal surgery has been performed, maintain sterile technique with dressing changes and all invasive procedures.

E. Patient – Family Teaching and Discharge Planning

Provide patient and SOs with verbal and written information for the following:

1. Medications, including the drug name, dosage, schedule, purpose, precautions, and potential side effects.

2. Activity precautions as prescribed by MD, such as avoiding heavy lifting (>5 lb), resting after periods of fatigue, getting maximum amounts of rest, gradually increasing activities to tolerance.
3. Advise on signs of recurrence: fever, chills, abdominal pain, vomiting, abdominal distention. If patient has undergone surgery, the signs of wound infection: fever, pain, chills, incisional swelling, persistent redness, purulent drainage.
4. Importance of follow-up medical care; confirm date and time of next medical appointment.

3.2.5 Appendicitis

Appendicitis is the most commonly occurring inflammatory lesion of the bowel and one of the most frequent reasons for abdominal surgery. The appendix is a blind, narrow tube that extends from the inferior portion of the cecum and does not serve any useful function.

Causes obstruction of the appendiceal lumen by a fecalith, inflammation, a foreign body, or a neoplasm.

Pathophysiology

Obstruction prevents drainage of secretions that are produced by epithelial cells in the lumen, thereby increasing intraluminal pressure and compressing mucosal blood vessels. This tension causes impaired viability, which can lead to necrosis and perforation. Inflammation and infection result from normal bacteria invading the devitalized wall. Mild cases of appendicitis can heal spontaneously, but severe inflammation can lead to a ruptured appendix which can cause local or generalized peritonitis

A. Assessment

Clinical manifestations: Vary because of differences in anatomy, size, and age of the individual.

- Early stage: Abdominal pain (either epigastric or umbilical) that is vague and diffuse and later becomes generalized; nausea and vomiting.
- Intermediate, “acute” stage: Pain that shifts from epigastrium to RLQ at McBurney’s point (halfway between umbilicus and right iliac crest) and is aggravated by walking or coughing. Anorexia, malaise, constipation (or occasionally, diarrhea), and diminished or absent peristalsis also can occur.
- Acute appendicitis with perforation: Increasing, generalized pain; recurrence of vomiting.

Physical exam

- Intermediate, “acute” stage: Pain in RLQ elicited by light palpation of abdomen; presence of rebound tenderness; RLQ guarding, rigidity, and muscles spasms; tachycardia; fever; absent or diminished bowel sounds; pain elicited with rectal exam. A palpable, tender mass may be felt in the peritoneal pouch if the appendix lies within the pelvis.
- Acute appendicitis with perforation: Increasing fever; generalized abdominal rigidity. Typically, patient remains still or rigid in either a side-lying or supine position with flexed knees. Presence of abscess can result in a tender, palpable mass.

B. Diagnostic Tests

1. WBC with differential: Will reveal presence of leukocytosis and an increase in neutrophils.

2. Urinalysis: To rule out genitourinary conditions mimicking appendicitis; may reveal microscopic hematuria and pyuria.
3. Abdominal xray: May reveal presence of a fecalith.
4. IVP: May be performed to rule out ureteral stone or pyelitis.

C. Medical Management and Surgical Interventions

Preoperative care:

1. **Bed rest**: For observation.
2. **NPO status**: Parenteral fluids are begun if surgery is imminent.
3. **Pharmacologic therapy**: Narcotics are avoided until diagnosis is certain because they mask clinical manifestations.
 - Antibiotics: To prevent systemic infection.
 - Tranquilizing agents: For sedation.
4. **Obtain consent for operation**
5. **Ice packs**: May be used for some patients to help relieve pain and decrease blood flow to the area, impeding inflammatory response.
6. **NG tube**: Inserted for gastric suction and lavage, if needed. Surgery:
7. **Appendectomy**: Performed as soon as the diagnosis is confirmed and fluid imbalance and systemic reactions have been controlled.

Postoperative care

8. **Activities**: Ambulation begins either the day of surgery, or the first postoperative day; normal activities are resumed 2–3 weeks after surgery.
9. **Diet**: Advances from clear liquids to soft solids during the second through fifth postoperative day; parenteral fluids are continued if required.
10. **Pharmacotherapy**
 - Antibiotics: Continued in the presence of infection.
 - Analgesics: For postoperative pain.

D. Nursing Diagnoses and Interventions

Potential for infection related to risk of rupture, peritonitis, and abscess formation secondary to inflammatory process.

Desired outcomes: 1. Patient does not exhibit signs of infection; or, if they appear, they are detected and reported promptly, resulting in immediate treatment. 2. Patient can verbalize the rationale for not administering enemas or laxatives preoperatively and enemas postoperatively, and demonstrates compliance with the therapeutic regimen.

1. Assess and document quality, location, and duration of pain. Be alert to pain that becomes accentuated and generalized or to the presence of recurrent vomiting, and note whether patient assumes side-lying or supine position with flexed knees. Any of these can signal impending rupture.
2. Monitor VS for elevated temperature, increased pulse rate, hypotension, and shallow/rapid respirations; and assess the abdomen for presence of rigidity, distention, and decreased or absent bowel sounds, any of which can occur with rupture. Report significant findings.
3. Caution patient about the danger of preoperatives self-treatment with enemas and laxatives because they increase peristalsis, which increases the risk of perforation.

Alteration in comfort: Acute pain and nausea related to the inflammatory process.

Desired outcome: Patient verbalizes a reduction in discomfort and does not exhibit signs of uncontrolled pain.

1. Assess and document quality, location, and duration of pain.
2. Medicate patient with antiemetics, sedatives, and analgesics as prescribed; evaluate and document response.
3. Preoperatively, apply ice packs to RLQ as prescribed.
4. Keep patient NPO before surgery; after surgery, nausea and vomiting usually disappear. If prescribed, insert NG tube for decompression.
5. Teach technique for slow, diaphragmatic breathing to reduce stress and help relax tense muscles.

E. Patient–Family Teaching and Discharge Planning

Provide patient and SOs with verbal and written information for the following:

1. Medications, including drug name, dosage, purpose, schedule, precautions, and potential side effects.
2. Care of incision, including dressing changes
3. Observe for signs of infection: fever, chills, incisional pain, redness, swelling, and purulent drainage.
4. Postsurgical activity precautions: Avoid lifting heavy objects (>5 lb) for the first six weeks, gradually increase activities to tolerance.
5. Importance of avoiding enemas for the first few postoperative weeks.

3.2.6 Hemorrhoids

Hemorrhoids are distended, tortuous veins found in the rectum and anus.

Types

Internal hemorrhoids are found proximal to the anal sphincter and are not visible unless they become large enough to protrude through the anus.

External hemorrhoids are distal to the anal sphincter and can become thrombosed if the vein ruptures.

Causes ; conditions that precipitate increased intra-abdominal pressure and/or obstruct venous return, for example, pregnancy, chronic constipation, portal hypertension, infiltrating carcinoma, physical exertion, infection, and ulcerative colitis.

Risk factors: Diet low in fiber, obesity, long-term constipation and straining, lifestyle or career that requires constant sitting or standing.

A. Assessment

Internal hemorrhoids: Bleeding with defecation, anemia if blood loss occurs over an extended period of time, narrowing of stool, mucus discharge. Discomfort is minimal.

External hemorrhoids: Perianal pain and itching, bleeding, and intense pain if a vein ruptures and thromboses.

Physical exam: Detection of internal hemorrhoids with proctoscopy and digital palpation; grapelike appearance of external hemorrhoids on inspection.

B. Diagnostic Tests

1. **Stool test:** To assess for the presence of blood.
2. **CBC:** To assess for anemia from chronic blood loss.

C. Medical Management and Surgical Interventions

1. **Regulation of bowel movements:** Bulk cathartic, stool softener, high-fiber diet, exercise, augmenting fluid intake.
2. **Treat pain and itching:** Warm or cold compresses and warm sitz baths.
3. **Pharmacotherapy:**
--Topical anesthetics such as dibucaine hydrochloride (Nupercainal) ointment,
---anti-inflammatory preparations such as hydrocortisone ointment is given to relieve pain and itching and shrink mucous membranes.
4. **Manual reduction of prolapsed and strangulated hemorrhoids:** Returning hemorrhoids to rectum with a lubricated, gloved finger.
5. **Sclerosing agent:** Injection into the submucosal tissue surrounding the hemorrhoids to produce an inflammatory response, which leads to tissue shrinkage. This is a palliative and temporary measure.
6. **Rubber band ligation:** Nonsurgical method of constricting the circulation of the hemorrhoid and causing tissue necrosis, separation, and sloughing to occur.
7. **Hemorrhoidectomy:** Removal by cautery, clamp, or excision, of hemorrhoids that do not respond to the above therapies.

D. Nursing Diagnoses and Interventions

Alteration in comfort: Postoperative pain and itching.

Desired outcome: Patient relates a reduction in discomfort and does not exhibit signs of uncontrolled pain.

1. Administer topical anesthetics, astringents, and anti-inflammatory preparations as prescribed.
2. Administer cold or warm compresses to rectal area. Provide warm sitz baths 3–4 times a day, or as prescribed.
3. Administer narcotics for severe postoperative pain as prescribed.
4. Ensure that the patient takes stool softeners during the early postoperative period in preparation for the first bowel movement; administer oil-retention enema if prescribed.
5. Provide warm sitz baths after each bowel movement to minimize discomfort and promote healing.

Alteration in bowel elimination: Constipation related to fear of pain with postoperative defecation.

Desired outcomes: 1.Patient has bowel movements without straining during the early postoperative period. 2.Patient can verbalize the rationale for the importance of postoperative bowel movements and complies with the therapeutic regimen.

1. Explain to patient that discomfort is common with the first bowel movements after surgery.
2. Administer stool softeners and bulk cathartics as prescribed.
3. Administer analgesic ½–1 hour before the patient attempt defecation.
4. Encourage ambulation the day of surgery or first postoperative day to enhance peristalsis.

5. In nonrestricted patients encourage fluid intake of at least 2–3 L/day to help soften stools and promote elimination.
6. Document the first bowel movement, which should occur by the third or fourth postoperative day.
7. Stay with the patient or stand just outside the bathroom door because dizziness and fainting are common at this time due to dilation of the pelvic blood vessels.
8. Record the amount and character of the stool and the patient's response.

Alteration in pattern of urinary elimination: Anuria or dysuria related to local swelling and/or presence of rectal packing secondary to hemorrhoidectomy.

Desired outcome: Patient relates the resumption of the normal pattern of voiding.

1. If patient has difficulty voiding in the early postoperative period because of local swelling and/or rectal packing, encourage patient to get out of bed to void.
2. Encourage sitz baths or warm showers, which stimulate the voiding reflex.

Potential fluid volume deficit related to abnormal blood loss secondary to slipped ligatures.

Desired outcomes: Patient can verbalize knowledge of the therapeutic and preventive measures for healing of the operative site and does not exhibit signs of abnormal bleeding or hypovolemia.

1. Observe for signs of pallor, diaphoresis, hypotension, and increasing pulse and respiration rates which are indicative of rectal bleeding.
2. Assist with insertion of a Foley catheter into the rectum and inflation of balloon to provide pressure to bleeding site.
3. Assess for rectal bleeding. After surgery and into the first or second postoperative day, the patient will have rectal packing. Inspect the perianal area for evidence of fresh bleeding. Be alert to excess bleeding, as evidenced by >2 saturated pads in 8 hours and the presence of a frequent, unrelieved urge to defecate, which can signal sequestered hemorrhage.
4. Advise patient to avoid straining or sitting on the toilet longer than necessary.
5. Instruct patient to keep perianal area clean but to avoid vigorous wiping after bowel movement. Encourage sitz baths after every bowel movement to cleanse the rectal area and relieve local irritation.
6. As appropriate, advise the patient to abstain from anal intercourse until proper healing has taken place.
7. Explain to patient that some bleeding can be expected about 8–12 days postoperatively when the sutures begin to dissolve.

Knowledge deficit: Potential for recurrence of hemorrhoids

Desired outcome: Patient can verbalize of the potential for recurrence and can list preventive measures.

1. Advise patient to use mild, bulk cathartics and/or stool softeners if constipation recurs and to avoid straining with defecation.
2. Encourage a diet high in fiber content such as whole grain products (breads, cooked grains, and cereals), apples, peas, and kidney and other dried beans.
3. For nonrestricted patients, explain that a minimum fluid intake of 2–3 L/day is necessary to soften the stool and promote elimination.

4. Encourage daily exercise, which enhances peristalsis and promotes elimination. Advise patient to provide prolonged standing and sitting.

E. Patient–Family Teaching and Discharge Planning

Provide patient and SOs with verbal and written information for the following:

1. Medications, including drug name, dosage, schedule, purpose, precaution, and potential side effects.
2. Methods for preventing constipation such as exercise, diet high in fiber content, augmenting fluid intake, and taking stool softeners or mild, bulk cathartics if necessary.
3. Awareness that some bleeding can occur about 8–12 days postoperatively, when sutures begin to dissolve.

3.3 Intestinal Neoplasms and Inflammatory Processes

3.3.1 Diverticulosis/Diverticulitis

Diverticulosis is acquired small pouches or sacs (diverticula) in the colon formed by the herniation of mucosal and submucosal linings through the muscular layers of the intestine. Diverticulitis is a complication of diverticulosis, and it occurs when one or more diverticula perforate through the bowel wall, resulting in inflammation and infection, which can include peritonitis. If untreated, death can ensue.

Although diverticula can be found anywhere in the colon, they are seen most frequently in the sigmoid colon. It is theorized that diverticula develop secondary to a low-residue diet and increased intracolonic pressure, such as that created with straining to have a bowel movement.

3.3.2 Colorectal Cancer

Colorectal cancer is second only to lung cancer in the annual number of newly diagnosed cancer cases. Over 90% of colorectal cancers are adenocarcinomas, of which 50% are located in the rectum, 20% in the sigmoid colon, 6% in the descending colon, 8% in the transverse colon, and 16% in the cecum and ascending colon. Many arise from malignant degeneration of benign adenomatous polyps. Metastatic disease occurs through lymph nodes, direct extension to adjacent tissues, and the bloodstream.

Causes

Unknown,

Risk factors include ;family history of colorectal cancer, familial polyposis coli, ulcerative colitis and/or granulomatous colitis, and the presence of adenomatous colon polyps.

Locations

Right colon

Left colon

Rectal

A. Assessment

Right colon cancer: Vague, dull abdominal pain. The patient may be asymptomatic.

Physical exam: Possible presence of a palpable mass in the RLQ, black or dark-red stools, and presence of abdominal distention.

Left colon cancer: Increasing abdominal cramping (“gas pains”), change in bowel elimination patterns, decrease in caliber of stools, constipation, vomiting, obstipation, and acute large bowel obstruction causing progressive increase in abdominal pain. Patient may be asymptomatic.

Physical exam: Possible absence of stool felt on rectal examination, presence of bright red blood coating the surface of the stool, and abdominal distention.

Rectal cancer: Sense of incomplete evacuation, tenesmus, and pain (a late manifestation). Patient may be asymptomatic.

Physical exam: Potential presence of palpable mass; bright red blood coating surface of the stool.

History of (for all types of colorectal cancer): Blood on or in stools, change in stool elimination pattern, vague abdominal discomfort or pain.

B. Diagnostic Tests

1. Occult blood test of three serial stool specimens: To detect presence of blood associated with tumor mass bleeding.
2. Proctosigmoidoscopy and/or colonoscopy: To examine areas of intestine visually.
3. Biopsy: To confirm diagnosis.
4. Barium enema with air contrast: To detect colon irregularities suspicious of tumor.
5. Carcinoembryonic antigen (CEA): Serum elevation can be indicative of intestinal tumor.

C. Medical Management and Surgical Interventions

1. **Surgery:** Resection of tumor mass and lymph nodes that drain the area, with reanastomosis of colon. If bowel ends cannot be reanastomosed, a colostomy is created.
2. **Radiation therapy:** To eliminate cancer cells, reduce tumor mass, and/or decrease pain from advanced disease.
3. **Chemotherapy:** For advanced disease, usually fluorouracil (5-FU), alone or in combination with other agents to eliminate cancers cells and provide relief from pain with advanced disease.
4. **Nutritional management:** May include elemental fluid supplements and/or parenteral nutrition if oral intake is inadequate.

3.3.3 Ulcerative Colitis

Ulcerative colitis is a nonspecific, chronic, inflammatory disease of the mucosa and submucosa of the colon.

Causes

The etiology of ulcerative colitis is unknown, but theories include infection, allergy, immunologic abnormalities, psychosomatic factors, and heredity.

Pathophysiology

the disease begins in the rectum and sigmoid colon, but it can extend proximally and uninterrupted as far as the cecum. In some instances, a few centimeters of distal ileum are affected. This is sometimes referred to as “backwash ileitis,” and it occurs in only about 10% of the patients with ulcerative colitis involving the entire colon.

Individuals with ulcerative colitis develop colonic adenocarcinomas at ten times the rate of the general population.

A. Assessment

Clinical manifestations: Bloody diarrhea is the the cardinal symptom. The clinical picture can vary, from acute episodes with frequent discharge of watery stools mixed with blood, pus, and mucus accompanied by fever, abdominal pain, rectal urgency, and tenesmus, too loose or

frequent stools, to formed stools coated with a little blood. Remissions and exacerbations are common.

Physical exam: With severe disease, the abdomen will be tender, especially in the LLQ; and distention and a tender and spastic anus also may be present. With rectal examination, the mucosa might feel gritty, and the examining gloved finger may be covered with blood, mucus, or pus.

B. Diagnostic Tests

1. **Stool examination:** Reveals the presence of frank and/or occult blood and rule out bacterial and parasitic disorders.
2. **Sigmoidoscopy:** Reveals red, granular, hyperemic, and extremely friable mucosa; strips of inflamed mucosa undermined by surrounding ulcerations, which form pseudopolyps; and thick exudates composed of blood, pus, and mucus. **Colonoscopy:** Will help determine the extent of the disease and differentiate ulcerative colitis from Crohn's disease.
3. **Rectal biopsy:** Will aid in differentiating ulcerative colitis from carcinoma and other inflammatory processes.
4. **Barium enema:** Reveals mucosal irregularity from fine serrations to ragged ulcerations, narrowing and shortening of the colon, presence of pseudopolyps, loss of haustral markings, and the presence of spasms and irritability.
5. **Blood tests:** Anemia is common because of iron deficiency and chronic inflammation; leukocytosis and elevated sedimentation rate are common, and hypoalbuminemia and electrolyte disturbances are often found.

C. Medical Management and Surgical Interventions

Medical therapy is symptomatic. The goals are to terminate the acute attack, reduce symptoms, and prevent recurrences.

1. **Parenteral replacement of fluids, electrolytes, and blood products:** To maintain acutely ill patient, as indicated by laboratory test results.
2. **Bed rest and limitation of visitors to promote physical and emotional rest**
3. **Pharmacotherapy**
 - Sedatives and tranquilizers: To promote rest and reduce anxiety.
 - Hydrophilic colloids (eg, kaolin and pectin mixture) and anticholinergics and antidiarrheal preparations (eg, tinctures of belladonna and opium, diphenoxylate hydrochloride, loperamide, and codeine phosphate) to relieve cramping and diarrhea.
 - Anti-inflammatory agents: Corticosteroids to reduce mucosal inflammation.
 - Sulfasalazine: To help maintain remissions.
 - Immunosuppressive agents: To reduce inflammation in patients not responding to steroids and sulfasalazine and who are unwilling or unable to undergo colectomy.
 - Antibiotics: To limit secondary infection.
4. **Nutritional management:** Varies with the patient's condition. A bland, high-protein, high-calorie, low-residue diet with vitamin and mineral supplements and excluding raw fruits and vegetables provides good nutrition and decreases diarrhea. Milk and wheat products are restricted to reduced cramping and diarrhea in patients with lactose and gluten intolerances.
5. **Surgical interventions:** Indicated only when the disease is intractable to medical management or when the patient develops a disabling complication. Total proctocolectomy cures ulcerative colitis and results in construction of a permanent fecal diversion such as ileostomy.

- 6. Postoperative management** includes routine chest physiotherapy to prevent respiratory complications; IV fluid and electrolyte replacement NG tube for decompression until bowel sounds are present and the patient is eliminating flatus or stool; gradual resumption of diet as tolerated following NG tube removal and return of bowel function; aseptic incisional care to prevent infection; and fecal diversion care and teaching.

D. Nursing Diagnoses and Interventions

Alteration in comfort: Pain, abdominal cramping, and nausea related to inflammatory process of the intestines.

Desired outcome: Patient relates a reduction in discomfort and does not exhibit signs of uncontrolled pain, abdominal cramping, and/or nausea.

1. Monitor and document characteristics of pain, and assess whether it is associated with ingestion of certain foods or medications or with emotional stress.
2. As prescribed, maintain patient on NPO to provide bowel rest.
3. Provide nasal and oral care at frequent intervals to lessen discomfort from NPO status or presence of NG tube.
4. Keep patient's environment quiet and plan nursing care to provide maximum periods of rest.
5. Administer sedatives and tranquilizers as prescribed to promote rest and reduce anxiety.
6. Administer hydrophilic colloids, anticholinergics, and antidiarrheals as prescribed to relieve cramping and diarrhea.
7. Observe for intensification of symptoms, which can indicate the presence of complications.

Potential for injury related to risk of complications secondary to intestinal inflammatory disorder.

Desired outcome: If signs of complications appear, they are detected promptly, resulting in immediate medical intervention and absence of injury to the patient.

1. Monitor patient for fever, chills, diaphoresis, and increased abdominal discomfort, which can occur with perforation of the colon and potentially result in localized abscess or generalized fecal peritonitis.
2. Report any evidence of the sudden abdominal distention associated with the above symptoms, since they can signal toxic megacolon. Factors that can contribute to the development of this complication include hypokalemia, barium enema examinations, and use of opiates and anticholinergics.
3. Monitor patient for signs of hemorrhage: hypotension, increased pulse and respiratory rate, pallor, diaphoresis, and restlessness.
4. Assess stool for quality (eg, is it grossly bloody and liquid secondary to large amount of bleeding from mucosa?) and quantity (eg, is it mostly blood or mostly stool?). Report significant findings

Alterations in bowel elimination: Diarrhea related to intestinal inflammatory process.

Desired outcome: Patient experiences fewer episodes of diarrhea.

1. Provide covered bedpan, commode, that is easily accessible and ready to use at times.
2. Empty bedpan and commode promptly to control odor and decrease patient anxiety and self-consciousness.

3. Administer hydrophilic colloids, anticholinergics, and antidiarrheals as prescribed to decrease fluidity and number of stools.
4. Administer topical corticosteroid preparations and antibiotics via retention enema, as prescribed, to relieve local inflammation. If patient has difficulty retaining the enema for the prescribed amount of time, consult with MD regarding the use of corticosteroid foam, which is easier to retain and administer.

Potential impairment of skin integrity: Perineal/perianal area related to irritation secondary to persistent diarrhea.

Desired outcome: Patient's perineal/perianal skin remains intact.

1. Assist patient with cleansing and drying of perineal/perianal area after each bowel movement.
2. Apply protective skin care products, such, as skin preparations, gels, or barrier films, only to normal, unbroken skin. Petrolatum emollients, moisture barrier ointments, and vanishing creams also can be used to prevent irritation from frequent liquid stools.
3. Administer hydrophilic colloids, anticholinergics, and antidiarrheals as prescribed to decrease fluidity and number of stools.

Fluid volume deficit related to abnormal loss secondary to diarrhea.

Desired outcome: Patient does not exhibit signs of dehydration.

1. If the patient is acutely ill, maintain parenteral replacement of fluids, electrolytes, and vitamins as prescribed.
2. Administer blood products and iron as prescribed to correct existing anemia.
3. Monitor I&O; weigh patient daily; and monitor laboratory values to evaluate fluid and electrolyte status.
4. Monitor frequently and consistency of stool. Assess and record presence of blood, mucus, fat, or undigested food.
5. Monitor patient for signs of dehydration: thirst, poor skin turgor, dryness of mucous, membranes, fever, and concentrated and decreased urinary output.
6. When patient is taking food by mouth, provide bland, high-protein, high-calorie, and low-residue diet, as prescribed.
7. Assess tolerance to diet by determining incidence of cramping, diarrhea, and flatulence.

E. Patient–Family Teaching and Discharge Planning

Provide patient and SOs with verbal and written information for the following:

1. Medications, including name, rationale, dosage, schedule, route of administration, precautions, and potential side effects.
2. Dietary management to promote nutritional and fluid maintenance and prevent abdominal cramping, discomfort, and diarrhea.
3. Importance of perineal/perianal skin care after bowel movements.
4. Care of incision, dressing changes, and permission to take baths or showers once sutures/drains are removed.
5. Care of stoma, peristomal/perianal skin, or perineal wound; use of ostomy equipment; and method for obtaining supplies. Sitz baths may be indicated for perineal wound.
6. Medications that are contraindicated (eg, laxatives) or that may not be well tolerated or absorbed (eg, antibiotics, enteric-coated tablets, or long-acting tablets).

7. Gradual resumption of ADLs, excluding heavy lifting (>5 lb), pushing, or pulling for 6–8 weeks to prevent incision herniation.
8. Importance of reporting clinical manifestations that require medical attention, such as change in stoma color from the normal bright and shiny red; peristomal or perineal/perianal skin irritation; signs irritation; diarrhea; incisional pain, drainage, swelling, or redness; clinical manifestations of fluid and electrolyte imbalance; and clinical manifestations of mechanical or functional obstruction.

3.3.4 Fecal Diversions

A. Surgical Interventions

It is sometimes necessary to interrupt the continuity of the bowel because of intestinal disease and/or its complications. The resulting fecal diversion can be located anywhere along the bowel, depending on the location of the diseased and/or injured portion; and it can be permanent or temporary. The most common sites for fecal diversion are the colon and ileum.

1. **Colostomy** is created when the surgeon brings a portion of the colon to the abdominal skin surface. An opening in the exteriorized colon permits elimination of flatus and stool through the stoma. The continuity of the colon can be interrupted anywhere along its length.
2. **Ileostomy:** Ileostomy is created by bringing a distal portion of the ileum up and out onto the surface of the surface of the skin of the abdominal wall. A permanent ileostomy is matured by the same procedure discussed with a permanent colostomy. Surgical indications include ulcerative colitis, Crohn's disease, and familial polyposis requiring excision of the entire colon and rectum.
3. **Ileoanal reservoir:** is a two-stage surgical procedure developed to preserve fecal continence and avoid the need for a permanent ileostomy. an ileal reservoir is constructed just above the junction of the ileum and anal canal. This procedure is an option for patients requiring colectomy for ulcerative colitis or familial polyposis. It is contraindicated in patients with Crohn's disease and incontinence problems.

3.4 Obesity

Obesity is excessive body fat.

Causes

Although overeating is the primary cause, other factors can predispose an individual to obesity, including genetics, the environment, inactivity, endocrine disorders, and excessive weight gain during pregnancy.

Types

Juvenile-onset (hyperplastic developmental) obesity

Is characterized by a marked increase in the number of adipose tissue cells (hyperplasia) and triggered by increased caloric intake during infancy and early childhood. Hyperplasia occurs rapidly in the early years and continues (with excess caloric intake) until growth and development are completed. The fat cells do not disappear, and the number remains constant. Eighty percent of obese children become obese adults. Adult-onset (hypertrophic/mature/reactive) obesity is characterized by enlargement of individual adipose cells; the total number remains constant.

Adult-onset obesity

Is not as severe as juvenile obesity, and weight loss is usually more successful. This type of obesity often occurs in response to traumatic or stressful life events.

Obesity affects multiple organ systems and is a major contributing factor to male premature death, fatal myocardial infarctions in males under age 40, coronary artery disease in males and females over age 40, hypertension and hyperlipidemia, cerebrovascular accidents, gynecologic irregularities, toxemia during pregnancy, postoperative complications, orthopedic problems in the lower extremities and spine, and peripheral vascular disorders. Mortality rate is higher for obese individuals, and most morbidly obese people live to less than age 50. Obesity can precipitate diabetes mellitus and the majority of diabetics over age 40 are obese when the disorder is diagnosed. Obese individuals also can suffer social and emotional problems associated with their weight problems.

A. Assessment

Overweight: Weight that is greater than the average (ideal) weight given in insurance tables for given sex and height in the United States.

Obesity: Weight > 20% over ideal body weight.

Morbid obesity: Weight >45 kg (100 lb) above normal weight for 3 or more years.

B. Diagnostic Tests

There is no test specific for obesity. Obtaining weight, height, and anthropometric measurements in conjunction with weight and diet history provides evidence for the diagnosis of obesity.

C. Medical Management and Surgical Interventions

- 1. Diet:** This is the cornerstone of obesity therapy. The caloric intake must be lowered below the caloric expenditure. A caloric deficit of 3500 kcal is required to lose one pound. Diet education is crucial to diet therapy because new eating habits must be formed before therapy can be successful on a long-term basis. Starvation/semistarvation is not a diet of choice because lean body mass is lost to a significant extent rather than adipose tissue.
- 2. Exercise:** This is also an integral part of weight loss/maintenance program. It increases caloric expenditure, keeps tissues firm, and aids circulation and digestion. It must be coupled with diet to afford significant weight reduction.
- 3. Pharmacotherapy:** Is used only as an adjunct to diet and exercise.
 - Anorexiant agent: aid in “reprogramming” eating habits.
 - Thyroid hormones: Used if there is evidence of hypometabolism.
 - Maintenance vitamin supplements: Often indicated, especially if the patient reports use of “fad” diets. Amphetamines are avoided.
- 4. Psychotherapy:** Used in conjunction with diet and exercise therapy program is helpful. Group therapy can help to reinforce motivation. Diets approved by these groups have been proven to be nutritionally sound. Behavior modification is also useful.
- 5. Surgical treatment (bariatric operations):** Used as a last resort when all medical therapies for successful weight loss have failed. Usually, it is reserved for individuals who are morbidly obese.

D. Nursing Interventions

- 1.** Because weight may show daily fluctuations due to water loss/retention, weigh patient no more than once or twice a week. Ensure a consistent schedule, however.
- 2.** Discourage SOs from bringing in food to the patient. A thorough explanation of the diet often alleviates this problem.
- 3.** Assess cultural and economic influences of family diet. Formulating a diet regimen based on realistic family information leads to best results.

