COURSE TITLE: MEDICINE AND MEDICAL NURSING 11

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LIST OF FIGURES

LIST OF ABBREVIATION

TDF- Tenofivir

XTC- Emitrictabine and Lamuvidine

NVP- Niverapine

EFV- Efavirenz

AZT- Azidovudine

LPV-r- Lopinavir/ritonavir

ABC- Abacavir

ddI- Didanosine

COURSE OVERVIEW

Introduction

You are welcome to our course on Medicine and Medical Nursing II. In year 1 you learnt four units covering introduction to medicine, the gastrointestinal tract conditions, conditions of the respiratory system and the cardiovascular system. This course equally intends to equip you with knowledge which is essential in nursing patients with medical conditions. Knowing the medical conditions will assist you to provide nursing care to your patients effectively and efficiently as you know, nursing care contributes much in the recovery of the patients.

Medicine and medical nursing II has 4 units. The splitting of the course in different units is to enable you understand each aspect of the course step by step. This course is associated with some of the courses that you studied in your foundation block such as anatomy and physiology, microbiology and nutrition.
The systems that you will learn are briefly described below before we discuss them in detail later.

CONTENT

Unit 1: The Urinary System

This is the first unit in Medicine and Medical nursing II. In this section you will review the anatomy and physiology of the urinary system, outline the role of the nurse in investigations and procedures which are done in investigatingthe urinary system disorders and describe the management of patients with urinary disorders.

Unit 2: Nervous System

This unit reviews the anatomy and physiology of the Nervous system, outlines the neurological assessment and role of the nurse in investigations and procedures which are done in neurological disorders and management of a patient with neurological disorders.

Unit 3: Endocrine System

The unit reviews anatomy and physiology of the endocrine system, outlines the role of the nurse in investigations and procedures which are done in endocrine disorders. It also describes the

management of a patient with metabolic disorders, pancreatic disorders, pituitary disorders, thyroid disorders and adrenal disorders.

Unit 4: HIV/AIDS And Antiretroviral Therapy

This unit will review the HIV/AIDS information and management issues and outline the goals and general principles of Antiretri viral (ARV) therapy. It will also discuss the antiretroviral drug information, counselling and education, patient assessment, initiating ARV therapy, management of a patient on ARV therapy, managing HIV/AIDS in special populations, Post Exposure Prophylaxis and record keeping, monitoring and evaluation of patiets on ART.

COURSE AIM:

The aim of the course is to equip the student with knowledge and skills in the management of clients with medical disorders in the hospital, health centre and community.

COURSE OBJECTIVES

At the end of the course the student should be able to:

1. Describe the common medical conditions affecting various systems of the body.
2. Demonstrate knowledge and skills in creating awareness in the management, prevention and control of common infectious diseases in the hospital, health centre and community settings.
3. Interpret medical diagnostic investigations and prescribe relevant drugs and other interventions.
4. Apply the appropriate nursing models in the management of patients/clients.

Assessment

Theory 100%

- Continuous Assessment
  - Test 2 20%
  - Group Assignment 2 20%

Total **40%**

- Final Theory Examination 1 **60%**

Practical 100%

Clinical Assessment 2 40%

Final Practical Examination 1 60%

UNIT 1: URINARY SYSTEM (14 Hours)

1.1 Intorduction

Dear learner welcome towhich focuses on the urinary system. You will recall that the urinary system is the system that is responsible for the excretion of metabolic wastes through urine. As such, any disease or condition that affects this system will eventually lead to accumulation of theses waste products and cause problems to the body. In this unit we are going to review the anatomy and physiology of the urinary system and discuss your role as a nurse in investigations

and procedures performed during investigation of urinary disorders. We shall also discuss common disorders of the urinary system. Let us start by reviewing our objectives for this course.

1.2 Unit Objectives

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

1. Describe anatomy and physiology of the urinary system
2. Explain the roles of the nurse in investigations and procedures
3. Discuss the management of patients with disorders of the urinary system

1.3 Applied Anatomy And Physiology Of The Urinary System

You will now start by reviewing the anatomy and physiology of the urinary system. Before you continue reading find out how much you still remember about this system by completing the following activity.

Activity

List down in your notebook the organs of the urinary system and their functions.

We hope that recalled that the urinary system comprises structures that precisely maintain the internal chemical environment of the body through the following functions of excretion, regulation, and secretion. The main structures are the kidneys, ureters, bladder and urethra.

Kidneys

The kidneys are a pair of brownish-red structures located retroperitoneally (behind and outside the peritoneal cavity) on the posterior wall of the abdomen from the 12th thoracic vertebra to the 3rd lumbar vertebra in the adult. An adult kidney weighs 120 to 170 g (about 4.5 oz.) and is 12 cm (about 4.5 inches) long, 6 cm wide, and 2.5 cm thick. The kidneys are well protected by the ribs, muscles, Gerota's fascia, perirenal fat, and the renal capsule, which surround each kidney. The kidney consists of two distinct regions, the renal parenchyma and the renal pelvis. The renal parenchyma is divided into the cortex and the medulla. The cortex contains the glomeruli,

proximal and distal tubules, and cortical collecting ducts and their adjacent peritubular capillaries. The medulla resembles conical pyramids. The pyramids are situated with the base facing the concave surface of the kidney and the apex facing the hilum, or pelvis. Each kidney contains approximately 8 to 18 pyramids. The pyramids drain into 4 to 13 minor calices that, in turn, drain into 2 to 3 major calices that open directly into the renal pelvis. The hilum, or pelvis, is the concave portion of the kidney through which the renal artery enters and the renal vein exits. The renal artery (arising from the abdominal aorta) divides into smaller and smaller vessels, eventually forming the afferent arteriole. Figure 1 below shows the structure of the kidney.

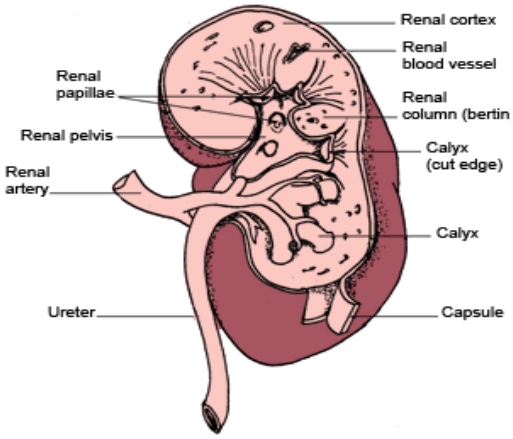

Figure 1.Showing the Kidney

The afferent arteriole branches to form the **glomerulus**, which is the capillary bed responsible for glomerular filtration. Blood leaves the glomerulus through the efferent arteriole and flows back to the inferior vena cava through a network of capillaries and veins. Each kidney contains about 1 million **nephrons**, the functional units of the kidney. Each kidney is capable of providing adequate renal function if the opposite kidney is damaged or becomes non-functional. The nephron consists of a glomerulus containing afferent and efferent arterioles, Bowman's capsule, proximal tubule, loop of Henle, distal tubule, and collecting ducts. Collecting ducts converge into papillae, which empty into the minor calices, which drain into three major calices that open directly into the renal pelvis.

Nephrons are structurally divided into two types: cortical and juxtamedullary. Cortical nephrons are found in the cortex of the kidney, and juxtamedullary nephrons sit adjacent to the medulla. The juxtamedullary nephrons are distinguished by their long loops of Henle and the vasa recta,

long capillary loops that dip into the medulla of the kidney. Figure 2 below shows the structure of the nephron.

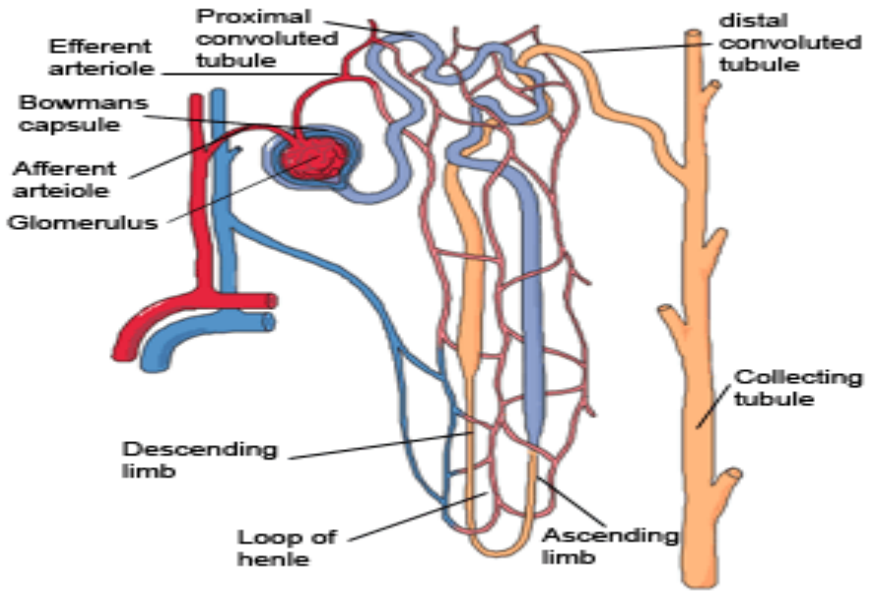

Figure 2.Showing the Nephron

The glomerulus is composed of three filtering layers: the capillary endothelium, the basement membrane, and the epithelium. The glomerular membrane normally allows filtration of fluid and small molecules yet limits passage of larger molecules, such as blood cells and albumin. of yearsKidney function begins to decrease at a rate of approximately 1% each year beginning at approximately age 30.

The discussion above reviewed the anatomy of the upper urinary system; in the next subheading we will discussof the lower urinary system.

Ureters, Bladder, and Urethra

Urine, which is formed within the nephrons, flows into the ureter, a long fibromuscular tube that connects each kidney to the bladder. The ureters are narrow, muscular tubes, each 24 to 30 cm long, that originate at the lower portion of the renal pelvis and terminate in the trigone of the bladder wall. There are three narrowed areas of each ureter: the ureteropelvic junction, the ureteral segment near the sacroiliac junction, and the ureterovesical junction.

The angling of the ureterovesical junction is the primary means of providing antegrade, or downward, movement of urine, also referred to as efflux of urine. This angling prevents vesicouretera reflux, which is the retrograde, or backward, movement of urine from the bladder, up the ureter, toward the kidney. During voiding (micturition), increased intravesical pressure keeps the ureterovesical junction closed and keeps urine within the ureters. As soon as micturition is completed, intravesical pressure returns to its normal low baseline value, allowing efflux of urine to resume. Therefore, the only time that the bladder is completely empty is in the last seconds of micturition before efflux of urine resumes.

The three areas of narrowing within the ureters have a propensity toward obstruction because of renal calculi (kidney stones) or stricture. Obstruction of the ureteropelvic junction is the most serious because of its close proximity to the kidney and the risk of associated kidney dysfunction. The left ureter is slightly shorter than the right. The lining of the ureters is made up of transitional cell epithelium called urothelium. As in the bladder, the urothelium prevents reabsorption of urine. The flow of urine from the renal pelves through the ureters into the bladder is facilitated by peristaltic waves (occurring about one to five times per minute) from contraction of the smooth muscle in the ureter wall (Smeltzer et al., 2010).

The urinary bladder is a muscular, hollow sac located just behind the pubic bone. Adult bladder capacity is about 300 to 600 mL of urine. In infancy, the bladder is found within the abdomen. In adolescence and through adulthood, the bladder assumes its position in the true pelvis. The bladder is characterized by its central, hollow area called the vesicle, which has two inlets (the ureters) and one outlet (the urethrovesical junction), which is surrounded by the bladder neck. The wall of the bladder comprises four layers. The outermost layer is the adventitia, which is made up of connective tissue. Immediately beneath the adventitia is a smooth muscle layer known as the detrusor. Beneath the detrusor is a smooth muscle tunic known as the lamina propria, which serves as an interface between the detrusor and the innermost layer, the urothelium. The urothelium layer is specialized, transitional cell epithelium, containing a membrane that is impermeable to water. The urothelium prevents the reabsorption of urine stored in the bladder. The bladder neck contains bundles of involuntary smooth muscle that form a

portion of the urethral sphincter known as the internal sphincter. The portion of the sphincteric mechanism that is under voluntary control is the external urinary sphincter at the anterior urethra, the segment. The urethra arises from the base of the bladder. In the male it passes through the penis; while in the female, it opens just anterior to the vagina. In the male, the prostate gland, which lies just below the bladder neck, surrounds the urethra posteriorly and laterally.

Having looked the anatomy of the urinary system we, will now shift our focus to the physiology of the urinary system.

Activity
List down **in your notebook** the steps of urine formation.

Now compare your answer with what you read in the following discussion.

Physiology of the Urinary System

The urinary system performs various roles that are essential for normal bodily homeostasis. These functions include urine formation; excretion of waste products; regulation of electrolyte, acid, and water excretion; and auto regulation of blood pressure.

Urine Formation

Urine is formed in the nephrons through a complex three-step process: glomerular filtration, tubular reabsorption, and tubularsecretion. The various substances normally filtered by the glomerulus, reabsorbed by the tubules, and excreted in the urine include

- sodium,
- chloride,
- bicarbonate,
- potassium,
- glucose,
- urea,
- creatinine, and
- uric acid.

Within the tubule, some of these substances are selectively reabsorbed into the blood. Others are secreted from the blood into the filtrate as it travels down the tubule. Some substances, such as glucose, are completely reabsorbed in the tubule and normally do not appear in the urine.

Amino acids and glucose are usually filtered at the level of the glomerulus and reabsorbed so that neither is excreted in the urine. Glucose, however, appears in the urine (glycosuria) if the amount of glucose in the blood and glomerular filtrate exceeds the amount that the tubules are able to reabsorb. Normally, glucose is completely reabsorbed when the blood glucose level is less than 200 mg/dL (11 mmol/L). In diabetes, when the blood glucose level exceeds the kidneys' reabsorption capacity, glucose appears in the urine. Glycosuria is also common in pregnancy. Protein molecules are also generally not found in the urine; however, low-molecular-weight proteins (globulins and albumin) may periodically be excreted in small amounts. Transient proteinuria in amounts less than 150 mg/dL is considered normal and does not require further evaluation. Persistent proteinuria usually signifies damage to the glomeruli.

Process of Urine Formation

Process of urine formation is as follows:

- Glomerular filtration: The normal blood flow through the kidneys is about 1,200 mL/min. As blood flows into the glomerulus from an afferent arteriole, filtration occurs. The filtered fluid, also known as filtrate or ultrafiltrate, then enters the renal tubules. Under normal conditions, about 20% of the blood passing through the glomeruli is filtered into the nephron, amounting to about 180 L/day of filtrate. The filtrate normally consists of water, electrolytes, and other small molecules, because water and small molecules are allowed to pass, whereas larger molecules stay in the bloodstream. Efficient filtration depends on adequate blood flow maintaining a consistent pressure through the glomerulus. Many factors can alter this blood flow and pressure, including hypotension, decreased oncotic pressure in the blood, and increased pressure in the renal tubules from an obstruction.
- Tubular reabsorption and tubular secretion: The second and third steps of urine formation occur in the renal tubules and are called tubular reabsorption and tubular secretion. In

tubular reabsorption, a substance moves from the filtrate back into the peritubular capillaries or vasa recta. In tubular secretion, a substance moves from the peritubular capillaries or vasa recta into tubular filtrate. Of the 180 L (45 gallons) of filtrate that the kidneys produce each day, 99% is reabsorbed into the bloodstream, resulting in 1,000 to 1,500 mL of urine each day. Although most reabsorption occurs in the proximal tubule, reabsorption occurs along the entire tubule. Reabsorption and secretion in the tubule frequently involve passive and active transport and may require the use of energy. Filtrate becomes concentrated in the distal tubule and collecting ducts under the influence of **antidiuretic hormone (ADH)** and becomes urine, which then enters the renal pelvis.

Excretion of Waste Products

The kidney functions as the body's main excretory organ, eliminating the body's metabolic waste products. The major waste product of protein metabolism is urea, of which about 25 to 30 g is produced and excreted daily. All of this urea must be excreted in the urine; otherwise it will accumulate in body tissues. Other waste products of metabolism that must be excreted are creatinine, phosphates, and sulfates. Uric acid, formed as a waste product of purine metabolism, is also eliminated in the urine. The kidneys serve as the primary mechanism for excreting drug metabolites (Smeltzer et al., 2010).

CHECKPOINT QUESTIONS

Encircle the most appropriate answer.

1. The functional unit of a kidney is
   - **a.** The calyx
   - **b.** Pelvis
   - **c.** Pyramids
   - **d.** The nephron
2. The first step in urine formation is
   - **a.** Excretion
   - **b.** Selective reabsorption

c. Glomerular filtration

d. Active transportation

3. Anatomically the kidneys are

a. Laterally positioned

b. Anterior to the peritoneum

c. Retroperitoneal

d. Lowly set

Answers: Q1 D. Q2 C. Q3 C.

This marks the end of our review of the anatomy and physiology of the urinary system. We will now discuss the roles of the nurse in investigations and procedures that help to diagnose urinary conditions.

1.4 role OF The Nurse In Investigations And Procedures

As a nurse you will be carrying out or assisting in carrying out investigative procedures. Therefore it is important to know what your roles are as you do these procedures. The following is a set of investigations and procedures done in diagnosing urinary conditions.

- Urine examination

  You are responsible for collecting a clean urine specimen from the patient in clean receptacle (urinal/bedpan). Places the urine in a jug and later in test tube, in order for you to clearly see the physical properties of urine. The collection can either be immediate "on the spot" or 24 hour urine sample.

  - Physical: check for the physical appearance of urine, urine has an amber colour and formof particles with an aromatic smell. Obtain first urinated morning specimen. Ensure that the urine is examined within 1 hour of urinating. Wash the perineal area if soiled with menses or faecal material.
  - Chemical: using uristix during urinalysis it will reveal the chemical properties in a urine specimen (protein, urea, the pH of urine, leucocytes etc.).

- Microscopic: collect urine in the sterile container. Touch only the outside of the container. You should examine microscopically and observe microbes as seen under a microscope. He/she may see parasites and their ova in a specimen of stool/urine e.g. in schistosomiasis. For women separate labia with one hand and clean the meatus with the other hand using at least three sponges. For the male retract the foreskin and clense glans with at least three cleansing. After cleaning, instruct the patient to start urinating and then continue voiding in a sterile container.

- **Renal function tests:** blood urea and nitrogen (BUN), urea and creatinine and electrolyte tests can also be done. As a nurse you are required to collect the sample in the absence of a laboratory scientist and you need to know the normal values of the parameters. You are required to assist the Medical Officer in collecting the specimens and ensure that the patient's results are filed when ready. You should also explain the test and watch for postpuncture bleeding.

- Radiological examinations
  - Plain x-ray

    You need to prepare the patient for the radiological examinations listed below both physical and psychologically and assist the radiology staff in placing the patient in the required positions for each of the procedures. You will need to protect yourself from x-rays by putting on the lead made aprons and keep away from sources of radioactive material
- Intravenous pyelogram

  This is an X- ray examination, which visualises the urinary tract after IV injection of the contrast media (*Lewis et al., 2007).* As a nurse you need to give an enema evening before the procedure to empty colon of faeces and gas. Keep patient on NPO status 8 hours before the procedure. assess the patient for iodine sensitivity to avoid anaphylaxic reaction. Inform the patient that the procedure involves lying on the table and having serial x-rays taken. After the procedure, force fluids to flush out contrast material.

- **Retrograde pyelogram**

This is an x- ray of the urinary tract which is taken after injection of contrast material into kidneys cystoscope is inserted and ureteral catheters are inserted through it into renal pelvis. Prepare the patient as for IVP. Inform the patient that pain may be experienced from distention and distention and discomfort from cystoscope. Inform the patient that anaesthesia may be given for the procedure.

- **Cysto-gram**

In this procedure, contrast media is injected into the bladder via cystoscope or catheter. The purpose of the procedure is to visualise the bladder and evaluate vesico-ureteral reflux. Explain the procedure to the patient.

- Urethrogram

  A retrograde urethrogram is a routine radiologic procedure (most typically in males) used to image the integrity of the urethra. Hence a retrograde urethrogram is essential for diagnosis of urethral injury, or urethral stricture. The procedure involves the insertion of a Foley catheter into the distal urethra and minimally inflating it. This is followed by instillation of 30mL of water soluble contrast and a plain radiograph is obtained. It is used when there is suspicion of urethral trauma.

  The most common indication for a retrograde urethrogram in the setting of trauma is the presence of blood at the urethral meatus after blunt or penetrating trauma. Penile fracture with gross haematuria is also an indication for a RUG to elucidate the presence of a urethral injury. Another relative indication for a RUG is the finding of a “floating prostate” on digital rectal examination, which may indicate urethral disruption. The prostate normally feels fixed on digital rectal examination, and if the prostate is mobile or “floating,” a urethral disruption may have occurred. Lower urinary tract symptoms, male patients with a previous history of urethral stricture who havewith symptoms of urinary urgency, urinary frequency, and poor bladder emptying are at risk for a urethral stricture.Postoperative evaluation - A RUG is often performed for the imaging and evaluation of the urethral after a surgical procedure such as urethroplasty.

- ***Cystoscopy***

This is visualization of the internal structure of the bladder by use of an endoscope. As a nurse you will need to prepare the equipment and the room where the procedure will be done. You will need to prepare IV fluids such as normal saline as it aids in accurate visualization and bladder washout during the procedure. Ensure privacy and reassure the patient. Figure 3 shows a cytoscopic examination.

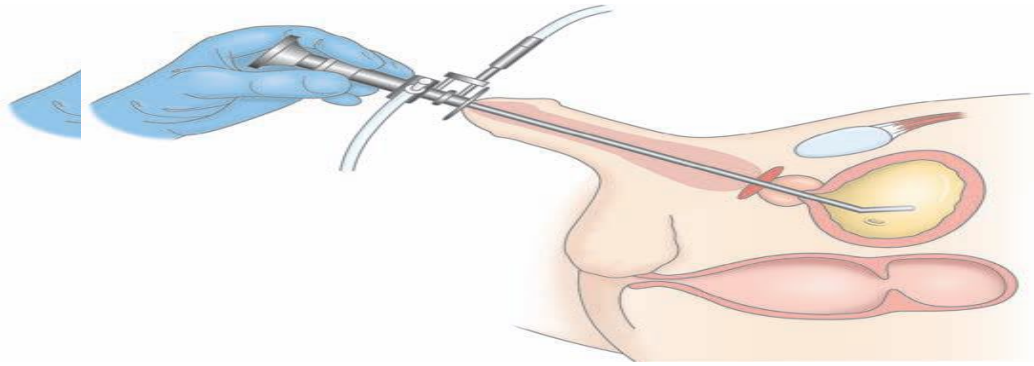

Figure 3: An illustration of a cystoscopic examination

- **Ultra sound**

ultrasound enables visualization of the various organs of urinary system. As a nurse you need to prepare the patient by taking adequate fluid/water as these organs and their internal structures are better visualized when fluid filled e.g. the bladder.

PERITONEAL DIALYSIS/HAEMODIALYSIS

Definition of dialysis: this is used to remove fluid and uremic waste products from the body when the kidneys cannot do so. In dialysis a semi permeable membrane, osmosis and diffusion initiate normal kidney function by eliminating excessive body fluids maintaining or restoring plasma electrolyte and acid base balances and removing waste products and dialyzable toxins from the blood. Dialysis is most often used in patients with acute/chronic renal failure. The most common types are:

- Peritoneal and
- Hemodialyses

Peritoneal Dialysis

Peritoneal **dialysis** (PD) is a treatment for patients with severe chronic kidney disease. The process uses the patient's peritoneum in the abdomen as a membrane across which fluids and dissolved substances (electrolytes, urea, glucose, albumin and other small molecules) are exchanged from the blood. Fluid is introduced through a permanent tube in the abdomen and flushed out either every night while the patient sleeps (automatic peritoneal dialysis) or via regular exchanges throughout the day (continuous ambulatory peritoneal dialysis).

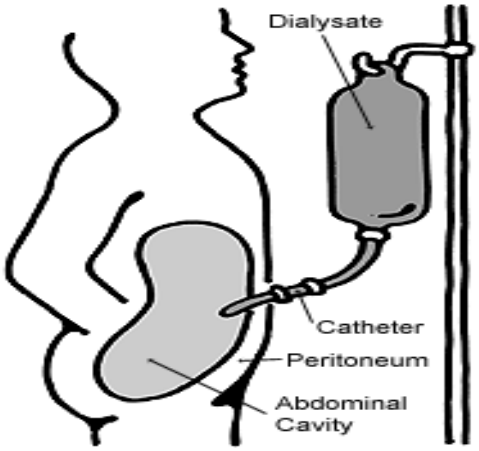

Figure 4: Peritoneal dialysis (Source: Wikipedia)

Heamodialysis

This is a process used for patients who are acutely ill and require short-term dialysis (days-weeks) or patients with end stage renal diseases who require long-term therapy. A synthetic semi permeable membrane replaces the renal glomeruli and tubules and acts as the filter for the impaired kidneys. Haemodialysis is a method that is used to achieve the extracorporeal removal of waste products such as creatinine and urea and free water from the blood when the kidneys are in a state of renal failure.

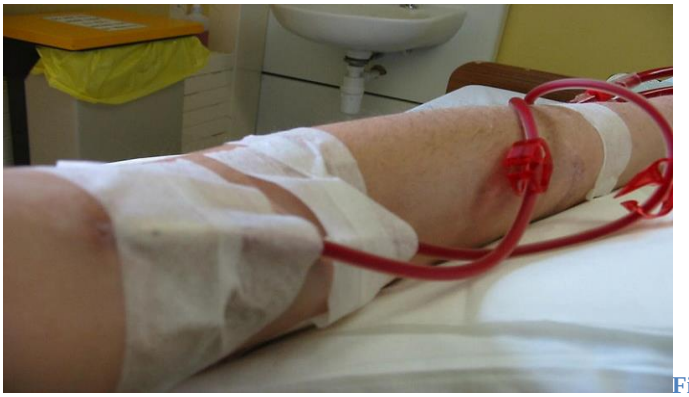

Figure 5: Haemodialysis (Source: Wikipedia)

Roles of the nurse

- Explain procedure and obtain a signed consent.
- Prepare the room for dialysis
- Ensures the patient is prepared for the procedure
- Ensure equipment and needed fluids are readily available
- Assess baseline vital signs, weight and serum electrolyte levels. Encourage emptying of bladder and bowels to minimize risk of puncturing the internal organs and structures. Give pt necessary support and instruction to allay anxiety.
- Promote patient comfort during procedure i.e. provide physical comfort measures e.g. pressure area care, keep pt informed of progress and results, provide psychological care throughout procedure
- Maintain dialysate infusion and drainage i.e. if fluid is not draining properly, move pt from side to side to facilitate the removal of peritoneal drainage. Never push in the catheter.
- Monitor changes in fluid and electrolyte balance, weight changes, vital signs and I and O record.
- Monitor for complications, i.e. peritonitis, watch for nausea and vomiting, anorexia, abdominal pain, tenderness, rigidity and cloudy dialysate drainage.

- ✓ Bleeding where you observe the catheter site and the blood drainage
- ✓ Respiratory difficulty
- ✓ Leakage on the site of drainage
- ✓ Constipation
- ✓ Low serum albumin

Role of the nurse

Assignment

During your clinical placement on a medical ward carryout the following investigations.

1. Urinalysis and record on the patient's chart.

2. Collect a urine specimen and do microscopic examination in the laboratory and report your findings.

You have finished looking at the roles of the nurse during investigations and procedures, the next subheading will discuss the management of patients with urinary disorders.

1.5 Management Of Patients With Urinary Disorders

Urinary disorders are common problems in many individuals and some may be mild while others can lead to complete renal shut down. They are classified as the upper or lower urinary tract disorders and further classified as complicated or uncomplicated. The lower urinary tract disorders include infections affecting the bladder and structures below the bladder and the upper urinary tract disorders include the kidneys and ureters disorders. In this discussion our attention will be on common disorders of the lower and upper urinary tract.

5**Lower Urinary Tract Disorders.**

Urinary tract infections are one of the most common infections affecting humans. Urinary Tract Infections involves an inflammation of some portion of the urinary tract usually caused by bacteria.

CYSTITIS

Cystitis is one of the diseases of the urinary tract infections.

Definition of Cystitis

Cystitis is an inflammation of the bladder

Causes of Cystitis

The cause of cystitis is pathological bacteria such as:

- Escherichia coli
- Gram positive cocci, e.g. staphylococcus & streptococcus

Predisposing factors to cystits

1. Residual urine in the bladder
    - Neurogenic bladder
    - Urethral stricture
    - Prostatic hypertrophy
    - Renal calculi
    - Prostatitis
    - Postcoital bladder trauma can also cause cystitis.
2. Instrumentation of the urinary tract
    - Indwelling catheter
    - Catheterization
    - Urethral dilation
    - Cystoscopy

Clinical Picture

- Dysuria
- Frequent micturition
- Urgency
- Supra pubic pain
- Urine often appears grossly cloud
- HaematuriaSystemic manifestations such as fever, nausea, and vomiting
- Burning sensation
- Urine incontinence

Management Of Patient With Cystitis

Investigations

- History taking and clinical pictureUrine examination may reveal WBCs and bacteria

- Mid-stream urine for culture and sensitivity
- Cystocopy to diagnose the problem if the infection is not improving.

Medical Management

- Treatment depends on culture and sensitivity
- Appropriate antibiotics may be prescribed

Nursing Management

Assessment*:*

- Health history taking
- Physical examination

Information Education and Communication

Hygiene

- Advise the patient to shower rather than bathe in the tub because bacteria in the bath water may enter the urethra.
- Advise the patient, to clean the perineum and urethral meatus after each bowel movementfrom front to back. This will help reduce concentrations of pathogens at the urethral opening in, in women, the vaginal opening.

Fluid Intake

- Tell the patient to be drinking liberal amounts of fluids daily to flush out bacteria.
- Patient should avoid coffee, tea, colas, alcohol, and other fluids that are urinary tract irritants.

Voiding

Explain the importance of voiding every 2 to 3 hours during the day and completely empty the bladder. This prevents over distention of the bladder and compromised blood supply to the bladder wall. Both predispose the patient to UTI. Precautions expressly for women include voiding immediately aftersexual intercourse.

Therapy

- Take medication *exactly* as prescribed. Special timing of administration may be required.

- If bacteria continue to appear in the urine, long-term antimicrobial therapy may be required to prevent colonization of the periurethral area and recurrence of infection

Follow up

Follow up patient and emphasize importance of review date

Prevention of recurrent infections

Educate patient on signs and symptoms of cystitis

Complications

- Septicaemia
- Kidney failure
- Scar tissue formation
- Cancer of the bladder

We will now discuss the disorders of the Upper Urinary Tract. We will begin with pyelonephritis.

5Upper Urinary Tract

PYELONEPHRITIS (PYELITIS, NEPHRITIS)

This is inflammation of the renal pelvis and this can be acute or chronic inflammatory process (Smeltzer et al., 2010). The inflammation includes the tubule and interstitial tissue of the kidney. This condition occurs as an ascending infection.

Causes

- Bacterial ascending infections secondary to ureterovesical reflux
- Urinary tract obstruction
- Bladder tumours
- Strictures

- Benign prostatic hypertrophy
- Urinary calculi

Clinical presentation (Acute pyelonephritis)

- Enlarged kidney with interstitial infiltration of inflammatory cells
- Abscess in renal capsule
- Atrophy and glomeruli destruction
- Fever and chills
- Flank pain
- Low back pain
- Abdominal tenderness
- Pyuria
- Bacteriuria
- Leucocytosis
- Painful urination
- Nausea and vomiting

Clinical presentation (chronic pyelonephritis)

- Fatigue
- Headache
- Poor appetite
- Polyuria
- Increased thirst
- Weight loss
- Scarred, contracted and non-functional kidney

Management

Investigations

- Ultrasound of the kidney will show enlarged kidney and obstruction
- Radionuclide imaging to show sites of infection

- Urine culture and sensitivity test to identify the causative organism
- IV pyelogram may be done in acute pyelonephritis if functional and structural renal abnormalities are suspected.
- IV urogram and measurements of creatinine clearance, blood urea nitrogen, and creatinine levels may be performed in chronic pyelonephritis.

Drug therapy

1. Ciprofloxacin 500mg twice daily
2. Ampicillin 500mg four times daily
3. Third generation cephalosporin

Nursing Management

The nursing care of the patient focuses on:

***Pain relief*:** Pain in pyelonephritis is due to the inflammatory process. Antispasmodics may be prescribed to ease on pain. Asprin and application of warm compresses may help in the relieving of pain and spasms.

Fluid intake*:* the patient is encouraged to take a lot of fluid which will help in the flushing out of bacteria and promote renal blood flow. Avoid drinking of irritants such as alcohol and highly caffeinated drinks.

Psychological care*:*, as a nurse you will need to explain to the patient the disease process and treatment outcomes and that resuming normal life is possible if the patient complies with treatment. Allow the patient to verbalise their concer and worries regarding the disease. Involve the significant other in the care of the patient to promote sense of belonging.

Observations: observe the vital signs, temperature, pulse, bloob pressure and respirations to monitor the progress and response to treatment. Observe the urine for the colour, amount and consistency, weigh the patient daily to rule out oedema which may indicate complication like renal failure. Observe the intensive of pain and intervene.

Patient teaching

1. Hygiene
    - Shower rather than bathe in tub because bacteria in the bath water may enter the urethra.

- After each bowel movement, clean the perineum and urethral meatus from front to back. This will help reduce concentrations of pathogens at the urethral opening and the vaginal opening.

2. Fluid Intake
   - Drink liberal amounts of fluids daily to flush out bacteria.
   - Avoid coffee, tea, colas, alcohol, and other fluids that are urinary tract irritants.
3. Voiding Habits
   - Advise the patient to void every 2 to 3 hours during the day and completely empty the bladder. This prevents over distention of the bladder and compromised blood supply to the bladder wall both predispose the patient to UTI. Precautions expressly for women include the following: Void immediately after sexual intercourse. Take the prescribed single dose of an oral antimicrobial agent after sexual intercourse.

4. Therapy

- Take medication *exactly* as prescribed. If bacteria continue to appear in the urine, long-term antimicrobial therapy may be required to prevent colonization of the periurethral area and recurrence of infection. The medication should be taken after emptying the bladder just before going to bed to ensure adequate concentration of the medication during the overnight period.
- For recurrent infection, consider acidification of the urine through ascorbic acid (vitamin C), 1,000 mg daily or cranberry juice.
- If prescribed, test urine for bacteria with recommended test devices, such as dip-slides (Microstix), as follows:
  i. Wash around the urethral meatus several times, using different washcloths.
  ii. Collect a midstream urine specimen.
  iii. Remove a slide from its container, dip it into the urine sample, and return it to the container.
  iv. Incubate the slide at room temperature according to product directions.
  v. Read the results by comparing the slide with the colony density chart provided with the product.
  vi. Begin therapy as directed, and complete the full prescribedcourse of medication.

vii. Notify the health care provider if fever occurs or if signs and symptoms persist.

- Consult the health care provider regularly for follow-up, recurrenceof symptoms, or infections nonresponsive to treatment.

TUBERCULOSIS OF KIDNEY

This is a complication of pulmonary tuberculosis and the cause is mycobacterium bacilli. Usually a blood-borne infection presents with lesions in the renal cortex. It later ulcerates into the renal pelvis and involves the ureters and bladder, epididymis, seminal vesicles and prostate. Calcifications and ureter strictures are common. Other clinical feature include:

- Haematuria
- Malaise
- Fever
- Night sweats. Lassitude, weight loss, loin pain
- Associated genital disease and
- Chronic renal failure. (Boon, Colledge and Walker, 2006)

Take

The investigations and treatment of the condition is the same as for pulmonary tuberculosis and renal diagnostic tests.

GLOMERULONEPHRITIS (ACUTE AND CHRONIC)

Glomerulonephritis also known as glomerular nephritis is a renal disease (usually of both kidneys) characterized by inflammation of the glomeruli. Glomeruli are very small blood vessels in the kidneys that act as tiny little filters - there are about one million glomeruli in each kidney. The disease damages the kidneys' ability to remove waste and excess fluids from the body. It may present with isolated haematuria and /or proteinuria (blood or protein in the urine) or as a nephritic syndrome, acute renal failure, or chronic renal failure. They are categorized into several

different pathological patterns, which are broadly grouped into: non-proliferative or proliferative types.

Primary causes are ones which are intrinsic to the kidney, whilst secondary causes are associated with certain infections (bacterial, viral or parasitic pathogens), drugs, systemic disorders (Systemic Lupus Erythromatosis, vasculitis) or diabetes.

s

Glomerulonephritis is an inflammation of the glomerularcapillaries that can occur in acute and chronic forms (Smeltzer et al., 2010).

Acute Glomerulonephritis

Causes

1. Group A beta hymolytic streptococci infection of the throat may precede the onset of glomerulonephritis by 2 to 3 weeks Post-streptococcal glomerulonephritis - strep infections of the throat
2. Impetigo (a skin infection) may cause glomerulonephritis. Impetigo is a much less common cause than throat infection. As treatment for most streptococcal infections improve, this cause is becoming much less common.
3. Acute viral infection (upper respiratory tract infection, mumps, varriscellazoster virus, epstern barr virus)
4. Medications

Pathophysiology

Acute Glomerulonephritis results from entrapment and collection of antigen-antibody complexes produced as an immunologic mechanism in response to bacteria in the glomerular capillary membranes inducing inflammatory damage and impeding glomerular function. When the glomeruli are inflamed the patient has Glomerulonephritis which can damage the kidneys. Sometimes the damage undermines the kidney's ability to filter blood properly, resulting in an accumulation of waste in the blood stream - kidney failure. The damage may also starve the blood of protein, which ends up being expelled from the body in urine, instead of entering the

bloodstream. The damaged and inflamed glomeruli lose the ability to be selectively permeable, allowing red blood cells proteins to filter through. As the glomerular filtration rate falls, uremic poisoning may result.

Signs and symptoms

- Decreased urination
- Hematuria (blood in urine) due to inflammatory response
- Protenuria due to increased permeability in the glomerular membrane
- Smoky or coffee coloured urine due to bleeding in the upper urinary tract
- Oedema due to decreased glomerular filtration
- Oliguria with output less than 400ml/24hrs
- Patient may also experience shortness of breath, dyspnoea or thopnea. These are symptoms of pulmonary oedema resulting from hypervolaemia.
- Signs of hypertension resulting from sodium or water retention caused by decreased GFR or inappropriate rennin release
- Non-specific symptoms like nausea, malaise, and fatigue

Investigations

- History and physical examinations
- Urinalysis will show protenuria, haematuria and red blood cell casts.
- BUN, serum creatinine and albumin Blood test will show elevated blood urea nitrogen, creatinine will be raised and decreased serum protein.
- Urine test will show proteins, red blood cells and white blood cells
- Throat culture may show group A beta haemolytic streptococci
- Kidney ureter – bladder x-rays will reveal bilateral kidney enlargement but in chronic glomerulonephritis, it shows symmetrically contracted kidneys with normal pelves and calyces
- Kidney ultrasound
- Renal biopsy will establish the underlying disease and provide data to plan therapy

Drugs therapy

Aims of treatment

Treatment is essentially nonspecific and symptomatic. Goals of treatment include:

- To relieve symptoms
- To prevent complications
- To Control hypertension
- Correcting fluid and electrolyte imbalance

1. Antihypertensives such as hydralazine or nifedipine which are vasodilators
2. Angiotensin-converting enzyme (ACE) inhibitors which help to relax the blood vessels, reducing the workload of the heart.
3. Diuretics like furosemides to reduce extracellular fluid overload
4. Antibiotics may be given for symptomatic UTI, however they should be given cautiously
5. Dialysis.

Chronic Glomerulonephritis

Chronic glomerulonephritis is a slowly progressive disease characterized by inflammation of the glomeruli which results in sclerosis, scarring and eventually renal failure. It remains subclinical until the progressive phase begins.

Causes

Primary renal disorders

- Membranoproliferative glomerulonephritis
- Membranous glomerulopathy
- Focal glomerulosclerosis

Systemic disorders that may cause glomerulonephritis include:

- Systemic lupus erythematous
- Goodpatures syndrome
- Haemolytic uremic syndrome

Others include:

- Chronic glomerulonephritis may be due to repeated attacks of acute glomerulonephritis
- Hypertensive nephrosclerosis
- Hyperlipedimia Glumerulo sclerosis
- Chronic tubulointerstitial injuryy, or
- Hemodynamically mediated glomerular sclerosis.

Pathophysiology

The kidneys are reduced to as little as one fifth of their normal size consisting largely of fibrous tissue. The cortex shrinks to a layer 1-2 mm or less. Bands of scar tissue distort the remaining cortex making the surface of the kidney rough and irregular. Numerous glomeruli and their tubules become scared and the branches of the renal artery are thickened. The result is severe glomerular damage that results in end stage renal disease (Smeltzer et al., 2010).

Signs and symptoms

In most patients, chronic glomerulonephritis develops insidiously. However some patients present with non-specific complaints such as:

- Loss of appetite
- Anaemia
- Vomiting or weakness
- The condition may bediscovered when hypertension or elevated BUN and serumcreatinine levels are detected.
- Most patients report general symptoms, such as:
- Loss of weight and strength,

- Increasing irritability,and
- An increased need to urinate at night (nocturia).
- Headaches,
- Dizziness
- Digestive disturbances are also common (Smeltzer et al., 2010).

Investigations

- Routine Urinalysis
- Lab tests may reveal anaemia or signs of reduced kidney
- Ultrasound of the pelvis (Kidney) will show a reduced kidney
- IVP (Intravenous Pyelogram)
- 24 hours urine protein analysis (Total protein)
- Kidney function test (uric acid, Urine concentration, urine creatinine)
- Chest X-ray
- Kidney biopsy confirm the diagnosis

Treatment

- Treatment varies depending on the cause of the disorder, and the type and severity of symptoms. High blood pressure may be difficult to control and it is generally the most important aspect of treatment.
- Angiotensin receptor blockers are most commonly prescribed.
- Medicines that suppress the immune system may also be prescribed, depending on the cause of the condition.
- Plasmapheresis may be done due to immune-related causes. The fluid part of the blood containing antibodies is removed and replaced with intravenous fluids or donated plasma (without antibodies). Removing antibodies may reduce inflammation in the kidney tissue.
- Dietary restrictions on salt, fluids, protein and other substances may be recommended.
- Person with this condition should be closely watched for signs that they are developing kidney failure.
- Dialysis or a kidney transplant may eventually be necessary.

Nursing Interventions

- Provide bed rest during the acute phase.
- Perform passive range of motion exercises for the patient on bed rest.
- Allow the patient to resume normal activities gradually as symptoms subside.
- Consult the dietician about a diet high in calories and low in protein, sodium, potassium and fluids.
- Protect the debilitated patient against secondary infection by providing good nutrition and hygienic technique and preventing contact with infected people.
- Check the patients' vital signs and electrolyte values. Monitor intake and output and daily weight.
- Report peripheral oedema or the formation of ascites.
- Explain to the patient taking diuretics that he may experience orthostatic hypotension and dizziness when he changes positions quickly.
- Provide emotional support for the patient and his family. If the patient is scheduled for dialysis, explain the procedure fully.

Information education and communication

- Advise the patient get immediate treatment for a strep infection that causes a sore throat or impetigo.
- If the patient is diabetic, advise them on the importance of adhering to the treatment plan.
- Emphasize the need for regular blood pressure, urine protein and renal function assessment to detect reoccurrence.
- Stress the importance of follow up examinations to detect complications like renal failure
- Advise the patient to take prescribed anti-hypertensives and diuretics as scheduled, advise the patient to take diuretics during the day so that his night sleep is not disturbed
- Stress the importance of compliance with prescribed diet

- Teach the patient the signs of infection especially those of UTI and advise the patient to report immediately.

Assessment And Physical Examination

Question the patient about an untreated respiratory tract infection that has occurred in the last 1-3 weeks. Ask the patient about the medical history to identify any multisystem diseases. Because patients often describe a history of weight gain and oedema of the hands and face; ask the patient if his or her rings are tight than usual.

Some patients may also describe decreased urine volume, changes in colour (dark, smoky), increased fatigue and activity intolerance, muscle and joint achiness, shortness of breath and orthopnoea. In an elderly patient the symptoms may be more vague and nonspecific such as achiness and nausea.

Note any sign of fluid retention, such as oedema in the face and hands. As you speak to the patient, you may notice dyspnoea and laboured breathing. Inspect the neck veins to determine if engorgement is present. The patient's urine output is usually decreased and is often dark or even coffee colour.

When you auscultate the patient's heart and lungs, you may hear basilar crackles and an S3 heart sound. Most patients have an elevated arterial pressure. Weigh the patient each day and monitor abdominal girth.

Provide on-going monitoring for visual changes, vomiting adventitious breath sounds, abdominal distension and seizure activity. These signs and symptoms indicate the potential onset of the complications and need to be reported to the physician. Patients and families may be anxious about changes in the patients' appearance, an uncertain prognosis, and the possibility of lifestyle changes. Older children and adults may be concerned about their appearance. Assess the patient's and family's coping mechanisms, support systems and stress levels. Most patients with acute glomerulonephritis recover spontaneously. During the acute phase, when urine is grossly hematuric and blood pressure is elevated, the patient is placed on bed-rest and symptoms are managed pharmacologically. A dietary consultation is necessary to implement dietary

restrictions that can manage increased blood pressure, decreased urine output, and the presence of nitrogenous products in the urine.

Usually, sodium and fluid restriction is instituted to manage hypertension and edema. Depending on the course of their disease some patients also need potassium and protein restrictions. If the patient is on fluid restriction, work with the patient and family to devise a schedule of fluid intake that maximizes patient preference and comfort.

Focus on reducing discomfort, reducing complications and providing patient education. Work with the patient to develop a schedule for daily hygiene that limits fatigue and overexertion. Cluster care to provide for rest periods, and assist the patient with relaxation technique.

Complications

- Acute kidney failure: Loss of function in the filtering part of the nephron may cause waste products to accumulate rapidly. This condition may mean you will need emergency dialysis.
- Chronic kidney failure: in this extremely serious complication, the kidneys gradually lose function. Kidney function at less than 10 percent of normal capacity indicates end-stage kidney disease.
- High blood pressure: Damage to the kidneys and the resulting build-up of waste in the blood stream can raise blood pressure.
- Nephrotic syndrome: This is a group of signs and symptoms that may accompany glomerulonephritis and other conditions that affect the filtering ability of the glomeruli. Nephrotic syndrome is characterized by high protein levels in the urine, resulting in low protein levels in the blood.Anaemia due to alteration in the rennin hormone production
- Cardiac hypertrophy
- Congestive cardiac failure due hypertension

Having looked at glomerulonephritis its causes, pathophysiology and management of a patient with this disorder we will shift our attention to yet another disorder nephrotic syndrome.
NEPHROTIC SYNDROME

Activity

In your own words what is nephrotic syndrome?.

Now compare your answer with what your read in the following section.

The term nephrosis, or nephrotic syndrome, had its origin in the early 20th century and was introduced primarily to distinguish it from nephritis, a label used to denote a clinical state associated with haematuria, proteinuria, and a cellular proliferation of the glomerulus. It describes a clinical condition of oedema and proteinuria in which the renal histology (light microscopy) demonstrates fatty degeneration of the tubules associated with normal appearing glomeruli

DEFINITIONS

This is a glomerular disease characterized by the following

- Marked increase in protein in urine (proteinuria)
- Decrease in albumin in blood (Hypoalbuminemia)
- Oedema
- High serum cholesterol and low-density lipoproteins (lipidemia (Smeltzer et al., 2010).

Causes

Nephrotic syndrome has many causes and may either be the result of a disease limited to the kidney, called *primary* nephrotic syndrome, or a condition that affects the kidney and other parts of the body, called *secondary* nephrotic syndrome.

Primary causes

- Membraneous proliferative glomerulonephritis
- Primary nephrotic syndrome
- Focal glomerulonephritis
- Inherited nephrotic disease

Secondary/extrarenal causes

Multisystem disease

- Systemic lupus erythematosus (SLE)
- Diabetes mellitus
- Sarcoidosis
- Amyloidosis

Infections

- Bacterial infections, e.g. streptococcal, syphilis
- Protozoal infections, e.g. malaria
- Viral (hepatitis, Human Immunodeficiency Virus infection HIV

Neoplasms

- Malignancy (cancer) such as Hodgkin's lymphoma
- Leukaemia
- Multiple myeloma

Allergies

- Insect bite
- Poison ivy
- Pollen

Drugs

- Corticosteroids,
- Gold,
- Intravenous heroin
- Drugs, especially NSAIDs in the elderly
    - Captopril

Pathophysiology

Heavy proteinuria (albuminuria) is the hallmark (characteristic/feature) of this condition and the primary abnormality in nephrotic syndrome. The degree of proteinuria varies considerably from patient to patient Some patients will excrete as much as 15 g/m2/24 hours, and the minimal excretion compatible with the diagnosis is around 1 g/m2/24 hours (approximately 40 mg/m2/hour).

The initiating event that produces proteinuria remains unknown. Nephrotic syndrome is believed to have an immune pathogenesis, hence the glomerular capillary permeability to albumin is selectively increased, and this increase in filtered load overcomes the modest ability of the tubules to reabsorb protein. This selective proteinuria is quite different than the more unselective proteinuria observed in cases of glomerulonephritis. Hypoalbuminemia is the result of the increased urinary loss of protein. Other factors, however, may contribute to the Hypoalbuminemia, among them decreased synthesis, increased catabolism, and increased gastrointestinal losses.

Oedema is the classical presentation of the Hypoalbuminemia. The explanation for oedema formation is a decrease in plasma oncotic pressure (as a consequence of low serum albumin) causing an extravasation (leakage and spread of or fluid from vessels into surrounding tissues) of plasma water into the interstitial space. The resulting contraction in plasma volume in turn leads to a decrease in renal perfusion and hence the stimulation of the renin-angiotensin system. This hormonal effect coupled with an increase in the synthesis and secretion of antidiuretic hormone (related to the decrease in effective plasma volume) resultsinto an increase in renal tubular reabsorption of sodium and water. Figure 6 below illustrates the pathophysiology of nephrotic syndrome.

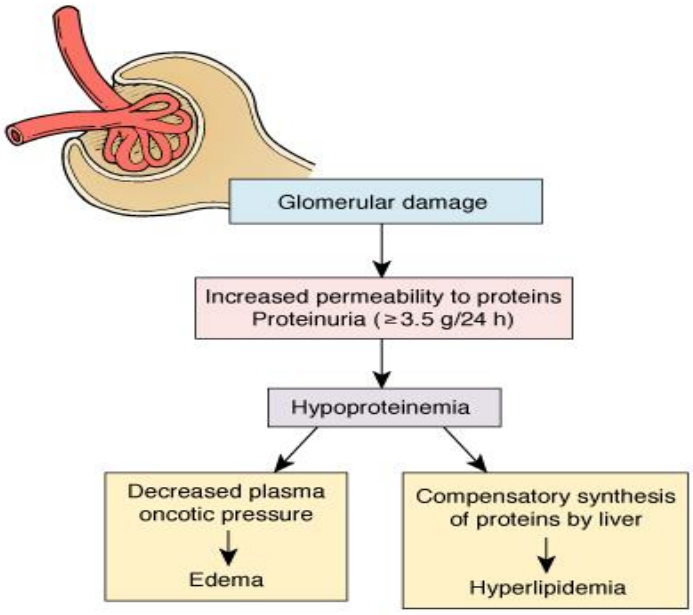

Figure 35-10 Pathophysiology of the nephrotic syndrome.

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Figure 6: Pathophysiology of nephrotic syndrome

Clinical Manifestations

- The history of clinical manifestation is oedema, which is the presenting symptom in about 95% of the patients. The oedema in the early phase is intermittent and insidious; even its very presence may not be appreciated. It is typically dependent in nature, more noticeable in the face in the morning (upon arising) and predominately in lower extremities later in the day. It is pitting in nature. Other signs and symptoms include:
- Severe generalized oedema (Anasarca) that begins in the face.
- Present with gross haematuria.
- Oliguria is a common occurrence whatever the aetiology.
- Anorexia, irritability, fatigue, abdominal discomfort, and diarrhoea are common.
- If ascites is marked, respiratory distress is not uncommon.
- Occasional fever and septic picture;
- The peritoneum is often the site of the infection.
- Streptococcus pneumoniae is the most frequent organism responsible for peritonitis in this population, but Staphylococcus aureus and Escherichia coli are commonly recovered.
- Symptoms of a urinary tract infection are occasionally present.

- A history of prior allergic events is common, and atopy has been reported in approximately 40-50% of children

Physical Examination

- The most common clinical finding is oedema which is present in more than 95% of cases.
- It may be mild and localized only to those areas where tissue resistance is low (e.g., periorbital area, scrotum, labia). Generalized oedema is common and is dependent and pitting in character.
- Ascites is common, and anasarca may be present. In those patients with marked ascites, there may be mechanical restriction to breathing and the child may manifest compensatory tarchypnoea.
- The patient usually demonstrates a pallor that is greater than laboratory evidence of anaemia would suggest.
- Hypertension may be present

Signs of a concurrent upper respiratory tract infection may be present, and some patients will have overt evidence of an atopic state with varying degrees of eczema. Abdominal tenderness is unusual in the absence of a peritoneal infection.

Medical Management

Investigations

The key to determining that renal disease is responsible for the initial clinical presentation is an examination of the urine for protein and cellular elements.

- Hypoalbuminemia is the second of the cardinal laboratory features of NS.Renal function is normal in the majority of patients at onset of the condition, but, approximately 25-30% will have mild to moderate reduction of glomerular filtration rate as evidenced by a rise of serum creatinine above the normal range (>95th percentile for age
- Full blood count -Elevated white blood cell counts are occasionally seen even in the absence of infections.

- Blood chemistry will reveal decreased serum albumin, decreased total serum protein and elevated serum cholesterol.
- Serum protein and lipids analysis this will reveal increased levels of cholesterol, phospholipids, triglycerides and decreased albumin levels
- Renal biopsy can be done to categorize specific kidney tissue damage
- Renal ultrasonography usually reveals normal to slightly enlarged kidneys.

Treatment

Treatment is aimed at preserving renal function. Treatment that appears specific (steroids) and treatment that is non-specific (observation for infections, diet, diuretics, antihypertensive) is discussed here.

