COURSE GUIDE

NSC 402
MEDICAL-SURGICAL NURSING IV

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Printed 2018

ISBN: 978-058-900-11

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GENERAL INTRODUCTION

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

COURSE OVERVIEW

Medical Surgical Nursing (IV)

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

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

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

STUDY UNITS

Module 1 Caring for Patients with Urinary System Disorders

Unit 1 Assessment and Diagnostic Evaluation of Urinary Disorders of the Urinary System
Unit 2 Review of Related Anatomy & Physiology of the Urinary System
Unit 3 Caring for Patients with Fluid and Electrolyte Disorders in Renal Disorders
Unit 4 Caring for Patients with Dysfunctional Voiding Patterns; Congenital Voiding Dysfunction

Module 2 Caring For Patients with Eye and Vision Disorders

Unit 1 Assessment and Diagnostic Evaluation of Disorder of the Eye and Vision
Unit 3 Caring for Patients with Impaired Vision: Refractive Errors; Low Vision and Blindness; Glaucoma; Catarac
Unit 4 Caring for Patients with Corneal and Retinal Disorders: Corneal Dystrophies; Keratoconus; Corneal Surgeries; Refractive Surgeries; Retinal Detachment; Retinal Vascular Disorders; Macular Degeneration

Module 3 Caring for Patients with Inflammatory Conditions

Unit 1 Caring for Patients with Infection/Inflammatory Conditions: Orbital and Ocular Trauma, Dry Eye Syndrome, Conjunctivitis; Uveitis; Orbital Cellulitis

Unit 2	caring for patients with orbital tumors and orbital Surgeries/ Enucleation
Unit 3	Caring for Patients with Ocular Consequences of Systemic Disease: Diabetic Retinopathy; Cytomegalovirus; Retinitis; Hypertension-Related Eye Changes
Unit 4	Concept in Ocular Medication Administration

COURSE IMPLENTATION
DOING THE COURSE

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

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

COURSE REQUIREMENTS AND EXPECTATIONS OF YOU

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

1. Be versatile in basic computer skills
2. Participate in all laboratory practical up to 90% of the time

3. Submit personal reports from laboratory practical sessions on schedule
4. Log in to the class online discussion board at least once a week and contribute to ongoing discussions.
5. Contribute actively to group seminar presentations.

EQUIPMENT AND SOFTWARE NEED TO ACCESS COURSE

You will be expected to have the following tools:

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

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

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

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

COURSE EVALUATION

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

- **In-Course Examination:**

In line with the university's regulation, in-course examination will come up in the middle of the semester these would come in form of Computer

Marked Assignment. This will be in addition to 1compulsory Tutor Marked Assignment (TMA's) and three Computer marked Assignment that comes after every module…..

- **Laboratory practical:** Attendance, record of participation and other assignments will be graded and added to the other scores form other forms of examinations.

- **Final Examination:** The final written examination will come up at the end of the semester comprising essay and objective questions covering all the contents covered in the course. The final examination will amount to 60% of the total grade for the course.

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

GRADING CRITERIA

Grades will be based on the following Percentages		
Tutor Marked Individual Assignments	10%	
Computer marked Assignment	10%	
Group assignment	5%	40%
Discussion Topic participation	5%	
Laboratory practical	10%	
End of Course examination	60%	

GRADING SCALE
A = 70-100
B = 60 - 69
C= 50 - 59
F = $\leq$ 49

SCHEDULE OF ASSIGNMENTS WITH DATES

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

SPECIFIC READING ASSIGNMENTS

To be provided by each module

REFERENCES AND FURTHER READING

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

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

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

MAIN COURSE

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MODULE 1 CARING FOR PATIENTS WITH URINARY SYSTEM DISORDERS BISOLA BANKOLE

UNIT 1 ASSESSMENT AND DIAGNOSTIC EVALUATION OF URINARY DISORDERS OF THE URINARY SYSTEM

CONTENTS

1.0 INTRODUCTION

The urinary system is a very important system. Urine production and elimination are one of the most important mechanisms of body homeostasis all body systems are directly or indirectly affected by kidney function e.g. composition of blood is determined more by kidney function than by diet main function of kidneys is to get rid of metabolic wastes !typically referred to as “excretory system” excretory wastes = metabolic wastes. chemicals & toxins produced by cells during metabolism. Some other functions include removal of metabolic wastes & toxins but we have several organs that serve an excretory function other than kidneys: skin, sweat glands rid body of water, minerals, some nitrogenous wastes (ammonia), lungs rid body of CO2 from energy metabolism of cells, liver; liver excretes bile pigments, salts, calcium, some toxins, elimination of excess nutrients & excess hormones, helps to regulate blood volume & pressure. blood pressure is directly affected by the volume of fluids retained or removed from body: eg. excessive salts promote water retention greater volume increases BP e.g. dehydration lower volume decreases BP, regulation of electrolytes & body pH any compromise to this system causes various deteriorating medical ailments. It is therefore important to assess

MODULE 2 CARING FOR PATIENTS WITH EYE AND VISION DISORDERS

UNIT 1 ASSESSMENT AND DIAGNOSTIC EVALUATION OF DISORDER OF THE EYE AND VISION

CONTENTS

1.0 INTRODUCTION

Examination of the eye is essential to the diagnosis and management of ocular disease and trauma. Eye examination entails a systematic assessment of the eye and its accessory structures (adnexia). The accessory structures include the eye lids, eye lashes, eye brow, lacrimal apparatus, fibrous/fatty tissue and extra-ocular muscles of the eye

2.0 OBJECTIVES

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

- discuss steps in caring for patients with vision disorder**.**

3.0 MAIN CONTENT

3.1 Pen-Torch Examination of the Eye

This reveals findings that are significant to making a diagnosis. It may demonstrate that the eye is in fact healthy or diseased, or detect the presence of trauma or a foreign body.