4. Instruct patient to record typical 24-hour dietary intake, including the time, amount, type of food, and feeling prior to eating. This will help patient determine whether the eating response is stimulated by internal or external factors.
5. Reinforce for patient and SOs that food is not reward or solace and that being overweight is not a sign of happiness or prosperity.
6. Provide lists of low-calorie nutrition foods for snacks, if they are allowed
7. Provide exercise (unless contraindicated) as an adjunct to diet therapy.
8. Review diet plan with patient and SOs and answer questions.

Table 1 Suggested Low-Calorie Snacks

any fresh fruit (bananas and kiwi fruit are acceptable in small quantities)	skim milk (½ cup)
raw vegetables: carrots, broccoli, celery, cauliflower, snowpeas, tomatoes	sugar-free beverages
plain yogurt	sugar-free gelatins
plain herb tea	crackers (2)
plain coffee	
low-fat cottage cheese (½ cup)	

4.0 Conclusion

Obstruction of the GI tract is a condition in which the normal peristaltic transport of GI contents does not take place. Digestion and absorption of foods and fluids and the elimination of wastes are impaired or totally blocked. Subsequently, nutritional and fluid and electrolyte status are compromised and distention occurs.

5.0 Summary

Peptic ulcer is an erosion of the stomach (gastric ulcer) or duodenum (duodenal ulcer). Hiatal hernia occurs when there is a weakening of the muscular collar around the esophageal and diaphragmatic junction, permitting a portion of the lower esophagus and stomach to rise up into the chest during an increase in intra-abdominal pressure.

Achalasia (cardiospasm) is a chronic, progressive motor disorder that affects the lower two-thirds of the esophagus.

6.0 Tutor Marked Assignment

Identify the causes of peptic ulcer diseases, and describe the nursing care of a client with this condition.

7.0 Further Reading and Other Resources

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UNIT II: HEPATO-BILIARY SYSTEM

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1.0 Introduction

The liver lies directly beneath the diaphragm and occupies most of the right upper quadrant of the abdomen. It governs the formation and secretion of bile. The gall-bladder, which lies directly beneath the right lobe of the liver, and the hepatic, cystic, and common bile ducts comprise the biliary system. The biliary duct system transports bile from the liver to the gallbladder. Bile is concentrated and stored in the gallbladder and released to the small intestine (duodenum), where it facilitates the absorption of fats, fat-soluble vitamins, and certain minerals, and also activates the release of pancreatic enzymes. If an obstructive lesion is present in the biliary ducts, the flow of bile is blocked, resulting in hemoconcentration. When this occurs, a variety of clinical manifestations can surface, including obstructive jaundice, dark-amber urine, and clay-colored stools. Pruritus occurs because of the deposition of bile salts in skin tissue. Steatorrhea and bleeding tendencies result from the inability of the duodenum to absorb fats and fat-soluble vitamins A, D, E, and K. Vitamin K is necessary for adequate clotting of the blood.

2.0 Objective

1. List common findings manifested by clients with hepatic disorders.
2. Discuss four complications that often accompany cirrhosis.
3. Identify a factor that contributes to cholecystitis.
4. Discuss the nursing management of clients undergoing medical or surgical treatment of a gallbladder disorder.

3.1 Hepatic and Biliary Disorders

3.1.1 Hepatitis

Causes

Viral hepatitis is caused by one of the hepatitis viruses: A, B, non-A or non-B,

Pathophysiology

Although their symptoms is similar, immunologic and epidemiologic characteristics are different. When hepatocytes are damaged, necrosis and autolysis can occur, which in turn lead to abnormal liver functioning. Generally, these changes are completely reversible after the acute phase. In some cases, however, massive necrosis can lead to liver failure and death. Jaundice may be seen in any patient with decreased hepatic function. It is classified as prehepatic (hemolytic), caused by increased production of bilirubin; hepatic (hepatocellular), which is caused by the dysfunction of the liver cells; or posthepatic (obstructive), caused by an obstruction of the flow of bile out of the liver.

A. Assessment

Clinical manifestations: Nausea, vomiting, anorexia, signs of URI, fatigue, irritability, distaste for cigarettes in smokers, slight to moderate temperature increases, epigastric discomfort, dark urine, clay-colored stools, pruritus.

Physical exam: Inspection of the skin, mucous membranes, and sclera may reveal yellow coloration; palpation of lymph nodes and abdomen may reveal lymph-adenopathy, hepatomegaly, and splenomegaly.

History of: Blood dyscrasia, multiple blood transfusions, alcohol or drug abuse, exposure to hepatotoxic chemicals.

B. Diagnostic Tests

1. **Hematologic tests:** Anti-HAV IgM will be present with hepatitis A, HbsAg, hepatitis B. SGOT (frequently called serum aspartate transaminase, AST) and SGPT initially will be elevated and then drop. Total bilirubin will be elevated and the PT will be prolonged. Differential WBC count will reveal leukocytosis, monocytosis, atypical lymphocytes, and plasma cells.
2. **Bromsulphalein (BSP) excretion test:** Dye is injected intravenously. In a normal liver it is almost completely cleared from the blood; in hepatitis the blood level is elevated.
3. **Urine tests:** Will reveal elevation of urobilinogen.

C. Medical Management

1. **Bed rest:** During acute phase and when patient is fatigued.
2. **Diet:** No alcohol for 6 months. Parenteral and/or enteral nutrition may be initiated if anorexia is severe. Vitamins are usually given.

Table 2 Comparison of Characteristics of Types of Viral Hepatitis.

	Type A	Type B	Non-A Non-B
Mode of Transmission	Fecal-oral route; large-scale outbreaks caused by contamination of food or water	Percutaneous inoculation (needle stick); usually through blood, but may result from saliva or semen	Usually blood; also semen and saliva
Population Affected	More common in children and in overcrowd areas with poor sanitation	All ages. Drug addicts, male homosexuals and sexual partners of infected individuals. Patients and staff in hemodialysis units are at high risk.	All ages. Highest risk in recipients of blood transfusions. Also at risk are drug addicts and hemodialysis patients. Nosocomial spread possible.
Diagnosis of Acute Disease	Anti-hepatitis A virus (IgM) antibody in serum (anti-HAV IgM). 2 – 6 weeks	Hepatitis B surface antigen in serum (HbsAg). 6 weeks – 6 months	When causes or type A and type B are ruled out. 2 weeks – 6 months
Incubation Period			
Carrier State	No	Yes	Yes
Chronicity	No	Yes	Yes
Measures for Reducing Exposure	Handwashing, stool precautions first 2–3 weeks	Handwashing, wearing gloves when handling body fluids and masks when fluids may splatter, using care when discarding needles and syringes; autoclaving all nondisposable items. Patient can never become a blood donor.	Same as for type B.
Prophylaxis	IG (immune globulin) before or within 1–2 weeks after exposure.	HBIG (hepatitis B immune globulin) within 24 hours after exposure and 1 month later.	Still controversial, but currently a single dose of IG is recommended.

Hepatitis B vaccine recommended for medical and laboratory personnel, male homosexuals, neonates of infected mothers, and sexual partners of chronic HbsAg carriers.

Table 3 Hepatotoxic Drugs

Generic/Category Name	Common Trade Names
Acetaminophen	Tylenol
acetylsalicylic acid	Aspirin
Chlorpromazine	Thorazine
dantrolene sodium	Dantrium
Isoniazid	Isotamine
Methyldopa	Aldomet
nitrofurantoin macrocrystals	Macrochantin
phenytoin sodium	Dilantin
propylthiouracil (PTU)	Propyl-Thyracil
Sulfonamides	Bactrim, Septra, Gantrisin

3. **Manage pruritis:** Restrict alkaline soaps; prescribe emollients. Antihistamines and tranquilizers, if used, are administered with caution and in low doses, because they are metabolized by the liver. Table shows the lists of hepatotoxic drugs.
4. **Pharmacotherapy**
 - Parenteral vitamin K: For those patients with prolonged PT.
 - Antiemetics: For patients with nausea.
5. **Restrict hepatotoxic drugs:** See Table 6-3.

D. Nursing Diagnoses and Interventions

Sleep pattern disturbance related to agitation secondary to hepatic dysfunction (faulty absorption, metabolism, and storage of nutrients)

Desired outcome: Patient relates the attainment of adequate sleep and rest.

1. Provide rest periods before and after activities and treatments.
2. Keep frequently used objects within easy reach.
3. Promote rest and sleep by decreasing environmental stimuli, providing back massage and/or relaxation tapes, speaking with patient in short, simple terms.

Knowledge deficit: Causes of hepatitis and modes of transmission.

Desired outcome: Patient can verbalize knowledge of the causes of hepatitis and measures that help prevent transmission.

1. Assess patient's knowledge of the disease process and educate as necessary.
2. Make sure patient knows you are not making moral decisions regarding alcohol/drug use and/or sexual behavior.
3. Teach patient and SOs the importance of good handwashing.

4. If appropriate, advise patients with hepatitis A that crowded living conditions with poor sanitation should be avoided to prevent recurrence.
5. Remind patients with hepatitis B and non-A non-B hepatitis that sexual relations should be avoided.
6. Explain that blood donation is no longer possible.
7. Advise patients with hepatitis B that their sexual partners should receive hepatitis B vaccine.
8. Refer patient to alcohol/drug treatment programs.

Alteration in nutrition: Less than body requirements related to decreased intake secondary to anorexia, nausea, and gastric distress.

Desired outcome: Patient does not exhibit signs of malnutrition or weight loss.

1. Take a diet history to determine food preferences.
2. Monitor and record intake.
3. Offer mouth care prior to meals to alleviate unpleasant taste and thereby enhance appetite.
4. Encourage small, frequent feedings and provide emotional support during meals.
5. Give vitamin and mineral supplements, if appropriate.
6. Administer antacids, antiemetics, antidiarrheals, and cathartics as prescribed to minimize gastric distress.
7. Encourage SOs to bring in desirable foods, if permitted.

Impairment of skin integrity related to pruritus secondary to hepatic dysfunction.

Desired outcome: Patient's skin remains intact.

1. Keep skin moist by using tepid water or emollient baths, avoiding soap, and applying emollient lotions at frequent intervals.
2. Encourage patient not to scratch skin and to keep nails short and smooth. Suggest the use of the knuckles if patient must scratch. Wrap or place gloves on the hands of comatose patients.
3. To prevent infection, treat any skin lesion promptly.
4. Encourage patient to wear loose, soft clothing; provide soft linens (cotton is best).
5. Keep the environment cool.
6. Change wet linen often.

Disturbance in self-concept related to alteration in body image secondary to jaundice.

Desired outcomes: Patient verbalizes feelings and concerns. Patient can verbalize knowledge of measures for enhancing appearance and demonstrates an interest in daily grooming.

1. Encourage patient and SOs to verbalize feelings, concerns.
2. Encourage patient to maintain daily grooming.
3. Explain that wearing yellow and green intensifies yellow skin tone. Suggest wearing bright reds and blues or black instead.
4. Provide privacy as necessary.

Potential for injury related to increased risk of bleeding secondary to decreased vitamin-K absorption.

Desired outcomes: Patient does not exhibit signs of bleeding due to handling, invasive procedures, or sharp objects. If bleeding does occur, it is detected promptly, resulting in immediate treatment and absence of injury to the patient.

1. Monitor PT levels daily.
2. Handle patient gently (eg, when turning or transferring).
3. Minimize IM injections. Rotate sites, and use small-gauge needles. Apply moderate pressure after an injection, but do not massage the site.
4. Observe for ecchymotic areas. Inspect the gums and test the urine and feces for the presence of bleeding. Report significant findings.
5. Teach patient to use electric razor and soft-bristled toothbrush.
6. Administer vitamin K as prescribed.

E. Patient–Family Teaching and Discharge Planning

Provide patient and SOs with verbal and written information for the following:

1. Importance of rest and getting adequate nutrition.
2. Hepatotoxic agents, especially OTC drugs.
3. Prescribed medications, eg, multivitamins, including the name, purpose, dosage, schedule, potential side effects, and precautions.
4. Potential complications, including delayed healing, skin injury, and bleeding tendencies.
5. Referral to alcohol/drug treatment programs as appropriate.

3.1.2. Cirrhosis

Cirrhosis is a chronic, serious disease in which there are structural changes in the liver. Although pathologic changes do not occur for many years, structural changes gradually lead to total liver dysfunction. Complications include portal hypertension, ascites, esophageal varices, hemorrhoids, splenomegaly, bleeding tendencies, jaundice, hepatorenal syndrome, and hepatic encephalopathy (hepatic coma).

Types

- **Laennec’s (alcoholic/portal) cirrhosis:** Associated with chronic alcohol abuse
- **Postnecrotic cirrhosis:** Associated with history of viral hepatitis or hepatic damage from industrial chemicals;
- **Biliary cirrhosis:** Associated with post-hepatic biliary obstruction

A. Assessment

Chronic indicators: Lassitude, anorexia, nausea, vomiting (especially early in the morning), dyspepsia, flatulence, change in bowel habits, slight weight loss, discomfort in epigastric area or RUQ.

Acute indicators: Hepatocellular failure resulting in jaundice, peripheral edema, fetor hepaticus (a musty, sweetish odor on the breath), hepatic encephalopathy (can progress from slight changes in personality and behavior to coma); hematologic disorders, including bleeding tendencies, anemia, leukopenia, thrombocytopenia; excess circulating estrogen as evidenced by spider angiomas, testicular atrophy, gynecomastia, pectoral and axillary alopecia, palmar erythema; and portal hypertension complications, including splenomegaly, esophageal and gastric varices, hemorrhoids, and ascites.

History of: Exposure to hepatotoxic agents, viral hepatitis, alcoholism, poor nutrition.

B. Diagnostic Tests

1. Hematologic tests: SGOT (AST) and SGPT will be elevated.
2. Bromsulphalein (BSP) test: Usually elevated.
3. Urine tests: Urine bilirubin will be increased; urobilinogen will be normal or increased.
4. Liver biopsy: Provides a microscopic picture of hepatocytes and aids in confirming a diagnosis. It is contraindicated in patients with clotting abnormalities, in cases of obstructive jaundice, or in the presence of local infection at biopsy site or ascites. and rigid abdomen.
5. Barium swallow: Verifies the presence of esophageal or gastric varices.
6. Radiologic studies: Ultrasound differentiates hemolytic and hepatocellular jaundice from obstructive jaundice and shows hepatomegaly and intrahepatic. Cholangiography reveals the extent of obstruction via contrast dye. perature.
7. Esophagoscopy: A fiberoptic technique used to verify presence of esophageal varices and/or bleeding.

C. **Medical Management and Surgical Interventions**

1. **Identify and treat underlying causes:** For example, exposure to hepatotoxins, use of alcohol, biliary obstruction.
2. **Pharmacotherapy**
 - Diuretics: To reduce edema.
 - Antibiotics: To control intestinal flora.
 - Hematinics: To control anemia.
 - Blood coagulants and vasopressors: To control bleeding.
 - Laxatives and stool softeners: To prevent straining.
 - Antidiarrheals: As necessary to control diarrhea.
 - Antipruritics: For pruritus.
 - Topical anesthetics: For hemorrhoids.
 - Supplemental vitamins and minerals: Such as folic acid for macrocytic anemia and vitamin K for prolonged PT.
3. **Dietary management:** With fluid retention and ascites, sodium and fluids are restricted. Usually, half the calories are supplied as carbohydrates. Protein is restricted in hepatic coma or precoma. Parenteral or enteral nutrition is administered in the presence of bleeding or coma.
4. **Bed rest:** In the presence of fever, infection.
5. **Treatment of complications**
 - Hemorrhage from esophageal varices: Usually, a 4-lumen Minnesota sump tube or 3 lumen Sengstaken-Blakemore tube is used for tamponade, and surgical management includes a portocaval shunt, the shunts divert blood from the portal system to the vena cava.
 - Ascites: Dietary management may include sodium and fluid restrictions.
 - Diuretics, usually aldosterone antagonists, antagonists, are often given to minimize fluid collection.
 - Pharmacologic management includes antibiotics to inhibit intestinal bacteria and magnesium sulfate or enemas to cleanse the intestines after GI bleeding. The following drugs are contraindicated: barbiturates, narcotics, potassium-depleting diuretics, and ammonia-containing medications.

D. Nursing Diagnoses and Interventions

Alteration in nutrition: Less than body requirements related to decreased intake secondary to anorexia and nausea.

Desired outcomes: Patient does not exhibit signs of malnutrition or weight loss. Patient can verbalize knowledge of foods that are permitted and restricted.

1. Explain dietary restrictions to the patient.
2. Sodium and/or fluids are restricted, if the ammonia level rises, protein also will be restricted.
3. Encourage small, frequent meals to ensure adequate nutrition.
4. Encourage SOs to bring in desirable foods as permitted.
5. Administer vitamin and mineral supplements, as prescribed.
6. Administer the following medications, as prescribed, to decrease gastric distress
antacids, antiemetics, antidiarrheals and cathartics.
7. Monitor I&O; weigh patient daily.
8. Promote bed rest to reduce metabolic demands on the liver.
9. Provide soft diet if patient has esophageal varices,

Impaired gas exchange related to decreased lung expansion secondary to pressure on the diaphragm caused by ascites.

Desired outcome: Patient's ARTERIAL BLOOD GASES values and physical findings are within acceptable range.

1. During complaints of dyspnea or orthopnea, assist patient into semi- or high Fowler's position to enhance gas exchange.
2. Administer oxygen as prescribed.
3. Monitor ARTERIAL BLOOD GASES values; notify MD of significant findings.
4. Encourage patient to change positions and deep-breathe at frequent intervals to enhance gas exchange. If secretions are present, ensure that the patient coughs frequently.
5. Observe for signs of infection such as spiking temperatures, chills, diaphoresis, and adventitious breath sounds.
6. Obtain baseline abdominal girth measurement, and then measure girth either daily or q shift. Measure around the same circumferential area each time; mark the site with indelible ink.

Potential for injury related to increased risk of bleeding secondary to altered clotting factors and portal hypertension.

Desired outcomes: 1. Patient does not exhibit signs of bleeding due to irritating foods, actions that increase intra-abdominothoracic pressure, or defecation. 2. Patient can verbalize knowledge of actions or foods that can cause bleeding.

1. Instruct patient to avoid swallowing foods that are chemically or mechanically irritating (eg, rough or spicy foods, hot foods, hot liquids).
2. Advise patient to avoid actions that increase intra-abdominothoracic pressure, such as coughing, sneezing, lifting, or vomiting.

3. Administer stool softeners, as prescribed, to prevent patient from straining with defecation.
4. Inspect stools for presence of occult blood; perform a guaiac test as indicated.
5. Instruct patient about alcohol's role in causing esophageal varices.

Potential for injury related to risk of hepatic coma secondary to cerebral accumulation of ammonia and/or GI bleeding.

Desired outcome: Potentially precipitating causes of hepatic coma, if they occur, are detected and reported promptly, resulting in immediate treatment and absence of injury to the patient.

1. Perform a baseline assessment of patient's personality characteristics. Enlist the aid of SOs to help determine slight changes in personality or behaviour.
2. Have patient demonstrate his or her signature daily. If the writing deteriorates, ammonia levels may be increasing. Be alert to generalized muscle twitching and asterixis Report significant findings
3. Remind patient to avoid protein and foods high in ammonia, such as gelatin, onions, and strong cheeses. The diseased liver is unable to convert ammonia to urea
4. Observe for signs of GI bleeding, including melena or hematemesis. GI bleeding can precipitate hepatic coma. Report bleeding
5. Protect patient against injury that can be precipitated by confused state.

Fluid volume excess: Edema and ascites related to retention secondary to portal hypertension and hepatocellular failure.

Desired outcomes: Patient can verbalize knowledge of food and nonfood items that increase swelling and exhibits physical findings and laboratory values within acceptable range.

1. Obtain baseline abdominal girth measurement. Mark abdomen with indelible ink to ensure serial measurements from the same circumferential site. Measure girth daily or q shift as appropriate.
2. Monitor I&O; weigh patient daily.
3. Give frequent mouth care
4. Monitor electrolyte values and report abnormalities
5. Remind patient to avoid food and nonfood items that contain sodium (eg, antacids, baking soda, and some mouthwashes).

Knowledge deficit: Factors that precipitate or aggravate cirrhosis.

Desired outcome: Patient can verbalize knowledge of factors that can aggravate or precipitate cirrhosis.

1. Determine patient's pattern of alcohol use. Inform patient that a major survival factor is cessation of alcohol.
2. refer patient to an alcohol treatment program
3. Assist patient with identifying hepatotoxins other than alcohol
4. Encourage patient to adhere to medical regimen, including dietary restrictions and pharmacologic management.

E. Patient–Family Teaching and Discharge Planning

Provide patient and SOs with verbal and written information for the following:

1. Medications, including drug name, purpose, dosage, schedule, precautions, and potential side effects.
2. Dietary restrictions.
3. Potential need for life style changes, including cessation of alcohol.
4. Awareness of hepatotoxic agents..

3.1.3 Cholelithiasis and Cholecystitis

Cholelithiasis is a condition characterized by the presence of stones in the gallbladder and/or the biliary ducts. Gallstones can be composed of cholesterol, calcium bilirubinate, or calcium carbonate. Precipitating factors include disturbances in metabolism, biliary stasis, obstruction, and infection. Gallstones are especially prevalent in women who are multiparous, on estrogen therapy, or who use oral contraceptives. Other risk factors include obesity, dietary intake of fats, sedentary lifestyle, and familial tendencies. The incidence increases with age, and it is estimated that one out of every three persons who reach age 75 has gallstones. Cholelithiasis is frequently seen in disease states such as diabetes mellitus, regional enteritis, inflammatory disease of the terminal ileum, and certain blood dyscrasias. Usually, cholelithiasis is asymptomatic until a stone becomes lodged in the biliary tract. If the obstruction is unrelieved, biliary colic and cholecystitis can ensue.

Cholecystitis is most commonly associated with bile duct obstructions. Acute cholecystitis is typically caused by a gallstone that obstructs the cystic duct. With obstruction, structural changes can occur such as hypertrophy of the gallbladder and a swelling and thickening of the gallbladder walls. If the edema is prolonged, the walls become scarred and fibrosed and the constant presence of bile can lead to mucosal irritation. As a complication of the impaired circulation and edema, pressure ischemia and necrosis can develop, resulting in gangrene or perforation. With chronic cholecystitis, stones almost always are present, and the gallbladder walls are thickened and fibrosed.

A. Assessment

Cholelithiasis: History of intolerance to fats and occasional discomfort after eating. As the stone moves through the duct or becomes lodged, a sudden onset of mild, aching pain will occur in the midepigastrium after eating and increase in intensity during a colic attack, potentially radiating to the RUQ and right subscapular region. Nausea, vomiting, tachycardia, and diaphoresis also can occur. This condition may also be asymptomatic.

Cholecystitis: History of intolerance to fats and discomfort after eating, including regurgitation, flatulence, belching, epigastric heaviness, indigestion, heartburn, chronic upper abdominal pain, and nausea. Amber-colored urine, clay-colored stools, pruritus, jaundice, steatorrhea, and bleeding tendencies can be present if there is bile obstruction. Symptoms may be vague.

Physical exam

- Cholelithiasis: Palpation of RUQ will reveal a rigid abdomen during colic attack, with flaccidity between pains.
- Cholecystitis: Palpation will elicit tenderness localized behind the inferior margin of the liver. With progressive symptoms, a tender, globular mass might be palpated behind the lower border of the liver.

B. Diagnostic Tests

1. **Radiologic studies:** Oral cholangiogram, IV cholangiogram, and percutaneous trans-hepatic cholangiogram assess the patency of the gallbladder and biliary ducts and help to rule out other conditions that mimic cholelithiasis or cholecystitis. Chest, abdominal, upper GI, and barium enema x-rays are often used to rule out pulmonary or other GI disorders.

2. EKG: To rule out cardiac disease.
3. Ultrasonography of the gallbladder and biliary tract: To detect gallstones and tumors and help distinguish between intrahepatic and extrahepatic jaundice.
4. CT scan: To detect dilated bile ducts and the presence of gallbladder cysts.
5. Endoscopic retrograde cholangiopancreatography: To visualize and evaluate the biliary tree.
6. CBC: To assess for presence of infection and/or blood loss.
7. PT: To assess for a prolonged clotting time secondary to faulty vitamin K absorption.
8. Bilirubin tests (serum and urine) and urobilinogen tests (urine and fecal): To differentiate between hemolytic disorders, hepatocellular disease, and obstructive disease. Usually there is an increase of bilirubin in the plasma and urine with biliary disease.
9. Serum liver enzyme test: Will show a small elevation in SGOT, SGTP, and LDH. With biliary obstruction, the alkaline phosphatase and 5 nucleotidase will have values ten times normal.

C. Medical Management and Surgical Interventions

1. Pharmacologic therapy:

- Analgesics and antacids: For pain.
 - Anticholinergics: To prevent smooth muscle contraction.
 - Antispasmodics: To relieve spasms.
 - Antibiotics: For infection.
 - Antiemetics: For nausea and vomiting.
 - Bile salts: To facilitates absorption of fats and fat-soluble vitamins.
 - Fat-soluble vitamins such as A, D, E, and K: Replacement is necessary because of faulty vitamin absorption in the small intestine.
 - Hyperlipidemic agents: Bind with bile salts in the intestine to facilitate excretion, and may be given to provide relief from pruritus.
2. **Chemical dissolution of cholesterol gallstones with a solvent**: An oral preparation of bile salts, eg, chenodeoxycholic acid, may be administered to dissolve cholesterol stones. The solvent is infused via a T-tube.
 3. **Dietary management**: Varies according to the patient's condition. During an acute attack, NPO with IV fluids may be instituted. With severe nausea and vomiting, an NG tube is inserted and attached to low, intermittent suction. Diet of small, frequent feedings of a low-fat diet are recommended for both the acute and chronic conditions.
 4. **Non-operative biliary stone removal**: This involves the extraction of stone, which is performed under fluoroscopy in the radiology department.
 5. **Surgical interventions**: Usually required for relief of long-term symptoms of cholelithiasis and acute cholecystitis. The type of surgery depends on the severity and length of illness and site of obstruction. The following procedures may be performed:
 - Cholecystostomy: Opening and draining the gallbladder of gallstones.
 - Choledochotomy: Opening the common bile duct to remove stones.
 - Choledochoduodenostomy: Anastomosis of the common bile duct to the duodenum.
 - Choledochojejunostomy: Anastomosis of the common bile duct to the jejunum.
 6. **Cholecystectomy (removal of the gallbladder)**: Is the most commonly performed procedure for biliary disease, which accounts for one-third of all surgical procedures that are performed. A right subcostal incision is made, allowing exploration of the common duct. The stones are removed and a T-tube is often inserted to maintain patency of the common duct and drain bile, and a drain (usually Penrose) is inserted and brought out through a stab wound for drainage of blood, serum, and bile.

4.0 Conclusion

An obstruction the biliary ducts leads to a blockage of the flow of bile. When this occurs, a variety of clinical manifestations surface, including obstructive jaundice, dark-amber urine, and clay-colored stools. Pruritus occurs because of the deposition of bile salts in skin tissue. Steatorrhea and bleeding tendencies result from the inability of the duodenum to absorb fats and fat-soluble vitamins A, D, E, and K.

5.0 Summary

Viral hepatitis is caused by one of the hepatitis viruses: A, B, non-A or non-B,

Cholelithiasis is a condition characterized by the presence of stones in the gallbladder and/or the biliary ducts.

Gallstones are composed of cholesterol, calcium bilirubinate, or calcium carbonate.

6.0 Tutor Marked Assignment

- List common findings manifested by clients with cirrhosis.
- Discuss the nursing management of clients undergoing medical or surgical treatment of a gallbladder disorder.
- Compare the types of Viral Hepatitis

7.0 Further Reading and Other Resources

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UNIT III: CARE OF CLIENTS WITH MUSCULO-SKELETAL DISORDERS

1.0	Introduction
2.0	Objective
3.0	Main Content -----
3.1	Inflammatory Bone Disorders
3.2	Dislocation/Subluxation
3.3	Meniscal Injuries
3.4	Skeletal Disorders
3.4.1	Osteomyelitis
3.4.2	Fractures
3.4.3	Benign Neoplasms
3.4.4	Malignant Neoplasms
3.4.5	Osteoporosis
3.5.2	Amputation
3.5.3	Bone Grafting
4.0	Conclusion
5.0	Summary
6.0	Tutor Marked Assignment
7.0	Further Reading and Other Resources

1.0 Introduction

This unit deals with the disorders of the musculo-skeletal systems with special emphasis on fractures.

2.0 Objective

On completion of this unit you will be able to:

1. Differentiate strains, contusions, and sprains.
2. Describe the signs and symptoms of a fracture and the common treatments.
3. State the pathophysiology of gout, bursitis, and ankylosing spondylitis.
4. Identify the causes of osteomyelitis.
7. Discuss the nursing management for a client with a sprain, dislocation, or cast; who is in traction or undergoing orthopedic surgery; or with an amputation.

3.1 Inflammatory Bone Disorders

Arthritis is inflammation of a joint. There are many forms, the common ones are osteoarthritis, gouty arthritis, rheumatoid arthritis, and are therefore discussed in this unit.

3.1.1 Osteoarthritis

Osteoarthritis, also known as degenerative joint disease (DJD), is an extremely prevalent disorder. It is a chronic, progressive disease characterized by increasing pain, deformity, and loss of function. It can be found in any age group, usually following trauma or as a complication of congenital malformation. True joint inflammation is seldom present (except in the distal interphalangeal joints). Hereditary and mechanical factors are suspected to be the primary causes of this process.

Primary osteoarthritis is idiopathic and occurs in the distal interphalangeal joints (DIP), proximal interphalangeal joints (PIP), the carpometacarpal joints (CMC), hip, knee, first metatarsophalangeal joint (MTP), and the cervical and lumbosacral spine. Secondary arthritis can occur in any joint and usually follows some form of intra-articular injury or extra-articular cause that affects joint dynamics. Examples include joint fractures and chronic insults such as poor posture, obesity, occupational abuse, or a metabolic disease that affects the joint (ochronosis, osteitis deformans, or hyperparathyroidism).