Specific Therapy

- Initial management

Glucocorticoid (steroid) therapy has so changed the morbidity and mortality of NS as to make it almost specific and is considered standard.

Oral prednisone or prednisolone is started in a dosage of 2 mg/kg/day (60 mg/m2/d). The total daily dose is usually split into two doses and given daily for 4-8 weeks.

- Maintenance therapy

Maintenance glucocorticoid therapy is still controversial, not about the need for some period of continuing prednisone but about especially the duration that such should be given Thus, the author's/us recommendation for management is as follows:

- 4 weeks: intensive (daily) treatment-as above
- 8 weeks: 1.5 mg/kg/d (one dose every other morning)
- 8 weeks: 1.0 mg/kg/d (one dose every other morning)
- 8 weeks: 0.5 mg/kg/d (one dose every other morning)
- Therapy stopped

Non-specific therapy

Observation for infection : The patient with nephrotic syndrome is a prime candidate for infection, and the potential for dissemination is increased if steroids are administered

indiscriminately. Diuretic therapy may be beneficial, but there is risk of reducing the plasma volume which may lead to acute renal failure. The loop diuretics (furosemide) given orally in usual amounts (20 mg) are safe and moderately effective; If the oedema is sufficiently intense that intravenous diuretic therapy seems indicated, then salt-poor albumin should be infused (usually at 1 gram/kg body weight given IV over 2-4 hours)

Antihypertensive therapy should be given when hypertension is present and particularly if it persists, but caution should be exercised. In some patients the hypertension will respond to diuretics. Angiotensin-converting enzyme inhibitors (ACEI) agents are the preferred therapy even though calcium channel blocking agents have been used effectively in short term therapy e.g. enalapril. Other medications include cyclophosphamide and immunosuppressant's Azathioprine

Nursing Care

Environment: tPatient is nursed in a well-ventilated room, free from infection, reverse barrier nursing is appropriate because of the condition and effects of steroid therapy which are both immunosuppressive.

Position*:* tPatient is nursed in upright position especially with oedema and respiratory distress, but attends to the limbs as well by elevating them on a pillow. Use the airing to relieve pressure off the pressure areas thus preventing pressure sores.

Rest: timportant to improve the immunity

Observations: observe the vital signs especially temperature to rule out infection, pulse and blood pressure to monitor hypertension, urine protein, oedema, respiratory rate, nutrition status, mental status. Weigh the patient everyday preferably on the same scale.

Nutrition*:* the kidneys of patient with active nephrotic syndrome exhibit the usual tubular mechanisms for sodium conservation and total body sodium is uniformly increased. With the addition of prednisone therapy, renal sodium excretion is further curtailed. Because free access to salt is known to increase oedema, dietary salt intake should be restricted. Increase on protein intake.

Psychological care: explain the disease process, importance of medication, explain all procedures of the patient and involve significant others in the care to reduce apprehension and gain co-operation. The person with nephrotic syndrome is often ashamed of the oedematous appearance and they need support in dealing with an altered body image. Reassure the patient that, body image change is as a result of oedema, which may be normal if the oedema reduces.

Exercise: encourage deep breathing exercise especially with pulmonary oedema, change of position to prevent constant pressure on the oedematous skin which is likely to break and excoriate.

Pressure sores: turn the patient in bed, allows the patient to sit up and encourage patient to ambulate to improve blood circulation.

Hygiene: assist patient with activities of daily living, bathing, oral toilet, nail care to avoid skin lacerations due to scratching with long nails.

Medication: as a nurse, ensure that you give the prescribed medications accordingly. You have to know the mode of actions, side effects of the drugs and possible drug interactions.

Patient teaching

Teach the patient to:

1. Take drugs as per schedule as this is the main stay treatment.
2. Take plenty of protein and reduce on salt intake.
3. Avoid infections and seek early medical attention if sick.
4. Avoid injury as wounds may take long to heal due to oedema and compromised immunity.
5. Observe general personal hygiene to avoid infections.

Complications

1. Glomerular lesion
2. Acute renal failure has been noted in individuals with all types of Nephrotic Syndrome
3. Hypertension is a frequent
4. HypoproteinemiaHyperlipidemia is the direct result of increased hepatic production of lipids and lipoproteins and is related to the degree and duration of the hypoproteinemia.

5. Pulmonary oedema due to fluid leak, sometimes it leaks into lungs causing hypoxia and dyspnoea.
6. Growth retardation due to protein deficiency from the loss of protein in urine, anorexia (reduced protein intake), and steroid therapy (catabolism) loss of glucose in the urine.
7. Vitamin D deficiency can occur, vitamin D binding protein is lost.

You have come to the end of nephrotic syndrome and I hope you have learnt valid points in the management of a patient suffering from this condition, the next topic of discussion will be renal failure, take time to read on this topic so that our discussion will be a fruitful one.

RENAL FAILURE (ACUTE/CHRONIC)

As earlier discussed at the end of nephrotic syndrome that our next topic will be renal failure, I welcome you to this discussion and I urge you to participate effectively. We will start by defining renal failure.

Renal failure results when the kidneys cannot remove the body's metabolic wastes or perform their regulatory functions. The substances normally eliminated in the urine accumulate in the body fluids as a result of impaired renal excretion, affecting endocrine and metabolic functions as well as fluid, electrolyte, and acid–base disturbances. Renal failureis a systemic disease and is a final common pathway of many different kidney and urinary tract diseases. (Smeltzer et.al. 2010).

Definition:

Types of kidney failure

Kidney failure may either be acute or chronic depending on the causes

Acute renal failure

Acute renal failure (ARF) refers to the abrupt loss of kidney function over a period offew hours to a few days, with a fall in glomerula filtration rate (GFR) accompanied by a rise in serum creatinine and urea nitrogen.

Acute renal failure (ARF)

Acute renal failure is a rapid loss of renal function due to damage to the kidneys.

Depending on the duration and severity of ARF, a wide range of potentially life-threatening metabolic complications can occur, including metabolic acidosis as well as fluid and electrolyte imbalances. It results from obstruction, reduced circulation or renal parenchyma disease. ARF is often associated with three distinct phases which are Oliguria which is the decrease in urinary output to less than 400mls per day, diuresis and recovery.

Aetiology

The aetiology of acute renal failure is classified into three groups:

1. **Pre-Renal Causes:** Thesecauses interfere with renal perfusion. The kidney depends on an adequate delivery of blood to be filtered by the glomeruli. Therefore, a reduced renal blood flow obviously decreases the GFR.

- Hypovolaemia - resulting from dehydration, hemorrhage, excessive diuresis, diarrhoea and vomiting and burns.
- Decreased cardiac output- resulting from cardiac arrhythmias, cardiogenic shock, congestive heart failure, myocardial infarction, pulmonary oedema and pericardial tamponade.
- Decreased peripheral resistance- due to anaphylaxis, neurologic injury, and some hypertensive drugs.
- Decreased renal vascular blood flow- due to bilateral renal vein thrombosis, embolism and renal artery thrombosis.
- Obstruction

2. **Renal/ Intra Renal Causes**: Refers to parenchymal changes from disease or nephrotoxic substances resulting in impaired nephron function.

- Acute tubular necrosis

- Trauma (Rhabdomyolysis)
- Severe muscle exertion
- Certain genetic conditions
- Infectious diseases
- Metabolic disorders
- Glomerulonephritis
- Vascular lesions

3. **Post-Renal Causes**: Arises from obstruction in the urinary tract, anywhere from the tubules to the urethral meatus.

- Prostatic hypertrophCalculi failure
  - ✓ Urethral strictures
  - ✓ Benign prostate hyperplasia
  - ✓ Bladder cancer
  - ✓ Neuro muscular disorder
  - ✓ Prostate cancer
  - ✓ Spinal cord disease
  - ✓ Strictures
- Trauma (back, pelvis, perineum

Pathophysiology

The damaged tubule cannot conserve sodium normally, which leads to renin-angiotensin aldosterone system activation. Regardless of severity most forms of ARF are reversible. The resulting ischemia may cause an increase in vasopressin, cellular swelling and inhibition of prostaglandin synthesis. The reduced blood flow decreases glomerular pressure, GFR and tubular flow, thus oliguria occurs. Decreased renal blood flow leads to decreased O_2oxygen delivery to the proximal tubules. This will result in increase in metabolic end products such as urea, creatinine, etc.

Phases of Acute Renal Failure

There are four phases of ARF: initiation, oliguria, diuresis, and recovery.

1. The initiation period begins with the initial insult and ends when oliguria develops.

2 The oliguria period is accompanied by an increase in the serum concentration of substances usually excreted by the kidneys (urea, creatinine, uric acid, organic acids and the intracellular cations [potassiumand magnesium]. The minimum amount of urineneeded to rid the body of normal metabolic wasteproducts is 400 ml. In this phase uremic symptoms first appear and life-threatening conditions such as hyperkalemia develop. Some patients have decreased renal function with increasing nitrogen retention, yet actually excrete normal amounts of urine (2 L/day or more). This is the nonoliguric form of renal failure and occurs predominantly after exposure of the patient to nephrotoxic agents, burns, traumaticinjury, and the use of halogenated anesthetic agents.

The diuresis period is marked by a gradual increase in urine output, which signals that glomerular filtrationhas started to recover. Laboratory values stabilize andeventually decrease. Although the volume of urinary output may reach normal or elevated levels, renal function may still be markedly abnormal. Because uremic symptoms may still be present, the need for expert medical and nursing management continues. The patient must be observed closely for dehydration during this phase; if dehydration occurs, the uremic symptoms are likely to increase.

3 The recovery period signals the improvement of renal function and may take 3 to 12 months. Laboratory values return to the patient's normal level. Although a permanent 1% to 3% reduction in the GFR is common,it is not clinically significant.

The effects of ARF are widespread. The major consequences are:

- Fluid and electrolyte imbalances (fluid overload or depletion, hyponatremia, hypocalcaemia and hyperkalemia)
- Acidosis
- Increased susceptibility to infections
- Anemia
- Platelet dysfunction
- Gastrointestinal complications (anorexia, nausea, vomiting, diarrhea, constipation)

- Uremic encephalopathy

General Signs A*nd Symptoms o*f Arf

- Muscle weakness
- Dysrhythmias
- Pruritus
- Oliguria
- Pitting edema
- Hypertension
- Pulmonary edema
- Metabolic acidosis with kussmal respirations (hyperventilation) and pulmonary edema.
- Altered mental state
- Anorexia
- Nausea
- Dry skin
- Headache
- Seizures

Diagnostic Tests

- BUN and Serum creatinine values.
- Creatinine clearance: measures the kidney's ability to clear the blood of creatinine and approximate the glomerular filtration rate.
- Urinalysis
- Renal ultrasound
- Renal scan
- Renal BiopsySerum electrolyte analysis will show increased levels of potassium due to decreased GFR and increased phosphate concentration.
- CT scan or MRI retro grade pyelogram
- Haemoglobin levels will reduce to due to reduced erythropoietin production.

Treatment

The goal of treatment is to remove the precipitating factors, maintain homeostatic balance and prevent complications until the kidneys are able to resume function.

a. Treat the cause
b. Restrict fluids: Replace losses plus 400ml/24hrs.
c. Medications that are handled primarily by the kidney will require modification of dosage or frequency to prevent medication toxicity.
d. Diuretics: In oliguric ARF for fluid removal, e.g. Furosemide or Mannitol.
e. Antihypertensive: to control blood pressure. Aldoment or atenolol
f. Aluminium hydroxide antacids to control hyperphosphatemia.
g. Sodium bicarbonate to control acidosis
h. Intravenous calcium to reverse the cardiac effects of life-threatening hyperkalemia.
i. Vitamins B and C to replace losses if patient is on dialysis
j. Packed cells for active bleeding or if anaemia is poorly tolerated.
k. Give hypertonic glucose and insulin infusion and sodium bicarbonate to treat hyperkalemia
l. Diet: Increase carbohydrates, reduce protein and reduce Potassium and reduce sodium intakes, however, because of loss of K^+ during the diuretic phase, K^+ may need to be increased during that timePeritoneal dialysis or haemodialysis may be done.

Nursing Care

nursing care focuses on the three broad objectives:

- Monitoring for signs of fluid overload
- Maintaining the patient's energy expenditure at a level compatible with the individual's health.
- Controlling or helping to control fluid intake.
- Prevention of superimposed infections

Environment/Prevention Of Infection

Maintain bed rest in the acute phase. Assist patient activities of daily living to conserve energy. Most patients in the acute stage of ARF will be cared for in the intensive care unit because of the

need for constant monitoring of blood pressure, electrocardiogram, pulmonary status and mental status Prevent injury; assess orientation and reorient confused patient. During bed-rest, keep side rails raised and use padded rails as necessary. Avoid sources of infection, limit visitors and maintain asepsis for indwelling catheter. Invasive procedures should be done under strict aseptic measures because the patient has reduced immunity.

Observations

When patient is ambulant, assess motor skills and monitor ambulation, assist patient if necessary. Assess patient for signs of bleeding. Protect patient from bleeding; instruct patient to use soft toothbrush. Assess for signs and symptoms of infection. Assess the level of consciousness (LOC) of the patient. If the patient is in a stuporous state, put him or her in recovery position, and turn him every 2 hours to prevent pressure sores. Assess pressure areas for any sore or swelling. Establish baseline information of vital signs. Take vital signs every after 4 hours. Follow a strict intake t and output programme and record accurately. Monitor weight daily. Observe for indicators of fluid volume excess, including oedema, rayles, and tachycardia. Observe for indicators of volume depletion, including poor skin turgor. Avoid use of indwelling urinary catheters because they are a common source of infection. Use intermittent catheterization instead. Monitor IV fluids carefully. Monitor serum electrolytes and assess for presence of irregular apical pulse indicative of hypokalaemia.

Nutrition/Fluids

Most patients with ARF are too ill to tolerate oral feedings. Oral intake can worsen nausea as a result of the altered biochemical environment and GIT irritation. Nurses, dietician and physicians should work together to institute a diet that provides enough calories to avoid catabolism while preventing a surplus of nitrogen. Catabolism will lead to an increased BUN levels because of the breakdown of muscle for protein.

Protein is restricted to 0.5g/kg of body weight per day. Carbohydrate intake should be maintained at around 100g/day. Na^+ and K^+ are restricted. Fluids are restricted especially in oliguric phase. Provide fluid in form of ice chips to minimize thirst. Dietary supplements are usually prescribed. Patients who are unable to take in sufficient nutrients are given total

parenteral nutrition and administration of fat emulsion which provide a non-protein source of calories.

Hygiene

Provide meticulous skin care. Assist patient with ADL. Bathe patient with super fat soap. Administer antipruritic agents and perform oral care as often as possible.

Skin Care

The skin may be dry or susceptible to breakdown as a result of edema; therefore, meticulous skin care is important. Additionally, excoriation and itching of the skin may result from the deposit of irritating toxins in the patient's tissues. Bathing the patient with cool water, frequent turning, and keeping the skin clean and well moisturized and the fingernails trimmed to avoid excoriation are often comforting and prevent skin breakdown.

Elimination

Monitor and record the quality and number of bowel movements. Provide prescribed stool softeners and bulk-building supplements as necessary. Suggest alternate dietary sources of fibre, such as unsalted popcorn. Provide fleet, oil retention or tap water enemas as prescribed.

Psychological Care

Reassure patient and family that mental capacities will return with recovery of kidney function. Structuring the environment and activities may help with coping in the initial phase. Assist patient to explore feelings concerning the nature of the illness. The patient with ARF may require treatment with hemodialysisor Peritoneal Dialysis, The length of time that these treatments are necessary varies with the cause and extent of damage to the kidneys. The patient and family need assistance, explanation, and support during this period. The physician should explain the purpose of the treatment to the patient and family. However, high levels of anxiety and fear may necessitate repeated explanation and clarification by the nurse. The family members may initially be afraid to touch and talk to the patient during these procedures but should be encouraged and assisted to do so. In an intensive care setting, many of the nurse's functions are devoted to the

technical aspects of patient care; however, it is essential that the psychological needs and other concerns of the patient and family be addressed. Continued assessment of the patient for complications of ARF and precipitating causes is essential.

Patient And Family Teaching

- Teach the patient about the cause of renal failure and problems with recurrent failure.
- Identification of preventable environmental or health factors contributing to the illness, such as hypertension and nephrotoxic drugs is taught.
- Teach patient about medication regimen, including name of medication, dosage reason for taking and side effects.
- Teach patient about prescribed dietary regimen.
- Explain the risk of hypokalemia and to report symptoms (muscle weakness, anorexia, nausea and vomiting, lethargy).
- Teach about signs and symptoms of returning renal failure (decreased urine output, without decreased fluid intake). Teach about signs and symptoms of infection; methods to avoid infection.
- Emphasize the need for ongoing follow-up care.
- Give information about options for future; explanation of transplantation kidney and dialysis if these are a possibility.
- If patient requires dialysis after discharge, co-ordinate discharge planning with dialysis unit staff. Arrange visit to dialysis unit if possible.

CHRONIC RENAL FAILURE

Definition: Chronic renal failure is a progressive, irreversible loss of kidney function that develops over days to years.

Causes Of Chronic Renal Failure

Congenital And Inherited Diseases

- Polycystic kidney disease (infantile or adult)
- Alpot's syndrome

- Congenital hypoplasia

Glomerular Dysfunction

- Glomerulonephritis
- Diabetic nephropathy
- Hypertensive nephrosclerosis

Systemic Diseases

- Sickle cell anaemia
- Systemic lupus erythematosus
- Human Immunodeficiency Virus
- Systemic lupus erythematosus

Urinary Tract Obstruction

- Prostatic and bladder tumours
- Lymphadenopathy
- Urethral obstruction
- Calculi

Others

- Chronic pyelonephritis
- Nephrotic Syndrome
- Renal infarction

In chronic renal failure the damage to the kidneys is progressive and irreversible. Progression of CRF is through four stages:

Decreased renal reserve, renal insufficiency, renal failure and end stage renal disease. **Chronic renal failure progresses into four stages that merge into one another:**

- **Diminished renal reserve**- In this situation the GFR is about 50%efficiency of normal. Serum BUN (Blood Urea Nitrogen) and creatinine values are normal and the patients are asymptomatic. However, they are more susceptible to developing azotaemia with an additional renal insult.
- **Renal insufficiency**- The GFR is 20% to 50%efficiency of normal. Azotaemia appears, usually associated with anaemia and hypertension. Polyuria and nocturia occur as a result

of decreased concentrating ability. Sudden stress (e.g. with nephrotoxins) may precipitate uraemia.

Renal failure – The GFR is less than 20% to 25%efficiency of normal. The kidneys cannot regulate volume and solute composition, and patients develop oedema, metabolic acidosis, and hypocalcaemia. Overt uremia may ensue, with neurologic, gastrointestinal and cardiovascular complications.

- **End – stage renal disease** – The GFR is less than 5% of normal; this is the terminal stage of uremia.

Pathophysiology

The specific pathophysiologic mechanism depends on the underlying disease causing kidney destruction. During CKF some of the nephrons are thought to remain intact while others are destroyed. The intact nephrons hypertrophy and produce an increased volume of filtrate with increased tubular reabsorption, despite a decreased GFR. This adaptive method permits the kidney to function until about three fourths (3/4) of the nephrons are destroyed. The solute load then becomes greater than can be reabsorbed, producing an osmotic dieresis with polyuria and thirst. Eventually as more nephrons are damaged, oliguria occurs, resulting in retention of waste products (uraemia).

Clinical Manifestations

CRF affects all the body systems and signs and symptoms results from disordered fluid and electrolytes balance, alterations in regulatory functions of the body and retention of solutes.

1. Hematopoietic System
    - Anaemia
    - Fatigue
    - Defects in platelet function
    - Thrombocytopenia
    - Ecchymosis
    - Bleeding
2. Cardiovascular System

- Hypervolemia
- Hypertension
- Tachycardia
- CHF

3. Respiratory System
    - Tachypnoea
    - Kussmaul's respirations
    - Uremic halitosis
    - Tenacious sputum
    - Pulmonary oedema
4. Gastrointestinal Tract
    - Anorexia
    - Nausea and vomiting
    - GIT bleeding
    - Abdominal distension
    - Diarrhoea
    - Constipation
5. Neurological System
    - Lethargy and confusion
    - Convulsion
    - Stupor, coma
    - Sleep disturbances
    - Muscle irritability
6. Skeletal System
    - Renal rickets
    - Joint pains
    - Retarded growth
7. Skin
    - Pallor
    - Pigmentation

- Pruritus
- Ecchymosis
- Excoriation
- Uremic frost

8. Urinary System
    - Reduced urine output
    - Reduced urine specific gravity
    - Proteinuria
    - Casts and cells in urine
    - Reduced urine sodium
9. Reproductive System
    - Infertility
    - Reduced libido
    - Erectile dysfunction
    - Amenorrhea
    - Delayed puberty

Diagnostic Tests

- History and physical examination
- BUN and serum creatinine will beboth elevated.
- Creatinine clearance test
- X-ray of the kidneys, ureters and bladder
- IVP depending on the levels of blood chemistry
- Renal ultrasound
- Renal biopsyBlood chemistry (Na^+, K^+, Calcium, Phosphorus, Magnesium and serum PH)
- Urinalysis may show proteins, glycosuria, RBCs, leucocytes casts and crystals depending on the cause.
- Urine culture to isolate the causative organism

Treatment

Initial management of the patient with CRF is focused on controlling symptoms, preventing complications and delaying the progression of renal failure.

Diet: CHO are increased in protein-restricted patients to ensure adequate caloric intake and prevent catabolism. Depending on existing renal function, Na^+ is limited to prevent thirst and fluid retention. K^+ is limited because of the kidney's inability to excrete excess K^+, and protein is limited to minimize retention of nitrogenous wastes.

Fluid Restriction: For patients at risk for developing fluid volume excess fluids are restricted to insensible loss plus urine output only.

Pharmacological

- Aluminium hydroxide or calcium carbonate to control hyperphosphatemia
- Antihypertensive to control BP
- Multivitamins and Folic acid for patients with dietary restrictions or who are on dialysis (H_2O- soluble vitamins are lost during dialysis)
- Ferrous sulphate or recombinant human erythropoietin (Epogen) to treat anaemia. Subcutaneously 3 x per week: 50u/kg body weight.
- Diphenhydramine to treat pruritus.
- Sodium bicarbonate to treat acidosis
- Vitamin D preparations and calcium supplements: to treat hypocalcaemia and prevent bone disease (bone demineralization).
- Packed cells to treat severe or symptomatic anaemia.
- Renal transplantation or dialysis; i.e. if the above therapies are inadequate.

Guidelines For Care (Nursing Care)

1. Maintain Fluid And Electrolyte Balance
   A. Monitor for fluid and electrolyte excess:

- Assess intake and output every 8 hours
- Weigh patient every day
- Assess presence of oedema and extent
- Auscultation breath sounds
- Monitor cardiac rhythm and blood pressure every 8 hours

B. Encourage patient to remain within prescribed fluid restrictions
C. Provide small quantities of fluid spaced over the day to stay within fluid restrictions.
D. Encourage a diet high in carbohydrates and within the prescribed sodium, potassium, phosphorus and protein limits.
E. Administer phosphate-binding agents with meals as prescribed.

2. Prevent Infection And Injury
   a. Promote meticulous skin care
   b. Encourage activity within prescribed limits but avoid fatigue.
   c. Protect person from injury

Nutritional Therapy

Dietary intervention is necessary with deterioration of renal function and includes careful regulation of protein intake, fluid intake to balance fluid losses, sodium intake to balance sodium losses, and some restriction of potassium. At the same time, adequate caloric intake and vitamin supplementation must be ensured. Protein is restricted because urea,uric acid, and organic acids—the breakdown products of dietary nd tissue proteins —accumulate rapidly in the bloodwhen there is impaired renal clearance. The allowed protein must be of high biologic value (dairy products, eggs,meats). High -biologic -value proteins are those that are complete proteins and supply the essential amino acids necessary for growth and cell repair. Usually, the fluid allowance per day is 500 mL to 600 mLmore than the previous day's 24-hour urine output. Calories are supplied by carbohydrates and fat to prevent wasting. Vitamin supplementation is necessary because a protein restricted diet does not provide the necessary complement of vitamins.

Complications

- Hyperkalemia due to decreased excretion, metabolic acidosis, catabolism, and excessive intake (diet, medications, fluids)
- Pericarditis, pericardial effusion, and pericardial tamponade due to retention of uremic waste products and inadequate dialysis
- Hypertension due to sodium and water retention and malfunction of the renin–angiotensin–aldosterone system
- Anemia due to decreased erythropoietin production, decreased RBC lifespan, bleeding in the GI tract from irritating toxins and ulcer formation, and blood loss during hemodialysis
- Bone disease and metastatic and vascular calcifications due to retention of phosphorus, low serum calcium levels, abnormal vitamin D metabolism, and elevated aluminum levels

You have finished looking at renal failure which is both acute and chronic renal failure. Our next topic of discussion will be schistosomiasis; in your spare time please find out all you can on schistosomiasis, so we can discuss the condition effectively.

Activity

1. In your note books write the nursing care for a patient on dialysis (peritoneal and haemodialysis).
2. You will need to visit the renal unit so that you appreciate the procedure of dialysis and how to care for a patient on dialysis.

URAEMIA

Uraemia or **uraemia** denotes "urea in the blood". Urea is one of the primary components of urine. By definition uraemia is an excess of amino acid and protein metabolism end products. It is a complication of renal failure.

Manage this condition as renal failure

http://en.wikipedia.org/w/index.php?search=Pyelitis&title=Special%3ASearch&fulltext=1

http://en.wikipedia.org/wiki/Uremia

SCHISTOMIASIAS

Schistosomiasis is a disease that is caused by parasites (genus Schistosoma) that enter humans by attaching to the skin , penetrating it , Schistosomiasis is a very common disease in the tropics especially swampy areas and farming areas with damand it is the second most prevalent tropical disease in the world. This disease is also known as bilharziasis and snail fever or, in the acute form, Katayama fever. Theodore Bilharz identified the parasite Schistosoma hematobium in Egypt in 1851. The infection is purely parasitic as you will see in the discussion.

Definition: a chronic disease caused by trematodes of the genius schistosoma which infect the large bowel or the urinary bladder depending on the species.

Incidence-This is related to water use.

- Common in low socioeconomic levels
- Those working in irrigation plantation or wading in water
- Development of water projects for irrigation or electricity provides the habitat for the snail vectors

Incubation Period

p4-6 weeks after infection, symptoms appear

Vector Snail

- Schistosoma .haematobium: Bulinus genus
- Schistosoma mansoni: Biomphalaria

Epidemiology

In endemic areas schistosome infection is acquired in childhood. Infection increases in prevalence and intensity with age, peaking in the age group of 15 to 20 years. In older people a drastic decline in intensity but not in prevalence has been demonstrated

Causes

There are three main species of schistomes that infect humans, these are:

- Schistosoma mansoni found in Africa
- Schistosoma haematobium found in Africa
- Schistosoma japonicum found in East Asia.

Others are:

- Schistosoma intercalatum (intestinal)
- Schistosoma Mekongi voge

Pathophysiology (Life Cycle)

Schistomiasis is caused by tissue reaction against the eggs of the schistosome worm. Depending on the species, the eggs are excreted in urine faeces. When the eggs reach water,miracidia hatch out. Miracidia are free swimming larvae which have to reach a snail within 24hours or they will die. In the snail,the miracidia develop and multiply into many cercariae. The cercariae are infective agents of schistosomiasis. They are shed off from the snails in 4 to 7 weeks. They can only live up to 48hours unless they infect man. Man gets infected with cercariae when he wads, bath, play, cultivate or fish in infected water.

The cercariae penetrate the skin and enter the blood stream from where they carried to the liver or the bladder where they develop into adult worms. The adult worms will start laying hundreds of eggs each day. The eggs are the cause of the disease. They lodge in the lining of the urinary bladder (S. haematobium) or in the lining of the bowel (S. mansoni) . Here they cause inflammation, bleeding, scars and after several years even cancer S. Mansoni lives in the mesenteric plexus of the large intestine. Figure 7 below shows the disease progression cyle of schistosomiasis.

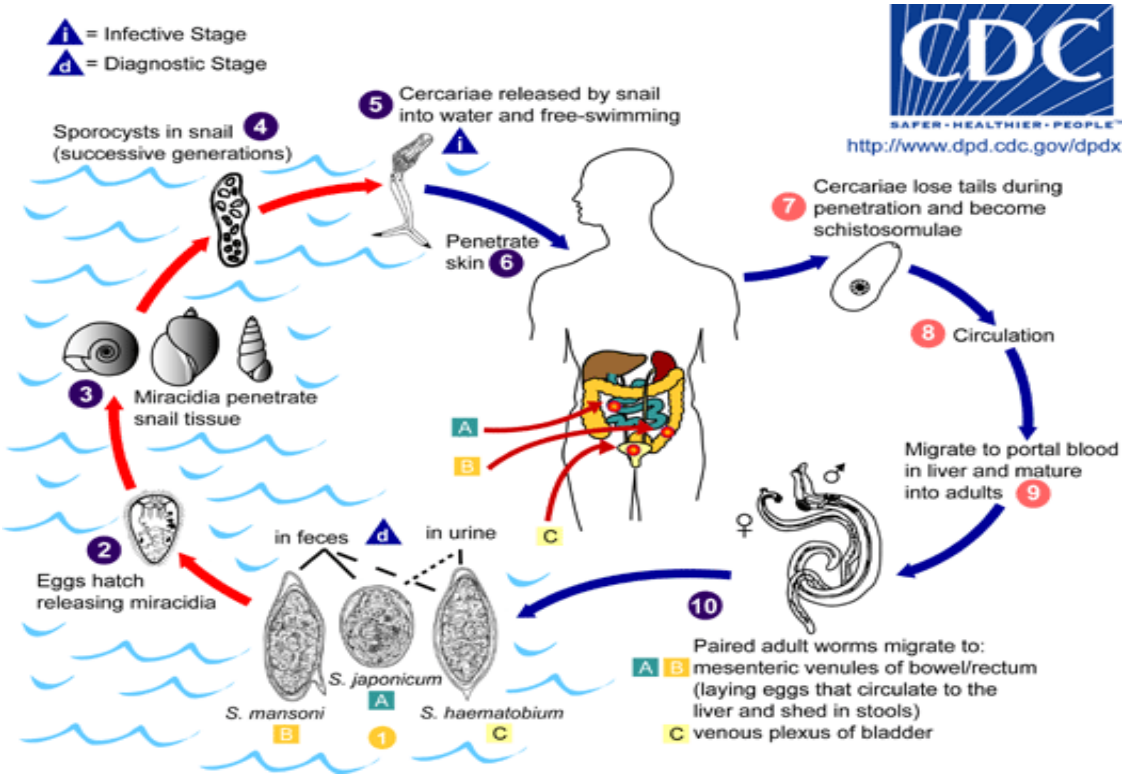

Figure 7: The disease progression cycle of Schistosomiasis

Pathology

The cercaria from the infected snail on penetrating the skin of man gets into the liver as explained in the life cycle. The superficial lesions within the bladder may become numerous and large. They often ulcerate and become infected. Due to erosion of some blood vessels , either in the bladder or intestine, bleeding occurs with some pus. In severe cases, constriction of the urethra may occur with consequent hydronephrosis. In females, the pelvic organs are also affected. In general, organs liable to be affected are the liver, kidneys, intestines, ureters, lungs and the bladder.

Clinical Features

1. Bloody diarrhoea (Mansoni dysentery or in Japonicum)
2. Terminal haematuria (Haematobium)
3. Cercarial dermatitis or swimmers itch; which is localized and local oedema.
4. High fever after a couple of weeks as a result of allergic reaction to the parasites and this is called “KATAYAMA FEVER”.

5. Weakness
6. Tiredness
7. Cough and laboured breathing
8. Loss of weight
9. Abdominal swelling
10. Frequency of micturition

Medical Management

Investigations

- History of blood in urine or stool
- Urine Stix will show blood in urine in S. haematobium.
- Stool/urine for microscopic examination for schistosome eggs
- Pelvic x-ray in case of bladder calcification which is a characteristic of chronic infection.
- Rectal biopsy may demonstrate eggs when stool and urine examinations are negative.

Drugs

1. For S.haematobium: Metrifonate (Bilarcil) 7.5mg/kg body weight once every 2-3 weeks, total of 3 doses.
2. S.Mansoni: Oxamniquine (Vansil) 15-30mg/kg body weight for 1-2 days. In children 20mg/kg body weight.
3. Praziquantal (Biltricide) 40mg/kg body weight one dose; Treats both mansoni and haematobium.
4. Niridazole (Ambilhar) 25mg / kg in divided doses for 7 days

Preventive And Control Measures

1. Reduction or elimination of vector snail: Application of a chemical in stagnant water to kill the snails.
2. Elimination of snail habitant by burying quarries or pools of water.
3. Sanitation measures to prevent human excreta from contaminating local water sources; e.g. use of toilets, repairing all leaking sewer pipes.

4. Provision of safe fresh water supplies to reduce contact with snail infestated water sources
5. Use of protective foot wear or clothing during cultivation in rice pads or when wadding in water. Or use of medicated salves to prevent the cercareal from reaching the skin.
6. Use of periodic drugs to limit infection intensity in exposed populations.
7. Mass-screening in endemic areas and treating those that show symptoms

Complications

1. Cancer of urinary bladder
2. Cancer of liver
3. Liver cirrhosis
4. Malnutrition
5. Anaemia
6. Retardation of growth and development
7. Cystitis and urethritis
8. Glomerulonephritis

Let us now review what you have learnt.

In this unit your,who have these

learn

SELF TEST MCQ

1. Cystitis is said to be
   - **a.** Inflammation of the gall bladder
   - **b.** Infection of the urinary bladder
   - **c.** Obstruction of the urinary bladder
   - **d.** Inflammation of the small bowels

2. The cause of urinary schistosomiasis is

a. Schistosoma Japonicum

b. Plasmodium Vivax

c. Schistosoma Mansoni

d. Schistosoma Haematobium

1.

a.

b.

c.

d.

2.

a. cuss is a common cause of UTI

b. ichia coli is a common cause

c.

MATCHING QUESTIONS

The the conditions in column I with the statements in column II

3. Glomerulanephritis — a) Inability of the kidney to adequately excrete toxic substances from the body.

4. Cystitis — b) Characterised by protenuria, oedema and hypolipidemia

5. Schistosomiasis — c) It is caused by group A beta haemolytic streptococci

6. Nephrotic syndrome — d) Presents with terminal haematuria

7. Renal failure — e) Inflammation of the bladder

Answers: **MCQ**, Q1 B. . Q3 D

MATCHING Q3C, Q4E, Q5D, Q6B, Q7

1.5 Summary

You have now come to the end of unit 1 and you learnt the urinary system. You started by reviewing the anatomy and physiology of the urinary system then you looked at the investigations and procedures done in the Urinary system disoders and the roles of the nurse during these investigations. Further you discussed the conditions that affect the system and how to manage them. Conditions such as cystitis, pyelonephritis, renal failures etc. are some of the common conditions that were discussed and you are likely to encounter them and as a nurse you need to fully be equipped with this knowledge in order to help patients as you see them.

In the next unit you will be learning about conditions that affect the nervous system and how to manage them. Below is the self-test for you to attempt.

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UNIT 2: NERVOUS SYSTEM

2.1 Introduction

Welcome to the second unit 2 of medicine II. In the previous unit we discussed the urinary system and the management of various disease/conditions of this system. In this unit you will

focus on diseases/conditions that affect the nervous system. The nervous system is a system that controls and regulates a lot of functions in our bodies. It is therefore important to understand this system and the diseases/conditions that affect it so that you can provide effective management to clients/patients with such conditions. Let us start by reviewing our objectives for this unit.

2.2 Objectives

1. Describe the anatomy and physiology of the nervous system
2. Conduct a neurological assessment
3. Explain the role of the nurse in investigations and procedures of the nervous system
4. Describe the management of patient with neurological disorders

2.3 Applied Anatomy And Physiology

The nervous system detects and responds to changes inside and outside the body.
It coordinates with the endocrine system to control the body function hence maintaining homeostasis.It consists of a vast number of cells called neurones. Its stimulation provides an immediate response while that of the endocrine is slower and prolonged.
The brain is divided into three major areas:

1. The cerebrum,

The brain stem,

2. The cerebellum

Figure 1 below shows the main structures of the brain.
Let us further look at each area in turn.

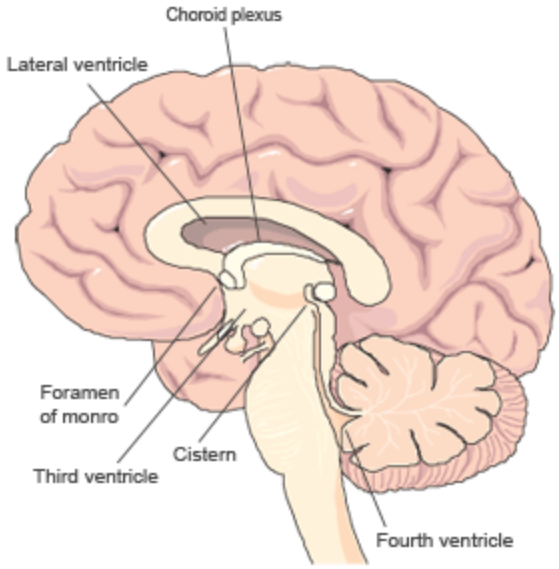

Figure 8: Diagram showing parts of the brain

Cerebrum

The cerebrum consists of two hemispheres that are incompletely separated by the great longitudinal fissure. This sulcus separates the cerebrum into the right and left hemispheres. The two hemispheres are connected at the lower portion of the fissure by the corpus callosum. The outside surface of the hemispheres has a wrinkled appearance that is the result of many folded layers or convolutions called gyri, which increase the surface area of the brain, accounting for the high level of activity carried out by such a small-appearing organ.

- The external or outer portion of the cerebrum (the cerebral cortex) is made of grey matter; it contains billions of neurons/cell bodies, giving it a grey appearance. White matter makes up the innermost layer and is composed of nerve fibres and neuroglia (support tissue) that form tracts or pathways connecting various parts of the brain with one another (transverse and association pathways) and the cortex to lower portions of the brain and spinal cord (projection fibres). The cerebral hemispheres are divided into pairs of frontal, parietal, temporal, and occipital lobes. The four lobes are as follows:
    a. Frontal—the largest lobe. The major functions of this lobe are concentration, abstract thought, information storage or memory, and motor function. It also contains Broca's

area, critical for motor control of speech, affect judgement, personality and inhibitions.

b. Parietal—largely sensory lobe in function. The primary sensory cortex, which analyses sensory information and relays the interpretation of this information to the thalamus and other cortical areas, is located in the parietal lobe.

c. Temporal—it has the auditory receptive areas. It contains a vital area (interpretive) area that provides integration of somatization, visual, and auditory areas and plays the most dominant role of any area of the cortex in cerebration.

- Occipital—this is the posterior lobe of the cerebral hemisphere and it is responsible for visual interpretation.

The basal ganglia are masses of nuclei located deep in the cerebral hemispheres that are responsible for control of fine motor movements, including those of the hands and lower extremities.

The thalamus lies on either side of the third ventricle and acts primarily as a relay station for all sensation except smell. All memory, sensation, and pain impulses also pass through the thalamus.

The hypothalamus is located in front but below the thalamus. The hypothalamus lies immediately beneath and lateral to the lower portion of the wall of the third ventricle. It includes the optic chiasm (the point at which the two optic tracts cross) and the mamillary bodies (involved in olfactory reflexes and emotional response to odours). The infundibulum of the hypothalamus joins it to the posterior pituitary gland. The hypothalamus functions to regulate the pituitary secretion of hormones that influence metabolism, reproduction, stress response, and urine production.

The hypothalamus works with the pituitary to maintain fluid balance and maintains temperature regulation by promoting vasoconstriction or vasodilatation. Found in the hypothalamus is the site of the hunger centre and appetite control. Present are also centres that regulate the sleep–wake cycle, blood pressure, aggressive and sexual behaviour, and emotional responses. The autonomic nervous system is also controlled by the hypothalamus.

The pituitary gland is located in the sella turcica at the base of the brain and is connected to the hypothalamus. The gland is known for cancerous changes that often present in adults and detected by physical signs and symptoms that include hormonal imbalance or visual disturbances that are secondary to pressure on the optic chiasm.

Cerebellum.

3. and
4. The cerebellum which composes two hemispheres, the thalamus, the hypothalamus, and the basal ganglia. It also has connections for the olfactory (cranial nerve I) and optic (cranial nerve III) nerves are found in the cerebrum.

The brain stem includes the following structure

1. The midbrain
2. The pons
3. The medulla
4. The connections of cranial nerves II and IV through XII.

The cerebellum is located underneath the cerebrum and posterior the brain stem. The brain accounts for about 2% of the total body weight; it weighs approximately 1,400 g on average in a young adult (Hickey, 2003 in Brunner and Suddarth, 2010). In the elderly, the average brain weighs approximately 1,200g.

The cerebellum is separated from the cerebral hemispheres by a fold of dura mater, the tentorium cerebelli. The cerebellum has both excitatory and inhibitory actions and is largely responsible for coordination of movement. It also controls fine movement, balance, **position sense** (awareness of where each part of the body is), and integration of sensory input Brunner and Suddarth, 2010).

The Brain stem:

The brain stem, is located beneath the limbic system. It is responsible for vital life functions such as breathing, heartbeat, and blood pressure. It

Self test questions

1. How many areas is the brain divided into
   a. 2 b. 4 c. 3
2. The cerebellum is one of the three main areas of the brain.
   a. True b. False
3. The stem of the brain includes the medulla oblongata
   a. True b. False

Answers to the self test questions

1. c
2. a
3. a

Below is a further discussion on the protection to the brain

Protection To The Brain

Bone structure

The brain is contained in a vault called skull, which protects it from injury. The following bones make the skull frontal, temporal, parietal, and occipital bones which are joined together by suture lines.

Covering of the brain

The meninges (fibrous connective tissues that cover the brain and spinal cord) provide protection, support, and nourishment to the brain and spinal cord. The meningeal coverings are:

- Dura mater—the outermost and tough layer that covers the brain and the spinal cord.
- Arachnoid— a thin middle membrane which is delicate membrane that closely resembles a spider web (hence the name arachnoid).
- Pia mater—the innermost membrane; a thin, transparent layer that closely fixed to the brain. The space between the arachnoid and pia matter is the subarachnoid space in which the CSF flows.

You can remember the coverings of the brain by using the acronym **DAP**

Cerebrospinal Fluid (Csf)

CSF, a clear and colourless fluid with a specific gravity of 1.007, andis produced in the ventricles and is circulated around the brain and the spinal cord through the ventricular system. In the brain are fourventricles namely

- The right and left lateral, and
- The third and fourth ventricles.

  The two lateral ventricles open into the third ventricle at the interventricular foramen and the third ventricle open into the fourth ventricle.

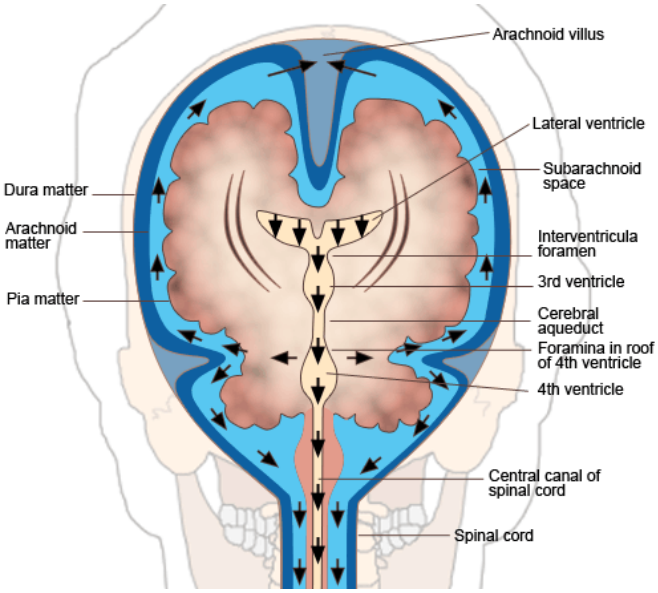

Figure 9: Showing covering of the brain and CSF flow

Cerebral Circulation

The cerebral circulation receives approximately 15% of the cardiac output, or 750 mL per minute. Typically the brain does not store nutrients therefore; it requires constant blood flow to meet its high metabolic demands. In comparison to other organs that may tolerate decreasein blood flow, the brain lacks additional collateral blood flow, which may result in irreversible tissue damage when blood flow is obstructed for long periods.

Arteries. Two internal carotid arteries and two vertebral arteries and their vast system of branches provide the blood supply to the brain. At the base of the brain surrounding the pituitary gland, is a ring of arteries called the circle of Willis which formed from the branches of the internal carotid arteries, anterior and middle cerebral arteries, and anterior and posterior communicating arteries.

Veins. Venous drainage for the brain does not follow the arterial circulation as in other body structures. The veins reach the brain's surface, join larger veins, then cross the subarachnoid space and empty into the dural sinuses, which are the vascular channels lying within the tough dura mater. The network of the sinuses carries venous outflow from the brain and empties into the internal jugular vein, returning the blood to the heart. Cerebral veins and sinuses are unique because, unlike other veins in the body, they do not have valves to prevent blood from flowing backward and depend on both gravity and blood pressure (Smeltzer et al, 2010).

Blood–Brain Barrier

Access to the Central Nervous System is practically impossible because of a barrier known as the blood brain barrier. This barrier blocks off many substances (eg, dyes, medications, and antibiotics) that would otherwise reach the brain cause irritation. The barrier is formed by the endothelial cells of the brain's capillaries, which form continuous tight junctions, creating a barrier to macromolecules and many compounds. All substances following along with CSF must be filtered capillary endothelial cells and astrocytes.

Anatomy Of The Spinal Cord

The spinal cord and medulla form a continuous structure extending from the cerebral hemispheres and serving as the connection between the brain and the periphery. The spinal cord is almost 45cm long and about the thickness of a finger, it extends from the foramen magnum at the base of the skull to the lower border of the first lumbar vertebra, where it forms a fibrous band called the conus medullaris. Continuing below the second lumbar space are the nerve roots that extend beyond the conus, which are called the cauda equine. Just like the brain, the spinal cord consists of grey and white matter. Gary matter in the brain is external and white matter is

internal; in the spinal cord, grey matter is in the centre and is surrounded on all sides by white matter and surrounded by the meningies encased in the vertebral column.

The spinal cord is an H-shaped structure with nerve cell bodies (grey matter) surrounded by ascending and descending tracts (white matter). The lower portion of the H is broader than the upper portion and corresponds to the anterior horns. The anterior horns has cells with fibres that form the anterior (motor) root end and are essential for the voluntary and reflex activity of the muscles they innervate.

Sensory and Motor Pathways: The Spinal Tracts. The white matter of the cord is composed of myelinated and unmyelinated nerve fibres. The fast-conducting myelinated fibres form bundlesthat also contain glial cells. Fibre bundles with a common function are called tracts. There are six ascending tracts. Two conduct sensation, principally the perception of touch, pressure, vibration, position, and passive motion from the same side of the body. Before reaching the cerebral cortex, these fibres cross to the opposite side in the medulla. The two spinocerebellar tracts conduct sensory impulses from muscle spindles, providing necessary input for coordinated muscle contraction. They ascend essentially uncrossed and terminate in the cerebellum.

The Peripheral Nervous System

The peripheral nervous system includes the cranial nerves, the spinal nerves, and the autonomic nervous system.

Cranial Nerves

There are 12 pairs of cranial nerves that emerge from the lower surface of the brain and pass through the foramina in the skull. Refer to neurologic examination on page 56.

Spinal Nerves

The spinal cord is composed of 31 pairs of spinal nerves: 8 cervical, 12 thoracic, 5 lumbar, 5 sacral, and 1 coccygeal. Each spinal nerve has a ventral root and a dorsal root.

Autonomic Nervous System

The **autonomic nervous system** regulates the activities of internal organs such as the heart, lungs, blood vessels, digestive organs, and glands. Maintenance and restoration of internal homeostasis is largely the responsibility of the autonomic nervous system. There are two major divisions: the **sympathetic nervous system**, with predominantly excitatory responses, most notably the "fight or flight" response, and the **parasympathetic nervous system**, which controls mostly visceral functions (Smeltzer et al, 2010).

Read and review the anatomy of the CNS

Now that we have finished discussing the applied anatomy and physiology, we are now going to see how we can assess the client's neurological status.

2.4 Neurological Assessment

The assessment in this sub unit refers to the activities you need to carry out in order to make a diagnosis on a client who has neurological complaints. To do this we need to define the terminologies used and identify the parts of the assessment we need to consider when carrying out the activities.

Definition: a method of obtaining specific data in relation to the function of a patient's nervous system and is typically performed by the attending physician (Restrepo, 2014).

Requirements (for the requirements, refer to your procedure manual).

Health History

An important aspect of the neurologic assessment is the history of the present illness. The first interview provides an opportunity to systematically explore the patient's current condition and related events while simultaneously observing overall appearance, mental status, posture, movement and affect. Depending on the patient's condition, as a nurse may need to rely on question and answer information, a review of the medical record, or input from the family or a combination of these.

The health history therefore includes details about the onset, character, severity, location, duration, and frequency of symptoms and signs; associated complaints; precipitating,

aggravating, and relieving factors; progression, remission, and exacerbation; and the presence or absence of similar symptoms among family members (Smeltzer et al., 2010)

The neurologic examination will focus on the testing of cranial nerves and major functions assessment. I am sure you remember the cranial nerves from your Anatomy and physiology.

I Olfactory Sensory Smell Have patient identify a familiar scent with eyes closed (usually deferred).

II Optic Sensory Vision (acuity and field of vision). Pupil reactivity to light and accommodation (afferent impulse).Have patient read from a card, one eye at a time. Test visual fields by having patient cover one eye, focus on your nose, and identify the number of fingers you are holding up in each of four visual quadrants.

III Oculomotor- Motor Eyelid elevation Pupil size and reactivity (efferent impulse). Check pupillary responses by shining a bright light on one pupil; both pupils should constrict (consensual reflex). Do the same for the other eye. To check for accommodation, move your finger toward the patient's nose; the pupils should constrict and converge.

IV Trochlear this nerve allows turning of eyes downward and laterally. Have patient look down and in.

V Trigeminal allows us to both Chew, Facial and mouth sensation Corneal reflex (sensory)
Ask patient to hold the mouth open while you try to close it and to move the jaw laterally against your hand. With patient's eyes closed, touch her face with cotton and have her identify the area touched. In comatose patients, brush the cornea with a wisp of cotton; the patient should blink.

VI Abducens Motor - allows turning of eyes laterally. Have the patient move the eyes from side to side.

VII Facial allows both Facial expression, Taste Corneal reflex (motor) Eyelid and lip closure. Ask patient to smile, raise eyebrows, and keep eyes and lips closed while you try to open them. Have patient identify salt or sugar placed on the tongue.

VIII Acoustic Sensory Hearing and equilibrium. To test hearing, use tuning fork, rub your fingers, or whisper near each ear.

IX Glossopharyngeal Both Gagging and swallowing (sensory) and taste

Touch back of throat with sterile tongue depressor or cotton-tipped applicator. Have patient swallow.

X Vagus - Both Gagging and swallowing (motor), speech (phonation). Assess gag and swallowing with CN IX and assess vocal quality.

XI Spinal accessory Motor Shoulder movement, head rotation. Have patient shrug shoulders and turn head from side to side.

XII Hypoglossal Motor Tongue movement, speech (articulation). Have patient stick out tongue and move it internally from cheek to cheek and assess articulation (Restrepo, 2014).

TABLE 6-6

Sensory Examination

Clinically, there are two major somatosensory pathways that are examined. The first is the spinothalamic (ST) part of the anterolateral system, and the second is the dorsal column-medial lemniscus (DCML) system. The principle sensory modalities for the ST system are pain and temperature. The principle sensory modalities for DCML system are vibratory, position sense, and discriminatory or integrative sensation. Spinal cord and lower brainstem lesions can result in **sensory dissociation**, which means one sensory system is affected but the other is not. Sensory evaluation is performed by having the patient respond to stimuli at a specific location. It evaluates the ability to perceive and identify specific sensations with the patient's eyes closed. The patient must be able to cooperate with the examination by communicating whether or not the sensation is felt and whether both sides of the body feel it equally. The assessment of light touch, pinprick, and temperature sensation can be achieved by applying a cotton swab, clean pin, and a cold or warm object, respectively, to various parts of the body. The clinician should begin with the patient's feet and move upward. Comparing one side with the other is valuable in localizing the specific site of abnormality. To test vibratory sensation, use a low-frequency tuning fork.

To test **proprioception**, or position sense, have the patient with his eyes closed distinguish whether his finger or toe are moved up or down. Patients should be able to discriminate between two different points 2 to 10 mm apart on their fingers and hands and up to 75 mm on their thigh and back.

Graphesthesia examines the patient's ability to identify numbers written on their palm and ***stereognosis*** tests for recognition of objects placed in their hands with their eyes closed.

Motor Examination

A bedside neurologic assessment almost always includes an evaluation of motor function. Because the clinician assesses the patient's ability to move on command, they must be awake, willing to cooperate, and able to understand what the examiner is asking. Motor strength is assessed bilaterally by having the patient flex and extend his/her arm against your hand, squeezing your fingers, lifting his/her leg while you press down on the thigh, holding his/her leg straight and lifting it against gravity, and flexing and extending his/her foot against your hand.

Each extremity is graded by using a motor scale from 0 (no movement) to +5 (full range of motion with full strength). In an unconscious patient, the assessment of motor response is performed by applying a noxious stimulus and observing the patient's response to it. Central stimulation, such as sternal pressure, produces an overall body response and is more reliable than peripheral stimulation. In an unconscious patient, peripheral stimulation, such as nail bed pressure, can elicit a reflex response, which is not a true indicator of motor activity. If central stimulation is necessary, it should be performed judiciously because deep sternal pressure can easily bruise the soft tissue above the sternum. A less traumatic alternative to sternal pressure is to squeeze the trapezius muscle. Supraorbital pressure should not be used for central stimulation on patients with facial fractures or vagal nerve sensitivity. The response to pain varies depending on the level of neurologic function. Normally, pain causes the patient to attempt to remove the source of the pain or to withdraw from the painful stimulation. If the cerebral cortex is functioning, there is a withdrawal from painful stimuli in a predictable and reflexive manner. The symmetry and pattern of the motor response to noxious stimuli, as well as associated neurologic symptoms, should be documented for all patients suspected of having a neurologic disease.
Subtle central weakness (such as with early CNS malignancy) can be tested via **pronator drift.**

Ask your patient to hold his/her arms forward with palms up. In mild cortical weakness, the patient's hand on the weak side pronates and drifts down. The motor system is usually reduced to

the direct corticospinal tract or **upper motor neuron** (UMN) and **lower motor neuron** (LMN). The corticospinal tract has its main influence on the motor neurons that innervate the muscles of the distal extremities such as the hand and the foot.