Examination of the eye should be systematic.it is usual to look first at the eye that is thought to be diseased or injured. However if it a general check-up, the systematic process is followed and the right eye is first examined. Also, the eye is examined from outward inward shining the light intermittently
General overview of the face and eye- does the eye look normal? Is there facial symmetry? Are there any signs of disease or trauma? Are the eye proptosed? Is the patient using the eye brow to open the eye lids? Does the patient adopt a particular inclination of the head to see?

Eyelids- observe the lids for abnormalities such as ptosis, inflammation, redness, oedema, trauma (old and new), swelling. Examine the eye lid margin for redness, scales, dandruff, orientation of the eyelashes – are they turning in (entropion) or out (ectropion), are they scanty? Are the margin swollen? Is the blink rate normal (every 3-6seconds), are the lids and puncta in good apposition to the globe?

Lacrimal apparatus – for pus, for absence or blocked punctum, swelling or tenderness, foreign body

Conjunctiva- the following areas should be examined carefully; bulbar area, palpebral area and the fornices.

To examine the bulbar conjunctiva, gently pull down the lower lids to expose the lower bulbar conjunctiva. After viewing this, gently push up the upper lid, asking the patient to look down to expose the superior bulbar areas. By asking the patient to look in all directions of gaze, all the area can be viewed.
To examine the palpebral conjunctiva, it is necessary to evert the lids, then, check for presence of papillae, follicles and cysts.

Sclera- for the colour, growth etc.
Cornea- check for opacity, curvature, encroachment of conjunctival tissue. Using a loupe after staining with fluorescein for ulceration, pannus formation, haziness, opacity, foreign body, laceration etc.

Anterior Chamber- AC examination is possible using a pen-torch but a better estimation is done using a slit-lamp. Check for presence of pus (hypopyon), presence of blood (hyphaema), depth etc.

Iris- Note the colour, size, regularity, reaction to light
Pupils- assess the pupils for shape and size, reaction to light, pupil response
Lens- assess for opacity, aphakia (no lens), pseudophakia (artificial lens)

Globe – palpated to estimate intra ocular pressure, alignment symmetry, phithis bulbi Fundus – to assess disc colour and cupped disc ratio

3.2 Visual Acuity

This can be define as the ability of an eye to identify letters or objects of a given size at a given distant. This is done to determine the accurateness or clearness of the central vision, both distant and near. Determination of visual acuity should be part of every eye examination.

There are basically three types of visual acuity; Distance, Near and Colour. Reasons for taking visual acuity include;

- Pre-operative assessment,
- Post-operative assessment,
- Routine medical examination,
- To assess the improvement or otherwise after any ocular surgery (e.g. cataract extraction) or medical procedure,
- To assess the seeing and reading ability of our patients,
- For compensation in cases of individual injury (in time of litigation),
- To detect any ocular defect,
- To ensure safety at work and vocation e.g. driving, pilot

3.3 Distance Visual Acuity

i. Locate patient 6metres away from the chart sitting down.
ii. Occlude the left eye and ask him/her to read with the right eye - record outcome
iii. Occlude the right eye and ask him/her to read with the left eye - record outcome
iv. If VA is less than 6/9, repeat step (ii) and (iii) using a pinhole or glasses
v. If VA is less than 6/60; reduce distance by 1 meter by moving the chart toward the patient till you reach 1 meter
vi. If patient cannot perceive hand movement then determine if patient could perceive light . record outcome
vii. If patient cannot perceive light then record outcome as being NPL- No perception of light

The VA is recorded as a fraction, the numerator representing the distance at which patient can read the chart, over the denominator which represents the distance at which the normal eye could see or read e.g. 6/60 means that

what a normal eye could read at 60m the patient could read this at a much nearer distance of 6metres.

3.4 Near Visual Acuity

Under good illumination, Patient is ask to read the Romans test type or Snellen's near test type chart at the normal reading distance of 33cm from the eye. Record the findings.
Patient who wears glasses for reading are also tested with their glasses on.

3.5 Colour Visual Acuity

This is ability of the patient to recognize different colours. The preferred book of choice is the Ishihara book. Ishihara test type is a book of colour plates having confusion dots against multi-colours.

The patient is requested to hold colour chart at 33cm reading distance, then identify the figures or trace the dots of colours on the book.

Inability to trace the confusion dots indicates colour blindness
If thirteen – seventeen or more plates are traced out of twenty-five, the vision is normal; but less than that there is deficiency in the vision

TONOMETRY
It is a method of measuring IOP based on the principle of indentation and flattening.

Intraocular pressure can be assessed in various ways. The simplest form is the use of digital palpation. Place two fingers on the closed eye lids and feel the eye.

Other ways include the use of an instrument called a "Tonometer" and are of two types:

1. **The Schiotz Tonometer:** this measures the pressure from the resistance of the cornea to indentation. The higher the pressure the greater the resistance.

2. **The Applanation Tonometer:** This is a more accurate instrument in that it works by piercing a flat, round surface against the cornea and measuring the size of the circle it flatters. This method deforms the eye much less and therefore given a much more accurate result.

The Goldman's applanation tonometer is always attached to a slit lamp while the Perkin's tonometer is hand-held and hence portable.

OPHTHALMOSCOPY

Ophthalmoscopy is the examination of the eye with the aid of an ophthalmoscope. It is done primarily to assess the state of fundus and detect opacities of ocular media. Ophthalmoscopy can either be direct (providing a binocular view) or indirect (providing a stereoscopic view).

DIRECT OPHTHALMOSCOPY

An ophthalmoscope is a portable piece of equipment that provides sources of magnification, illumination and light filters. It is usual to dilate the patient's pupils before the examination. This is the most commonly practiced method for routine fundus examination.

INDIRECT OPHTHALMOSCOPY

This piece of equipment is a binocular, stereoscopic, head-set device that allows examiner to gain a wide-field view of both the vitreous and the retina. The ophthalmoscope is used with a hand-held, 20-diopter lens.