A. Assessment

Involvement can range from incidental findings on x-ray to pervasive disease that affects the patient's independence in the performance of ADLs.

Clinical manifestations: Onset is insidious, beginning with joint stiffness. It evolves into joint pain, which worsens with activity and is relieved with resting the joint. Signs of local inflammation are usually absent, except occasionally in the DIP, PIP, and CMC joints. Usually, there are no specific systemic signs or symptoms.

Physical exam: Characteristic findings include Heberden's nodes (enlargement of the DIP joint), Bouchard's nodes (enlargement of the PIP joint), varus or valgus deformity of the knee, bony enlargement of the joint, and flexion contracture of the knee. Frequently, crepitation is found.

B. Diagnostic Tests

There are no characteristic laboratory studies associated with this disorder.

X-ray studies: May reveal narrowing of the joint space, osteophytosis (bony projections) of the joint margins, bone cysts, bony erosions, and dense subchondral bone.

C. Medical Management and Surgical Interventions

1. **Rest:** This is the principal therapy for preventing progression. The patient is advised to avoid activities that will further stress the joint. Use of ambulatory assistive devices, splints, or orthotics may be prescribed to allow rest or decrease stress on affected joints, and the patient is instructed in methods that prevent postural strain. Regular rest periods of 30 – 60 minutes are often advised for patients prone to overworking.
2. **Weight reduction:** For patients for whom excessive weight contributes to the pathology.
3. **Local moist heat:** To decrease stiffness and provide some subjective pain relief. Hydrotherapy with warm water is especially useful in aiding range of motion (ROM) exercises. Patients who cannot afford to purchase a device to supply moist heat may be required to use a traditional heating pad.
4. **ROM and muscle strengthening exercises:** May be useful in selected cases to increase joint function and supplement joint strength. Exercises may include passive ROM, active ROM, active-assisted ROM, and isometric and isotonic exercises.
5. **Intraarticular steroids:** Used to provide transient relief of symptoms.
6. **Pharmacotherapy:** Includes the use of analgesics and anti-inflammatory agents. Analgesics may be necessary to combat the pains associated with DJD and the nonsteroidal anti-inflammatory agents(Aspirine, acetaminophen).
7. **Surgical interventions:** Various orthopedic surgeries may be utilized to correct underlying congenital anomalies or defects created by trauma. Arthroplastic surgery allows damaged joints surface to be augmented, repaired, or replaced, while particular tissues may be repaired to improve joint strength. Joint replacement has been used to replace most joints, but the greatest success has been found with the hip and knee.
8. **Splints and orthotic devices:** May be used to supplement joint strength or protect the joint from excessive strain.
9. **Assistive devices:** Great varieties have been developed to help patients perform ADLs independently, even in cases of significant joint function loss. Examples include stocking helpers, built-up eating utensils, pickup sticks, and raised toilet seats.

3.1.2 Gouty Arthritis

Gout is an inherited metabolic disorder, crystal-induced synovitis.

Causes

- Excess production or underexcretion of urates
- Conditions in which uric acid is retained or excessively produced, such as lead poisoning, use of thiazide diuretics, chronic renal disease, myeloproliferative disease, hemoglobinopathies, cancer chemotherapy, and multiple myeloma.

Pathophysiology

The pathophysiology involves the formation of tophus (nodular deposition of monosodium urate monohydrate crystals), which causes a pronounced inflammatory response. Tophi may be found in synovial tissues, cartilage, periarticular tissues, tendon, bone and the kidneys. Although the relationship between high serum levels of uric acid (hyperuricemia) and gouty arthritis is unclear, there appears to be some relationship between rapid fluctuations in the level of serum uric acid and an acute gouty attack. Uric acid renal calculi, nephrosclerosis, and gouty nephritis can accompany this process.

A. Assessment

Chronic indicators: Joint changes such as severe joint deformity and loss of function. Hypertension, renal calculi, and renal failure also may be seen.

Acute indicators: Sudden onset, acute inflammation, and an excruciatingly painful joint that presents with erythema, joint effusion, restricted motion, warmth, and tenderness. Systemic indicators include tachycardia, anorexia, fever, headache, and malaise. Although it is usually monoarticular, polyarticular attacks have been noted with this disorder.

Physical examination: Tophi may be noted as subcutaneous nodules on the hands, feet, olecranon bursa, prepatellar bursa, and ears. The most commonly affected joint is the metatarsophalangeal joint of the great toe, but the feet, ankles, and knees are also commonly affected.

History of: Recent surgery, oral intake high in purines, alcoholic excess, infection, and use of diuretics.

B. Diagnostic Tests

1. **Serum tests:** Uric acid is frequently elevated above the normal 7.5 mg/dL there also will be leukocytosis and an elevated sedimentation rate.
2. **Joint fluid aspiration:** Allows compensated polariscopic examination of wet smears to reveal presence of sodium urate crystals.
3. **Tophus aspiration:** Provides identification of typical sodium urate crystals.
4. **X-ray:** Will demonstrate no change early in the disease, but with chronic disease there will be radiolucent urate tophi (which look like punched-out areas on the x-ray) adjacent to soft tissue tophi.

C. Medical Management and Surgical Intervention

1. Pharmacotherapy

- Colchicine is the drug of choice during the acute phase. It is believed that it mediates the inflammatory response caused by the urate crystals. Usual dosage is 0.5 mg qh or 1 mg q2h until either the pain is controlled or side effects appear. Side effects include nausea, vomiting, abdominal cramping, and diarrhea. Colchicine is contraindicated in patients with inflammatory bowel disorders, significant hepatic disease, or renal disease.
 - Corticosteroids: May control acute attacks but must be combined with colchicines because discontinuation of the steroid often results in a relapse.
 - Nonsteroidal anti-inflammatory agents: May be used for chronic or acute forms of the disease.
2. **Analgesics such as acetaminophen with codeine or oxycodone preparations: To control the pain of gout.**
 3. **Joint rest:** Mandatory and should include complete bed rest with elevation of the inflamed joint. Sometimes, topical cooling (ice applications to the joint) is prescribed to aid in reducing inflammation.
 4. **Management between attacks:** May be accomplished with the following:
 - Colchicine: Prophylactic or interim use.
 - Uricosuric agents such as probenecid or sulfinpyrazone: The dosage is determined by the patient's serum uric acid levels.
 5. Allopurinol, a xanthine oxidase inhibitor lowers serum uric acid, decreases the concentration of uric acid in the urine, and mobilizes the uric acid crystals to tophi. Dosage varies, depending on serum uric levels.
 6. **Diet therapy:** alcoholic beverage intake is frequently curtailed because of its connection with the precipitation of acute attacks.

7. **Surgical intervention:** Gouty tophi are excised when they erode through the skin or cause mechanical impairment. Chronic joint involvement may require surgical interventions

D. Nursing Interventions

- Elevate the inflamed joint with pillows, above the level of the heart. Explain the rationale to the patient.
- Perform passive ROM of the joints bid (or have the patient perform assisted ROM). Explain the rationale to the patient.
- Instruct patient to increase joint ROM as the inflammation subsides, following the process described under the nursing diagnosis.
- Assess patient's knowledge of the disease process, medication regimen, and potential side effects of the drugs. As appropriate, teach the pathophysiology of the disease. In addition, when secondary gout is suspected, inform patient about the primary disease causing the gout.
- Provide thorough instructions for the medication therapy, including rationale, dosage, schedule, precautions, and potential side effects.
- Caution patients taking narcotic analgesics of the potential of altered sensorium, and advise them to avoid using machinery, driving, or performing other activities requiring alertness.
- Intake of purine-containing foods may be restricted in severe cases. High-purine-content foods include broth, gravy, organ meats, mackerel, yeast, poultry, meats, fish, shellfish, beans, mushrooms, peas, and spinach. Ensure that the patient receives consultation, with verbal instruction in the dietary regimen as well as written instructions to take home.
- Inform patient that excessive use of alcohol can precipitate gout attacks.

3.1.3 Rheumatoid Arthritis

Rheumatoid arthritis (RA) is a systemic disease characterized by remissions and exacerbations of inflammation of the connective tissue throughout the body. RA most commonly affects the synovial joints, but the effects of this disease are highly variable. The disease onset and progression can be rapid and fulminating or slow and chronic.

Causes

The etiology is unknown, predisposing factors include autoimmune process, food allergies, hereditary and viral infections.

The inflammatory process results in chronic synovitis with the formation of pannus, inflammatory exudates that accumulates over the surface of the synovial membrane, eventually eroding cartilage, bone, ligaments, and tendons. Involvement of connective periarticular tissues results in loss of support structures and leads to characteristic joint changes, which further contribute to the pathology.

A. Assessment

Acute indicators: Morning stiffness lasting >30 minutes, symmetrical joint involvement, joint effusion, periarticular edema, pain, local warmth, and erythema. Joint stiffness is usually worsened by stress placed on the joint, and it can follow periods of inactivity as well. Prodromal clinical manifestations may include malaise, weight loss, vague periarticular pain, low-grade fever, and vasomotor disturbances resulting in paresthesia and Raynaud's phenomenon. Sometimes, an acute exacerbation is related to stress such as infection, surgery, trauma, or emotional strain.

Chronic indicators: Progressive thickening of the periarticular tissues, subluxation, fibrous ankylosis, atrophy of skin and muscle, severe limitation of ROM with progressive loss of

function, joint and muscle contractures, dryness of the eyes and mucous membranes, and subcutaneous nodules. Some patients develop splenomegaly and enlarged lymph nodes.

B. Diagnostic Tests

1. **Serologic and other blood studies:** These are performed to detect certain macroglobulins, which make up the rheumatoid factor.
WBC count: Usually normal or slightly elevated, but leukopenia can be present, especially in the presence of splenomegaly.
2. **Fluid aspiration from the involved joint:** May reveal synovial fluid greater in volume than normal, opaque and cloudy yellow in appearance, glucose level lower than serum level, and elevated WBCs and leukocytes in the presence of RA.
3. **X-ray studies of the involved joints:** will illustrate soft tissue swelling, erosion of joint surfaces normally covered by articular cartilage, and osteoporosis of adjacent bone.

C. Medical Management and Surgical Interventions

1. **Bed rest:** This is essential throughout all phases of this disease until significant clinical joint findings have decreased for 2 weeks. During this period, proper joint positioning is essential to prevent contractures. Concurrent physical therapy is prescribed to put joints through passive ROM at least once a day. During remissions, the patient should receive 8 hours of sleep each night and 1–2 hours rest at mid-day. Any increase in symptomatology necessitates increasing the amount of rest.
2. **Emotional support:** To lessen stress and help patients deal with fear, disability, and the many losses they will incur.
3. **Rest of inflamed joints:** Imperative. Unstable joints should be splinted or braced to provide support and put through passive ROM at least daily while inflamed.
4. **Joint exercise:** Essential to maintain joint function and muscle strength, with the amount increasing as inflammation decreases. A graded exercise program should include the following: passive ROM, active-assisted ROM, active ROM, and resistive ROM with gradually increasing levels of resistance. Isometric exercise is used to maintain muscles strength during active joint inflammation. Any signs of increasing joint inflammation are causes for regression to a less stressful exercise until joint inflammation has again decreased. Inflamed weight-bearing joints should be protected from stress by orthotics and ambulatory adjuncts such as cane, crutches, and walker, or a wheel chair.
5. **Thermotherapy:** To relax muscles and reduce pain. Cold therapy is used during inflammatory stages to reduce pain. Moist heat (especially warm tub baths) is useful to exercise stiffened joints, using the heat and buoyancy to aid motion.
6. **Anti-inflammatory agents: More potent anti-inflammatory agents,** also used in the treatment of RA, include the following:
 - Antimalarials (chloroquine phosphate, 250 mg/day; or hydroxychloroquine sulfate, 200 mg/day):
 - Chrysotherapy (use of medicinal gold salts): Useful but the mechanism of action is not clearly understood. Contraindications include hepatotoxicity, or hematologic pathology.
 - Corticosteroids: Control the symptoms of RA, but they do not substantially alter the progression of the disease.
 - Penicillamine: Used only after all other methods have been ineffective in controlling symptoms. Fifty percent of patients taking this drug will develop side effects, including thrombocytopenia, leukopenia, aplastic anemia, nephritic syndrome, and immune complex disease (myasthenia gravis). This medication is taken between meals to aid absorption.

7. Surgical interventions

- Synovectomy: To remove the inflamed synovium and prevent pannus formation. This may be performed either by surgical excision or instillation of a radioactive solution to “burn” the synovium.
- Osteotomy: To correct disruptive force vectors placed on the joint surfaces or to correct bony malalignments.
- Carpal tunnel release, tarsal tunnel release, ganglionectomy, tendon repair, and removal of Baker’s cyst: Examples of surgeries performed to correct concurrent connective tissue defects associated with RA.

D. Nursing Diagnoses and Interventions

1. Rest of inflamed joints, exercise, and thermotherapy are the corner stone of treatment.
2. Teach patient to use and care for splints and orthotics

Table 1 Nonsteroidal Anti-Inflammatory Agents

Generic Name	Common Trade Names	Usual Daily Dosage (mg)
acetylsalicylic acid	Aspirin	650–1300 q4h
choline salicylate	Arthropan	650 q4–6h
choline magnesium trisalicylate	Trilisate	1000–1250 bid
fenopfen calcium	Nalfon	300–600 qid
Ibuprofen	Motrin, Advil, Nuprin	200–400 qid
Indomethacin	Indocin	20–50 tid
magnesium salicylate	Mobidin	600–1200 tid or qid
meclofenamate sodium	Meclomen	50 tid or qid
mefenamic acid	Ponstel	250 qid
Naproxen	Naprosyn	250–375 qid
naproxen sodium	Anaprox	275 q6–8h
Oxyphenbutazone	Tandearil	100 tid
Piroxicam	Feldene	20 qd
sulindac	Clinoril	150–200 bid
tolmetin sodium	Tolectin	200–400 mg tid

3.2 Dislocation/Subluxation

A dislocation occurs when the joint surfaces are completely out of contact. A subluxation is an incomplete dislocation in that some of the joint surfaces remain in contact. Most dislocations and subluxations are the result of trauma and can involve significant periarticular damage, including fractures.

3.3 Meniscal Injuries

Meniscal injuries involve the intra-articular fibrocartilages on the medial or lateral side of the knee’s tibial plateau. These halfmoon-shaped cartilages facilitate joint motion while also absorbing some of the stress placed on the joint. There are a variety of cartilage injuries that can occur, and all involve a tear to varying degrees. Most commonly, a meniscal injury is the result of trauma to the knee or, less frequently, degeneration of the joint secondary to arthritis. Medial meniscus injuries are the most common and usually follow a knee movement involving internal rotation. Injuries to the lateral meniscus are more commonly associated with external rotation that occurs while the knee is partially flexed.

3.4 Skeletal Disorders

3.4.1 Osteomyelitis

Osteomyelitis is an acute or chronic infection involving a bone. Although osteomyelitis often remains localized, it can spread through the marrow, cortex, and periosteum.

Causes

- Recent bone trauma
- Bone with low oxygen tension such as found in sickle cell anemia.
- Acute hematogenic osteomyelitis is most frequently caused by Staphylococcus aureus (90–95%), Escherichia coli, Pseudomonas species, Klebsiella, Enterobacter, Proteus, Streptococcus (group A), and Hemophilus influenzae.

Types

Primary osteomyelitis

Is a direct implantation of micro-organisms into bone via compound fractures or penetrating wounds, or from surgery.

Secondary or acute hematogenic osteomyelitis

Is an infection of bone that occurs through its own blood supply or from infection from contiguous soft tissues or joints involved with septic arthritis.

A. Assessment

Acute osteomyelitis: Abrupt onset of pain in the involved area, fever, malaise, and limited motion. Pseudoparalysis is especially indicative of osteomyelitis in children who refuse to move an adjacent joint because of pain.

Chronic osteomyelitis: Edema and erythema over the involved bone, weakness, irritability, and generalized signs of sepsis can occur. Sometimes the only symptom is persistent purulent drainage from an old pocket or sinus tract.

History of: Total joint replacement, compound fracture, use of external fixator, vascular insufficiency (eg, with diabetes mellitus), recurrent UTIs.

B. Diagnostic Tests

1. CBC: Will reveal leukocytosis and anemia in the presence of osteomyelitis.
2. ESR: Elevated in the presence of osteomyelitis.
3. Blood or sequestrum cultures: To identify the causative organism. Sequestrum is a piece of necrotic bone that is separated from surrounding bone as a result of osteomyelitis.
4. X-rays: May reveal subtle areas of radiolucency (osteonecrosis) and new bone formation. No x-ray changes will be evident until the disease has been active at least 5 days in infants, 8–10 days in children, and 2–3 weeks in adults.
5. Radioisotope scanning: May reveal areas of increased vascularity (called “hot spots”), which are indicative of osteomyelitis.

C. Medical Management and Surgical Interventions

1. **Anti-microbial therapy:** This is continued is for at least 6 weeks.
2. **Bed rest.**

3. **Immobilization of affected extremity:** With splint, cast, or traction to relieve pain and decrease the potential for pathologic fracture.
4. **Blood transfusions:** To correct any accompanying anemia.
5. **Removal of internal fixation device or endoprosthesis, if present:** To help control the infection.
6. **Surgical decompression of infected bone:** to stop further spread of infection and promote healing
7. **Drains:** May be inserted into the affected bone to drain the site or act as ingress-egress tubes to funnel topical antibiotics directly into the area of infection.
8. **Topical antibiotics:** May be used via continuous or intermittent infusion and are continued until three successive drain cultures have been negative.
9. **Long-term IV antibiotic therapy:** May be continued for 3–6 months.
10. **Amputation:** Although rarely performed, it may be required for extremities in which persistent infection severely limits function.

3.4.2 Fractures

A fracture is a break in the continuity of a bone. It occurs when stress is placed on the bone that exceeds the bone's biologic loading capacity. Most commonly, the stress is the result of trauma. Pathologic fractures are the result of decreased biologic loading capacity, so that even normal stress can result in a break.

A. Assessment

Acute indicators: Patient complains of sudden pain, which is usually associated with trauma or physical stress such as jogging or strenuous exercise. In pathologic fractures, the patient may describe clinical manifestations associated with the underlying pathology. Nonunion is demonstrated by nonalignment and lost function secondary to lost bony rigidity.

Physical examination: Loss of normal bony or limb contours, edema, ecchymosis, limb shortening, decreased ROM of adjacent joints, false motion (movement that occurs outside of a joint), and crepitus. Complicated and complex fractures can present with clinical manifestations of perforated viscus, neurovascular deficit, joint effusion, or excessive joint laxity.

B. Diagnostic Tests

Bone scans, tomograms, CT scans, stereoscopic films, and arthrograms also can be used.

C. Medical Management and Surgical Interventions

The choice of treatment varies with the complexity of the fracture, the patient's age and concurrent health problems, and functional goals. The goal of treatment is to provide immobilization of the bone until healing occurs. The length of time for immobilization varies with the type of fracture. The following is a brief overview of common examples of treatment interventions that may be utilized.

1. **Bed rest:** May be all that is required to maintain reduction for simple, uncomplicated fractures.
2. **Traction**
 - Cervical fractures: Skeletal traction via Turner, Corn, Vinke, or Crutchfield tongs, which are inserted into the outer plate of the cranial vault. Cervical collars can be used to provide skin traction for simple fractures.
 - Humeral fractures: Dunlop's side arm or overhead 90/90 traction.
 - Pelvic fractures: Pelvic sling or pelvic belt may be used for nondisplaced fractures, while skeletal traction with pins in the ilium and/or femur may be required for displaced fractures.

- Femoral fractures: Skin traction (Buck's extension, Russell's traction, or balanced suspension traction) may be applied until skeletal traction can be set or the fracture is internally fixated. Skeletal traction may involve a Steinmann pin or Kirshner wire positioned through the distal femur or proximal tibia. When skeletal traction is used, it is provided in combination with balanced suspension or Russell's traction and is used for 1–4 months.
 - Tibial fractures: Temporary traction can be accomplished with Buck's extension or, for longer periods of time, with a pin placed through the distal tibia or calcaneus, augmented with balanced suspension or Russell's traction.
3. **Immobilization devices**
 - Soft or hard cervical collars or
 - Plaster of Paris body cast.
 - Velpau cast, shoulder
 - Spica cast
 4. **Closed reduction:** Allows for manipulation of displaced fragments to their normal anatomic alignment. It can be done under general, regional, local, or hematoma-block anesthesia.
 5. **Open reduction with internal fixation (ORIF):** Indicated for fractures that are grossly unstable or for patients who cannot tolerate prolonged bed rest or traction. Internal fixation may be accomplished with screws, pins, wires, plates, bone grafts, methylmethacrylate, or rods.
 6. **External fixation:** Consists of skeletal pins that penetrate the fracture Fragments and are attached to universal joints, which in turn are attached to rods to provide stabilization. These rods form a frame around the fractured limb for immobilization. The external fixator is left in place until sufficient soft tissue repair or bony callus formation allows either application of a cast or complete removal of any form of immobilization. Sometimes the skeletal pins are left in place (after removing the external fixation rods) and incorporated into a cast that immobilizes the limb until the fracture has healed. The external fixator can be used to treat massive open comminuted fractures with extensive soft tissue injury and/or neurovascular injury in which there is increased risk of infection. It is also the treatment of choice for infected non-union, segmental bone loss, limb-lengthening procedures, arthrodesis (joint fusion), and multiple trauma with injuries involving other body systems.
 7. **Coaptation splint:** Provides immobilization for patients with significant nerve damage or edema while compensating for the edema to prevent iatrogenic compartment syndrome
 8. **Progressive ROM and muscle-strengthening exercises:** Begun after the designated period of immobilization to help the patient regain joint function.
 9. **Continuous passive motion (CPM):** A motor-driven device developed to place a joint through repeated extension and flexion. It is used as an adjunctive treatment for such injuries as femoral condyle and tibial plateau fractures.

Specific nursing diagnoses for patients with casts, traction, open reduction and internal fixation, and external fixators:

- **Self-care deficit:** Inability to perform ADLs related to physical limitations secondary to cast and/or surgical procedure.
- **Potential impairment of physical mobility** related to inactivity secondary to prolonged bed rest
- **Alteration in comfort**

- **Potential impairment of skin integrity** related to irritation secondary to the presence of a cast (applies to patients with casts and ORIF)
- **Potential alteration in tissue perfusion:** Peripheral, cerebral, or cardiopulmonary, related to impaired circulation secondary to fat embolization (applies to patients with multiple trauma, multiple fractures, or surgical repair of fractures)
- **Knowledge deficit:** Potential for infection related to orthopedic procedure and/or presence of internal or external device (appropriate for patients with ORIF, external fixators)

E. Patient–Family Teaching and Discharge Planning

Provide patient and SOs with verbal and written information for the following:

1. Medications, including the name, dosage, purpose, schedule, precautions, and potential side effects.
2. Importance of rest, elevation, and use of thermotherapy.
3. Ways in which patient can control discomfort.
4. Use of assistive devices and/or ambulatory aids. Ensure that the patient can perform a return demonstration.

3.4.3 Benign Neoplasms

The three most common benign bone tumors are osteochondromas, enchondromas, and giant cell tumors. Osteochondromas are the most common, representing 45% of all benign tumors. Usually they are found in the metaphysis (wider portion of the shaft) of the long bones, typically the distal femur or proximal humerus, although they also can occur in a rib or vertebra. Individuals under the age of 20 are most commonly affected. Some osteochondromas are the result of an inheritable autosomal dominant trait that causes concurrent growth retardation and bowing of the long bones. *Enchondromas* (chondromas) are most commonly found in the hand (metacarpals or phalanges) or the proximal humerus. They represent approximately 10% of all diagnosed benign tumors. Although they occur most commonly in thirties, they may be seen at any time. *Giant cell tumors* are found most often around the proximal humerus, distal radius, or the knee in the area of the fused epiphysical growth plate. Ten percent of these tumors degenerate into malignancy with a potential for metastasis. Tumors recur 40% of the time.

3.4.4 Malignant Neoplasms

The most common malignant tumors affecting bones are osteogenic sarcoma, primary chondrosarcoma, and myeloma. Osteogenic sarcoma is the most common of the three, occurring most frequently in adolescents. It is found in the distal femoral metaphysis, proximal tibial metaphysis, proximal humeral metaphysis, pelvis, and proximal femur. This tumor is associated with early metastasis to the lung, lymph involvement, and rapid death unless rigorous treatment is begun early in the disease process.

3.4.5 Osteoporosis

Osteoporosis is a condition in which the amounts of bony mass decreases while the size of the bone remains constant. It is a major health problem causing over a million fractures a year in people over the age of 45. The risk of osteoporosis increases with age and is higher in females than in males. Factors in the development of osteoporosis include menopause, reduced activity, and decreased dietary intake of calcium.

A. Assessment

Clinical manifestations: Documented loss of bone density, most commonly found in conjunction with pathologic fractures secondary to osteoporosis. Most fractures occur in the dorsal (thoracic) and lumbar vertebral bodies (usually D8 through L2), the neck and intertrochanteric regions of the femur, and the distal radius. Vertebral compression fractures can develop gradually, resulting in loss of height, kyphosis, back discomfort, and constipation. Fractures of the hip result in significant morbidity and mortality.

B. Diagnostic Tests

1. Standard AP and lateral x-rays of the spine: Provide a diagnosis for osteoporotic fractures.

C. Medical Management and Surgical Interventions

1. **Oral estrogen:** Doses as low as 0.625 mg have been shown to be effective in preventing osteoporosis in postmenopausal women. Use of cyclic estrogen/progesterone also may reduce the risk of endometrial cancer.
2. **Calcium intake:** Should exceed 1000–1500 mg/day for women approaching menopause
3. **Vitamin D:** Necessary to allow adequate intestinal absorption and usage of calcium. The recommended daily intake of this vitamin is 600–800 U twice daily.
4. **Moderate weightbearing exercise:** To stress bones and activate osteoblastic bone formation, because inactivity has been shown to result in disuse osteoporosis.
5. **A variety of treatment modalities under investigation for use in combating osteoporosis:** Include sodium fluoride, calcitrol, calcitonin, weekly androgenic anabolic steroids, thiazides, and bisphosphonates. However, their efficacy has not been proven yet.

3.5 Musculoskeletal Surgical Procedures

3.5.1 Bunionectomy

Bunionectomy is surgery to correct hallux valgus. Hallux refers to the great toe and valgus means that it is bent outward, away from the midline. The bunion is a prominence of the first metatarsal head, resulting from altered joint dynamics, can also occur because of hereditary intrinsic joint weakness, the most frequent cause is improperly fitting footwear.

3.5.2 Amputation

Today, amputation is less frequently required as an orthopedic surgical intervention than it was before the advent of antibiotics and microsurgery techniques. However, amputation is still required for certain disorders, such as atherosclerotic arterial occlusive disease, osteomyelitis, severe trauma, malignant tumors, or congenital anomalies. Most amputations are performed especially in patients with diabetes mellitus or over age 60. The majority of amputations are of the lower extremity.

3.5.3 Bone Grafting

A bone graft procedure refers to the transfer of cancellous and/or cortical bone from one site to another. The bone can be from the patient (autogenic), another human (homogenic), or another species (heterogenic). Bone grafts can be required to create bony fusion of a joint (arthrodesis), compensate for lost or inadequately developed bone, or correct bony non-union of fractures.

Current microsurgical techniques permit myocutaneous-bone or muscle-bone grafts that involve bone, overlying muscle, and/or skin. These complex grafting techniques are used to rebuild large areas from which tissue has been lost due to trauma or necessary surgical resection.

3.5.4. Total Hip Arthroplasty

Total hip arthroplasty (THA) is surgery involving resection of the hip joint and its replacement with an endoprosthesis. THA is performed when the joint has been severely affected by disease, resulting in significant pain and a femoroacetabular articulation without useful function. THA is done for clients with osteoarthritis, rheumatoid arthritis, ankylosing spondylosis, and severe hip trauma. Usually,

3.5.5 Total Knee Arthroplasty

Total knee arthroplasty (TKA) is surgery that involves resection of the knee joint and its replacement with an endoprosthesis. TKA is done for clients with osteoarthritis, rheumatoid arthritis, gouty arthritis, hemophilic arthritis, and severe knee trauma.

3.6 Phimosi s and Paraphimosis

Phimosi s and paraphimosis are conditions that occur among uncircumci sed male clients when the opening of the foreskin is constricted. Phimosi s refers to an inability to retract the foreskin (prepuce); paraphimosis is a strangulation of the glans penis due to an inability to replace the retracted foreskin. These phimotic conditions are often caused by a congenitally small foreskin; however, chronic inflammation at the glans penis and prepuce secondary to poor hygiene or infection are also etiologic factors. Clients with phimosi s report pain with erection and intercourse and difficulty cleaning under the foreskin. Clients with paraphimosis experience painful swelling of the glans. If the condition continues, severe edema and urinary retention may occur. Circumcision is recommended to permanently relieve these conditions.

3.7 Hydrocele, Spermatocele, and Varicocele

The suffix, cele, indicates a swelling. Hydrocele, spermatocele, and varicocele all present as a swelling of the scrotum but in each case, the conditions are somewhat different as seen in the Table below. Often, hydrocele and spermatocele are not clinically significant and do not require treatment; however, varicoceles are thought to be an underlying cause of male infertility and may be surgically repaired

Condition and Etiology	Description	Signs and Symptoms	Diagnostic Aids	Medical and Surgical Management
Hydrocele Congenital defect; injury; infection; lymph obstruction; tumor; side effect of radiation; or unknown cause	Accumulation of as much as 100 mL of lymphatic fluid between the testis and tunica vaginalis	Swollen testicle; heaviness in scrotum or lower back; may be asymptomatic; pain if testicular blood flow is impaired	Palpation; transillumination	No treatment if asymptomatic; aspiration of fluid as a temporary measure; surgical excision of fluid-filled sac; treatment of primary condition (ie, infection)
Spermatocele Unknown cause	Epididymal, sperm-containing	Small, freely movable mass;	Palpation; transillumination	No treatment unless cyst is

	cyst	usually asymptomatic, may be painful if large		large and causes pain
Varicocele Incompetent valves in the spermatic veins	Venous dilation with damage to elastic fibers and hypertrophy of vein walls	Feeling of heaviness in scrotum; may be asymptomatic or have pain and swelling	Palpation; auscultation of venous rush; ultrasound; blood flow studies	No treatment, surgical ligation, or sclerosing

4.0 Conclusion

A fracture may be treated by any one or a combination of the following: traction, closed reduction, open reduction that may include internal or external fixation, and cast application.