The clinical findings from an UMN lesion include loss of distal extremity strength, dexterity, and a Babinski's sign (see the next section) plus increased tone, hyperreflexia, and the clasp-knife phenomenon (from loss of control of the indirect brainstem centers). An UMN lesion above the level of the red nucleus will result in **decorticate posture** (thumb tucked under flexed fingers in fisted position, pronation of forearm, and flexion at elbow with the lower extremity in extension with foot inversion), whereas a lesion below the level of the red nucleus will result in **decerebrate posture**, in which the upper extremity is in pronation and extension and the lower extremity is in extension. The reason for this is that the red nucleus output reinforces antigravity flexion of the upper extremity. When its output is eliminated, the unregulated tracts reinforce extension tone of both upper and lower extremities. If there is a lesion in the medulla, all of the brainstem motor nuclei, as well as the direct corticospinal tract, would be out and the patient would be acutely flaccid. If the patient were to survive, tone would return because of interneuronal activity at the spinal cord level (Restrepo, 2014)

Deep Tendon Reflexes

Deep tendon reflexes evaluate spinal nerves and include the triceps, biceps, brachioradialis, patellar, and the Achilles tendon. Although deep tendon reflexes are not routinely assessed, they should be tested in any patient with a spinal cord injury or symptoms consistent with a neurologic problem.

The **patellar reflex** or "knee-jerk" is tested by tapping on the patellar tendon with a reflex hammer while the patient's leg hangs loosely at a right angle with the thigh. Normally, the lower leg jerks forward when this reflex is intact. The absence of this reflex is known as **Westphal's sign**. The reflexes are graded on a scale from 0 to 5+, with 0 being no reflex, 2+ being normal, and 5+ being hyperreflexia with clonus (repeated rhythmic contractions). Abnormal or absent deep tendon reflexes indicate abnormalities in anatomic components required for the reflex arc to occur. These structures include the muscle, the nerve fibers going from the tendon to the spinal

cord, and the nerve fibres returning from the spinal cord to the muscle. **Myastheniagravis** and **botulism** are diseases characterized by abnormal deep tendon reflexes caused by abnormalities of the neuromuscular junction that impair the normal impulse transmission. Absent deep tendon reflexes may be a sign that the patient is at risk for respiratory failure (Restrepo, 2014).

Superficial Reflexes

The **plantar reflex** is the only superficial reflex that is commonly assessed and should be tested in comatose patients and in those with suspected injury to the L4 to L5 or S1 to S2 areas of the spinal cord. To assess the plantar reflex the examiner strokes the lateral plantar aspect of the foot with the handle of a reflex hammer or thumbnail. The stroke should begin at the heel and move up the foot, in a continuous motion, along the outer aspect of the sole and then across the ball to the base of the big toe. The normal response is plantar flexion (curling under) of the toes. Dorsiflexion of the great toe with fanning of remaining toes, **Babinski's sign**, is abnormal, except in children up to 12 to 18 months of age. The presence of Babinski's sign could indicate a brain disease caused by damage to the corticospinal tract (Restrepo, 2014).

Brainstem Reflexes

Brainstem reflexes are evaluated in stuporous or comatose patients to determine if the brainstem is intact. Protective reflexes, such as coughing, gagging, and the corneal response, are usually evaluated as part of the assessment of the CNS.

Gag Reflex

Cranial Nerves IX and X are especially important to thebecause they control a variety of functions. CN IX controls the muscles of swallowing that are needed to prevent aspiration. This function of CN IX is evaluated by testing the patient's **gag reflex**.

This test is performed by gently inserting a tongue depressor into the back of the throat. Although some healthy individuals have a minimal or absent gag reflex, its absence may increase the risk for aspiration and endotracheal intubation may be necessary to protect the lungs. CN IX also has a branch that extends to the carotid sinus and plays a major role in the control of blood pressure. The ability to cough with suctioning can be tested in an intubated patient and implies

an intact CN X. This test should not be attempted on nonintubated patients in the ICU because of the risk of aspiration. Stimulation of CN X while suctioning the airway may result in the presence of bradycardia caused by to vagal stimulation. This could become clinically significant in hemodynamically unstable patients (Restrepo, 2014)

Pupillary Reflex

Pupillary light reflexes provide information regarding the status of the brain and the sympathetic and parasympathetic nervous systems. Pupillary function is controlled by the midbrain and evaluates CNS II and III. **Pupillary reflex** is determined by briefly passing a bright light in front of both open eyes while carefully watching the iris in both eyes for movement. Pupil size, congruency, and response to light and accommodation should be described. The acronym **PERRLA** is commonly used to refer tonormal **pupils** that are qual, ound, and eactive to ight and ccommodation (movement). Any visible change in the pupils' size is noted. **Anisocoria** is a neurologic term indicating that one pupil is larger than the other. **Mydriasis**, or pupillary dilation, may be caused by serious brain injury or inadvertent exposure of the eyes to inhaled anticholinergics. **Miosis** or small "pinpoint" pupils usually result from pontine (Restrepo, 2014). hemorrhage or from ingestion of narcotics or organophosphates. Pupillary responses almost always remain intact in metabolic causes of coma. Mid position and fixed pupils often indicate severe cerebral damage.

Corneal Reflex

The **corneal reflex** is used to test the afferent CN V and the efferent CN VII. The test is performed by lightly touching the cornea with a cotton swab. The normal response is that the patient should blink both eyes. The presence of this response implies an intact ipsilateral fifth cranial nerve, intact central pons, and intact bilateral seventh cranial nerves. Testing must be performed bilaterally to evaluate both afferent components of the fifth cranial nerve. Most clinicians omit the corneal reflex test unless there is sensory loss on the face as per history or examination, or if cranial nerve palsies are present at the pontine level.

Oculocephalic and Oculovestibular Reflexes

Abnormalities of extraocular movement (CN II, III, IV, and VI) have prognostic importance in the ICU. 3 Normal movement of the eyes requires an intact midbrain connection. The resting position of the gaze, the presence of **nystagmus**, and the response to head movements and coldtympanic membrane stimulation should be identified.To test the **oculocephalic reflex** or **doll's eyes** reflex,turn the patient's head briskly from side to side the eyesshould turn to the left while the head is turned to the right and vice versa. If this reflex is absent, there will be no eyemovement when the patient's head is moved side to side.Cervical spine stability must be ensured before oculocephalicmaneuvers are performed. To test the **oculovestibular reflex,** also known as the ice caloric or cold caloricreflex, a physician instils at least 20 ml of ice water intothe ear of a comatose patient. In patients with an intact.
brainstem, the eyes will move laterally toward the affected ear. In patients with severe brainstem injury, the gaze will remain at midline (Restrepo, 2014).

Coordination, Balance, And Gait Exam

The principal area of the brain that is examined by the coordination, balance, and gait examination is the cerebellum. Cerebellar dysfunction results in decomposition of movements and undershooting and overshooting of goal-directed movements (dysmetria). Decomposition of movement and dysmetria are the main elements of ataxia.

Dysfunction of different systems of the cerebellum may result in a myriad of signs and symptoms that include nystagmus, truncal instability (titubation), truncal ataxia, ataxia of speech (scanning dysarthria), and ataxia of the extremities (appendicular ataxia). Ataxia caused by disease of the cerebellar hemispheres will be ipsilateral to the dysfunctional hemisphere.

Cerebellar assessment may not be necessary in a problemfocused examination, and it cannot be done if the patient cannot or does not follow commands. Coordination may be simply assessed by holding up your finger and having the patient quickly and repeatedly moving his/her finger back and forth from your finger to his/her nose. Ask the patient to alternately touch his/her nose with his/her right and left index fingers. Finally, have the patient repeat these tasks with his/her eyes closed. The movements should be rapid, smooth, and accurate.

Balance can be assessed using the Romberg test if the patient is able to stand and is not restricted to bed. Have the patient stand with his/her feet together, arms at the sides, and eyes open; the patient should be able to stand upright with no swaying. If the patient can do that, have him/her close his/her eyes and stand the same way. If the patient falls or breaks the stance after closing the eyes, the Romberg test is positive, indicating proprioceptive or vestibular dysfunction.

All levels of the neural axis contribute to **gait**, although most gait abnormalities are motor in nature. In assessing gait, it is important to watch not only the lower extremities but also the upper extremities for normal associated movements. To assess gait, ask the patient to walk without shoes around the examining room or down the hallway, first with his/her eyes open, then closed. A smooth, regular gait rhythm and symmetric stride length is expected (Restrepo, 2014).

Vital Signs And The Nervous System The nervous system is intricately connected to the mechanics of respiration. From the cerebral cortex to the LMNs, the nervous system regulates respiratory effort. Automatic breathing is regulated primarily by lower brainstem nuclei via the **pneumotaxic** and **apneustic** autonomic respiratory centers. The most vital neurons are located in the ventral respiratory group (VRG) of the medulla. The VRG, the dorsal respiratory group (DRG), and the pontine respiratory group (PRG) form the pontomedullary regulatory generator. This generator works at a subconscious level and results in rhythmic contraction and relaxation of the respiratory muscles but may be modified by pulmonary and cardiovascular reflexes. Lesions at various levels from the cerebrum to the upper cervical cord cause abnormal changes of the breathing pattern.

The most common abnormal respiratory pattern seen in patients with neurologic disorders is **Cheyne-Stokesrespiration**, which consists of phases of hyperponea that regularly alternate with episodes of apnoea (Restrepo, 2014).

1. Intracranial Pressure Monitoring
2. There are three primary reasons to measure **intracranial pressure** (ICP):
3. To monitor patients at risk oflife-threatening intracranial hypertension
4. To monitorfor evidence of infection.

5. To assess the effectsof therapy aimed at reducing ICP. Mean ICP of a supinepatient is normally 10 to 15 mm Hg.

Although small fluctuationsare normal during the cardiac cycle, variabilitygreater than 10mmHg is suggestive of serious neurologic compromise. Elevations in ICP to 15 to 20 mm Hg compress the capillary bed and compromise microcirculation. At ICP levels of 30 to 35 mm Hg, venous drainage is impeded and oedema develops in uninjured tissue. Even when autoregulatory mechanisms are intact, cerebral perfusion cannot be maintained if ICP increases to within 40 to 50 mm Hg of the mean arterial pressure (MAP). When ICP approximates MAP, perfusion stops and the brain dies.

Finally neurologic examination will be used to assess the mental status of a patient (Restrepo, 2014).

Self test on neurological assessment:

1. How many cranial nerves have we discussed?
   a. 10 b. 12 c. 15
2. Olfactory nerve is intracranial nerve number…….
   a. 6 b. 2 c. 1
3. To monitor evidence of infection is one of the reasons intra cranial pressure is done
   a. True b. false

Now that we have finished assessing our client's neurological state we also need to look at what the nurse' role during investigations and procedures as she/he works in the next unity.

What do you think is the role of the nurse in investigations and procedures of the nervous system?

2.5 Role Of The Nurse In Investigations And Procedures

- nsnervousCerebral **Angiography**

In this procedure a catheteris inserted into an artery usually the femoral artery and indirectly threaded up to the carotid artery. Then, a radiopaque dye is injected, which allows X-ray visualization of the cerebral vasculature.

The nurse is responsible for patient preparation, before the procedure. She/ he put up the IV line if required and prepares the papers where the physician will write.

- Myelography

This follows a lumbar puncture and CSF removal, then a radiologic dye is instilled. X-ray determines spinal cord compression related to back pain or extremity weakness and show spinal abnormalities.

The role of the nurse is related to patient welfare. Cleanliness and the general preparation of the patient before the procedures are done.

- Echoencephalography ECG)

This diagnostic technique determines if the midline structures in the brain have shifted, indicating a lesion. The role of the nurse remains preparatory.

- Electroencephalography (EEG)

This is a test that is used to screen the brain abnormalities and visualises the electrical brain wave pattern disturbances or anatomical alterations. Abnormal results indicate organic disease

Nurse's role is to prepare the client for the procedure to include a total hygiene care.(Loeb S. et al1993)

- **Lumbar puncture-** this collection of cerebral spinal fluid at 3rd and 4th lumber spaces on the vertebral column. The procedure is sterile procedure carried out by the doctor and your role as a nurse to firstly explain the procedure to the patient and reassure the patient that the procedure is for diagnostic purposes. The nurse prepares the necessary equipment needed (procedure manual) and places the patient in the preferred position (lateral position with flexed knees and neck to allow for maximum exposure of the lumber region.

Cryptoccoco Antigen Test (CAT)

Test done to detect the cryptococcal organism if present in a CSF in the laboratory.

Radiological Examinations

The nurse explains the procedure to the patient and reassures the patient concerning the procedure. The nurse makes an appointment with the radiology department. Patient preparation requires that all metallic objects be removed from the patient and internal devices and other metal implants must be noted, as these will be captured as abnormalities on the films. A patient with internal devices (pace-makers) may not under MRI and other alternatives of imaging may

be necessary as MRI interferes with the function of such devices. Below are some radiologic examinations:

- Computer Tomography Scanning (CTSCAN)- special x-ray imaging and images recorded on compact discs
- Computerized Axial Tomography (CAT SCAN)- special x-ray imaging and images recorded on compact discs
- Magnetic Resonance Imaging (MRI)- special imaging machine which uses high magnetic field to capture images.

We have come to the end of the sub unit. Answer the following questions before you move to management of patient with neurological disorders.

1. CTSCAN	a. MAGNETIC RESEARCHIMAGING
2. CATSCAN	b. COMPUTERISED AXIL TOMOGRAPHY
3. MRI	c. COMPUTER TOMOGRAPHY SCANING

2.6management Of A Patient With Neurological Disorders

The sub unit looks at how you will manage the patient with neurological disorders. We and these includes' maigraine headaches, CVA, brain tumours, epilepsy and other diseases affecting the nervous system**.**

MIGRAIN (HEADACHE)

Definition: Thisis a symptom complex characterized by periodic and recurrent attacks of severe headache. The cause of migraine has not been clearly demonstrated, but it is primarily a vascular disturbance that occurs more commonly in women and has a strong familial tendency. The typical time of onset is puberty, and the incidence is highest in adults 20 to 35 years of age (Smeltzer et al, 2010). OR

It is a syndrome characterized by severe pain on one side or both sides of the head (which may transfer from one side of the head to the other) accompanied by nausea, dizziness and visual disturbances caused by dilatation and constriction of blood vessel in the head.

Clinical Manifestations

i. Fatigue, nausea and vomiting and fluid imbalance precede headache by about a day.
ii. Sensitivity to light and noise (most prominent feature).
iii. Unilateral or bilateral, aching or throbbing headache which last longer than in classic migraine and last 6 to 48 hours
iv. Numbness, tingling, or weakness
v. Warning signs (auras) that can precede a migraine include seeing stars or zigzag lines, tunnel vision, or a temporary blind spot.
vi. Symptoms that may linger even after the migraine has gone away include:
vii. Feeling mentally dull, like your thinking is not clear or sharp
viii. Increased need for sleep
ix. Neck pain

However, migraine with aura can be divided into four phases: prodrome, aura, the headache, and recovery (headache termination and postdrome).

Prodrome. The prodrome phase is experienced by 60% of patients with symptoms that occur hours to days before a migraine headache. Symptoms include depression, irritability, feeling cold, food cravings, anorexia, change in activity level, increased urination, diarrhoea, or constipation. Patients usually experience the same prodrome with each migraine headache.

Aura Phase. Aura occurs in up to 31% of patients who have migraines (Goadsby et. al., 2002 andSmeltzer ea tl, 2010). The aura lasts less than an hour and may provide enough time for the patient to take the prescribed medication to avert a full-blown attack (described ina later section). Aura is characterized by focal neurologic symptoms. Visual disturbances (i.e. light flashes and bright spots)are common and may be hemianopia (affecting only half of the visual field). Other symptoms include numbness and tingling of the lips, face, or hands; mild confusion with slight weakness of an extremity, drowsiness and dizziness.

Aura corresponds to the painless vasoconstriction which the initial physiologic change characteristic of classic migraine.

Headache Phase. As vasodilation and a decline in serotonin levels occur, a throbbing headache (unilateral in 60% of patients) intensifies over several hours. This headache is severe and

incapacitating and is often associated with photophobia, nausea, and vomiting. Its duration varies, ranging from 4 to 72 hours

(Goadsby et al., 2002 and Smeltzer, 2010)

Recovery Phase. In the recovery phase (termination and postdrome), the pain gradually subsides. Muscle contraction in the neck and scalp is common, with associated muscle ache and localized tenderness, exhaustion, and mood changes. Any physical exertion exacerbates the headache pain. During this post headache phase, patients may sleep for extended periods.

Medical Management

Therapy for migraine headache is divided into two phases

1. Abortive (symptomatic) and
2. Preventive approaches.

The abortive approach, best used in patients who suffer less frequent attacks, it is aimed at relieving or limiting a headache at the onset or while it is in progress.

The preventive approach is used in patients who experience more frequent attacks at regular or predictable intervals and may have medical conditions that preclude the use of abortive therapies (Evans & Lipton, 2001 and Smeltzer et al, 2010).

Drug therapy

Serotonin receptor agonists e.g. Ergotamine

1 – 2mg at onset repeated after 30 minutes if necessary. Maximum 8mg in 24 hours at intervals of not less than 4 days. Maximum 10 tablets per week.) The drug is effective in aborting the headache if taken early in the migraine (MoH, 2013).

Nursing Management

When migraine has been diagnosed, the goals of nursing management are to enhance pain relief. It is reasonable to try non-pharmacologic interventions first, but the use of pharmacologic agents should not be delayed. The goal is to treat the acute event of the headache and to prevent recurrent episodes. Prevention involves patient education regarding precipitating factors, possible lifestyle or habit changes that may be helpful and pharmacologic measures.

Relieving Pain

Individualized treatment depends on the intensity of the migraine Nursing care is directed toward treatment of the acute episode. A migraine in the early phase requires abortive medication therapy instituted as soon as possible.Analgesics like acetaminophen, ibuprofen, or aspirin are often used to relieve headache.

Home And Community-Based Care

Teaching Patients Self-Care. Headaches, especially migraines, are more likely to occur when the patient is ill, overly tired, or stressed. Non-pharmacologic therapies are important and include patient education about the type of headache, its mechanism (if known), and appropriate changes in lifestyle to avoid triggers.

Regular sleep, meals, exercise, avoidance of peaks and troughs of relaxation, and avoidance of dietary triggers may be helpful in avoiding headaches.

Patients and their families need to be reminded of the importance of following the prescribed treatment regimen for headache and keeping follow-up appointments. In addition, they are reminded of the importance of participating in health promotion activities and recommended health screenings to promote a healthy lifestyle.

You have just finished learning about migraine headache.You will now look at cerebral vascular accident (CVA).

CEREBRAL VASCULAR ACCIDENT (CVA)

Definition: is a sudden loss of function resulting from disruption of the blood supply to a part of the brain (Smeltzer et al, 2010). In other words it is the sudden disruption of O_2 supply to the nerve cells, generally caused by obstruction or rupture in one or more of the blood vessels that supply the brain.

Cerebral Vascular Accident (CVA) is a sudden interruption of cerebral circulation in one or more of the blood vessels supplying the brain resulting in permanent or temporary neurological dysfunction lasting longer than 24hours.

Activity:2.6.1

In your own understanding what are the causes of CVA?

Write douwn your answers in your note book and compare with the notes afterwards.

Causes

Stroke also now known as “brain attack” is be caused by three different lesions in the cerebral arteries, all of which produce a similar clinical picture:

1. **Cerebral Thrombosis**: In elderly people, the cerebral arteries are affected by arteriosclerosis in which the lining of the arteries becomes thickened and roughened. The flow of blood is obstructed and clotting occurs. This clot (thrombus) blocks the artery and deprives part of the brain of its blood supply.
2. **Cerebral Haemorrhage**: Rupture of a blood vessel produces haemorrhage into the brain. This event is more common in case of hypertension.
3. ***Cerebral Embolism*:** An embolus, or detached clot, may lodge in one of the cerebral arteries and produce a stroke. This variety of stroke is seen in diseases where a clot forms on the left side of the heart and is carried up in the blood stream to lodge in one of the cerebral vessels. The diseases which most frequently cause a clot in the left side of the heart are:
    - Mitral stenosis with atrial fibrillation
    - Myocardial infarction
    - Subacute bacterial endocarditis

Transient Ischemic Attack (Tia)

A transient ischemic attack (TIA, or ministroke) is a brief episode of symptoms similar to a stroke. It is caused by a temporary decrease in blood supply to part of the brain. Most attacks last just a few minutes. TIA has the same cause as an ischemic stroke but in contrast to a stroke, which involves a more prolonged lack of blood supply and causes some permanent damage to the brain tissue. TIA does not leave lasting effects to the brain but there is alikelihood of putting one at a greater risk of a full-blown stroke that could cause permanent damage.

Risk Factors

- Age: 60-75% of all strokes occur in person over 65 years of age.
- Sex: Men have a slightly increased incidence of stroke.
- Race: It is believed that Negroes are more prone.
- Hypertension: Hypertension is a major risk factor for stroke, particularly in combination with atherosclerosis.
- Heart disease: One of major contributor to stroke both from atherosclerosis and emboli.

- Diabetes Mellitus: Associated with microvascular and macrovascular changes that contribute to atherosclerosis.
- Others are; includecigarette smoking, oral contraceptive use (especially if also a smoker), alcohol intake, family history and previous stroke or TIA.

Pathophysiology

The brain must receive a steady supply of nutrients from the blood because it has no capacity to store either O_2 or glucose. It is supplied with blood from two major pairs of vessels, the internal carotids and vertebrals.

The complex processes of cerebral auto regulation maintain blood flow to the brain at a fairly constant rate of 750mls per minute. The cerebral vessels dilate and constrict in response to changes in blood pressure and carbon dioxide tension. Ischemia will cause primary death of cerebral cells or cerebral infarction, which creates a core of necrotic tissues. There is secondary area of tissue damage in which cells are temporarily unable to function but may remain viable. Ischemia will cause the following:

- Impaired movement of calcium and potassium. High levels of calcium are believed to trigger the activation of enzymes that attack neuron cell membranes.
- The accumulation of O_2 free radicals, which further disrupt calcium metabolism.
- The presence of glucose is low: perfusion area enhances lactate production, which worsens cellular damage and acidosis.
- An influx of fluid-activated white cells and coagulation factors further clog the microcirculation.

Stroke associated with haemorrhage is primarily related to an abrupt rise in intracranial pressure and ischemia followed by cerebral oedema. With intracerebral bleeding blood is forced into the adjacent brain tissue, where a hematoma forms. This will result in compression of tissue and even result in brain tissue displacement or herniation.

Let us look how the patient with CVA presents.

Clinical Manifestations

Specific symptoms will reflect the site and severity of ischemic damage.

Motor Effects

- Hemiparesis or hemiplegia of the side of the body opposite the site of ischemia.

- Initially flaccid, progressing to spastic.
- Dysphagia : swallowing reflex may also be impaired
- Dysarthria

Bowel And Bladder

- Frequency, urgency and urinary incontinence
- Constipation
- Bowel incontinence

Language

- Aphasia: difficulty or inability to express self verbally or difficulty or inability to comprehend speech.
- Alexia: inability to understand written word
- Agraphia: inability to express self in writing

Sensory-Perceptual

- Diminished response to superficial sensation i.e. touch, pain, pressure, heat and cold.
- Diminished proprioception: loss of awareness of where various body parts are in relationship to each other and the environment.

Cognitive-Emotional

- Emotional lability and unpredictability
- Depression
- Memory loss
- Short attention span
- Loss of reasoning,(judgment and abstract thinking ability)

As the cerebral edema increases; there will be changes in mentation including apathy, irritability, disorientation, memory loss, withdrawal, drowsiness, stupor or coma.

Other clinical features are:

- Numbness or loss of sensation
- Weakness or paralysis on part or one side of the body.
- Headache, neck stiffness and rigidity
- Vomiting
- Seizures

- Dizziness or syncope

Management

Medical Management

A. Investigations
   a. CT scan: To reveal site of infarction, hematoma and shift of brain structure.
   b. MRI: Magnetic Resonance Imaging to reveal site of infarction
   c. EEG: Shows abnormal nerve impulse transmission
   d. Lumbar Puncture for CSF analysis: Not done routinely especially if there is IICP.
   e. Cerebral Angiography: To pin point site of rupture or occlusion and identify collateral blood circulation.
   f. History may reveal risk factor e.g. hypertension
   g. Coagulation studies may show coagulation problems

Medical Treatment

Medical management commonly includes physical rehabilitation, dietary and drug regimes and care measures to help patient adapt to specific deficits such as speech impairment and paralysis.

- *Respiratory Support*: Maintenance of air-way and delivery of O_2 as needed. Intermittent positive pressure breathing (IPPB) and chest physiotherapy are important.
- *IV fluids:* To maintain fluid and electrolyte balance. Fluids may be limited while IICP is a risk.
- *Positioning:* Bed rest during acute stage. Activity level is increased as patient's condition improves. Elevate HOB at 30° as prescribed. HOB may be kept up with hemorrhagic stroke or patients with IICP to decrease cerebral perfusion and improve venous outflow.

Diet: NPO status and possible gastric tube if swallow and gag reflexes are diminished or if patient has decreased LOC. A low-Na^+ and lowand - fat diet may be prescribed to minimize other risk factors.***Pharmacotherapy***

- Tissue plasminogen activator.
- Patients with stroke can benefit from an injection of tissue plasminogen activator (TPA).TPA is a potent clot-busting drug that helps patients to recover more fully.However, the drug can only be given to patients within a three-hour window of the

stroke occurring, and it can only be given in situations in which doctors are certain that giving TPA will not worsen bleeding in the brain.

- Anticoagulants: In patients with CVS or transient Ischemic attacks Heparin sodium and Warfarin Sodium to help prevent further thrombosis.
- Antihypertensive agents e.g. Nifedipine to control very high Blood pressure, which may cause cerebral edema and IICP.
- Antiplatelet medications e.g. Aspirin in conjuction with dipyridamole or Sulfinpyrazone; to prevent platelet aggregation that may lead to thrombus formation.
- Glucocorticosteroids: e.g. Dexamethasone and Osmotic diuretics (Mannitol) to prevent or reduce cerebral oedema.
- Antacids and histamine H_2-Receptor blockers (e.g. ranitidine) to reduce the risk of GI hemorrhage from gastric ulcer caused by stress.
- Ant epilepsy drug e.g. phenytoin or Phenobarbital; to control and prevent seizures.
- Sedatives/Tranquilizers e.g. diphenhydramine; to promote rest. These are used cautiously to avoid further impairment of neurologic function.
- Analgesics e.g. Acetaminophen; to control headache
- Stool softeners e.g. Acetaminophen to control headache
- Stool softeners e.g. Docusate or Bisocondayle to prevent straining which can result in IICP

Hemodilution: e.g. albumin, Crystalloid fluids. Hydration is promoted via fluids and volume expanders to decrease blood viscosity in order to improve cerebral blood flow.

Activity:2.6.2
How do you think the patient with CVA is nursed?

Nursing Care

AIMS

1. To prevent complications associated with stroke
2. To maintain a patent airway
3. To provide a nutritious diet
4. To bring back the patient to his/her normal or near normal functional state.

The nursing care of a patient with stroke calls for total nursing care in which all activities of daily living are done on behalf of the patient.

Environment/Position

The patient is best nursed in the intensive care unit where he/she will be closely monitored during the acute/critical stage.

The patient is placed on bed rest with the head of the bed elevated at 30° and positioned well, to prevent the tongue from falling back. Supplemental O_2 may be administered preferably 100% O_2 from the cylinder.

The environment is kept as quiet and restful as possible and all activities that are known to increase intracranial pressure, such as coughing, straining, lying prone, muscle contraction, emotional upset and abrupt head or neck flexion are avoided or minimized.

The patient is assisted to change positions every 2 hours and encouraged to move independently in bed as soon as possible. The affected arm is positioned with the hand elevated above the wrist and the wrist above the elbow to support venous return and minimize oedema.Shoulders are placed in neutral position with support as needed. Pillows, rolled towels and sand bags are used to support normal body alignment with particular attention to preventing external rotation of the hip.

Special care to avoid excess pressure or pull on the shoulder joint, which is extremely vulnerable to joint subluxation and adduction contractures should be put in place.

Heels should be elevated off the mattress to avoid pressure injury and foot positioning to prevent foot drop.

Hands are splint firmly. Supine position is avoided to prevent aspiration. A side-lying position with the head of the bed elevated 10 to 20° should be maintained.

Pressure area care and prevention of sore formation with the use of elbow and heel protection is done.

Observation

Immediately after the patient's admission the focus of nursing care is on monitoring the patient's neurological status and preventing complications, while assessing the severity of stroke. Vital signs and neurological checks are performed regularly to rule out the presence of IIP. The Glasgow coma scale is used to assess the level of consciousness and neurological responses. Assess for signs of pressure, shearing or friction damage during each position change. Change

the patient's position every 2 hourly and record on the chart. Ensure strict input and output recording. The patient will have an indwelling urethral catheter, therefore assess for any presence of infection. Get urine samples for examination.

Nutrition And Fluids

Depending on the condition of the patient, feeding may be through NGT. Assess the swallowing reflexes of the patient. Ensure that the tube is in the right place to prevent chocking the patient.

A highly nutritious diet should be given through an NGT such as milk, soup, custard and light porridge. Ensure that feeds are at an acceptable temperature; prevent burning the gut of the patient. Fluids are strictly given to prevent cerebral oedema.

Elimination

Ensure that the patient is not constipated. Stool softeners may be given to prevent straining.

Patient may have uncontrolled bowel movements, ensure that a barrier-cream is applied to the buttock (perineal area) to prevent skin excoriation.

Hygiene

Since the patient depends totally on the nurse, all hygienic needs should be done for her, i.e. bedpans, pressure area care, mouth care and catheter toilet. Ensure that the patient is in a dry, clean environment.

Exercises And Rehabilitation

The rehabilitation plan incorporates active physical therapy, but the nurse needs to incorporate avariety of intervention into the patients daily care routines.

Positioning is fundamental to preventing complications such as contractures and skin breakdown. Coughing and deep breathing exercises and frequent position changes prevent pooling of mucus and encourage ventilation of all areas of the lungs. Encourage patient with hemiplegia to exercise while they are still in bed not only prepares them for later activities but also offers hope and a sense of optimism about recovery.

Perform passive range of motion (ROM) exercises four times daily after the first 24hours following a stroke. Frequent ROM prevents joint immobility, tendon contractures, and muscle

atrophy and weakness. They also stimulate circulation and help re-establish neuromuscular pathways.

Help patient out of bed as soon as it is medically permitted. Involve physiotherapist to assist with exercises. Help patient use walker with guidance/ use of crutches. Assist patient to be on a wheel chair or use a wheel chair.

Speech therapist should be consulted to re-train the patient on speech if it has been affected. The client may be taught to use the other hand which is not affected.

Health Education

1. If the patient is to be discharged home, the family needs clear understanding of the residual deficits. The family and patient need to have realistic expectations about the patient's abilities.
2. Emphasis on the need of physiotherapy if there's residue disability as rehabilitative measures
3. Patient should avoid high cholesterol diet and high Sodium intake.
4. Patient has to reduce weight if he is obese.
5. Reduce or stop smoking/alcohol beverages. Patient has to avoid prolonged bed rest and stressful life-styles.
6. Involve Speech Therapist if available to assist patient with speech problems.
7. Teach patient and family on drugs,diet and importance of reducing weight if obese.
8. Emphasize the importance of review dates in order to promote full recovery.

Complications

1. Contractures-disabilities
2. Paralysis-limb of facial
3. Pressure sores
4. Pneumonia
5. Speech defects
6. Altered gait

Activity 2.6.3

Identify five problems a patient with CVA may have, and manage them using the nursing care

plan.

Well as you can see, there are many conditions affecting the nervous system.You have just finished looking at CVA. Am sure there people in your community h=who have suffered from it and you can now give the good IEC for them to stay healthy.

Let us now look at brain tumours.

BRAIN TUMORS

A **brain tumour** is an intracranial solid neoplasm, within the brain or the central spinal canalwhich can either be cancerous (malignant) or non-cancerous (benign). Brain tumours include all tumours inside the cranium or in the central spinal canal. They are created by an abnormal and uncontrolled cell division, usually in the brain itself, but also in lymphatic tissue, in blood vessels, in the cranial nerves, the brain envelopes the meninges, skull, pituitary gland, or pineal gland. Within the brain itself, the involved cells may be neurons or glial cells which include astrocytes, oligodendrocytes, and ependymal cells. Brain tumours may also spread from cancers primarily located in other organs (metastatic tumors).

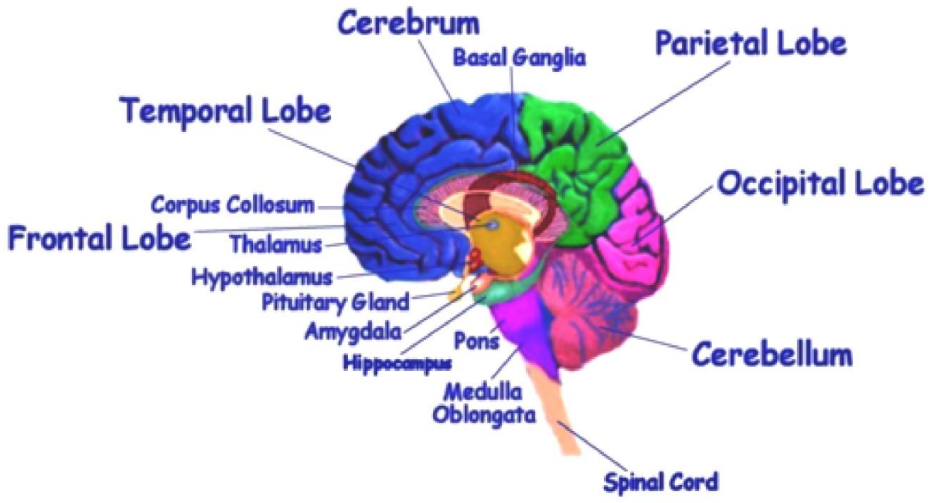

Figure 10: The structures of the brain

Causes /predisposing factors s

A risk factor is something that may increase the chance of getting a disease. Some of the risk factors for brain tumours are:

Ionizing radiation: Ionizing radiation from high dose x-rays (such as radio therapy from a large machine aimed at the head) and other sources can cause cell damage that leads to a tumour. People exposed to ionizing radiation may have an increased risk of a brain tumour, such as meningioma or glioma.

Family history: It is rare for brain tumours to run in a family. Only a very small number of families have several members with brain tumours.

Metastatic cancers are far more common than primary tumors of the brain and spinal cord.

Mutations and deletions of so-called tumour suppressor genes are thought to be the cause of some forms of brain tumors.

Types Of Brain Tumours

Primary brain tumours can be benign or malignant:

Benign brain tumoursBenign brain tumors do not contain cancer cells:Usually, benign tumours can be removed, and they seldom grow back.Benign brain tumors usually have an obvious border or edge. Cells from benign tumours rarely invade tissues around them. They don't spread to other parts of the body. However, benign tumours can press on sensitive areas of the brain and cause serious health problems.

Unlike benign tumours in most other parts of the body, benign brain tumours are sometimes life threatening.

Benign brain tumours may become malignant.

Malignant brain tumours

Malignant brain tumors also called brain cancer contain cancer cellsMalignant brain tumours are generally more serious and often are a threat to life.

They are likely to grow rapidly and crowd or invade the nearby healthy brain tissue.

Cancer cells may break away from malignant brain tumours and spread to other parts of the brain or to the spinal cord. They rarely spread to other parts of the body.

Tumour Grading

Physicians group brain tumours by grade. The grade of a tumour refers to the way the cells look under a microscope and these include the following;

Grade I: The tissue is benign. The cells look nearly like normal brain cells, and they grow slowly.

Grade II: The tissue is malignant. The cells look less like normal cells than do the cells in a Grade I tumour.

Grade III: The malignant tissue has cells that look very different from normal cells. The abnormal cells are actively growing (anaplastic).

Grade IV: The malignant tissue has cells that look most abnormal and tend to grow quickly.

Cells from low-grade tumours (grades I and II) look more normal and generally grow more slowly than cells from high-grade tumours (grades III and IV).

Over time, a low-grade tumour may become a high grade tumour. However, the change to a high-grade tumour happens more often among adults than children.

Among adults, the most common types are:

Astrocytomas: The tumour arises from star – shaped glial cells called astrocytes. It can be any grade. In adults, an astrocytoma most often arises in the cerebrum.

Grade I or II astrocytoma: It may be called a low-grade glioma.

Grade III astrocytoma: It's sometimes called a high-grade or an anaplastic astrocytoma.

Grade IV astrocytoma: It may be called a glioblastoma or malignant astrocytic glioma.

Meningioma: The tumour arises in the meninges. It can be grade I, II, or III. It is usually benign (grade I) and grows slowly.

Oligodendroglioma: The tumour arises from oligodendrocytes,cells that make the fatty substance that covers and protects nerves. It usually occurs in the cerebrum. It's most common in middle – agedadults. It can be grade II or III.

Among children, the most common types of brain tumours include the following;

Medulloblastoma: The tumour usually arises in the cerebellum. It's sometimes called a primitive neuroectodermal tumour. It is grade IV.

Grade I or II astrocytoma: In children, this lowgrade tumour occurs anywhere in the brain. The most common astrocytoma among children is juvenile pilocytic astrocytoma. It's grade I.

Ependymoma: The tumor arises from cells that line the ventricles or the central canal of the spinal cord. It's most commonly found in children and young adults. It can be grade I, II, or III.

Brain stem glioma: The tumour occurs in the lowest part of the brain. It can be a low-grade or high-grade tumour. The most common type is diffuse intrinsic pontine glioma.

Symptoms **Of Brain Tumors**

i) The most common symptoms of brain tumours include:
ii) Headaches (usually worse in the morning)
iii) Nausea and vomiting
iv) Changes in speech, vision, or hearing
v) Problems balancing or walking
vi) Changes in mood, personality, or ability to concentrate
vii) Problems with memory
viii) Muscle jerking or twitching (seizures or convulsions)
ix) Numbness or tingling in the arms or legs
x) Most often, these symptoms are not due to a brain tumor. Another health problem could cause them.

Diagnosis

One or more of the following tests can be done to diagnose a brain tumour:

Neurologic exam: involves checking of vision, hearing, alertness, muscle strength, coordination, and reflexes. The physician also checks for eye swelling caused by a tumor pressing on the nerve that connects the eye and the brain.

MRI: A large machine with a strong magnet linked to a computer is used to make detailed pictures of areas inside the head. Sometimes a special dye (contrast material) is injected into a blood vessel in the arm or hand to help show differences in the tissues of the brain. The pictures can show abnormal areas, such as a tumor.

CT scan: An x-ray machine linked to a computer takes a series of detailed pictures of the head.

Angiogram: Dye injected into the bloodstream makes blood vessels in the brain show up on an x-ray. If a tumour is present, the x-ray may show the tumour or blood vessels that are feeding into the tumour.

Spinal tap: the physician may remove a sample of cerebrospinal fluid (the fluid that fills the spaces in and around the brain and spinal cord). This procedure is performed with local anaesthesia. The physician uses a long, thin needle to remove fluid from the lower part of the spinal column.

Biopsy: The removal of tissue to look for tumour cells is called a biopsy. A pathologist looks at the cells under a microscope to check for abnormal cells. A biopsy can show cancer, tissue changes that may lead to cancer, and other conditions. A biopsy is the only sure way to diagnose a brain tumour.

Brain **Tumour Treatment**

People with brain tumours have several treatment options. The options may involve;

Surgery, complete or partial resection of the tumour with the objective of removing as many tumour cells as possible

Radiation therapy, the most commonly used treatment for brain tumours; the tumour is irradiated with beta, x rays or gamma rays.

Chemotherapy is a treatment option for cancer, however it is seldom used to treat brain tumours as the blood and brain barrier prevents the drugs from reaching the cancerous cells. Chemotherapy can be thought of as a poison that prevents the growth and division of all cells in the body including cancerous cells.

Many clients get a combination of treatments.The choice of treatment depends mainly on the following:

The type and grade of a brain tumour

Its location in the brain

Its size

The patient's age and general health

Rehabilitation

Rehabilitation of a tumour patient after treatment may involve several types of therapists who can help. These may be;

- ***Physical therapists***: Brain tumours and their treatment may cause paralysis. They may also cause weakness and problems with balance. Physical therapists help these patients regain strength and balance.
- ***Speech therapists***: Speech therapists help patients who have trouble speaking, expressing thoughts, or swallowing.

- ***Occupational therapists***: Occupational therapists help patients learn to manage activities of daily living, such as eating, using the toilet, bathing, and dressing.
- ***Physical medicine specialists***: Medical personnel with special training help patients with brain tumours stay as active as possible. They can help them recover lost abilities and return to daily activities.

Follow – upcare after brain tumour treatment

The patient needs regular check-ups after treatment for a brain tumour. For example, for certain types of brain tumours, check-ups may be every 3 months. Check-ups help ensure that any changes in patient's health are noted and treated if needed. You have finished learnig about brain tumors, Nowyouwill be looking at conditions which affect the coverings of the brain.**MENINGITIS (FUNGAL, BACTERIAL, VIRAL)**

Definition: meningitis is the inflammation of the meninges, the protective membranes that surround the brain and spinal cord (Smelzer et al, 2010).

OR

It is the inflammation of the meninges covering the brain and spinal cord caused by bacterial,viral or fungi characterized by fever, neck stiffness, fit and altered level of consciousness

Classification Of Meningitis According To Causative Agents

a. Bacterial meningitis (Sepitc meningitis)
    - Haemophilus influenza
    - Neisseria Meningitidis
    - Streptococcus Pneumoniae
    - Staphylococcus aureus
    - Escherichia Coli
    - Mycobacterium tubercle
    - Klebsiella
    - Proteus
    - Pseudomonas
    - Listeria monocytogen

Mode Of Transmission

Meningitis generally is transmitted in one of the following four ways:

- Airborne droplets or contact with oral secretions from infected individuals.
- From direct contamination (from a penetrating skull wound or skull fracture).
- Via the blood stream (Pneumonia, endocarditic, rotten tooth Otitis media)Bacteria Meningitis is transmitted through: d
- Direct extension from the ears, nasopharynx, sinuses, a cranial injury or congenital meningeal defect and spread via the blood stream. Chronic suppurative otitis media is a common problem in children and an important source of bacterial meningitis.

Pathophysiology

Most cases of bacteria meningitis are caused by an infectious agent that has colonized or established a localized infection elsewhere in the host body.Potential sites of colonization or infection include the skin,the nasal-pharyx,the respiratory tract ,and the Genitourinary tract.A bacteria agent can gain access to the central nervous system and cause meningeal disease through the invasion of bacteria in the bloodstream and subsequent haematogenous seeding of the CNS

Direct contiguous spread (e.g sinusitis,otits media,trauma,or during inoculation during intracranial manipulation.The brain is naturally protected from the body's immune system by the barrier that the meninges create between the bloodstream (BBB) and the brain.However, in bacteria meningitis, the BBB can be disruptedand blood vessels become leaky and allow fluid, white blood cells,and other infection-fighting particles enter the meninges and brain causing them to swelland infection spread quickly through the CSF that circulates around the brain and spinal cord.The inflammatory process may remain confined to the subarachnoid space. In less severe forms, the pia matter is not penetrated, and the underlying parenchyma remains intact.However,in more severe forms of bacterial meningitis ,the pia matter is breached ,and the underlying parenchyma is invaded by the inflammatory process.This leads to obstruction of CSF flow and decreased reabsorption causing increased intracranial pressure severe headache and fever.

Clinical Features

- In meningococcal meningitis, a pink macular rash, petechiae will appear.

- Kening's signs – with the hip joint flexed, extension at the knee causes spasm in the hamstring muscles.
- Brudzink's sign – passive flexion of the neck causes flexion of the thighs and knees.
- Confusion due to increased ICP
- Restlessness due to headache and neck stiffness
- Convulsions due to interruption of normal cerebral functions.
- Fever due to systemic infection.
- Headache due to increased intracranial pressure as a result of infection of CSF.
- Neck stiffness due to meningeal irritation.
- Photophobia due to damage and irritation to the optic nerve.
- Vomiting due to autonomic disturbances
- Cerebral Hypoxia which result from reduced blood flow to the brain

Management

Investigations

1. History taking
2. Physical assessment: Positive Brudzinskis signs, Positive kerning's signs and neck rigidity.

Diagnostic tests

- Lumbar puncture: CSF analysis and gram stain/culture
- Culture and sensitivity testing of blood, sputum, urine and other body secretions.
- Blood slide to rule out cerebral malaria

Treatment

a. **Respiratory precautions**: Patient with N. Meningitidis, H. Influenzae or in whom the causative organism is in doubt requires observation with special respiratory Isolation for 24hours after initiation of the appropriate antibiotic therapy.

b. **Parenteral antibiotics in bacterial meningitis**: The antibiotic must penetrate the blood-brain barrier into the CSF. Antibiotics include the following:

-
- Chloramphenicol 500-1000mg qid for 7/7 plus
- Benzyl penicillin 4-8 mega units qid for 7/7 OR

- Cephotaxime injection 1g by injection every 12 hours increased in severe infections to 8g daily in 4 divided doses (meningitis) higher doses may be required up to 12g daily in 3 – 4 divided doses.
- Antipyretic will be given e.g. Paracetamol 1g tds for 3/7days to reduce fever

c. **Viral meningitis** is self-limiting however, there arises a need to put the patient on antibiotic treatment to prevent bacterial invasion which would otherwise thrive in viral infection.

d. **Fungal meningitis**- the patient will be prescribed on Amphotericin-B or Ketaconazole.

e. Supportive treatment includes:
    - Glucocorticosteroids: Dexamethasone- High dose therapy to stabilize the cell membrane and reduce inflammation and cerebral oedema.
    - Osmotic diuretic: Mannitol to reduce cerebral oedema.
    - Diazepam to control seizures.
    - Analgesics e.g. paracetamol for headache and as well as to control fever.
    - Limitation of fluid to about 1500ml to keep patient under hydrated and reduce cerebral oedema and effects of inappropriate ant diuretic hormone secretion.

Nursing Care

Nursing Diagnosis

- Pain related to headache/neck stiffness secondary to meningitis.
- Fluid volume: excess related to SIADH.
- Risk for impaired tissue integrity related to unconsciousness/immobility.
- High risk for fluid volume deficit related to less oral intake or fever.
- Altered nutrition; less than body requirement related unconsciousness.
- Self-care deficit related patient's unconsciousness

Activity

Identify the appropriate nursing model using the identified problems above write the nursing care for a patient suffering from meningitis

Aims

1. To prevent spread of infection
2. To prevent pressure sore formation/reduce possibility of occurrence of complications.

3. To maintain good nutrition status.

Environment

Provide a quiet environment and a darkened room. Restrict visitors as necessary to reduce noise. Sun shields may be used to promote comfort from photophobia. If patient is unconscious, promote bed rest and assist patient with Activities of Daily Living as needed to reduce movements that may cause pain.

Respiratory isolation is required for meningococcal infections only until the pathogen can no longer be cultured from naso-pharynx. The patient may need to be in a railed bed to prevent falls during seizures. Elevate the head of bed to promote venous drainage, this helps to reduce cerebral congestion and oedema.

Comfort Measures

Apply ice bag to the head or cool cloth to the eyes to help diminish the headache. Support patient in a position of comfort. The HOB should be elevated at 30 degrees to promote venous return.

Keep the neck in alignment during position changes. Provide gentle passive ROM and massage to the neck and shoulder joints and muscles to help relieve stiffness. If patient is afebrile, apply moist heat to the neck and back to promote muscle relaxation and reduce pain. Keep communication simple and direct; in a soft and calm tone of voice. Loosen constricting bed clothing and avoid restraining the patient.

Observations

Observe measure and record the input and output. The patient usually will have an indwelling catheter especially if he/she is unconscious. Weigh the patient on alternate days to monitor the nutritional status. The urine specific gravity and electrolyte serum studies should be recorded and reported. Vital signs should be observed every 4 to 6 hours and gradually reduced as the patient's condition improves.

Observe the level of consciousness and the mental status. Observe and record the status of the skin for any pressure sores. Monitor patient for symptoms of increased intracranial pressure and institute measures for precaution maintain fluid restriction as prescribed. Administer hypertonic saline (3%) as prescribed. Hypotonic intravenous solutions such as 5% dextrose in water are avoided because they increase cerebral oedema.

Nutritional Support And Fluids

Parenteral or enteral feeding or modified diet, depending on patient's LOC and ability to swallow.

Prevent constipation, by giving stool softeners and laxatives to avoid IICP. Intravenous fluids are given to maintain a balanced electrolyte status. Generally, fluids are limited to 1,500mls to a state of under hydration.

Hygienic Measure

Help with ADL such as bed baths, oral toilet, pressure area care etc. Turning of patient should be 2 hourly. Perform catheter care as necessary. Ensure that the patient is lying on dry linen to avoid skin peeling off.

Psychological Care

Explain the disease process to the patient and significant others to alley anxiety. Explain specific respiratory precautions to prevent spread of infection to others. If they must leave the room for a procedure or test, explain that a mask must be worn to protect others from contact with airborne droplets. Reassure patient the special respiratory precaution are temporary and will be discontinued once patient has been on the appropriate antibiotic for 24 to 48 hours.

Health Education

Give the patient and relatives information in writing and verbal such as:

- Transmission & preventive measures
- Avoid overcrowding areas especially in meningococcal meningitis.
- Completion of medication so as to avoid resistance
- Early treatment of any infection including respiratory tract infection
- Meningococcal vaccine should be emphasized in epidemic period.
- Heamophilus Influenza vaccine should be emphasized.

Complications

- DIC (Disseminated Intravascular Coagulation)
- Hydrocephalusdue to adhesions formed after inflammation causing CSF brockage
- Syndrome of inappropriate secretion of ant diuretic hormone
- Impaired of hearing due to compression of vestibulocochlear nerve by the inflamed meninges.
-
- Brain abscess due to presence of the bacteria

- Mental retardationdue to severe inflammation of the brain tissue
- Encephalitis due to invasion of bacteria in the brain tissue
- Visual impairment: due to compression of nerves by the inflamed meninges.
- Brain damage: due to dissemination of the infection to the brain. From the meninges.
- Optic neuritis: due to the infection to the optic nerve.
- Paralysis: due to nerve damage
- Gangrene due to toxins(poisons) produced by bacterias when they enter the blood that kill healthy tissues.
- Cerebral Oedema;due to some exudate that can seep away from the blood vessels

VIRAL MENINGITIS (ASEPTIC MENINGITIS).

Definition

Viral meningitis is the inflammation of the outer covering of the brain and spinal cord meninges caused by different types of viruses e.g. of measles,small pox,herpes simplex virus and coxsac virus evidenced by fever,headache,neck stifnes,brared vision,convulsions and confusion.

It is the most common type of meningitis as compared to bacterial and fungal meningitis but it is not fatal or deadly.It can resolve on its own without treatment.

It usually attacks young and old aged people. Malnourished children,HIV positive and patients on cytoxic drugs are predisposed to this type of meningitis because of their compromised immunity.

Monde of transmission

Infection can enter through a direct entry such as penetrating head injury, fracture of the base of the skull and complication of the upper respiratory tract infection.

Causes/Predisposing Factors

It is commonly caused byEcho virus,coxsac, enterovirusvirus [intestinal],herpes zoster virus,polio virus,rabies virus,measles viruse.

People whose immunity is compromised due to HIV infection and people on cytotoxic drugs and malnourished children are prone to viral meningitis.

Pathophysiology

The organism will gain entry to the central nervous system through the upper respiratory tract, blood stream[wound, fracture of the skull and brain abscess. The virus irritates the meninges through infecting the central spinal fluids leading to inflammatory response within the meninges. The inflammatory response tend to increase the cerebral spinal fluids leading to inflammatory response within the meninges.The inflammatory response tend to increase the cerebral spinal fluids production, with an increase in intracranial pressure, celebral oedema swelling around the dura matterng to someone manifest all those signs and symptoms, such as, confusion, headache etc.

Signs And Symptoms

i) Fever related to infection
ii) Vomiting related to irritation by the virus to the vomitus centre
iii) Photophobia related to compressed optic nerves as a result of intracranial pressure.
iv) Headache related to increased intracranial pressure and irritation of the brain meninges
v) Seizures related to disturbed transmissions of motor impulses due to toxins produced by the virus
vi) Neck stiffness related to inflamed meninges
vii) Neck stiffness related to the inflamed meninges
viii) Dizziness related to increased intracranial pressure.

Investigations

Same as for bacterial meningitis

Treatment

Treatment in viral meningitis is symptomatically, it is treated according to the symptoms manifested].

The chances of survival and recovery largely depend on the type of the virus causing the meningitis.

Acyclovir for herpes virus can be given 200mg tds for 5 days.

For headache give analgesia e.g. paracetamol 1g tds for 3 days

Vomiting, give antiemetic's such as promethazine 25mg od for 3 days

For cerebral oedema give manitol an osmotic diuretic drug, 200mg/kg body weights

To suppress the inflammation, give Dexamethasone 0.5mg/kg body weight for 4 days.

Nursing Care

AIMS

To prevent infection spread

To promote quick recovery

Alley anxiety

Maintain optimal nutritional status

Promote rest

Nursing Care

Enviroment

Nurse the patient in a quiet room to in order to promote rest.Ensure the room has dim light in order to prevent photophobia.Nurse the patient in an acute bay for easy and close observation.Nurse the patient in a clean environment to prevent upper respiratory infections. Ensure that the room has all the emergence equipment for resuscitation for any eventuality.