SLIT-LAMP EXAMINATION

The slit lamp is a binocular microscope mounted on a table. This instrument enables the user to examine the eye with magnification of 10 to 40 times the real image. The illumination can be varied from a broad to a narrow beam of light for different parts of the eye. For example, by varying the width and intensity of the light, the anterior chamber can be examined for signs of inflammation. Cataracts may be evaluated by changing the angle of the light. When a hand-held contact lens, such as a three-mirror lens, is used with the slit lamp, the angle of the anterior chamber may be examined, as may the ocular fundus.

GONIOSCOPY

Gonioscopy visualizes the angle of the anterior chamber to identify abnormalities in appearance and measurements. The gonioscope uses a refracting lens that can be a direct or indirect lens. The indirect lens views the mirror image of the opposite anterior chamber angle and can be used only with a slit lamp. The direct gonioscopic lens gives a direct view of the angle and its structures.

FLUORESCEIN ANGIOGRAPHY

Fluorescein angiography evaluates clinically significant macular edema, macular capillary non-perfusion, and identifies retinal and choroidal neovascularization (i.e. growth of abnormal new blood vessels) in age-

related macular degeneration. It is an invasive procedure in which fluorescein dye is injected, usually into an ante-cubital area vein. Within 10 to 15 seconds, this dye can be seen coursing through the retinal vessels. Over a 10 minute period, serial black-and-white photographs are taken of the retinal vasculature. The dye may impart a gold tone to the skin of some patients, and urine may turn deep yellow or orange. This discoloration usually disappears in 24 hours.

ULTRASONOGRAPHY

Lesions in the globe or the orbit may not be directly visible and are evaluated by ultrasonography. A probe placed against the eye aims the beam of sound. High-frequency sound waves emitted from a special transmitter are bounced back from the lesion and collected by a receiver that amplifies and displays the sound waves on a special screen. Ultrasonography can be used to identify orbital tumors, retinal detachment, and changes in tissue composition.

UNIT 2 CARING FOR PATIENTS WITH IMPAIRED VISION: REFRACTIVE ERRORS; LOW VISION AND BLINDNESS; GLAUCOMA; CATARACT

CONTENTS

1.0 INTRODUCTION

This unit will focus on caring for patients with impaired vision. You are to note the issues raised.

2.0 OBJECTIVE

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

- Highlight steps in caring for patients with impaired vision:

3.0 MAIN CONTENT

3.1 Refractive Errors

The refractive state of the eye is determined by the combination of corneal power, lens power, anterior chamber (AC) depth and axial length of the eyeball.

Normal (emetropic) eye is the refractive state of an eye where parallel rays of light from near and distant objects are focused on the retina. The image formed by an emmetropic eye is usually clear.

In refractive error (ammetropia), the optical system of the eye fails to focus parallel rays of light on the retina.

In farsightedness (hypermetropia), a near image is focused behind the retina because the eye ball is too short. The total power of the eye is abnormal i.e. inadequate. Distant objects are focused normally, but close vision is blurred. A biconvex lens corrects this anomaly.

In nearsightedness (myopia), the eyeball is too long and distant objects are focused in front of the retina. The total power of the eye is abnormal i.e. excessive. Close objects are focused normally, but distant vision is blurred. Correction is achieved using a biconcave lens.

Astigmatism is the abnormal curvature of part of the cornea or lens. This interferes with the light path and prevents focusing of light on the retina, causing blurred vision. Correction requires cylindrical lenses. It may coexist with hypermetropia, myopia or presbyopia.

DIAGNOSIS
A refractive error may be roughly estimated by use of Snellen's chart or by determining the individual's vision at different distances and comparing it with that of the examiner.

To obtain a more definitive refractive error measurement, a retinoscopic examination is necessary. Before this examination, a cycloplegic drug is often instilled. A cycloplegic drug dilates the pupil and temporarily paralyzes the ciliary muscle, thus preventing accommodation.

3.2 Low Vision and Blindness

Low vision is a general term describing visual impairment that requires patients to use devices and strategies in addition to corrective lenses to perform visual tasks.

Blindness can be defined as inability to count fingers in day-light at a distance of 3meters OR a visual acuity of less than 3/60 or its equivalent OR visual field of less than 10° in the better eye with the best possible correction (WHO, 1979)

In the 9^{th} revision of the International Classification of Diseases (ICD), the visual impairment (maximum vision less than 6/18 Snellen) has been divided into 5 categories. Categories 1 and 2 constitute "low vision" and categories 3, 4 and 5 constitute "blindness". Patients with the visual fields

between 5° and 10° are placed in category 3 and those with less than 5° in category 4.

Category of visual impairment		**Level of visual acuity (Snellen)**
Normal vision	0	6/6 to 6/18
Low vision	1	Less than 6/18 to 6/60
	2	Less than 6/60 to 3/60
Blindness	3	Less than 3/60 (FC at 3 m) to 1/60 (FC at 1m) or Visual field between 5°and 10°
	4	Less than 1/60 (FC at 1 m) to light perception or visual field less than 5°
	5	No light perception

Categories of visual impairment (WHO, 1977)

Blindness can also be categorized into two, namely;

1. **Avoidable blindness:** the concept of avoidable blindness includes both preventable blindness and curable blindness

   i. Preventable blindness- is that which can be easily prevented by attacking the causative factor at an appropriate time. For example, trachoma, onchocerciasis, corneal blindness due to vitamin A deficiency can be prevented by timely measures.
   ii. Curable blindness- is that in which vision can be restored by timely intervention. For example, leprosy, cataract blindness can be cured by surgical treatment.

2. **Non-avoidable blindness:** these are causes of blindness that are due to degenerative changes, metabolic disorders, congenital disorders. For example, diabetes, retinopathy, macular degeneration, haemorrhagic changes etc.

CAUSES OF BLINDNESS

Globally, there are major causes of blindness. They are;
Cataract
Glaucoma
Corneal scarring including trachoma
Age related macular degeneration
Diabetic retinopathy
Childhood blindness including xerophthalmia

Onchocerciasis
Refractive errors and low vision etc.

MANAGEMENT OF LOW VISION

Managing low vision involves magnification and image enhancement through the use of low-vision aids and strategies and through referrals to social services and community agencies serving the visually impaired.