5.0 Summary

When using traction, it is essential to (1) maintain continuous pull, (2) sustain countertraction, (3) preserve skeletal alignment in the line of pull, (4) keep splints and slings suspended, (5) ensure that ropes move freely through each pulley, (6) apply the prescribed amount of weight, and (7) keep weights hanging free.

6.0 Tutor Marked Assignment

- Identify the causes of osteomyelitis.
- Describe methods to prevent or reduce low back pain due to poor posture and body mechanics.
- Discuss the nursing management for a client with a sprain, dislocation, or cast; who is in traction or undergoing orthopedic surgery; or with an amputation.

7.0 Further Reading and Other Resources

Walsh M., Watson's (1997). Clinical Nursing and Related Sciences. (5th Edition)

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MODULE THREE

UNIT 1: CARE OF CLIENTS WITH RENAL SYSTEM DISORDERS

1.0	Introduction
2.0	Objective
3.0	Main Content.....
3.1	Renal Disorders
3.1.1	Glomerulonephritis
3.1.2	Nephrotic Syndrome
3.1.3	Acute Pyelonephritis
3.1.4	Renal Calculi
3.1.5	Hydronephrosis
3.2	Renal Failure
3.2.1	Acute Renal Failure
3.2.2	Chronic Renal Failure
3.3	Renal Dialysis
3.3.1	Care of the patient on dialysis t
3.4	Disorders of the Urinary Tract
3.4.1	Ureteral Calculi
3.4.2	Urinary Tract Obstruction
3.4.3	Cancer of the Bladder
3.5	Urinary Disorders Secondary to Other Disease Processes
3.5.1	Urinary Incontinence
3.5.2	Urinary Retention
4.0	Conclusion
5.0	Summary
6.0	Tutor Marked Assignment
7.0	Further Reading and Other Resources

1.0 Introduction

This unit focuses on renal disorders with emphasis on the care of a client on dialysis.

2.0 Objective

At the end of this unit , you will be able to;

- Differentiate between pyelonephritis and glomerulonephritis.
- Explain the difference between acute and chronic renal failure.
- Explain the purpose of dialysis and name two methods for performing the procedure.
- Discuss nursing care of a client undergoing dialysis.

3.0 Main Content

Blood urea nitrogen

Creatinine

Cystoscope

Intravenous pyelogram

Anasarca

Anuria

Uremia

3.1 Renal Disorders

3.1.1 Glomerulonephritis

Glomerulonephritis (GN) is the name of a group of diseases that damage the renal glomeruli. When the glomerulus is injured, protein and RBCs are allowed to enter the renal tubule and be excreted in the urine. GN can be acute or chronic. Most individuals with acute GN recover completely within 1 – 2 years, but renal damage continues to progress for those with chronic GN. Chronic GN is the most common cause of chronic renal failure. Most forms of GN are the result of immunological processes.

A. Assessment

History of: Recent URI or other infection; or other autoimmune disease; bloody urine.

Clinical manifestation: Hematuria, proteinuria, oliguria, dull bilateral flank pain, headache, low-grade fever, fatigue, lethargy, anorexia, nausea, nocturia, Presence of edema (peripheral, periorbital, sacral), crackles elevated BP.

B. Diagnostic Tests

1. Urinalysis and 24-hour urinary protein excretion: Red cell casts and protein excretion >3.5 g/day are common with GN.
2. BUN and serum creatinine: Will increase in the presence of decreased renal function.
3. throat and blood cultures, hepatitis B antigen, and immunoelectrophoresis of the serum and urine: to determine cause of GN.

C. Medical Management

1. **Bed rest:** For patients with acute GN.
2. Treat infections with antibiotics
3. **Pharmacotherapy**
 - Corticosteroids and cytotoxic agents: To suppress the immune system and reduce antibody formation.

- Anticoagulants: To reduce non-immunological mediators of glomerular damage.
 - Diuretics: To remove excess fluid.
 - Antihypertensives: To control BP
4. **Diet:** Restricted sodium and fluids if edema or hypertension is present. A high-carbohydrate diet is encouraged to maintain nutrition and prevent tissue catabolism. If renal function is markedly decreased, protein and potassium may be limited to prevent hyperkalemia and retention of excess nitrogenous wastes.
 5. **Peritoneal dialysis or hemodialysis:** To maintain homeostasis or prevent uremic complications if renal function is markedly decreased .

3.1.2 Nephrotic Syndrome

Nephrotic syndrome (NS) is a complex of symptoms that can occur due to glomerular damage and is characterized by increased urinary excretion of protein, decreased serum albumin, increased serum lipids, and edema. In adults, NS usually progresses to chronic renal failure.

Causes

The two main causes of NS are glomerulonephritis and diabetic nephropathy, and its course and prognosis depend on the status of the disease that caused it.

A. Assessment

History of: Glomerulonephritis or diabetes mellitus.

Clinical manifestations: Pallor; edema (periorbital, abdominal, sacral, anorexia, nausea, diarrhea, lethargy, fatigue. Patient also may have ascites, pleural effusion, decreased urinary output, and weight gain and either hypertension or hypotension.

B. Diagnostic Tests

1. 24-hour protein excretion: To diagnose the syndrome. NS is defined as the urinary excretion of >3.5 g/day of protein. Protein loss can be >10 g/day.
2. Urinalysis: Will show sediment-containing casts, over fat bodies, and RBCs.
3. Serum tests: Will show low albumin, elevated cholesterol and triglycerides, and low total calcium. BUN and creatinine may be elevated. Additional lab tests might be performed, depending on the suspected cause of NS.
4. Renal biopsy: Often necessary of NS. See “Glomerulonephritis,”.

C. Medical Management

1. **Bed rest.**
2. **Diet:** Low in sodium, rich in value protein (1.5 g/kg body weight/day), with adequate caloric intake. Liberal protein intake is required to provide necessary amino acids for albumin synthesis.
3. **Pharmacotherapy** may include the following:
 - Diuretics: Used cautiously to reduce edema .
 - Antibiotics: To treat infection.
 - Corticosteroids, anticoagulants, or cytotoxic agents: To treat glomerulonephritis.
 - Antihypertensive agents: To treat hypertension.

D. Nursing Diagnoses and Interventions

Potential alterations in fluid volume: Deficit related to vascular dehydration secondary to pharmacotherapy; Excess related to retention secondary to decreased serum albumin and renal retention of sodium and water.

Desired outcome: 1. Patient does not exhibit signs of dehydration or overhydration. 2. Patient can verbalize knowledge of foods high in sodium and the rationale for avoiding them.

1. Monitor I&O closely
2. Monitor weight daily. Report unusual or steady gains or losses.
3. Limit sodium intakes as prescribed. Instruct patient about foods that are high in sodium
4. Measure abdominal girth in patients with ascites.
5. Auscultate lung fields, and be alert to signs of respiratory distress.
6. Observe for indicators of decreased effective circulating volume (eg, hypotension, tachycardia, and decreased CVP), which can occur as a result of sodium restriction and the administration of diuretic or certain anti-hypertensives.

Alteration in nutrition: Less than body requirements related to decreased intake secondary to anorexia and increased need secondary to urinary losses of protein.

Desired outcome: Patient maintains a diet high in protein, does not exhibit signs of weight loss or malnutrition, and can verbalize knowledge of foods high in protein.

1. Provide prescribed high-protein diet in small, frequent feedings.
2. Teach patient about foods high in protein (eg, meat, poultry, fish, cheeses, nuts, lentils, and eggs).
3. Develop diet plan with dietitian, patient, and SOs, adjusting it to patient preference.
4. Encourage SOs to bring in patient's favorite high-protein foods as allowed.
5. Record calorie intake and weigh patient daily.

Potential for infection related to vulnerability secondary to treatment with immuno-suppressive agents, prolonged immobility, invasive procedures, and disease process.

Desired outcome: Patient does not exhibit signs of infection. Patient can verbalize knowledge of the indicators of infection and the importance of seeking medical attention promptly should they occur.

1. Minimize the risk of exposing patient to individuals with infections by providing a private room, if possible.

3.1.3 Acute Pyelonephritis

Acute pyelonephritis is an infection of the renal parenchyma and pelvis, which usually occurs secondary to an ascending UTI. UTIs typically result from anatomic or functional obstruction to urine flow, eg, from prostatic hypertrophy or renal calculi, or instrumentation such as catheterization or cystoscopy. Hematogenous infection also can occur in acute pyelonephritis when bacteria reach the kidney via the bloodstream. The incidence of acute pyelonephritis increases with advancing age is almost exclusively a disease of females.

Causative organism

Is usually a type of fecal flora such as Escherichia, Klebsiella, or Enterobacter.

A. Assessment

Clinical manifestations: Fever, chills, flank pain, nausea, vomiting, malaise, frequency and urgency of urination, dysuria, cloudy and foul-smelling urine.

Physical exam: Tender, enlarged kidneys; abdominal rigidity; and costovertebral tenderness.

History of: UTI and/or obstruction; recent urologic procedure.

B. Diagnostic Tests

Unless an anatomic or pre-existing renal disease is present, renal function should remain normal.

1. Urine culture: Should be positive for the causative organism. **Note:** Asymptomatic bacteriuria is common in the elderly.
2. Urinalysis: Will reveal presence of WBCs, WBC casts, RBCs, and bacteria.
3. Blood culture: for the causative organism.
4. IVP or retrograde pyelogram: May be performed for recurrent episodes

C. Medical Management and Surgical Interventions

1. Bed rest.

2. Pharmacotherapy

- Antibiotics: For the infection; initially parenteral, then oral.
- ASA or acetaminophen: To control the fever and treat the discomfort.

3. Surgical intervention: May be necessary if an obstruction is present.

3.1.4 Renal Calculi

The kidneys excrete several substances that singly or in combination are highly insoluble. Normally, these substances are excreted with minimal crystal formation; but diet, medications, metabolic abnormalities, systemic disease, or infection can increase the tendency for crystals to form and stones to develop. Altered urine pH and concentration also can be important factors in stone formation.

Stones can lodge and cause obstruction or be passed in the urine. Giant staghorn calculi occasionally develop and fill the entire renal pelvis. The most common types of stones are made of calcium, struvite, cystine, and uric acid. Renal calculi are often recurrent and a common medical problem. Complications include infection and hydronephrosis.

A. Assessment

Clinical manifestations: The primary symptom is pain, with location and severity depending on the area in which the stone has lodged. Vague back pain occurs when the stone lodges in the calyces or pelvis. Renal colic (severe flank pain radiating to the groin) is typical when the stone has lodged at the junction of the pelvis and ureter. Other indicators are hematuria, nausea, vomiting, syncope, and fever.

Physical exam: Diaphoresis, pallor, and obvious distress.

History of: Previous stone formation, UTI, or urinary tract obstruction; diet high in calcium, purine, or oxalate.

B. Diagnostic Tests

Same as Ureteral Calculi.

C. Medical Management and Surgical Interventions

1. Same as Ureteral Calculi.

2. Surgical interventions: Indications for surgery include complete obstruction; persistent infection; severe, uncontrollable pain; renal pelvic calculus that is too large to pass spontaneously any of the following surgery can be done; .

- Pyelolithotomy (incision into the renal pelvis).
- Nephrolithomy (incision into the renal parenchyma).
- Nephrectomy (removal of the kidney if it is severely damaged).

3.1.5 Hydronephrosis

Hydronephrosis is the dilatation of the renal pelvis and calyces secondary to the obstruction of urinary flow. It results from any condition or abnormality that causes urinary tract obstruction. If the obstruction is not corrected, the affected kidney eventually atrophies and fails. Obstruction in the urethra or bladder will affect both kidneys, while obstruction in a single ureter or kidney will affect only the involved kidney. Dramatic postobstructive diuresis can occur as a result of measures to relieve kidney obstruction. Inappropriate loss of sodium and water can in turn lead to volume depletion.

Major causes of obstruction are calculi, neoplasms and prostatic hypertrophy

A. Assessment

Indicators are determined by the level, severity, and duration of obstruction.

Kidney/ureteral obstruction: Flank pain and abdominal tenderness, renal colic, gross hematuria, paralytic ileus.

Bladder neck/urethral obstruction: Frequency, hesitancy, dribbling, incontinence, nocturia, clinical manifestations of renal insufficiency, suprapubic pain, anuria.

Physical exam: Enlarged kidney(s) and distended bladder if bladder neck obstruction is present; crackles (rales) and possibly hypertension and edema if patient is fluid overload.

History of: UTI or obstruction.

B. Diagnostic Tests

1. BUN and serum creatinine: To determine level of renal function.
2. Urinalysis: To determine the presence of stone formation or infection.
3. Renal ultrasound: Noninvasive technique that uses high-frequency sound waves to assess renal size, contour, and structural changes. Because it does not rely on dye uptake, it can be used to evaluate poorly functioning kidneys.
4. Abdominal x-ray, IVP, and/or retrograde pyelogram: To identify cause of obstruction.

C. Medical Management and Surgical Interventions

Management of hydronephrosis depends on the cause and duration of the urinary tract obstruction. Management of hydronephrosis might include the insertion of a nephrostomy tube into the renal pelvis to drain urine and relieve pressure. It is inserted percutaneously under local anesthesia or in an open surgical procedure.

3.2 Renal Failure

3.2.1 Acute Renal Failure

Acute renal failure (ARF) is a sudden decrease in renal function that may or may not be accompanied by oliguria. The kidney loses its ability to maintain biochemical homeostasis, causing dramatic alterations in fluid and electrolyte and acid – base balance. Although the renal damage is usually reversible, ARF has a high mortality rate. The mortality rate of ARF varies greatly with etiology, the patient's age, and other medical problems. The overall mortality rate for ATN is 40–60%. The clinical course of ATN can be divided into the following three phases: oliguric (lasting approximately 7–21 days); diuretic (7–14 days) and recovery (3–12 months).

Causes

Acute tubular necrosis (ATN), due to ischemia or nephrotoxins
Transfusion reactions,
Septic abortions,
Acute poststreptococcal glomerulonephritis,
Malignant hypertension, and
Hepato-renal syndrome.
Crush injuries.

A. Assessment

Electrolyte disturbance: Muscle weakness, dysrhythmias, pruritis.

Fluid volume excess: Oliguria, pitting edema, hypertension, pulmonary edema.

Metabolic acidosis: Kussmaul respirations (hyperventilation), lethargy, headache.

Uremia: Altered mental state, anorexia, nausea, diarrhea, pale and sallow skin, purpura, decreased resistance to infection, anemia, fatigue.

Physical exam: Pallor and edema (peripheral, periobital, sacral); crackles (rales) and elevated BP in patient who has fluid overload.

History of: Exposure to nephrotoxic substances, recent blood transfusion, prolonged hypotensive episodes or decreased renal perfusion, abortion, or a recent URI.

B. Diagnostic Tests

1. **BUN and serum creatinine:** Assess the progression and management of ARF. Although both BUN and creatinine will increase as renal function decreases, creatinine is a better indicator of renal function because it is not affected by diet, hydration, or tissue catabolism.
2. **Creatinine clearance:** Measures the kidney's ability to clear the blood of creatinine and approximates the glomerular filtration rate. It will decrease as renal function decreases.
3. **Urinalysis:** Can provide information about the cause and location of renal diseases as reflected by abnormal urinary sediment (casts and cellular debris).
4. **Urinary osmolality and urinary sodium:** To rule out renal perfusion problems
5. **Renal ultrasound:** Provides information about renal anatomy and pelvic structures, evaluates renal masses, and detects obstruction and hydronephrosis.
6. **Renal scan:** Provides information about the perfusion and function of the kidneys.

C. Medical Management

The goal is to remove the precipitating cause, maintain homeostatic balance, and prevent complications until the kidneys are able to resume function. Initially, a trial of fluid and diuretics may be used to rule out pre-renal problems.

1. **Restrict fluids:** Replace losses plus 400 mL/24h.
2. **Packed cells:** For active bleeding or if anemia is poorly tolerated.
3. **Pharmacotherapy**
 - Diuretics: In non-oliguric ARF for fluid removal
 - Antihypertensives: To control BP.
 - Aluminum hydroxide antacids: Bind phosphorus to control hyperphosphatemia.
 - Calcium or vitamin D supplements: For hypocalcemic patients.

- Sodium bicarbonate: To treat acidosis. It is used cautiously in hypocalcemic or fluid-overloaded patients.
 - Vitamins B or C: To replace losses if patient is on dialysis.
4. **Diet:** High carbohydrate, low protein, low potassium, and low sodium. Sodium is limited to prevent thirst and fluid retention. Protein is limited to minimize retention of nitrogenous wastes. Because of the loss of potassium during the diuretic phase, potassium might need to be increased during this time
 5. **TPN:** May be necessary for patients unable to maintain adequate oral/enteral intake.
 6. **Peritoneal dialysis or hemodialysis:** Administered if the above therapy is inadequate for maintaining homeostasis or preventing complications

3.2.2 Chronic Renal Failure

Chronic renal failure (CRF) is a slowly progressive, irreversible loss of kidney function, which can develop over months to years. Eventually it may progress to end-stage renal disease (ESRD). The patient with ESRD requires dialysis or a kidney transplant to sustain life. Prior to ESRD, the patient can lead a relatively normal life with CRF, managed by diet and medications. This period can last from days to years, depending on the cause of renal failure and the patient's renal function at the time of diagnosis.

Causes of CRF include; glomerulonephritis, diabetes mellitus, polycystic kidney disease, and hypertension. In some patients, the etiology of CRF is unknown.

A. Assessment

Fluid volume abnormalities: Crackles (rales), hypertension, edema, oliguria, or anuria.

Electrolyte disturbances: Muscle weakness, dysrhythmias, pruritis, tetany.

Uremia—retention of metabolic wastes: Weakness, malaise, anorexia, dry and discolored skin, peripheral neuropathy, irritability, clouded thinking, ammonia odor to breath. **Note:** Uremia adversely affects all body systems.

Metabolic acidosis: Rapid respirations, lethargy, headache.

Physical exam: Pallor, dry and discolored skin, edema (peripheral, periorbital, sacral). With fluid overload, crackles and elevated BP may be present.

History of: Glomerulonephritis, diabetes mellitus, polycystic kidney disease, hyper-tension, systemic lupus erythematosus, chronic pyelonephritis, or analgesic abuse, especially the combination of phenacetin and aspirin.

B. Diagnostic Tests

1. BUN and serum creatinine: Both will be elevated.
2. Creatinine clearance: Measure the kidney's ability to clear the blood of creatinine and approximates the glomerular filtration rate. Creatinine clearance will decrease as renal function decreases.
3. Urinalysis: Can provide information about the cause and location of renal disease as reflected by abnormal urinary sediment.
4. X ray of the kidneys, ureters, and bladder (KUB): Documents changes in size or shape, and some forms of obstruction.
5. IVP, renal ultrasound, renal biopsy, renal scan (using radionuclides), and CT scan:

6. Serum chemistries, chest and hand x rays, and nerve conduction velocity test: To assess for development and progression of uremia and its complications.

C. Medical Management and Surgical Interventions For renal failure

Prior to ESRD, medical management is aimed at slowing the progression of CRF. Once the patient reached ESRD, management is aimed at avoiding complications, alleviating uremic symptoms, and providing dialysis or renal transplantation.

1. **Diet:** Carbohydrates are increased in protein-restricted patients to ensure adequate caloric intake and prevent catabolism sodium is limited to prevent thirst and fluid retention, potassium is limited because of the kidney's inability to excrete excess potassium, and protein is limited to minimize retention of nitrogenous wastes.
2. **Pharmacotherapy**
 - Aluminum hydroxide antacids: Bind phosphorus to control hyperphosphatemia.
 - Antihypertensives: To control BP.
 - Multivitamins and folic acid: For patients with dietary restrictions or who are on dialysis (water-soluble vitamins are lost during dialysis).
 - Anabolic steroids, parenteral iron, or ferrous sulfate: To treat anemia.
 - Diphenhydramine: To treat itching.
 - Sodium bicarbonate: To treat acidosis.
 - Vitamin D preparations: To treat hypocalcemia.
 - Deferoxamine: To treat iron or aluminum toxicity (experimental use).
3. **Packed cells:** To treat severe or symptomatic anemia.
4. **Maintain homeostasis and prevent complications** by avoiding the following: volume depletion, hypotension, use of radiopaque contrast medium, and nephrotoxic substances. Pregnancy is contraindicated.
5. **Renal transplant or dialysis:** If the above therapies are inadequate.

D. Nursing Diagnoses and Interventions

Activity intolerance related to fatigue and weakness secondary to anemia and uremia.

Desired outcome: Patient verbalizes decreases in weakness and fatigue and exhibits evidence of improving endurance.

1. Typically, these patients are not transfused unless hematocrit drops below 20% or anemia is poorly tolerated.
2. Observe for increased weakness, fatigue, dyspnea, chest pain, or further decreases in hematocrit.
 - Provide and encourage optimal nutrition.
 - Administer anabolic steroids (eg, nandrolene) if prescribed. Tell female patients about side effects, including facial hair, deepening voice, and menstrual irregularities.
 - Observe for and report evidence of occult blood and blood loss.
 - Do not administer ferrous sulfate at the same time as aluminum hydroxide antacids. The two medications should be given at least 1 hour apart to maximize absorption of the ferrous sulfate.
3. Administer parenteral iron if prescribed. Assist patient with identifying activities that cause increased fatigue and adjusting those activities accordingly.
4. Assist the patient with ADLs while encouraging maximum independence.

Impairment of skin integrity related to pruritus is controlled and can verbalize knowledge of the cause, preventive factors, and treatment.

Desired outcome: Patient states that pruritus is controlled and can verbalize knowledge of the cause, preventive factors, and treatment.

1. Pruritus is common in uremic patients, causing frequent, intense scratching.
2. Keep patient's fingernails short.
3. Because uremia retards wound healing, instruct the patient to monitor scratches for evidence of infection
4. Encourage the use of skin emollients, uremic skin is often dry and scaly because of reduction in oil gland activity.
5. Patients should avoid hard soaps and excessive bathing.
6. Advise patient to bathe every other day and use bath oils as needed if dry skin is a problem.

Knowledge deficit: Need for frequent BP checks and compliance with antihypertensive therapy and the potential for change in insulin requirements for diabetics.

Desired outcomes: Patient can verbalize knowledge of the importance of frequent BP checks and compliance with antihypertensive therapy. Diabetic patient can verbalize knowledge of the potential for change in insulin requirements.

1. Teach patient the importance of getting BP checked at frequent intervals and complying with the prescribed antihypertensive therapy.
2. Teach diabetic patients that insulin requirements often decrease as renal function decreases. Instruct diabetic patients to be alert to indicators of hypoglycemia, including confusion, diaphoresis, and hypotension.

Potential for injury related to sensorimotor and medication alterations secondary to electrolyte and acid – base imbalance and uremia.

Desired outcome: Patient does not exhibit signs of injury and can verbalize orientation to time, place, and person. Patient can relate knowledge of the importance of avoiding foods and products that contain potassium.

1. Do not give potassium-containing medications such as potassium penicillin G.
2. observe for indicators, of hyperkalemia if the patient requires multiple blood transfusions
3. Use fresh-packed cells when possible.

3.3 Renal Dialysis

These are lifesaving procedures used to treat severely decreased or absent renal function. Dialysis can be either temporary, until the kidneys are able to resume adequate function, or permanent. Dialysis is defined as the selective movement of water and solutes from one fluid compartment to another across a semipermeable membrane. The two fluid compartments are the patient's blood and the dialysate (electrolyte and glucose solution). With hemodialysis, the semipermeable membrane is an artificial one; with peritoneal dialysis, the peritoneum serves as a natural dialysis membrane.

Indications for dialysis include: Acute renal failure or acute episodes of chronic renal failure, medications, and fluid restriction; ESRD; drug overdose; hyperkalemia; fluid overload; or metabolic acidosis.

Forms of dialysis,

There are two peritoneal dialysis and hemodialysis.

Functions of dialysis: Correction of electrolyte abnormalities; removal of fluid and metabolic wastes; correction of acid–base abnormalities.

Peritoneal dialysis: Slow; does not require heparinization; requires a minimum of equipment.

Hemodialysis: Fast; requires heparinization, expensive and complex

3.3.1 Peritoneal Dialysis

Peritoneal dialysis utilizes the peritoneum as the dialysis membrane. Dialysate is instilled into the peritoneal cavity via a special catheter, and movement of solutes and fluid occurs between the patient's capillary blood and the dialysate. At set intervals the peritoneal cavity is drained and new dialysate is instilled.

A. Components of Peritoneal Dialysis

1. **Catheter:** Silastic tube that is either implanted as a surgical procedure for chronic patients or inserted at the bedside for acute dialysis.
2. **Dialysate:** Sterile electrolyte solution similar in composition to normal plasma. The electrolyte composition of the dialysate can be adjusted according to individual need. The most commonly adjusted electrolyte is potassium. Glucose is added to the dialysate in varying concentrations to remove excess body fluid via osmosis.

B. Types of Peritoneal Dialysis

1. Intermittent peritoneal dialysis (IPD): The patient is dialyzed for 8–10-hour periods, 4–5 times per week. This type of dialysis usually requires a critical care setting. The patient is restricted to bed.
2. Continuous ambulatory peritoneal dialysis (CAPD): The patient attaches a specialized bag of dialysate to the peritoneal catheter; allows the dialysate to drain in; clamps the catheter, leaving the bag attached and goes about his or her daily routine. After 4 hours (8 hours at night) the clamp is opened and the dialysate is allowed to drain out.
3. Continuous cycling peritoneal dialysis (CCPD): This is a combination of IPD and CAPD. The patient is ambulatory by day and restricted to bed at night.

C. Nursing Diagnoses and Interventions

Potential for infection related to increased vulnerability secondary to direct access of the catheter to the peritoneum.

Desired outcome: Patient does not exhibit signs of infection. Patient can verbalize understanding of the clinical manifestations of infection and demonstrates sterile technique for bag, tubing, and dressing changes.

1. The most common complication of peritoneal dialysis is peritonitis. Observe for and report indications of peritonitis, including fever, abdominal pain, cloudy outflow, nausea, and malaise.
2. The dialysate must remain sterile.
3. Maintain sterile technique when adding medications to the dialysate.
4. Observe for and report redness, drainage, or tenderness at exit site.
5. Culture any exudates and report the results

Potential alterations in fluid volume: Excess related to fluid retention or inadequate exchange secondary to catheter problems and/or peritonitis; Deficit related to abnormal loss secondary to hypertonicity of the dialysate.

Desired outcome: Patient does not exhibit signs of dehydration, fluid overload, or retained dialysate.

1. Observe for and report indicators of fluid overload, such as hypertension, tachycardia, distended neck veins, or increased CVP. Fluid retention can occur because of catheter complications that prevent adequate outflow, or a severely scarred peritoneum that prevents adequate exchange.
2. Monitor I&O observe for incomplete dialysate returns.
3. Weight daily. A steady weight gain is indicative of fluid retention.
4. Elevate the HOB if respiratory distress occur because of compression of the diaphragm by the dialysate.
5. Bloody outflow may appear with initial exchanges. Report gross bloody outflow.

Alteration in nutrition: Less than body requirements related to increased need secondary to protein loss in the dialysate.

Desired outcome: Patient does not exhibit signs of malnutrition and can verbalize knowledge of the prescribed dietary regimen.

1. Ensure adequate dietary intake of protein: 1.2–1.5 g/kg body weight daily. An increased intake of protein is necessary to prevent excessive tissue catabolism.
2. Provide a list of restricted and encouraged food

Potential for injury related to sensorimotor and mentation alterations secondary to uremia and serum electrolyte imbalance.

Desired outcomes: Patient does not exhibit signs of injury and can verbalize orientation to person, place, and time and knowledge of the clinical manifestations of uremia and serum electrolyte imbalance.

1. Observe for and report the following:
 - Confusion, lethargy, and restlessness.
 - Muscle cramps and muscle weakness.
 - Abdominal cramps, lethargy, and dysrhythmias.

3.3.2 Hemodialysis

During hemodialysis, a portion of the patient's blood is removed via a special vascular access, heparinized, pumped through an artificial kidney (dialyzer), and then returned to the patient's circulation. Hemodialysis is either a temporary, acute procedure performed as needed, or it is performed chronically three times a week for 3–6 hours each treatment.

A. Components of Hemodialysis

1. **Artificial kidney (dialyzer):** Composed of a blood compartment and dialysate compartment, separated by a semipermeable membrane that allows the diffusion of solutes and the filtration of water. Protein and bacteria do not cross the artificial membrane.

2. **Dialysate:** An electrolyte solution similar in composition to normal plasma. Each of the constituents may be varied according to patient need. The most commonly altered component is potassium. Glucose may be added to prevent sudden drops in serum osmolality and serum glucose during dialysis.
3. **Vascular access:** Necessary to provide a blood flow rate of 200 – 300 mL/min for an effective dialysis.

B. Nursing Diagnoses and Interventions

Potential alterations in fluid volume: Excess related to fluid retention secondary to renal failure; Deficit related to excessive fluid removal secondary to dialysis.

Desired outcome: Patient does not exhibit signs of overhydration or dehydration. Patient can verbalize understanding of the clinical manifestations of overhydration and dehydration.

1. Monitor I&O and daily weight as indicators of fluid status. A steady weight gain is indicative of retained fluid.
2. Observe for and report signs of fluid overload: edema, hypertension, crackles (rales), tachycardia, distended neck veins, SOB, and increased CVP.
3. After dialysis observe for and report of volume depletion, including hypotension, decreased CVP, and tachycardia.

Potential for injury related to increased risk of bleeding secondary to heparinization with dialysis.

Desired outcome: Patient does not exhibit signs of excessive bleeding.

1. Observe for bleeding
2. Do not give IM injection for at least 1 hour postdialysis to prevent hematoma formation.
3. Observe the stool for evidence of bleeding. Report findings.

Potential for injury related to risk of complications secondary to the creation of the vascular access for hemodialysis.