Observations

Check vital sign and BP to act as the base line data in order to know if the condition is improving or deteriorating. Observe the level of consciousness using the Glasgow coma scale to monitor patient's response to therapy. Pay particular attention to pressure area to detect on set of pressure sore development. Observe the patient's facial expressions to detect pain and administer prescribed analgesics like paracetamol

Ppsychological Care

Reassure the patient that the medical team is doing everything possible to manage the condition and thus allaying anxiety.Explain to the patient as well as to the relatives that the causative

organism is self-limiting and treatment will only be given symptomatically, also to prevent complications.Explain to the patient and relatives every procedure done to the patient in order to gain cooperation. Encourage the patient to ask question and answer accordingly Explain all procedures you conduct on the patient togain cooperatin and allay anxiety. Explain to the relatives that the confusion and delirium are temporal, they will desapear.

Position

If the patient is in an unconscious state, nurse him/her in a lateral position to promote drainage of secretions from the mouth. Nurse the patient in a railed bed to prevent accidental falls in cases of confusion or seizures.

Nutriton/Intravenous Fluids

Offersmall frequentnutrious meals to enhance the healing process. If patient cannot take food and fluids orally, insert a nasal gastric tube for feeding to maintain the nutritional status. As the condition improves, encourage the patient to take small frequent meals which should be supplemented with parenteral fluids in order to promote healing.Offer prescribed laxative mild or stool softeners to prevent constipation and minimize the risk of increased intra cranial pressure resulting from straining during defecating.

Hygiene

If patient is unconscious do a bed bath to remove dead epithelial cells and promote comfort. Change soiled linen whenever necessary to promote comfort .Encourage oral care to prevent halitosis.

Rest And Exercise

Nurse the patient in aquiet environment to promote rest.Ensure that you restrict visitors to avoid disturbing the patient.Carry out procedures in blocks to allow the patient have enough rest. Carry our passive exercise to prevent muscle wasting and promote blood circulation.

Ellimination

If the patient is unconscious catheterize him/her to prevent him/her from soiling the beddings.Ensure that catheter care is done to prevent urinary tract infections. Offer a lot of fluids and roughage to prevent constipation.

Complication

The following are complications of meningitis:-

i) Visual impairment related to compressed optic nerve.
ii) Hydrocephalus in children related to oedema and increased intra cranial pressure.
iii) Brain abscess related to infection.
iv) Encephalitis related to the body response to infection.
v) Convulsions related to disturbed transmision of neurons [electric impulses]
vi) Cerebral palsy related to disturbed brain motor functions.
vii) Delayed milestone related to motor disturbance in the brain

FUNGAL MENINGITIS

Definition

Cryptococcal meningitis is a fungal infection which affects the membranes covering the brain and the spinal cord (meninges) caused by cryptococcus Neoformans, characterised by the blurred vision, cerebral oedema and fever.

OR

It is the inflammation of the meninges caused by cryptococcus neoformans characterised by pyrexia, blurred vision, irritation of the brain and spinal cord.

Predisposing Fators

Cryptococcosis is an opportunistic infection for HIV/Aids.

Other conditions that pose an increased risk in contraction the infection include:-

Certain lymphomas, e.g. Hodgkin. Lymphoma

Sarcoidosis, liver cirrhosis

Patients on long – term corticosteroids therapy.

People who come in contact with pigeons dropping(or contaminated with those droppings.

Eucalyptus trees, blue gum tree.

Pathophysiology

Cryptococcus Neoformas usually gain entry to the (CNS) central Nervous system through the upper respiratory tract or blood stream (wounds, fracture of the skull, brain abscess, otitis media).

Infection of cerebral spinal fluid (CSF) leads to an inflammatory response within the meninges.

Inflammatory response tends to increase CSF production, with an increase in intracranial pressure.

In fungal/cryptococcal meningitis the purulent secretions quickly spread to others areas of the brain through cerebral spinal fluid (CSF).

Due to An inflammatory process, there will be cerebral oedema (swelling around Dura) and increased intracranial pressure.

Signs And Symptoms

Same as for bacterial meningitis

Investigations Cont

i) History and physical examination: will reveal sign and symptoms suggestive of the disease.
ii) Lumber puncture: shows typical cerebral spinal fluid (CSF) will reveal elevated cerebral spinal fluid (CSF) pressure, cloudy or milky white (CSF) high protein level gram stain and culture that usually identifies and infecting organism, unless it's a virus and depressed glucose concentration.
iii) White blood cell count: indicates leucocytosis and serum electrolyte levels often are abnormal.
iv) Computed Tomography scan: can rule out cerebral haematoma, haemorrhage or tumour.
v) Cerebrospinal fluid (CSF) CRAg important tool to diagnose meningeal disease but can be negative in Non meningeal cryptococcosis.
vi) Serum cryptococcal Antigen (Gag) positive in>99% of AIDS patient with cryptococcal meningitis less often with isolated pulmonary disease.

vii) Cryptoccoco Antigen Test(CAT) this is a test which is done to detect cryptoccoco antigen (capsular material) by culture of cerebral spinal fluid , sputum and urine in most cases CNS or disseminated diseases.

viii) Blood slide: to rule out malaria.

Treatment

First line

The drug of choice for treatment of fungal meningitis isAmphotericin B

Dose: Amphotericin B 0.7 – 1.0 milligram per kg body weight per day intravenously plus flucytosine (5-fc) 100mg/kg body weight per day orally divided over 4 doses for 2weeks the maintenance dose fluconazole 400mg/day for 8weeks.

Second line:

Amphotericin B 0.7-1mg/kg/day intravenous plus flu cytosine 100mg/kg body weight/day per oral divided over 4 doses for 6-10weeks; amphotericin B 0.7-mg/kg body weight/day/per oral for 6-10 weeks ; fluconazole 400-800mg in day per oral for 10-12 weeks ; fluconazole 400-800mg/day per oral with 5-fc 100-150mg/kg body weight/day per oral divided over 4 doses for 6 weeks; lipid formulation of amphotericin B 4-5mg/kg body weight/day intravenously for 6-10 weeks. Haconazole 400mg/day per oral (liquid formulation preferred) for10-12weeks can be given but is less effective than fluconazole.

Prophylaxis: fluconazole 200mg/kg body weight until CD4 count goes up to 200 then stop.

Nursing Care

OBJECTIVE

To relieve anxiety

To maintain hydration status of patient.

To reduce patient body temperature within normal ranges

To relieve pain

To prevent complications.

Environment

Nurse the patient in a quiet and dim room to promote rest and decrease photophobia.

The room should be well ventilated and warm so as to promote comfort.

Nurse the patient in dim light to prevent photophobia.

Position

Nurse a patient in coma lateral position to allow postural drainage of secretions from the month

If the patient is conscious , nurse him\her in a position he/she is comfortable.2hourly turning must be done to prevent presure sore formation.

Observations

Access the patients' clinical status, neurological function and vital signs of increased intracranial pressure, for instance vomiting, seizures, a change in motor function and vital signs.

Also watch for signs of cranial nerve involvement such as (ptosis, strabismus, diplopia double visitation.Watch for signs of confusion. Observe the intake and output of fluids to assess the level of hydration.Observe the patient by using glasgow coma scale which is a tool for assessing a patients response to stimuli, also to measure the consciousness of a patient.

Rehydration

Maintain adequate fluid intake to avoid dehydration but avoid fluid overload because of the damage of cerebral oedema.

Record intake and output on a fluid balance chart.

Medication

- Administer prescribed drugs and note their effects.Watch the adverse reactions of the drugs.
- Watch for side effects e.g. kidney damage, chills stiffness.

Rehydration

Maintain adequate fluid intake to avoid dehydration but avoid fluid overload because of the damage of cerebral oedema.Record intake and output on a fluid balance chart.***Medication***

Administer prescribed drugs and note their effects.

Watch the adverse reactions of the drugs.

Watch for side effects e.g kidney damage, chills stiffness.

Psychological Care

Provide reassurance and support. Explain all procedures done to the patient to gain the patients cooperation. Reassure the family that the delirium and behaviour changes caused by meningitis

usually disappear. Explain the cause of the disease top make the patient understand alley anxiety.
Encourage patient to verbalize and express then fears.
Include patient and family when making decisions as regards patient care.
Complications
Same as for other types of meningitis

BRAIN ABSCESSES

Brian abscess is an accumulation of pus within the brain tissue that can result from a local or a systemic direct extension from an ear, tooth, mastoid or sinus infection.

Predisposing Factors/ Causes

A weakened immune system (such as HIV and aids)
Chronic disease such as cancer
Drug that suppress the immune system such as (corticosteroids or chemotherapy)
Right to left heart shunts, usually they result of congenital heart disease
Pulmonary infections
Bacterial endocarditis
Skull fracture due to trauma
Surgery

Pathophysiology

Common predisposing conditions for abscesses in healthy adults otitis media and rhinosinusitis, dental infections, and systemic infections. An abscess can result fromintracranial surgery and penetrating head injury. Pathogens causing brain abscess reach thebrain by hematologic. Brain abscesses in immunocompromisedpeople may result from various pathogens present in the body.

Signs And Symptoms

A brain abscess can cause many different symptoms depending on the location.
Headache due to increased intracranial pressure
Nausea
Vomiting
Sleepiness

Seizures

Fever due to infection

Increased intracranial pressure

Investigations

Computed tomography which will show the location of the abscess

Magnetic resonance imaging will show the size and location of the abscess

Biopsy of the abscess done for microscopic and culture

Skull x-ray to show the abscess

Full blood count will show elevated white blood cells and ensophills

Testing for the presence of antibodies to organism such as toxoplasma gondi and taenia solium

Treatment

AIM

Treatment is aimed at controlling increased ICP,

Antimicrobial therapy directed at combating infection

The choice ofantibiotic medication is based on culture andsensitivity testing of the causative organism.

Administer prescribed drugs such as corticosteroids andmannitolto help reduce the inflammatory cerebraledema

If the patient shows evidence of an increasing neurologicdeficit, antiseizure medications (phenytoin, phenobarbital)may be prescribed to prevent or treat seizures.

Nursing Management

Nursing care is aimed at:

Continuing to assess the neurologicstatus of the patient.

Administering medications and assessing the response to treatment,

Providing supportive care.

TAKE NOTE: If the patient is unconscious,nursing management will be as for an unconscious patientapplies including the following:

Environment:

Nurse the patient in a quiet, darkened room until photophobia and cerebral irritation subside. The patient should also be nursed in a padded, railed bed to prevent patient from falling during seizures. visitors should be restricted to promote rest.

Position:

The patient is nursed in a lateral position with the head elevated to prevent and reduce cerebral oedema. Turn the patient every 2 hours to prevent pressure sore formation and hypostatic pneumonia. When consciousness is gained and patient's condition is stable, encourage patient to assume a position of comfort.

Observations:

Monitor vital signs i.e. temperature, pulse, respirations and blood pressure are checked 2 hourly.

Assess levels of consciousness using the Glasgow coma scale is used to record patient's observations. Neurologic deficits such as impaired hearing, vision, sensory and motor weakness are also observed 2 hourly. Monitor the patient's fluid intake and output and balanced every 24hours. Observation for side effects of the high dose antibiotics must be done.

Nutrition

Feed the patient through a nasogastric tube to maintain nutrition status until he is able to take orally. Fluid intake should be sufficient to prevent dehydration or avoid fluid overload which could lead to increasing cerebral oedema. A balanced diet is essential to quicken recovery. Give small, frequent meals to prevent nausea. Added fibre in the diet will assist in the prevention of constipation which should be avoided because of the danger of a rise in intracranial pressure caused by straining at stool.

Hygiene:

Bath the patient daily to stimulate blood circulation and promote comfort.

Do oral care is frequently to prevent oral infection, bad smell and dental carries. Change soiled linen to promote comfort and prevent the skin from breaking. This will prevent pressure sore formation. Eye care should be done and eyes prevented from drying especially when patient is unconscious.

Rest and activity:

Nurse the patient in a quiet, darkened room to promote rest. Conduct your procedures collectively to ensure long periods of rest for the patient. Position the patient well to ensure rest. Carry out passive limb exercises to prevent muscle wasting and joint stiffness.It also helps to promote blood circulation.

Elimination:

If patient is unconscious, catheterize him. This will facilitate monitoring of urine output and will also prevent soiling of the beddings. If conscious, provide bedpan/urinal as requested.

Thank you for going through meningitis.You now know the different types of meningitis and how they are treated. Let us now look at malaria which affects the brain

CEREBRAL MALARIA

Definition

It is a complication of a type of malaria caused by a parasite called plasmodium falciparum characterized by impaired consciousness, delirium, abnormal neurological signs, convulsions, hyperthermia and headache

Presence of unarousable coma, after exclusion of other encephalopathies (hypoglycemia, bacterial meningitis and viral encephalitis) and confirmation of P. falciparum infection.

Pathophysiology

The basic underlying defect seems to be clogging of the cerebral microcirculation by the parasitized red cells. The parasitized cell develop knobs on their surface and develop increased cytoadherent properties, as a result of which they tend to adhere to the endothelium of capillaries and venules. This results in sequestration of the parasites in these deeper blood vessels.Obstruction to the cerebral microcirculation results in hypoxia and increased lactate production due to anaerobic glycolysis. The adherent erythrocytes may also interfere with gas and substrate exchange throughout the brain. Increased cerebral anaerobic glycolysis interferes with neurotransmission causing impairment of cerebral function.

Signs and symptoms

Coma that lasts for 24-72 hours, initially rousable and then unrousable. (a Glasgow coma scale of <7/15.)

Impaired consciousness

Fever due to parasiteamia

Generalized convulsions and neurological abnormalities

Severe headache followed by drowsiness

Delirium

Mild neck stiffness may be seen, however, neck rigidity and photophobia and signs of raised intracranial tension are absent

Coma that persists for 24-72 hours, initially rousable and then unrousable

Management

Diagnosis

Thick and thin blood smears / RDT for malaria parasites will reveal P. falciparum

Blood glucose to rule out other conditions like Diabetes mellitus

Blood slide for malarial parasites will be positive

Lumbar puncture to rule out meningitis

Random blood sugar test to rule out hypoglycaemia

MRI to rule out encephalitis

Blood for culture to rule out septicaemia

Clinical picture may show impaired cerebral functions

Treatment

Administer prescribed quinine 20mg/kg body weight intravenously in 1 litre of 5% dextrose loading dose (infused over 4 hours), then patient rests for another four hours.

Followed by a maintenance dose 10mg/kg body weight in 500mls of 5% dextrose 8 hour for 5/7

Analgesic and antipyretics like paracetamo lg tds for three days

Oxygen 2-5litre/minute may be given.

As condition improve patient may be given the following:

Folic acid 5mg od for 14/7

Anti-emetics e.g. Plasil

Multivit 2tablets bd for 7/7

Nursing management

Aims

Eliminate P. Falciparum.

Combat hyperpyrexia.

Control convulsions.

Correct dehydration.

Environment

Nurse the patient in a quiet and dim room to promote rest and decrease photophobia. The room should be well ventilated and warm so as to promote comfort. Nurse patient in a padded railed bed to prevent injury and falling

Position

Nurse a patient in coma lateral position to allow postural drainage of secretions from the month

If the patient is conscious , nurse him\her in a position he/she is comfortable. 2hourly turning must be done to prevent presure sore formation.

Observations

Monitor the patients' clinical status, neurological function and vital signs of increased intracranial pressure, for instance vomiting, seizures, a change in motor function and vital signs.

Also watch for signs of cranial nerve involvement such as (ptosis, strabismus, diplopia double visitation. Watch for signs of confusion. Observe the intake and output of fluids to assess the level of hydration. Observe the patient's levels of consciousness by using glasgow coma scale which is a tool for assessing a patients response to stimuli to determine response to treatment.

If temperature is high, Tepid sponge the patient, put on the fan and giving of fluids. Give paracetamol or aspirin if temperature >38.5 ^{o}c.

Nutrition

Assess the hydration status. Strict 24 hour intake and output chart should be maintained to monitor patients feeds. Provide Liquid balanced diet to maintain the patient's nutritional status

If patient is unconscious, insert NGT and feed the patient 3 hourly. In case of dehydration or hypovolaemic shock give 20 – 30ml/kg body weight of normal saline and reassess patient within 30 minutes to decide on next fluid requirement according to degree of dehydration.

Rehydration

Maintain and adequate fluid intake to avoid dehydration. Avoid fluid overload to prevent cerebral oedema. Record intake and output on a fluid balance chart.

Medication

Administer prescribed drugs and note their effects.

Watch the adverse reactions of the drugs.

Watch for side effects e.g. kidney damage, chills stiffness.

Psychological care

Explain the condition of the patient to the relatives to provide reassurance and support. Explain all procedures done to the patient to gain the patients cooperation. Reassure the family that the delirium and behaviour changes caused by meningitis usually disappear. Explain the cause of the disease to the patirnt and relatives to alley anxiety. As the condition improves,encourage patient to verbalize and and respond accordingly to gain their cooperation. Include patient and family when making decisions as regards patient care.

Hygiene

Catheter toilet should be done as indicated to prevent ascending of infections. Change linen frequently to promote comfort. Bath patient daily in bed to promote comfort and self-esteem

Care for the mouth to prevent halitosis and promote appetite. Perform 2 hourly changing of position to prevent pressure sore formation.

Exercise

Perform passive exercises to promote blood circulation. Use splints or foam boots to prevent foot drop. Nurse patient on special beds to prevent pressure on bony prominences

Elimination

Insert an indwelling catheter and strictly measure urine output to assess renal function. This will also prevent soiling patients linen. Offer enough fluids to prevent constipation

Possible Complications

Renal failure.

This will result from renal injury in acute tubular necrosis. There is reduced oxygen consumption of the kidneys. Acute tubular necrosis presumably results from renal microvascular obstruction and cellular injury consequent upon sequestration in the kidneys and filtration of free haemoglobin, myoglobin and other cellular materials.

Anaemia –

There is marked destruction of red blood cells containing parasites at merozoites stage. There is accelerated destruction of non-parasitized (blood cells without parasite) red blood cells that parallels disease severity and also bone marrow dyserythropoeisis (non formation of red blood cells). In severe malaria, anaemia develops rapidly. The rapid haemolysis of unparasitized red blood cells is the major contributor to the decline in haematocrit. Most patients with malaria have iron deficiency anaemia. At times routine iron supplementation following malaria promotes recovery from anaemia.

Pulmonary oedema

Pulmonary oedema in malaria results from a sudden increase in pulmonary capillary permeability. Hypoglycaemia- Hypoglycaemia occurs in approximately 8% of adults and up to 30% of children with cerebral malaria. This is due to increased glucose use and impaired glucose production caused by the inhibition of gluconeogenesis.

Metabolic acidosis

Acidosis may be associated with renal failure in adults, but in the acute infection there is also a lactic acidosis. The outlook for persistent acidosis is poor. Although blood pressure and tissue perfusion is usually adequate initially, hypotension commonly ensues.

Gastro – Intestinal/ Liver complications

This may result from both gut sequestration and visceral vasoconstriction. Gut permeability is increased and this may be associated with reduced defences against bacterial toxins or even whole bacteria in severe disease.

Black water fever (haemoglobinaemia).

This happens when there is massive intravascular haemolysis and the passage of 'Coca-Cola' coloured urine. Black water is usually transient and resolves without complications, but in severe cases renal failure may develop.

Same as for other types of meningitis

Activity:2.6.4 Write down two the differencesbetween malaria and meningitis

You are through with cerebral malaria,you will nowlearn about multiple sclerosis

MULTIPLE SCLEROSIS (MS)

Definition

Multiple sclerosis (MS) is an immune-mediated progressive demyelinating disease of the CNS (Smeltzer et al., 2010). Demyelination refers to the destruction of myelin, the fatty and protein material that surrounds certain nerve fibres in the brain and spinal cord; it results in impaired transmission of nerve impulses.

Causes

Autoimmune activity results in demyelination, but the sensitized antigen has not been identified.

A viral infection which affects the white matter of the brain and spinal cord, producing demyelinated lesions that prevent normal conducting of nerve impulses.

Physical injury, excessive fatigue and poor state of health

Risk factors

1. Multiple factors play a role in the initiation of the immune process. Geographic prevalence is highest in northern Europe, southern Australia, the northern United States, and southern Canada.
2. It is believed that an environmental exposure at a young age may play a role in the development of MS later in life.
3. Genetic predisposition is indicated by the presence of a specific cluster (haplotype) of human leukocyte antigens (HLA) on the cell wall. The presence of this haplotype may promote susceptibility to factors, such as viruses, that trigger the autoimmune response activated in MS.

Pathophysiology

Sensitized T cells typically cross the blood–brain barrier; their function is to check the CNS for antigens and then leave. In MS, the sensitized T cells remain in the CNS and promote the infiltration of other agents that damage the immune system. The immune system attack leads to inflammation that destroys myelin (which normally insulates the axon and speeds the conduction of impulses along the axon) and oligodendroglial cells that produce
myelin in the CNS.

Plaques of sclerotic tissue appear on demyelinated axons, further interrupting the transmission of impulses. Demyelination interrupts the flow of nerve impulses and results in a variety of manifestations, depending on which nerves are affected. Demyelinated axons are scattered irregularly throughout the CNS. The areas most frequently affected are the optic nerves, chiasm, and tracts; the cerebrum; the brain stem and cerebellum; and the spinal cord. Eventually the axons themselves begin to degenerate, resulting in permanent and irreversible damage (Bashir & Whitaker, 2002; Halper, 2001 in Smeltzer , 2010).

Clinical Manifestations

The course of MS may assume many different patterns. In some patients, the disease follows a benign course, with a normal life span and symptoms so mild that patients do not seek health care and treatment.

Primary progressive MS is characterized by continuous decline, with the potential development of quadriparesis, cognitive dysfunction, visual loss, and brain stem syndromes.

The signs and symptoms of MS are varied and multiple, reflecting the location of the lesion (plaque) or combination of lesions.

The primary symptoms commonly reported are

- Fatigue. Fatigue impairs optimal function throughout the course of the disease. Fatigue is exacerbated when febrile illness, environmental temperature, hot showers, and normal circadian rhythms during the afternoon elevate body temperature.
- Depression
- Weakness
- Numbness
- Difficulty in coordination,
- Loss of balance, and pain.

- Visual disturbances due to lesions
- In the optic nerves or their connections may include blurring of vision, **diplopia**, patchy blindness (scotoma), and total blindness.
- Depression may relate to the pathophysiology or may occur as a reaction to the diagnosis.
- Suicide as the cause of death occurs 7.5 times more frequently among persons diagnosed with MS.
- **Spasticity** (muscle hypertonicity) of the extremities and loss of the abdominal reflexes are due to involvement of the main motor pathways (pyramidal tracts) of the spinal cord.
- Disruption of the sensory axons may produce sensory dysfunction (paresthesias, pain).
- Cognitive and psychosocial problems may reflect frontal or parietal lobe involvement; some degree of cognitive change (eg, memory loss, decreased concentration) occurs in about half of patients, but severe cognitive changes with dementia (progressive organic mental disorder) are rare.
- Involvement of the cerebellum or basal ganglia can produce ataxia (impaired coordination of movements) and tremor.
- Loss of the control connections between the cortex and the basal ganglia may occur and cause emotional lability and euphoria.
- Bladder, bowel, and sexual dysfunctions are common.

Management

Investigations

1. MRI is the primary diagnostic tool for visualizing plaques, documenting disease activity, and evaluating the effect of treatment.
2. Electrophoresis of CSF identifies the presence of oligoclonal banding (several bands of immunoglobulin G bonded together, indicating an immune system abnormality).
3. Neuropsychological testing may be indicated to assess cognitive impairment.
4. A sexual history helps to identify changes in sexual function.

Medical Management

No cure exists for MS. An individualized, organized, and rational treatment program is indicated to relieve the patient's symptoms and provide continuing support, particularly for individuals with cognitive changes.

Drug therapy

1. Avonex
2. Betaseron
3. Copaxone

Nursing Diagnoses

Based on the assessment data, the patient's major nursing diagnoses may include the following:

- Impaired physical mobility related to weakness, muscle paresis, spasticity.
- Risk for injury related to sensory and visual impairment.
- Impaired urinary and bowel elimination (urgency, frequency, incontinence, constipation) related to nervous system dysfunction.
- Impaired speech and swallowing related to cranial nerve involvement.
- Disturbed thought processes (loss of memory, dementia, euphoria) related to cerebral dysfunction.
- Ineffective individual coping related to uncertainty of course of MS.
- Impaired home maintenance management related to physical, psychological, and social limits imposed by MS.
- Potential for sexual dysfunction related to spinal cord involvement or psychological reactions to condition.

Complications of MS

- Urinary tract infections,
- Constipation
- Pressure ulcers
- Contracture deformities
- Dependent pedal edema
- Pneumonia
- Depression
- Decreased bone mass.
- Emotional, social, marital, economic, and vocational problemsmay also be a consequence of the disease.

Having learnt about multiple sclerosis you will now look at epilepsy

EPILEPSY

Definition: Epilepsy is paroxysmal neurologic disorder characterized by 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 diffused, a generalized seizure occurs. Symptoms vary widely, depending on the involved area of the cerebral cortex (Smeltzer et al.,,2010).

Epilepsy is a neurological disorder characterised by recurrent seizures with or without loss of consciousness due to an abnormal electrical discharge in the brain.

Take note: Seizures are episodes of abnormal motor, sensory, autonomic,or psychic activity (or a combination of these) that result from sudden excessive discharge from cerebral neurons (Smeltzer et al,.2010)

Pathophysiology

Normally brain electrical activities are non-synchronous, but in epileptic patients due to functional or structural problems in the brain a group of neurons begins firing in an abnormal, excessive and synchronized manner resulting in a seizure. The impulses occur in bursts whenever a nerve cell has a task to perform causing the ells or groups of cells to continue firing after a task is finished. During the period of unwanted discharges, parts of the body controlled by the misbehaving cells may perform irregularly. If these uncontrolled, abnormal discharges occur repeatedly, there is failure of the inhibitory synaptic contact between neurons resulting in a seizure. This causes high voltage spikes and wave activity on the EEG. a person is said to have an epileptic syndrome. This may occur in small groups and remain local causing a local seizure or the small groups may spread causing a generalized seizure.

Potential causes for lowered seizure threshold include:

a. Congenital defects
b. Head injury
c. Subarachnoid haemorrhage
d. Brain tumours/Intracranial tumour
e. Infections (meningitis or encephalitis)

f. Exposure to toxins (lead)
g. Hypoxia
h. Cerebral vascular disease.
i. Metabolic and endocrine disorders (hypoglycaemia, hypocalcaemia, uraemia, hyperthyroidism, excessive hydration and fever)
j. Drugs and alcohol withdraw
k. Allergies

For susceptible individuals, triggers may include emotional tension or stress; physical stimulation, such as loud music or bright, flashing lights, lack of sleep or food, fatigue, menses or pregnancy and excessive drug/alcohol use.

Seizure activity increases cerebral O_2 consumption by 60% and cerebral blood flow by 250%. Instances of prolonged and repeated generalized seizures; status epilepticus can be life-threatening because exhaustion, anoxia, respiratory arrest and cardiovascular collapse can occur.

Classifications Of Epilepsic Seizures

Types of epilepsies are differentiated by how the seizure activity manifests

1. **Generalized seizures**: Formerly termed as grand mal- a seizure characterized by loss of consciousness and tonic spasms of the trunk and extremities, rapidly followed by repetitive generalized clonic jerking.
2. **Partial seizures** (formerly termed as petit mal): attacks of brief impairment of consciousness often associated with flickering of eyelids and slight twitching of the mouth.
3. **Psychomotor seizures**: attacks characterized clinically by impaired consciousness and amnesia for the episode; may be accompanied by motor and psychic activity that is irrelevant for time and place.
4. **Focal seizures**: seizures beginning with a focal disturbed area of cerebral function.
5. **Jacksonian seizures**: focal motor or sensory convulsions

Clinical Symptoms

Depending on the location of the discharging neurons, seizures may range from a simple staring spell to prolonged convulsive movements with loss of consciousness.

The initial pattern of the seizures indicates the region of the brain in which the attack originates.

Simple Partial Seizures,

Begin with electrical discharge in a small area of the brain and remain confined to the area.

The person experience abnormal sensations, movement or psychic aberrations depending on the part of the brain affected For example if an electrical discharge occurs in the part of the brain that controls the right arms muscle movements the arm will begin to shake and jerk.

but without loss of consciousness.

Jacksonian seizure (motor)

Symptoms begin in one isolated part of the body such as hand or foot and then "march up" the limb as the electrical activity spreads in the brain.

Complex partial seizures

Begins within one to two minutes during which the person loses touch with surrounding and either remains motionless or moves automatically but inappropriately for time and place. The person may experience excessive emotions of fear, anger, elation, or irritability. Confusion lasts for several minutes followed by full recovery it is also referred to as temporal lobe seizure.After the manifestations, the person does not remember the episode when it is over.

Generalized seizures

Its commonly referred to as grand mal seizures, involve both hemispheres of the brain, causing both sides of the body to react. This is due to abnormal discharges of impulses over a large area of the brain which causes wide spread malfunction. There may be intense rigidity of the entire body followed by jerking alternations of muscle relaxation and contraction. Often the tongue is chewed; stools and urine may be passed involuntarily. Afterwards the person may have headache, be temporally confused and feel extremely tired. Usually the person does not remember what happened during the seizure.

Stages Of A Major Seizure

1. Usually occurs in children and rarely continues beyond adolescence. This type of seizures may cease altogether as the child matures, or it may evolve into another type of seizures. The typical clinical manifestation is a brief staring spell that lasts only a few seconds so it often occurs unnoticed. There may be an extremely brief loss of consciousness, when untreated

seizures may occur more than 50 times a day and often precipitated by hyperventilation and flushing lights.

2. *Aura Stage*: A sensory warning stage, such as a sound, odor or a flash of light. A prodromal phase of increased irritability, tension, mood changes or headache preceding the seizure by hours or days. The seizure usually does not last more than 2-6 minutes.
3. *Tonic (Rigid/Contracted):* Often lasts only 15 seconds, usually subsiding in less than a minute. There is loss of consciousness, clenched jaws (potential for tongue to be bitten), apnea (may hear cry as air is forced out of lungs) and cyanosis. The patient may be incontinent and the pupils may dilate and become non-reactive to light.
4. *Clonic (Rhythmic Contraction And Relaxation Of Extremities And Muscles)*: The patient experience violent jerking of the muscles (limbs), production of excessive saliva and sweating profusely. This phase may last for several minutes.May subside in 30 seconds but can last 2-5 minutes. The eyes roll upward and excessive salivation results in foaming at mouth.
5. Stupor: May last 5 minutes. The individual is limp and unresponsive. The pupils begin to react to light and return to their normal size.
6. Postictal: Is the period immediately after the seizure, the patient may be sleepy, semiconscious, and unable to speak clearly, The patient may complian of headache, muscle aches andtiredness, He/she may appear confused, and has no memory of the seizure and may sleep for several hours.
7. Myoclonic :A myoclonic seizure is characterized by a sudden excessive jerk of the limbs. The jerk may be forceful enough to hurl the person to the ground. These seizures are brief and may occur in clusters.
8. Atonic:Involves either a tonic episode or a paroxysmal loss of muscle tone and begins suddenly with the person falling to the ground. Consciousness usually returns by the time the person hit the ground and normal activity can resume immediately.

Status Epilepticus: State of continuous or rapidly recurring seizures in which the individual does not completely recover baseline neurologic functioning between seizures. This is a medical emergency.

Management

Investigations

1. History taking whichill reveal that it runs in families or recurrent seizures.
2. Encephalography will show increased electrical activity in the brain.
3. Serum electrolytes to rule out metabolic causes, such as hypoglycaemia or hypocalcaemia.
4. E.E.G: May reveal abnormal pattern of electrical activity
5. C.T.scan: May reveal presence of a space-occupying lesion.
6. SKULL X-RAY: To reveal fractures, tumours etc.
7. Lumbar puncture and CSF analysis: to rule out Increased Intracranial Pressure or infection such as meningitis.

Drug Therapy

Treatment aim

Treatment is aimed at controlling seizure not necessarily cure but some cases respond well and seizures disappear.

.

Hydantoin derivative (Phenytoin for tonic-clonic, partial simple and partial complex seizures).

PHENYTOIN

Dose: 3-4mg/Kg body weight or 150-300mg daily increasing gradually to the usual dose of 200-500mg daily.

Action: blocks sodium ion channels and stabilizing the cell hence preventing action potentials and rapid firing of neurons.

S/E: dizziness, insomnia, headache, blurred vision, nausea and vomiting, skin rash and hyperglycaemia.

Contra Indications: myocardial insufficiency, Hepatic dysfunction, diabetes and respiratory depression.

PHENOBARBITONE

Dose: 30-60mg PO daily. 100-200mg iv/im daily

Action: causes reversible depression of the activity of all excitable tissues , it facilitates inhibitory neurotransmission in the central nervous system by interacting with the gamma amino butyric acid receptors.

S/E: dizziness, drowsiness, hypotension, constipation, headache, liver damage and hypersensitivity.

CARBAMAZEPINE

Dose: 100-300 od or bd.

Action: blocks sodium channels thereby reducing action potential formation and the rate of firing. It inhibits uptake and release of norepinephrine in the brain. It inhibits polysynaptic response and the spread of seizure discharge and shortens the duration of after discharge.

S/E: vertigo, headache, blurred vision, anaemia, impotence and dry mouth.

Contra Indications: history of bone marrow suppression and hypersensitivity.

Contra Indication: acute intermittent porphyria, severe lung insufficiency, liver and kidney disease.

SODIUM VALPROATE

Dosage: 10-15mg/kg /day in two to three divided doses increasing gradually to 5mg/kg at weekly intervals.

*Action***:** acts by inhibition of gamma amino butyric acid transaminase thereby increasing increasing

Side Effects**:** nausea and vomiting, headache, thrombocytopenia and acute pancreatitis.

DIAZEPAM

*Dosage***:** 5-10mg intravenously repeated every 15 minutes till seizures are controlled, maximum dose of 30mg.

Action: action is mediated by enhancement of the activity of gamma amino butyric acid (GABA) a major neurotransmitter in the brain especially in the limbi system thalamus, hypothalamus and reticular.

Surgery: brain tumor or hematomas may be excised.

Management Of Status Epilepticus

Respiratory management: O_2 therapy, oral air-way suctioning and intubation as needed to maintain airway patency and prevent hypoxia.

Assessment of blood glucose and administration of intravenous glucose. Serum laboratory studies are done.

Slow administration of intravenous diazepam of 2 mg/min. Initially as bolus. If seizures continue, diazepam is given in a continuous IV drip of 10-15mg/l. Monitor for signs of respiratory depression and hypotension.

Administer IV phenytoin if diazepam is unsuccessful or phenobarbitone if phenytoin is unsuccessful.

Thiamine; if alcohol withdrawal occurs.

Nursing Measures

AIMS

Prevent injury as a result of the seizure.

To allay anxiety.

To maintain clear airway.

To prevent complications**.**

Usually patient is managed at home /OPD.

Pre-seizure precautions

Pad side rails with blankets or pillows. Keep side rails up and the bed in its lowest position when the patient is in bed. Keep the wheel chair, or stretcher brakes locked.

Keep suction and O_2 equipment readily available. Consider a heparin lock for IV access for high risk patients.

Avoid using glass or other breakable oral thermometers when taking patient's temperature.

Caution patient to lie down and push the call button if they experience a prodromal or aural warning. Encourage patient to empty the mouth of dentures or foreign objects.

Do not allow unsupervised smoking.

During Seizure

Remain with patient; observe for, record and report type, duration and characteristics of seizure activity and any post seizure response. This should include precipitating events, aura, automatisms, type and duration of movement. Changes in **Level of Consciousness**eye movement, bladder and bowel movement.

Prevent or break the fall and ease patient to the floor if the seizure occurs while patient is out of bed.

If patients' jaws are clenched, do not force an object between the teeth to avoid breaking the teeth.

Protect patients head from injury during seizure activity.

Do not restrain patient to prevent injuring the patient.

Roll patient into a side lying position to promote drainage of secretion and maintain a patent airway.

Loosen tight clothing to promote blood circulation.

Suction any secretion from the mouth with suctioning machine and wipe out any froth at the mouth.

Maintain patient's privacy. Clear nonessential people out of the room.

Administer anti-epilepsy drug as prescribed.

After The Seizure

Reassure and reorient patient, check neurologic status vital signs.

Provide a quiet, calm environment because sounds and stimuli can be confusing to the awakening patient. Keep talk simple and to a minimum. Speak slowly and with pauses between sentences. Do not offer food or drink until patient is fully awake.

Check patient's tongue for lacerations and body for injuries. Monitor urine for red or cola, color, which may signal myoglobinuria from muscle damage.

Monitor for the presence of weakness or paralysis.

Monitor for status epilepticus.

Iec/Rehabilitation

Assess patient's knowledge of measures that can prevent seizures and environmental hazards that can be life-threatening in the presence of seizure activity.

Advise patient to check into state regulation about automobile operation as regard to epileptic clients.

Caution patient to refrain from operating heavy or dangerous equipment, swim mining and possibly even tub bathing until he or she is seizure free.

Advice patient not sit near fire alone. Encourage stress management, progressive relaxation techniques.

Advise patient that some activities, such as climbing or bicycle riding, require careful risk-benefit evaluation.

Teach patient that use of stimulants and depressants should be avoided. Withdrawal from stimulants and depressants can increase the likelihood of seizures.

Teach patient adequate amounts of rest, avoiding physical and emotional stress, and maintaining a nutritious diet.

Encourage individuals where seizure that occur without warning not to be chewing gum or sucking on sweets.

Encourage patient to wear medic-alert bracelet for identification or to carry a medical information card.

Stress importance of taking prescribed medication regularly and on schedule and not discontinuing the medication without doctor's guidance.

Nursing **Care Problems**

1. Risk of injury related to seizure activity
2. Fear related to the possibility of seizures
3. Ineffective individual coping related to stress imposed by epilepsy
4. Deficient knowledge related to epilepsy and its control
5. Risk of respiratory failure related to airway blockage

Complications

The complications are divided into two

- Those that occur as a result of the condition epilepsy
- Permanent brain damage/neurologic deficits

a. Those that occur due to events of an epileptic attack
    - Injuries
    - Car accidents
    - Drowning
    - Medication side effects
    - Psychological problems
    - Pregnancy problems

Thank you for your full participation.Do not get tired.You will continue learning about conditions of the nervous system.

Let us now look at intra spinal tumours.

INTRASPINAL TUMOURS

Defionition

Intraspinal tumour is a growth of cells (mass) within or surrounding the spinal cord.

Causes

Any type of tumour may occur in the spine, including:

- Leukemia (blood cancer)
- Lymphoma(tumour in lymph nodes)
- Myeloma(tumour of bone marrow)

A small number of spinal tumours occur in the nerves of the spinal cord itself. Most often these are gliomas.(tumours of connective tissues)Tumours that start in spinal tissue are called primary spinal tumours. Tumours that spread to the spine from some other place (metatastasis) are called secondary spinal tumours. Tumours may spread to the spine from the breast, prostate, lung, and other areas.The cause of primary spinal tumours is unknown. Some primary spinal tumours occur with genetic defects.

Spinal tumours can occur:

Inside the cord (intramedullary)Tumours that occur within the spinal cord exert pressure on it cause symptoms ranging from localized or shooting pains and weakness and loss of reflexes above the tumor level to progressive loss of motor function and paralysis. These tumours cause sensory deficitys below the level of the lesion.

In the membranes (meninges) covering the spinal cord (extramedullary - intradural)

Between the meninges and bones of the spine (extradural) or tumours may extend from other locations. Most spinal tumours are extradural.

As it grows, the tumour can affect the:

Blood vessels

Bones of the spine

Meninges

Nerve roots

Spinal cord cells

The tumour may press on the spinal cord or nerve roots, causing damage. With time, the damage may become permanent.

Symptoms

Tumours in the spinal cord usually cause symptoms, sometimes over large portions of the body. Tumours outside the spinal cord may grow for a long time before causing nerve damage.

Symptoms may include:

Sensations abnormalities

Especially in the legs (may be in the knee or ankle, with or without shooting pain down the leg).

Cold sensation of the legs, cool fingers or hands, or coolness of other areas

Back pain: which worsen over periods of time.

In any area -- middle or low back are most common

Is usually severe and not relieved by pain medication

Is worse when lying down

Is worse with strain, cough, sneeze

May extend to the hip, leg, or feet (or arms), or all extremities

May stay in the spine

Incontinence of urine and feaces

Muscle spasms and loss of muscle function and weakness

Exams and Tests (Investigations)

A neurological examination may help pinpoint the location of the tumour. The following should be noted:

Abnormal reflexes

Increased muscle tone

Loss of pain and temperature sensation

Muscle weakness

Tenderness in the spine

Confirmatory diagnostic tests

Cerebrospinal fluid (CSF) examination

Cytology (cell studies) of CSF

Myelogram

Spinal CT scan

MRI and spinal X-rays

Treatment

The goal of treatment is to reduce or prevent nerve damage from pressure on (compression of) the spinal cord.

Treatments include:

Treatment of specific intraspinal tumors depends on thetype and location of the tumor and the presenting symptomsand physical status of the patient.Surgical interventionis the primary treatment for most spinal cord tumors.Other treatment modalities include partial removal of the tumor, decompression of the spinal cord, chemotherapy,and radiation therapy, particularly for intramedullary tumorsand metastatic lesions

Corticosteroids (dexamethasone) may be given to reduce inflammation and swelling around the spinal cord.

Surgery may be needed to relieve compression on the spinal cord. Some tumours can be completely removed. In other cases, part of the tumour may be removed to relieve pressure on the spinal cord.

Radiotherapy may be used with, or instead of, surgery.

Anticancer drugs have proven to be effective against most spinal tumours, but it may be recommended in some cases.

Possible Complications

i. Incontinence
ii. Spinal nerve compression
iii. Loss of sensation
iv. Paralysis
v. Permanent damage to nerves, disability from nerve damage.

You have finished learning about intaspinal tumours.Let us continue looking at Bell's palsy

BELL'S PALSY

Bell's palsy (facial paralysis) is due to unilateral inflammation of the seventh cranial nerve, which results in weakness or paralysis of the facial muscles on the affected side (Smeltzer., 2010).

It is a disorder where impulses from the seventh cranial nerve, which is responsible for motor innervation of facial muscles are blocked resulting in an inflammatory reaction around the nerve and produces unilateral facial weakness or paralysis.

Causes

The cause is unknown, although possible predisposing factors include:Vascular ischemia,

Viral disease (herpes simplex, herpes zoster),

Chronic ear infections

Autoimmune disease, or a combination of all of these factors.

The incidence is 13 to 34 cases per 100,000; it increases with age and among pregnant women in the third trimester (Campbell & Brundage, 2002; Shmorgun, Chan & Ray, 2002 in Smeltzer, 2010).

Pathology

Bell's palsy is considered to be a type of pressure paralysis. The inflamed, oedematous nerve becomes compressed to the point of damage, or its nutrient vessel is occluded, producing ischemic necrosis of the nerve.

The face is distorted from paralysis of the facial muscles leading to increased lacrimation (tearing), painful sensations in the face, behind the ear, and in the eye.The patient may

experience speech difficulties and may be unable to eat on the affected side because of weakness or paralysis of the facial muscles.

Clinical manifestation

Let us now look at how the patient with Bell's palsy present.

i. Pain on the affected side around the angle of the jaw or behind the ear for a few hours.
ii. Difficulty eating on the affected side because of the relaxation of the facial muscle.
iii. Difficulty in speaking resulting from facial muscle relaxation.
iv. Inability to make facial expressions e.g smiling or frowning
v. Drooling of saliva from the corner of the mouth due to mouth droops on the affected side.
vi. Increased insensitivity to sound.
vii. Patient fails to close the eye on the weak side.
viii. Taste perception is distorted over the affected anterior portion of the tongue.

Management

Investigations

Magnetic resonance imaging(MRI) & CT scan may show the inflammation and the possible cause.

Clinical presentations.

Electromyography to confirm presence of damage & determine severity.

Electrophysiological tests such as electromyography and Nerve conduction study, in which a muscle or nerve is artificially stimulated, may be used to assess the condition of facial muscles and the facial nerve.

Treatment

Aim

The objectives of treatment are to maintain the muscle tone of the face and to prevent or minimize denervation.

Acyclovir used to fight viral herpes infection, also benefits of shortening the course of the disease. Acyclovir 400mg bd

Analgesics e.g aspirin or ibuprofen may be used to relieve pain.

Drug: ibuprofen 400mg

- Action: control facial pain & discomfort.
- NOTE: If paralysed facial muscle prevent the eyes from closing completely, the eye must be protected from dryness by lubricating using eye drops.

Nursing care

OBJECTIVES

To improve nutrition.

To relieve pain.

To improve body image(self esteem)

Psychological care

The patient should be reassured that no stroke has occurred and that spontaneous recovery occurs within 3 to 5 weeks in most patients.Tell patient that spontaneous recovery usually occurs within two months toreduce anxiety. Help the patient adjust to the temporary change in his or her body.

Medication

Corticosteroid therapy (prednisone) may be prescribed to reduce inflammation and oedema; this reduces vascular compression and permits restoration of blood circulation to the nerve.

Pain control

Facial pain is controlled with analgesic agents such is diclofenac 50mg or brufen 400mg. Warm compresses may be applied to the involved side of the face to promote comfort and blood flow through the muscles.**Other treatment modalities**

Electrical stimulation may be applied to the face to prevent muscle atrophy. Although most patients recover with conservative treatment, surgical exploration of the facial nerve may be indicated in patients who are suspected of having a tumour or for surgical decompression of the facial nerve and for surgical treatment of a paralyzed face.

Nutrition

Advise the patient to chew using the unaffected part of the mouth to prevent pain. Educate patient the importance of eating a balanced diet.Arrange for privacy during meal times to reduce embarrassment.

Information, Education & Communication.

Teach the patient about the condition, its signs & symptoms and treatment.

Advise the patient to protect his or her affected eye by covering it with an eye patch, especially when out doors.

Teach patient to exercise the facial muscles by grimacing in front of a mirror.

Advise patient to chew food on the unaffected side of his mouth& to eat semi solid foods.

Complications

i. Blindness
ii. Tears when eating, known as crocodile tears
iii. Corneal ulceration
iv. Impaired vision
v. Speech problem occurs as a result of damage to the facial muscles.
vi. Loss of reduced sense of taste- if nerves do not repair properly.
vii. A contracture is where your facial muscles are permanently tense.
viii. Untreatable damage to the cranial nerve.

PERIPHERAL NEUROPATHIES

Thanks very much for your concentration. You have just finished looking at Bell's palsy and now you will looking at peripheral neuropathies.

Definition

A peripheral **neuropathy** (disorder of the nervous system) is a disorder affecting the peripheral motor, sensory, or autonomic nerves (Smeltzer et al., 2010). Peripheral nerves connect the spinal cord and brain to all other organs. They transmit motor impulses from the brain and relay sensory impulses to the brain. A mononeuropathy affects a single peripheral nerve; multiple mononeuropathy or mononeuritis multiplex indicates the involvement of multiple single

peripheral nerves or their branches. Polyneuropathies are characterized by bilateral and symmetric disturbance of function, usually beginning in the feet and hands. (Most nutritional, metabolic, and toxic neuropathies take this form.)

Causes

The most common causes of peripheral neuropathy are diabetes, alcoholism, and occlusive vascular disease. These disorders result in hypoxia or atrophy of the peripheral nerve. Many bacterial and metabolic toxins and exogenous poisons also cause peripheral neuropathy. Because of the growing use of chemicals in industry, agriculture, and medicine, the number of substances causing peripheral neuropathies and the incidence of peripheral neuropathies have increased. In developing countries, leprosy is a major cause of severe nerve disease because *Mycobacterium laprae* invade the peripheral nervous system.

Clinical Manifestations

The major symptoms of peripheral nerve disorders are loss of sensation, muscle atrophy, weakness, diminished reflexes, pain, and paresthesia (numbness, tingling) of the extremities. The patient frequently describes some part of the extremity as numb.

Autonomic features include decreased or absent sweating, orthostatic hypotension, nocturnal diarrhoea, tachycardia, impotence, and atrophic skin and nail changes.

Management

Peripheral nerve disorders are diagnosed by history, physical examination, and somatosensory evoked potentials.

1. Drug therapy
2. Pyridoxine (Vitamin B 6)

Nursing care

The care focuses on relieving the symptoms and passive exercises of the affected limb.

TOXOPLASMOSIS

Let us now look at toxoplasmosis.Have you heard about this condition before?Well, you will learn about it today.

Definition:

This is an infection caused by toxoplasma gondii an intracellular protozoan parasite found widely in the environment.

The protozoa is predominantly found in Cats, Birds and Domesticated animals.

This condition also presents in immune compromised people

Mode of transmission

Mode of transmission in humans is **faeco-oral** (mostly through contaminated water and raw/uncooked meat). It can be transmitted through uncooked meat and cat faeces.

This microbe infects the brain and can cause raised intracranial pressure, which leads to headaches and vomiting.

Clinical manifestation

Vomiting and headache due to increased intracranial pressure. Confusion, motor weakness and fever. In the absence of treatment, disease progression results in seizures, stupour and coma.

Disseminated toxoplasmosis is less common, but can affect the eyes and cause pneumonia.

Management

Definitive diagnosis of toxoplasmosis requires radiographic testing (usually an MRI scan).

Drug therapy

The infection is treated with drugs such as pyrimethamine, sulfadiazine and clindamycin. Leucovorin may also be used to prevent the side-effects of pyrimethamine.

Prophylaxis against toxoplasmosis is through taking Trimethoprime-Sulphadoxine.

Recommendationsto advise HIV-positive individuals to:

- Avoid ingestion of undercooked meat
- To wash hands after any contact with soil
- To avoid emptying cat litter trays, or to empty trays daily and wash hands thoroughly after every disposal.

Thank you for your attention. You have just finished learning about Toxoplasmosis. Now you are going to learn about a deforming condition which used to be common but rarely seen today.

What comes to your mind when the term Leprosy is mentioned?

LEPROSY

Definition: Leprosy is an infectious chronic granulomatous disease principally affecting the skin and peripheral nervous system, caused by ***Mycobacterium leprae.*** Mycobacterium.leprae is an obligate intracellular acid-fast gram-positive bacillus with an affinity for macrophages and

Schwann cells, has slow replication within the Schwann cells, hence the long incubation period of leprosy.

Epidemiology

Globally, the number of leprosy cases is declining. During 2011, about 219,000 new cases were reported. At the beginning of 2012, 180,000 people mainly in Asia and Africa. The majority of cases are now centred in Southeast Asia, Africa and South America, with 64% of all cases occurring in India. (WHO, 2012)

Causes And Predisposing Factors

- Humans are the primary reservoir of Mycobacterium. leprae. Animal reservoirs of leprosy have been found in 3 species: 9-banded armadillos, Chimpanzees, and Mangabey monkeys.
- Leprosy is not a highly infectious disease. The principle means of transmission is by aerosol spread from infected nasal secretions to exposed nasal and oral mucosa. Leprosy is not generally spread by means of direct contact through intact skin, though close contacts are most vulnerable. The incubation period is 6 months to 40 years or longer. The mean incubation period is 4 years for tuberculoid leprosy (TT) and 10 years for lepromatous leprosy (LL).
- Most persons are immune to leprosy. Subclinical disease is common in endemic areas, and the infection progresses to clinical disease in only a selected few.
- Exposure to nasal discharge of those that remain untreated for years is thought to be the main cause of infection. Transmission is not completely understood. In addition to exposure to respiratory secretions, exposure to insect vectors and infected soil has been suspected as possible as a possible mode of transmission. In endemic countries, household contacts of patients are at increased risk for leprosy. The relative risk is 8-10 for LL and 2-4 for TT. In none endemic countries, household contacts rarely acquire the disease. HIV infection is not a risk factor for acquiring leprosy, nor does it increase the clinical symptoms or virulence of leprosy.

Incidence And Prevalence

- Race: Leprosy occurs in all races. African blacks have a high incidence of the tuberculoid form of leprosy. People with light skin and Chinese individuals tend to have the lepromatous type of leprosy. Leprosy is endemic in Asia, Africa, the Pacific basin, and Latin America (excluding Chile). It is more a rural than urban disease.

- Sex: In adults, the lepromatous type of leprosy is more common in men than in women after puberty, with a male-to-female ratio of 2:1. In children, the tuberculoid form predominates, and no sex preference exists. Women tend to have a delayed presentation, which increases rates of deformity.
- Age: Leprosy has a bimodal age distribution, with peaks at ages 10-14 years and 35-44 years. The disease is rare in infants. Children appear to be most susceptible to disease and tend to have the tuberculoid form.

Classification

One classification classifies leprosy as either tuberculoid or lepromatous. These are further broken down into intermediate or borderline.

Another classification classifies leprosy as either paucibacillary or multibacillary depending on the number of lesions and bacterial index (BI).

- *Paucibacillary* disease (Indeterminate leprosy [IL] and TT) has fewer than 5 lesions and no bacilli on smear testing. Five or more lesions with or without bacilli (Borderline) leprosies and LL) is considered ***Multibacillary*** disease.
- ***IL:*** This early form causes one to a few hypopigmentated or sometimes erythematous macules. Sensory loss is unusual.
- ***TT:*** Skin lesions are few. One erythematous large plaque is usually present, with well-defined borders that are elevated and that slope down into an atrophic centre.
- ***Borderline Tuberculoid leprosy (BT):*** Lesions in this form are similar to those in the tuberculoid form, but they are smaller and more numerous.
- ***Borderline borderline leprosy (BB):*** Cutaneous lesions consist of numerous, red, irregularly shaped plaques that are less well defined than those in the tuberculoid type.
- ***Borderline lepromatous leprosy (BL):*** Lesions are numerous and consist of macules, papules, plaques, and nodules.
- ***LL:*** Early cutaneous lesions consist mainly of pale macules. Late infiltrations are present with numerous bacilli. Macular lesions are small, diffuse, and symmetric.

Pathophysiology

In general, leprosy affects the skin, peripheral nerves, and eyes. Systemic symptoms may occur. Specific symptoms vary with the severity of the disease.

Leprosy commonly affects the superficial peripheral nerves, skin, mucous membranes of the upper respiratory tract, anterior chamber of the eyes, and testes. These areas tend to be cool parts of the body.