The goals are to enhance visual function and assist patients with low vision to perform customary activities. Low-vision aids include optical and non-optical devices.

The optical devices include convex lens aids, such as magnifiers and spectacles; telescopic devices; anti-reflective lenses that diminish glare; and electronic reading systems, such as closed-circuit television and computers with large print. Continuing advances in computer software provide very useful products for patients with low vision. Scanners teamed with the appropriate software enable the user to scan printed data into the computer and have it read by computer voice or to increase the magnification for reading. Magnifiers can be hand-held or attached to a stand with or without illumination. Telescopic devices can be spectacle telescopes or clip-on or hand-held loupes.

Non-optical aids include large-print publications and a variety of writing aids. The Internet continues to expand, and a telephone system has been developed that allows access to the Internet and e-mail using voice commands

MANAGEMENT OF BLINDNESS

Medical treatment for blindness centers on treating the underlying condition and preventing further impairment. Depending on the cause of the blindness, treatment may include medication prescription, surgical intervention, corrective eyewear prescription, and referral to supportive services.

NURSING PROBLEMS

- Disturbed sensory perception: visual related to altered sensory reception
- Self-care deficit (specify area) related to visual impairment
- Risk for injury related to visual impairment
- Risk for impaired home maintenance related to lack of assistance, lack of rehabilitation, or other factors

- Interrupted family processes related to change in role secondary to visual impairment
- Ineffective role performance related to visual impairment, lack of rehabilitation
- Deficient knowledge related to disease process, prevention, and treatment due to lack of prior exposure
- Deficient diversional activity related to transition from sighted to visually impaired
- Fear related to blindness
- Anxiety related to sensory perception changes (visual)

3.3 Glaucoma

Glaucoma is a group of eye disorders characterized by a progressive optic neuropathy resulting in cupping of the optic disc, irreversible visual field defect and increased intraocular pressure

It is a silent progressive disease and is one of the leading preventable eye diseases if arrested before significant effects on vision occur.

CLASSIFICATION OF GLAUCOMA

The classification of glaucoma depends on the following factors:

1. Congenital/Developmental glaucoma
2. According to the appearance of the drainage angle (open angle or closed angle)
3. Presence of any other factors that may contribute to the rise in IOP;
    i. Primary glaucoma has no other ocular disorders associated with a rise in IOP
    ii. Secondary glaucoma is associated with another condition such as inflammation, neo-vascular disease, etc. and accounts for one-third of all glaucoma cases

CONGENITAL/DEVELOPMENTAL GLAUCOMAS

These are group of diverse disorders in which there is abnormal high intraocular pressure as a result of developmental abnormalities of the angle of anterior chamber obstructing the drainage of aqueous humour.

Sometimes, glaucoma may not occur until several years after birth; therefore, the term developmental glaucoma is preferred to describe such disorders

Depending upon the age of onset, developmental glaucoma is termed as follows:

1. **New born glaucoma**, also called true congenital glaucoma. Is labeled when IOP is raised during intrauterine life and child is born with ocular enlargement. It accounts for 25% of cases

2. **Infantile glaucoma**, is labeled when the disease manifests prior to the child's third birthday. It accounts for about 65% of cases

3. **Juvenile glaucoma**, is labeled when there is increased IOP after 3years but before adulthood.

When the disease manifests prior to age of 3 years, the eyeball enlarges and so the term 'buphthalmos' (bull-like eyes) is used.

SIGNS AND SYMPTOMS
Lacrimation (excessive tears)
Photophobia (fear of light)
Blepharospasm
Corneal oedema and enlargement
Sclera appears thin and bluish
Deep anterior chamber
Cupping of the optic disc especially after third year
Raised IOP
Axial myopia

DIAGNOSIS
A complete examination under general anaesthesia (EUA) should be performed on child suspected of having congenital glaucoma. The examination should include the following;

i. Measurement of the IOP
ii. Measurement of the corneal diameter
iii. Slit lamp examination
iv. Ophthalmoscopy to evaluate optic disc
v. Gonioscopic examination of angle of anterior chamber

MANAGEMENT
Treatment of congenital glaucoma is primarily surgical. However, IOP must be lowered by medical treatment with hyperosmotic agents, acetazolamide and beta-blockers till surgery is taken up.

Surgical procedures are:

i. Incisional angle surgery; it can be performed by either internal approach (goniotomy) or external approach (trabeculectomy)
ii. Filteration surgery is required in many cases;
    1. trabeculotomy with antimetabolites,
    2. Combined trabeculectomy and trabeculotomy with antimetabolites has been accepted as the standard procedure
iii Glaucoma drainage devices (GDD) are required in incalcitrant cases

PRIMARY OPEN ANGLE GLAUCOMA

This is the most common. It is when the optic nerve damage results in a progressive loss of visual field. There is a gradual rise in the IOP. The increased pressure is caused by trabecular blockage which is where the aqueous humour of the eye drains out. The pressure builds up in the eye and causes imperceptible gradual visual loss. Peripheral vision is affected first but eventually the entire vision will be lost if not treated.

SIGNS AND SYMPTOMS

Asymptomatic until it has caused a significant loss of visual field. Therefore, periodic eye examination is required after middle age.
Painless
Scotoma (defect in the visual field)
Difficulty in reading near print which usually may lead to frequent changing of presbyopic glasses
Loss of vision usually the peripheral vision is lost before the central vision
The patient bumps into objects
Slowly progressive raised IOP
Cupping of the optic disc
On gonioscopic examination, the anterior chamber (AC) is widely opened.