Desired outcomes: Patient does not exhibit signs of injury from vascular access complications.

1. Monitor the vascular access as follows: Assess for patency, auscultate for bruit, and palpate for thrill.
2. Report severe or unrelieved pain, and observe for and report numbness, tingling, and swelling of the extremity distal to the access, any of which can signal inadequate blood supply.
3. Observe the extremity distal to vascular access for decreased capillary refill, or is discolored, as these problems can occur with vascular insufficiency.
4. Follow the three principles of nursing care common to all types of vascular access: Prevent bleeding, prevent clotting, and prevent infection.

3.4 Disorders of the Urinary Tract

3.4.1 Ureteral Calculi

Ureteral calculi are common urologic condition. Although the cause of stones is unknown in 50% of reported cases, it is believed that they originate in the kidney and are passed through the kidney to the ureter; 90% of all stones pass from the ureter into the bladder and out of the urinary system spontaneously.

A. Assessment

Clinical manifestations: Pain that is sharp, sudden, and intense or dull and aching. Pain can be intermittent as the stone moves along the ureter, subsiding when it enters the bladder. Nausea, vomiting, diarrhea, abdominal pain, and paralytic ileus can occur. Patient may experience frequency, void in small amounts, and have hematuria.

Physical exam: Pallor and diaphoresis may be noted; chills and fever may be present in the acute stage. There can be absence of bowel sounds secondary to ileus, and the abdomen may be distended and tympanic.

History of: Sedentary lifestyle, residence in geographic area in which water supply is high in stone-forming minerals, vitamin A deficiency, vitamin D excess, hereditary cystinuria.

B. Diagnostic Tests

1. Serum tests: To assess calcium, phosphorus, and uric acid levels.
2. Serum and urine creatinine tests: To evaluate renal function. Abnormalities are reflected by high serum creatinine and low urine creatinine.
3. Urinalysis: To test urine pH and specific gravity and for presence of RBCs, WBCs, crystals, and casts.
4. Urine culture: To assess for infection.
5. 24 -hour urine collection: To test for high levels of uric acid, cystine, oxalate, calcium, phosphorus, or creatinine.
6. Kidney, ureter, bladder (KUB) x-ray: To outline gross structural changes in the kidneys and urinary system.
7. IVP/excretory urogram: To outline radiopaque stones within the ureters.
8. CT scan with or without injection of contrast medium: To delineate cyst, tumors, calculi, and other masses; ureteral dilation; and bladder distention.

C. Medical Management and Surgical Interventions

1. Pharmacotherapy during the acute stage

- Narcotic and antispasmodic agents: To relieve pain and ureteral spasms.
- Antiemetics: For nausea and vomiting.
- Antibiotics: For infection.

2. Prophylactic pharmacotherapy

- For uric acid stones: Allopurinol, sodium bicarbonate, potassium or sodium citrate, or citric acid is given to reduce uric acid production or alkalinize the urine.
- For calcium stones: Ascorbic acid, ammonium chloride, potassium acid phosphate, sodium and potassium phosphates, or hydrochlorothiazide is given to acidify the urine, produce solubility, or reduce urinary calcium.
- For cystine stones: Penicillamine is given to lower cystine levels in the urine.

3. IV therapy: For patients who are dehydrated.

4. Increase fluid intake: To help flush stone from ureter to the bladder and out through the system

3.4.2 Urinary Tract Obstruction

Urinary tract obstruction is usually the result of blockage from pelvic tumors, calculi, and urethral strictures. Additional causes include neoplasms, benign prostatic hypertrophy, ureteral or urethral trauma, inflammation of the urinary tract, and pelvic or colonic surgery in which ureteral damage has occurred.

Pathophysiology

The obstruction acts like a dam, causing urine to collect and pool. Muscles in the area contract to push urine around the obstruction, resulting in the dilation of the structures behind the obstruction. Hydrostatic pressure increases, and filtration and concentration processes within the urinary system are compromised. Obstructions can occur anywhere along the urinary tract, but the most common sites are the uretero-pelvic and ureterovesical junctions, bladder neck, and urethral meatus. Obstructions in the upper urinary tract can lead to bilateral involvement of the ureters and kidneys as well as of the bladder.

A. Assessment

Clinical manifestations: Anuria, pain that is sharp and intense or dull and aching, nausea, vomiting, local abdominal tenderness, hesitancy, straining to start a stream, dribbling, oliguria, and nocturia.

Physical exam: Bladder distention, mass in flank area, and “kettle drum” sound over bladder with percussion.

History of: Recent fever (possibly caused by the obstruction); hypertensive episodes (caused by increased hormone production from the body’s attempt to increase renal blood flow).

B. Diagnostic Tests

1. Serum electrolytes, BUN, and creatinine: To assess renal function.
2. Hemoglobin and hematocrit: To assess for systemic bleeding and/or anemia, which may be related to decreased renal secretion of erythropoietin.
3. Urinalysis, urine culture, IVP, and KUB radiology: .

C. Medical Management and Surgical Interventions

1. **Catheterization:** To establish drainage of urine.
2. **Pharmacotherapy**
 - Narcotics: For pain relief.
 - Antispasmodics: For relief of spasms.
 - Antibiotics: For bacterial infection.
 - Corticosteroids: For reduction of local swelling.
3. **IV therapy:** For acutely ill, dehydrated patients.
4. **Serial urine and serum tests:** For ongoing analysis of electrolytes and osmolality.
5. **Cystoscopy:** To determine degree of bladder outlet obstruction.
6. **Surgical removal of obstruction or dilation of strictures.**

D. Nursing Interventions

1. Do not allow rapid emptying of the bladder. Rapid decompression can lead to hemorrhage, acute fluid and electrolyte imbalance, and shock.
2. Monitor VS for signs of hypotension: decreasing BP, changes in LOC, tachycardia, tachypnea, tready pulse.
3. Record accurate output
4. Observe for and report indicators of the following:
 - Hypokalemia: Abdominal cramps, lethargy, dysrhythmias.
 - Hyperkalemia: Diarrhea, colic, irritability, nausea, muscle cramps, weakness, irregular apical or radial pulses.
 - Hypocalcemia: Muscle weakness and cramps, complaints of tingling in fingers, positive Trousseau and Chvostek signs.
 - Hyperphosphatemia: Excessive itching.

5. Monitor mentation, noting signs of disorientation, which can occur with electrolyte imbalance.
6. Weigh patient daily using the same scale and at the same time of day, eg, before breakfast. Weight fluctuations of 2–4 lb (0.9–1.8 kg) normally occur in a diuresing patient.

3.4.3 Cancer of the Bladder

Cancer of the bladder is the second most common form of urinary system cancer. Causes are not clearly understood, but industrial exposure to certain chemicals such as b-naphthylamine is believed to be a factor, as is smoking and the consumption of large amounts of coffee and saccharin.

Bladder cancer often begins in the bladder lumen, but the bladder neck and ureteral orifices also can be involved. Cellular proliferation can occur throughout the transitional epithelium that lines the kidneys, ureters, and mucosa of the bladder. Metastasis can appear in the lymph nodes and spread to the bones, liver, and lungs.

A. Assessment

Clinical manifestations: Dysuria, painless hematuria, burning with urination. Depending on tumor size, the patient may experience suprapubic pain.

Physical exam: Presence of a mass, which may be palpated by using both hands on the abdomen in an effort to feel the outline of the tumor.

B. Diagnostic Tests

1. Urinalysis, urine culture, IVP:
2. Urine cytology: To assess for abnormal cells.
3. WBC, Hgb, and Hct: To check for presence of infection, bleeding, and anemia.
4. Alkaline phosphatase test: To assess for metastasis to the bones.
5. Biopsy in conjunction with cystoscopy: to visualize the structures.
6. Cystogram (cystography): To outline tumors that are present in the bladder.

C. Medical Management and Surgical Interventions

1. **Grading and staging the disease:** The degree of grading and staging is determined by the extent of metastasis and tissue involvement: the greater the metastastasis, the higher the grade and stage.
2. **Chemotherapy:** Methotrexate, doxorubicin, fluorouracil, cyclophosphamide, mitomycin C, and cisplatin may be used. Intravesical chemotherapy, which is used for superficial bladder cancers, involves instillation of medication into the bladder via an indwelling catheter.
3. **Palliative radiation therapy:** Used primarily in the late stages for pain relief, but it also can be used early in treatment.
4. **Segmental resection:** Performed if the dome of the bladder is involved. The top half of the bladder is removed via an abdominal incision.
5. **Urinary diversion:** This is another surgical intervention done commonly via catheter installation

3.5 Urinary Disorders Secondary to Other Disease Processes

3.5.1 Urinary Incontinence

Urinary incontinence is the physiologic or psychologic inability to control urine. Usually it results from an interruption in the signal from the musculature and sphincter of the bladder as it transmits through the spinal cord to the brain. General causes include diminished cerebral functioning, UTI, bladder weakness, medications (eg, anticholinergic drugs), and interference with the urethrobladder reflex. Diseases and trauma that can cause incontinence include cerebrovascular accident (CVA), traumatic brain injury, and meningitis. With stress incontinence, which occurs as the result of a weakness of the musculature and sphincter of the bladder, there is loss of urine with sneezing, laughing, coughing. Urinary incontinence occurs most frequently in individuals over age 65.

3.5.2 Urinary Retention

When urine is produced and accumulates in the bladder but is not released, the condition is called urinary retention. The major cause is obstruction, for example, from benign obstructive hypertrophy, tumor, calculi, urethral stricture, fibrosis, or meatal stenosis. Other causes include decreased sensory stimulation to the bladder, anxiety, or muscular tension. Medications such as opiates, sedatives, antihistamines, antispasmodics, major tranquilizers and antidepressants, and antidyskinetics also can interfere with the normal micturition reflex.

A. Assessment

Clinical manifestations: Sudden inability to void, intense suprapubic pain, restlessness, diaphoresis, voiding small amounts (20 – 50 mL) at frequent intervals.

Physical exam: “Kettle drum” sound with bladder percussion, bladder distention, bladder displacement to one side of the abdomen.

B. Diagnostic Tests

1. Urinalysis, urine culture, serum electrolytes, BUN, serum creatinine, KUB, and IVP: All may be performed to evaluate renal–urinary function and structure and assess for infection and other problems.
2. Cystometrograms: To evaluate the neuromusculature of the bladder by measuring the efficiency of the detrusor muscle reflex, intravesical pressure and capacity, and the bladder’s reaction to thermal stimulation. During this procedure the patient voids into a funnel attached to a machine that graphically measures the amount, time, and flow of voiding.

C. Medical Management

1. **Catheterization:** For drainage of urine.
2. **Pharmacotherapy**
 - Cholinergics: To stimulate bladder contractions.
 - Analgesia: For pain relief.
 - Antibiotics If infection is present.
3. **IV therapy:** For hydration of the acutely ill patient.
4. **Serial urine and serum electrolyte testing:** For ongoing evaluation of electrolyte and osmolality status.
5. **Surgery:** Performed if obstruction is the cause of the retention.

D. Nursing Interventions

1. Assess the bladder for distention by inspection, percussion, and/or palpation; measure and document I&O.

2. Try noninvasive measures for release of urine: Have patient listen to the sound of running water or place hands in a basin of warm water. If these measures are ineffective, try pouring warm water over the perineum.
3. Notify the doctor if patient is unable to void, has bladder distention, and/or has suprapubic or urethral pain.
4. If catheterization is prescribed, maintain aseptic technique and ensure that the bladder is slowly decompressed to prevent acute fluid and electrolyte imbalance, shock, and/or hemorrhage.

4.0 Conclusion

The kidneys excrete several substances that singly or in combination are highly insoluble. Normally, these substances are excreted with minimal crystal formation; but diet, medications, metabolic abnormalities, systemic disease, or infection can increase the tendency for crystals to form and stones to develop. Altered urine pH and concentration also can be important factors in stone formation.

5.0 Summary

Acute renal failure is a sudden, rapid decrease in renal function that is potentially reversible. Chronic renal failure is a progressive and irreversible condition that eventually requires lifesaving treatment measures such as dialysis or a kidney transplant in end-stage disease. Dialysis is a technique for cleaning and filtering the blood; there are two different methods: hemodialysis and peritoneal dialysis.

6.0 Tutor Marked Assignment

- Explain the difference between acute and chronic renal failure.
- Explain the purpose of dialysis and name two methods for performing the procedure.

7.0 Further Reading and Other Resources

Walsh M., Watson's (1997). *Clinical Nursing and Related Sciences*. (5th Edition)

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UNIT II : CARE OF CLIENTS WITH NEUROLOGIC DISORDERS

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1.0 Introduction

This unit focuses on the care of clients with neurologic disorders

2.0 Objective

On completion of this unit you will be able to:

- Differentiate strains, contusions, and sprains.
- Describe the signs and symptoms of multiple sclerosis,
- State the pathophysiology of Alzheimers disease
- Identify the causes of epilepsy.
- Discuss the nursing management for a client with epilepsy

3.0 Main Content

3.1 Inflammatory Disorders of the Nervous System

Inflammation of nervous system tissue results from a wide variety of causes, including bacterial or viral infections, autoimmune processes, and chemical toxins. The inflammatory response may cause increased vascular permeability with exudation of fluids from the vessels, resulting in swelling, which in turn can cause increased intracranial pressure (ICP).

3.1.1 Multiple Sclerosis

Multiple sclerosis (MS) is an inflammatory disorder of the central nervous system (CNS) myelin. In response to the inflammation, the myelin nerve sheaths peel off the axon cylinders. This demyelination interrupts electric nerve transmission and causes the wide variety of symptoms associated with MS. If the myelin regenerates, electric nerve impulse transmission may be restored. Symptoms will decrease or disappear; that is, the patient may go into remission. If the inflammation is severe and causes irreversible destruction of myelin, the involved areas are replaced by dense glial scar tissue that forms areas of sclerotic plaque, which permanently damage the conductive pathways of the CNS.

Etiology of MS

Although the etiology of MS is unknown, causes include; slow-acting viral infections, allergic reactions to infections agents such as viruses, and autoimmune processes are suspected causes. Infection, trauma, and pregnancy, fatigue and physical or emotional stress, heat and fever are common precipitating factors

A. Assessment

Onset of MS can be extremely rapid, causing disability within days, or it can be insidious, with exacerbations and prolonged remissions allowing an active life. Clinical manifestations vary widely, depending on the site and extent of demyelination, and they can change from day to day. Usually, early symptoms are mild.

Clinical manifestations

- Damage to motor nerve tracts: Weakness, paralysis, and/or spasticity. Fatigue is common. Diplopia may occur secondary to ocular muscle involvement.
- Damage to cerebellar or brain stem regions: Intention tremor, nystagmus, or other tremors; uncoordination, ataxia; weakness of facial and throat muscles resulting in difficulty chewing, dysphagia, and dysarthria.
- Damage to sensory nerve tracts: Decreased perception of pain, touch, and temperature; paresthesias such as numbness and tingling; decrease or loss of proprioception; and/or decrease or loss of vibratory sense. Optic neuritis may cause partial or total loss of vision.
- Damage to cerebral cortex (especially frontal lobes): Mood swings, inappropriate affect, euphoria, apathy, irritability, depression, and/or hyperexcitability.
- Damage to motor and sensory control centers: Urinary frequency, urgency, or retention; urinary and fecal incontinence; constipation.
- Sacral cord lesions: Impotence; diminished sensations that result in inhibit sexual response.

Physical exam: Ophthalmoscopic inspection may reveal temporal pallor of optic disks. Reflex assessment may show increased deep tendon reflexes (DTRs) and diminished abdominal skin and cremasteric reflexes.

B. Diagnostic Tests

1. Magnetic resonance imaging (MRI): To reveal presence of plaques.
2. EEG: Abnormal in a third of patients with MS
3. CT scan: To demonstrate presence of plaques and rule out mass lesions.
4. Lumber puncture and CSF analysis:

C. Medical Management and Surgical Interventions

1. **Bed rest**: During acute exacerbation.

2. Pharmacotherapy

- Anti-inflammatory agents: Prednisone, dexamethazone, adrenocorticotrophic hormone (ACTH), etc. may be prescribed during an exacerbation in an attempt to decrease inflammation and associated edema of the myelin and hasten onset of a remission.
- Antispasmodics and muscle relaxants such as baclofen or dantrolene sodium: May be given to decrease spasticity.
- Smooth muscle relaxants such as propantheline bromide: To decrease urinary frequency and urgency.
- Smooth muscle stimulants such as bethanechol chloride: May be given to help prevent urinary retention.
- Tranquilizers such as diazepam: May be given for both their anxiety-reducing and muscle-relaxant effects.

3. **Physical medicine**: Physical therapy, occupational therapy, and assistive devices or braces may be prescribed so that patient can maintain mobility and independence with ADLs. Muscles-strengthening and conditioning exercises and gait training are also frequently indicated.

4. **ROM exercises**: To maintain or increase joint function and prevent contractures.

5. **Counseling and/or psychotherapy**: To help patient and SOs adapt to the disability and deal with emotions and feelings that are either a direct or indirect result of the disease process.

6. **Surgical interventions**: To treat complications such as contractures, spasticity, decreased mobility, and pain. Interventions may include tenotomy, myotomy, peripheral neurectomy, and rhizotomy.

3.1.2 Bacterial Meningitis

Bacterial meningitis is an infection that results in inflammation of the meningeal membranes covering the brain and spinal cord. It can be transmitted in one of four ways; via airborne droplets from infected individuals; from direct contamination (eg, from a penetrating skull wound, lumbar puncture, ventricular shunt, or surgical procedure); via the bloodstream (eg, pneumonia, endocarditis); or from direct contact with an infectious process that invades the meningeal membranes such as that which can occur with osteomyelitis, sinusitis, otitis media, mastoiditis, or brain abscess. Common infecting agents include Neisseria meningitides, Diplcoccus pneumoniae, Hemophilus influenzae, Staphylococcus aureus, and Escherichia coli. The prognosis is good if the disorder is recognized early and antibiotic treatment is initiated promptly.

A. Assessment

Infection: Fever, chills, malaise.

Increased ICP: Severe headache, irritability, drowsiness, stupor, coma, nausea and vomiting, decreased pupillary reaction to light, pupillary dilation or inequality.

Meningeal irritation: Back stiffness and pain, nuclear rigidity.

Other: Generalized seizures and photophobia; joint pain (in the presence of H. influenzae.)

Physical exam: A positive Brudzinski's sign is elicited: When the neck is passively flexed forward, both legs flex involuntarily at the hip and knee. A positive Kernig's sign also may be found: When the thigh is flexed, the individual is unable to extend the leg completely without pain. In the presence of meningococcal meningitis, a pink macular rash, petechiae, ecchymosis, purpura, and increased DTRs also may occur.

B. Diagnostic Tests

1. Lumbar puncture, CSF analysis, and Gram stain and culture: To identify causative organism.
2. Culture and sensitivity testing of blood, urine, and other body secretions: To identify infective organism and determine appropriate antibiotic.
3. counterimmunoelectrophoresis (CIE): For detection of antigens of pneumococci, meningococci, and H.influenzae in the CSF, blood, and urine.
4. Sinus and chest x-rays: Taken after treatment is started to rule out sinusitis and pneumonia.

C. Medical Management

1. Strict respiratory isolation:

2. **Parenteral antibiotics:** antibiotics may include the following: penicillin G, ampicillin, nafcillin, oxacillin, chloramphenicol, gentamicin, kanamycin; or vancomycin.

3. Other pharmacotherapy

- Osmotic diuretics (eg, mannitol): To decrease cerebral edema.
 - Anticonvulsant agents (eg, diazepam and phenytoin): To control seizures.
 - Analgesics and antipyretics (eg, aspirin and acetaminophen): For headache and/or fever.
 - Sedatives and tranquilizers: To promote rest.
4. **Bed rest:** During acute stage of the disease.
 5. **Limitation of fluids to two-thirds maintenance (about 1500 mL):** To keep patient underhydrated and reduce cerebral edema and effects of inappropriate ADH secretion.
 6. **Support respirations:** Via oxygen, suctioning, or intubation as necessary.
 7. **Treatment of complications:** Examples include disseminated intravascular coagulation, respiratory or heart failure, and septic shock.
 8. **Nutritional support:** Enteral or parenteral feedings as required for patients who are stuporous or comatose.

3.1.3 Encephalitis

Encephalitis is an inflammation of the brain that can cause severe neuronal dysfunction. It is often the result of an arbovirus infection that is transmitted by an infected mosquito or tick. Post-infectious encephalitis occurs after a vaccination or as a complication of other infections such as measles, chickenpox, and herpes virus. The prognosis varies according to the type of infection.

A. Assessment

Infection: Fever, chills, malaise.

Increased ICP: Headache; changes in LOC such as irritability, confusion, and drowsiness that can progress to stupor and coma; nausea and vomiting; pupillary changes such as inequality and decreased reaction to light.

Meningeal irritation: Neck stiffness/rigidity; pain.

Focal: Symptoms vary. Patient may have seizures or photophobia, ataxia, and sensorimotor deficits. Eastern equine encephalitis, for example, can destroy major portions of a lobe or hemisphere and leave the individual with hemiplegia, aphasia, blindness, deafness, and/or seizures. Herpes simplex encephalitis has a special affinity for the frontal and temporal lobes of the brain, resulting in alterations in senses of smell and/or taste, seizures, aphasia, organic psychosis, and dementia.

B. Diagnostic Tests

1. **Lumbar puncture and CSF analysis:** May reveal increased CSF pressure, increased WBC and protein levels, and normal glucose. **CT scan:** To rule out other neurologic problems.
2. **EEG:** May show presence of abnormal electrical activity.

C. Medical Management

1. **Antiviral agent (eg, vidarabine):** For herpes encephalitis. Side effects may include tremor, dizziness, hallucinations, anorexia, nausea, vomiting, diarrhea, itching, rash, and anemia.
2. **Supportive pharmacotherapy**
 - Anticonvulsants such as phenytoin.
 - Glucocorticosteroids such as dexamethasone: To reduce cerebral edema and inflammation.
 - Sedatives: For restlessness.
 - Analgesics and antipyretics such as aspirin and acetaminophen: For headache and fever.
3. **Limitation of fluids to two thirds maintenance (about 1500 ml):** To maintain a state of underhydration, which helps reduce cerebral edema.
4. **Support respirations:** Via oxygen, suctioning, or intubation.
5. **Nutritional support:** Enteral or parenteral feedings for stuporous or comatose patients, as needed.

3.2 Degenerative Disorders of the Nervous System

3.2.1 Parkinsonism

Parkinson's disease is a slowly progressive, degenerative disorder of the CNS affecting the brain centers that regulate movement. For unknown reasons, cell death occurs in the substantia nigra of the midbrain. This leads to an abnormally low concentration of dopamine in the corpus striatum, which causes symptoms of Parkinson's disease. Possible causes include viral encephalitis, neurotoxins, cerebrovascular disease, head injury, phenothiazide use, and exposure to carbon monoxide. Approximately 1% of all individuals over age 50 have this disease. Parkinsonism is usually progressive, and death can result from aspiration pneumonia or choking. Parkinsonian crisis, a medical emergency, is usually precipitated by emotional trauma or failure to take the prescribed medications.

A. Assessment

Initially, symptoms are mild and include stiffness or slight hand tremors. They gradually increase and can become disabling.

Bradykinesia: Slowness, stiffness, and difficulty with initiating movement. The patient may have a mask-like facial expression, unblinking stare, difficulty chewing and swallowing,

drooling, and a high-pitched, monotone, weak voice. The patient also has loss of automatic associated movements, such as swinging the arms when walking.

Loss of postural reflexes: Causes the typical shuffling, propulsive gait with short steps; stumbling; falling.

Increased muscles rigidity: Limb muscles become rigid on passive motion. Typically, this rigidity results in jerky (“cogwheel”) motions.

Tremors: Increase when the limb is at rest and stop with voluntary movement and during sleep (non-intention tremor). “Pill-rolling” tremor of the hands and “to and fro” tremor of the head are typical.

Autonomic: Excessive diaphoresis, seborrhea, postural hypotension, and decreased libido.

Physical exam: Usually a positive blink reflex is elicited by tapping a finger between the patient’s eyebrows. A positive palmomental reflex (contraction of muscles of the chin and corner of mouth) can be elicited by stroking patient’s palm. Diminished postural reflexes are present on neurologic exam; however, there is risk of injury with this test because the patient may quickly lose balance and fall.

Parkinsonian crisis: This sudden and severe increase in bradykinesia, rigidity, and tremors can lead to tachycardia, hyperpnea, and inability to swallow or maintain a patent airway.

B. Diagnostic Tests

Diagnosis is usually made on the basis of physical exam and characteristic symptoms, and after other neurologic problems have been ruled out.

1. Urinalysis: May reveal decreased dopamine level, which supports the diagnosis.
2. Medication withdrawal: Long-term therapy with large doses of medications such as halperidol or phenothiazines can produce Parkinson-like symptoms. If caused by these medications, symptoms will disappear when the drug is discontinued.
3. EEG: Often shows abnormalities.

C. Medical Management and Surgical Interventions

1. Pharmacotherapy

- Dopamine replacement, eg, levodopa or levodopa-carbidopa combination: Given in increasing amounts until symptoms are reduced or patient’s tolerance is reached. Side effects include anorexia, nausea, postural hypotension and urinary retention.
 - Vitamin B₆ (pyridoxine) can reverse the effects of levodopa.
 - Antiviral agents, eg, amantadine hydrochloride: Less effective than levodopa, but has less severe side effects. Side effects include dizziness, insomnia, orthostatic hypotension, and ataxia.
 - Anticholinergics, eg, trihexyphenidyl hydrochloride, cycrimine hydrochloride and procyclidine hydrochloride: To help relieve tremors and rigidity. Side effects include dryness of the mouth, blurred vision, constipation, mental dullness, confusion, and urinary retention or overflow incontinence.
2. **Physical therapy:** Massage; muscle stretching; active/passive ROM, especially on hands and feet; and walking and gait–training exercises.
 3. **Treatment for Parkinsonian crisis:** This crisis necessitates respiratory and cardiac support. The patient is placed in a quiet, calm environment with subdued lighting. Sodium Phenobarbital or sodium amobarbital is given IM or IV.

4. **Stereotaxic surgery:** Performed in selected patients. Electrical coagulation, freezing, radioactivity, or ultrasound is used to destroy portions of the globus pallidus of the ventrolateral nucleus of the thalamus to prevent involuntary movement and help relieve tremors and rigidity of the extremities.

3.2.2 Alzheimer's Disease

Alzheimer's disease is a progressive, degenerative disorder of the brain characterized by changes and degeneration of the cerebral cortical nerve cells and nerve endings, resulting in abnormal neurofibrillary tangles and neuritic plaques. This process causes irreversible impairment of memory and degeneration of intellectual functions. Although etiology is unknown, aluminum poisoning, viruses, genetics, auto-immune disease, and neurotransmitter deficiency of choline acetyltransferase are possible causes, of which the last is considered the most probable. The onset of Alzheimer's disease is insidious, and it can strike individuals as young as 40 years of age. The disease progresses to total disability and eventually results in death from problems such as infection or aspiration, usually within 3–15 years.

A. Assessment

The appearance and severity of clinical manifestations vary from individual to individual. Initial indicators are mild, and it may take several years before a definite diagnosis can be made.

Memory: Early short-term memory loss, with longer retention of long-term memory; the past can become “the present.” Eventually, long-term memory is lost as well.

Cognitive process: Inability to think through problems, poor decision-making ability, shortened attention span, lack of insight, inability to perform arithmetic calculations, inability to recognize or name common objects, disorientation to time and place, inability to recognize own reflection in the mirror. Hallucinations also may occur.

Mood: Apathy, lack of initiative, irritability, emotional lability, panic, fear, bewilderment, perplexity, exaggeration of any previous psychotic traits, depression, and anxiety. As the ability to communicate lessens and the world becomes more frightening, the potential for violence and agitation increases. The patient may have catastrophic reactions and emotional outbursts when faced with a complex task.

Social behavior: Decreased ability to handle social interaction, loss of social graces, loss of inhibitions, helplessness, dependency.

Speech patterns: Difficulty finding words, loss of spontaneity in speech, inability to express thoughts, incoherent speech.

Sleep pattern: Restlessness, pacing, decreased need for sleep, nocturnal awakening, reversal of normal sleep pattern.

Self-care: Neglect of routine tasks and personal hygiene; weight loss; increasing inability to dress, bathe, toilet, and feed self or recognize where to urinate or defecate.

Mobility/posture: Stooped and shuffling gait, balance and coordination problems, falling, inability to walk.

Alzheimer's disease progresses through seven distinct stages that correspond with those identified in the Global Deterioration Scale (Table 1).

Table 1 Global Deterioration Scale

Stage	Characteristic	Manifestations
1	Normal mentation	None
2	Forgetfulness	Concern for self-identified memory changes such as forgetting familiar names. No objective demonstration of memory loss.
3	Early confusion	One or more of the following: getting lost in an unfamiliar location others notice a decline in work performance deficit in word and name finding little retention of what has been read difficulty remembering names of new acquaintances loss or misplacement of valuable objects impaired ability to concentrate. Objective demonstration of memory deficit Denies memory and cognitive deficits Mild to moderate anxiety
4.	Late confusion	Deficits in the following areas: current and recent events personal history counting backward in series of numbers traveling and handling finance. Inability to perform complex tasks such as preparing dinner for guests. Strongly denies impairment. Affect becomes blunted Retreats from challenges
5	Early dementia	Needs assistance of others. Memory loss for important information like address and telephone number. Some disorientation to time or place. Difficulty counting backward by 4s or 2s. May have difficulty choosing proper clothing.
6	Middle dementia	May forget name of spouse or others who are significant. Unaware of recent events and experiences. Memory of past sketchy. Unaware of date and surroundings. Difficulty counting backward and sometimes forward in 10s. Requires assistance with activities of daily living. May be incontinent Needs assistance to travel. Confuses day and night. Personality and emotional changes such as: delusional thinking repetition of cleaning anxiety, agitation, violence cannot keep a thought long enough to carry it out.
7.	Late dementia	Loss of verbal ability. Grunting may be evident. Incontinent; requires help with toileting and feeding. Loss of motor skills for walking, sitting, head control, and smiling

Adapted from Reisberg, B., Ferris, S. H., Leon, J. J., & Crook, T. (1982). The global deterioration scale (GDS): An instrument for the assessment of primary degenerative dementia. *American Journal of Psychiatry*, 139, 1136–1139.