Tissue damage depends on the degree to which cell-mediated immunity is expressed, the type and extent of bacillary spread and multiplication, the appearance of tissue-damaging immunologic complications (i.e. lepra reactions), and the development of nerve damage and its sequelae. The strength of the host's immune system influences the clinical form of the disease. A strong cell-mediated immunity and a weak humoral response results in mild forms of disease, with a few well-defined nerves involved and lower bacterial loads. A strong humoral response but relatively absent cell-mediated immunity results in LL, with widespread lesions, extensive skin and nerve involvement, and high bacterial loads. Therefore, a spectrum of disease exists such that cell-mediated immunity dominates in mild forms of leprosy and decreases with increasing clinical severity. Meanwhile, humoral immunity is relatively absent in mild disease and increases with the severity of diseases.

Types Of Reactions

Leprosy reactions;During the course of untreated or even treated leprosy, the immune system may produce inflammatory reactions. There are two (2) types.

Type 1: Results from a spontaneous increase in cell-mediated immunity. These reactions can cause fever and inflammation of the pre-existing skin and peripheral nerve lesions, resulting in skin oedema, erythema, and tenderness and worsening nerve functioning. These reactions particularly if not treated early contribute significantly to nerve damage. Because the immune response is increased, these reactions are termed reversal reactions, despite the apparent clinical worsening.

Treatment for type 1: patients with type 1 reaction (except minor skin reaction) are given predinisolone 40-60mg per oral once per day initially, followed by low maintenance dose of as low as 10-15mg per day for a few months. Minor skin inflammation should not be treated.

Type 2: Also known as erythema nodosum leprosum or (ENL) are systemic inflammatory reaction that appears to be a vasculitis or panniculitis and probably involve circulating immune complex deposition or increased T-helper cell function. There have become less common since clofazimane was added to the drug regimen. Patients may develop erythematous and painful papules or nodules that may postulate and ulcerate and cause fever, neuritis, lymphadenitis,

orchitis, arthritis (particularly in large joints usually knees), and glomerulonephritis. Hemolytic or bone marrow suppression may cause anemia, and hepatic inflammation may cause mild abnormalities in liver function test.

Treatment for type 2: First and second episodes of ENL may be treated if mild with Aspirin or, if significant, with one week of predinisolone 40-60mg once per day plus antimicrobials. For recurrent cases Thalidomide 100-300mg orally once per day. However because of its teratogenicity, thalidomide should not be given to women who may become pregnant. Adverse effects are mild constipation, mild leucopenia and sedation.

Signs And Symptoms

Specific symptoms vary with the severity of the disease

- Prodromal symptoms are generally so slight that the disease is not recognized until a cutaneous eruption is present. However, 90% of patients have a history of numbness first, sometimes years before the skin lesions appear.
- Temperature is the first sensation that is lost. Patient cannot sense extremes of hot or cold. The next sensation lost is light touch, then pain, and finally deep pressure. These losses are especially apparent in the hands and feet; therefore, the chief complaint may be a burn or ulcer in an anesthetic extremity.
- Other parts of the body that might be affected are the cool areas: superficial peripheral nerves, anterior chamber of the eyes, testes, chin, malar eminences, earlobes, and knees. From this stage, most lesions evolve into the tuberculoid, borderline, or lepromatous types.
- Important physical signs generally affect 3 general areas: cutaneous (skin) lesions, neuropathies, and eyes.
- For cutaneous lesions, assess the number and distribution of skin lesions. A hypo pigmented macule with a raised border is often the first cutaneous lesion. Plaques are also common. Lesions may or may not be hypo esthetic. Borderline disease often appears with lesions on the buttocks.
- Regarding neuropathies, assess for areas of hypo esthesia (light touch, pinprick, temperature and anhidrosis), especially peripheral nerve trunks and cutaneous nerves. The most common nerve affected is the posterior tibial nerve. Others commonly damaged are the ulnar, median, lateral popliteal, and facial nerves. Bedsides sensory loss, there may be associated tenderness and motor loss.

- Eye damage is most often seen with facial lesions. **Lagophthalmos** (inability to close the eye), a late finding in LL, results from involvement of the zygomatic and temporal branches of the facial nerve (cranial nerve, CN VII. Involvement of the ophthalmic branch of the trigeminal nerve (CN V2) can result in reduced corneal reflex, leaving dry eyes and reduced blinking.
- Others Saddle nose, madolosis.

Diagnosis

The diagnosis of leprosy is primarily a clinical one. Diagnosis was based on 1 of 3 signs:

1)- Hypopigmented or reddish patches with definite loss of sensation

2)- Thickened peripheral nerves

3)- Acid-fast bacilli on skin smear or biopsy material

Management

A. Investigations

- **Tissue Smear Testing/Slit-Skin Smear (Skin Biopsy**): An incision is made in the skin, and the scalpel blade is used to obtain fluid from a lesion. The fluid is placed on a glass slide and stained by using the Ziehl-neelsen acid-fast method or the fite method to look for organisms per 100 bacilli. Skin smears have specificity but low sensitivity because 70% of all patients with leprosy have negative smears. However, this test is useful because it detects the most infectious patients.
- **Histamine Testing:** This is used to diagnose postganglionic nerve injury. Histamine diphospate is dropped on normal skin and affected skin, and a pinprick is made through each site. The site forms a wheal on normal skin but not where nerve damage is present.
- **Methacholine Sweat Testing:** An intradermal injection of methacholine demonstrates the absence of sweating in leprosy lesions. This test is useful in dark-skinned patients in whom the flare with the histamine test cannot be seen.
- **Others include:** leprimin, serology and PCR testing.

B. Treatment

- The management of leprosy includes early pharmacotherapy and physical, social, and psychological rehabilitation. The goals of pharmacotherapy are to stop the infection, reduce morbidity, prevent complications and eradicate the disease. Since 1981, MDT (Multiple drug

therapy) has been advocated by WHO and The USA. MDT prevents dapsone resistance, quickly reduces contagiousness, and reduces relapses, reactions, and disabilities.

Treatment Plans

1. Non- Pharmacological/ Patient Education

Patients first need an explanation of the diagnosis and prognosis. Their fears should be addressed because of the cultural stigma associated with leprosy. The physician should also refute any myths that the patient may have about leprosy. Patients may need psychological counseling because they may have difficulty in coming to terms with the disease or in feeling rejected by society. The patient should be reassured that, within a few days of starting MDT, they are not infectious and can lead a normal life. Patients need education about how to deal with anesthesia of a hand or foot. They must learn to carefully inspect their extremities for trauma daily. Patients should also be told to wear proper footwear and protective equipment as necessary. Inexpensive canvas shoes with protective insoles are as effective as special orthopaedic shoes. Inspecting limbs and eyes for onset of anesthesia or weakness is also important. Physical therapy and occupational therapy are important tools in rehabilitation. Patients must learn how to recognize the onset of lepra reactions, and they should be told to seek immediate medical attention if these reactions develop. Potential deformities can be prevented by educating patients about how to deal with existing nerves damage and by treating any sequelae of this damage.

2. Pharmacological Treatment

- The length of treatment ranges from 6 months to 2 years. Patients are considered non-infectious within 1-2 weeks of treatment (usually after the first dose). These drugs are conveniently packaged in monthly calendar blister packs. Monitor for drug resistance and adverse reactions to medications.
- Paucibacillary disease can be treated with a combination of 2 drugs, whereas Multibacillary disease requires triple-drug therapy. Single skin lesions (Paucibacillary) can be treated with a single dose of 3 drugs. The length of treatment depends on the type of disease and on the access to drugs.

Current WHO recommendations for treatment of leprosy are as follows:

- Paucibacillary disease: Dapsone 100mg/day plus rifampicin 600mg once a month for 6 months.

- Multibacillary disease: Dapsone 100mg/day plus rifampicin 600mg once a month plus Clofazimine 300mg once a month and 50mg/day for 1 year.
- Single skin lesion: A single dose of rifampicin 600mg, Ofloxacin 400mg, and Minocycline 100mg.

3. Surgical Treament

Emergency surgery may be necessary if a patient with profound nerve inflammation presents with a nerve abscess or loss of nerve function secondary to compression. Prompt recognition and surgical drainage of the abscess can often restore nerve function. Elective surgery may be required for correction of lagophthalmos (i.e. inability to close the eye).

Reconstructive surgery can be used to repair nasal collapse in LL.

Other surgery may be needed to improve function or for cosmesis.Contractures can be surgically repaired.

Complications

1. If severe and left untreated, leprosy can cause clinically significant and debilitating deformity such as loss of digits.
2. Lagophthalmos
3. Madalosis
4. Saddle nose
5. Nerve damage
6. Reversal reactions.

Rehabilitation Of A Patient With Leprosy

Rehabilitation is the important in leprosy control. Prompt treatment and detection is important. This may include accessories and corrective surgical procedures.

Socio-economic and vocational rehabilitation also forms an integral part in the care of leprosy patient.

Thank you for your enthusiasm to learning more.You were looking at leprosy and you will now be looking at Guillain-Barre syndrome

GUILLAIN-BARRÉ SYNDROME

Guillain-Barre syndrome is an autoimmune attack of the peripheral nerve myelin. The result is acute, rapid segmental demyelination of peripheral nerves and some cranial nerves, producing ascending weakness with **dyskinesia** (inability to execute voluntary movements), hyporeflexia, and **paresthesias** (numbness(Smeltzer et al., 2010).

Predisposing factors

1. Respiratory or gastrointestinal infection, although vaccination, pregnancy and surgery have also been identified as antecedent events (Bella & Chad, 1998 in Brunner and Suddarth, 2010).
2. Infection with *Campylobacter jejuni* (a relatively common gastrointestinal bacterial pathogen) precedes Guillain-Barreé syndrome in a few cases (Ho & Griffin, 1999; Lindenbaum, Kissel & Mendel, 2001 in Brunner and Suddarth, 2010).

Pathophysiology

Myelin is a complex substance that covers nerves, providing insulation and speeding the conduction of impulses from the cell body to the dendrites. The cell that produces myelin in the peripheral nervous system is the Schwann cell. In Guillain-Barré the Schwann cell is spared, allowing for remyelination in the recovery phase of the disease.

Guillain-Barré is the result of a cell-mediated immune attack on peripheral nerve myelin proteins (Ho & Griffin, 1999 in Brunner and Suddarth, 2010). The best-accepted theory is that an infectious organism contains an amino acid that mimics the peripheral nerve myelin protein. The immune system cannot distinguish between the two proteins and attacks and destroys peripheral nerve myelin. Studies indicate that an exact location within the peripheral nervous system, the ganglioside GM1b, is the most likely target of the immune attack(Yuki, Ang, Koga et al., 2000 in Brunner and Suddarth, 2010). With the autoimmune attack there is an influx of macrophages and other immune-mediated agents that attack myelin, cause inflammation and destruction, and leaves the axon unable to support nerve conduction.

Clinical Manifestations

1. Classic Guillain-Barré begins with muscle weakness and diminished reflexes of the lower extremities.

2. Hyporeflexia and weakness progress and may result in quadriplegia.
3. Demyelination of the nerves that innervate the diaphragm and intercostal muscles results in neuromuscular respiratory failure.
4. Sensory symptoms include paresthesias of the hands and feet and pain related to the demyelination of sensory fibers.
5. Cranial nerve demyelination can result in a variety of clinical manifestations.
6. Optic nerve demyelination may result in blindness.
7. Bulbar muscle weakness related to demyelination of the glossopharyngeal and vagus nerves results in an inability to swallow or clear secretions.
8. Vagus nerve demyelination results in autonomic dysfunction, manifested by instability of the cardiovascular system.
9. The presentation is variable and may include tachycardia, bradycardia, hypertension, or orthostatic hypotension.

The symptoms of autonomic dysfunction occur and resolve rapidly. Guillain-Barré does not affect cognitive function or level of consciousness. While the classic clinical features include areflexia and ascending weakness, variation in presentation occurs. There may be
a sensory presentation, with progressive sensory symptoms, an atypical axonal destruction, and the Miller-Fisher variant, which includes paralysis of the ocular muscles, ataxia, and areflexia.

Management

Investigations

A history of a viral illness in the previous few weeks suggests the diagnosis.

Changes in vital capacity and negative inspiratory force are assessed to identify impending neuromuscular respiratory failure.

Serum laboratory tests are not useful in the diagnosis. However, elevated protein levels are detected in CSF evaluation, without an increase in other cells.

Medical Management

Because of the possibility of rapid progression and neuromuscular respiratory failure, Guillain-Barré is a medical emergency, requiring intensive care unit management. Careful assessment of changes in motor weakness and respiratory function alert the clinician to the physical and respiratory needs of the patient.

Respiratory therapy or mechanical ventilation may be necessary to support pulmonary function and adequate oxygenation. Mechanical ventilation may be required for an extended period.
The patient is weaned from mechanical ventilation when the respiratory muscles can again support spontaneous respiration and maintain adequate tissue oxygenation.

Other interventions are aimed at preventing the complications of immobility. These may include the use of anticoagulant agents and thigh-high elastic compression stockings or sequential compression boots to prevent thrombosis and pulmonary emboli.

Plasmapheresis and is used to directly affect the peripheral nerve myelin antibody level. The therapy decreases circulating antibody levels and reduces the amount of time the patient
is immobilized and dependent on mechanical ventilation.

Other considerations
The cardiovascular risks posed by autonomic dysfunction require continuous ECG monitoring. Tachycardia and hypertension are treated with short-acting medications such as alpha-adrenergic blocking agents. Hypotension is managed by increasing the amount of IV fluid administered. The use of short-acting agents is important because autonomic dysfunction is very labile.

Nursing Process:
Assessment
Ongoing assessment for disease progression is critical. The patient is monitored for life-threatening complications (respiratory failure, cardiac dysrhythmias, DVTs) so that appropriate interventions can be initiated. Because of the threat to the patient in this sudden, potentially life-threatening disease, the nurse must assess the patient's and family's ability to cope and their use of appropriate coping strategies.
Nursing Diagnoses
Based on the assessment data, the patient's major nursing diagnoses may include the following:

- Ineffective breathing pattern and impaired gas exchange related to rapidly progressive weakness and impending respiratory failure.
- Impaired physical mobility related to paralysis.

- Imbalanced nutrition, less than body requirements, related to inability to swallow.
- Impaired verbal communication related to cranial nerve dysfunction.

Activity

Write the nursing care plan based on identified problems above

MYASTHENIA GRAVIS

Myasthenia gravis, an autoimmune disorder affecting the myoneural junction, is characterized by varying degrees of weakness of the voluntary muscles (Smeltzer et al., 2010).

Women tend to develop the disease at an earlier age (20 to 40 years of age) compared to men (60 to 70 years of age), and women are affected more frequently.

Pathophysiology

Normally, a chemical impulse precipitates the release of acetylcholine from vesicles on the nerve terminal at the myoneural junction. The acetylcholine attaches to receptor sites on the motor end plate, stimulating muscle contraction. Continuous binding of acetylcholine to the receptor site is required for muscular contraction to be sustained.

In myasthenia gravis, autoantibodies directed at the acetylcholine receptor sites impair transmission of impulses across the myoneural junction. Therefore, fewer receptors are available for stimulation, resulting in voluntary muscle weakness that escalates with continued activity. These antibodies are found in 80% to 90% of the people with myasthenia gravis. Eighty percent of persons with myasthenia gravis have either thymic hyperplasia

or a thymic tumour , and the thymus gland is believed to be the site of antibody production. In patients who are antibody negative, it is believed that the offending antibody is directed at a portion of the receptor site rather than the whole complex.

Clinical Manifestations

The initial manifestation of myasthenia gravis usually involvess the ocular muscles. Diplopia (double vision) and ptosis (drooping of the eyelids) are common.

Other symptoms include

- weakness of the muscles of the face and throat (bulbar symptoms) and
- generalized weakness.
- Weakness of the facial muscles will result in a bland facial expression.

- Laryngeal involvement produces **dysphonia** (voice impairment) and increases the patient's risk for choking and aspiration. Generalized
- weakness affects all the extremities and the intercostal muscles, resulting in decreasing vital capacity and respiratory failure.

Myasthenia gravis is purely a motor disorder with no effect on sensation or coordination.

Management

Investigations

1. An anticholinesterase test is used to diagnose myasthenia gravis.
2. The thymus gland, which is a site of acetylcholine receptor antibody production, is enlarged in myasthenia gravis. MRI demonstrates this enlargement in 90% of cases

Medical Management

Management of myasthenia gravis is directed at improving function and reducing and removing circulating antibodies. Therapeutic modalities include administration of anticholinesterase agents and immunosuppressive therapy, plasmapheresis, and thymectomy.

Drug therapy

a. Anticholinesterase agents such as
   - pyridostigmine bromide (Mestinon)
   - neostigmine bromide (Prostigmin)

These provide symptomatic relief by increasing the relative concentration of available acetylcholine

at the neuromuscular junction.

b. Immunosuppressive therapy is to reduce the production of the antibody. Corticosteroids suppress the patient's immune response, thus decreasing the amount of antibody production e.g. Prednisolone

Nursing Management

Because myasthenia gravis is a chronic disease and most patients are seen on an outpatient basis, much of the nursing care focuses on patient and family teaching.

You have just been learning aboutmyasthenia gravis. Now let us look at Parkinson's disease

PARKINSON'S DISEASE

Definition: this a progressive disease characterized by muscle rigidity, akinesia and involuntary tremors. The disease is also called paralysis agitans or shaking palsy.

Causes

The cause is unknown, however, is associated with the following:

1. Dopamine deficiency that prevents the brain cells from performing their normal inhibitory function.
2. Exposure to toxins such manganese and carbon monoxide that destroy cells in the substantia nigra.
3. Genetics
4. Environmental factors
5. Endotoxin (lipopolysaccharide)

Clinical Manifestations

- Muscle rigidity
- Akinesia
- Tremors usually begins with fingers
- Fatigue with activities
- Muscle cramps of legs, neck and trunk
- Oily skin
- Increased perspiration
- Insomnia and mood changes
- Mask like facial presentation
- Difficulties in walking

Stages of Parkinson Disease

Stage 1: Initial stage

- Unilateral limb involvement
- Minimal weakness
- Hand and arm trembling

Stage 2: Mild stage

- Bilateral limb involvement
- Masklike facies

- Slow, shuffling gait

Stage 3: Moderate disease

- Increased gait disturbance

Stage 4: Severe disability

- Akinesia
- Rigidity

Stage 5: complete dependence

Management

Investigations

1. Urinalysis
2. CT scan
3. MRI

Treatment

The aim of treatment is to relieve symptoms but there is no specific treatment.

a. Anticholinergics- benztropine
b. Dopamine agonists- bromocriptine, cabergoline
c. Levodopa combinations- carbidopa/levodopa and a lot more other drugs as indicated.

Nursing care

The nursing care you will give to patient with this condition focuses on

1. Exercise and ambulation
2. Meeting self-care needs
3. Injury prevention
4. Balanced nutrition
5. Improved communication skills
6. Psychosocial support

Thank you for your patience.you have just been learning about Parkinson's disease. You will now be learning about Rabies.

RABIES

Definition: this is a deadly viral infection that primarily affects the brain and the spinal cord .

Causes: rabies is caused by a bullet shaped virus known as lyssaviruses of the rhabdoviridae family.

Transmission:

It is a zoonotic disease (i.e., transmitted by animals), most commonly by a bite from an infected animal but occasionally by other forms of contact. A bite or a scratch introduces the virus which is usually present in the saliva of rabid animal. Rabies is almost invariably fatal if post-exposure prophylaxis is not administered prior to the onset of severe symptoms.The animal hosts include dogs, cats, foxes, bats etc.

Incubation period: The incubation period ranges from a few days to several years (most commonly 3-8 weeks(WHO, 2009).

Pathophysiology

Following entry into the body for example; After a rabbit bite, the virus start multiplying in the myocytes at the bite site and later moves to the central nerves system and reaches the brain .

Multiplication continues in the brain and can spread outwards along the peripheral nerves causing interstitial neuritis.

It may also reach the muscles and salivary grand's (this shows that it is not strictly neurotropic) and also to other organs such as the lungs, adrenals, renal s, bladder and the testicles causing priapism.Rabies virus will cause widespread changes throughout the central nervous system and cause an immune reaction wherever it goes giving rise to the typical signs and symptoms of Rabies. This causes neural necrosis and mononuclear cellular infiltration especially in the thalamus, hypothalamus, pons and medulla.Cranial nerve nuclei's are also extensively damaged and neural changes are present in the spinal cord especially in posterior horns.The virus causes an immune reaction where ever it goes giving rise to the typical signs and symptoms of rabies.

Clinical Manifestation

The clinical manifestation are classified into three; Prodromal or Invasive, Excitement or Neurological and Terminal or Paralytic phase.

Prodromal/Invasive

This phase is characterized by;

- Fever due to damage to the hypothalamus by the virus and necrosis.
- Restlessness due to neural necrosis.
- Sore throat due to regurgitation of gastric contents.

- Headache due to encephalitis.
- Numbness, anesthesia, burning and cord sensation may be felt along the peripheral nerves involved and the site of the bite due to damage of the neurons by the virus.
- insomnia
- Reflexes are increase
- Pupils are dilated

Excitement/Neurological

This phase is characterized by:

i. Marked excitation which may occur due to increased sensitivity and stimulation on the brain cells.
ii. There is delirium associated with generalized convulsion due to neural necrosis and increased destruction of the brain cells.
iii. There is tonic or clonic contraction of the muscles due to impaired nerve supply to the muscles.
iv. Nausea and vomiting
v. Painful spasms of the pharynx and larynx worsened by drinking water
vi. Episthotonus due to muscle spasms
vii. Voice become hoarse

- Mucus collects in the mouth
- Spasms of other muscle are present
- There is an exaggerated fear of water that even the mention can trigger spasms (Hydrophobia
- There is profuse drooling of saliva due to lack of invasion of the salivary grand's by the glossal and hypoglossal nerve as they are damaged by the virus.
- Hallucination
- Convulsion will also set in
- Parasthesiae of the trunk, limbs and face sets in.
- Sexual excitement with priapism is common
- Squinting may develop

Face may lack expression

There may be inability to close mouth and eyes**Terminal/Paralytic**

i. The patient becomes quiet and unconscious due to the damage of the brain cells and spinal cord.
ii. There is loss of bowel and urinary control due to damage to the vagus nerve.
iii. Spasms ceases with progressive paralysis which is due to motor neural damage.
iv. The patient is depressed and apathetic
v. Fever is present
vi. Malaise
vii. Weakness
viii. Ataxia
ix. Paralysis due acute progressive ascending myelitis. Paralysis begins in the legs
x. Encephalitis develops and patient may go into coma.
xi. Death may not take place for a month but is innevitable

Management

Investigations

1. Detection of viral antigens by direct fluorescent antibody test (FAT) or by ELISA in clinical specimens, preferably brain tissue (collected post mortem).
2. Detection by FAT on skin biopsy (ante mortem).
3. Detectable rabies-neutralizing antibody titre in the serum or the CSF of an unvaccinated person.
4. Detection of viral nucleic acids by PCR on tissue collected post mortem or intra vitam in a clinical specimen (brain tissue or skin, cornea, urine or saliva(WHO, 2009)

Drug therapy

First aid: the wound bite site must be thoroughly washed under running water and leave the site uncovered.

Rabies Vaccine Bp Pasteur Merieux (Pasteur Merieux)

Freeze-dried inactivated Wistar rabies virus strain PM/WI 38 1503-3M cultivated in human diploid cells. Single dose vial with syringe containing diluent.

IMPORTANT. Studies have shown that when this vaccine is injected into the gluetal region there is a poor response. Concomitant administration of chloroquine may also affect the antibody response. Because of the potential consequences of inadequately treated rabies exposure and because there is no indication that foetal abnormalities have been associated with rabies

vaccination, pregnancy is **not** considered a contra-indication to post-exposure prophylaxis. If there is substantial risk of exposure to rabies, pre-exposure prophylaxis may also be indicated during pregnancy.

Dose: Prophylactic, 1ml by deep subcutaneous or intramuscular injection in the deltoid region, on days 0, 7, and 28; also booster doses every 2-3 years to those at continued risk; see above for 2 dose schedule.

Post-exposure, 1ml by deep subcutaneous or intramuscular injection in the deltoid region5 doses; first dose day 0 followed by injection on day 3,7,14,28..Anti tetanus toxoid 0.5mls im start

*Side effects***:** Rash, hallucination, nausea and vomiting.

Prevention

- Immunize all dogs and cats owned by an individual or by the community and reduce the size of the ownerless dog population by reproduction control, reduction of the carrying capacity of the environment and law enforcement when needed.
- Immunize any person with proven or probable exposure to rabies and administer rabies immunoglobulin in case of severe exposure (WHO category 3).
- Humans at high risk (e.g. laboratory personnel, professions at high risk) must receive pre-exposure immunization: 3 injections of an intramuscular dose on days 0, 7, and 28.
- Pre-exposure vaccine regimen: 1 dose of a cell culture or purified duck embryo vaccine on days 0, 7, 28. A variation of a few days is acceptable. The dose is 1 standard intramuscular dose (1 ml or 0.5 ml according to vaccine type). vaccine may be given intradermally (0.1 ml on days 0, 7, 28) but intramuscular injections are preferable if antimalarial chemoprophylaxis (e.g. chloroquine) is being used concurrently or there is a possibility of an immune-compromised state (antibody response may be impaired if the intradermal method is used (WHO, 2009). Below is self-test for you to attempt.

SELF TEST

1. Intracranial infections include the following except

a. Fangi

b. Bacteria

c. Viral

d.infestations

2. CVA stands for

a. Congestive Vascular Accident

b. Cerebral Vascular Accident

c. Cerebral Ventral Accident

d. Congestive Ventral Accident

3. The causes of meningitis include the following except

a. Fungal infections

b. Bacterial infections

c. Parasitic infestations

d. Viral infection

4. Peripheral neuropathies are

a. Disease of the fingers

b. Disorders of the nervous system

c. affects the knees

True/False

5. A dog bite causes rabies T/ F

6. Myaesthernia Gravis is a neurological disorder T/ F

7. Toxoplasmosis is a disease of the nerve T/ F

8. Leprosy belongs to endocrine disorders T/ F

Answers: Q1 D Q2 B. Q3 C. Q4B Q5 F. Q6 T. Q7 T. Q8 F.

2.7 SUMMARY

You have come to the unit of this, in unit you have learnt about the central nervous systems, the investigation and role of the in carrying various investigations and procedures.

You have also learnt about various medical conditions that affect the central nervous system and how they impact on human health. Some of the conditions learnt include cerebral vascular accident, meningitis, Bell's palsy, etc. It is very important for you to know these conditions so that you can effectively manage patients/clients who come under care.

In the next unit you will learn about conditions that affect the endocrine system. Please take to revise the on the anatomy and physiology of the endocrine system.

2.8 References

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UNIT 3: ENDOCRINE SYSTEM

3.1 Introduction

The system works like the fuel and electrical system of a motor vehicle, it is the electrical and hormonal system of the body. The multiple activities of the cells, tissues and organs of the body are coordinated by the interplay of several types of chemical messenger systems such as endocrine hormones which are released by glands or specialized cells into the circulating blood and influence the functions of cells at another location in the body. You have also learnt that the endocrine system is an integrated chemical communication and coordination system that enables reproduction, growth and development, and regulation of energy. Therefore, in this unit you will learn the various disorders of the endocrine system.

3.2 Objectives

At the end of this unit you should be able to

1. Describe the anatomy and physiology of the endocrine system
2. Explain the role of the nurse in investigations and procedures of the endocrine system
3. Discuss the management of patient with metabolic disorders
4. Discuss the management of a patient with pancreatic disorders
5. Discuss the management of a patient with pituitary disorders
6. Discuss the management of a patient with thyroid disorders

Discuss the management of a patient with adrenal disorders

3.3 Applied Anatomy And Physiology of the Endocrine System

In this subunit we briefly revise the structures and function of the endocrine system. From your anatomy and physiology lessons in year 1, you agree with me that the endocrine system plays a major role in regulating body functions and as such, any abnormality in this system results in malfunctioning of our bodies.12 below

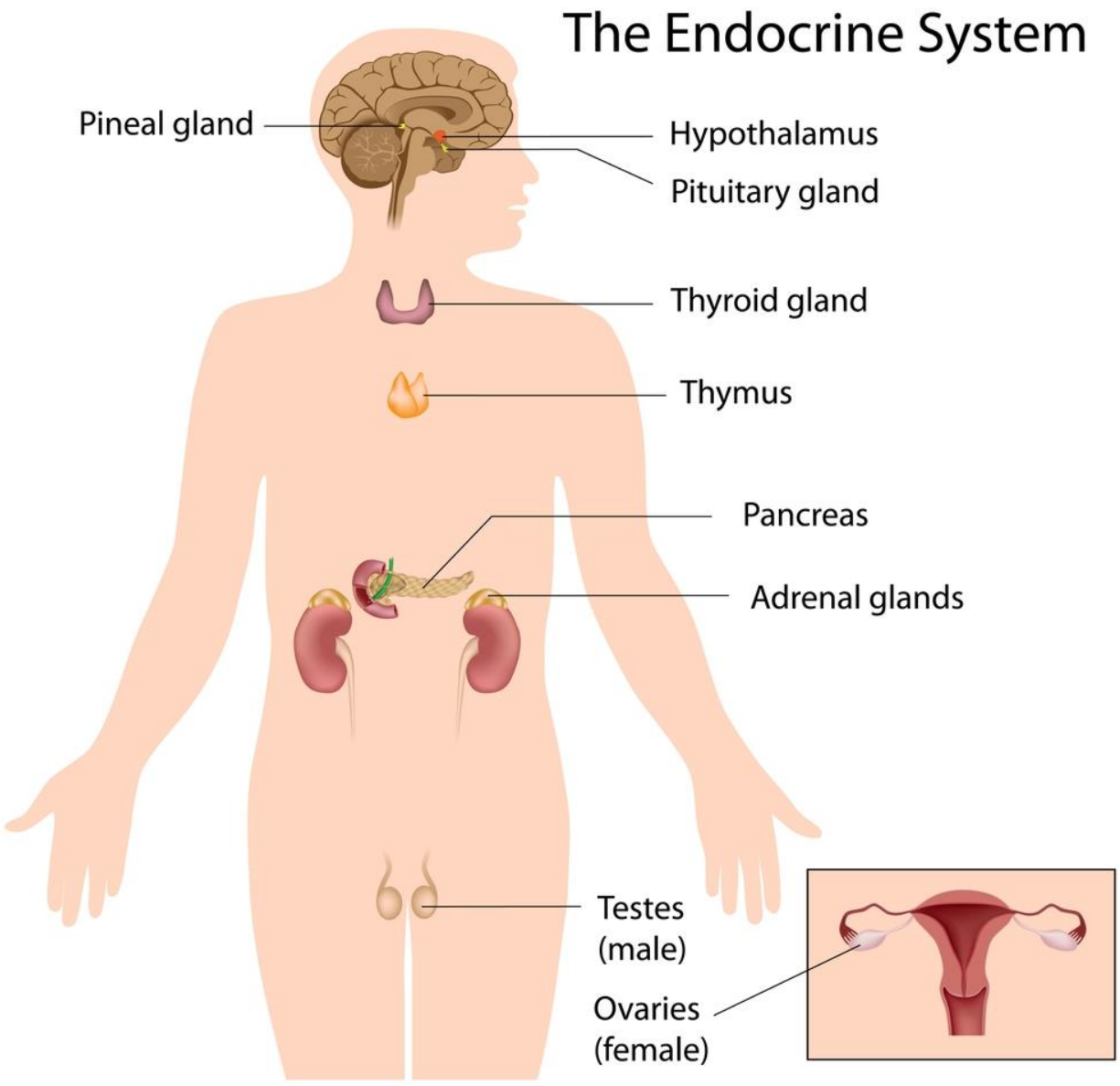

Figure 11: Showing Endocrine glands

You just reviewed the anatomy and physiology of the endocrine system; we will now proceed to look at the role of the nurse during investigation and procedures

3.3 Role of the Nurse in investigations and procedures

As a nurse you will be required to carry out certain investigations and procedures and on the other hand you may assist the Physician in carrying out these investigations and procedure. Therefore it is imperative that you are equipped with knowledge on your roles during these investigative procedures.

Blood tests*Glucose tolerance test*: this is a test that determines one's ability to clear excess sugar from the blood stream. The investigation is used in diagnosing diabetes mellitus in patients who present with diabetes mellitus for the first time.As a nurse your role is to prepare the glucose tablets that will be administered to the patient and glucometer which is used to monitor serum glucose levels. You are also required to collect samples of blood at 2 hourly intervals and test for the presence of glucose. The normal serum glucose level is 140mg/dl or 7.8mmol/l. A reading above 200mg/dl or 11.1mmol/l indicates a provisional diagnosis.

Caution: never give glucose sweets to known diabetic patients

Hormonal function test- hormonal profile test are carried out to detect any abnormalities. Of concern are the pituitary, thyroid and parathyroid hormones.

Your role in these investigations and procedures is to prepare the patient psychologically and collect blood were necessary.

- **Urine tests-** mostly this is urinalysis. Collect a urine specimen and test the urine using uristix, interpret the results and record.
- Radiological examinations:-

Porto-splenography

Portal splenography demonstrates the site and often the cause of portal venous obstruction and is performed prior to surgical intervention. The nurse needs to prepare the patient and reassure the patient that it is a diagnostic test.

Assignment

During your clinical placement on a medical ward, follow up a patient with diabetes mellitus and check the type of investigations he/she underwent to come up with the diagnosis.

3.4 Management Of A Patient With Metabolic Disorders

Metabolic disorders vary in our setting today and account for many loss of lives and complications that cause irreparable damage to patient/client. In our discussion we will concentrate on two metabolic disorders commonly encountered. These are diabetes mellitus and diabetes insipidus.

DIABETES MELLITUS

What is diabetes Mellitus? I am sure you have heard people saying they have sugar disease. That is a layman's language of saying diabetes mellitus.

DEFINITION: Diabetes Mellitus is not a single disease entity, but rather a group of metabolic disorders sharing the common underlying feature of hyperglycaemia. Hyperglycaemia in diabetes results from defects in insulin secretion, insulin action or most commonly, both.

Classification

Although all forms of diabetes mellitus share hyperglycaemia as a common feature, the pathogenic process involved in the development of hyperglycaemia vary widely. The previous classification schemes of diabetes mellitus were based on the age at onset of the disease or on the mode of therapy; in contrast, the recently revised classification reflects our greater understanding of the pathogenesis of each variant. The vast majority of cases of diabetes fall into one of two broad classes:

1. *Type 1 Diabetes Mellitus:* this is characterized by an absolute deficiency of insulin caused by pancreatic β-cell destruction. It accounts for approximately 10% of all cases.
2. *Type 2 Diabetes Mellitus:* is caused by a combination of peripheral resistance to insulin action and an inadequate secretory response by the pancreatic β-cells ("relative insulin deficiency"). Approximately 80% to 90% of patients have type 2 diabetes.

Aetiology of Diabetes Mellitus

The exact cause of DM is unknown. However, the aetiology of each type is still unfolding. At least 4 sets of factors influence the development of diabetes and these are:-

- Genetic factors
- Metabolic factors
- Microbiological
- Immunological factors

1. Genetic Factors

Patients with a family history of diabetes are at far greater risk of developing diabetes especially type 2. Diabetes runs in the family even though research has not yet pin pointed the responsible gene.

2. Metabolic Factors

Metabolic factors involved are many and complex in the aetiology of the disease. Emotional or physical stress can unmask an inherited predisposition to DM, probably as a result of gluconeogenesis induced by increased production of hormones from the adrenal cortex especially the glucocorticoids. The majority of the diabetic patients who develop the disease after the age of 10 years are obese. This is due to decreased number of insulin receptors on the tissues, which increase the level of serum insulin.

3. Microbiological Factors

Infectious agents like viruses predispose individuals to diabetes mellitus, especially type 1, resulting from dysfunction of beta cells e.g. Coxsackie virus. Supportive evidence of viruses as causative factors includes the following: -

- Both type 1 DM and viral infections tend to have sudden onset

Seasonal fluctuations in the onset of type 1, late autumn and early spring correspond with the times of the year when flu and other viral illness are most common.

Viral infections can and often attack the pancreas; many viral infections are characterized by the inflammation of pancreatic beta cells.

4. Immunological Factors

In type 1 DM there is evidence of an auto-immune response. This is an abnormal response in which antibodies are directed against normal tissue of the body responding to these tissues as if they are foreign. Auto antibodies against islet cells and against endogenous (internal) insulin have been detected in people at the time of diagnosis and even several years prior to the development of clinical signs of type 1 diabetes. Such antibodies are not found in normal individuals. Diabetics that continue to produce insulin will eventually stop producing normal amounts of the hormone and islet cells antibodies remain in the blood.

Table 1: Showing Differences between DM type1 and type 2

Comparative Clinical Features Of Type 1 And Type 2 Diabetes		
	TYPE 1	**TYPE 2**
Typical age at onset	<40 years	>50 years

Duration of symptoms	weeks	Months to years
Body weight	Normal or low	Obese
Ketonuria	Yes	No
Rapid death without treatment with insulin	Yes	No
Autoantibodies	Yes	No
Diabetes complications At diagnosis	No	25%
Family history of diabetes	uncommon	common
Other autoimmune disease	Common	Uncommon

Normal Physiology

Insulin is secreted by beta cells, which are one of the four types of cells of the Islets of langerhans in the pancrease. Insulin is an anabolic or storage, hormone. When a meal is eaten, insulin secretion increases and moves glucose from the blood into muscle, liver and fat cells. In those cells insulin has the following effects:

- Transports and metabolizes glucose for energy.
- Stimulates storage of glucose in the liver and muscle (in the form of glycogen)
- Enhances storage of dietary fat in adipose tissue.
- Accelerates transport of amino acids (derived from dietary protein) into cells.

Insulin also inhibits the breakdown of stored glucose, protein and fat. During fasting periods (between meals and overnight), the pancrease continuously releases a small amount of insulin; another pancreatic hormone called glucagon (which is secreted by the alpha cells of the Islets of Langerhans) is released when blood glucose levels decrease. The insulin and the glucagon together maintain a constant level of glucose in the blood by stimulating the release of glucose from the liver.

Initially, the liver produces glucose through the breakdown of glycogen (glycogenolysis). After 8 to 12 hrs without food, the liver form glucose from the breakdown of non-carbohydrate substances, including amino acids (gluconeogenesis).

Type 1 Diabetes Mellitus

Type 1 is characterized by destruction of the pancreatic beta-cells. It is thought that a combination of genetic, immunologic and possibly environmental (e.g.viral) factors contributes to beta cell destruction.

Regardless of the specific etiology, the destruction of the beta cells results in unchecked glucose production by the liver and fasting hyperglycemia. In addition, glucose derived from food cannot be stored in the liver but instead remains in the blood stream and contributes to postprandial (after meal) hyperglycemia. If the concentration of glucose in the blood exceeds the renal threshold for glucose, usually 180 to 200mg/dl the kidneys may not reabsorb all of the filtered glucose; the glucose then appears in the urine (glycosuria). When excess glucose is excreted in the urine, it is accompanied by excessive loss of fluids and electrolytes. This is called osmotic diuresis.

Because insulin normally inhibits glycogenolysis (breakdown of stored glucose) and gluconeogenesis (production of new glucose from amino acids and other substrates) in people with insulin deficiency, these processes occur unrestrained and contribute further to hyperglycemia. In addition, fat breakdown occurs, resulting in an increased production of ketone bodies, which are the by-products of fat breakdown.

Type 2 Diabetes Mellitus

The two main problems related to insulin in type 2 diabetes are insulin resistance and impaired insulin secretion. Insulin resistance refers to a decreased sensitivity of the tissues to insulin. Normally, insulin binds to special receptors on cell surfaces and initiates a series of reaction involved in glucose metabolism. Intracellular reactions are diminished, thus rendering insulin less effective at stimulating glucose uptake by the tissues. The exact mechanisms that lead to insulin resistance and impaired insulin secretion in type 2 diabetes are unknown, although genetic factors are thought to play a role.

Despite the impaired insulin secretion that is characteristic of type 2 diabetes, there is enough insulin present to prevent the breakdown of fat and the accompanying production of ketone bodies. Therefore DKA (diabetic ketoacidosis) does not occur in type 2 diabetes.

Clinical Manifestations

The cardinal signs and symptoms of diabetes mellitus: -

- Hyperglycaemia as a result of failure by the liver and skeletal muscles to use glucose
- Polyuria (increased urination) – water not reabsorbed from the renal tubules secondary to osmotic activity of glucose, leads to loss of water, glucose, and electrolytes.
- Polydipsia (excessive thirst) - this occurs secondary to polyuria.
- Polyphagia – (excessive hanger) starvation secondary to tissue breakdown (catabolism) causes hunger.
- Weight loss – initial loss secondary to depletion of water, glycogen, and triglyceride stores; chronic loss secondary to decreased muscle mass as amino acids are diverted to form glucose and ketone bodies.
- Recurrent blurred vision – secondary to chronic exposure of ocular lens and retina to hyperosmolar fluids.
- Pruritus, skin infections, vaginitis – bacterial and fungal infections of skin seem to be more common.
- Ketonuria – when glucose cannot be used for energy in type 1 DM, fatty acids are used for energy, and broken down into ketones in blood and excreted by kidneys.
- Weakness, fatigue and dizziness – decreased plasma volume leads to postural hypotension; potassium loss and protein catabolism contribute to weakness.

Other symptoms include:

- Tingling or numbness in hands or feet due neuropathy
- Dry skin
- Skin lesion or wounds that are slow to heal

Management

Aim

The aim of management is to normalize insulin activity and blood glucose levels to reduce the development of vascular and neuropathic complications.

Medical management

Diagnosis

- History of diabetes in the family and clinical features

• Random blood sugar – this is taken any time of the day regardless of whether any meal was taken or not. Readings above 2000mg/dl with presence of any classic symptom is indicative of diabetes

• Fasting blood sugar – (normal range 70 – 100mg/dl) alteration may indicate hyperglycaemia or hypoglycaemia.

• Postprandial blood glucose – (normal readings are less than 120mg /dl) blood sample is taken 2 hours after meals.

• Glucose tolerance test- evaluates insulin response to glucose load. The blood sample is taken before ingestion of 50-200g of glucose and samples taken half hourly, 1 hourly, 2 hourly and 3 hourly. The diagnosis of diabetes is made if there is a two hours value of 200mg/dl or greater.

• Glycosylated haemoglobin- normally glucose attaches itself to the haemoglobin molecule on the red blood cell. Once attached it cannot disassociate, therefore the higher the blood glucose levels have been the higher are the glycosylated haemoglobin results. Glycosylated haemoglobin can be sampled anytime during the day.

• Urine ketone levels- urine levels of ketones (acetone, aceto-acetic acid, and beta-hydroxybutyric acid) can be tested by dip strips or tablets. The presence of ketones indicates that the body is using fat as a source of energy.

Treatment

Objectives

1. To keep the sugar levels within normal range as much as possible.

2. To alleviate symptoms and minimize the risk of long term complications by appropriate control.

There are five components of diabetes management namely;

- - Nutrition management
- - Exercise
- - Blood glucose monitoring
- - Education
- - Pharmacological therapy

Nutrition Management (Food Guide Pyramid)

Food guide pyramid is a tool used to develop meal plans, which places the basic four food groups. It is commonly used for patients with type 2 diabetes who have difficult time complying with a calorie-controlled diet. The food pyramid consists of six food groups.

1. Bread, cereal, rice and pasta
2. Fruits
3. Vegetables
4. Meat, poultry, fish, dry beans, eggs and nuts
5. Milk, yogurt and cheese
6. Fats, oils, and sweets

The pyramid shape was chosen to emphasize that the foods in the largest area, the base of the pyramid (starches, fruits and vegetables) are lowest in calories and fat; and highest in fiber and should make up the basis of the diet. For those with diabetes as well as for the general population, 50% to 60% of daily caloric intake should be from these three groups.

Glycaemic Index

One of the main goals of diet therapy in diabetes is to avoid sharp rapid increase in blood glucose levels after food is eaten. The term glycaemic index is used to describe how much a given food raises the blood glucose level compared with an equivalent amount of glucose.

Alcohol Consumption

The ingestion of alcohol by diabetic patients need not be completely restricted. It is important, however for patients and health care professionals to be aware of the potential adverse effects of alcohol specific to diabetes. The main danger with the use of alcohol by a diabetic patient is hypoglycaemia, especially those taking insulin.

Exercises

Exercise is extremely important in the management of DM, because of its effects on lowering blood glucose and reducing cardiovascular risk factors. Exercise lower blood glucose by increasing the uptake of glucose by body muscles and by improving insulin utilization. It also improves circulation and muscle tone.

However, patient with blood glucose levels of more than 250mg/dl (14mmol)dl) who have ketones in their urine should not begin exercising until the urine ketone test is negative and the blood glucose level is closer to normal. Exercising with elevated blood glucose levels causes increased secretions of glucagon, growth hormone and catecholamine's. The liver then releases more glucose, resulting in an increase in blood glucose.

Blood Glucose Monitoring

Blood glucose monitoring is a corner stone of diabetes management. Frequent self monitoring of blood glucose enables people with diabetes to adjust the treatment regimen to obtain optimal blood sugar control. This allows for detection and prevention of hypoglycaemia and hyperglycaemia. Close monitoring of glucose levels is therefore very important in the control of diabetes mellitus. Finger – stick monitoring and urine testing may be useful in self monitoring of glucose levels. The tests should be done regularly.

Education

Components of education include teaching patient on diet, exercises, self monitoring, medications and modification of life style.

Pharmacologic Therapy

Insulin therapy: frequently insulin injections are taken two times per day (even more often) to control blood glucose. Because the insulin dose required by the individual patient is determined by the level of glucose in the blood, accurate monitoring of blood glucose levels is essential.

A number of insulin preparations are available. They vary according to three main characteristics;

- Time course of action
- Species source
- Manufacturer

Time: insulin may be grouped into several categories based on the onset, peak and duration of action.

Table 2: Categories of Insulin

Time course	Agent	Onset	Peak	Duration	Indication
Rapid-acting	Humalog	10-15min	1hr	3hrs	Used for rapid reduction of glucose levels to treat post prandial hyperglycaemia and to prevent nocturnal hypoglycaemia
Short-acting (Crystalline Zinc Insulin) (CZI)	Regular ("R")	½-1 hour	2-3 hours	4-6 hours	Usually administered 20-30 minutes before meal, may be taken alone or in combination with longer-acting insulin
Intermediate-acting (Lente Insulin)	NPH (Neutral Protamine Hagedorn lente "L")	3-4 hours	4-12 hours	16-20 hours	Usually taken after food
Long-acting	Ultralente ("UL")	6-8 hours	12-16 hours	20-30 hours	Used primarily to control fasting glucose level

Complications Of Insulin Therapy

- Local allergic reaction (redness, swelling, tenderness and indurations) Physician may prescribe an antihistamine to be taken 1 hour before the injection if such a local reaction

occurs. Use of alcohol to cleanse the skin is no longer recommended. (use boiled/distilled water)

- Systemic allergic reaction (generalized urticaria). Desensitization with small doses of insulin administered in gradually increasing amounts.
- Insulin lipodystrophy (localized reaction in the form of either lipoatrophy or lipohypertrophy, occurring at the site of insulin injections. Lipoatrophy is loss of subcutaneous fat and appears as slight dimpling or more serious pitting of subcutaneous fat. Lipohypertrophy, development of fibrofatty mass at the injection site, is caused by repeated use of an injection site.
- Insulin resistance, because of obesity

1. Morning hyperglycemia:
2. An elevated blood glucose level on arising in the morning may be caused by an insufficient level of insulin due to several causes, such as;

– Insulin waning- progressive rise in blood glucose from bedtime to morning.
– Dawn phenomenon: relatively normal blood glucose until about 3AM, when the level begins to rise.

Somogyi effect: normal or elevated blood glucose at bedtime, caused by the production of counterregulatory hormones (growth hormone).

Dosages: 5-20 units bid, subcutenous injection.

Diabetic ketoacidosis (use regular insulin only): 0.33units/kg as an IV bolus followed by 0.1units/kg/hour by continous infusion until blood glucose level drops to 250mg/dl.

Oral Antidiabetic Agents

Oral antidiabetic agents may be effective for type 2 diabetic patients who cannot be treated by diet and exercise alone.

Sulfonylureas: Exert their primary action by directly stimulating the pancreas to secrete insulin. Therefore, a functioning pancreas is necessary for these agents to be effective and they cannot be used in patients with type 1.

It also improves insulin action at cellular level. Examples are:

- Diabinese (Chlorpropamide) 250-1500mg in divided doses.
- Tolbutamide (Orinase) 500-2000mg divided doses

1. Biguanides: Metformin (Glucophage) produce its antidiabetic effects by facilitating insulin's action on peripheral receptor sites. Therefore, it can be used only in the presence of insulin.

Biguanides used in combination with sulfonylurea agents may enhance the glucose lowering effect more than either medication used alone. Dosage is 1500mg in divided dosage.

2. Alpha Glucoside inhibitors: Acarbose (Precose) is a member of this drug category used in managing type 2. AGI work by delaying the absorption of glucose in the intestinal system, resulting in a lower postprandial blood glucose level.

Nursing Care

To nurse a patient with diabetes mellitus we will use a case study on a patient who has been newly diagnosed with diabetes mellitus.

Think about the case and identify four (4) problems whether actual or potential problems that you ctions and Son

Case Study

Mrs. Bwali, a 52-year-old married woman with no history of diabetes mellitus is admitted to the ward where you are working with complaints of poor vision, excessive thirst, frequent passage of urine, loss of sensation in the feet, and she has an ulcer that has taken long to heal. The patient states that she has an inconsistent sleep-activity schedule and does not exercise regularly. When you conduct physical examination, you find that her feet and hands are warm and dry, with colour consistent with the rest of the body, decreased perception to touch in lower extremities, and decreased vibration and pin prick sensation to great toes bilaterally. She weighs 40kg. Her blood pressure is 140/90mmHg, Heart rate 92 beats/minute, and respirations 24 breaths/minute. Her urine is negative for microalbinuria. Describe how Mrs. Bwali will be nursed using a nursing care plan.

We

NURSING CARE PLAN

PROBLEM	NURSING DIAGNOSIS	OBJECTIVES / GOALS	INTERVENTIONS	EXPECTED OUTCO
Fluid volume deficit	Fluid volume deficit related to polyuria secondary to hyperglycaemia evidenced by excessive thirst, dry skin and poor skin turgor.	Patient will maintain normal fluid balance	- Monitor blood glucose levels. - Assess and monitor for fluid volume deficit (assess the skin turgor, dry skin and other signs of dehydration). - Administer fluids as prescribed up to 2 to 3 litres per day to replace lost fluid and prevent hypovolaemia. - Monitor intake and output using a fluid balance chart to prevent fluid overload. - Assess for signs of hypovolemic shock and electrolyte deficiency e.g. sunken eyes, cold, clammy skin, muscle cramps.	Patient demons adequate hydr evidenced by stable signs, appropriate u out put, good skin tur
Altered nutrition	Altered nutrition less than body requirements, related to insulin deficiency, hyper-metabolic state, evidenced by weight loss, weakness and increased ketones	Patient will have improved nutrition and achieve weight gain.	- Weigh patient daily or as indicated to assess adequacy of nutritional intake - Monitor blood glucose levels - Ascertain patient's dietary programme and usual patterns to identify deficits and deviations from therapeutic needs. - Together with nutritionist	Patient will achieve weight gain and select appropriate amount of calories and nutrients.

			help patient to adjust to required dietary regulations. - Teach patient on foods that increase blood glucose levels e.g. carbohydrates - Identify food preferences including ethnic and cultural foods to be incorporated into the meal plan to enhance cooperation after discharge.	
Fatigue	Fatigue related to decreased metabolic energy production, altered body chemistry and increased energy demands manifested by activity intolerance, decreased performance and inability to concentrate.	Patient will tolerate activity and experience an increase in energy levels	- Discuss with patient the need for activity and identify activities with patient that lead to fatigue. - Alternate activity with periods of rest to prevent excessive fatigue - Monitor pulse, respiratory rate and blood pressure before and after activity to indicate physiological levels of tolerance. - Encourage patient's participation in activities of daily living as tolerated so as to increase confidence and self esteem.	Patient verbalizes increase in energy level and displays improved ability participate in d activities
Knowledge deficit	Knowledge deficit of self administration of insulin, care of	Patient will verbalize disease	- Work with patient in setting mutual goals for learning to	Patient verbalizes demonstrates ac knowledge of proper

	equipment and home monitoring of blood glucose related to lack of previous exposure to information and skill as manifested by questions/request for information and verbalization of the problem.	process, need for blood glucose monitoring and accurately demonstrate self-administration of insulin	promote enthusiasm and cooperation. - Demonstrate finger stick testing, insulin administration and urine testing and have patient do return demonstration. - Stress importance of adhering to recommended diet to prevent hyperglycemia and hypoglycemia. - Review self administration of insulin and care of equipment. - Have patient demonstrate procedure to verify understanding and correctness of procedure.	insulin administratio and the prescribed dietary regimen.

Now that we have discussed our nursing care plan, go ahead and practice writing another care plan for the following problems:

1. Anxiety
2. Risk for infection

(Remember when writing a potential or risk nursing diagnosis, there is no evidence, so you don't write "evidenced by or manifested by")

Other specific nursing care includes:

Health Education On Discharge

Teaching should start as early as possible during admission. Arrange for teaching lessons and demonstrations.

Teach management, including the significance of insulin or oral hypoglycemic for disease control. Demonstrate injection techniques and observe patient performance.

Initiate documentation of rotating sites for subcutaneous injections including the abdomen.

Link medication needs to other factors, such as diet and exercise. Ensure that the family is aware of signs and treatment of hypoglycemia as well as the protocol for managing persistent hyperglycemia. Involve family members in all teaching.

Coordinate involvement of the patient/family and dietician in planning a therapeutic diet for disease control. Reinforce nutritional guidelines.

Encourage supervised weight loss if the patient is over-weight.

Teach blood and urine glucose testing methods for home use. Observe patient demonstrations for accuracy of testing, interpretation of results and documentation. Encourage the patient to keep a daily record of glucose monitoring.

Emphasize the importance of regular activity and exercise and of maintaining an approximately equivalent level of activity from day to day.

Tell the patient to be aware of increased susceptibility to infections, and discuss ways to avoid exposure. Emphasize the importance of prompt, appropriate treatment of even minor injuries to avoid serious complications.