MANAGEMENT

Therapeutic choices are; medical therapy, laser trabeculoplasty, and filtration surgery

A. MEDICAL THERAPY

1. Single drug therapy: drugs under this category include topical beta-blockers (e.g. timolol maleate 0.25%, 0.5%), prostaglandin analogus (e.g. latanoprost 0.005%), adrenergic drugs (e.g. epinephrine hydrochloride 0.5%, 1%, 2%), topical carbonic anhydrase inhibitor (e.g. dorzolamide 2%), and pilocarpine 1%, 2%, 4%
2. Combination topical therapy; this is instituted whenever it is observed that one drug is no more effective.
3. Oral carbonic anhydrase inhibitors; this is used to control the IOP on a short term basis

4. Hyperosmotic agents like mannitol 1-2gm/kg body weight may be used initially when patients present with very high IOP

B. LASER TRABECULOPLASTY

Laser trabeculoplasty is used in patients where the IOP could not be controlled despite maximal tolerated medical therapy. It is also considered when there is non-compliance to medical therapy. Commonly laser procedures include argon laser trabeculoplasty (ALT), or diode laser trabeculoplasty (DLT) and selective laser trabeculoplasty (SLT).

C. SURGICAL THEREAPY

This is indicated when there is;

1. Uncontrolled glaucoma despite maximal medical therapy and laser trabeculoplasty
2. Non-compliance of medical therapy and non-availability of ALT/SLT
3. Failure of medical therapy and ineffective ALT
4. Eyes with advanced disease i.e. having very high IOP, advanced cupping and advanced field loss should be treated with filtration surgery as primary line of management.

PRIMARY ANGLE CLOSURE GLAUCOMA

PACG results from gradual synechial closure of the angle of anterior chamber. It is characterized by apposition of peripheral iris against the trabecular meshwork resulting in obstruction of aqueous outflow by closure of an already narrow angle of the anterior chamber. This is the most common in people over 40years of age and usually affects one eye.

SIGNS AND SYMPTOMS

Sudden severe pain
Photophobia
Headache
Nausea and vomiting
Raised IOP
Glaucomatous cupping of the optic disc
Visual field defect
Gonioscopy reveals more than 270 degree of angle closure along with peripheral anterior synechiae

MANAGEMENT

Acute angle closure is a serious ocular emergency condition and needs to be managed aggressively as below:

A. Immediate medical therapy to lower the IOP,
B. Definitive treatment,
C. Prophylaxis of the fellow eye, and
D. Long term glaucoma surveillance and IOP management of both eyes

A: Immediate medical therapy:

a) Hyperosmotic agents; this can either be systemic e.g. intravenous mannitol (1gm/kg body weight) or oral e.g. glycerol 1gm/kg body weight
b) Systemic carbonic anhydrase inhibitors e.g. acetazolamide (Diamox) 500mg intravenous stat then 250mg orally tds
c) Topical anti-glaucoma drugs. For examples prostaglandin analogue (e.g. latanoprost 0.005%), beta blockers (e.g. timolol 0.5%), pilocarpine 2%
d) Analgesics and anti-emetics to alleviate the symptoms
e) Topical steroid e.g. prednisolone acetate 1%

B: Definitive therapy:

a) Laser Peripheral iridotomy or laser iridotomy (with Nd:YAG lase or Argon Laser)
b) Filtration surgery i.e. trabeculectomy
c) Clear lens extraction by phacoemulsification especially in the presence of cataract

C: Prophylactic treatment of the other eye

The use of prophylactic laser iridotomy

NURSING PROBLEM

- Pain related to increased intraocular pressure
- Disturbed sensory perception: visual related to altered sensory reception
- Self-care deficit related to decreased vision
- Anxiety related to partial or total visual loss
- Risk for injury related to decreased vision
- Impaired home maintenance related to decreased vision
- Deficient knowledge related to medical regimen, disease process due to no prior experience

3.4 Cataract

Cataract is the opacity of the crystalline lens of the eye following changes in the physical and chemical characteristics. It is a common cause of

treatable blindness. The lens is normally transparent to allow light to enter the eye. It alters its shape in order to focus objects at varied distance (accommodation). The lens refracts light rays and brings them to focus on the retina.

CLASSIFICATION

Cataract can be classified according to the following:
1. Causes- traumatic, metabolic, inflammatory, radiation
2. Age of onset- congenital, senile, juvenile
3. Degree of Maturity- incipient, hyper-mature
4. Position of opacity- nuclear, cortical, zonular

CAUSES

Aging/ Senility
Heredity
Congenital
Trauma/ injury
Exposure to radiation
Metabolic e.g. diabetes mellitus
Drugs like steroids, anti-psychotics e.g. phenothiazine

SIGNS AND SYMPTOMS

Blurred vision
White speck in the eye
Diplopia
Refractive changes
Visual field loss
Changes in colour of object seen i.e. colour shift
Difficulties in reading small print
Difficulties in seeing distant objects

MANAGEMENT

This depends on the cause, maturity and whether cataract is responsible for reduced vision or whether surgery will improve the vision.

A developing cataract can be managed with changes in the spectacle prescription.

In a patient with cataract accompanied with retinal disorders or diabetes mellitus, the level of vision in relation to the patient's lifestyle and functional capabilities is really the only determining factor that indicates whether or not surgery is necessary.

Surgical management is the only proven way, but even at that, a number of investigations must be undertaken to obtain the best possible outcome for vision, as part of a preoperative assessment.

INDICATIONS FOR SURGERY

Optical- to improve vision
Therapeutic- as in leaking lens causing uveitis, glaucoma
Prophylactic- to prevent complication as in hyper-mature
Cosmetic- where opacity is disfiguring

CHOICE OF SURGICAL TECHNIQUES

1. Intracapsular cataract extraction (ICCE) - Obsolete
2. Extracapsular cataract extraction (ECCE)
3. Small incision cataract surgery (SICS)
4. Phacoemulsification
5. Irrigation and aspiration (in Children)
6. Lensectomy (in Children)

PREOPERATIVE ASSESSMENT

A comprehensive preoperative assessment is required and this may take place on the same day as the patient's visitation to the clinic. This includes;

- History taking- family history, past medical history, details of current medication, history of allerges etc.
- General examination from head to toe
- Complete ocular examination
- Investigations including, visual acuity, biometry, conjuctival swab for microscopic culture and sensitivity, blood sugar test
- Identification of social problems, which may require support so that services may be arranged and surgery is not delayed.