B. Diagnostic Tests

Many disorders that can cause a progressive dementia syndrome (eg, head injuries, brain tumors, depression, arteriosclerosis, drug toxicity, and alcoholism) need to be ruled out. This is especially important because some dementias are reversible.

1. Mental status exam: To test orientation, memory, calculation, abstraction, judgement, and mood.
2. Magnetic resonance imaging (MRI):
3. EEG: May reveal slowed brainwave activity.
4. CT scan: May reveal brain atrophy and ventricular enlargement. It also helps rule out other neurologic problems, particularly mass lesions.

C. Medical Management

Generally, the treatment is supportive only.

Pharmacotherapy: Medications, if prescribed, are used to treat behavioral manifestations. These include:

- Tricyclic or other antidepressants such as doxepin hydrochloride and trazodone hydrochloride.
- Antipsychotic agents such as haloperidol: Used for psychiatric patients.
- Tranquilizers or sedatives such as chloral hydrate, triazolam, and diazepam.

Table 2 Drug Therapy for Alzheimer’s Disease

Drug Category/Mechanism of Action	Side Effects	Nursing Consideration
Tacrine (Cognex) Provides for increased levels of acetylcholine in the cortex by inhibiting cholinesterase, the enzyme that breaks down acetylcholine.	Headache, fatigue, confusion, dizziness, nausea, vomiting, diarrhea, gastrointestinal upset, abdominal pain, loss of appetite, skin rashes, hepatotoxicity.	Administer on an empty stomach on an around-the-clock schedule. Do not abruptly discontinue. Arrange for regular blood tests to determine transaminase levels.
Donepezil (Aricept) Inhibits the breakdown of acetylcholine	Nausea, vomiting, diarrhea, bradycardia, possibly worsens asthma and chronic obstructive pulmonary disease.	Inform client and family of side effects and to report any that occur. Tell client to exercise caution if performing tasks that require alertness. Tell client that frequent small meals may minimize gastrointestinal upset. Monitor heart rate and report bradycardia (heart rate < 60).

D. Nursing Management

The major focus of nursing management is to help the client and caregiver in maintaining the highest possible quality of life by supporting mental and physical functions and ensuring safety. Most clients are initially cared for in their homes and a home health nurse can instruct the family about physical care, the disease process, and treatment. He or she also will provide emotional support and intervene if the caregiver becomes overburdened. Risk for Caregiver Role Strain is related to being overwhelmed by responsibilities, fatigue, and depression. The goal of nursing intervention is to have the caregiver feel comfortable and knowledgeable about implementing plans that will provide needed relief. To accomplish this, the nurse can:

- Assess the caregiver's strengths, limitations, and ability to manage caretaking activities.
- Suggest scheduling respite care, brief relief from caretaking responsibilities, with family and friends on a regular rotating basis.
- Provide a list of agencies that offer social services such as the county's commission on aging and society security and medicare agencies.
- Develop a list of people who may be contacted in any emergency, including a 24-hour hotline for the home health nursing agency.
- Recommend that the caregiver and client take care of legal matters such as wills, transferring titles, and preparing an advanced directive..
- Suggest establishing durable power of attorney designating who may make decisions regarding finances or health care when the client becomes incompetent, the legal term for the inability to understand the risks or benefits of decisions.
- Advise the caregiver to obtain guardianship or conservatorship, court-appointed responsibility, for managing the client's care and assets if the client is already incompetent.
- Encourage the caregiver to place the client in a long-term nursing facility while taking a well deserved vacation.
- The nurse should routinely evaluate the caregiver's use of respite care. He or she should seek relief at least 1 or 2 days a week.

3.3 Traumatic Disorders of the nervous Disorders

3.3.1 Spinal Cord Injury

The spinal cord injuries (SCIs) are caused by vertebral fractures and/or dislocations that sever, lacerate, or compress the spinal cord and interrupt neuronal function and transmission of nerve impulses. The spinal cord swells in response to injury, and this, along with hemorrhage, can cause additional compression and compromised function. Neurologic deficits resulting from compression may be reversible if the resulting edema and ischemia do not lead to spinal cord degeneration, common causes of injury include motor vehicle accidents, diving or other sporting accidents, falls, and gunshot wounds.

Prognosis: an SCI does not cause immediate death unless it is at C-1 through C-3, which results in respiratory muscle paralysis. Individuals who survive these injuries require a ventilator for the rest of their lives. If the injury occurs at C-4, respiratory difficulties may result in death, Injuries below C-4 also can be life-threatening because of ascending cord edema, which can cause respiratory muscle paralysis. Other potential complications of SCIs include autonomic dysreflexia, decubitus ulcers, and UTI, any of which can be life-threatening.

A. Assessment

Acute indicators: Loss of sensation, weakness, and/or paralysis below the level of the injury; localized pain or tenderness over the site of injury; headache; hypothermia or hyperthermia; and alterations in bowel and bladder function.

Chronic indicators: Chronic autonomic dysfunction may be manifested as fever, mild hypotension; anidrosis; and alterations in bowel, bladder, and sexual function. Injuries below L-1 or L-2 may result in permanent flaccid paralysis muscle spasticity.

Physical exam

- Acute (spinal shock): Absence of DTRs below level of injury, absence of cremasteric reflex (scratching or light stroking of the inner thigh for male patients causes the testicle on that side to elevate) for T-12 and L-1 injuries; absence of penile reflex.

- Chronic: Generally, increased DTRs occur if the spinal cord lesion is off the upper motor neuron type.

B. Diagnostic Tests

1. X-ray of spine: To delineate fracture, deformity, or displacement of vertebrae, as well as soft tissue masses such as hematomas.
2. Magnetic resonance imaging (MRI): Reveals changes in the spinal cord and surrounding soft tissue.
3. Myelography: Shows blockage or disruption of the spinal canal
4. ARTERIAL BLOOD GASES/pulmonary function tests: To assess effectiveness of respirations.
5. Cysometry: To assess capacity and function of the bladder.
6. CT scan: To reveal changes in the spinal cord, vertebrae, and soft tissue surrounding the spine.

C. Medical Management and Surgical Interventions

Acute care:

1. **Immobilization of injury site:**
2. **Bed rest on a firm surface:** For example, Stryker wedge turning frame or Roto Rest Kinetic Treatment Table.
3. **Pharmacotherapy**
 - Anti-inflammatory agents and corticosteroids such as dexamethasone: To reduce cord edema after the initial injury and minimize ascending cord edema.
 - Osmotic diuretics such as 20% mannitol: Sometimes used for 10 days to reduce cord edema after the initial injury and minimize ascending cord edema.
 - Analgesics and sedatives: To decrease pain and anxiety.
4. **Aggressive respiratory therapy:** For all patients with SCIs. Patient with injuries above C-5 are intubated and put on a ventilator.
5. **Nasogastric decompression during spinal shock phase:** Necessitated by the presence of paralytic ileus.
6. **Bladder decompression during spinal shock phase:** Either intermittent catheterization or paralytic ileus.
7. **Surgery/immobilization:** May include traction, fusion, laminectomy, and closed or open reduction of fractures. The surgical goal is to immobilize the spine and, if indicated, decompress the spinal cord to help prevent additional neurologic deficit. If indicated, bone fragments are removed and the spine is surgically fused within 5–10 days of the injury.
8. **Physical and occupational therapy:** Passive ROM is started on all joints. After the injury is stabilized, an aggressive rehabilitation program is initiated, including muscle-strengthening exercises; conditioning exercises; massage; and instruction in adaptive devices, equipment, and transfer techniques as appropriate. Patients with sacral injuries have the potential to walk and should be instructed in the use of braces, crutches, or a cane as appropriate.
9. **Counseling and psychotherapy:** To help patient and SOs adjust to the disability.

Chronic care:

1. **Pharmacotherapy**
 - Muscle relaxants such as diazepam.
 - Antispasmodics: To decrease spasms.
 - Antibiotics: To prevent bladder infection.

- 2. Dietary management:** Limiting milk and other dairy products to minimize the risk of renal calculi, give juices that leave an acid ash in the urine and decrease urinary pH, which reduces the potential for infection.

3.3.2 Head Injury

Head injuries can cause varying degrees of damages to the skull and/or brain tissue. Primary injuries that result from head injury include skull fracture, concussion, contusion, scalp laceration, brain tissue laceration, and tear or rupture of cerebral vessels. Problems that arise soon after the primary injury include hemorrhage and hematoma formation from the tear or rupture of vessels, ischemia from interrupted blood flow, cerebral swelling and edema, and infection, any of which can interrupt neuronal function.

A. Assessment

Concussion: Mild head injury in which there is temporary neurologic impairment involving loss of consciousness and possible amnesia of the event. After the concussion the patient may have headache, dizziness, nausea, lethargy, and irritability. Although full recovery usually occurs in a few days, a postconcussion syndrome with headaches, dizziness, and lethargy may continue for several weeks.

Contusion: Bruising of the brain tissue producing a long-lasting neurologic deficit than concussion. Traumatic amnesia often occurs, causing loss of memory not only of the trauma, but also of events occurring prior to the incident. Loss of consciousness is common, and it is generally more prolonged than that with concussion. Changes in behavior such as agitation or confusion can last for several hours to days. Headache, nausea, lethargy, motor paralysis, paresis, and possibly seizures can occur as well. Depending on the extent of damage, there is the potential for either full recovery or permanent neurologic deficit such as seizures, paralysis, paresis, or even coma and death.

Skull fracture: Can be closed (simple) or open (compound). Skull fractures are further classified as linear (hairline), comminuted (fragmented, splintered), or depressed (pushed inward toward the brain tissue). A blow forceful enough to fracture the skull is capable of causing significant brain tissue damage, and therefore, close observation is essential. With a penetrating wound or basilar fracture there is the potential for meningitis, encephalitis, brain abscess, cellulites, or osteomyelitis.

Rupture of cerebral blood vessels.

These appear in four forms

Epidural (extradural) hematoma or hemorrhage:

Subdura hematoma or hemorrhage:

Intracerebral hemorrhage

Subarachnoid hemorrhage

- **Epidural hematoma or hemorrhage** involves bleeding between dura mater (outer meninges) and skull. Clinical manifestation are; increased ICP: headache, unilateral pupil dilation (on same side as the lesion), and possibly hemiparesis. Typically, the patient loses consciousness for a short period of time immediately after injury, regains consciousness, and has a lucid period.
- **Subdura hematoma or hemorrhage:** This involves accumulation of venous blood between dura mater (outer meninges) and arachnoid membrane (middle meninges) that is not reabsorbed. This type of hematoma is classified as acute, subacute, or chronic depending on how quickly the signs occur. In acute subdural hematomas, signs appear within 24 hours, resulting from focal neurologic deficit (hemiparesis, pupillary dilation) and increased ICP (headache, decreased LOC). When it occurs 2 – 10 days later, the

hematoma is considered subacute. When indicators occur several weeks or more later, it is considered chronic.

- **Intracerebral hemorrhage:** Involves arterial venous bleeding into the white matter of the brain. Signs include aphasia, hemiparesis, hemiplegia, hemisensory deficits, and loss of consciousness.
- **Subarachnoid hemorrhage:** This is bleeding into the subarachnoid space below the arachnoid membrane (middle meninges) and above the pia mater (inner meninges next to brain). The signs include; a severe headache, vomiting, restlessness, seizures, and loss of consciousness. Signs of meningeal irritation include nuchal rigidity and a positive Kernig's sign.

B. Diagnostic Tests

1. **Skull and cervical spine x-rays:** To locate skull and neck fractures. If the fracture crosses the groove of the meningeal artery, epidural hematoma is likely to be found.
2. **Magnetic resonance imaging (MRI):** To identify the presence of blood in the intracranial area.
3. **CT scan:** To identify any accumulation of blood and/or a shift of midline structure caused by increased ICP.
4. **Brain scan:** To identify hematoma with chronic subdural hematoma. Generally, it is not done for more acute disorders because of the lengthy uptake time of the radioactive isotope.
5. **EEG:** May reveal electrical activity, indicating the presence of a hematoma.

C. Medical Management and Surgical Interventions

1. **Support respirations and other vital functions:** Oxygen therapy delivery, suction, intubation, and ventilation. Initially, patient may have an NG tube for gastric decompression to prevent vomiting and aspiration.
2. **Monitor vital signs/neurologic status:** Baseline assessment is established and patient is monitored frequently for changes.
3. **Bed rest with HOB elevated (or as prescribed):** To promote venous drainage and help reduce cerebral congestion and edema.
4. **Fluid restrictions:** NPO status for 8 – 24 hours (or longer if patient is unresponsive). Then, fluids are limited to decrease cerebral edema. Hypotonic IV solutions such as 5% dextrose in water are contraindicated because they increase cerebral edema.
5. **Diet:** Hyperalimentation/intralipids or progressive diet, depending on patient's LOC and ability to swallow.
6. **Treat secondary complications:** For example, cerebral edema, increased ICP, inappropriate ADH secretion, disseminated intravascular coagulation (DIC), adult respiratory distress syndrome (ARDS), and diabetes insipidus.
7. **Pharmacotherapy:** Note: Narcotics and other medications that alter mentation are contraindicated.
 - Anticonvulsants: Prophylaxis for seizures with or following penetrating wounds. These drugs may include phenytoin, Phenobarbital, and IV diazepam.
 - Glucocorticosteroids such as dexamethasone and osmotic diuretics such as mannitol: To decrease cerebral edema.
 - Antibiotics and tetanus prophylaxis: In the presence of penetrating wounds and basilar fractures.
 - Antipyretics: For fever.
 - Analgesics such as acetaminophen: For pain.
 - Mild sedatives such as chloral hydrate or diphenhydramine: For restlessness.

8. **Hypothermia:** If indicated, a hypothermia blanket is used to reduce body temperature to 87 – 90F and thereby minimize metabolic needs.
9. **Surgical procedures**
 - Suturing: To repair superficial laceration or dural tears.
 - Craniotomy: To remove bone fragment or elevate depressed fractures.

3.4 Nervous System Tumors

3.4.1 Brain Tumors

The abnormal and uncontrolled cell growth of neoplastic or benign tumors can have a wide variety of effects on the brain. Most significant is the disruption of neuronal function caused by infiltration of the tissue, compression of brain tissue and blood vessels, and/or obstruction of normal flow of CSF. Primary brain tumors, composed of nervous system tissue, rarely metastasize outside the CNS. Secondary brain tumors arise from cells that have metastasized from other parts of the body such as the lung, breast, and skin. Although benign tumors tend to be more treatable than neoplastic tumors, they are considered as serious because they are equally capable of increasing ICP, which in turn compromises vital centers.

3.5 Vascular Disorder of the Nervous System

3.5.1 Aneurysm; See vascular disorders

3.5.2 Cerebrovascular Accident

A cerebrovascular accident (CVA) is the sudden disruption of oxygen supply to the nerve cells caused by obstruction or rupture in one or more of the blood vessels that supply the brain. Occlusive CVA, which can be caused by thrombosis or embolism, results in blockage of blood supply to the brain tissue. The resulting ischemia, if prolonged, causes brain tissue necrosis (infarction) as well as cerebral edema and increased ICP.

A. Assessment

General findings:

Classically, symptoms appear on the side of the body opposite that of the damaged site. For example, a CVA in the left hemisphere of the brain will produce symptoms in the right arm and leg. However, when the CVA affects the cranial nerves, the symptoms of cranial nerve deficit will appear on the same side as the site of injury. An obstruction of an anterior cerebral artery may produce bilateral symptoms, as will severe bleeding or multiple emboli. Hemiplegia is fairly common. Initially, the patient usually has flaccid paralysis. As spinal cord depression resolves, more normal tone is seen and hyperactive reflexes occur.

Clinical manifestations:

Vary with the size and site of injury and may improve in 2 – 3 days as the cerebral edema decreases. Headache; neck stiffness and rigidity; vomiting; seizures; dizziness and/or syncope; fever; changes in mentation, including apathy, emotional lability, irritability, disorientation, memory loss, withdrawal, drowsiness, stupor, or coma; bowel and bladder incontinence numbness or loss of sensation; weakness or paralysis on part or one side of the body; and aphasia can occur.

Physical examination: Papilledema, arteriosclerotic retinal changes are seen on ophthalmic examination. A positive Kernig's sign and positive Babinski's sign also may be present.

History of: Rheumatic heart disease, hypertension, atherosclerosis, high serum cholesterol and/or triglycerides, diabetes mellitus, gout, smoking, heart disease, oral contraceptive use, family predisposition for arteriovenous malformation

B. Diagnostic Tests

1. **Magnetic resonance imaging (MRI):** To reveal site of infarction, hematoma, shift of brain structure, and cerebral edema.
2. **CT scan:** To reveal site of infarction, hematoma, and shift of brain structures.
3. **Lumbar puncture and CSF analysis:** May reveal increase in CSF pressure; clear to bloody CSF, depending on the type of stroke; and presence of infection or other nonvascular cause for bleeding.
4. **Cerebral angiography:** To pinpoint site of rupture or occlusion and identify collateral blood circulation, aneurysms,

C. Medical Management and Surgical Interventions

1. **Respiratory support:** Maintenance of airway and delivery of oxygen, as needed.
2. **IV fluids:** To maintain fluid and electrolyte balance.
3. **Bed rest during acute stage:** Activity level is increased as patient's condition improves.
4. **Diet:** NPO if swallow and gag reflexes are diminished or if patient has decreased LOC. A low-sodium and/or low-fat, low-cholesterol diet may be prescribed to minimize other risk factors. Diet may consist of fluids and pureed, soft, or chopped foods, or tube feedings, depending on patient's LOC and ability to chew and swallow.
5. **Pharmacotherapy**
 - **Anticoagulants:** May be used for patients with thrombotic CVAs. Medications include aspirin, heparin sodium, and warfarin sodium to help prevent further thrombosis. If the stroke or neurologic deficit is in evolution (still progressing), anticoagulants may be useful for 24 – 72 hours. Once the stroke is completed and neurologic status is stable, anticoagulants are no longer useful. Anticoagulants are contraindicated with hemorrhagic CVA.
 - **Antihypertensive agents:** To control high BP.
 - **Vasopressors:** To treat low BP.
 - **Corticosteroids (eg, dexamethasone) and osmotic diuretics (eg, mannitol):** To prevent or reduce cerebral edema.
 - **Cimetidine or antacids:** To reduce the risk of GI hemorrhage from gastric ulcer caused by stress or corticosteroid therapy.
 - **Anticonvulsants such as phenytoin or Phenobarbital:** To control and prevent seizures.
 - **Sedatives/tranquilizers:** To promote rest. These are used cautiously to avoid further impairment of neurologic function.
 - **Analgesics such as acetaminophen:** To control headache. If CVA is hemorrhagic, aspirin is avoided because it can cause an increase in bleeding.
 - **Stool softeners:** To prevent straining, which can result in increased ICP.
6. **Physical medicine:** Muscle-strengthening exercises, conditioning exercises and gait training are also frequently prescribed.
7. **ROM:** To maintain or increase joint function and prevent contractures. Exercises may include passive ROM, or active-assistive ROM. Passive ROM is started immediately for all joints.
8. **Speech therapy:** For aphasic patients.
9. **Carotid endarterectomy:** Surgical removal of plaque in the obstructed artery to increase blood supply to the brain.

D. Nursing Diagnoses and Interventions

Sensory-perceptual alterations related to neglect of the affected side secondary to neurologic deficit.

Desired outcome: Patient does not neglect the affected side.

1. Assess patient's ability to recognize objects to the right or left of his or her visual midline; perceive body parts as his or her own; perceive pain, touch, and temperature sensations; judge distances; orient self to changes in the environment; differentiate left from right; maintain posture sense; and identify objects by sight, hearing, or touch. Document deficits.
2. Assess patient for neglect of the affected side as follows:
 - Encourage patient to make a conscious effort to care for neglected body parts and/or check them for proper position to ensure against contractures and skin breakdown.
 - When patient is in bed or up in a chair, provide safety measures such as bedrails and restraints to prevent patient from attempting to get up, which can occur because of unawareness of the affected side.
 - Teach patient to use unaffected arm to perform ROM exercises on the affected side.
3. Arrange the environment to maximize performance of ADLs by keeping necessary objects on patient's unaffected side.

Impaired physical mobility related to alterations in the upper or lower limbs secondary to hemiparesis or hemiplegia.

Desired outcome: Patient can demonstrate techniques that enhance ambulating and transferring.

1. Teach patient methods for turning and moving, using the stronger extremity to move the weaker extremity.
2. Instruct patient always to lead with the stronger side when transferring by maintaining weight on the stronger side and pivoting, using the stronger arm for support.
3. Encourage patient to make a conscious attempt to look at the extremities and check their position before moving. Remind patient to make a conscious effort to lift and then extend the foot when ambulating.
4. Instruct patient with impaired sense of balance to compensate by leaning toward the stronger side.

Impaired verbal communication related to aphasia and/or dysarthria secondary to cerebrovascular insult.

Desired outcome: Patient demonstrates ability for self-expression and two-way communication.

1. Evaluate the nature and severity of the patient's aphasia.
2. Assess patient's ability to point or look toward a specific object, follow simple directions, understand yes/no questions, understand complex questions, repeat both simple and complex words, repeat sentences, name objects that are shown, demonstrate or relate the purpose or action of the object, fulfill written requests, and write requests. Use this assessment as the basis for a communication plan.
3. Obtain a referral to a speech therapist/pathologist as needed.

3.6 Seizure Disorders

Seizures result from an abnormal, uncontrolled, electrical discharge from the neurons of the cerebral cortex in response to a stimulus. If the activity is localized in one portion of the brain the individual will have a partial seizure, but when it is widespread and diffuse a generalized seizure occurs. Symptoms vary widely, depending on the involved area of the cerebral cortex. Although a seizure itself is generally not fatal, instances of prolonged and repeated generalized seizures, status epilepticus, can be life-threatening because exhaustion, respiratory arrest, and cardiovascular collapse can occur. Seizure threshold refers to the amount of stimulation needed to cause the neural activity.

Causes

These include; congenital defects; head injury; intracranial tumors; infections such as meningitis and encephalitis; exposure to toxins such as lead; hypoxia; and metabolic and endocrine disorders such as hypoglycemia, hypocalcemia, uremia, hypoparathyroidism, and excessive hydration. For susceptible individuals, “triggers” may include physical stimulation such as loud music or bright, flashing lights; lack of sleep or food; fatigue; emotional tension or stress; and excessive drug/alcohol use.

A. Assessment

Type of seizures,

Toni-clonic (grand mal): Usually preceded by an aura (visceral sensation, visual disturbances, sounds, or odors preceding the seizure); seizure can last 2–5 minutes and includes the following phases:

- **Tonic (rigid/contracted):** Can last 1 – 2 minutes. Symptoms include loss of consciousness, eyes rolling upward, clenched jaws (potential for tongue to be bitten), apnea (may hear cry as air is forced out of the lungs), cyanosis, and excessive salivation resulting in foaming at the mouth.
- **Clonic (rhythmic contraction and relaxation of the extremities and muscles):** Can last 1–2 minutes. Patient may be incontinent. During this phase, the potential is greatest for biting the tongue.
- **Post-ictal:** Patient may be sleepy, semiconscious, confused, unable to speak clearly, uncoordinated, have a headache, and/or have little recollection of the seizure event.

Absence (petit mal): Patient has momentary loss of awareness, appear to be daydreaming, and ceases voluntary muscle activity. Patient may experience facial, eyelid, or hand twitching. There is usually no memory of the seizure, and the patient may have difficulty reorienting after the seizure event. This type of seizure can last 1–10 seconds and may occur up to 100 times per day.

Myclonic: Sudden, very brief contraction of muscles or muscle groups with no loss of consciousness or post-ictal state.

Status epilepticus: This is a state of continuous or rapidly recurring seizures. This is a medical emergency, resulting in potential complications such as cerebral edema, aspiration, hyperthermia, exhaustion, and respiratory and cardiovascular collapse. Patient may not regain consciousness between seizures. Death may result.

B. Diagnostic Tests

Because a variety of problems can precipitate seizures, testing may be extensive. Common tests include the following:

1. Serum electrolytes: To rule out metabolic causes such as hypoglycemia or hypocalcemia.
2. EEG, both sleeping and awake: May reveal abnormal patterns of electrical activity, particularly with stimuli such as flashing lights.
3. Skull x-rays: To reveal fractures, tumors, calcifications, or congenital anomalies (pineal shift, ventricular deformity).
4. CT scan: May reveal presence of a space-occupying lesion such as a tumor or hematoma.
5. Brain scan: To rule out lesions such as a tumor.
6. Lumbar puncture and CSF analysis: To rule out increased ICP or infection.

C. Medical Management and Surgical Interventions

1. **Anticonvulsants and sedatives:** To help prevent seizure activity.
 - Hydantoin derivatives such as phenytoin, mephenytoin, or ethosuximide: For grand mal seizures.
 - Carbamazepine: For grand mal seizures.
 - Valproic acid: For petit mal seizures.
 - Succinimide derivatives such as ethosuximide: For petit mal seizures.
 - Barbiturate derivatives such as Phenobarbital or primidone: May be used in conjunction with one of the anticonvulsants above.
2. **Treatment of underlying causes:** Such as metabolic disorder or infectious process.
3. **Counseling or psychotherapy:** For patients with poor self-concept or coping difficulties related to the diagnosis.
4. **Surgery:** May include a craniotomy.
5. **Management of status epilepticus**
 - Maintenance of patent airway.
 - Monitor for signs of respiratory depression and hypotension.
 - Assessment of serum glucose and administration of IV glucose if indicated.
 - Slow administration of IV diazepam, 1 mg/min.
 - Administration of thiamine: If alcohol withdrawal occurs.
 - Administration of paraldehyde: May be given if other medications are unsuccessful. : Because the solution reacts negatively with plastic, use a glass syringe for IM and IV routes or a rubber catheter if it is given via retention enema.
 - Intubation and general anesthesia For severe cases.

D. Nursing Diagnoses and Interventions

Potential for injury secondary to seizure activity.

Desired outcomes: Patient does not exhibit signs of oral and/or musculoskeletal injury or airway compromise. SOs can verbalize knowledge of actions that are necessary during seizures activity.

Seizures precautions:

1. Pad siderails with blankets or pillows.
2. Tape an airway or padded tongue blade to the bedside.
3. Avoid using glass or other breakable oral thermometers when taking patient's temperature.

During the seizure:

4. Remain with patient. Observe for, record, and report type, duration, and characteristics of seizure activity and any postseizure response.
5. Prevent or break the fall, and ease patient to the floor if the seizure occurs while he or she is there.
6. If the patient's jaws are clenched, do not force an object between the teeth as this can break teeth or lacerate oral mucous membranes. If able to do so safely and without damage to oral tissue, insert an airway or tongue blade.
7. Protect patient's head from injury during seizure activity. A towel folded flat may be used to cushion the head from striking the ground. Be sure the head's position does not occlude the airway.
8. Do not restrain patient.
9. Roll patient into a side-lying position to promote drainage of secretions and maintain a patent airway.
10. Loosen tight clothing.
11. Reassure and reorient patient after the seizure. Ask patient if an aura preceded the seizure activity; record this information.
12. Provide SOs with verbal and written information for the above interventions.

Knowledge deficit: Life-threatening environmental factors and preventive measures for seizures.

Desired outcomes: Patient can verbalize accurate information regarding measures that may prevent seizures and environmental factors that can be life-threatening in the presence of seizures.

1. Assess patient's knowledge of measures that can prevent seizures and/or environmental hazards that can be life-threatening in the presence of seizure activity. Provide or clarify information as indicated.
2. Advise patient to refrain from operating heavy or dangerous equipment, swimming, and possibly even tub bathing until he or she is seizure-free for the amount of time specified by MD. Teach patient never to swim alone, regardless of the amount of time he or she has been seizure-free.
3. Teach patient that withdrawal from stimulants (eg, caffeine) and depressants (eg, alcohol) can increase the likelihood of seizures and therefore these drugs should be avoided. In addition, depressants can potentiate the effects of the anticonvulsant medication.
4. Teach patient that getting adequate amounts of rest, avoiding physical and emotional stress, and maintaining a nutritious diet may help prevent seizure activity. If stimuli such as flashing lights appear to trigger seizures, advise patient to avoid environments that are likely to have these stimuli.
5. Encourage patient to wear a Medic-Alert bracelet or stimuli identification.

Knowledge deficit: Purpose, precautions, and side effects of anticonvulsant medications.

Desired outcome: Patient can verbalize accurate information regarding the prescribed anticonvulsant medication.

1. Stress the importance of taking the prescribed medication regularly and on schedule
2. Explain that anticonvulsants may make people drowsy.
3. Advise patient to avoid activities that require alertness until his or her CNS response to the medication has been determined.
4. Caution patients who are taking phenytoin that there are two types of this drug. Dilantin Kapseal is absorbed more slowly and is longer acting. It is important not to confuse this extended-release phenytoin with prompt-release phenytoin. Doing so may cause dangerous underdosage or overdosage. Generic phenytoin should not be substituted for Dilantin Kapseal.
5. Instruct patient to report any significant weight gain or weight loss because it may necessitate a change in dosage or scheduling.

Noncompliance with the therapy related to frustration secondary to negative side effects of anticonvulsant medications and difficulty with making necessary lifestyle changes, and/or denial of the illness.

Desired outcome: Patient can verbalize knowledge and feelings about the disease process and treatment plan.

1. Assess patient's understanding of the disease process, medical management, and treatment plan. Explain or clarify information as indicated.
2. Assess for causes of noncompliance, such as medication side effects and/or difficulty with making significant lifestyle changes or with following the medication schedule.
3. Evaluate patient's perception of his or her vulnerability to the disease process, and be alert to signs of denial of the illness. Determine if a value, cultural, or spiritual conflict is causing noncompliance.