- Teach foot care, skin care, leg exercises and assessment of circulatory status. If patient smokes, emphasize the importance of quitting. Ensure that the patient and family receive a written foot care protocol.
- Discuss the eye disorders associated with DM. Help the patient understand the significance of careful disease control in preventing or slowing development of diabetic retinopathy. Emphasize the importance of early reporting of vision changes.
- Explain peripheral symptoms such as paresthesia, pain and sensory loss. Encourage patient to be carrying with her/him a card for identification and sweets.

Complications

1. Hyperglycemic coma (diabetic coma)
2. Hypoglycemic coma
3. Retinopathy, cataracts, blindness.
4. Peripheral neuropathy-paresthesia, numbness.
5. Prone to infections-Moniliasis

6. Wounds don’t heal early (takes long to heal)
7. Arteriosclerosis- gangrene (amputation of lower limbs)
8. Nephropathies.

Acute Complications Of Diabetes Mellitus

A. Hypoglycemia (Insulin Reaction)

Definition: This is abnormally low blood sugar levels; occurs when the blood glucose falls to less than 50 to 60 mg/dl (2.7 to 3.3mmol/L). Hypoglycemia can occur at any time of day or night.

Causes:

- Too much insulin or oral hypoglycemic agents
- Too little food
- Excessive physical activity

It often occurs before meals, especially if meals are delayed or snacks are omitted. For example, midmorning hypoglycemia may occur when the morning regular insulin is peaking.

Clinical Manifestation

- As the blood glucose level falls, the sympathetic nervous system is stimulated, resulting in a surge of adrenalin. This causes symptoms such as sweating, tremors, tachycardia, palpitation, nervousness and hunger.
- In moderate hypoglycaemia, the fall in blood glucose level deprives the brain cells of needed fuel for functioning. Inability to concentrate, headache, light-headedness, confusion, memory lapses, numbness of the lips and tongue, slurred speech, impaired coordination, emotional changes, irrational or combative behavior, double vision and drowsiness. In severe hypoglycemia; disoriented behavior, seizures, difficulty arousing from sleep or loss of consciousness.

Management

Immediate treatment must be given when hypoglycaemia occurs.

10 to 15gm of a fast acting simple carbohydrate such as:

- Three or four commercially prepared glucose tablets.
- 2 to 3 teaspoons of sugar or honey

For patients who are unconscious and cannot swallow; an injection of glucagon 1mg can be administered either subcutaneously or intramuscularly. After an injection of glucagon, it may take up to 20 minutes for the patient to regain consciousness.

A simple sugar followed by a snack should be given to the patient on awakening to prevent recurrence of hypoglycaemia and to replenish liver stores of glucose.

In an emergency department, patient who is unconscious or cannot swallow may be treated with 25 to 50ml of 50% dextrose (D50) administered intravenously.

B: Diabetic Ketoacidosis

DKA is a complication of type I diabetes mellitus as a result of an absence or markedly inadequate amount of insulin resulting in disorders in the metabolism of carbohydrates, fats and proteins. It is characterized by three main features; Hyperglycaemia, dehydration and electrolyte loss, acidosis.

Causes Of Dka

There are three main causes of DKA;

- Decreased or missed dose of insulin
- Illness or infection
- Undiagnosed and untreated diabetes

A decrease in insulin may result from an insufficient dosage of insulin prescribed or from insufficient insulin being administered by the patient.

Errors in insulin dosage by patients who are ill, who assume that if they are eating less or if they are vomiting, they must decrease their insulin doses. Other causes of decreased insulin include:

- Error in drawing up or injecting insulin (especially in patients with visual impairment).
- Intentionally skipping of insulin doses
- Equipment problems (occlusion of insulin pump tubing)

Pathophysiology

When insulin is lacking, the amount of glucose entering the cells is reduced and glucose production by the liver increases. Both factors lead to hyperglycaemia. In an attempt to rid the body of the excess glucose, the kidneys excrete the glucose along with water and electrolytes (Sodium and potassium). This osmotic diuresis, which is characterized by excessive urination (polyuria), leads to dehydration and marked electrolyte loss. Patients with severe DKA may lose

an average of 6.5litres of water and up to 400 to 500MEq each of sodium, potassium and chloride over a 24 hour period.

Another effect of insulin deficiency is the breakdown of fat (lipolysis) into free fatty acids and glycerol. The free fatty acids are converted into ketone bodies by the liver. In DKA there is excessive production of ketones bodies because of the lack of insulin that would normally prevent this from occurring. Ketone bodies are acids; when they accumulate in the circulation, this leads to metabolic acidosis.

Clinical Manifestations

1. Hyperglycaemia of DKA leads to polyuria and polydipsia.
2. In addition patient may experience blurred vision, weakness and headache.
3. Drop in systolic blood pressure on standing due to intravascular volume depletion.
4. Ketosis and acidosis lead to gastrointestinal symptoms such as anorexia, nausea, vomiting and abdominal pain.
5. Acetone breath (a fruity odour) which occurs with elevated ketone levels.
6. Hyperventilation (very deep, but not laboured respirations) called kussmaul respirations; representing the body's attempt to decrease the acidosis, counteracting the effect of the ketone builds up.
7. Mental changes such as, lethargic or comatose.

Management

Investigations:

- Rule out other causes of comatose if patient is in coma and if you're not sure that the patient is diabetic.
- Blood glucose levels may vary from 300 to 800mg/dl (16.6 to 44.4mmol/dl)
- Evidence of ketoacidosis reflected in low serum bicarbonate (0 to 15 MEq/L) and low PH (6.8 to 7.8) values.
- Accumulation of ketone bodies (which precipitates acidosis) is reflected in blood and urine ketone measurements.

In addition to treatment of hyperglycemia, management of DKA is aimed at correcting dehydration, electrolyte loss and acidosis.

Dehydration: Rehydration is important for maintaining tissue perfusion. Fluid replacement enhances the excretion of excessive glucose by the kidneys. Patient may

need up to 6 to 10 liters of IV fluids to replace fluid losses caused by polyuria, hyperventilation, and diarrhoea and vomiting.

Initially 0.9% normal saline is administered at a high rate, usually 0.5 to 1 liter per hour for 2 to 3 hours. Hypotonic normal saline (0.45%) may be used for patients with hypertension or hypernatremia or those at risk for congestive heart failure.

After the first few hours 0.45% normal saline is fluid of choice for continued rehydration, if the blood pressure is stable and the sodium level is not low. When the blood glucose level reaches 300mg/dl or less, the IV fluid may be changed to D5W to prevent a precipitous decline in the glucose level.

Electrolyte Loss:

The major electrolyte of concern during treatment of DKA is potassium cautions but timely K^+ replacement is vital to avoid dysrhythmias that may occur with hypokalemia. 40meq per hour is needed for several hours. Potassium must be infused even if the plasma K^+ level is normal.

Acidosis:

Ketone bodies (acids) accumulate as a result of fat breakdown. The acidosis that occurs in DKA is reversed with insulin, which inhibits fat breakdown, thereby stopping acid build-up.

Insulin is usually infused intravenously at a slow, continuous rate (5 units per hr). Hourly blood glucose values must be measured. Dextrose is added to intravenous fluids, such as normal saline (D5N/S or D50.45N/S) when blood glucose levels reach 250 to 300mg/dl to avoid too rapid a drop in the blood glucose level.

Insulin is often infused separately from the rehydration solution to allow frequent changes in the rate and content of rehydration solution. Insulin must be infused continuously until subcutaneous administration of insulin resumes. Any interruption in administration may result in the re-accumulation of ketone bodies and worsening acidosis.

Blood glucose levels are corrected before the acidosis is corrected. Thus, intravenous insulin may be continued for 12 to 24 hours, until the serum bicarbonate level improves (to at least 15 to 18meq/l) and until the patient can eat.

Nursing Management

Nursing care of the patient with DKA focuses on monitoring fluid and electrolyte status as well as blood glucose levels, administering fluids, insulin and other medications and preventing other complications such as fluid overload.

Urine output is monitored to ensure adequate renal function before K^+ is administered to avoid hyperkalaemia.

The patient's ECG is monitored for dysrhythmias indicating abnormal K^+.

Vital signs, arterial blood gases and other clinical findings are recorded on a flow sheet. The nurse documents the lab values and the frequent changes in fluids and medications that are prescribed and monitors the patient's responses.

Monitoring fluid volume status involves frequent measurements of vital signs, lung assessment, and monitoring venous fluid intake as dehydration is corrected.

Monitoring for signs of fluid overload is especially important for older patients, those with renal impairment or that risk for Congestive Heart Failure. As DKA resolves, the potassium replacement rate is decreased, for safety, the nurse makes sure that:

- There is no sign of hyperkalaemia on the electrocardiogram.
- Lab results of potassium are normal or low.
- The patient is urinating (no renal shutdown)

As the patient recovers, the nurse reassesses the factors that may led to DKA and teaches the patient and family strategies to prevent its recurrence.

DIABETES INSIPIDUS

Definition: Diabetes insipidus is a disorder of the posterior lobe of the pituitary gland characterized by a deficiency of antidiuretic hormone (ADH), or vasopressin. Great thirst (polydipsia) and large volumes of dilute urine characterize the disorder. It may be secondary to head trauma, brain tumor, or surgical ablation or irradiation of the pituitary gland. It may also occur with infections of the central nervous system (meningitis,

encephalitis, tuberculosis) or tumors (eg, metastatic disease, lymphoma of the breast or lung). Another cause of diabetes insipidus is failure of the renal tubules to respond to ADH; this nephrogenic form may be related to hypokalemia, hyperkalaemia, and a variety of medications (eg, lithium, demeclocycline [Declomycin]).

Clinical Manifestations

- Without the action of ADH on the distal nephron of the kidney, an enormous daily output of very dilute, water-like urine with a specific gravity of 1.001 to 1.005 occurs. The urine contains no abnormal substances such as glucose and albumin. Because of the intense thirst, the patient tends to drink 2 to 20 liters of fluid daily and craves cold water. In the hereditary form of diabetes insipidus, the primary symptoms may begin at birth. In adults, the onset of diabetes insipidus may be abrupt or insidious. The disease cannot be controlled by limiting fluid intake because the high-volume loss of urine continues even without fluid replacement. Attempts to restrict fluids cause the patient to experience an insatiable craving for fluid and to develop hypernatremia and severe dehydration.

Assessment and Diagnostic Findings

- The fluid deprivation test is carried out by withholding fluids for 8 to 12 hours or until 3% to 5% of the body weight is lost. The patient is weighed frequently during the test. Plasma and urine osmolality studies are performed at the beginning and end of the test. The inability to increase the specific gravity and osmolality of the urine is characteristic of diabetes insipidus. The patient continues to excrete large volumes of urine with low specific gravity and experiences weight loss, rising serum osmolality, and elevated serum sodium levels. The patient's condition needs to be monitored frequently during the test, and the test is terminated if tachycardia, excessive weight loss, or hypotension develops. Other diagnostic procedures include concurrent measurements of plasma levels of ADH (vasopressin) and plasma and urine osmolality, a trial of desmopressin (synthetic vasopressin) therapy and intravenous infusion of hypertonic saline solution.
- When the diagnosis is confirmed and the cause is not obvious (eg, head injury), the patient is carefully assessed for tumors that may be causing the disorder.

Medical Management

- The objectives of therapy are (1) to replace ADH (which is usually a long-term therapeutic program), (2) to ensure adequate fluid replacement, and (3) to identify and correct the underlying intracranial pathology. Nephrogenic causes require different management approaches.

Pharmacologic Therapy

Desmopressin (DDAVP), a synthetic vasopressin without the vascular effects of natural ADH, is particularly valuable because it has a longer duration of action and fewer adverse effects than other preparations previously used to treat the disease. It is administered intranasally; the patient sprays the solution into the nose through a flexible calibrated plastic tube. One or two administrations daily or every 12 to 24 hours usually control the symptoms (Tierney, McPhee, & Papadakis, 2001). Another form of therapy is the intramuscular administration of ADH, or vasopressin tannate in oil, which is used when the intranasal route is not possible. It is administered every 24 to 96 hours. The vial of medication should be warmed or shaken vigorously before administration. The injection is administered in the evening so that maximum results are obtained during sleep. Abdominal cramps are a side effect of this medication. Rotation of injection sites is necessary to prevent lipodystrophy. Clofibrate, a hypolipidemic agent, has been found to have an antidiuretic effect on patients with diabetes insipidus who have some residual hypothalamic vasopressin. Chlorpropamide (Diabinese) and thiazide diuretics are also used in mild forms of the disease because they potentiate the action of vasopressin. The patient receiving chlorpropamide should be warned of the possibility of hypoglycemic reactions. If the diabetes insipidus is renal in origin, the previously described treatments are ineffective. Thiazide diuretics, mild salt depletion, and prostaglandin inhibitors (ibuprofen, indomethacin, and aspirin) are used to treat the nephrogenic form of diabetes insipidus.

Nursing Management

- The patient with possible diabetes insipidus needs encouragement and support while undergoing studies for a possible cranial lesion. The nurse needs to inform the patient and family about follow-up care and emergency measures. The nurse also needs to provide specific verbal and written instructions, show the patient how to administer the

medications, and observe return demonstrations as appropriate. The nurse also advises the patient to wear a medical identification bracelet and to carry medication and information about this disorder at all times. Vasopressin must be administered with caution if the patient has coronary artery disease because the medication causes vasoconstriction.

You have come to the end of discussing metabolic disorders in which we looked at diabetes mellitus and diabetes insipidus. Read further to understand the two conditions and how to manage them. In our next topic we will be dealing with pancreatic disorders.

SELF TEST

1. The normal value of glucose tolerance test is
    a. 12.1mmol/l
    b. 11.5mmol/l
    c. 11.1mmol/l
    d. 10.9mmol/l
2. Diabetes is a
    a. Metabolic disorder of all nutrients
    b. Syndrome
    c. Pituitary disorder
    d. Malabsorption disorder
3. The two major hormones released by the thyroid gland are
    a. T5 and T6
    b. T3 and T5
    c. T3 and T4
    d. T4 and T5

Answers Q1 .C Q2 A. Q3 C

3.5 Management f Patient With Pancreatic Disorders

We hope you remembered that the pancreas is located in the abdominal cavity behind the stomach. The pancreas is responsible for the production of enzymes that aid in digestion and it also produces hormones that regulate normal body from its specialized cell namely, alpha, beta and gammas. As a nurse you should know about these condition/disorders so that you can effectively manage these patients. Among the many conditions of the pancreas, we shall only discuss two; Pancreatitis and pancreatic tumours.

PANCREATITIS

Definition: this is the inflammation of the of the pancreas which is either acute or chronic inflammation (Loeb, 1993)

Causes

1. The most common are biliary tract diseases and alcoholism
2. Abnormal organ structure
3. Metabolic and endocrine disorders (hyperlipidaemia etc.)
4. Pancreatic cysts/tumours
5. Penetrating peptic ulcers and trauma

Predisposing factors

1. Drug use such as glucocorticoids
2. Renal failure
3. Kidney transplant
4. Heredity

Pathophysiology

Acute pancreatitis is an inflammatory process that is caused by premature activation of pancreatic enzymes that destroy ductal tissue and pancreatic cells resulting in auto-digestion and fibrosis of the pancreas. The pathologic changes vary but these changes can lead to oedema and necrotizing haemorrhagic pancreatitis. The primary endocrine disorder resulting from the pancreatic changes is diabetes. The release of pancreatic enzymes prematurely and their

activation results in an inflammatory process, direct toxic injury to the pancreatic cells and further release of pancreatic enzymes.

With obstruction of the pancreatic duct, there is increase in pressure within the pancreas leading to rapture of the duct and spillage of the pancreatic juice within the pancreatic parenchymal tissue. In acute pancreatic four major pathophysiologic processes occur namely:

a. Lipolysis in which there is pancreatic necrosis
b. Proteolysis in which there is auto-digestion with localized pancreatic destruction.
c. Necrosis of blood vessels which is activated by trypsin and causes elastic fibbers of the vessels and duct s to dissolve.
d. Inflammation which happens when leucocytes cluster around the haemorrhagic and necrotic areas of the pancreas. Bacterial invasion may lead to suppuration or formation of an abscess (Ignatavicius and Workman, 2006).

Clinical Features

1. Sudden onset of severe abdominal pains radiating to the back. Later this pain becomes generalized
2. Nausea and vomitting may occur and persist
3. Abdominal distension and rigidity due to development of peritonitis
4. Shock- attributed to severity of pain, distension of the abdomen and loss of blood
5. Vital signs- temperature may be elevated initially but becomes subnormal with peritonitis. Pulse is rapid and blood pressure falls as peritonitis and shock develops
6. Blood changes- Serum bilirubin levels may rise after 2-3 days. Prothrombin levels will fall due to lack of absorption of vitamin K.

Medical Management

1. Analgesics- Pethidine is used for acute pain
2. Anticholinergics such as probanthine is given to inhibit stimulation if enzymes and promote relaxation of sphincter of oddi.
3. Vitamin K may be administered to prevent bleeding tendencies

Nursing Management

The specific points that you need to consider as you nurse a patient with pancreatitis include the following:

- Pass an NGT for aspiration of the stomach content to relieve vomitting and distension
- Nutrition- Keep the patient nil orally until condition improves. Start with light carbohydrates when fluids are swallowed, avoid fats, total abstinence from alcohol is advised.
- Fluids- Keep on IVF, give plasma or plasma expanders or whole blood depending on the shock and need of blood.
- Advise patient to abstain from alcohol
- Psychological care is given as patients are usually ill

Complications

- Paralytic ileus
- Renal failure
- Diabetes mellitus

CHRONIC PANCREATITIS

This may develop or occur following an initial acute episode. It develops slowly and is frequently associated with alcohol abuse. In the chronic disease there is progressive fibrosing and calcification of areas in the pancrease following inflammation and necrosis

Signs nd Symptoms

1. Recurrent attacks of pain in the Epigastric region
2. Anorexia, nausea, Flatulence and constipation
3. Bulky greasy and offensive or stertorrhoea stool
4. Progressive weight loss

Treatment

- Low fat diet to help reduce the diarrhoea
- Give multivitamin
- Insulin maybe required if glucose levels in the blood is high
- Analgesic for pain

PANCREATIC TUMOURS

Definition: this abnormal growth of pancreatic cells. The cancer usually arises from the head of the pancreas and almost always these are adenocarcinomas.

The tumours are known for obstructing the ampulla of vater and common bile duct and metastasize to the duodenum.

Causes

Cancer of the pancreas is closely related to the inhalation or absorption of carcinogens such as

- Cigarette smoking
- Excessive fat and protein intake
- Food additives
- Industrial chemicals e.g. urea, benzidine
- Others include
- Diabetes Mellitus and chronic pancreatitis and alcohol abuse

Clinical Manifestations

1. Intermittent epigastric pain
2. Pain that radiates to right upper quadrant as disease progresses
3. Anorexia, nausea, vomiting and loss of weight.
4. Jaundice
5. Palpable well defined abdominal mass
6. Abdominal bruit with compression of splenic artery

Complications

- Malabsorption of nutrients
- Insulin dependent diabetes
- Liver and GIT anomlies
- Change in mental status

Management

Investigations

- Aspiration biopsy
- Ultrasound
- Serum bilirubin
- Serum amylase

- Plasma insulin immunoassay

Treatment

1. Surgery is the most effective that includes pancreatectomy and cholecysjejunostomy.
2. Chemotherapy e.g. Fluorouracil, combined with spectinomycin, mitomycin and doxorubicin
3. Radiotherapy
4. Supportive treatment with antibiotics, antiacids, insulin.

Nursing Care

When nursing diabetes mellitus we used a nursing care plan. We will try and use the same to nurse patients with pancreatic tumours.

Some of the problems that a patient with pancreatic tumours may present with are:

1. Altered nutrition less than body requirements
2. Anxiety
3. Constipation
4. Fluid volume deficit
5. Impaired skin integrity
6. Ineffective individual coping
7. Knowledge deficit
8. Risk for infection

Activity

Using the identified nursing problems write the nursing care plan

You have finished looking and pancreatic disorder of which we defined pancreatitis, looked at the causes and predisposing factors to the condition and also looked at the basic management of the condition. We further looked at pancreatic cancer as well. In our next topic we will focus on pituitary disorders, but before that here is a self-test.

SELF TEST

1.Pancreatitis is mainly associated with

a. Bacteria
b. Adenoviruses
c. E-coli
d. Autoimmune reaction

2.The of pancreatitis progression is as follows

e. Inflammation, lipolysis, proteolysis and necrosis

f. Necrosis, proteolysis, inflammation, lipolysis

g. Lipolysis, proteolysis, necrosis and inflammation

h. Proteolysis, inflammation, necrosis and lipolysis

Answers: Q1 D. Q2 C.

3.6 Management Of A Patient With Pituitary Disorders

The pituitary gland is a gland that is responsible for the release of many hormones that are important in the regulation of body functions. It is divided into the anterior and posterior lobes and it is also connected to the hypothalamus.

HYPOPITUITARISM

The hormones released by the pituitary gland (and their functions) are:

- Adrenocorticotropic hormone (ACTH) - stimulates the adrenal gland to release cortisol; cortisol helps to maintain blood pressure and blood sugar
- Antidiuretic hormone (ADH) - controls water loss by the kidneys
- Follicle stimulating hormone (FSH) - controls sexual function and fertility in males and females
- Growth hormone (GH) - stimulates growth of tissues and bone
- Luteinizing hormone (LH) - controls sexual function and fertility in males and females
- Oxytocin - stimulates the uterus to contract during labor and the breasts to release milk
- Prolactin - stimulates female breast development and milk production
- Thyroid stimulating hormone (TSH) - stimulates the thyroid gland to release hormones that affect the body's metabolism

Lack of a hormone leads to loss of function in the gland or organ the hormone controls. For example, lack of TSH leads to loss of normal function of the thyroid gland.

- Hypopituitarism may be caused byaccidental or neurosurgical trauma; vascular events such as apoplexy;pituitary or hypothalamic neoplasms such as pituitary adenomas, craniopharyngiomas, or metastatic tumors;inflammatory disease such as lymphocytic

hypophysitis;infiltrative disorders such as sarcoidosis and tuberculosis hemochromatosis (Kasper et al, 2005)

Presentation And Diagnosis The clinical manifestations of hypopituitarism depend on which hormones are lost and the extent of the hormone deficiency. GH deficiency causes growth disorders in children and leads to abnormal body composition in adults.

Gonadotropin deficiency causes menstrual disorders and infertility in women and decreased sexual function, infertility, and loss of secondary sexual characteristics in men. TSH and ACTH deficiency usually develop later in the course of pituitary failure. TSH deficiency causes growth retardation in children and features of hypothyroidism in children and in adults. The secondary form of adrenal insufficiency caused by ACTH deficiency leads to hypocortisolism with relative preservation of mineralocorticoid production. PRL deficiency causes failure of lactation. When lesions involve the posterior pituitary, polyuria and polydipsia reflect loss of vasopressin secretion. Epidemiologic studies have documented an increased mortality rate in patients with longstanding pituitary damage, primarily from increased cardiovascular and cerebrovascular disease.

Management

Laboratory investigations

Laboratory tests measure levels of the hormones produced by the pituitary gland as well as levels of hormones produced by the target organs. For example, a person with hypothyroidism due to failure of the pituitary gland has low levels of the thyroid hormone and low levels of the thyroid stimulating hormone, which is produced by the pituitary gland.

Treatment

Hormone replacement therapy is the treatment of choice. Treatment regimens that mimic physiologic hormone production also allow for maintenance of satisfactory clinical homeostasis.

Patients in need of glucocorticoid replacement require careful dose adjustments during stressful events such as acute illness, dental procedures, trauma, and acute hospitalization (Kasper et al, 2005).

Nursing Care

Identified problem after assessment:

1. Disturbed body image
2. Sexual dysfunction
3. Anxiety
4. Ineffective coping
5. Deficient knowledge

Assignment

Using the problems stated above develop a nursing care plan for a patient suffering from hypopituitarism

PITUITARY TUMOURS

Pituitary adenomas are the most common cause of pituitary hormone hypersecretion and hyposecretion syndromes in adults. They account for 10% of all intracranial neoplasms. At autopsy, up to a quarter of all pituitary glands harbour an unsuspected microadenoma (10 mm diameter). Similarly, pituitary imaging detects small pituitary lesions in at least 10% of normal individuals (Kasper et al, 2005).

Pathogenesis

Pituitary adenomas are benign neoplasms that arise from one of the five anterior pituitary cell types. The clinical and biochemical phenotype of pituitary adenomas depend on the cell type from which they are derived. Thus, tumours arising from lactotrope (PRL), somatotrope (GH),

corticotrope (ACTH), thyrotrope (TSH), or gonadotrope (LH, FSH) cells hypersecrete their respective hormones (Kasper et al, 2005).

Clinical presentation

Usually visual disturbance, loss of bodyb hair, diabetes insipidus, sterility, and headaches.

Management

Investigations

Confirmatory – biopsy others are supportive

Surgery is the most effective, however dopamine agonists and somatostatin analogues.

Nursing care: use the same problems as for hypopituitarism.

You have come to the end of this subunit which dealt with disorders of the pituitary gland; our subunit will focus on managing a patient with thyroid disorders.

SELF TEST

1. The best modality of treatment for a patient with pituitary tumours is
    a. Dopamine agonist
    b. ADH therapy
    c. Radiotherapy
    d. Surgery
2. Apoplexy causes
    a. Thyroid tumours
    b. Hypopituitarism
    c. Hypothalamus deficiency
    d. Adrenal insufficiency

Answers: Q1 D. Q2 C.

3.7 Management Of A Patient With Thyroid Disorders

The thyroid gland is a butterfly-shaped organ located in the lower neck anterior to the trachea. It consists of two lateral lobes connected by an isthmus. The gland is about 5 cm long and 3 cm wide and weighs about 30 g. The blood flow to the thyroid is very high (about 5 mL/min per

gram of thyroid tissue), about five times the blood flow to the liver. This reflects the high metabolic activity of the thyroid gland. The thyroid gland produces three hormones: **thyroxine** (T4), **triiodothyronine** (T3), and **calcitonin**. Thyroxine and triiodothyronine are referred to collectively as thyroid hormone.

Thyroid Function And Dysfunction

Various hormones and chemicals are responsible for normal thyroid function. Key among them are thyroid hormone, calcitonin, and iodine.

Thyroid Hormone

The two separate hormones, thyroxine (T4) and triiodothyronine (T3), that are produced by the thyroid gland and that make up thyroid hormone, are amino acids that have the unique property containing iodine molecules bound to the amino acid structure. T4 contains four iodine atoms in each molecule, and T3 contains only three. These hormones are synthesized and stored bound to proteins in the cells of the thyroid gland until needed for release into the bloodstream. About 75% of bound thyroid hormone is bound to thyroxine-binding globulin (TBG); the remaining bound thyroid hormone is bound to thyroid-binding prealbumin and albumin.

Role Of Iodine

From your secondary school I am sure you remember one of the elements in the periodic table which is Iodine.

Iodine is essential to the thyroid gland for synthesis of its hormones. In fact, the major use of iodine in the body is by the thyroid, and the major derangement in iodine deficiency is alteration of thyroid function. Iodide is ingested in the diet and absorbed into the blood in the gastrointestinal tract (remember in Zambia all table salt must be iodised). The thyroid gland is extremely efficient in taking up iodide from the blood and concentrating it within the cells, where iodide ions are converted to iodine molecules, which react with tyrosine (an amino acid) to form the thyroid hormones.

Regulation Of Thyroid Hormone

The secretion of T3 and T4 by the thyroid gland is controlled by thyroid-stimulating hormone (TSH, or thyrotropin) from the anterior pituitary gland. TSH controls the rate of thyroid hormone release. In turn, the level of thyroid hormone in the blood determines the release of TSH. If thyroid hormone concentration in the blood decreases, the release of TSH increases, which causes increased output of T3 and T4. This is an example of negative feedback. Thyrotropin-releasing hormone (TRH), secreted by the hypothalamus, exerts a modulating influence on the release of TSH from the pituitary. Environmental factors, such as a decrease in temperature, may lead to increased secretion of TRH, resulting in elevated secretion of thyroid hormones. Figure 42-4 shows the hypothalamic-pituitary-thyroid axis, which regulates thyroid hormone production.

Function Of Thyroxine And Triiodothyronine

The primary function of the thyroid hormone is to control the cellular metabolic activity. T4, a relatively weak hormone, maintains body metabolism in a steady state. T3 is about five times as potent as T4 and has a more rapid metabolic action. These hormones accelerate metabolic processes by increasing the level of specific enzymes that contribute to oxygen consumption and altering the responsiveness of tissues to other hormones. The thyroid hormones influence cell replication and are important in brain development. Thyroid hormone is also necessary for normal growth. The thyroid hormones, through their widespread effects on cellular metabolism, influence every major organ system.

Calcitonin

Calcitonin, or thyrocalcitonin, is another important hormone secreted by the thyroid gland. It is secreted in response to high plasma levels of calcium, and it reduces the plasma level of calcium by increasing its deposition in bone.

Assessment and Diagnostic Findings

The thyroid gland is inspected and palpated routinely on all patients. Inspection begins with identification of landmarks. The lower neck region between the sternocleidomastoid muscles is inspected for swelling or asymmetry. The patient is instructed to extend the neck slightly and swallow. Thyroid tissue rises normally with swallowing. The thyroid is then palpated for size,

shape, consistency, symmetry, and the presence of tenderness. The examiner may examine the thyroid from an anterior or a posterior position. In the posterior position, both hands encircle the patient's neck. The thumbs rest on the nape of the neck, while the index and middle fingers palpate for the thyroid isthmus and the anterior surfaces of the lateral lobes. When palpable, the isthmus is perceived as firm and of a rubber-band consistency. The left lobe is examined by positioning the patient so that the neck flexes slightly forward and to the left. The thyroid cartilage is then displaced to the left with the fingers of the right hand. This maneuver displaces the left lobe deep into the sternocleidomastoidmuscle, where it can be more easily palpated. The left lobe is thenpalpated by placing the left thumb deep into the posterior area of the sternocleidomastoid muscle, while the index and middle fingers exert opposite pressure in the anterior portion of the muscle. Having the patient swallow during the maneuver may assist the examiner to locate the thyroid as it ascends in the neck. The procedure is reversed to examine the right lobe. The isthmus is the only portion of the thyroid that is normally palpable. If a patient has a very thin neck, two thin, smooth, nontender lobes may also be palpable. If palpation discloses an enlarged thyroid gland, both lobes are auscultated using the diaphragm of the stethoscope. Auscultation identifies the localized audible vibration of a bruit. This abnormal finding indicates increased blood flow through the thyroid gland and necessitates referral to a physician. Tenderness, enlargement, and nodularity within the thyroid also require referral for additional evaluation (Table 42-2).

Nursing Implications

When thyroid tests are scheduled, it is necessary to determine whether the patient has taken medications or agents that contain iodine because these may alter the test results. Iodine-containing medications include contrast agents and those used to treat thyroid disorders. Less obvious sources of iodine are topical antiseptics, multivitamin preparations, and food supplements frequently found in health food stores; cough syrups; and amiodarone, an antiarrhythmic agent. Other medications that may affect test results are estrogens, salicylates, amphetamines, chemotherapeutic agents, antibiotics, corticosteroids, and mercurial diuretics. The nurse ask the patient about the use of these medications and notes their use on the laboratory requisition. Chart 42-1 gives a partial list of agents that may interfere with accurate testing of thyroid gland function.

Abnormal Thyroid Function

Inadequate secretion of thyroid hormone during fetal and neonatal development results in stunted physical and mental growth (**cretinism**) because of general depression of metabolic activity. In adults, hypothyroidism manifests as lethargy, slow mentation, and generalized slowing of body functions. Over secretion of thyroid hormones (hyperthyroidism) is manifested by a greatly increased metabolic rate. Many of the other characteristics of hyperthyroidism result from the increased response to circulating catecholamines (epinephrine and norepinephrine). Hypothyroidism and hyperthyroidism are discussed in detail in the following sections of this chapter. Over secretion of thyroid hormones is usually associated with an enlarged thyroid gland (**goiter**). Goiter also commonly occurs with iodine deficiency. In this latter condition, lack of iodine results in low levels of circulating thyroid hormones, which causes increased release of TSH; the elevated TSH causes overproduction of thyroglobulin and hypertrophy of the thyroid gland. The term **euthyroid** refers to thyroid hormone production that is within normal limits.

THYROIDITIS

Thyroiditis, inflammation of the thyroid gland, can be acute, subacute, or chronic. Each type of thyroiditis is characterized by inflammation, fibrosis, or lymphocytic infiltration of the thyroid gland.

Acute Thyroiditis

Acute thyroiditis is a rare disorder caused by infection of the thyroid gland by bacteria, fungi, mycobacteria, or parasites. *Staphylococcus aureus* and other staphylococci are the most common causes. Infection typically causes anterior neck pain and swelling, fever, dysphagia, and dysphonia. Pharyngitis or pharyngeal pain is often present. Examination may reveal warmth, erythema (redness), and tenderness of the thyroid gland. Treatment of acute thyroiditis includes antimicrobial agents and fluid replacement. Surgical incision and drainage may be needed if an abscess is present.

Subacute Thyroiditis

Subacute thyroiditis may be subacute granulomatous thyroiditis (deQuervain's thyroiditis) or painless thyroiditis (silent thyroiditis or subacute lymphocytic thyroiditis). Subacute granulomatous thyroiditis is an inflammatory disorder of the thyroid gland that predominantly affects women between 40 and 50 years old (Smallridge, 2000).

The condition presents as a painful swelling in the anterior neck that lasts 1 to 2 months and then disappears spontaneously without residual effect. It often follows a respiratory infection. The thyroid enlarges symmetrically and may be painful. The overlying skin is often reddened and warm. Swallowing may be difficult and uncomfortable. Irritability, nervousness, insomnia, and weight loss—manifestations of hyperthyroidism—are common, and many patients experience chills and fever as well.

Treatment aims to control the inflammation. In general, nonsteroidal anti-inflammatory drugs (NSAIDs) are used to relieve neck pain. Acetylsalicylic acid (aspirin) is avoided if symptoms of hyperthyroidism occur because aspirin displaces thyroid hormone from its binding sites and increases the amount of circulatinghormone. Beta-blocking agents (eg, propranolol [Inderal]) may be used to control symptoms of hyperthyroidism. Antithyroid agents, which block the synthesis of T3 and T4, are not effective in thyroiditis because the associated thyrotoxicosis results from the release of stored thyroid hormones rather than from their increased synthesis. In more severe cases, oral corticosteroids may be prescribed to reduce swelling and relieve pain; however, they do not usually affect the underlying cause. In some cases, temporary hypothyroidism may develop and may necessitate thyroid hormone therapy.

Follow-up monitoring is necessary to document the patient's return to a euthyroid state. Painless thyroiditis (subacute lymphocytic thyroiditis) often occurs in the postpartum period and is thought to be an autoimmune process. Symptoms of hyperthyroidism or hypothyroidism are possible. Treatment is directed at symptoms, and yearly follow-up is recommended to determine the patient's need for treatment of subsequent hypothyroidism.

Chronic Thyroiditis (Hashimoto's Disease)

Chronic thyroiditis, which occurs most frequently in women between 30 and 50 years old, has been termed Hashimoto's disease, or chronic lymphocytic thyroiditis; its diagnosis is based on the histologic appearance of the inflamed gland. In contrast to acute thyroiditis, the chronic forms are usually not accompanied by pain, pressure symptoms, or fever, and thyroid activity is usually normal or low rather than increased.

Cell-mediated immunity may play a significant role in the pathogenesis of chronic thyroiditis, and there may be a genetic predisposition to it. If untreated, the disease runs a slow, progressive course, leading eventually to hypothyroidism.The objective of treatment is to reduce the size of the thyroid gland and prevent hypothyroidism. Thyroid hormone therapy is prescribed to reduce thyroid activity and the production of thyroglobulin. If hypothyroid symptoms are present, thyroid hormone therapy is prescribed. Surgery may be required if pressure symptoms persist.

THYROID TUMORS

Tumors of the thyroid gland are classified on the basis of beingbenign or malignant, the presence or absence of associated thyrotoxicosis, and the diffuse or irregular quality of the glandular enlargement. If the enlargement is sufficient to cause a visible swelling in the neck, the tumor is referred to as a goiter. All grades of goiter are encountered, from those that are barely visible to those producing disfigurement. Some are symmetric and diffuse; others are nodular. Some are accompanied by hyperthyroidism, in which case they are described as toxic; others are associated with a euthyroid state and are called nontoxic goiters.

Endemic (Iodine-Deficient) Goiter

The most common type of goiter, encountered chiefly in geographic regions where the natural supply of iodine is deficient (eg, the Great Lakes areas of the United States), is the so-called simple or colloid goiter. In addition to being caused by an iodine deficiency, simple goiter may be caused by an intake of large quantities of goitrogenic substances in patients with unusually susceptible glands. These substances include excessive amounts of iodine or lithium, which is used in treating bipolar disorders. Simple goiter represents a compensatory hypertrophy of the thyroid gland, caused by stimulation by the pituitary gland.

The pituitary gland produces thyrotropin or TSH, a hormone that controls the release of thyroid hormone from the thyroid gland. Its production increases if there is subnormal thyroid activity, as when insufficient iodine is available for production of the thyroid hormone. Such goiters usually cause no symptoms, except for the swelling in the neck, which may result in tracheal compression when excessive. Many goiters of this type recede after iodine imbalance is corrected. Supplementary iodine, such as SSKI, is prescribed to suppress the pituitary's thyroid-stimulating activity.

When surgery is recommended, the risk for postoperative complications is minimized by ensuring a preoperative euthyroid state by treatment with antithyroid medications and iodide to reduce the size and vascularity of the goiter. Providing children in iodine-poor regions with iodine compounds can prevent simple or endemic goiter. If the mean iodine intake is less than 40 fg/day, the thyroid gland hypertrophies. The World Health Organization recommends that salt be iodized to a concentration of 1 part in 100,000, which is adequate for the prevention of endemic goiter. In the United States, salt is iodized to 1 part in 10,000. The introduction of iodized salt has been the single most effective means of preventing goiter in at-risk populations.

Nodular Goiter

Some thyroid glands are nodular because of areas of hyperplasia (overgrowth). No symptoms may arise as a result of this condition, but not uncommonly these nodules slowly increase in size, with some descending into the thorax, where they cause local pressure symptoms. Some nodules become malignant, and some complete listing of inherited genetic conditions are associated with a hyperthyroid state. Thus, the patient with many thyroid nodules may eventually require surgery.

Management **Of Thyroid Storm**

- Immediate objectives are reduction of body temperature and heart rate and prevention of vascular collapse. The following are measures to accomplish these:A hypothermia mattress or blanket, ice packs, a cool environment, hydrocortisone, and acetaminophen (Tylenol). Salicylates (eg, aspirin) are not used because they displace thyroid hormone from binding proteins and worsen the hypermetabolism.
- Humidified oxygen is administered to improve tissue oxygenation and meet the high metabolic demands. Arterial blood gas levels or pulse oximetry may be used to monitor respiratory status.
- Intravenous fluids containing dextrose are administered to replace liver glycogen stores that have been decreased in the hyperthyroid patient.
- Methimazole is administered to impede formation of thyroid hormone and block conversion of T4 to T3, the more active form of thyroid hormone.
- Hydrocortisone is prescribed to treat shock or adrenal insufficiency.
- Iodine is administered to decrease output of T4 from the thyroid gland. For cardiac problems such as atrial fibrillation, dysrhythmias, and heart failure, sympatholytic agents may be administered. Propranolol, combined with digitalis, has been effective in reducing severe cardiac symptoms.

THYROID CANCER

Cancer of the thyroid is much less prevalent than other forms of cancer; however, it accounts for 90% of endocrine malignancies. According to the American Cancer Society (2002 in Brunner and Suddarth, 2010), about 20,700 new cases of thyroid cancer are diagnosed each year. Women account for 15,800 of the new cases and men 4,900. About 800 women and 500 men die annually from this malignancy.

There are several types of cancer of the thyroid gland; the type determines the course and prognosis. External radiation of the head, neck, or chest in infancy and childhood increases the risk of thyroid carcinoma. Between 1940 and 1960, radiation therapy was occasionally used to shrink enlarged tonsillar and adenoid tissue, to treat acne, or to reduce an enlarged thymus.

For people exposed to external radiation in childhood, there appears to be an increased incidence of thyroid cancer 5 to 40 years after irradiation. Consequently, people who underwent such treatment should consult a physician, request an isotope thyroid scan as part of the evaluation, follow recommended treatment of abnormalities of the gland, and continue with annual check-ups.

Assessment and Diagnostic Findings

Lesions that are single, hard, and fixed on palpation or associated with cervical lymphadenopathy suggest malignancy. Thyroid function tests may be helpful in evaluating thyroid nodules and masses; however, their results are rarely conclusive.

Needle biopsy of the thyroid gland is used as an outpatient procedure to make a diagnosis of thyroid cancer, to differentiate cancerous thyroid nodules from noncancerous nodules, and to stage the cancer if detected. The procedure is safe and usually requires only a local anaesthetic. Patients who undergo the procedure are followed closely, however, because cancerous tissues may be missed during the procedure.

A second type of aspiration or biopsy uses a large bore needle rather than the fine needle used in standard biopsy; it may be used when the results of the standard biopsy are inconclusive, or with rapidly growing tumours. Additional diagnostic studies include ultrasound, MRI, CT scans, thyroid scans, radioactive iodine uptake studies, and thyroid suppression tests.

Medical Management

The treatment of choice for thyroid carcinoma is surgical removal. Total or near-total thyroidectomy is performed when possible. Modified neck dissection or more extensive radical neck dissection is performed if there is lymph node involvement.

Surgical Management

Efforts are made to spare parathyroid tissue to reduce the risk for postoperative hypocalcemia and tetany. After surgery, ablation procedures are carried out with radioactive iodine to eradicate residual thyroid tissue if the tumor is radiosensitive.

Radioactive iodine also maximizes the chance of discovering thyroid metastasis at a later date if total-body scans are carried out. After surgery, thyroid hormone is administered in suppressive doses to lower the levels of TSH to a euthyroid state (Thyroid Carcinoma Guidelines, 2001). If remaining thyroid tissue is inadequate to produce sufficient thyroid hormone, thyroxine is required permanently.

Several routes are available for administering radiation to the thyroid or tissues of the neck, including oral administration of radioactive iodine and external administration of radiation therapy. The patient who receives external sources of radiation therapy is at risk for mucositis, dryness of the mouth, dysphagia, redness of the skin, anorexia, and fatigue (see Chap. 16 for a discussion of these side effects of radiation).

Chemotherapy is infrequently used to treat thyroid cancer. Patients whose thyroid cancer is detected early and who are appropriately treated usually do very well. Patients who have had papillary cancer, the most common and least aggressive tumour, have a 10-year survival rate greater than 90%. Long-term survival is also common in follicular cancer, a more aggressive form of thyroid cancer (Tierney et al., 2001). Continued thyroid hormone therapy and periodic follow-up and diagnostic testing, however, are important to ensure the patient's well-being (Thyroid Carcinoma Guidelines, 2001).

Postoperatively, the patient is instructed to take exogenous thyroid hormone to prevent hypothyroidism. Later follow-up includes clinical assessment for recurrence of nodules or masses in the neck and signs of hoarseness, dysphagia, or dyspnoea. Total body scans are performed 2 to 4 months after surgery to detect residual thyroid tissue or metastatic disease.

Thyroid hormones are stopped for about 6 weeks before the tests. Care must be taken to avoid iodine-containing foods and contrast agents. A repeat scan is done 1 year after the initial surgery. If measurements are stable, a final scan is obtained in 3 to 5 years. FT4, TSH, serum calcium, and phosphorus levels are monitored to determine whether the thyroid hormone supplementation is adequate and to note whether calcium balance is maintained.

Although local and systemic reactions to radiation may occur and may include neutropenia or thrombocytopenia, these complications are rare when radioactive iodine is used. Patients who undergo surgery that is combined with radioiodine have a higher survival rate than those undergoing surgery alone. Patient teaching emphasizes the importance of taking prescribed medications and following recommendations for follow-up monitoring.

The patient who is undergoing radiation therapy is also instructed in how to assess and manage side effects of treatment. Partial or complete **thyroidectomy** may be carried out as primary treatment of thyroid carcinoma, hyperthyroidism, or hyperparathyroidism. The type and extent of the surgery depend on the diagnosis, goal of surgery, and prognosis. Thyroidectomy may be the treatment of choice for patients with symptomatic hyperparathyroidism (see later discussion), kidney stones, or bone disease.

The patient undergoing surgery for treatment of hyperthyroidism is given appropriate medications to return the thyroid hormone levels and metabolic rate to normal and to reduce the risk for thyroid storm and hemorrhage during the postoperative period. Medications that may prolong clotting (eg, aspirin) are stopped several weeks before surgery to minimize the risk for postoperative bleeding.

Nursing Management

Important preoperative goals are to gain the patient's confidence and reduce anxiety. Often, the patient's home life has become tense because of his or her restlessness, irritability, and nervousness secondary to hyperthyroidism.

Efforts are necessary to protect the patient from such tension and stress to avoid precipitating thyroid storm. If the patient reports increased stress when with family or friends, suggestions are made to limit contact with them. Quiet and relaxing forms of recreation or occupational therapy may also be helpful.

Providing Preoperative Care

As a nurse you instruct the patient about the importance of eating a diet high in carbohydrates and proteins. A high daily caloric intake is necessary because of the increased metabolic activity and rapid depletion of glycogen reserves.

Supplementary vitamins, particularly thiamine and ascorbic acid, may be prescribed. The patient is reminded to avoid tea, coffee, cola, and other stimulants.

You should also inform the patient about the purpose of preoperative tests, if they are to be performed, and explains what preoperative preparations to expect. The information should help to reduce the patient's anxiety about the surgery.

In addition, special efforts are made to ensure a good night's rest before surgery, although many patients are admitted to the hospital on the day of surgery.

Your preoperative teaching includes demonstrating to the patient how to support the neck with the hands after surgery to prevent stress on the incision. This involves raising the elbows and placing the hands behind the neck to provide support and reduce strain and tension on the neck muscles and the surgical incision.

Providing Postoperative Care
you should periodically assess the surgical dressings and reinforce them when necessary.

When the patient is in a recumbent position, observe the sides and the back of the neck as well as the anterior dressing for bleeding.

In addition to monitoring the pulse and blood pressure for any indication of internal bleeding, it is also important to be alert for complaints of a sensation of pressure or fullness at the incision site. Such symptoms may indicate haemorrhage and hematoma formation subcutaneously and should be reported.

Difficulty in respiration occurs as a result of oedema of the glottis, hematoma formation, or injury to the recurrent laryngeal nerve. This complication requires that an airway be inserted. Therefore, a tracheostomy set is kept at the bedside at all times, and the surgeon is summoned at the first indication of respiratory distress. If the respiratory distress is due to hematoma, surgical evacuation is required.

The intensity of pain is assessed and analgesic agents are administered as prescribed for pain. The nurse should anticipate apprehension in the patient and should inform him or her that oxygen will assist breathing.

When moving and turning the patient, the nurse carefully supports the head and avoids tension on the sutures. The most comfortable position is the semi-Fowler's position, with the head elevated and supported by pillows. Intravenous fluids are administered during the immediate postoperative period.

Water may be given by mouth as soon as nausea subsides. Usually, there is a little difficulty in swallowing; initially, cold fluids and ice may be taken better than other fluids. Often, patients prefer a soft diet to a liquid diet in the immediate postoperative period.

The patient is advised to talk as little as possible to reduce oedema to the vocal cords, but when the patient does speak, any voice changes are noted because they might indicate injury to the recurrent laryngeal nerve, which lies just behind the thyroid next to the trachea.

An overbed table may be used to provide easy access to items that are needed frequently, such as paper tissues, water pitcher and glass, and a small emesis basin. These are kept within easy reach so that the patient will not need to turn the head to reach for them. It is also convenient to use this table when vapour-mist inhalations are prescribed for the relief of excessive mucous secretions.

The patient is usually permitted out of bed as soon as possible and is encouraged to eat foods that are easily eaten. A well-balanced, high-calorie diet may be prescribed to promote weight gain. Sutures or skin clips are usually removed on the second day.

The patient is usually discharged from the hospital the day of surgery or soon afterward if the postoperative course is uncomplicated.

Monitoring and Managing Potential Complications

Haemorrhage, hematoma formation, oedema of the glottis, and injury to the recurrent laryngeal nerve are complications that have been reviewed previously in this chapter.

Occasionally in thyroid surgery the parathyroid glands are injured or removed, producing a disturbance in calcium metabolism. As the blood calcium level falls, hyperirritability of the nerves occurs, with spasms of the hands and feet and muscle twitching. This group of symptoms is termed tetany, and the nurse must immediately report its appearance because laryngospasm, although rare, may occur and obstruct the airway. Tetany of this type is usually treated with intravenous calcium gluconate. This calcium abnormality is usually temporary after thyroidectomy.

Promoting Home and Community-Based Care

Teaching Patient's Self-Care - The patient may be discharged the evening of surgery or within 1 or 2 days. Therefore, the patient and family need to be knowledgeable about the signs and symptoms of the complications that may occur and those that should be reported.

Strategies are suggested for managing postoperative pain at home and for increasing humidification. The nurse explains to the patient and family the need for rest, relaxation, and nutrition. The patient is permitted to resume his or her former activities and responsibilities completely once recovered from surgery.

Continuing Care - If indicated, a referral to home care is made. The home care nurse assesses the patient's recovery from surgery. The nurse also assesses the surgical incision and reinforces instruction about limiting activities that put strain on the incision and sutures.

Family responsibilities and factors relating to the home environment that produce emotional tension have often been implicated as precipitating causes of thyrotoxicosis. A home visit provides an opportunity to evaluate these factors and to suggest ways to improve the home and family environment.

The nurse gives specific instructions regarding follow-up visits to the physician or the clinic, which are important for monitoring the thyroid status.

HYPERPARATHYROIDISM

Hyperparathyroidism, which is caused by overproduction of parathyroid hormone by the parathyroid glands, is characterized by bone decalcification and the development of renal calculi (kidney stones) containing calcium. Primary hyperparathyroidism occurs two to four times more often in women than in men and is most common in patients between 60 and 70 years of age.

About 100,000 new cases of hyperparathyroidism are detected each year in the United States. The disease is rare in children younger than 15 years, but the incidence increases tenfold between the ages of 15 and 65 years. Half of the patients diagnosed with hyperparathyroidism do not have symptoms. Secondary hyperparathyroidism, with manifestations similar to those of primary hyperparathyroidism, occurs in patients with chronic renal failure and so-called renal rickets as a result of phosphorus retention, increased stimulation of the parathyroid glands, and increased parathyroid hormone secretion.

Clinical Manifestations

The patient may have no symptoms or may experience signs and symptoms resulting from involvement of several body systems.

- Apathy
- fatigue,
- muscle weakness,
- nausea,
- vomiting,

- constipation,
- hypertension, and
- cardiac dysrhythmias may occur;

All are attributable to the increased concentration of calcium in the blood.

Psychological manifestations may vary from irritability and neurosis to psychoses caused by the direct effect of calcium on the brain and nervous system. An increase in calcium produces a decrease in the excitation potential of nerve and muscle tissue.

The formation of stones in one or both kidneys, related to the increased urinary excretion of calcium and phosphorus, is one of the important complications of hyperparathyroidism and occurs in 55% of patients with primary hyperparathyroidism.

Renal damage results from the precipitation of calcium phosphate in the renal pelvis and parenchyma, resulting in renal calculi (kidney stones), obstruction, pyelonephritis, and renal failure.

Musculoskeletal symptoms accompanying hyperparathyroidism may result from demineralization of the bones or bone tumours composed of benign giant cells resulting from overgrowth of osteoclasts.

The patient may develop skeletal pain and tenderness, especially of the back and joints; pain on weight bearing; pathologic fractures; deformities; and shortening of body stature. Bone loss attributable to hyperparathyroidism increases the risk for fracture.

The incidence of peptic ulcer and pancreatitis is increased with hyperparathyroidism and may be responsible for many of the gastrointestinal symptoms that occur.

Assessment and Diagnostic Findings

- Primary hyperparathyroidism is diagnosed by persistent elevation of serum calcium levels and an elevated level of parathormone

- Radioimmunoassays for parathormone are sensitive and differentiate primary hyperparathyroidism from other causes of hypercalcemia in more than 90% of patients with elevated serum calcium levels.
- An elevated serum calcium level alone is a nonspecific finding because serum levels may be altered by diet, medications, and renal and bone changes.
- Bone changes may be detected on x-ray or bone scans in advanced disease.
- The double antibody parathyroid hormone test is used to distinguish between primary hyperparathyroidism and malignancy as a cause of hypercalcemia.
- Ultrasound, MRI, thallium scan, and fine-needle biopsy have been used to evaluate the function of the parathyroids and to localize parathyroid cysts, adenomas, or hyperplasia.

Complications: Hypercalcemic Crisis

Acute hypercalcemic crisis can occur with extreme elevation of serum calcium levels. Serum calcium levels higher than 15 mg/dL (3.7 mmol/L) result in neurologic, cardiovascular, and renal symptoms that can be life-threatening.

Treatment includes rehydration with large volumes of intravenous fluids, diuretic agents to promote renal excretion of excess calcium, and phosphate therapy to correct hypophosphatemia and decrease serum calcium levels by promoting calcium deposit in bone and reducing the gastrointestinal absorption of calcium.

Cytotoxic agents (mithramycin), calcitonin, and dialysis may be used in emergency situations to decrease serum calcium levels quickly.

A combination of calcitonin and corticosteroids has been administered in emergencies to reduce the serum calcium level by increasing calcium deposition in bone. Other agents that may be administered to decrease serum calcium levels include bisphosphonates (eg, etidronate [Didronel], pamidronate).

The patient requires expert assessment and care to minimize complications and reverse the life-threatening hypercalcaemia.

Medications are administered with careand attention is given to fluid balance to promote return of normal fluid and electrolyte balance. Supportive measures are necessary for the patient and family.

Medical Management

The insidious onset and chronic nature of hyperparathyroidism and its diverse and commonly vague symptoms may result in depression and frustration. The family may have considered the patient's illness to be psychosomatic.

An awareness of the course of the disorder and an understanding approach by the nurse may help the patient and family to deal with their reactions and feelings.