This assessment facilitates better opportunities to provide more detailed information in relation to the perioperative management of cataract for patients. Increased patients involvement and subsequent cooperation in care planning lead to the negotiation of much more flexible approaches to perioperative care.

COMPLICATION OF CATARACT SURGERY

Retrobulbar haemorrhage
Vitreous loss
Retinal detachment

Hyphaema
Shallow anterior chamber
Iris prolapsed
Secondary glaucoma.

UNIT 3 CARING FOR PATIENTS WITH CORNEAL AND RETINAL DISORDERS: CORNEAL DYSTROPHIES; KERATOCONUS; CORNEAL SURGERIES; REFRACTIVE SURGERIES; RETINAL DETACHMENT; RETINAL VASCULAR DISORDERS; MACULAR DEGENERATION

CONTENTS

1.0 INTRODUCTION

Corneal dystrophies are inherited disorders in which the cells have some inborn defects due to which pathological changes may occur with passage of time leading to development of corneal haze in otherwise normal eyes that are free from inflammation or vascularization. You are going to learn more about this as you continue the study.

2.0 OBJECTIVES

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

- Discuss the nitty-gritty of caring for patients with corneal and retina disorders.

3.0 MAIN CONTENT

3.1 Corneal Dystrophies

Corneal dystrophies are inherited disorders in which the cells have some inborn defects due to which pathological changes may occur with passage of time leading to development of corneal haze in otherwise normal eyes that are free from inflammation or vascularization. There is no associated systemic disease. Dystrophies occur bilaterally, manifesting occasionally at birth, but more usually during first or second decade and sometimes even later in life.

CLASSIFICATION
The International Committee for Classification of Corneal Dystrophies (ICCCD) in 2008 classified corneal dystrophies as below:

I. Epithelial and Sub-epithelial Dystrophies
- i. Epithelial basement membrane dystrophy (EBMD)
- ii. Epithelial recurrent erosion dystrophy (ERED)
- iii. Subepithelial mucinous corneal dystrophy (SMCD)
- iv. Mutation in keratin genes: Messmann corneal dystrophy (MECD)
- v. Lisch epithelial corneal dystrophy (LECD)
- vi. Gelatinous drop-like corneal dystrophy (GDLD)

II. Bowman Layer Dystrophies
- i. Reis-Bucklers corneal dystrophy (RBCD) – Granular corneal dystrophy type 3
- ii. Thiel-Behnke corneal dystrophy (TBCD)
- iii. Grayson-Wilbrandt corneal dystrophy (GWCD)

III. Stromal Dystrophies
- i. Lattice corneal dystrophy (LCD)
- ii. Granular corneal dystrophy (GCD)
- iii. Macular corneal dystrophy (MCD)
- iv. Schnyder corneal dystrophy (SCD)
- v. Congenital stromal corneal dystrophy (CSCD)
- vi. Fleck corneal dystrophy (FCD)
- vii. Posterior amorphous corneal dystrophy (PACD)
- viii. Central cloudy dystrophy of Francois (CCDF)
- ix. Pre-Descemet corneal dystrophy (PDCD)

IV. Descemet Membrane and Endothelial Dystrophies

i. Fuchs endothelial corneal dystrophy (FECD)
ii. Posterior polymorphous corneal dystrophy (PPCD)
iii. Congenital hereditary endothelial dystrophy (CHED)
iv. X-linked endothelial corneal dystrophy (XECD)

3.2 Keratoconus

It is a non-inflammatory bilateral form of corneal degeneration in which the cornea becomes thin and protrudes anteriorly (cone-shaped central cornea). The hereditary condition has a higher incidence among women. Onset occurs at puberty; the condition may progress for more than 20 years and is bilateral. Corneal scarring occurs in severe cases. Blurred vision due to progressive myopia and irregular astigmatism is a prominent symptom which does not improve fully despite full correction with glasses.

MANAGEMENT

Spectacles
Use of hard contact lens
Keratoplasty may be require at a later stage when contact lens correction is no longer effective.

3.3 Corneal Surgery

KERATOPLASTY

This is an operation in which the patient's diseased cornea is replaced by the healthy clear cornea. It is also known as corneal grafting or corneal transplantation. The donor eye should be removed within 6 hours of death and should be stored under sterile conditions.

TYPES

1. Autokeratoplasty – using self-cornea
2. Allografting or Allo-keratoplasty- using the donor's healthy cornea

INDICATIONS

Optical – to improve vision as seen in corneal opacity, corneal dystrophies, advanced keratoconus
Therapeutic – to replace inflamed cornea not responding to conventional therapy
Cosmetic – to improve the appearance of the eye
Tectonic graft – to restore the integrity of the eye ball as seen in corneal perforation, marked corneal thinning.

COMPLICATIONS

Early complications are flat anterior chamber, iris prolapsed, infection, secondary glaucoma, primary graft failure

Late complications are graft rejection, recurrence of disease and astigmatism

4.4 Refractive Corneal Surgeries

Surgery and/or laser is used to reshape the main refractive surface of the eye, (anterior corneal surface), and to bring light rays in focus on the retina without the need for glasses or contact lenses. In myopia the corneal surface is flattened so that the image focuses onto the retina. The effect in hypermetropia is not always stable.

Major refractive corneal surgery includes;

i. Radial keratotomy (RK)
ii. Astigmatic keratotomy (AK)
iii. Photorefractive keratotomy (PRK)
iv. Laser assisted in-situ keratomileusis (LASIK) and its varieties
v. Thermal laser keratoplasty (TLK)
vi. Conductive keratoplasty (CK)
vii. Orthokeratoplasty – it refers to the molding of the cornea with overnight wear of unique rigid gas permeable contact lens. It is a non-surgical reversible method.
viii. Intracorneal ring (ICR) implants
ix. Phototherapeutic keratectomy (PTK) – it refers to the ablation of superficial corneal lesion with thehelp of excimer laser (198nm). It is used for patients with superficial corneal scars, corneal degenerations, and recurrent corneal erosions.
x. Keratoprosthesis – it refers to the use of an artificial corneal device in patients unsuitable for keratoplasty.