Table 2 Common Anticonvulsants

Name	Seizure Type	Side Effects	Precautions
Phenytoin (Dilantin)	Grand mal	Mental, dullness, ataxia, diplopia, gingival hypertrophy, nystagmus, nausea, vomiting and increased body hair	Ensure frequent oral hygiene. Take drug with food or large amounts of liquid
Carbamazepine (Tegretol)	Grand mal	Blood dyscrasias, ataxia, rash, diplopia, nausea, vomiting and liver damage	Check CBC frequently. Patient should report fever, mouth ulcers, sore throat, bruising, or bleeding immediately. Take drug with food.
Mephenytoin (Mesantoin)	Grand mal	Blood dyscrasias, rash, drowsiness, ataxia, diplopia,	Check CBC frequently. Patient should report fever,

		gastric distress, and gingival hyperplasia	mouth ulcers, sore throat, bruising, or bleeding immediately. Take drug with food. Good oral hygiene, gum massage, and gentle flossing are important
Phenobarbital (Luminal)	All types, generally in combination with other medications	Drowsiness and lethargy	Do not stop abruptly as this may cause withdrawal seizures
Primidone (Mysoline)	Grand mal, generally in combination with other drugs	Drowsiness, emotional changes including depression, irritability, ataxia, decreased muscle coordination, nausea, vomiting and impotence	Do not stop abruptly as it may cause withdrawal seizures. Take with food or large amounts of fluid
Ethosuximide (Zarantin)	Petit mal	Gastric distress, nausea, vomiting, dizziness, drowsiness	Take with food or large amounts of fluid
Valproic acid (Depakene)	Petit mal	Sedation, dizziness, nausea/vomiting, anorexia and liver damage	Do not chew as it may irritate mucous membranes. Take with food. Monitor liver function through periodic lab tests. May produce false-positive test for ketones in the urine.

4.0 Conclusion

The primary aims of medical treatment of a neurologic deficit involves the use of drugs and various types of therapies to treat the original disorder, prevent and treat complications, and restore maximum sensory and motor functions. Surgery may be necessary to rectify or treat the original cause or a complication of the deficit.

5.0 Summary

Nursing management of a client with a neurologic deficit includes providing skin care to prevent breakdown and infection; assisting with ROM exercises; promoting regular bowel movements, bladder emptying, physical activity, and sexual health; and preventing injury.

6.0 Tutor Marked Assignment

- State the pathophysiology of Alzheimers disease
- Identify the causes of epilepsy.
- Discuss the nursing management for a client with epilepsy

7.0 Further Reading and Other Resources

Suzanne C. Smeltzer, Brenda Bare (2004). Medical Surgical Nursing. Lippincott Williams & Wilkins

Ethelwynn L. Stellenbery, Juditt C. Bruce (2007). Nursing Practice: Medical-Surgical Nursing for Hospital and Community. Elsevier Edinburgh

UNIT III: CARE OF CLIENTS WITH ENDOCRINE DISORDERS

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1.0 Introduction

This unit focuses on the care of clients with endocrine disorders

2.0 Objective

On completion of this unit you will be able to:

1. Define and distinguish the two types of diabetes mellitus.
2. Identify the three classic symptoms of diabetes mellitus.
4. List common complications of diabetes mellitus.
5. Differentiate between the symptoms of hypoglycemia and hyperglycemia.
6. Discuss the nursing management of the client with diabetes mellitus.

3.0 Main Contents

Diabetes mellitus

Diabetic coma

Glycosuria

Hyperglycemia

Polydipsia

Polyphagia

3.1 Disorders of the Thyroid Gland

The thyroid gland produces three hormones: thyroxine (T_4), triiodothyronine (T_3), and thyrocalcitonin (calcitonin). Secretion of T_3 and T_4 is regulated by the anterior pituitary gland via a negative feedback mechanism. When serum T_3 and T_4 levels decrease, thyroid-stimulating hormone (TSH) is released by the anterior pituitary. This stimulates the thyroid gland to secrete more hormones until normal levels are reached. T_3 and T_4 affect all body systems by regulating overall body metabolism, energy production, and fluid and electrolyte balance and controlling tissue utilization of fats, proteins, and carbohydrates. Thyrocalcitonin inhibits mobilization of calcium from bone and reduces blood calcium levels.

3.1.1 Hyperthyroidism

Hyperthyroidism is a clinical syndrome caused by excessive circulating thyroid hormone. Because thyroid activity affects all body systems, excessive thyroid hormone exaggerates normal body functions and produces a hypermetabolic state. Family history of hyperthyroidism is a significant factor for development of this disorder. Hyperthyroidism also can be caused by nodular toxic goiters in which one or more thyroid adenomas hyperfunction autonomously.

Graves' disease (diffuse toxic goiter) accounts for approximately 85% of reported cases of hyperthyroidism. It is characterized by spontaneous exacerbations and remissions that appear to be unaffected by therapy. The cause of Graves' disease is unknown.

The most severe form of hyperthyroidism is **thyrotoxic crisis or thyroid storm**, which results from a sudden surge of large amounts of thyroid hormones into the bloodstream, causing an even greater increase in body metabolism. This is medical emergency. Precipitating factors include infection, trauma, and emotional stress, all of which place greater demands on body metabolism. Thyrotoxic crisis also can occur following subtotal thyroidectomy because of manipulation of the gland during surgery. Despite vigorous treatment, thyroid storm cause death in approximately 20% of affected patients.

A. Assessment

Clinical manifestations: Oligomenorrhea or amenorrhea; increased appetite with weight loss; diarrhea or frequent defecation; increased perspiration, especially on the palms of the hands; hyperglycemia; and generalized muscle weakness. In addition, heat intolerance, anxiety, excitability, restlessness, tremors, insomnia, and atrial fibrillation with congestive heart failure (CHF) are sometimes noted.

Physical exam: Tachycardia with irregular, bounding pulse; increased respirations; elevated temperature; enlargement of the thyroid gland 2–4 times greater than normal; bruit with auscultation over gland; atrophy of skeletal muscles; enlargement of the thymus and lymph nodes; hyperreflexia; and gynecomastia in males. In addition, these patients are usually thin and hyperkinetic with warm, moist skin and fine, silky hair that will not curl.

Thyrotoxic crisis (thyroid storm): Severe tachycardia, high temperature, CNS irritability, and delirium.

B. Diagnostic Tests

1. Serum thyroxine and triiodothyronine tests: Will show elevation of T₃ and T₄ in the presence of disease.
2. Thyroid stimulating hormone (TSH) test: Decreased in the presence of disease.
3. Free thyroxine index (FTI): Elevated in the presence of disease.
4. Thyroid scanning: Uses radionuclear scanning to determine function of thyroid gland and presence of nodules.

C. Medical Management and Surgical Interventions

1. Pharmacotherapy

- Propranolol (beta-adrenergic blocking agent): To relieve tachycardia, anxiety, and heat intolerance. It is usually contraindicated in patients with CHF
 - Antithyroid agents (thioamides), including prophyllthiouracil and methimazole: To prevent the synthesis and release of thyroxine. Patients are continued on thioamides for a period that ranges from 6 months to several years.
 - Iodides: May be given in conjunction with thioamides to inhibit thyroid hormone release. Adverse reactions include skin rash and fever.
 - Barbiturates and tranquilizers: To minimize anxiety and promote rest.
 - Antidiarrheals: To decrease peristalsis and increase absorption of nutrients from the GI tract.
2. **Diet:** High in calories, protein, carbohydrates, and vitamins to restore a normal nutritional state.
 3. **Subtotal thyroidectomy:** Surgical removal of part of the gland. To minimize postoperative complications, the patient is prepared with antithyroid (thioamide) agents for approximately 6 weeks until euthyroid. Nutritional status is maximized and approximately 2 weeks before surgery the patient is started on iodides to decrease vascularity of the gland and make it firmer, which facilitates removal. The most frequent postoperative complication is hemorrhage at the operative site. Damage to the laryngeal nerve occurs in 1–4% of cases.

3.1.2 Hypothyroidism

Hypothyroidism is a condition in which there is an inadequate amount of circulating thyroid hormone, which causes a decrease in metabolic rate that affects all body systems.

Causes

- Pathologic changes in the thyroiditis, thyroid atrophy
- It can also be caused by pituitary tumors, postpartum necrosis of the pituitary gland, or hypophysectomy.
- Hypothalamic deficiency in the release of thyrotropin-releasing hormone (TRH).

Myxedema occurs when hypothyroidism is untreated. It is a life-threatening condition. The clinical picture of myxedema coma is that of exaggerated hypothyroidism, with dangerous

hypoventilation, hypothermia, hypotension, and shock. Coma and seizures can occur as well. Myxedema coma usually develops slowly, has a >50% mortality rate, and requires prompt and aggressive treatment.

A. Assessment

Clinical manifestations can progress from mild early in onset to life-threatening.

Clinical manifestations: Early fatigue, weight gain, anorexia, constipation, menstrual irregularities, muscle cramps, lethargy, inability to concentrate, hair loss, cold intolerance, and hoarseness. Some usually placid patients may become depressed or extremely agitated.

Physical exam: Possible presence of goiter, cardiomegaly, bradycardia, hypothermia, peripheral nonpitting edema, and periorbital puffiness. Patients are often obese with cool, dry, yellowish skin. The voice is hoarse and the speech is frequently slurred. The hair is thin, coarse, and brittle and the tongue is enlarged. Slow mentation and reflexes are usually present.

B. Diagnostic Tests

1. **Serum tests:** TSH will be elevated in the presence of primary hypothyroidism and low or normal in other forms of the disease. This is the most significant test for differentiating primary from secondary or tertiary hypothyroidism. Serum T₃ and T₄ will be decreased.
2. **Iodine-131 uptake:** Will be less than 10% in 24 hours. In secondary hypothyroidism, uptake increases with administration of exogenous TSH.
3. **Thyroid antibody tests:** Positive in primary hypothyroidism.

C. Medical Management

1. **Oral thyroid hormone:** Given early in treatment for primary hypothyroidism. To prevent hyperthyroidism caused by too much exogenous thyroid hormone, patients are started on low doses that are increased gradually, based on serial laboratory tests (T₃ and T₄) and the patient's response to medication. This therapy is continued for the patient's lifetime. For patients with secondary hypothyroidism, thyroid supplements can promote acute symptoms and are therefore contraindicated.
2. **Stool softeners:** To minimize constipation secondary to decreased gastric secretions and peristalsis.
3. **Diet:** High in roughage and protein to help prevent constipation; restriction of sodium to decrease edema; and reduction in calories to promote weight loss.

Treatment of myxedema coma:

1. **IV thyroid supplements:** Rapid IV administration of thyroid hormone can precipitate hyperadrenocorticism, but can be avoided by the concomitant administration of IV hydrocortisone.
2. **Intubation and mechanical ventilation:** To compensate for decreased ventilatory drive.
3. **Treat hyponatremia:** Restrict fluids and/or administer hypertonic (3%) saline.
4. **Treat associated illnesses such as infections.**
5. **Treat hypotension:** Administer IV isotonic fluids such as normal saline and lactated Ringer's solution. Hypotonic solutions such as 5% dextrose in water (D₅W) are contraindicated because they can further decrease serum sodium levels. Because of altered metabolism, these patients respond poorly to vasopressors.
6. **Treat hypoglycemia:** Administer IV glucose.

3.2 Disorders of the Parathyroid Glands

The parathyroid glands regulate serum calcium and phosphorus levels via release of parathyroid hormone (PTH). This is accomplished by a negative-feedback mechanism: When serum calcium levels rise, PTH secretions is suppressed. PTH acts on bone to decrease calcium binding, and it stimulates the kidneys to increase reabsorption of calcium and excretion of phosphate. PTH is also involved in the synthesis of a renal enzyme that catalyzes the formation of vitamin D, which in conjunction with PTH, increases absorption of calcium from the GI tract.

3.2.1 Hyperparathyroidism

Hyperparathyroidism is a clinical syndrome in which there is excessive secretion of PTH. Primary hyperparathyroidism is caused by pathology of one or more of the parathyroid glands. Eighty percent of these cases are caused by a benign adenoma of one gland, another 10% by hyperplasia of all four glands, and in rare cases, by carcinoma. In this disorder, excessive PTH acts on the skeletal, renal, and GI systems, and the overall effect is that of increased serum calcium and decreased phosphate. It is the second most common cause of hypercalcemia. Secondary hyperparathyroidism is usually caused by renal insufficiency with decreased glomerular filtration. The overall effect is that of decreased calcium and increased phosphate levels. Tertiary hyperparathyroidism occurs when secondary hyperparathyroidism progresses to a state in which excessive PTH is released independent of serum calcium levels.

A. Assessment

Clinical manifestations: Muscular weakness, fatigue, personality disturbances, emotional lability, constipation, weight loss, renal calculi, nausea, vomiting, anorexia, polyuria, hematuria, drowsiness, stupor, and coma. In addition, the patient may have kidney infections, anemia, arthralgia, pancreatitis, peptic ulcers, and pathologic fractures, as heart disease caused by calcium deposits in in the tissues.

Physical exam: Hypotonic muscles, enlarged parathyroid glands, hypertension. If the conditions is severe, renal failure also may be present.

B. Diagnostic Tests

1. Serum calcium: Elevated in primary hyperparathyroidism and low in secondary hyperparathyroidism.
2. Serum PTH: High or inappropriately high for serum calcium levels.
3. Plasma phosphorus: Decreased in primary hyperparathyroidism and elevated in secondary hyperparathyroidism.
4. 24-hour urine calcium: Elevated in primary hyperparathyroidsim. This test is often used to rule out other causes of hypercalcemia.
5. Skeletal x-rays: Will show diminution of bone mass in virtually all patients with hyperparathyroidism, as well as calcification of articular cartilage. X-rays of the hands will show subperiosteal resorption of the phalanges.
6. EKG: May show shortened Q–T interval, which is reflective of hypercalcemia.

C. Medical Management and Surgical Interventions

Surgical treatment for primary hyperparathyroidism: The most effective form of treatment for primary hyperparathyroidism is the surgical removal of one or more of the parathyroid glands (parathyroidectomy). In addition to the postoperative complications potentially found with a thyroidectomy, abnormalities in serum calcium levels also may be found.

Medical treatments for primary hyperparathyroidism: Reserved for patients who are prone to surgical risks and the goals of treatment are to provide adequate hydration and reduce serum calcium levels.

1. **Promote calcium excretion:** This is accomplished by forcing fluids orally, or providing IV normal saline for patients who are stuporous or nauseate. Volumes up to 1000 mL/h may be given for short periods.
2. **Increase salt intake:** Either via diet or salt tablets. Because sodium competes with calcium for excretion by the kidneys, increased sodium levels will cause the kidneys to excrete more calcium.
3. **Diet:** Limit dietary calcium such as milk, many cheeses, cottage cheese, to one serving per day.
4. **Pharmacotherapy**
 - Diuretics: To prevent volume overload Thiazide diuretics are contraindicated because they decrease calcium excretion.
 - Oral phosphate supplements: To decrease bone reabsorption of calcium and bind calcium in the intestine to limit calcium absorption.
 - IV mithramycin: To inhibit bone reabsorption and lower serum and urine calcium levels. This is the drug of choice for treatment of severe hypercalcemia because it is more effective and works more rapidly than calcitonin.
 - Oral steroids: For their calciuric effect and to decrease calcium absorption in the presence of vitamin D intoxication.
 - **Oral calcium supplements:** To increase serum levels.
 - **Aluminum-containing antacids (eg, AlternaGel, Amphojel):** For patients with chronic renal failure to bind phosphorus in the intestine and prevent resorption.
 - **Oral vitamin D supplements:** To correct deficiency.

3.2.2 Hypoparathyroidism

Hypoparathyroidism is a condition in which there is decreased production of parathyroid hormone (PTH). Most commonly, this disorder is iatrogenic, caused by damage to or accidental removal of the parathyroid glands during thyroid surgery or radioactive iodine treatment for hyperthyroidism. Damage can be temporary or permanent. If injury occurs in the absence of gland removal, the tissue generally recovers within a period of months and returns to normal function. Familial or auto-immune factors also can be significant in the development of hypoparathyroidism. Because the function of PTH is the regulation of serum calcium, the response to hypocalcemia.

A. Assessment

Clinical manifestations: Numbness and tingling around the mouth, fingertips, and sometimes in the feet; painful contractions or twitching of skeletal muscles; clonic and tonic spasms; grand mal seizures; laryngeal spasms; carpopedal spasm; nausea; vomiting; dysrhythmias; heart failure; cataracts (from calcium deposits); conjunctivitis; and photophobia.

Physical exam: Dry, scaly skin with increased pigmentation; thinning of scalp hair; loss of hair in axilla and pubic areas, eyebrows, and eyelashes; brittle fingernails and toenails, which may be deformed with horizontal ridges; and positive Chvostek's and Trousseau's signs.

B. Diagnostic Tests

1. Serum tests: Levels of calcium will be decreased; phosphate will be increased; and plasma PTH will be decreased.
2. Skull and skeletal x-rays: May show evidence of increased density and calcification of basal ganglia.

C. Medical Management

1. **Calcium supplements:** For cases of severe hypocalcemia, given either orally (route of choice) or IV.

2. **Parathyroid hormone injections:** To replace lost PTH.
3. **Vitamin D preparations:** To facilitate absorption of calcium from the GI tract.
4. **Sedatives (Phenobarbital) and magnesium sulfate:** To minimize tetany and seizures. **Phenytoin** may be given to control seizures.
5. **Aluminium hydroxide gels (eg, AlternaGel, Amphojel):** To bind phosphorus in the intestines and decrease serum phosphate levels.
6. **Diet:** High in calcium (1 quart milk/day) and low in phosphorus (limit meat, poultry, fish, eggs, cheese, dried beans, and cereals). If hyperphosphatemia persists, it may be necessary to restrict dairy products and egg yolks and provide oral calcium supplements. Foods high in oxalate, which binds to calcium, also should be avoided. These include beets, figs, nuts, spinach, black tea, and chocolate.

3.3 Disorders of the Adrenal Gland

Each of the two adrenal glands is composed of two distinct parts: the adrenal cortex and the medulla. Adrenocortical hormones include glucocorticoids (cortisol is the primary glucocorticoid), which are responsible for regulation of protein, fats, and carbohydrate metabolism and affect the immunologic and inflammatory responses; mineralocorticoids (aldosterone), which affect salt and water metabolism; and androgens, which affect sexual development. These hormones are released in response to serum levels of adrenocorticotropic hormone (ACTH), which functions via a negative-feedback mechanism: When serum cortisol levels decrease, ACTH release increases. The medulla secretes the catecholamines epinephrine and norepinephrine, which are released in response to sympathetic nervous system stimulation.

3.3.1 Addison's Disease

Addison's disease is a deficiency of adrenocortical hormones following destruction of the adrenal cortex, which can occur suddenly as a result of stress such as trauma, infection, or surgery. Eighty percent of reported cases involve an autoimmune factor.

A. Assessment

Clinical manifestations: Apprehension, headache, nausea, anorexia, abdominal pain, diarrhea, confusion, and restlessness. In addition, patients may have muscular weakness that becomes progressively worse throughout the day, fatigue, weight loss, postural hypotension, and emotional instability.

Physical exam: Possible presence of cyanosis, fever, pallor, weak pulse, and tachypnea. Often, there is emaciation with dehydration, generalized dark pigmentation of the skin with brown or black freckles, hypotension, and a small heart size.

Addisonian crisis: Headache, nausea, vomiting, fever, intractable abdominal pain, and severe hypotension, which can lead to vascular collapse and shock.

History of: Familial tendency, bilateral adrenalectomy, tuberculosis, any kind of trauma or infection, damage to the pituitary gland.

B. Diagnostic Tests

1. **Blood cortisol levels:** Drawn at 8:00a.m and 12 midnight. In normal individuals, blood cortisol levels are highest in the morning and gradually decrease until they reach their lowest level at 12 midnight. Patients with Addison's disease do not show this variation.
2. **Other blood studies:** Will reveal elevated potassium and BUN; decreased plasma aldosterone, sodium, and chloride levels; and decreased blood sugar. Serum ACTH will be increased in primary Addison's disease and decreased in secondary Addison's disease.

3. Urine sodium: Increased.

C. Medical Management and Surgical Interventions

1. Pharmacotherapy

- Antibiotics or antituberculosis therapy: If infection or tuberculosis is the cause.
- Maintenance doses of mineralocorticoids; oral supplementary sodium; and oral corticosteroids: Patients must take hormone replacements for life.

2. **Diet**: High in calories, carbohydrates, proteins, and vitamins, provided in small, frequent feedings to enhance nutritional state for these patients, who tend to be anorexic.

3. **Replace fluids**: To correct severe dehydration. 1–2 L D₅NS is given over a brief period of time, eg, 2 hours.

4. **Hydrocortisone sodium succinate**: To replace decreased cortisol. It is given 100 mg IV immediately and then q6h via infusion drip.

5. **Vasopressors**: To maintain adequate BP.

6. **Continuous cardiac monitoring**: For prompt identification of life-threatening dysrhythmias.

3.3.2 Cushing's Disease

Cushing's disease is a spectrum of symptoms associated with prolonged elevated plasma concentration of adrenal steroids.

Pathophysiology

Basically, there are two mechanisms by which this can occur. In normal individuals, the pituitary gland secretes adrenocorticotropic hormone (ACTH), which stimulates the adrenal glands to release the adrenal steroid hormones. This is regulated by a negative-feedback mechanism in which increasing levels of plasma cortisol suppress ACTH. In cases of pituitary pathology, this mechanism does not function and the pituitary gland continues to secrete excessive amounts of ACTH, with resultant abnormally high levels of adrenocorticoid hormones. This accounts for approximately 70% of reported cases and is termed Cushing's disease. Cushing's syndrome, on the other hand, is caused by pathology of the adrenal glands themselves, from ectopic ACTH-secreting tumors or from iatrogenic causes such as excessive ingestion of cortisol or ACTH.

A. Assessment

Clinical manifestations: Weight gain; muscle weakness; kyphosis and back pain; generalized osteoporosis, especially in the vertebrae; pathologic fractures of the long bones; mental and emotional disturbances; easy bruising; arteriosclerotic changes in the heart, brain, and kidney; renal calculi; thirst and polyuria; menstrual changes; and impotence.

Physical exam: Patients exhibit "central obesity" with pendulous abdomens and thin legs and arms; moon face; fat deposits on the neck and supraclavicular area (buffalo obesity); edema; hypertension; and thin, transparent skin with multiple ecchymoses. Androgen excess is most noticeable in females, as evidenced by changes in menstruation, as well as virilism and hirsutism. Patients frequently have stretch marks with red and purple striae showing through the stretched skin. Patients with Cushing's syndrome have hyperpigmentation of facial skin secondary to ectopic ACTH-secreting tumors.

History of: Excessive exogenous steroid ingestion, pituitary tumor.

B. Diagnostic Tests

1. **Blood cortisol:** Elevated at least part of the day. Normally, cortisol levels fluctuate throughout the day, with the highest level in the morning and a gradual decline until the lowest level is reached about midnight.
2. **Serum ACTH:** Measured in the same way as blood cortisol and will be abnormally high at least part of the day.
3. **24-hour urinary free cortisol:** Will be elevated. Cushing's syndrome is confirmed if the test is elevated in a patient with no cortisol suppression.
4. **High-dose dexamethasone test:** Patient is given 8 mg dexamethasone orally over a 24-hour period. A 24-hour urine analysis for 17-hydroxycorticosteroid (17-OHCS) is obtained and the results are interpreted as follows:-
 - High ACTH with no 17-OHCS suppression indicates an ectopic source of ACTH production.
 - Low ACTH with no 17-OHCS suppression indicates an adrenal tumor.
 - Normal to high ACTH with suppression of 17-OHCS indicates Cushing's disease.

C. Medical Management and Surgical Interventions

1. **Adrenocortical inhibitors:** are given to prevent hypocortisolism.
2. **Irradiation of the pituitary gland:** To decrease pituitary production of ACTH.
3. **Diet:** Low in calories and carbohydrates to reduce hyperglycemia. Salt is restricted to reduce BP; and foods high in potassium, including bananas, apricots, figs, dried peaches and prunes, oranges, and tomatoes are given to raise serum potassium levels.
4. **Transphenoidal hypophysectomy:** May be performed if the pathology involves the pituitary gland.

3.4 Disorders of the Pituitary Gland

The pituitary (hypophysis) is composed of two lobes: the anterior pituitary (adenohypophysis) and posterior pituitary (neurohypophysis). The anterior lobe is larger and its secretory activities are controlled by tropic hormones produced by and transmitted from the hypothalamus in response to negative feedback mechanisms. It secretes seven of the nine pituitary hormones. These include (1) adrenocorticotropic hormone (ACTH), which stimulates adrenal cortical growth and secretion of adrenocortical hormones; (2) thyrotropic hormone (TSH), which stimulates thyroid growth and secretion of thyroid hormones; (3) follicle-stimulating hormone (FSH), which stimulates ovulation in females and the production of sperm in males; (4) luteinizing hormone (LH), called the interstitial cell-stimulating hormone (ICSH) in males, in whom it stimulates production of testosterone, and in females stimulates ovulation and development of ovarian follicles; (5) melanocyte-stimulating hormone (MSH), which causes pigmentation; (6) luteotropic hormone (LTH), also called prolactin, which stimulates secretion of milk in females; and (7) growth hormone (GH) or somatotrophic hormone (STH), which accelerates body growth.

Posterior pituitary secretion is regulated by nerve impulses originating in the hypothalamus in response to stimuli from other parts of the body. It produces two hormones: antidiuretic hormone (ADH) or vasopressin, which acts on the renal tubules to increase reabsorption of water; and oxytocin, which stimulates milk "letdown" and contraction of the uterus.

3.4.1 Diabetes Insipidus

Diabetes insipidus results from a defect in the release or synthesis of ADH from the hypothalamus, or a defect in renal tubular response to ADH causing impaired renal conservation of water. The onset is usually insidious, with progressively increasing polydipsia and polyuria, but it can develop rapidly following an injury or infectious disease. Depending on the degree of injury, the condition can be either temporary or permanent.

There are three phases associated with diabetes insipidus. The first phase of polydipsia and polyuria immediately follows the injury and lasts 4 – 5 days. In the second phase, which lasts about 6 days, the symptoms disappear. At the third phase, the patient experiences permanent polydipsia and polyuria. The chief danger to these patients is dehydration from the inability to take in adequate fluids to balance the excessive output of urine.

A. Assessment

Clinical manifestations: Polydipsia, polyuria with dilute urine.

Physical exam: Usually within normal limits; however, patient may show signs of dehydration if fluid intake is inadequate.

History of: Cranial injury, especially basilar skull fracture; meningitis; primary or metastatic brain tumor; surgery in the pituitary area; cerebral hemorrhage; encephalitis; syphilis; or tuberculosis.

B. Diagnostic Tests

1. **Urine osmolality:** Decreased (50–200 mOsm/kg) in the presence of disease.
2. **Specific gravity:** Decreased (1.000 – 1.005) in the presence of disease.
3. **Serum osmolality:** Increased in the presence of disease.
4. **Water deprivation test:** Baseline measurements of body weight, serum and urine osmolalities, and urine specific gravity are obtained. Fluids are not permitted and the above measurements are repeated hourly. The test is terminated when urine specific gravity exceeds 1.020 and osmolality exceeds 800 mOsm/kg, a normal response; or when 5% of body weight is lost. Because the most serious side effects of this test is severe dehydration, the test should be performed early in the day so the patient can be more closely monitored.

C. Medical Management

1. **Administer exogenous vasopressin (Pitressin):** Replacement therapy for ADH. Potential side effects include hypertension secondary to vasoconstriction, myocardial infarction secondary to constriction of coronary vessels, uterine cramps, and increased peristalsis of the GI tract.
2. **Achieve a mild antidiuretic effect:** For example, with chlorpropamide.

3.5 Disorders of the Pancreas

The pancreas serves both exocrine (nonhormonal) and endocrine functions. The exocrine portion comprises 98% of tissue mass. Its function is the secretion of potent enzymes that act to reduce proteins, fats, and carbohydrates into simpler chemical substances. Pancreatic lipase acts on fats to produce glycerides, fatty acids, and glycerol; pancreatic amylase acts on starch to produce disaccharides. The pancreas also secretes sodium bicarbonate to neutralize the strongly acidic gastric contents as they enter the duodenum.

3.5.1. Pancreatitis

Pancreatitis can involve edema, hemorrhage, or necrosis of the pancreas and its blood supply. It is characterized by varying degrees of pancreatic insufficiency, which results in decreased production of enzymes and bicarbonate and malabsorption of fats and proteins. The digestion of fat is affected most severely. As a result, a high fat content in the bowel stimulates water and electrolyte secretion, which produces diarrhea. The action of bacteria on fecal fat produces flatus, steatorrhea, and abdominal cramps. Autodigestion, the activation of pancreatic enzymes

within the pancreas, is the pathologic process in pancreatitis. It destroys pancreatic tissues, causes vascular permeability, and results in edema and pain. Although the cause of this process is unknown, the backflow of pancreatic secretions into the biliary and pancreatic ducts is a response to high pressure within the ducts, such as that caused by a gallstone or cancer of the pancreatic head. Diabetes mellitus often occurs as a result of chronic pancreatitis because of damage to the beta cells, which results in alterations in insulin production.

A. Assessment

Acute pancreatitis: Sudden onset of severe epigastric pain following a large meal or alcohol intake. The pain radiates to the back and is unrelieved by vomiting. The patient also may have persistent vomiting, extreme malaise, restlessness, cold and sweaty extremities, dehydration, left pleural effusion, adult respiratory distress syndrome (ARDS), and jaundice.

Physical examination: Diminished or absent bowel sounds, suggesting presence of ileus; crackles (rales) at the lung bases related to persistent hypoventilation associated with splinting and guarding with pain. In addition, edema in and around the pancreas impinges on the diaphragm and prevents full expansion of the lungs with inspiration. Patients also may have low-grade fever (100 – 102F) and an abdominal mass.

Chronic pancreatitis: Constant, dull epigastric pain; steatorrhea (foamy, foul-smelling stools) resulting from malabsorption of fats and protein; severe weight loss; and onset of symptoms of diabetes mellitus: polydipsia, polyuria, polyphagia. In addition, chemical addiction is often seen because of the chronic pain.

History of: Biliary tract disease, chronic excessive alcohol consumption, duodenal ulcer, coxsackie virus, mumps, hypothermia, and use of estrogen –containing oral contraceptive, glucocorticoids, sulfonamides, chlorothiazides, and azothioprine.