The recommended treatment of primary hyperparathyroidism is the surgical removal of abnormal parathyroid tissue. In some patients without symptoms and with only mildly elevated serum calcium levels and normal renal function, surgery may be delayed and the patient followed closely for worsening of hypercalcaemia, one deterioration, renal impairment, or the development of kidney stones.

Hydration Therapy

Because kidney involvement is possible, patients with hyperparathyroidism are at risk for renal calculi. Therefore, a fluid intake of 2,000 mL or more is encouraged to help prevent calculus formation.

Cranberry juice is suggested because it may lower the urinary pH. It can be added to juices and ginger ale for variety.

The patient is instructed to report other manifestations of renal calculi, such as abdominal pain and haematuria. Thiazide diuretics are avoided because they decrease the renal excretion of calcium and further elevate serum calcium levels.

Because of the risk of hypercalcaemic crisis, the patient is instructed to avoid dehydration and to seek immediate health care if conditions that commonly produce dehydration (e.g. vomiting, diarrhoea) occur.

Mobility

Mobility of the patient, with walking or use of a rocking chair for those with limited mobility,s encouraged as much as possible because bones subjected to normal stress give up less calcium.

Bed rest increases calcium excretion and the risk for renal calculi.

Oral phosphates lower the serum calcium level in some patients. Longterm use is not recommended because of the risk for ectopic calcium phosphate deposits in soft tissues.

Diet and Medications

Nutritional needs are met, but the patient is advised to avoid a diet with restricted or excess calcium. If the patient has a coexisting peptic ulcer, prescribed antacids and protein feedings are necessary. Because anorexia is common, efforts are made to improve the appetite.

Prune juice, stool softeners, and physical activity, along with increased fluid intake, help to offset constipation, which is common postoperatively.

Nursing Management

The nursing management of the patient undergoing parathyroidectomy is essentially the same as that of a patient undergoing thyroidectomy.

However, the previously described precautions about dehydration, immobility, and diet are particularly important in the patient awaiting and recovering from parathyroidectomy.

Although not all parathyroid tissue is removed during surgery in an effort to control the calcium–phosphorus balance, the nurse closely monitors the patient to detect symptoms of tetany (which may be an early postoperative complication).

Most patients quickly regain function of the remaining parathyroid tissue and experience only mild, transient postoperative hypocalcemia. In patients with significant bone disease or bone changes, a more prolonged period of hypocalcemia should be anticipated. The nurse reminds the patient and family about the importance of follow-up to ensure return of serum calcium levels to normal.

HYPERTHYROIDISM (THYROTOXICOSIS)

Definition

The term thyrotoxicosis refers to the physiologic effects of hyper metabolism that results from excess secreting levels of thyroxin (T4), tri-iodothyronine (T3) or both. Hyperthyroidism and thyrotoxicosis usually occur together as in Graves disease. In some form of thyroiditis, thyrotoxicosis may occur without hyperthyroidism. Hyperthyroidism occurs in approximately 2% of women and only 0.2% men; the highest frequency is in the 30 to 50 year old age group.

Definition

Thyrotoxicosis is a clinical syndrome caused by an excess of circulating free T4 and T3 or both characterized by increased metabolic rate. It can also be defined as a clinical syndrome in which there is sustained increase in synthesis and release of thyroid hormones by the thyroid gland.

Predisposing factors

- Insufficient iodine supply
- Genetic
- Age
- Gender (more common in women than men)

Incidence

Occurs equally in men and women

Toxic multinodular goitre is greatest in people over 40 years of age.

The common causes of thyrotoxicosis include:

- Grave's disease (toxic diffuse goitre) due to autoimmune process; serum IgG antibodies bind to thyroid stimulating hormone receptor which stimulates thyroid hormone production.
- Toxic multinodular goitre; nodules secrete thyroid hormones and are independent of thyroid stimulating hormone.
- Toxic adenoma; causes about 5% of cases of hyperthyroidism; does not resolve after course of antithyroid drugs.
- Exogenous hyperthyroidism (excessive use of thyroid replacement hormones).
- Thyroiditis; acute inflammatory process of the thyroid gland which is viral in origin.
- Thyroid secreting hormone tumors.
- Ovarian teratomas.
- Molar pregnancies.

Pathophysiology

Excessive T3, T4 secretion leads to thyrotoxicosis. The thyroid hormones are synthesized as large precursor molecules called thyroglobulin which is the major constituent of colloid. The release of T3 and T4 into the blood is regulated by the thyroid stimulating hormone (TSH) from the anterior pituitary gland. Secretion of TSH is stimulated by thyroid releasing hormone (TRH) from the hypothalamus and secretion of the TRH is stimulated by exercise, stress, malnutrition, low plasma glucose and sleep.

The levels of secretion of TRH depend on the plasma levels of T3 and T4 because this hormone affects the sensitivity of the anterior pituitary. Any alteration in the normal function of the thyroid gland e.g. when the supply of iodine is deficient, excess TSH is secreted and there is proliferation of thyroid gland cells and enlargement of the gland. Nodules will form in the goiter and excessive T3, T4 production results.

These hormones increase metabolic rates in all body organs. They also increase sympathetic nervous system activity causing fine tremors and low heat tolerance. The thyroid hormones directly stimulate the heart increasing the heart rate and cardiac output and blood flow. The elevated thyroid hormone affect protein, lipid and carbohydrate metabolism. Protein synthesis and breakdown is also increased.

However, breakdown exceeds build-up causing a net protein known as negative nitrogen balance. Glucose levels are also increased and patient ends up with hyperglycaemia. Fat metabolism increases and body fat decreases. Although the client has an increased appetite, food intake does not meet energy demand and the client loses weight with prolonged hyperthyroidism and the patient has chronic nutritional deficiency.

The high thyroid hormone affects the production of stimulating hormones from the anterior pituitary and hypothalamus. In addition thyroid hormone influence the sex hormone production in both men and women. Women develop menstrual defects and infertility. Both women and men with hyperthyroidism have an increased libido.

Signs and Symptoms

- Palpitations; due to increased heart rate
- Heat intolerance; due to increased metabolic rate causing fever
- nervousness; due to stimulation of the sympathetic nervous system
- Insomnia; due to increased metabolic rate
- Breathlessness with or without exertion; due to increased metabolic rate and an increase in oxygen demand
- Rapid glucose absorption and utilization
- Diarrhea; due to increased peristaltic movements
- Light or absent menstrual periods due to the influence of the thyroid hormones on the sex hormone
- Fatigue; due to increased metabolic rate
- Hair loss; due to impaired protein metabolism
- Muscle weakness; due to tissue hypoxia
- Decreased concentration.
- Mood swings due to hormonal imbalance
- Trembling hands due to stimulation of the sympathetic nervous system
- Exophthalmos; due to piling of fats behind the eye
- Gynaecomastia; due to suppression of androgen production.

Investigations

- Clinical features such as exophthalmos may help to make a provisional diagnosis
- History taking may reveal weight loss despite good appetite
- Measuring the levels of thyroid stimulating hormone in the blood which is usually low due to inhibition by high thyroid hormones
- Blood for T3 and T4 will be elevated
- Sleeping pulse will be high
- A Radioactive Iodine Uptake (RAIU) test will be increased which may indicate hyper secretion by the thyroid gland. It also differentiates graves disease from other forms of thyroiditis
- Ultrasound scan of the thyroid gland may reveal enlarged glands
- X-ray of the neck
- T3 and T4 check-up in serum
- Increased levels of TSH.

Management

Three primary treatment options are available

Drug therapy

1. **Antithyroid medications (thyrostatics)**

Action
Inhibit the production of thyroid hormones
Also blocks peripheral conversion of T3 and T4

Carbimazole
Dose: 10-60mg/ day in two divided doses.
Side effects: rash, nausea, agranulocytosis and arthralgia.

Propthiouracil
Dose: 100-200mg tds
Side effects: as above

Methimazole (tapazole)
Dose: 10-60mg/ day in 2 divided doses
Side effects: nausea, vomiting.
A very high dose is often needed early in treatment but if too high dose is used persistently, patient can develop symptoms of hypothyroidism.

2. **Beta blockers**

Action: drugs which control symptoms only

Propranolol
Dose: 10-40mg tds po
Side effects: hypotension
Metoprolol
Dose:50mg qid

3. Iodine (LI or LS)

Action: in large doses it rapidly inhibits the synthesis of T3 and T4 and blocks release of these hormones. It also decreases vascularity of the thyroid gland, making surgery safer and easier.

B. Radioactive iodine therapy

Damages or destroys thyroid tissue, thus limiting thyroid hormone secretion.

C. Surgery

Indications

- **Large goitre**
- **Pressure symptoms**
- **Failed medical treatment**

The procedure done is thyroidectomy

A surgical procedure that involves partial or total removal of the thyroid gland.

D. Nutritional therapy

High calorie diet (4000 to 5000kcal/day): to satisfy hunger and prevent tissue breakdown.

No seasoned and high fibre foods or caffeine containing liquids.Six full meal a day and snacks high in proteins, carbohydrates, minerals, and vitamins, particularly vitamin A, thiamine, vitamin B6, and vitamin C.

Nursing Care

Environment

The patient must be nursed in a clean, well ventilated environment. The environment should be free of noise and must be cool. Nurse patient in a dim lit environment

Psychological care

The patient and relatives will be fearful and worried about the patient's appearance, emotional reaction and the condition itself. Therefore, let patient and relatives ventilate their fears, worries and also ask them to say what they know about this condition. Therefore, explain to them in simple terms that this condition is due to increased production of thyroxin by the thyroid gland in the body and that they should not worry too much because everything possible is being done to

control the situation. This will be done with the help of drugs. By so doing you will alley anxiety and gain cooperation.

Explain all procedures done on the patient to gain cooperation and promote patient nurse relationship. Explain that the condition is long term and so is drug therapy, therefore it is necessary to comply to it. Explain the side effects of drugs, like urticaria, dermatitis, nausea and arthralgia. This is to avoid patient from being alarmed when he experiences these effects without being told earlier.

Observations

Four (4) hourly observations of vital signs is very important in this patient to act as a guide to progress. Sleeping pulse is monitored to rule out hypertension and heart rate to rule out fibrillation and tachycardia. Observe side effects of drugs such as urticaria, dermatitis and nausea. Observe weight by weighing the patient daily to monitor whether the patient is gaining or loosing and also helps in assessing dietary treatment effectiveness. Fluid intake and urine output to be monitored in order to estimate fluid replacement and also to ensure proper kidney function. Measure the neck circumference to check for thyroid enlargement.

Observe for cardiac failure like jugular vein distension, pulmonary crackles, peripheral or sacral oedema. Observe stool for diarrhoea. Observe eyes for protrusion whether they are subsiding or not and cornea for ulcers.

Promotion of rest and comfort

Rest is essential in conserving the client's strength. The accomplishment of this objective may present a challenge to the nurse because if the client's tendency to be nervous and easily excited. A calm quite environment must be maintained and all stimuli must be kept at a minimum to promote rest. Change linen whether soiled to promote rest and comfort. Restrict stressful situations like very ill patients, dying patients and also stimulating visitors who will bring sad news to the patient to promote rest. Plan your nursing care to avoid disturbing the patient hence promoting adequate time for rest. Give prescribed sedatives like diazepam to calm the patient and encourage rest. Since heat is poorly tolerated by many of these patients/clients, a comfortable environmental temperature must be maintained. Clothing should be loose and it may be helpful to give frequent cool baths and cool drinks in order to cool patient's body. Open nearby windows, remove excess linen and give light linen to prevent over warming the patient. If possible fan can be provided to promote heat loss by evaporation.

Maintaining nutrition

Because of the increased metabolic rate there is an increased need for nutrients adequate in calories to supply the increased body needs. To compensate for the energy used up due to hyperactivity, dietician should be consulted to ensure that adequate calories are given to satisfy hunger and prevent tissue breakdown. Give calories of about 400 to 500 are given daily. Give six full meals a day with snacks in between high in protein, carbohydrates, minerals and vitamins (especially thiamine and ascorbic acid) are recommended to sustain the high rate of metabolism. Increased fluids are needed, as there is usually increased perspiration, polyuria and increased metabolic wastes. Highly seasoned or fibrous foods are discouraged because they stimulate the already hyper motile gastrointestinal tract. Stimulants such as coffee, tea or cola are also omitted since they increase the existing overactive autoimmune responses.

Eye care

If ophthalmosis is present, the cornea must be protected from ulceration, irritation and infection. Some clients may also suffer from orbital pain. There are a number of nursing measures to help reduce eye discomfort. Methylcellulose drops soothe the membranes and help prevent drying. Elevation of the head improves drainage and reduce congestion. Dark glasses reduces glare and prevent irritation from dust and dirt. If the eyelids cannot be closed they should be lightly tapped shut. To maintain flexibility, the client should be taught to exercise the intraocular muscles by turning the eyes in complete range of motions. Good glooming face can be helpful in reducing the loss of self esteem that occurs due to an altered body image. If the ophthalmos is severe, treatment may involve suturing the eyelids together.

Information Education and Communication

- **Advise the patient with this condition to seek medical treatment when they start feeling these signs and symptoms like tachycardia, vomiting, stupor and heat intolerance for prompt treatment and prevent recurrence.**

- **Encourage him to wear a medical identification for easy identification in case the patient gets sick**

- **Encourage patient to carry his medication with him at all times if discharged on medication.**

Instruct the patient taking PIV (propylthioracil) and methimazole to take these drugs with meals to minimise gastric intestinal tract distress and to avoid over the counter cough preparations because many contain iodine.

Advise the patient taking antithyroid drugs or radioisotope therapy to identify and report symptoms of hyperthyroidism.

- **Advise the patient with exophthalmos or other exopthalmopathy to wear sunglasses to protect his eye from light.**

- **Advise the patient if he has severe lid retraction, warn him to avoid sudden physical movements that might cause the lid to slip behind the eyeball.**

Nursing Care Plan

Problems identified

- **Fatigue**
- **Weight loss**
- **Risk of corneal ulcer development**
- **Raised body temperature**
- **Anxiety**

PROBLEM	NURSING DIAGNOSIS	OBJECTIVE	NURSING INTERVENTION AND RATIONALE	EVALUATION
Fatigue Risk of corneal ulcer developme nt	Fatigue related to agitations and increased activity Risk of corneal ulcer development related to exophthalmos levels	Patient will have his/her fatigue controlled throughout Patient will have the development of corneal ulcers prevented throughout hospitalization	I will provide the patient with bed rest in order to promote rest I will nurse the patient in a quite environment in order to allow patient to rest I will restrict visitors in order to provide more time for the patient to rest I will nurse patient in a side ward in order to provide comfort and rest I will administer prescribed analgesics to relieve pain and promote comfort	**The patient has had fatigue controlled throughout hospitalizati on as evidenced by activity tolerance and verbalization .** **Patient is free from**

			I will cover patients eyes with a mask, tape or sunglasses to prevent drying of the corneal and conjunctiva	**corneal ulceration throughout hospitalisation.**

Now that we have discussed problems, continue practicing with the rest of the problems that is, weight loss, raised body temperature and anxiety.

Complications

- Thyroid storm (thyroid storm); an acute rare life threatening emergency condition in which all hyperthyroid manifestations are heightened
- Injury following surgery
- Secondary hypertension; as an attempt by the heart to pump more blood to hyperactive cells to meet their metabolic demands
- Others as per specific systems e.g. infertility, malnutrition
- Cardiac failure

Recurrent Hyperthyroidism

No treatments for thyrotoxicosis is without side effects, and all three treatments (radioactive iodine therapy, antithyroid medications, and surgery)have share the same complications: relapse or recurrent hyperthyroidism and permanent hypothyroidism.

The rate of relapse increases in patients who had very severe disease, a long history of dysfunction, ocular and cardiac symptoms, large goiter, and relapse after previous treatment. The relapse rate after radioactive iodine therapy depends on the dose used in treatment.

Patients receiving a lower dose of radioactive iodine are more likely to require subsequent treatment than those being treated with a higher dose. Hypothyroidism occurs in almost 80% of

patients at 1 year and in 90% to 100% by 5 years for both the multiple low-dose and single high-dose methods. Although rates of relapse and the occurrence of hypothyroidism vary, relapse with antithyroid medications is about 45% by 1 year after completion of therapy and almost 75% by 5 years later (Larson et al., 2000).

Discontinuation of antithyroid medications before therapy is complete usually results in relapse within 6 months in most patients. The incidence of relapse with subtotal thyroidectomy is 19% at 18 months; an incidence of hypothyroidism of 25% has been reported at 18 months after surgery. The risk for these complications illustrates the importance of long-term follow-up of patients treated for hyperthyroidism.

You have come to the end of this subunit that focused on management of thyroid disorders. In the next subunit you look at management of adrenal disorders.

SELF TEST

1. Hyperexcitability is a cardinal feature in
    a. Addison's disease
    b. Hyperthyroidism
    c. Hypothyroidism
    d. Neurosis
2. Methimazole is indicated in the treatment of
    a. Malaria
    b. Hyponeirosis
    c. Hyperthyroidism
    d. Cushing' Syndrome

Answers: Q1 B. Q2 C.

3.8 Management Of A Patient With Adrenal Disorders

The adrenal glands are small glands located on top of each kidney. They produce hormones such as the sex hormone and cortisol. Cortisol helps us to respond to stress. The adrenal medulla functions as part of the autonomic nervous system. Stimulation of preganglionic sympathetic nerve fibers, which travel directly to the cells of the adrenal medulla, causes release of the catecholamine hormones epinephrine and norepinephrine.

About 90% of the secretion of the human adrenal medulla is epinephrine (also called adrenaline). Catecholamines regulate metabolic pathways to promote catabolism of stored fuels to meet caloric needs from endogenous sources. The major effects of epinephrine release are to prepare to meet a challenge (fight-or-flight response).

Secretion of epinephrine causes decreased blood flow to tissues that are not needed in emergency situations, such as the gastrointestinal tract, and causes increased blood flow to tissues that are important for effective fight or flight, such as cardiac and skeletal muscle. Catecholamines also induce the release of free fatty acids, increase the basal metabolic rate, and elevate the blood glucose level.

Adrenal Cortex

A functioning adrenal cortex is necessary for life. Adrenocortica secretions make it possible for the body to adapt to stress of all kinds. The three types of steroid hormones produced by the adrenal cortex are:

- **glucocorticoids**, the prototype of which is hydrocortisone;
- **mineralocorticoids**, mainly aldosterone; and
- sex hormones, mainly **androgens** (male sex hormones).

Without the adrenal cortex, severe stress would cause peripheral circulatory failure, circulatory shock and prostration. Survival in the absence of a functioning adrenal cortex is possible only

with nutritional, electrolyte and fluid replacement and appropriate replacement with exogenous adrenocortical hormones.

Glucocorticoids

The glucocorticoids are so named because they have an important influence on glucose metabolism. Increased hydrocortisone secretion results in elevated blood glucose levels. However, the glucocorticoids have major effects on the metabolism of almost all organs of the body. Glucocorticoids are secreted from the adrenal cortex in response to the release of ACTH from the anterior lobe of the pituitary gland.

This system represents an example of negative feedback. The presence of glucocorticoids in the blood inhibits the release of corticotropin-releasing factor from the hypothalamus and also inhibits ACTH secretion from the pituitary. The resultant decrease in ACTH secretion causes diminished release of glucocorticoids from the adrenal cortex. Glucocorticoids (in the form of **corticosteroids**) are administered frequently to inhibit the inflammatory response to tissue injury and suppress allergic manifestations.

Their side effects include the development of diabetes mellitus, osteoporosis, peptic ulcer, increased protein breakdown resulting in muscle wasting and poor wound healing, and redistribution of body fat. Large amounts of exogenously administered glucocorticoids in the blood inhibit the release of ACTH and endogenous glucocorticoids. Because of this, the adrenal cortex can atrophy. If exogenous glucocorticoid administration is discontinued suddenly, adrenal insufficiency results because of the inability of the atrophied cortex to respond adequately.

Mineralocorticoids

Mineralocorticoids exert their major effects on electrolyte metabolism. They act principally on the renal tubular and gastrointestinal epithelium to cause increased sodium ion absorption in exchange for excretion of potassium or hydrogen ions. ACTH only minimally influences aldosterone secretion. It is primarily secreted in response to the presence of angiotensin II in the bloodstream. Angiotensin II is a substance that elevates the blood pressure by constricting

arterioles. Its concentration is increased when renin is released from the kidney in response to decreased perfusion pressure.

The resultant increased aldosterone levels promote sodium reabsorption by the kidney and the gastrointestinal tract, which tends to restore blood pressure to normal. The release of aldosterone is also increased by hyperkalemia. Aldosterone is the primary hormone for the long-term regulation of sodium balance.

Adrenal Sex Hormones (Androgens)

Androgens, the third major type of steroid hormones produced by the adrenal cortex, exert effects similar to those of male sex hormones. The adrenal gland may also secrete small amounts of some estrogens, or female sex hormones.

ACTH controls the secretion of adrenal androgens. When secreted in normal amounts, the adrenal androgens probably have little effect, but when secreted in excess, in certain inborn enzyme deficiencies, masculinization may result. This is known as the **adrenogenital syndrome**.

Pheochromocytoma

Pheochromocytoma is a tumour that is usually benign and originates from the chromaffin cells of the adrenal medulla. In 80% to 90% of patients the tumour arises in the medulla (O'Connell, 1999 in Brunner and Suddarth, 2010). In the remaining patients, it occurs in the extra-adrenal chromaffin tissue located in or near the aorta, ovaries, spleen, or other organs. Pheochromocytoma may occur at any age, but its peak incidence is between ages 40 and 50 years (Rakel & Bope, 2001 in Brunner and Suddarth, 2010). It affects men and women equally. Because of the high incidence of pheochromocytoma in family members, the patient's family members should be alerted and screened for this tumour. Ten percent of the tumours are bilateral, and 10% are malignant.

Pheochromocytoma is the cause of high blood pressure in 0.2% of patients with new onset of hypertension (O'Connell cited in Brunner and Suddarth, 2010). Although it is uncommon, it is

one form of hypertension that is usually cured by surgery. Without detection and treatment, it is usually fatal.

Pheochromocytoma may occur in the familial form as part of multiple endocrine neoplasia type 2. Therefore, it should be considered a possibility in patients with medullary thyroid carcinoma and parathyroid hyperplasia or tumour.

Clinical Manifestations

The nature and severity of symptoms of functioning tumours of the adrenal medulla depend on the relative proportions of epinephrine and norepinephrine secretion. The typical triad of symptoms comprises headache, diaphoresis, and palpitations (Matthews et al., 1999 in Brunner and Suddarth, 2010). Hypertension and other cardiovascular disturbances are common. The hypertension may be intermittent or persistent. Only half of the patients with pheochromocytoma, however, have sustained or persistent hypertension. If the hypertension is sustained, it may be difficult to distinguish from other causes of hypertension.

Other symptoms may include tremor, headache, flushing, and anxiety. Hyperglycaemia may result from conversion of liver and muscle glycogen to glucose by epinephrine secretion. Insulin may be required to maintain normal blood glucose levels. The clinical picture in the paroxysmal form of pheochromocytoma is usually characterized by acute, unpredictable attacks lasting seconds or several hours. During these attacks, the patient is extremely anxious, tremulous, and weak.

The patient may experience headache, vertigo, blurring of vision, tinnitus, air hunger, and dyspnoea. Other symptoms include polyuria, nausea, vomiting, diarrhoea, abdominal pain, and a feeling of impending doom. Palpitations and tachycardia are common.

Blood pressures exceeding 250/150 mm Hg have been recorded. Such blood pressure elevations are life-threatening and may cause severe complications, such as cardiac dysrhythmias, dissecting aneurysm, stroke, and acute renal failure. Postural hypotension occurs in 70% of patients with untreated pheochromocytoma.

Assessment and Diagnostic Findings

Pheochromocytoma is suspected if signs of sympathetic nervous system overactivity occur in association with marked elevation of blood pressure. These signs can be associated with the "five Hs":

- hypertension,
- headache,
- hyperhidrosis (excessive sweating),
- hypermetabolism, and
- hyperglycemia.

The presence of these signs has a 93.8% specificity and a 90.9% sensitivity for pheochromocytoma. Absence of hypertension excludes pheochromocytoma with a 99% certainty. Paroxysmal symptoms of pheochromocytoma commonly develop in the fifth decade of life. Measurements of urine and plasma levels of catecholamines are the most direct and conclusive tests for overactivity of the adrenal medulla. Measurements of urinary catecholamine metabolites (metanephrines [MN] and vanillylmandelic acid [VMA]) or free catecholamines are the standard diagnostic tests used in the diagnosis of pheochromocytoma. Levels can be as high as three times the normal limits (O'Connell cited in Brunner and Suddarth, 2010).

A 24-hour specimen of urine should be collected to determine free catecholamines, MN, and VMA. The use of combined tests increases the diagnostic accuracy of testing. A number of medications and foods (e.g., coffee, tea, bananas, chocolate, vanilla, aspirin) may alter the results of these tests. Therefore, careful instructions to avoid restricted items must be given to the patient.

Urine collected over a 2 or 3-hour period after an attack of hypertension can be assayed for catecholamine content. Total plasma catecholamine (epinephrine and norepinephrine) concentration is measured with the patient supine and at rest for 30 minutes. To prevent elevation of catecholamine levels by the stress of venipuncture, a butterfly needle, scalp vein needle, or venous catheter may be inserted 30 minutes before the blood specimen is obtained.

Factors that may elevate catecholamine levels must be controlled to obtain valid results. These factors include: consumption of coffee or tea, use of tobacco, emotional and physical stress, and use of many prescription and over-the-counter medications (e.g., amphetamines, nose drops or sprays, decongestant agents, and bronchodilators).

Normal plasma values of epinephrine are 100 pg/mL (590 pmol/L); normal values of norepinephrine are generally less than 100 to 550 pg/mL (590 to 3,240 pmol/L). Values of epinephrine greater than 400 pg/mL (2,180 pmol/L) or norepinephrine values greater than 2,000 pg/mL (11,800 pmol/L) are considered diagnostic of pheochromocytoma. Values that fall between normal values and those diagnostic of pheochromocytoma indicate the need for further testing.

A clonidine suppression test may be performed if the results of plasma and urine tests of catecholamines are inconclusive. Clonidine (Catapres) is a centrally acting, antiadrenergic medication that suppresses the release of neurogenically mediated catecholamines. The suppression test is based on the principle that catecholamine levels are normally increased through the activity of the sympathetic nervous system. In pheochromocytoma, increased catecholamine levels result from the diffusion of excess catecholamines into the circulation, bypassing normal storage and release mechanisms. Therefore, in patients with pheochromocytoma, clonidine does not suppress the release of catecholamines. The results of the test are considered normal if 2 to 3 hours after a single oral dose of clonidine, the total plasma catecholamine value decreases by at least 40% from baseline. Patients with pheochromocytoma exhibit no change in catecholamine levels. False-positive results, however, may occur in patients with primary hypertension.

Imaging studies, such as CT scans, MRI, and ultrasound, may also be carried out to localize the pheochromocytoma and to determine whether more than one tumour is present.

Other diagnostic studies may focus on evaluating the function of other endocrine glands because of the association of pheochromocytoma in some patients with other endocrine tumours.

Medical Management

During an episode or attack of hypertension, tachycardia, anxiety, and the other symptoms of pheochromocytoma, the patient is placed on bed rest with the head of the bed elevated to promote an orthostatic decrease in blood pressure.

Pharmacologic Therapy

The patient may be moved to the intensive care unit for close monitoring of ECG changes and careful administration of alphaadrenergic blocking agents (e.g., phentolamine [Regitine]) or smooth muscle relaxants (e.g., sodium nitroprusside [Nipride]) to lower the blood pressure quickly. Other drugs include:

- Phenoxybenzamine (Dibenzyline): a long-acting alpha-blocker used when the blood pressure is stable to prepare the patient for surgery.

- Beta-adrenergic blocking agents, such as propranolol (Inderal): used in patients with cardiac dysrhythmias or those not responsive to alpha-blockers.

- Alphaadrenergic and beta-adrenergic blocking agents: used with caution because patients with pheochromocytoma may have increased sensitivity to them.

- Catecholamine synthesis inhibitors, such as alpha-methyl-p-tyrosine (metyrosine). These are occasionally used when adrenergic blocking agents do not reduce the effects of catecholamines.

Surgical Management

The definitive treatment of pheochromocytoma is surgical removal of the tumour, usually with **adrenalectomy**. Bilateral adrenalectomy may be necessary if tumours are present in both adrenal glands. Patient preparation includes control of blood pressure and blood volumes, usually carried out over a 7 to 10 days period. Phentolamine or phenoxybenzamine

(Dibenzyline) may be used safely without causing undue hypotension. Other medications (metyrosine [Demser] and prazosin [Minipress]) are used to treat pheochromocytoma.

The patient needs to be well hydrated before, during, and after surgery to prevent hypotension. Manipulation of the tumour during surgical excision may cause release of stored epinephrine and norepinephrine, with marked increases in blood pressure and changes in heart rate. Therefore, use of sodium nitroprusside (Nipride) and alpha-adrenergic blocking agents may be required during and after surgery. Exploration of other possible tumour sites is frequently undertaken to ensure removal of all tumour tissue. As a result, the patient is subject to the stress and effects of a long surgical procedure, which may increase the risk of hypertension postoperatively.

Corticosteroid replacement is required if bilateral adrenalectomy has been necessary. Corticosteroids may also be necessary for the first few days or weeks after removal of a single adrenal gland. Intravenous administration of corticosteroids (methylprednisolone sodium succinate [Solu-Medrol]) may begin the evening before surgery and continue during the early postoperative period to prevent adrenal insufficiency. Oral preparations of corticosteroids (prednisone) are prescribed after the acute stress of surgery diminishes.

Hypotension and hypoglycemia may occur in the postoperative period because of the sudden withdrawal of excessive amounts of catecholamines. Therefore, careful attention is directed toward monitoring and treating these changes. Blood pressure is expected to return to normal with treatment, however, one third of patients continue to be hypertensive after surgery. This may result if not all pheochromocytoma tissue was removed, if pheochromocytoma recurs, or if the blood vessels were damaged by severe and prolonged hypertension. Several days after surgery, urine and plasma levels of catecholamines and their metabolites are measured to determine whether surgery was successful.

Nursing Management

The patient who has undergone surgery to treat pheochromocytoma experiences a stressful preoperative and postoperative course and may remain fearful of repeated attacks. Although it is usually expected that all pheochromocytoma tissue has been removed, there is a possibility that

other sites were undetected and that an attack may recur. The patient is monitored for several days in the intensive care unit with special attention given to ECG changes, arterial pressures, fluid and electrolyte balance, and blood glucose levels. Several intravenous lines are inserted for administration of fluids and medications.

Promoting Home And Community-Based Care

Teaching Patients Self-Care. During the preoperative and postoperative phases of care, the nurse informs the patient about the importance of follow-up monitoring to ensure that pheochromocytoma does not recur undetected. After adrenalectomy, use of corticosteroids may be needed. Therefore, the nurse instructs the patient about their purpose, the medication schedule, and the risks of skipping doses or stopping their administration abruptly.

It is important to teach the patient and family how to measure the patient's blood pressure and when to notify the physician about changes in blood pressure. Additionally, the nurse provides verbal and written instructions about the procedure for collecting 24-hour urine specimens to monitor urine catecholamine levels.

Continuing Care. A follow-up visit from a home care nurse may be indicated to assess the patient's postoperative recovery, surgical incision, and compliance with the medication schedule. This may help to reinforce previous teaching about management and monitoring. The home care nurse also obtains blood pressure measurements and assists the patient in preventing or dealing with problems that may result from long-term use of corticosteroids.

Because of the risk of recurrence of hypertension, periodic checkups are required, especially in young patients and in patients whose families have a history of pheochromocytoma. The patient is scheduled for periodic follow-up appointments to observe for return of normal blood pressure and plasma and urine levels of catecholamines.

Having looked pheochromocytoma, our next topic will be Addison's disease

ADRENOCORTICAL INSUFFICIENCY (ADDISON'S DISEASE)

Pathophysiology

Addison's disease, or adrenocortical insufficiency, results when adrenal cortex function is inadequate to meet the patient's need for cortical hormones.

Autoimmune or idiopathic atrophy of the adrenal glands is responsible for 80% of cases (Rakel & Bope, 2001). Other causes include surgical removal of both adrenal glands or infection of the adrenal glands. Tuberculosis and histoplasmosis are the most common infections that destroy adrenal gland tissue. Although autoimmune destruction has replaced tuberculosis as the principal cause of Addison's disease, tuberculosis should be considered in the diagnostic workup because of its increasing incidence. Inadequate secretion of ACTH from the pituitary gland also results in adrenal insufficiency because of decreased stimulation of the adrenal cortex. Therapeutic use of corticosteroids is the most common cause of adrenocortical insufficiency (Coursin & Wood, 2002). The symptoms of adrenocortical insufficiency may also result from the sudden cessation of exogenous adrenocortical hormonal therapy, which suppresses the body's normal response to stress and interferes with normal feedback mechanisms. Treatment with daily administration of corticosteroids for 2 to 4 weeks may suppress function of the adrenal cortex; therefore, adrenal insufficiency should be considered in any patient who has been treated with corticosteroids.

Clinical Manifestations

Addison's disease is characterized by muscle weakness, anorexia, gastrointestinal symptoms, fatigue, emaciation, dark pigmentation of the skin, knuckles, knees, elbows, and mucous membranes, hypotension, and low blood glucose levels, low serum sodium levels, and high serum potassium levels. Mental status changes such as depression, emotional lability, apathy, and confusion are present in 60% to 80% of patients. In severe cases, the disturbance of sodium and potassium metabolism may be marked by depletion of sodium and water and severe, chronic dehydration. With disease progression and acute hypotension, the patient develops **addisonian crisis**, which is characterized by cyanosis and the classic signs of circulatory shock: pallor, apprehension, rapid and weak pulse, rapid respirations, and low blood pressure. In addition, the patient may complain of headache, nausea, abdominal pain, and diarrhea and show signs of confusion and restlessness. Even slight overexertion, exposure to cold, acute infections, or a decrease in salt intake may lead to circulatory collapse, shock, and death if untreated. The stress of surgery or dehydration resulting from preparation for diagnostic tests or surgery may precipitate an addisonian or hypotensive crisis.

Assessment and Diagnostic Findings

Although the clinical manifestations presented appear specific, the onset of Addison's disease usually occurs with nonspecific symptoms.

Diagnosis

The diagnosis is confirmed by laboratory test results.

Laboratory findings include:

Decreased blood glucose (hypoglycemia) and sodium (hyponatremia) levels,

An increased serum potassium (hyperkalemia) level, and

An increased white blood cell count (leukocytosis).

The diagnosis is confirmed by low levels of adrenocortical hormones in the blood or urine and decreased serum cortisol levels.

If the adrenal cortex is destroyed, baseline values are low, and ACTH administration fails to cause the normal rise in plasma cortisol and urinary 17-hydroxycorticosteroids. If the adrenal gland is normal but not stimulated properly by the pituitary, a normal response to repeated doses of exogenous ACTH is seen, but no response follows the administration of metyrapone, which stimulates endogenous ACTH.

Medical Management

Immediate treatment is directed toward combating circulatory shock: restoring blood circulation, administering fluids and corticosteroids, monitoring vital signs, and placing the patient in a recumbent position with the legs elevated. Hydrocortisone (Solu-Cortef) is administered intravenously, followed with 5% dextrose in normal saline. Vasopressor amines may be required if hypotension persists.

Drugs

Antibiotics may be administered if infection has precipitated adrenal crisis in a patient with chronic adrenal insufficiency. Additionally, the patient is assessed closely to identify other factors, stressors, or illnesses that led to the acute episode.

Fluids

Oral intake may be initiated as soon as tolerated. Gradually, intravenous fluids are decreased when oral fluid intake is adequate to prevent hypovolemia. If the adrenal gland does not regain function, the patient needs lifelong replacement of corticosteroids and mineralocorticoids to prevent recurrence of adrenal insufficiency. The patient will require additional supplementary

therapy with glucocorticoids during stressful procedures or significant illnesses to prevent addisonian crisis (Coursin & Wood, 2002 in Brunner and Suddarth, 2010).

Diet

Additionally, the patient may need to supplement dietary intake with added salt during times of gastrointestinal losses of fluids through vomiting and diarrhoea.

Nursing Management

Assessing The Patient

History

The health history and examination focus on the presence of symptoms of fluid imbalance and on the patient's level of stress. To detect inadequate fluid volume, the nurse monitors the blood pressure and pulse rate as the patient moves from a lying to a standing position.

Physical examination

The nurse assesses the skin colour and turgor for changes related to chronic adrenal insufficiency and hypovolaemia. Other key assessments include checking for weight changes, muscle weakness, and fatigue and investigating any illness or stress that may have precipitated the acute crisis.

Monitoring and Managing Addisonian Crisis

The patient at risk is monitored for signs and symptoms indicative of addisonian crisis. These symptoms are often the manifestations of shock: hypotension; rapid, weak pulse; rapid respiratory rate; pallor; and extreme weakness.

The patient with addisonian crisis is at risk for circulatory collapse and shock (Management of the patient is as for shock); therefore, physical and psychological stressors must be avoided. These include exposure to cold, overexertion, infection, and emotional distress.The patient with addisonian crisis requires immediate treatment with intravenous administration of fluid, glucose, and electrolytes, especially sodium; replacement of missing steroid hormones; and vasopressors. During acute addisonian crisis, the patient must avoid exertion; therefore, the nurse anticipates the patient's needs and takes measures to meet them.

Observations

Careful monitoring of symptoms, vital signs, weight, and fluidand electrolyte status is essential to monitor the patient's progress and return to a pre-crisis state. To reduce the risk of future

episodes of addisonian crisis, efforts are made to identify and reduce the factors that may have led to the crisis.

Fluid balance

To provide information about fluid balance and the adequacy of hormone replacement, the nurse assesses the patient's skin turgor, mucous membranes, and weight while instructing the patient to report increased thirst, which may indicate impending fluid imbalance. Lying, sitting, and standing blood pressures also provide information about fluid status. A decrease in systolic pressure (20 mm Hg or more) may indicate depletion of fluid volume, especially if accompanied by symptoms. The nurse encourages the patient to consume foods and fluids that will assist in restoring and maintaining fluid and electrolyte balance; along with the dietitian, the nurse assists the patient to select foods high in sodium during gastrointestinal disturbances and very hot weather. The nurse instructs the patient and family to administer hormone replacement as prescribed and to modify the dosage during illness and other stressful occasions. Written and verbal instructions are provided about the administration of mineralocorticoid (Florinef) or corticosteroid (prednisone) as prescribed.

Activities of daily living

Until the patient's condition is stabilized, the nurse takes precautions to avoid unnecessary activity and stress that could precipitate another hypotensive episode. Efforts are made to detect signs of infection or the presence of other stressors. Even minor events or stressors may be excessive in patients with adrenal insufficiency. During the acute crisis, the nurse maintains a quiet, nonstressful environment and performs all activities (eg, bathing, turning) for the patient. Explaining all procedures to the patient and family will reduce their anxiety. Explaining the rationale for minimizing stress during the acute crisis assists the patient to increase activity gradually.

Home based care

Teaching Patients Self-Care. Because of the need for lifelong replacement of adrenal cortex hormones to prevent addisonian crises, the patient and family members receive explicit verbal and written instructions about the rationale for replacement therapy and proper dosage. Additionally, they are instructed about how to modify the medication dosage and increase salt intake in times of illness, very hot weather, and other stressful situations. The patient also learns how to modify diet and fluid intake to help maintain fluid and electrolyte balance. The patient

and family are frequently prescribed preloaded, single-injection syringes of corticosteroid for use in emergencies. Careful instructions about how and when to use the injection are also provided. It is important to instruct the patient to inform other health care providers, such as dentists, about the use of corticosteroids, to wear a medical alert bracelet, and to carry information at all times about the need for corticosteroids. If the patient with Addison's disease requires surgery, careful administration of fluids and corticosteroids is necessary before, during, and after surgery to prevent addisonian crisis.

The patient and family need to know the signs of excessive or insufficient hormone replacement. The development of edema or weight gain may signify too high a dose of hormone; postural hypotension (decrease in systolic blood pressure, light headedness, dizziness on standing) and weight loss frequently signify too low a dose.

Continuing Care. Although most patients can return to their job and family responsibilities soon after hospital discharge, others cannot do so because of concurrent illnesses or incomplete recovery from the episode of adrenal insufficiency. In these circumstances,

a referral for home care enables the home care nurse to assess the patient's recovery, monitor hormone replacement, and evaluate stress in the home. The nurse assesses the patient's and family's knowledge about medication therapy and dietary modifications. A home visit also allows the nurse to assess the patient's plans for follow-up visits to the clinic or physician's office. The nurse reminds the patient and family about the importance of participating in health promotion activities and health screening.

Addison's disease has been covered and the next topic of discussion will be Cushing's Syndromes

CUSHING'S SYNDROME (ADRENAL INSUFFICIENCY)

Cushing's syndrome results from excessive, rather than deficient, adrenocortical activity. The syndrome may result from excessive administration of corticosteroids or ACTH or from hyperplasia of the adrenal cortex.

Cause and the Pathophysiology

Cushing's syndrome is commonly caused by use of corticosteroid medications and is infrequently due to excessive corticosteroid production by the adrenal cortex (Tierney et al., 2001). However, overproduction of endogenous corticosteroids may be caused by
several mechanisms, Including a tumor of the pituitary gland that produces ACTH and stimulates the adrenal cortex to increase its hormone secretion despite adequate amounts being produced.

Primary hyperplasia of the adrenal glands in the absence of a pituitary tumor is less common. Another less common cause of Cushing's syndrome is the ectopic production of ACTH by malignancies; bronchogenic carcinoma is the most common type of these malignancies. Regardless of the cause, the normal feedback mechanisms that control the function of the adrenal cortex become ineffective, and the usual diurnal pattern of cortisol is lost. The signs and symptoms of Cushing's syndrome are primarily a result of over secretion of glucocorticoids and androgens (sex hormones), although mineralocorticoid secretion also may be affected.

Clinical Manifestations
When overproduction of the adrenal cortical hormone occurs;
arrest of growth,
obesity, and musculoskeletal changes occur along with glucose intolerance. The classic picture of Cushing's syndrome in the adult is that of central-type obesity, with a fatty "buffalo hump" in the neck and supraclavicular areas, a heavy trunk, and relatively thin extremities.
The skin is thin, fragile, and easily traumatized;

- Ecchymoses (bruises) and striae develop.
- The patient complains of weakness and lassitude. Sleep is disturbed because of altered diurnal secretion of cortisol. Excessive protein catabolism occurs, producing muscle wasting and osteoporosis. Kyphosis, backache, and compression fractures of the vertebrae may result. Retention of sodium and water occurs as a result of increased mineralocorticoid activity, producing hypertension and heart failure. The patient develops a "moon-faced" appearance and may experience increased oiliness of the skin and acne. There is increased susceptibility to infection. Hyperglycaemia or overt diabetes may develop.
- The patient may also report weight gain, slow healing of minor cuts, and bruises.

- Women ages 20 to 40 years are five times more likely than men to develop Cushing's syndrome.
- In females of all ages, virilization may occur as a result of excess androgens.
- Virilization is characterized by the appearance of masculine traits and the recession of feminine traits.
- There is an excessive growth of hair on the face (hirsutism), the breasts atrophy, menses cease, the clitoris enlarges, and the voice deepens.
- Libido is lost in men and women.
- Changes occur in mood and mental activity;
- Psychosis may develop.
- Distress and depression are common and are increased by the severity of the physical changes that occur with this syndrome.

If Cushing's syndrome is a consequence of pituitary tumor, visual disturbances may occur because of pressure of the growing tumor on the optic chiasm.

Assessment and Diagnostic Findings

Indicators of Cushing's syndrome include an increase in serum sodium and blood glucose levels and a decreased serum concentration of potassium, a reduction in the number of blood eosinophils, and disappearance of lymphoid tissue. Measurements of plasma and urinary cortisol levels are obtained. Several blood samples may be collected to determine whether the normal diurnal variation in plasma levels is present; this variation is frequently absent in adrenal dysfunction. If several blood samples are required, they must be collected at the times specified and the time of collection must be noted on the requisition slip. An overnight dexamethasone suppression test is the most widely used screening test for diagnosis of pituitary and adrenal causes of Cushing's syndrome. It can be performed on an outpatient basis. Dexamethasone (1 mg) is administered orally at 11 pm, and a plasma cortisol level is obtained at 8 the next morning. Suppression of cortisol to less than 5 mg/dL indicates that the hypothalamic-pituitary-adrenal axis is functioning properly. Stress, obesity, depression, and medications such as antiseizure agents, oestrogen, and rifampin can falsely elevate cortisol levels. Other diagnostic studies include a 24-hour urinary free cortisol level and a high-dose or low-dose dexamethasone suppression test. High-dose and low-dose suppression tests are similar to the overnight test but

vary in dosage and timing. Measurement of plasma ACTH by radioimmunoassay is used in conjunction with the high-dose suppression test to distinguish pituitary tumors from ectopic sites of ACTH production as the cause of Cushing's syndrome. Elevation of both ACTH and cortisol level indicates pituitary or hypothalamic disease. Low ACTH with a high cortisol level indicates adrenal disease. A CT scan, ultrasound, or MRI may be performed to localize adrenal tissue and detect tumors of the adrenal gland.

Medical Management

If Cushing's syndrome is caused by pituitary tumors rather than tumors of the adrenal cortex, treatment is directed at the pituitary gland. Surgical removal of the tumor by transsphenoidal hypophysectomy (see Chap. 61) is the treatment of choice and has a 90% success rate (Rakel & Bope, 2001). Radiation of the pituitary gland also has been successful, although it may take several months for control of symptoms. Adrenalectomy is the treatment of choice in patients with primary adrenal hypertrophy. Postoperatively, symptoms of adrenal insufficiency may begin to appear 12 to 48 hours after surgery because of reduction of the high levels of circulating adrenal hormones. Temporary replacement therapy with hydrocortisone may be necessary for several months until the adrenal glands begin to respond normally to the body's needs. If both adrenal glands have been removed (bilateral adrenalectomy), lifetime replacement of adrenal cortex hormones is necessary. Adrenal enzyme inhibitors (e.g., metyrapone, aminoglutethimide, mitotane, ketoconazole) may be used to reduce hyperadrenalism if the syndrome is caused by ectopic ACTH secretion by a tumour that cannot be eradicated. Close monitoring is necessary because symptoms of inadequate adrenal function may result and because of possible side effects of these medications. If Cushing's syndrome is a result of the administration of corticosteroids, an attempt is made to reduce or taper the medication to the minimum dosage needed to treat the underlying disease process (e.g., autoimmune and allergic diseases and rejection of transplanted organs). Frequently, alternate-day therapy decreases the symptoms of Cushing's syndrome and allows recovery of the adrenal glands' responsiveness to ACTH.

Nursing Process: The Patient With Cushing's Syndrome

Assessment

The health history and examination focus on the effects on the body of high concentrations of adrenal cortex hormones and on the inability of the adrenal cortex to respond to changes in cortisol and aldosterone levels. The history includes information about the patient's level of activity and ability to carry out routine and self-care activities. The skin is observed and assessed for trauma, infection, breakdown, bruising, and edema. Changes in physical appearance are noted, and the patient's responses to these changes are elicited. The nurse assesses the patient's mental function, including mood, responses to questions, awareness of environment, and level of depression. The family is often a good source of information about gradual changes in the patient's physical appearance as well as emotional status.

Clinical Manifestations of Cushing's Syndrome

- ***Ophthalmic* :** ataracts, laucoma
- ***Cardiovascular*:** ypertension, Heart failure
- ***Endocrine/Metabolic*:** runcal obesity, Moon face, Buffalo hump, Sodium retention, Hypokalemia, Metabolic alkalosis, Hyperglycemia, Menstrual irregularities, Impotence,
- Negative nitrogen balance, Altered calcium metabolism, Adrenal suppression,
- ***Immune Function*:** ecreased inflammatory, responses, Impaired wound healing, Increased susceptibility to infections
- ***Skeletal*:**steoporosis, Spontaneous fractures, Aseptic necrosis of femur, Vertebral compression, fractures
- ***Gastrointestinal*:** Peptic ulcer, Pancreatitis
- ***Muscular*:** Myopathy,Muscle weakness
- ***Dermatologic*:** Thinning of skin, Petechiae, Ecchymoses, Striae, Acne
- ***Psychiatric*:** Mood alterations, Psychoses

Diagnosis

Nursing Diagnoses

Based on all the assessment data, the major nursing diagnoses of the patient with Cushing's syndrome include the following:

• Risk for injury related to weakness

• Risk for infection related to altered protein metabolism and inflammatory response

• Self-care deficit related to weakness, fatigue, muscle wasting, and altered sleep patterns

• Impaired skin integrity related to oedema, impaired healing, and thin and fragile skin
• Disturbed body image related to altered physical appearance, impaired sexual functioning, and decreased activity level
• Disturbed thought processes related to mood swings, irritability, and depression

Collaborative Problems/Potential Complications

Based on assessment data, potential complications may include the following:
• Addisonian crisis
• Adverse effects of adrenocortical activity

Planning and Goals

The major goals for the patient include decreased risk for injury,decreased risk for infection, increased ability to carry out self-care activities, improved skin integrity, improved body image, improved mental function, and absence of complications.

Nursing Interventions

Decreasing Risk For Injury

Establishing a protective environment will help to prevent falls, fractures, and other injuries to bones and soft tissues. The patient who is very weak may require assistance from the nurse in ambulating to prevent falls or bumping into sharp corners of furniture. Foods high in protein, calcium, and vitamin D are arerecommended to minimize muscle wasting and osteoporosis. Referral to a dietitian may assist the patient in selecting appropriate foods that are also low in sodium and calories.

Decreasing Risk For Infection

The patient should avoid unnecessary exposure to others with infections. The nurse frequently assesses the patient for subtle signs of infection because the anti-inflammatory effects of corticosteroids may mask the common signs of inflammation and infection.

Preparing the Patient For Surgery

The patient is prepared for adrenalectomy, if indicated, and the postoperative course (see later discussion in this chapter). If Cushing's syndrome is a result of a pituitary tumor, a transsphenoidal hypophysectomy may be performed . Diabetes mellitus and peptic ulcer are common in the patient with Cushing's syndrome. Therefore, insulin therapy and medication to

treat peptic ulcer may be initiated if needed. Before, during, and after surgery, blood glucose monitoring and assessment of stools for blood are carried out to monitor for appropriate intervention. If the patient has other symptoms of Cushing's syndrome, these are considered in the preoperative preparation. For example, if the patient has experienced weight gain, special instruction is given about postoperative breathing exercises.

Encouraging Rest And Activity

Weakness, fatigue, and muscle wasting make it difficult for the patient with Cushing's syndrome to carry out normal activities. Yet the nurse should encourage moderate activity to prevent complications of immobility and promote increased self-esteem. Insomnia often contributes to the patient's fatigue. It is important to help the patient plan and space rest periods throughout the day. Efforts are made to promote a relaxing, quiet environment for rest and sleep.

Promoting Skin Integrity

Meticulous skin care is necessary to avoid traumatizing the patient's fragile skin. Use of adhesive tape is avoided because it can irritate the skin and tear the fragile tissue when the tape is removed. The nurse frequently assess the skin and bony prominences and encourages and assists the patient to change positions frequently to prevent skin breakdown.

Improving Body Image

If the cause of Cushing's syndrome can be treated successfully, the major physical changes disappear in time. The patient may benefit from discussion of the effect the changes have had on his or her self-concept and relationships with others. Weight gain and oedema may be modified by a low-carbohydrate, low-sodium diet, and a high-protein intake may reduce some of the other bothersome symptoms.

Improving Thought Processes

Explanations to the patient and family members about the cause of emotional instability are important in helping them cope with the mood swings, irritability, and depression that may occur. Psychotic behaviour may occur in a few patients and should be reported. The nurse encourages the patient and family members to verbalize their feelings and concerns.

Monitoring And Managing/Potential Complications

Addisonian Crisis

The patient with Cushing's syndrome whose symptoms are treated by withdrawing corticosteroids, by adrenalectomy, or by removing a pituitary tumour is at risk for adrenal

hypofunction and addisonian crisis. If high levels of circulating adrenal hormones have suppressed the function of the adrenal cortex, atrophy of the adrenal cortex is likely. If the circulating hormone level is decreased rapidly because of surgery or by abruptly stopping corticosteroid agents, manifestations of adrenal hypofunction and addisonian crisis may develop. Therefore, the patient with Cushing's syndrome is monitored closely for hypotension; rapid, weak pulse; rapid respiratory rate; pallor; and extreme weakness. Efforts are made to identify factors that may have led to the crisis. The patient with Cushing's syndrome who experiences highly stressful events, such as trauma or emergency surgery, is at increased risk for addisonian crisis because of long-term suppression of the adrenal cortex. The patient may require intravenous administration of fluid and electrolytes and corticosteroids before, during, and after treatment or surgery. If addisonian crisis occurs, the patient is treated for circulatory collapse and shock.

Adverse Effects of Adrenocortical Activity

The nurse assesses fluid and electrolyte status by monitoring laboratory values and daily weights. Because of the increased risk for glucose intolerance and hyperglycaemia, blood glucose monitoring is initiated. The nurse reports elevated blood glucose levels to the physician so that treatment can be prescribed if indicated.

Promoting Home And Community-Based Care

Teaching Patients Self-Care

The patient with Cushing's syndrome and the patient's family require teaching and support to enable them to prevent problems associated with the syndrome and to manage those that cannot be prevented. The nurse presents information verbally and in writing. If the disorder is a result of corticosteroid use for treatment of a chronic disease, the patient and family need to understand that stopping the corticosteroid use abruptly and without medical supervision is likely to result in acute adrenal insufficiency and reappearance of the underlying symptoms of the chronic disease. The nurse emphasizes the need to ensure an adequate supply of the corticosteroid, because running out of the medication and skipping doses can precipitate addisonian crisis. Refer to the later discussion, Therapeutic Uses of Corticosteroids, for more information. The nurse stresses the need for dietary modifications to ensure adequate calcium intake without increasing the risk for hypertension, hyperglycaemia, and weight gain. The patient and family may be taught to monitor blood pressure, blood glucose levels, and weight. Wearing a medical alert bracelet and

notifying other health providers (e.g. dentist) are important to alert others that the patient has Cushing's syndrome.