3.5 Retinal Detachment

This is the separation of the neurosensory retina from the retinal pigment epithelium

TYPES

1. **Rhegmatogenous RD:** here, the separation is caused by a full-thickness break (holes, tears or breaks) in the continuity of the retina,

allowing fluid to collect between the neural retina and retinal pigment epithelium as a result of the tension between the vitreous and the retina. This type of retinal detachment can also be precipitated by moderate trauma, such as stooping or lifting weights, or by direct trauma to the eye.

It is the most common type of detachment.

2. **Non-rhegmatogenous RD:** it occurs in the absence of holes. It occurs when fibrous tissue in the vitreous humor attaches to the sensory retina and, as it contracts, pulls the retina away from its normal position, as seen in patients with sickle cell disease or diabetes mellitus.

   It is of two types: traction and exudative

PREDISPOSING FACTORS

1. Any condition that weakens the retina such as lattice degeneration of the retina
2. Any condition that cause proliferative retinal degeneration e.g. toxaemia, sickle cell disease
3. Retinoschisis- this is splitting of the neurosensory retina into two
4. Myopia- the globe is big in size resulting in the thinning of the wall leading to detachment
5. Trauma causing a hole or break as a result of vitreous pull on the retina
6. Aphakic eye- this results when there is vitreous loss during surgery

SIGNS AND SYMPTOMS

Sudden painful loss of vision
Quadratic field defect
Dark cotton/veil over the field of vision
Flashes of light
Floaters or specks in the field of vision

INVESTIGATIONS

Detailed history taking especially the duration of symptoms
Eye examination including visual acuity to detect low vision, visual field defect
Slit lamp examination to detect any abnormality

PRINCIPLE OF MANAGEMENT

1. Rest- strict bed rest to allow the retinal detachment to subside before surgery is carried out.
2. Position- the area of the detachment should be in dependent position
3. Surgery- through cryotherapy, LASER photocoagulation, Scleral explants, retinopexy

COMPLICATIONS

Infection
Blindness

3.6 Retinal Vascular Disorders

Common vascular disorders of the retina include: retinal artery occlusions, retinal vein occlusions, diabetic retinopathy, hypertensive retinopathy, sickle cell retinopathy and retinopathy of prematurity

3.6.1 Retinal Artery Occlusions

It is more common in males than females. It is usually unilateral but rarely may be bilateral (1-2% cases)

CAUSES

Atherosclerosis-related thrombosis at the level of lamina cribrosa is the most common cause (75%)
Carotid artery emboli
Retinal arteritis with obliteration
Raised intraocular pressure
Retinal migraine
Sickling haemoglobinopathies
Hypercoagulation disorders such as oral contraceptives, polycythemia

SIGNS AND SYMPTOMS

Sudden complete painless loss of vision
Reduced visual acuity
Absent direct papillary reflex
Marked narrowing of retinal arteries
Milky white retina due to ischaemic oedema
Cherry red spot on the fovea
Cattle tracking, i.e. segmentation of blood column is seen in the retinal vein

MANAGEMENT
It is an ophthalmic emergency as retinal tissue cannot survive ischaemia for more than a few hours
Immediate lowering of IOP by intermittent ocular massage and intravenous mannitol. It may also help in dislodging the embolus.
Vasodilators and inhalation of a mixture of 5% CO_2 and 95% O_2. Patient may breathe into a paper bag
Anterior chamber paracentesis may be considered
Intravenous acetazolamide 500mg should be given immediately
Anticoagulants may be helpful in some cases
Intravenous steroids are indicated in patients with giant cell arteritis

COMPLICATIONS

Neovascular glaucoma

3.6.2 Retinal Vein Occlusions

This is the second most common retinal vascular disease after diabetic retinopathy. It typically affects elderly patients in their sixth or seventh decade of life.

CAUSES
Systemic: contributing factors are; age, hypertension, diabetes mellitus, blood abnormalities (dyscrasias), e.g. sickle cell disease, raised IOP
Ocular: contributuing factors are; hypermetropia, congenital abnormalities, orbital cellulitis, orbital tumours, cavernous sinus thrombosis

CLASSIFICATION
1. Central retinal vein occlusion (CRVO). It may be non-ischaemic CRVO (venous stasis retinopathy) or ischaemic CRVO (haemorrhagic retinopathy)
2. Branch retinal vein occlusion (BRVO)

SIGNS AND SYMPTOMS
Reduced visual acuity
Swollen and reddish fundus with associated dilated tortuous vein
Retinal haemorrhage
Retinal oedema

MANAGEMENT
Panretinal photocoagulation (PRP) may be required to prevent neovascular glaucoma in patients with widespread capillary occlusion

Treatment of the underlying conditions
Intravitreal triamcinolone and anti VEGF (e.g. avastin, lucentis) may be considered for associated cystoids macular oedema (CME) and neovascularization

3.6.3 Retinopathy of Prematurity

It was earlier known as retrolental fibroplasias. Retinopathy of prematurity (ROP) is a bilateral proliferative retinopathy, occurring in premature infants with low birth weight who often have been exposed to high concentration of oxygen.