B. Diagnostic Tests

1. **Serum amylase:** When significantly elevated (>500 U/100 mL), rules out acute abdomen conditions such as cholecystitis, appendicitis, bowel infarction/obstruction, and perforated peptic ulcer.
2. **Serum calcium and magnesium:** Levels may be lower than normal. On EKG, hypocalcemia is evidenced by prolonged QT segment with a normal T wave.
3. **CBC:** Elevated WBCs and polymorphonuclear (PMN) bodies if bacterial peritonitis is present secondary to duodenal rupture.
4. **Urinalysis:** May show presence of glycosuria, which may signal the onset of diabetes mellitus. Elevated urine amylase levels are useful diagnostically when serum levels have dropped off. An elevated specific gravity reflects the presence of dehydration.

C. Medical Management and Surgical Interventions

Medical goals are to reduce stimuli for pancreatic secretion and rehydrate with fluids.

For acute pancreatitis:

1. **Fluid and electrolyte replacement:** To maintain adequate circulating blood volume.
2. **Bed rest:** To reduce metabolic demands on the body and thereby minimize need for pancreatic activity.
3. **Pharmacotherapy:** Meperidine for pain. **Note:** Both morphine and meperidine may cause spasms at the sphincter of Oddi. Atropine is often given to prevent this from occurring. Broad spectrum antibiotics: For infection, if present. Steroids: To reduce

inflammation. Anticholinergics: To impede impulses that stimulates pancreatic secretions.

4. **NPO status and NG suction:** To decrease stimulus for pancreatic secretions and alleviate pressure in the GI tract.
5. **Rule out underlying factors such as hyperparathyroidism and hyperlipoproteinemia:** Can contribute to the development of pancreatitis.
6. **Surgery:** May be performed for biliary pancreatitis or acute necrotizing hemorrhagic pancreatitis.
7. **Alcohol rehabilitation:** If alcoholism is the cause of pancreatitis.
8. **Long-term pain management:** With analgesics or the lowest effective dose of meperidine.
9. **Oral enzyme supplements such as pancreatin and pancrelipase:** To treat malabsorption.
10. **Diet:** High in carbohydrates and protein and low in fat.
11. **Insulin therapy:** May be required to ensure adequate carbohydrate metabolism if endocrine function is impaired.
12. **Surgical interventions:** Often indicated when pancreatitis is due to an obstructive process such as gallstone formation or cancer. When gallstones are the cause of the pancreatitis, surgical removal of the stone(s) and usually the gallbladder is performed

3.6 Diabetes Mellitus

Diabetes mellitus (DM) is a chronic, progressive, metabolic disorder characterized by various degrees of glucose intolerance stemming from the complete (type I) or relative (type II) lack of insulin. DM may be precipitated by any of the following factors: genetics, autoimmune defect, obesity, stress, pregnancy, or medications. Many cases are discovered when the urine is tested routinely, for an insurance examination or before an operation, for example. Since undiagnosed diabetes may do harm, every patient admitted to hospital or attending a clinic should have the urine tested to exclude glycosuria.

Pathophysiology

Carbohydrate foods (such as bread, potato and rice) are broken down in the bowel and absorbed into the blood as glucose. Glucose is then carried to the liver where it is stored as glycogen by the action of insulin. Only enough glucose is left in the blood for the provision of normal metabolism. Insulin is a hormone produced by special collections of cells in the pancreas known as the islets of Langerhans. The islets of Langerhans pour a lot of insulin into the blood stream after a large carbohydrate meal has been eaten, since large quantities of insulin are necessary to store excessive glucose in the liver. In diabetes, something goes wrong. Either the pancreas gland does not produce enough insulin, or if insulin is produced, it is neutralized and rendered ineffective by some other agent at present unknown. In either event, there is not enough insulin available, and instead of excess glucose being stored in the liver, it simply accumulates in the blood. When the sugar in the blood rises above a certain level or threshold, the kidneys excrete the excess sugar in the urine. Hence, large quantities of urine are passed to get rid of the excess sugar. This excessive urination soon leads to thirst, while the continuous drain of glucose from the body depletes the tissues of their vital energy supplies. In severe cases, since carbohydrates are no longer available for adequate metabolism, fat is used instead. Improper fat metabolism leads to the formation of acids (ketosis) which may lead to diabetic coma.

Symptoms and Signs

1. In mild cases there may be no symptoms and the disease is first diagnosed as the result of a routine examination for sugar in the urine.
2. Excessive output of urine (polyuria) leads to great thirst.
3. Hunger (polyphagia). The feeling of hunger is often unsatisfied by food.

4. Loss of weight.
5. Drowsiness and loss of energy.
6. In elderly women, itchiness round the vulva (pruritus vulvae) is very common and very troublesome since it disturbs sleep.
7. Infections such as boils or carbuncles are liable to occur. Diabetic patients are prone to tuberculosis.

Type I diabetes: Also known as insulin-dependent diabetes mellitus (IDDM) or juvenile DM, it most commonly develops in childhood or adolescence. Onset is sudden, with possible ketoacidosis when untreated. Type I diabetes accounts for 10% of cases of diabetes. These diabetes lack endogenous insulin because of the absence of beta-cell function and require exogenous insulin to meet the demands of glucose metabolism and normal physiologic function. Type I diabetics are totally dependent on insulin for survival.

Type II diabetes: Also known as non-insulin-dependent diabetes mellitus (NIDDM) or adult-onset diabetes, It most commonly begins after age 40. Normal or above normal quantities of insulin are present in the body fluids. It is ketosis-resistant because the presence of insulin prevents lipolysis. These diabetics may require insulin during times of stress, including surgery and infection, or when diet and oral hypoglycemic medications fail to control hyperglycemia. Ninety percent of type II diabetics are obese at the time of diagnosis. Their glucose intolerance relates to the failure of their bodies to use the normal, and sometimes above-normal, levels of insulin properly. Type II diabetics may become insulin-dependent when diet or diet plus oral hypoglycemic medications fail to maintain normoglycemia.

A. Assessment

Early indicators

- Type I: Fatigue, weakness, nocturnal enuresis, weight loss, and the cardinal symptoms of hyperglycemia: polydipsia, and polyphagia.
- Type II: Peripheral neuropathy, fatigue, polyuria, polydipsia.

Late indicators

- Type I: Dehydration, electrolyte imbalance, possible hypovolemic shock, changes in mentation, possible coma, Kussmaul's respirations, acetone breath, weak and rapid pulse, hypotension, hyperglycemia.
- Type II: Marked dehydration, hypovolemic shock, obtundation, shallow respirations, gross hyperglycemia. There is absence of ketosis.

B. Complications

1. Diabetic ketoacidosis (DKA)

2. Hypoglycemia; These complications are usually preventable. **Long-term complications:** The most important factor in prevention is the maintenance of consistent, stable blood glucose levels within normal physiologic range. The following describe the levels of vascular pathology.

- **Microangiopathy:** Thickening of the basement membrane of the capillaries. Diabetic microangiopathy is manifested by retinopathy, nephropathy, and neuropathy. It compounds the effects of macroangiopathy.
- **Macroangiopathy:** Affects the larger vessels of the brain, heart, and lower extremities, resulting in cerebrovascular, cardiovascular, and peripheral vascular disease. The risk factors are hyperglycemia, hypertension, hypercholesterolemia, smoking, aging, and extended duration of DM.

C. Diagnostic Tests

The diagnosis of diabetes is made from the clinical picture; and by testing the urine and the blood for sugar.

1. Urine.

- (a) The volume is greatly increased.
- (b) The urine is pale in color and has a high specific gravity (1,030 to 1,040) due to the sugar contained.
- (c) The tests for sugar are positive
- (d) Acetone and diacetic acid (ketone bodies) may also be present in the more severe cases.

2. Blood.

- (a) Normally, the fasting blood sugar is about 80 mg. per 100 ml., and this rises to 120 mg. after a meal. In diabetes, the fasting blood sugar may be over 200 mg. per 100 ml., and even higher after food.
- (b) Glucose Tolerance Test. The suspected diabetic is given a drink containing 50 G. of glucose, and blood is taken for the estimation of sugar at intervals thereafter. Normally, the blood sugar does not rise above 180 mg. per 100 ml., even after taking glucose, but this level is exceeded when diabetes is present

D. Medical Interventions

Diet: Dietary Regime.

In normal people the supply of insulin from the pancreas is regulated by the food eaten. If no food is taken, very little insulin is secreted. Following a large carbohydrate meal, the pancreas produces considerable amounts of insulin. In diabetics this mechanism is lost. A fixed amount of insulin is injected each day and hence the diet must not be allowed to vary in quantity. The principles of the diet in patients taking insulin must include:—

1. The diet should just be adequate to maintain weight at the normal standard for the patient's age and height. In practice, most patients in this category require a diet varying from 1,500 calories to 2,000 calories, which is less than is usual for diabetics taking insulin. A girl of slight physique leading a sedentary life may require a diet of 2,000 calories. A man doing a heavy laboring job may need 2,800 calories or more.
2. The diet must contain an adequate amount of protein (at least 75 G.) and carbohydrate (at least 180 G.). It must contain fruit and fresh vegetables.
3. Meals must be spaced during the day. Thus, in addition to the three main meals of breakfast, lunch and dinner, there should also be snacks in the middle to the morning, in the early afternoon and a bedtime.
4. Meals must be taken at regular times. Delayed meals may lead to hypoglycaemic attacks

Planning the Diet.

A diet is chosen suitable for the patient's size and activities, say about 2,200 calories. This will contain:—

230 G. carbohydrate	==	920 calories.
75 G. protein	==	300 calories.
110 G. fat	==	990 calories.
Total		<u>2,210 calories.</u>

Tables and charts are available which set out the composition and calorie values of common articles of food, and so a diet can be composed to suit the tastes of the individual.

2. Oral hypoglycemic medications (sulfonylureas): Used in symptomatic type II diabetics when diet alone cannot control hyperglycemia. Their primary action is to increase insulin production by affecting existing beta-cell function. The most serious side effect is hypoglycemia, particularly with chlorpropanamide (Diabinese), which has 72-hour duration and an average half-life of 36 hours. Hypoglycemia involving the oral hypoglycemics can be severe and persistent. Nursing monitoring needs to be diligent. Oral hypoglycemics should be omitted several days before planned surgery.

3. Diabetics Needing Insulin

In severe diabetes of this type, the initial treatment is best carried out in hospital since patients have to be instructed as to their diet, self-injection of insulin and how to test the urine for sugar. Patients are more likely to co-operate if they understand the nature of their complaint, and although there are many books on diabetes available for the public, nothing can take the place of a friendly and reassuring explanation as soon as possible after diagnosis. This is particularly important in children, when parents must be able to co-operate.

Insulin.

Insulin is prepared from the pancreas glands removed from cattle, pigs and sheep after slaughter. The glands are immediately frozen to prevent the destruction of insulin by the digestive enzymes also present, and insulin is later extracted by special methods. Insulin is destroyed by the gastric juices with the result that it cannot be given by mouth and has to be administered by subcutaneous injection. Pure insulin, known as soluble insulin, when injected subcutaneously leads to a fall in the blood sugar, but its effect only lasts a few hours. Hence, various forms of insulin have been prepared which prolong its action to last all day. There are now many different types of insulin but three remain in common use in this country.

Types of Insulin

1. **Soluble Insulin.** —This is clear insulin and its effect lasts about six hours. In order to control diabetes, it must be given at least twice a day. It is always used in the treatment of diabetic coma, and often in the treatment of unstable diabetes, such as occurs in children or during pregnancy.
2. **Insulin Zinc Suspension (Lente Insulin).** —This, too, is a clear insulin but the size of the suspended particles prolongs its action to last all day. In practice it is most used for diabetics who require less than 40 units of insulin a day. It is injected in the morning, half an hour before breakfast.
3. **Protamine Zinc Insulin (PZI).** —This is a milky solution of insulin combined with protamine. It is slow to act, but its effect lasts more than twenty-four hours. It is seldom used on its own, but is very successful when added to soluble insulin. The mixture of soluble and zinc protamine insulin can be used for diabetics needing more than 40 units a day, and the proportions can be adjusted to meet individual requirements. Usually, 2 units of soluble are used for 1 unit of PZI. Thus a patient requiring 60 units may use a mixture containing 40 units of soluble and 20 units of PZI drawn up in the same syringe. The soluble insulin is drawn up into the syringe first, and then the PZI is added to this.

Injection Insulin.

Insulin is prepared in solution of 40 or 80 units per ml, and the standard insulin syringe (B.S. 1619) has 20 marks per ml. Hence, if 40 unit strength of insulin is used, each mark on the syringe is worth 2 units. If 80 unit strength insulin is used, each mark is worth 4 units. Unfortunately, many syringes with different marking systems are available and can lead to considerable confusion.

New diabetics needing insulin must be fully instructed on the following points: —

1. The dose of insulin in terms of units and marks on the syringe.

2. The strength of insulin to be used, 40 or 80 units per ml.
3. The type of insulin or insulins to be used.
4. The technique of giving the subcutaneous injection and the sites suitable for injection. The site of injection should be varied from day to day.
5. The care and sterilization of needle and syringe.

If through illness the patient is unable to eat his food, he must inject his insulin and take a glucose drink instead of food. He must consult his doctor, but it is dangerous to omit insulin since this will lead to ketosis.

Treatment of Diabetes

Treatment will depend on the type of diabetes.

Hypoglycæmia. or Insulin coma

When the blood sugar level falls too low, symptoms of hypoglycæmia occur. Some diabetics, sometimes called 'brittle' or 'unstable,' are particularly liable to these attacks despite all precautions. Hypoglycæmia is most likely to occur: —

1. When meals are delayed or irregular.
2. When unusual exertion or exercise is undertaken.
3. When the insulin dose is excessive due to unwise attempts to ensure that all specimens of urine are free from sugar.

The earliest symptoms of hypoglycæmia are sweating, mental confusion, a feeling of hunger or weakness, palpitations and trembling. The patient may have a vacant look; he will be pale and sweating, with a rapid pulse. It is most important to institute treatment immediately since otherwise the patient will go into hypoglycaemic or insulin coma. The patient should be persuaded to take a glucose drink without delay, and only if he becomes too stuporous to swallow is it necessary to administer glucose by a stomach tube or by intravenous injection. Every diabetic patient taking insulin should be aware of the early symptoms of hypoglycæmia and must always carry lumps of sugar or glucose sweets to prevent this happening. Diabetics should carry a card or bracelet stating that they are diabetic and if found confused or unconscious must be sent to hospital as an emergency.

When patients are admitted to hospital in hypoglycæmia or 'insulin coma,' it is often not known whether the patient is a diabetic. Search should be made for evidence of insulin injections in the thighs or lower abdomen. The patient is usually sweating, with dilated pupils and normal blood pressure. The breathing is quiet and there is no evidence of dehydration or collapse. The urine, if it can be obtained, is free from sugar. This condition should not be confused with diabetic coma

Testing the Urine

Diabetics needing insulin should test the urine three times a day. Unstable diabetics may have to test the urine every day, but mild diabetics may find one or two days a week is adequate. Specimens should be collected before breakfast, before lunch and last thing at night. It is best not to test urine that has been in the bladder for many hours. The urine should be tested with Clinitest or Dextrostix and the results recorded on a chart kept for the purpose.

Diabetics Needing Tablets

Many adult patients who develop diabetes can be controlled without recourse to insulin. These diabetics are not overweight, but they are not thin or wasted. The urine contains sugar but no acetone.

Tablets.

Two types of sulphonylurea tablets are in common use to bring down the blood sugar. These compounds stimulate the pancreas to produce more insulin, and this explains why they are ineffective in the more severe type of diabetes where the pancreas is incapable of producing insulin at all.

1. **Tolbutamide** is the weaker tablet, and since it is effective only for a few hours it must be given two or three times a day. The usual dose is 0.5 G. (one tablet) three times a day.
2. **Chlorpropamide** is stronger and has a long action, so that it needs to be administered once daily. The usual dose is 250 mg. (one tablet) each morning.

Both these tablets reduce the blood sugar in mild cases, so that the urine soon becomes free from sugar. They are well tolerated, and only very rarely give rise to toxic effects such as skin rashes, agranulocytosis or jaundice.

Another group of hypoglycaemic agents are the **biguanides, of which phenformin and metformin** are available. These often give rise to nausea and vomiting, but they are sometimes used in small doses in combination with the sulphonylureas when these are not quite strong enough on their own.

Overweight Diabetics

These patients usually need neither tablets nor insulin, provided they are willing to reduce their weight by restricting the food intake. Overeating places a strain on the pancreas, and if the supply of insulin is limited diabetes will result. Hence the diet must be so restricted that the patient loses weight. Depending on the degree of obesity, the diet will vary from as little as 800 calories a day to 1,200 calories a day. Once the weight is reduced, the blood sugar falls to normal, the urine becomes free from sugar and the symptoms disappear. The patient must always keep to a diet, though not necessarily as severe as the original one.

Complication of Diabetes

These complications are: -

1. **Retinopathy.** —Degeneration of the retina at the back of the eye Haemorrhages may occur and may be of sufficient severity to lead to blindness.
2. **Nephropathy (Kimmelstiel – Wilson Syndrome).** —The kidneys are damaged by long-standing diabetes and this kidney disease may ultimately lead to albuminuria, œdema of the legs, high blood pressure and uræmia. This frequently is a cause of death in diabetes.
3. **Neuropathy.** —Involvement of the peripheral nerves leads to loss of the reflexes, pain in the legs, wasting of the muscles and weakness of gait.
4. **Arteriosclerosis.** —this is hardening of the arteries of the legs leads to an impoverishment of the blood supply to the feet. This may lead to gangrene, with consequent amputation of the whole leg. Hence elderly diabetic patients must be urged to look after their feet. They must avoid cutting the toe nails too short or digging into the corner of the toes. It is often best for a regular foot toilet to be undertaken by a trained chiropodist.
5. **Infections.** —Diabetic coma is often ushered in by an infection such as tonsillitis, pneumonia, pyelitis, appendicitis or phlebitis. Treatment of the infection must be vigorous and immediate.
6. Diabetic mothers tend to have large babies, often weighing more than 10 lb. at birth.

Diabetes Coma

Diabetes coma is nearly always due to an accompanying infection, such as pneumonia, pyelitis or gastro-enteritis. The patient may feel too ill to eat, and may mistakenly omit the usual

injection of insulin. The blood sugar rapidly rises, ketosis occurs and coma is often ushered in by vomiting.

Symptoms and Signs

1. At first drowsiness with great thirst and polyuria, followed by unrousable coma.
2. Deep sighing respiration with the breath smelling of acetone.
3. Cold extremities, sunken eyeballs, shriveled tongue, dry skin and a low blood pressure: these signs are due to dehydration (loss of fluid).
4. Urine contains heavy amounts of sugar and ketones. The picture of diabetic coma is quite different from that of coma due to too much insulin (hypoglycæmia), as set out in Table I.

Table I

Symptoms and Signs	Diabetic Coma	Insulin Coma (Hypoglycæmia)
1. Onset	Gradual. History of severe thirst and polyuria: abdominal pain and vomiting.	Sudden. Patient previously well and active, taking insulin.
2. Infection	Usually present (e.g., tonsillitis, enteritis, pyelitis).	Not usually present
3. Respirations	Deep, sighing. Breath smells of acetone.	Quiet regular breathing.
4. Skin	Dry, inelastic. Tongue dry and shrunken.	Sweating, moist.
5. Blood pressure	Very low; rapid thin pulse.	Normal. Full pulse.
6. Urine	Sugar and acetone.	No sugar

Treatment of Diabetic Coma

Diabetic coma is medical emergency and it is essential that all steps be taken as quickly as possible to restore the patient to consciousness.

1. **Insulin.** —This must be given in large doses and at frequent intervals. The first dose is usually about 100 units of soluble insulin, and of this 40 units or so will be given intravenously to ensure rapid action. In coma there is circulatory failure so that there is poor absorption from the subcutaneous route.
2. An intravenous saline drip is set up and 1,000 ml. are given rapidly within about an hour to overcome the severe dehydration. After this the drip may be slowed and continued at this slower rate till the patient is aroused and able to take fluids by mouth.
3. Urine is obtained every two hours, a retention catheter being useful for this purpose. The urine is tested for sugar and ketone bodies present the patient is in serious danger.
4. A blood sugar test is usually taken at the outset. The level of the blood sugar is a guide to treatment, as if the initial blood sugar is very high (500 mg. per cent. Or over) the first dose of insulin may have to be quickly supplemented by another large dose.
5. Gastric lavage and enemas to relieve the abdominal distension, is sometimes ordered. Circulatory failure and shock. These are always present in diabetic coma, and are treated by raising the foot of the bed and by giving intravenous saline to overcome the dehydration which causes the shock. It should be stressed that intravenous fluids are second in importance only to large doses of insulin in the treatment of diabetic coma.
6. When the patient regains consciousness, four-hourly feeds are given of 25 to 30 G. of carbohydrate in the form of glucose, fruit juices, milk or any suitable equivalent food the patient is able to take. Insulin will be ordered with each feed or less often, according to how much is needed to keep the urine sugar-free. When the patient can swallow, potassium chloride or potassium citrate 2 G. six-hourly is given by mouth. In most cases

of coma the onset is precipitated by some infection or septic focus, and so in all such cases any such sepsis must be treated from the start with penicillin or any other appropriate measures.

E. Nursing Diagnoses and Interventions

Potential for infection related to increased susceptibility secondary to disease process (eg, hyperglycemia, neurogenic bladder, poor circulation).

Desired outcome: Patient does not exhibit signs of infection.

1. Monitor temperature q4h.
2. Maintain meticulous sterile technique when changing dressings, performing invasive procedure, or manipulating indwelling catheters.
3. Monitor for signs of infection: dysuria, urgency, frequency, cloudy and/or foul-smelling urine; redness, local warmth, swelling, discharge, and pain from skin wounds or lesions; complaints of sore throat and swollen glands and inflamed pharynx; changes in the color, amount, or consistency of sputum; chest pain and SOB; fever; leukocytosis.

Potential impairment of skin integrity related to increased susceptibility secondary to peripheral neuropathy and vascular pathology.

Desired outcomes: Patient's skin remains intact. Patient can verbalize and demonstrate knowledge of proper foot care.

1. Assess integrity of the skin and evaluate reflexes of the lower extremities by checking knee and ankle deep tendon reflexes (DTRs), proprioceptive sensations, and vibration sensation using a tuning fork.
2. Monitor peripheral pulse, comparing the quality bilaterally.
3. Use foot cradle on bed, spaceboots for ulcerated heels, elbow protectors, and alternating air pressure mattress to prevent pressure points and promote patient comfort.
4. Teach patient the following steps for foot care:
 - Wash feet daily with mild soap and warm water; check water temperature with water thermometer or elbow.
 - Inspect feet daily for the presence of redness or trauma, using mirrors as necessary for adequate visualization.
 - Prevent infection from moisture or dirt by changing socks or stockings daily and wearing cotton or wool blends.
 - Prevent ingrown toenails by cutting toenails straight across after softening them during bath.
 - Do not self-treat corns or calluses; visit podiatrist regularly.
 - Attend to any foot injury immediately, and seek medical attention to avoid any potential complication.

Knowledge deficit: Procedure for insulin administration.

Desired outcome: Patient can demonstrate procedure for administration of insulin.

1. Teach patient to check expiration date on insulin vial and to avoid using it if outdated.
2. Demonstrate rolling the insulin vial between the palms to mix the contents. Caution patient that vigorous shaking produces air bubbles that can interfere with accurate dosage measurement.

3. Explain that insulin should be injected 30 minutes before mealtime.
4. Provide patient with a chart that depicts rotation of the injection sites. Explain that injection sites should be at least 1 inch apart.

Patient–Family Teaching and Discharge Planning

Provide patient and SOs with verbal and written information for the following:

1. Importance of carrying a diabetic identification card and wearing Medic-Alert bracelet or necklace.
2. Recognizing warning signs of both hyperglycemia and hypoglycemia and factors that contribute to both conditions. Remind patient that stress from illness or infection can increase insulin requirements and that increased exercise will necessitate additional food intake to prevent hypoglycemia when there is no change made in insulin dosage.
3. Home monitoring of blood glucose. Stress the need for careful control of blood glucose as a means of preventing long-term complications of DM.
4. Importance of daily exercise, good blood-glucose control, maintenance of normal body weight, and yearly medical evaluation, including visit to a podiatrist and ophthalmologist.
5. Necessity of rotating injection sites and injecting insulin at room temperature. Provide a chart showing possible injection sites and describe the system for rotating the sites.
6. Importance of good foot care.
7. Importance of annual eye examinations for early detection and treatment of retinopathy.

3.7 Diabetic Ketoacidosis

Diabetic ketoacidosis (DKA), a catabolic state, reflects glucose and ketone production from the breakdown of fats and protein acids. Hyperglycemia leads to osmotic diuresis with the loss of fluid and electrolytes. As ketones accumulate and bicarbonate excretion occurs, ketoacidosis develops. Coma and death will ensue if the condition remains untreated. Stress (eg, infection) is the most common precipitating causes of DKA.

4.0 Conclusion

The two major groups of diabetes mellitus are type 1, insulin-dependent diabetes mellitus. The three classic symptoms of diabetes mellitus are polyuria, polydipsia, and polyphagia. The treatment of diabetes mellitus involves diet, exercise, insulin or an oral hypoglycemic agent, and weight control.

5.0 Summary

Diabetes insipidus is a lack of ADH secretion in response to a decreased vascular volume and increased osmolarity, resulting in a lack of conservation of fluid by the kidneys and decreased concentration of urine.

Thyrotoxic crisis (thyroid storm) is a severe form of hyperthyroidism caused by oversecretion of T_3 and T_4 followed by a markedly increased metabolic rate.

The two major groups of diabetes mellitus are type 1, insulin-dependent diabetes mellitus.

6.0 Tutor Marked Assignment

- Differentiate between the symptoms of hypoglycemia and hyperglycemia.
- Discuss the nursing management of the client with diabetes mellitus.

7.0 Further Reading and Other Resources

Suzanne C. Smeltzer, Brenda Bare (2004). *Medical Surgical Nursing*. Lippincott Williams & Wilkins

Ethelwynn L. Stellenbery, Juditt C. Bruce (2007). *Nursing Practice: Medical-Surgical Nursing for Hospital and Community*. Elsevier Edinburgh.

APPENDIX I

Normal Values for Laboratory Tests Discussed in This Booklet

Urine		
Test	Specimen	Normal Values
Albumin	Random	Negative
	24-hour	10–100 mg/24 hr
Amylase	2-hour	35–260 Somogyi units/hr
	24-hour	80–5000 U/24 hr
Bence-Jones	Random	Negative
Protein		
Bilirubin	Random	Negative: 0.02 mg/dL
Calcium	Random	1+ turbidity
		10 mg/dL
	24-hour	50–300 mg/24 hr (depends on diet)
		25–200 mEq/24 hr
Catecholamines	Random	0–18 µg/dL
	24-hour	Less than 100 µg/24 hr (varies with activity)
Concentration test	Random after fluid restriction	1.025–1.035
Cortisol, free	24-hour	Men: 20–69 µg/24 hr
		Women: 8–63 µg/24 hr
Creatinine	24-hour	Male: 20–26 mg/kg/24 hr 1.0–2.0 g/24 hr Female: 14–22 mg/kg/24 hr 0.6–1.8 g/24 hr
Creatinine clearance	Serum or plasma and urine	Male: 107–141 mL/mm Female: 87–132 mL/mm
Glucose	Random	Negative: 15 mg/dL
	24-hour	130 mg/24 hr
Ketone	Random	Negative: 0.3–2.0 mg/dL
17-Ketosteroids	24-hour	Male: 8–25 mg/24 hr
(17-KS)		Female: 5–15 mg/24 hr
		Over 65: 4–8 mg/24 hr
		After 25 units of ACTH IM: 50%–100% increase
Microscopic examination	Random	RBC: 2–3/high-power field WBC: 4–5/high-power field Hyaline casts occasional Bacteria: fewer than 1000/mL
Osmolality	Random	Male: 390–1090 mOsm/kg Female: 300–1090 mOsm/kg
	24-hour	Male: 770–1630 mOsm/24 hr Female: 430–1150 mOsm/24 hr
PH	Random	46–80
Phenolsulfon- phthalein (PSP)	Timed collection after 6 mg of	15 minutes: 25%–35% of dye

Platelet count	150.000–400.000/ μ L	
Other Hematologic Studies	Specimen	Normal
Coagulation studies	-	
Bleeding time	Capillary blood	Duke method: 1–3 minutes Ivy method: 1–7 minutes
Fibrinogen assay	Plasma	200–400 mg/dL
Partial thromboplastin time (PTT)	Plasma	Activated: 30–40 seconds Nonactivated: 40–100 seconds
Platelet count	Whole blood	150,000–400,000/ μ L
Prothrombin time	Plasma	11–15 seconds
Whole blood clotting time	Whole blood	Siliconized tubes: 24–45 minutes Plain tubes: 5–15 minutes
Eosinophil count	Whole blood	50–400/ μ L
Erythrocyte Sedimentation rate (ESR)	Whole blood	
Wintrobe method		Male: 0–9 mm/hr Female: 0–20 mm/hr
Westergren method		Male: Under 50 years, 0–15 mm/hr Over 50 years, 0–20 mm/hr Female: Under 50 years, 0–20 mm/hr Over 50 years, 0–30 mm/hr
Volume, blood	Whole blood	Male: 69 mL/kg Female: 65 mL/kg
Volume, plasma	Whole blood	Male: 39 mL/kg Female: 40 mL/kg
Cerebraspinal Fluid		
Test	Normal Values	
Appearance	Clear and colorless	
Cell count	0–10 WBC/ μ L (60%–100% lymphocytes)	
Glucose	Adults 40–80 mg/dL 50%–80% of blood glucose	
Immunoglobulin		
IgA	0–06 mg/dL	
IgG	0–55 mg/dL	
IgM	0–13 mg/dL	
Protein	Adult 15–50 mg/dL	
Protein electrophoresis	Prealbumin 3%–6%	
	Albumin 45%–68%	
	α_1 -globulin: 3%–9%	
	α_2 -globulin: 4%–10%	
	β -globulin: 10%–18%	
	γ -globulin: 3%–11%	