Continuing Care

The need for follow-up depends on the origin and duration of the disease and its management. The patient who has been treated by adrenalectomy or removal of a pituitary tumour requires close monitoring to ensure that adrenal function has returned to normal and to ensure adequacy of circulating adrenal hormones. The patient who requires continued corticosteroid therapy is monitored to ensure understanding of the medications and the need for a dosage that treats the underlying disorder while minimizing the side effects. Home care referral may be indicated to ensure a safe environment that minimizes stress and risk for falls and other

side effects. The home care nurse assesses the patient's physical and psychological status and reports changes to the physician. The nurse also assesses the patient's understanding of the medication regimen and the patient's compliance with the regimen, and reinforces previous teaching about the medications and the importance of taking them as prescribed. The nurse emphasizes the importance of regular medical follow-up, the side effects and toxic effects of medications, and the need to wear medical identification with Addison's and Cushing's disease. Additionally, the nurse reminds the patient and family about the importance of health promotion activities and recommended health screening, including bone mineral density testing

3.3.SELF TEST

1. Pheochromocytoma increases the production of
    a. Catecholamines
    b. Dopamine
    c. Leutinizing hormone
    d. ADH

Hydrocortisone can be used to treat

    a. Schistosomiasis
    b. Addison's disease
    c. Leprosy
    d. Diabetes

Answers: Q1 A. Q2 B.

3.9 Summary

We have now come to the end of unit 3. We started out by first reviewing the anatomy and physiology of the endocrine system and we proceeded to look at the roles of the nurse in carrying out various investigative and diagnostic procedures in arriving at diagnosis for patients suffering from endocrine disorders.

We went a step further and dealt with conditions that arise from each specific endocrine gland namely; the disorders of metabolism such diabetes, disorders of the pancreas such as pancreatitis, disorders of the pituitary gland such as hypopituitarism, thyroid disorders which included thyroiditis, thyrotoxicosis etc.. and finally we ended by looking at the disorders of the adrenal gland such as pheochromocytoma.

I hope you learnt something and that you will apply this knowledge in managing your patient. In the next discussion will move to unit, which is the last unit of Medicine II, in this unit we shall focus our attention on HIV/AIDS and how to manage a patient suffering from HIV/AIDS.

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UNIT 4: HIV AND AIDS AND ANTIRETROVIRAL THERAPY (ART)

4.1 Introduction

HIV is one disease that affects millions of lives in the world and many are infected are infected and Zambia has not been spared from this scourge killing the youth generation. Statistics indicate that the HIV prevalence in Zambia stand at about 14.2% (CSO, 2010).

AIDS is the result of HIV infection, were there is complete immune breakdown and the patient reports attacks of various conditions/diseases. The introduction of ARV's is critical in prolonging the lives of those suffering from HIV/AIDS and improve their quality of life. Zambia has a robust programme of giving ARV's to People Living with HIV. Zambia has also implemented prevention of mother to child transmission of HIV and finally there is also in place prophylactic treatment protocol for those exposed to HIV accidently.

4.2 Objectives

At the end of this unit the student should be able to

1. Discuss HIV/AIDS information and management
2. Explain the goals and general principles of ARV therapy
3. Apply knowledge and information on antiretroviral drugs
4. Apply knowledge and skill in counselling and education to the patient HIV/AIDs
5. Describe assessment of patient with HIV/AIDs
6. Apply knowledge and skill in initiating of ARV therapy
7. Describe the management of a patient on ART
8. Explain the management of HIV/AIDs in special population.
9. Discuss the Post exposure prophylaxis
10. Discuss the record keeping, monitoring and evaluation of HIV and AIDS

4.3 HIV/AIDS INFORMATION AND MANAGEMENT

4.3.1 Definition of terms

HIV stands for Human Immuno-deficiency Virus which causes the disease AIDS lowering functioning of the Immune System

AIDS (Acquired Immune Deficiency Syndrome) is when the HIV infection is so massive that it destroys a large number of the immune system processes and leads to the patient having multiple opportunistic infections resulting in illness e.g chronic diarrhoea, persistent fever, weight loss, anaemia, a typical rash.

ART(**Antiretroviral Therapy)**: Management of the HIV disease including provision of ARVsAntiretroviral therapy.The drugs that specifically attack the HIV virus.

Highly Active Antiretroviral Therapy (HAART): antiretroviral regimens that use effective combinations of three or more agents, usually from two or more drug classes in order to achieve the greatest suppression of viral load for the most sustained period of time.
Antiretroviral (ARV) therapy Treatment with drugs that specifically attack the HIV virus.
Check your understanding here.

Self test: Cross match the following:

1. HIV	a. acquired immune deficiency syndrome
2. AIDS	b. world health organisation
3. ART	c. highly active anti-retroviral therapy
4. HAART	d. anteretrovirals
5. ARVS	e. ante retroviral therapy
6. WHO	f. human immunal virus

4.3.2 Modes of spread

HIV is transmitted primarily through exposure to HIV infected blood or exchange of HIV containing bodily fluids. The 3 primary modes of transmission are:

Blood-to-blood transmission. This is through:
Transfusion of HIV-infected blood or direct contact with the blood;
Exposure to HIV-contaminated needles, syringes, and other equipment;
Donated organs;
Traditional procedures involving scarification;
Sexual contact:

unprotected vaginal, oral, or anal intercourse;

direct contact with HIV-infected body fluids such as semen and cervical and vaginal secretions.

Perinatal transmission:

Mother to child transmission of HIV during pregnancy, labor and delivery

Mother to child transmission of HIV during breast-feeding

Checkpoint Questions. Answer whether the statements are true or false

Organ transplant can transmit HIV a. True b. False

Blood transfusion can transmit HIV a. Yes b. No

Contact with infected equipment and materials can spread HIV a. Yes b. No

Answers: 1)B; 2)A; 3)A

Now let us proceed to look at the viral replication of the HIV virus.

4.3.3 Viral Replication Stages of HIV Virus

This sub unit will make you understand the replication process and survival of the virus.

it begins the infection process of a susceptible host cell by binding or attaching itself to the CD4 receptor of the host cell, using the CCR5 and CXCR4 markers on the surface of the CD4 cell. CD4 is present on the surface of many lymphocytes, which are a critical part of the body's immune system.

Once attachment takes place, the membranes of the virus and host cell are fused and an open channel is established through which the viral core can enter into the host cell. Once the viral core enters the host cells, the viral core capsid is dissolved to release free viral RNA and viral enzymes. The viral enzymes released include RT, integrase, protease and RNAse-H.

Once the viral RNA and viral enzymes are released into the host cytoplasm, the single stranded viral RNA is converted to a double stranded DNA. This conversion is catalyzed by the enzyme *reverse transcriptase*.

Once the genetic material of HIV has been changed into DNA, this viral DNA enters the host cell nucleus where it can be integrated into the genetic material of the cell. The enzyme *integrase* catalyzes this process. Once the viral DNA is integrated into the genetic material of the host, HIV may persist in a latent state for many years. This ability of HIV to persist in certain latently infected cells is the major barrier to eradication or cure of HIV. That is why patients must remain on antiretroviral therapy for life.

After integration, the HIV virus turns the host cell into a 'machine' for producing more viruses. Synthesis of viral messenger RNA (mRNA) occurs. mRNA leaves the nucleus as complex multi-protein molecules and enters the cytoplasm where it is cleaved by the *protease* enzyme to produces less complex viral proteins (core, envelope, and regulatory proteins) and enzymes essential for HIV replication. The cleaved 'simple' proteins then assemble into functional groups at the cell membrane, and bud out of the host cell taking away with them part of the cell membrane to form the viral membrane. The newly produced, immature viruses (virions) are non-infectious or unable to infect other cells. However, once they mature, that is when the viral enzyme protease turns viral macro-proteins into functional proteins, they become infectious and infect other cells. Thus the process repeats itself. Figure 13 below illustrates the HIV viral replication process.

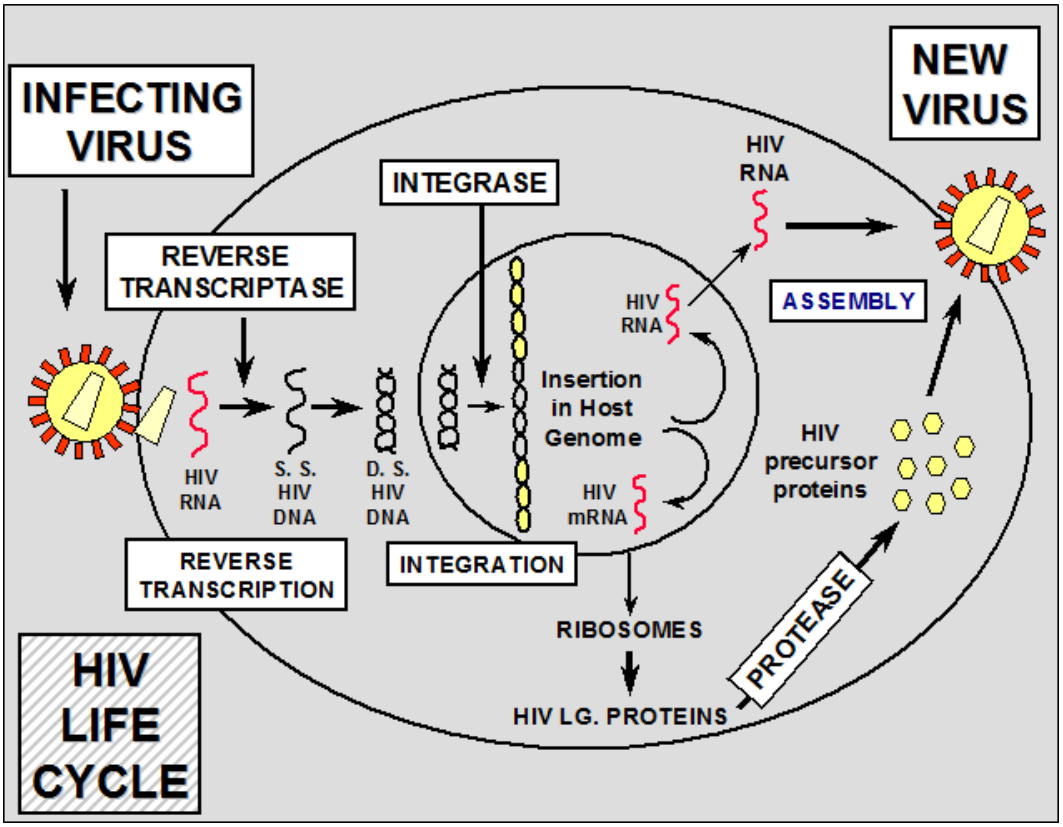

Figure 12: HIV viral replication process

You have now finished with sub unit 4.1.3 and can proceed to looking at the signs and symptoms of HIV using the WHO staging criterion.n

4.3.4 Signs And Symptoms (Pathophysiology)

The World Health Organization (WHO) has developed a staging system for HIV disease based on clinical symptoms. The system helps you to estimate the degree of immune deficiency that your patient has. In particular, the applications of the staging is meant to help you achieve the following:

- monitor patients and determine prognosis;
- prioritize the need for preventive therapies;
- provide guidance as to when to start or review ARV drug therapy;
- assess the clinical response to therapy in the absence of appropriate laboratory tests

How many stages arediscussed by 'WHO' stagi4.4 WHO STAGING OF HIV

1. Clinical Stage 1:
    - Asymptomatic (no present problems)
    - Persistent generalized lymphadenopathy (swollen nodes all over)

    Performance Scale 1
    - Asymptomatic, normal activity
2. Clinical Stage 2:
    - Weight loss < 10% body weight
    - Minor skin or mucus problems
    - Herpes zoster within last 5 years
    - Recurrent upper respiratory infections

    And/or Performance Scale 2
    - Symptomatic, normal activity
3. Clinical Stage 3:
    - Unexplained severe weight loss (over 10% of presumed)
    - Unexplained chronic Candidiasis for longer than one month
    - Unexplained persistent fever (intermittent or constant for longer than one month)
    - Persistent oral Candidiasis
    - Oral hairy leukoplakia
    - Pulmonary tuberculosis within the last year
    - Severe bacterial infections (e.g., pneumonia, pyomyositis)
    - Acute necrotizing ulcerative stomatitis, gingivitis or periodontitis
    - Unexplained anaemia (below 8 g/dl), neutropenia (below 500/mm3) and/or chronic thrombocytopenia (below 50,000 mm3).

    And/or Performance Scale 3
    - Bed-ridden <50% of days during last month
4. Clinical Stage 4:
    - HIV wasting syndrome: Weight loss of >10% of body weight, unexplained chronic diarrhoea (>1 month), chronic weakness and unexplained prolonged fever (>1 month)
    - Pneumocystis carinii pneumonia

- Toxoplasmosis of the brain
- Cryptosporidiosis with diarrhoea > 1 month
- Cryptococcosis, extrapulmonary
- Cytomegalovirus (CMV) disease
- Herpes simplex virus infection> 1 month
- Progressive multifocal leukoencephalopathy (PML)
- Any mycosis
- Candidiasis
- Atypical mycobacteriosis
- salmonella bacteraemia
- Extrapulmonary tuberculosis
- HIV encephalopathy, as defined by CDC
- Poor mental or physical function that prevents activities
- Lymphoma
- Kaposi's sarcoma (KS)

Performance Scale 4

- Bed-ridden >50% of days during last month
- HIV wasting syndrome
- Pneumocystis Jiroveci pneumonia (PCP)
- Recurrent severe bacterial pneumonia
- Chronic herpes simplex infection (orolabial, genital or anorectal of more than one month's duration or visceral at any site).
- Esophageal candidiasis (or candidiasis of trachea, bronchi or lungs)
- Extra pulmonary tuberculosis
- Kaposi sarcoma
- Cytomegalovirus infection (retinitis or infection of other organs)
- Central nervous system toxoplasmosis
- HIV encephalopathy
- Invasive cervical carcinoma
- Extrapulmonary cryptococcosis, including meningitis

- Disseminated non-tuberculous mycobacterial infection
- Progressive multifocal leukoencephalopathy
- Chronic cryptosporidiosis
- Chronic isosporiasis
- Disseminated mycosis (extrapulmonary histoplasmosis, coccidioidomycosis
- Lymphoma (cerebral or B-cell non-Hodgkin)
- Symptomatic HIV-associated nephropathy or cardiomyopathy
- Recurrent septicaemia (including nontyphoidal Salmonella)
- Invasive cervical carcinoma
- Atypical disseminated leishmaniasis

, drug information, counselling, patient assessment and when to start treatment. Then you will understand patient managementand care of those in special units. The subunits are 4.2 to 4.10 and are covered in 12 hours.

1. Oral hairy leukoplakia and Severe bacterial infections (e.g., pneumonia, pyomyositis) are stage 3 signs and symptoms. T/ F ans. T
2. Asymptomatic (no present problems) is one of the signs and symptoms in stage 4. T/F

Ans. F

5. You have come to the end of sub unit 4.1 and this time move to 4.2

You have now finished subunit 4.1 and have knowledge of what HIV is and acronyms associated with the disease. The following sub units will help you understand the goals and principles of ART

4.4 Goalsand General Principles of ART

We shall first look at the goals of ART followed by the principles.

4.4.1 Goals of ART

There are 5 main goals of ART, namely:

- Goal #1: Decrease the amount of virus in the body
  - To undetectable levels

  - Should be **sustained over time** to prevent drug resistance and disease progress
- Goal #2: Support and help the immune system
  - The CD4 cell count should increase approximately 100 cells/mm3 per year
  - In most cases, **immune function improves** so that development of new opportunistic infections is unlikely and prophylactic or preventive OI therapy can be stopped.
  - If **opportunistic infections** are already present, their **course may be shortened** or made less severe with ARV therapy.
- Goal #3: Improve the of quality of life
  - With improved immune function and suppression of viral replication, patients often **gain weight**, have **less fatigue**, and generally **feel better**.
  - They often can **return to work** and their other usual activities. Hope is restored.
- Goal #4: Reduction of HIV-related illness and death
  - Improved immune function and **suppression of viral replication stops the steady progression of HIV** disease and reverses the clinical course.
  - Development of **new opportunistic infections is unlikely** and patients are less likely to require hospitalization or to die from AIDS.
  - ARV therapy has been shown to benefit both adults and children.
- Goal #5: Possible reduction in transmission to others
  - Higher HIV viral loads are associated with increased risk of HIV transmission from mother to child and also between sexual partners.
  - ARV therapy has been has been shown to decrease risk to mother-to-child transmission of HIV
  - It is believed that use of effective ARV therapy will also **reduce the risk of sexual transmission** by lowering the viral load.

4.4.2 Principles of ART

The principles of ART are to:

1. Improve adherence to the ARV regimen

Taking ARV medication exactly as prescribed (correct dose, frequency, with appropriate food restrictions, etc.) is important to prevent the virus from multiplying. If the virus continues to multiply when taking ARV drugs, the virus changes and becomes resistant. When resistance develops to a specific ARV agent, that agent is no longer effective.

2. Use a combination of ARV drugs

   Effective ARV therapy generally includes a **combination of three or more drugs from at least two different classes of drugs**. Two nucleoside reverse transcriptase inhibitors (NRTI) with a protease inhibitor (PI), two PIs, or a non-nucleoside reverse transcriptase inhibitor (NNRTI). The rationale for combination therapy is to prevent or delay the development of drug resistance.

3. Rational sequencing of drugs

   The drug combination used is less important in predicting success than the **patient's ability to take the prescribed regimen correctly and consistently**.

   Drug combinations should be put together in such a way that they are:

   - As effective as possible in reducing viral load
   - Relatively free of side effects
   - As simple as possible to take (few pills, 1-2 times per day, minimal food restrictions)
   - Preserve future treatment options should they fail.

4. Delaying Resistance

   Delaying resistance is a critical consideration in any ARV treatment plan. This requires the right choice of regimen, continuous supply of drugs and patient adherence. Even in an ideal situation, 20-40% of patients will fail treatment within 5 years because of resistance. Resistance occurs when there is incomplete suppression of HIV.These resistant virus becomes dominant and overwhelms and outnumbers the remaining drug-sensitive virus.Continued treatment will result in increasing viral load and ultimately progressive disease.Once resistance develops to a specific drug, that drug can generally never be used effectively again. When resistance to one drug develops, cross resistance to other drugs in the same class may develop. Drug resistance develops more easily to certain ARV agents

than others and resistant viral strains can be transmitted to others.Those infected may have a reduced probability of successful ARV treatment.Strategies to prevent resistance involve using regimens that are as potent as possible and are relatively easily adhered to by patients.

Self test questions: what are the corresponding positions of the following gaols?

- 1. …….. Improve the of quality of life
- 2. ……. Reduction of HIV-related illness and death

Choose form here

a 2 b. 4 c. 3

Answers

1 . 3 2. B

Now you that you have finished this sub unit and answered the questions you can move on to sub unit 4.3.

4.5 Antiretroviral Drug Information

This sub unit will take you through to understand the treatment regimen for HIV infection. The sub unit will help you to have more information on HIV drugs a necessary prerequisite for sub units 4.6 to 4.10. read through and move on to the last sub unit and do all the activities as requested.

Modes of action of various ARV classes

ARV agents fall into categories based on where in the viral replication cycle they are effective.

a. Nucleoside reverse transcriptase inhibitors (NRTI): competitively inhibit the enzyme that facilitates the conversion of HIV RNA into HIV DNA through formation of faulty versions of the building blocks (nucleotides) used by reverse transcriptase to convert RNA to DNA. When reverse transcriptase uses these faulty blocks, the new DNA cannot be built correctly.

   Here are some examples of NRTI

   Zidovudine (ZDV, AZT/Retrovir),

   Didanosine (ddI/Videx),

   Stavudine (d4T/Zerit)

   Lamivudine (3TC/Epivir),

   Abacavir (ABC/Ziagen),

Emtricitabine (FTC/Emtriva)

Tenofovir (TDF/Viread).

XTC (3TC or FTC)

Combination Nrtis Include

Truvada (tenofovir/emtricitabine)

Combivir (zidovudine/lamivudine)

Epzicom (abacavir/lamivudine)

Trizivir (abacavir/zidovudine/lamivudine).

Non-nucleoside reverse transcriptase inhibitors (NNRTI): directly bind to and thereby inhibit the enzyme that facilitates the conversion of HIV RNA into HIV DNA

Examples of NNRTI include:

Efavirenz (EFZ) (Sustiva)

Nevirapine (NVP) (Viramune)

Delavirdine (Rescriptor)

Etravirine (Intelence).

Rilpivirine (Edurant), the newest member of this class of drugs, was approved by the U.S. FDA in May of 2011.

Two complete HIV treatment regimens that combine two NRTIs and one NNRTI in one pill taken once a day are available for convenience.

Atripla: a combination of efavirenz, emtricitabine, and tenofovir. (approved for use by the FDA in 2006). (Available in Zambia)

Complera: a combination of rilpivirine, emtricitabine, and tenofovir (Not yet available in Zambia)

- This combination pill was approved in August 2011 by the FDA as another first-line treatment for HIV infection in patients who need to start therapy.

b. ***Protease Inhibitors (PI):*** prevent the virus from making new copies of itself by blocking the protease enzyme that cuts up the proteins needed for new copies of the HIV virus.

PIs include:

- Ritonavir (Norvir),
- Lopinavir and ritonavir combination (Kaletra)
- Saquinavir (Invirase),

- Indinavir sulphate (Crixivan),
- Fosamprenavir (Lexiva),
- Darunavir (Prezista),
- Atazanavir (Reyataz),
- Tipranavir (Aptivus),
- Nelfinavir (Viracept).

Using PIs with NRTIs reduces the chances that the virus will become resistant to medications.

However, there are newer agents that keep the virus from entering the human cell which are not available in Zambia. These arecalled fusion and entry inhibitors:

Enfuvirtide (Fuzeon/T20) was the first drug in this group. It is given in injectable form like insulin.

Another drug called maraviroc (Selzentry) binds to a protein on the surface of the human cell and can be given by mouth. Both drugs are used in combination with other anti-HIV drugs.

Action: Prevent HIV from entering a host cell (CD4 cell).

Intergrase inhibitors (Not yet available)

Action: Inhibit/prevent integration of viral DNA into host cell DNA.

Integrase Inhibitors

Integrase inhibitors stop HIV genes from becoming incorporated into the human cell's DNA. This is a newer class of drugs recently approved to help treat those who have developed resistance to the other medications or used in initial treatment in combination with NRTIs.Raltegravir (Isentress) was the first drug in this class approved by the FDA in 2007. Elvitegravir is the latest integrase inhibitor developed and FDA-approved in 2012 as a component of a fixed-dose combination pill taken once daily called Stribild (elvitegravir/cobicistat/tenofovir/emtricitabine).

As you have gone through the HIV drugs you appreciate the fact that they have side effects, hence the reason you need to know individual drug side effects and how you can manage them.

Side Effects or Toxicity

The **significance of adverse effects** in ART management is critical as the drugs may not be taken correctly due to persistent nausea or vomiting prevents absorption. The drugs may worsen quality of life (weakness and due to long term effects i.e. high lipids, heart disease, diabetes,

etc).Some drugs may induce life-threatening effects such as Stevens-Johnson syndrome, pancreatitis, lactic acidosis and result into non-compliance to treatment.

Common Adverse Effects

- **Lactic acidosis**: Rare, life threatening. More likely with **pregnancy** and **ddI + d4T** therapy
- **Liver toxicity**: Up to 12% with long term **Nevirapine** use. More common in women and in first 12 weeks of therapy. Begin Nevirapine slowly first two weeks to prevent this.
- ***Hyperglycaemia***/Diabetes: Common with Protease Inhibitor (PI)use
- **Fat maldistribution** syndromes: Common with **PI** use
- Elevated serum lipids: More common with PI use
- **Bone disorders**: Loss of bone density more common in women and with **PI** use
- **Rash**: Common with Nevirapine (**NVP)** use, but only life-threatening 1% of the time
- **Pancreatitis**: More common with Didanosine (**ddI) + Stavudine (d4T) use**, more common in women, associated with lactic acidosis
- **Abacavir (ABC) sensitivity**: Life-threatening, may never use again
- **Bone Marrow suppression**: Most common with Zidovudine (**ZDV**) use
- Peripheral Neuropathy: More common with Didanosine (ddI) and/or Stavudine (d4T) use
- **Kidney stones**: Most common with Indinavir (**IDV)**
- CNS effects: fFrequent with Efavirenz
- GI intolerance: fFrequent with all drug classes

Managing Adverse Effects

- Educatepatientabout possible adverse effects
- Consider medical condition when selecting a regimen to decrease risks
- Follow recommendations for laboratory and clinical monitoring while on ARVs
- **Educate about danger signs** of life-threatening conditions
- **Make sure they know how to reach you** when they have questions or concerns

Drug Interactions

- Nelfinavir, Ritonavir, Lopinavir/Ritonavir, Nevirapine and Efavirenz may **decrease oral contraceptive effectiveness**
- **NNRTIs** and **PI** drugs should not be used with **Rifampin**can lead to liver damage
- Phenytoin, Phenobarbitol, Ketoconazole and Statin drugs all interact with ARVs

Self-test: True or False

The following are common side of ARVs

1. **Lactic acidosis**: rare, life threatening. ……
2. **Liver toxicity**: Up to 12% with long term **Nevirapine** use. …….
3. ***Hyperglycaemia***/Diabetes: Common with Protease Inhibitor (PI)use …….
4. **Fat *misdistribution*** syndromes: Common with **PI** use …….
5. Elevated serum lipids: More common with PI use ……

Answers.

1 to 5 are **All True**

4.6 Counselling And Education

Counseling and education are important elements in the management of patients on ART. Counseling refers to helping people make decisions while education refers to giving people new information. The table below shows areas of focus during a counseling and education session.

Table 3: Counseling Key issues

Topic	Initial Visit	Every Visit	At Intervals (as needed/appropriate)
Adherence		X	
HIV Disease Basics & Prevention	X		
Risk Reduction		X	
Disclosure and Support	X		X
Nutritional Support	X		X

Childbearing	X		X
Healthy Living	X		X
ARV Therapy	X		X
Plan of Care	X		

Adapted from HIV Module, 2004

1. Adherence

Every time you see a patient, reinforce adherence and help patient to solve their problems. Adherence is the most important factor in successful ARV therapy and this must assessed at each visit.

2. You must discuss with the patient **HIV disease basics** and prevention. Determine the simple understanding on;

- How the HIV virus affects the immune system
- The **natural history of HIV disease**, how HIV and AIDS are different
- The goals of HIV management
- HowHIV transmission and infection can be prevented

3. **Risk Reduction -discuss at every visit** on how to prevent HIV transmission and this includesthe following:

- Abstain from sexual relations
- Be faithful to a single partner with condom use or who is also faithful to you
- Use latex or polyurethane male or female Condoms with every act of intercourse (vaginal, anal or oral)
- Seek medical treatment if you have an STI; be sure your partner also seeks medical attention
- Do not share needles, razors, or other piercing instruments
- Practice safer sex even if your partner is HIV positive
- Avoid local beliefs and practices that may increase the risk of HIV transmission
- Practice dual protection – use condoms for both family planning and to prevent HIV

Disclosure and Support- discuss issues of disclosure of HIV status with someone for support. This be discussed during the initial visit and when there are changes in the patient's personal situation. Confidentiality must maintained throughout, include also disclosure to children at an appropriate time.

Nutritional Supportncourage the patient to eat a balanced diet consisting of carbohydrates, foods rich in iron, foods rich in vitamin A, foods rich in calcium, foods rich in magnesium and foods rich in vitamin C. The discussion must also include what foods to avoid (uncooked, not fresh), possible food-drug interactions, herbal remedies and also access to clean water.

4. ***Childbearing***- this applies to both female and male patients, best done at initial visit and throughout treatment plan. A more detailed discussion must include the following:
    - There is a change in the patient's relationship
    - The patient wants to have a child
    - The patient is using contraception inadequately or inappropriately
    - A woman is on medications that are dangerous to a fetus
    - There are important new developments in the area of HIV and pregnancy
    - Desires and plans for childbearing
    - Prevention of undesired pregnancies (family planning)

    Prevention of MTCT for those wanting to have a baby
5. **Healthy Living**- healthy living is a combination of choices to help HIV positive individuals live longer, feel better and enjoy their lives. Some areas of healthy living include:
    - Spirituality
    - Emotional support
    - Stress reduction
    - Adequate diet and exercise
    - Avoidance of tobacco
    - Avoidance of alcohol
6. **ARV Therapy-** this focuses on when the patient is ready to start medication. Explain to the patient this is a lifelong regimen and also include the following
        - Indications for starting the drug therapy.

- That if doses are missed the virus changes and then the drugs are not effective.
- How to take the drugs.
- Drug interactions
- The **importance of adherence** (including the chance for resistance; the need to start and stop drugs at the same time; possible problems such as work and daily lifestyle)
- Possibility and symptoms of **side effects**
- Management of side effects
- Symptoms of toxicity
- Possibility of a change in therapy if the patient becomes pregnant
- Give written instructions
- Assist the patient to start a diary for documenting treatment: symptoms, adherence, and missed doses.
- Emphasize that drugs should not be shared or sold.

7. **Plan of Care**- the following should be emphasised:
   a. Timing of the visits
      - 1st visit 2 weeks after starting therapy
      - Regular follow-up needed monthly for 3 months
      - Thereafter every 3 months
      - Unscheduled visits as needed
      - Stable patients may be seen every six months
   - How to contact the health worker
   - Need for testing of partners and children.

4.7 Patient Assessment

History taking

The components of the history that should be obtained from the HIV-infected patient include the following:

- Demographic data
- Presenting complaints

- Medical history
- Medication/drug/substance use history
- Reproductive history
- Socio-economic data
- Disclosure of illness

When taking history ensure privacy and confidentiality at all times.

Physical examination

Using the for four skills of physical examination (inspection, palpation, percussion and auscultation) conduct a head to toe full examination noting details and compare with history notes. Take vital signs (temperature, pulse, respirations and blood pressure. At the end perform a systems review to elicit more information on the patient's condition.

Laboratory tests used to diagnose HIV infection

A lot of laboratory investigation need to be done and as a nurse you need to know these investigations as they are critical to starting the patient on ART and continued management of the patient. Below is list of investigations commonly done;

1. Viral detection test such ELISA or Western Blot
2. CD_4 count to detect levels of CD_4 cells normal ranges above $500/mm^3$
3. Liver function test (serum alanine and aspartate detection)
4. Viral load tests
5. Viral detection test such Polymerase Chain Reaction test in children
6. Full blood count as some drugs depress bone marrow function such Zidovudine
7. RPR to rule out syphilis
8. Total lymphocyte count (TLC) of <1200 cells/mm^3 probably means a $CD4^+$ cell count <200/mm3.
9. Serum bilirubin as drugs such Indinavir cause increase in bilirubin levels
10. Serum creatinine detection test
11. Pregnancy test
12. Serum amylase test in case of pancreatitis

13. Serum lipid test as some drug like Protease Inhibitors cause increase in serum lipid etc.

Self-assessment test

Write True or False to the following statements:

What tests are done to assess the effectiveness of treatment for someone on ARVs

1. CD4 count to detect levels of CD4 cells normal ranges above 500/mm3
2. Sugar levels
3. Liver function test (serum alanine and aspartate detection)
4. Viral load tests
5. Viral detection test such Polymerase Chain Reaction test in children
6. Full blood count as some drugs depress bone marrow function such Zidovudine
7. RPR to rule out syphilis
8. Total lymphocyte count (TLC) of <1200 cells/mm3 probably means a CD4 + cell count <200/mm3.

Answers:1.T;2.F;3.T;4.T;5.T;6.T;7.T;8.T.

4.8 Initiating Art

When initiating a patient on ART the following must be considered carefully

WHO HIV Staging System

With parameters available the WHO staging must be looked as not all patients using this system may qualify to start ART. Those in stage 3 and 4 will require initiation of treatment. The other method is using the T-staging and those still in 3 and 4 with new and recurrent infections (indicative of treatment failure) will need ART initiation.

Recommendations for starting ARV therapy

The WHO staging, CD_4 results and national treatment protocol must be followed.

Activity

Read the Zambia national guidelines on the commencement of ARV therapy

Who should be initiated on ARV therapy

1. Those with CD_4 less than 350
2. Performance scale less 3 and 4
3. Readiness of the patient
4. ON request by the patient
5. Severe HIV neuropathy
6. When the health facility has the right combination of drugs

RECOMMENDED FIRST LINE TREATMENT FOR ADULTS

Table 4: Recommended first line treatment

Specific population	Description	1st line	Alternative
Adults	First line A once-daily fixed dose combination is recommended	TDF+XTC+EFV	TDF+XTC+NVP OR ABC+ 3TC+EFV

MoH, 2014

Indicate ture or false to thefollowing investigation if used to detect the HIV status

1. Viral detection test such ELISA or Western Blot ….. T/F
2. CD_4 count to detect levels of CD_2 cells normal ranges above 50 … T/F
3. Liver function test (serum alanine and aspartate detection) T /F
4. Viral load testslack ….. T/F
5. Viral detection test such Polymerase Chain Reaction test in children .. T/F
6. Full blood count as some drugs depress bone marrow function such Zidovudine …. T/F
7. RPR to rule out syphilis … T/F
8. Total lymphocyte count (TLC) of <1200 cells/mm^3 probably means a

CD4 $^{+}$ cell count <200/mm3. ,…. T/F

9. Serum bilirubin as drugs such as Indinavir cause increase in bilirubin levels . T/F

10. Serum creatinine detection test … T/F

11. Pregnancy test … T/F

Answers

1 . T 2. T 3. T 4. F 5. F 6. F 7. F 8. F 9. F 10. F

4.9 Management Of A Patient On Art

The following sub unit will discuss management of clients on ART and you will be required to pay attention.

2. Adherence

Every time you see a patient, reinforce adherence and help patient to solve their problems. Adherence is the most important factor in successful ARV therapy and this must assessed at each visit.

2. You must discuss with the patient **HIV disease basics** and prevention. Determine the simple understanding on;

- How the HIV virus affects the immune system
- The **natural history of HIV disease**, how HIV and AIDS are different
- The goals of HIV management
- How HIV transmission and infection can be prevented

1. **Risk Reduction -discuss at every visit** on how to prevent HIV transmission and this includes the following:
    - Abstain from sexual relations
    - Be faithful to a single partner with condom use or who is also faithful to you
    - Use latex or polyurethane male or female Condoms with every act of intercourse (vaginal, anal or oral)

- Seek medical treatment if you have an STI; be sure your partner also seeks medical attention
- Do not share needles, razors, or other piercing instruments
- Practice safer sex even if your partner is HIV positive
- Avoid local beliefs and practices that may increase the risk of HIV transmission
- Practice dual protection – use condoms for both family planning and to prevent HIV

2. Disclosure and Support- discuss issues of disclosure of HIV status with someone for support. This be discussed during the initial visit and when there are changes in the patient's personal situation. Confidentiality must maintained throughout, include also disclosure to children at an appropriate time.
3. **Nutritional Support**- Encourage the patient to eat a balanced diet consisting of **carbohydrates**, foods rich in **iron**, foods rich in **vitamin A**, foods rich in **calcium**, foods rich in **magnesium** and foods rich in **vitamin C. The discussion must also include w**hat **foods to avoid** (uncooked, not fresh), possible food-drug interactions, herbal remedies and also access to clean water.
4. Childbearing- this applies to both female and male patients, best done at initial visit and throughout treatment plan. A more detailed discussion must include the following:
    - There is a change in the patient's relationship
    - The patient wants to have a child
    - The patient is using contraception inadequately or inappropriately
    - A woman is on medications that are dangerous to a foetus
    - There are important new developments in the area of HIV and pregnancy
    - Desires and plans for childbearing
    - Prevention of undesired pregnancies (**family planning**)
    - **Prevention of MTCT** for those wanting to have a baby
5. **Healthy Living**- healthy living is a combination of choices to help HIV positive individuals live longer, feel better and enjoy their lives. Some areas of healthy living include:
    - Spirituality
    - Emotional support

- Stress reduction
- Adequate diet and exercise
- Avoidance of tobacco
- Avoidance of alcohol

6. ARV Therapy- this focuses on when the patient is ready to start medication. Explain to the patient this is a lifelong regimen and also include the following
    - Indications for starting the drug therapy.
    - That if doses are missed the virus changes and then the drugs are not effective.
    - How to take the drugs.
    - Drug interactions
    - The **importance of adherence** (including the chance for resistance; the need to start and stop drugs at the same time; possible problems such as work and daily lifestyle)
    - Possibility and symptoms of **side effects**
    - Management of side effects
    - Symptoms of toxicity
    - Possibility of a change in therapy if the patient becomes pregnant
    - Give written instructions
    - Assist the patient to start a diary for documenting treatment: symptoms, adherence, and missed doses.
    - Emphasize that drugs should not be shared or sold.
7. **Plan of Care**- the following should be emphasised:
    a. Timing of the visits
        - 1st visit 2 weeks after starting therapy
        - Regular follow-up needed monthly for 3 months
        - Thereafter every 3 months
        - Unscheduled visits as needed
        - Stable patients may be seen every six months
    b. How to contact you
    c. Need for testing of partners and children

Monitoring And Follow-Up

The purpose of monitoring and follow-up is to:

- Assess the effectiveness of therapy
- Evaluate potential side effects or toxicity
- Assess and reinforce adherence
- Evaluate for the development of other HIV-related illnesses

Minimum Clinical Assessment- there is need to clinically assess the condition of the patient and the following are what to focus on

- Signs and symptoms of possible drug toxicity
- Assessment of Adherence
- Assessment of response to therapy
- Weight
- Basic lab monitoring

Recommendations for Monitoring/Followup

Two weeks after beginning therapy:

- Clinical assessment
    - Coping
    - Adherence
    - Complaints
    - Fears
    - Side effects
    - New illnesses
    - Focused history and physical examination
    - Nevirapine dose escalation

Laboratory assessment

- Hemoglobin if on AZT
- Other tests depending on symptoms

Monthly for 3 months:

- Clinical assessment
- As at two week visit

- Laboratory assessment

- ALT if on NVP
- Hb if on AZT
- White blood cell count (WBC)

Every 3 months until stable:

- Clinical assessmentAs at two week visit
- Laboratory assessment
  - ALT if on NVP
  - White blood cell count (WBC) and Haemoglobin to check for anaemia (especially if on ZDV)
  - CD4 if available

Every 6 months when stable and no symptoms:

- Clinical assessment
- As at two week visit
- Laboratory assessment
  - ALT if on NVP
  - Creatinine if on TDF
  - White blood cell count (WBC) and Haemoglobin to check for anemia (especially if on ZDV)
  - CD4 if available
  - Chemistry panel
    - Other assessments
    - Check lipid profile every 3-6 months if abnormal cholesterol if possible

During return visits, be sure to:

- Make any necessary changes to the plan of care
- Provide more ARV drugs
- Care for side effects or other concerns as indicated
- Review and continue developing the treatment plan
- Reinforce key health messages and counselling
- Provide care and support, problem-solve with the patient

- Assess for opportunistic infections or the need for referral

Adherence to Therapy

  - Assess and reinforce adherence at every visit
  - Ask the patient about their adherence in a way that helps them feel comfortable
  - Acknowledge that:

- Everyone misses doses sometimes
- It is common for people to forget their drugs sometimes
- Sometimes people have trouble taking their pills
- Sometimes people are pressured by family or friends to share drugs

Other ways to assess adherence are to:

- Ask patients to bring in their pill bottles
- Ask patients to tell you how they take their pills and what they eat, starting in the morning of a typical day
- Ask patients to show you which pills they take at what times during the day
- Logistical Support Patients may need help solving problems with the logistics of ARV therapy, such as how to get more pills

Areas that may need to be addressed include:

- Contact information for the provider in case of emergency
- Where to go or who to call if a problem develops
- Issues of transport and outreach
- The need for outreach for patients that miss appointments or have trouble coming into the clinic

Assessing Effectiveness of Therapy

CD4 and viral load tests are a good way to assess the success of the therapy.

To assess effectiveness, ask the following:

- The patient has **gained weight**
- The patient is **able to do more** daily tasks
- The patient **feels better**
- The patient has not developed new opportunistic infections

If the patient is not responding, you may need to consider changing the drug regimen.

But first ask: How is the patient taking drugs? Is the patient adhering to the drug regimen? Carefully investigate this before changing therapy.

After about 6 weeks of therapy the patient may become sicker for a short time as their immune system improves. This is called "**immune reconstitution syndrome**" Do not change therapy if this occurs, rather, treat the problem and continue to observe.

Teatment Failure

There are3 types of treatment failure

1. Clinical failure
2. Immunologic failure
3. Virology failure

Clinical failure

Occurrence or re-occurrence of an AIDS-defining OI or malignancy after 6 months of HAART initiation. Immune Reconstitution Syndrome does not qualify as Clinical Failure and should be excluded

Immunologic failure

The following are the parameters for immunologic failure

CD4 increase of <50 cells/μL in the first year of HAART or CD4 drop by >50% from peak level on therapy without any concomitant infection to explain CD4 decrease or CD4 decrease below baseline while on HAART

Virologic failure

This is when there is confirmed detectable viral load after an undetectable viral load and failure to suppress to <400 copies/mL by 6 months of therapy or failure to suppress to <50 copies/mL by 1 year of therapy.

IN types of failure there is need to investigate so as to correct the situation. The following are some of the reason of treatment failure;

- Non- Adherence
- Reduced Drug levels
- Resistance

Changing Or Stopping Arv Therapy

- Change in treatment could be precipitated by the following
- Therapeutic failure? Before deciding that ART has failed it is important to ascertain that the patient's medication compliance has been good. If the patient has not been taking his drugs, ART failure is not the problem.

Changing Drug Regimens

The following situations are indications to change the drug regimen:

Failure which is

- Clinical: Occurrence or Re-occurrence of OI or malignancy after 6 months of HAART initiation.
- Immunologic: CD4 count drops (>50% from the peak value) or a return to, or below, the pre-therapy baseline. Failure of CD4 to increase by 50 cells/uL in first year
- Virologic: Confirmed detectable viral load after an undetectable V.L Failure to suppress to < 400 copies after 6 months or <50 copies after 1year of therapy.
- Additional indications to change the drug regimen: toxicity, poor adherence, occurrence of active TB, occurrence of pregnancy and new therapies

Immune Reconstitution Inflammatory Syndrome (IRIS): this is paradoxical clinical worsening of a known condition or the appearance of a new condition after initiating antiretroviral therapy in HIV-infected patients (Bosamiya, 2011).

Signs & symptoms of subclinical & unrecognized OI

- Fevers
- Sweats
- Loss of weight
- Lymphadenopathy
- Worsening pulmonary or CNS lesions
- Paradoxical worsening of treatment response: 8-12 weeks
- Autoimmune disease presentation

Risk factors for IRIS: the following the risk factors to developing IRIS

i. Starting ART near time of diagnosis of OI
ii. CD4 < 50
iii. Rapid decrease in VL

iv. Diagnosis is difficult: rule out OIs, TB, treatment failure, drug toxicity

Treatment for IRIS

i. Continue ART; its not an indication to stop ART or to change an ART regimen
ii. Treat OI
iii. Consider corticosteriods
iv. Selecting a New Regimen
  - In cases of treatment failure:
  - Change the whole regimen

WHO recommended second line ARV regime

- In other cases of side effects, interactions, toxicities, pregnancy, TB
  - Change only one drug instead of the whole regimen
  - If a single drug is stopped because of toxicity, remember:

Add another drug based on the specific side effect from the table in Managing ARV Therapy

OR

- Consult with an HIV expert if you are unsure or the patient's situation is serious

Indications for Referral or Consultation

Referral is determined by the level of care you are able to provide or any of the following:

Irreversible or severe drug toxicity

Unable to take the drugs despite change in therapy

Patient has no symptoms, no CD4 count available and patient wishes to start ARV therapy

Treatment failure after use of first line drugs

Patient with severe infection or any other conditions requiring a higher level of care.

Self test

1 . Health living is a combination of choice used to help HIV positive individual and among these are the following except:

a. Stress reduction
b. Adequate diet and exercise
c. Avoidance of tobacco
d. Non Avoidance of alcohol

2 .Treatment for IRIS include the following except:

a. Continue ART; its not an indication to stop ART or to change an ART regimen
b. Treat OI
c. Consider corticosteriods
d. Using the old Regimen

Take Note

The purposes of monitoring of ARV therapy and follow-up are to:

- Assess the effectiveness of therapy
- Evaluate potential side effects or toxicity

Assess and reinforce adherence

Evaluate for the development of other HIV-related illnesses

4.10 Managing Hiv And Aids In Special Populations

Activity

What are the special group of people do you expect to study in this unit?

Pregnant Women

Effect of Pregnancy on HIV

Administration of ARVs in pregnant women has been and it is still a challenge. The highly active ARV drugs may have developmental defects. However,there is evidence that pregnancy on its own worsens HIV infection or hastens its progression. CD4+ cell count declines during pregnancy of both HIV positive and HIV negative women due to increased plasma volume however, HIV infection can lead to adverse pregnancy outcomes e.g.

- Spontaneous abortion or miscarriage
- Intrauterine growth retardation
- Low birth weight
- Preterm delivery, especially with more advanced disease
- Increased stillbirth, perinatal and newborn mortality

WHO's 4-Prong Approach to PMTCT

- Core PMTCT interventions

- Antiretroviral prophylaxis
- Safer obstetric practices
- Safer infant feeding practices
- Prevention of Mother-to-Child Transmission (PMTCT)

There is evidence that HAART has decreased the rate of Mother to Child Transmission of HIV. If effective combination ARV therapy for maternal treatment is not available, use short-course ARV treatment, if available, to reduce the risk of MTCT.

Table 5: ARV treatment protocol for pregnant and breastfeeding mother

Specific population	Description	1st line	Alternative
Pregnant and breastfeeding women	First line	TDF+XTC+EFV	TDF+XTC+NVP or ABC+ 3TC+ EFV
	Previous sd-NVP exposure, or NVP mono-therapy exposure (NVP without 7 days of ZAT+3TC cover) or Unsure of tail coverage	TDF+XTC+LPV-r	TDF+XTC+ATV-r

NB: the pregnant woman must continue with antenatal visitsand all follow up care while on ART must be provided as per schedule

Children And Adolescents

Table 6: Treatment Guidelines for Children and Adolescents

Specific population	Description	1st line	Alternative
Children (6weeks to <3 months)	First line Maternal sd-NVP exposure, or maternal NVP mono-therapy exposure (NVP	AZT+3TC+LPV-r	After 3 months substitute to preferred 1 line with ABC

	without 7 days of ZAT+3TC cover) or mother unsure of tail coverage		
Children (3 months to <5 years)	First line	ABC+ 3TC+LPV-r	AZT+3TC+LPV-r After 5 years substitute to preferred 1st line with TDF+XTC+LPV-r
	HIV and Tuberculosis Co-infection	ABC+3TC+EFV	After completion of ATT substitute to preferred 1st line with LPV-r
Children (5 to <10 years old)	First line (No history of maternal sd-NVP, maternal NVP mono-therapy, mother unsure of tail coverage	TDF+XTC+EFV (weight-based dosing)	TDF+XTC+NVP (weight-based dosing) ABC+3TC+EFV
Adolescents (10 to <19 years old) weighing <35Kg			
Adolescents (10 to <20 years old) weighing > 35Kg	First line A once-daily fixed dose combination is recommended	TDF+XTC+EFV	TDF+XTC+NVP or ABC+3TC+EFV

MoH, 2014

Revise the above section before you move to the next topic on tuberculosis

Tuberculosis Patients

Treatment of tuberculosis in HIV possess a serious challenge in that the anti-TB drugs can be substituted and therefore there is always a need to modify ART treatment to meet the TB treatment regimen and produce less side effects and toxic effects.

Goals of ARV therapy in TB are to:

- Reduce HIV-related morbidity and mortality during anti-TB treatment
- Decrease the reoccurrence of TB
- Prolong life
- Improve the quality of life

All patients should be evaluated for TB prior to initiating ART. The diagnosis of TB in HIV patients is challenging especially when patients are severely immuno-suppressed. Management of HIV and tuberculosis co-infection is complicated because some ARV agents have drug interactions with anti-tuberculosis agents.

Newly diagnosed TB and HIV Co-infection

- If CD4 under 200/mm^3: Start TB therapy, then start ARV after the TB therapy is tolerated (2 weeks – 8weeks)
- If CD4 between 200-350/mm^3 : Start TB therapy, consider ARVs after 2 months of TB therapy
- If CD4 over 350: Start TB therapy, defer ARVs
- CD4 not available: Start ARV therapy after the TB therapy is tolerated (2 weeks – 2 months).

Table 7:Treatment guideline in HIV-TB Co-infections

Specific population	**1st line**	**Alternative**
HIV-TB Co-infections	TDF+XTC+EFV	ABC+3TC+EFV
	TDF+XTC+LPV-r Double the dose of LPV-r if on rifampicin) or switch rifampicin to rifabutin (avoid in pregnancy or breastfeeding others)	ABC+ 3TC+ LPV-R

MoH, 2014

Take Note

You must follow up the patients on ART and the patients must continue with TB treatment as recommended.

Self assessment test

1. The following are goals of ARV therapy in HIV except
    a. Prolong life
    b. Improve quality of life
    c. Treat HIV only
    d. Reduce HIV related deaths
2. All are adverse effects of HIV in pregnancy except
    a. Haemorrhage
    b. Low birth weight
    c. Still births
    d. Abortions
3. The alternative HIV treatment in pregnant women is
    a. ABC+ 3TC+ EFV
    b. Ddi+ AZT+ NVP
    c. D4t + ABC+ EFV
    d. AZT+ 3TC+ EFV

Answer: Q1 C.Q2 A. Q3 A.

4.11 Post Exposure Prophylaxis (Pep)

Post-exposure prophylaxis is the use of ART to prevent HIV transmission. Non-occupational exposure to HIV inchildren is mostly due to sexual abuse. In adults, exposure to HIV is mostly associated with occupational injuries. The risk of acquiring HIV infection after occupational exposure to HIV-infected blood is low (1:300 after percutaneous). There is no risk of transmission when the skin is intact. Factors associated with an increased risk include: deep injury, visible blood on the device which caused theinjury, injury with a large bore needle from artery or vein,and materials which pose a risk of HIV transmission are tissues and organs, vaginal secretions, semen, any other lesions. Other blood-borne infections are hepatitis B and

hepatitis C viruses. Thus, all HCWs should receive HBV vaccination. Management of occupational exposure to infectious substances includes the following steps:

Immediately after exposure

- Clean the site: wash skin wounds with soap and running water. If the exposed area is an eye or mucous membrane, flush with copious amounts of clean water. DO NOT USE BLEACH or other caustic agents' disinfectants to clean the site.
- Contact your In-Charge or supervisor
- Contact the medical officer who does the following
    - Determine the need for post exposure prophylaxis for high risk of transmission and risks and benefits of taking ART.
    - Counsel regarding PEP's risk and benefits. Start PEP preferably within 2 hours of the exposure. If 72 hours have passed since exposure, do not provide PEP because of lack of effectiveness.
    - For high risk exposure, arrange immediate HIV testingtesting and counseling.
    - Do not give PEP to exposed employees who refuse HIV testing or are HIV positive at initial test. Instead refer to ART clinic for assessment of ART eligibility. Observe confidentiality
    - Send baseline creatinine (FBC if starting AZT)
    - Complete the appropriate government PEP register

Follow up

- HIV testing on the day of the exposure.
- If negative, retest at 6 weeks, 3 months and 6 months after exposure.
- Retest for HIV whenever acute illness includes fever, rash, myalgia, fatigue, malaise, and lymphadenopathy

See Medical Officer within 72hours after starting PPEP and monitor for side effects for at least 2 weeks

Table 8: Recommended treatment protocol for PEP

Risk Category	ART	DURATION
No risk: intact skin	Not recommended	
Medium risk: invasive injury,	TDF+XTC+LPV-r	28 days

no blood visible on needle		
High risk: large volume of blood/fluid, known HIV infected patient, large bore needle, deep extensive injury		
Penetrative sexual abuse		

MoH, 2014

You have come to the end of the sub unit 4.9 and now are expected to check yourself if you have understood the unit well. The next sub unit is the last for this medicine II and looks at record keeping. Take interest to learn the requirements and see how you will help your clients in remembering how to take care of themselves.

Make a summary of what you have learnt in sub unit 4.9 and proceed to 4.10.

Self test questions

1.PEP is

a. Use of ART in the prevention if HIV transmission

b. Use of antibiotics in the prevention of HIV transmission

c. Use of Chemicals to prevent HIV transmission

b. Use if antimalarials to prevent HIV transmission

2. PEP must commenced within

a. 4hours

b. 2hours

c. 3hours

d. 1hour

Answers: Q1 A. Q2 B.

4.12 Record Keeping, Monitoring And Evaluation

The following systems must be in place for record keeping, monitoring and evaluation

1. Effective patient tracking system to avoid high attrition rate and treatment failure.

2. Monitoring andevaluation tools- these include documents for children, adolescent, pregnant and breastfeeding mothers and adults. Such documents include
    - Safe motherhood cards
    - ART files
    - Smartcard
    - Antenatal register
    - Postnatal register
    - Under five cards and registers etc.
3. Supply chain management systems- these help in ensuring functioning supply chain system to avoid stock out. Such records include
    - Report and requisition forms
    - Daily activity register
    - Interval monthly summary report etc.
4. Quality improvement tools.
5. Constant monitoring and supervision (MoH, 2014).

ACTIVITY:

what is the importance of record keeping, monitoring and evaluation.

4.13 SUMMARY

You have come to the end of this unit in which you learnt of HIV/AIDS and the end of Medicine II. In this unit we defined various concepts used in HIV/AIDS management including the modes of spread, viral replication and the clinical manifestation of HIV/AIDS using the WHO staging criteria.learnt

We have looked at the goals and principles of ART and related drug information. We also briefly looked and counseling and education, patient assessment before commencing ART.

In this unit we also focused on management of special groups these being pregnant and breastfeeding mothers, child and adolescents including TB patients in line with recommendation by Ministry of Health-Zambia.

Finally we also looked post-exposure prophylaxis and management of various records and document and evaluation practices related to HIV/AIDSmanagement.

Take time to review unit 4in totality before you move to the next topic.

4.14 References

MoH,(2014).Zambia Consolidated Guidelines for Treatment and Prevention of HIV, Ndeke House, Lusaka.

Bosamiya S.S. (2011) **The Immune Reconstitution Inflammatory Syndrome,** available @ www.ncbi.nlm.nih.gov.