RISK FACTORS

Low gestation age (especially < 32weeks)
Low birth weight (< 1.5kg)
Supplemental high concentration of oxygen administration

SIGNS AND SYMPTOMS
ROP has been divided into active ROP and cicatricial ROP
Active ROP can be divided into 5 stages clinically, namely;

Stage 1: there is formation of a demarcation line at the edge of the vessels which divides the vascular retina from the avascular retina.
Stage 2: the line structure in stage 1 acquires a volume to form a ridge with height and width
Stage 3: the ridge with extra-retinal fibrovascular proliferates into the vitreous. It is further sub-divided into mild, moderate and severe, depending on the amount of fibrovascular proliferation
Stage 4a: there is subtotal retinal detachment not involving the macula
Stage 4b: there is subtotal retinal detachment involving the macula
Stage 5: there is total retinal detachment which is always funnel shape

MANAGEMENT
Treatment of well-established disease is unsatisfactory, thus prophylaxis is very important.
To prevent ROP, premature babies should be given not more than 30% oxygen concentration.
Screening of all premature babies with ≤ 32 weeks gestational age or < 1.5kg weight for ROP
Protection of premature infants against infection and attacks of apnoea

Vitrectomy especially in stage 5 and 4b

3.7 Macular Degeneration

AGE RELATED MACULAR DEGENERATION
ARMD is also known as senile macular degeneration. It is a leading cause of blindness in developed countries, in population above the age of 65years. It is more prevalent among the Caucasians.
Common risk factors which may affect age of onset and/or progression include heredity, nutrition, and smoking, and hypertension, exposure to sunlight, hyperopia, blue eyes and cataract
It is of two types;

1. **Non-exudative ARMD**: it is also known as dry or atrophic ARMD. It is responsible for 90% of cases.
   [[[

   SIGNS AND SYMPTOMS
   Gradual loss of vision
   Complaint of distorted vision
   Difficulty in reading due to central shadowing
   Retinal pigment atrophy

2. **Exudative ARMD**: it is also known as wet or neo-vascular ARMD. It is responsible for only 10% cases of ARMD but is associated with comparatively rapidly progressive marked loss of vision. There is retinal pigment epithelial detachment (PED), choroidal neovascularization (CNV).

DIAGNOSIS
Slit lamp biomicroscopy with +90D/+78D non-contact lens
Fundus fluorescein angiography
Optical coherence tomography (OCT)

MANAGEMENT
Non-exudative ARMD:
There is no effective treatment, but patient may undergo behavioural modifications like cessation of smoking
Dietary supplements and antioxidants
Refraction and change of glasses may be helpful in the early cases while, low vision aid may be beneficial in advance cases.

Exudative ARMD:

The use of intravitreal anti-VEGF therapy (e.g. Avastin 1.25mg, Lucentis 0.5mg/0.05ml) at interval of 2-3months has been beneficial improving vision in 30-40% of cases

Photodynamic therapy (PDT)

Transpupillary thermotherapy (TTT)

the integrity of the urinary system constantly. The following diagnostic evaluation are useful.

2.0 OBJECTIVES

At the end of this lesson, you will be able to

- Discuss why assessment and diagnosis of the urinary system is important
- Describe the physical examination of the urinary system
- Describe laboratory investigations and other relavant examinations
- Discuss the nursing responsibilities of a patient undergoing any of the investigations

3.0 MAIN CONTENT

3.1 Physical Examination

A complete physical exam is important; however, certain aspects of the exam need to be emphasized. A focused physical examination should be performed to:

1. Assess the bladder for masses and fullness
2. Assess the external genitalia
3. Assess the pelvic floor, including anal sphincter tone, and thoroughly examine for support defects, prolapse, and other pelvic conditions in women
4. Assess the prostate in men
5. Demonstrate incontinence in patients with that symptom
6. Detect neurologic abnormalities that may contribute to dysfunction.

3.2 Abdominal Examination

An abdominal examination should be conducted. It includes examination of the flanks, begins with inspection for scars, masses, or hernias. Examination of the back should be performed to check for scars and scoliosis which may be an indication of potential spine abnormalities that may contribute to dysfunction of the urinary system. Supra pubic palpation is performed to determine if the patient has a distended bladder or pelvic mass.

In women, a systematic examination of the vagina and pelvis is important. This is first done in lithotomy position and may be repeated with the patient standing. The external genitalia are first inspected followed by evaluation of the vaginal mucosa for signs of atrophic vaginitis, indicating estrogen

deficiency, previous surgery, and vaginal discharge. The urethral meatus should be observed and the urethra palpated for any abnormalities. The anterior vaginal compartment is examined next. This can be aided by applying slight pressure wall with the posterior blade of a small vaginal speculum. The position of the urethra, bladder neck, and bladder can be observed at rest and with straining to evaluate support of these structures and determine the presence of urethral hypermobility and cystocele.

Also with coughing and straining, the urethra should be observed for urine loss and whether that loss occurs with hypermobility. The central vaginal compartment is examined next. The uterus and cervix should be evaluated at rest and with straining to determine prolapse. **Bimanual examination** is done to evaluate the presence of uterine, adnexal, or other pelvic masses. If the patient has hysterectomy, the vagina should be assessed for enterocele.

This is often best accomplished by first retracting the anterior vaginal wall and then the posterior wall. Finally the posterior vaginal compartment is examined by retracting the anterior vaginal wall with the speculum blade. A large rectocele is easily identifiable.

3.3 Laboratory Testing

1. Urine analysis; Urinalysis can screen for pyuria, bacte-uria hematuria, and the presence of glucosuria or proteinuria. When abnormalities are found on urine analysis, further testing may be conducted suchas urine culture.A urinalysis (urine analysis) is a commonly performed diagnostic test for the renal system. Urinalysis is an invaluable tool in the diagnosis of kidney disease and other systemic diseases that may affect the kidneys. The results of the urinalysis give information regarding kidney function and various body functions. A routine urinalysis specimen may be collected at any time of day; however, the first morning specimen is best. First morning specimens are usually concentrated and more likely to contain abnormal constituents if they are present. The specimen should be examined within 1 hour of urinating.

   Urine that cannot be examined promptly should be refrigerated. Urine standing at room temperature longer than 2 hours has more bacteria present, changes in pH, and hemolysis of RBCs. Urine collected for cytology should not be a first morning specimen due to changes in epithelial cells in urine held overnight. Random specimens are done for cytology.

To collect a voided specimen for urinalysis, the nurse has the patient wash the perineum using soap and water or a special towelette from a clean-catch midstream urine collection kit. Women should be directed to wash from the front to the back of the perineum. The patient is instructed to begin to void into the toilet, and then move the collection container under the stream, and then finish voiding into the toilet. This is called a clean-catch midstream specimen. It is used to obtain the cleanest possible specimen. Female patients should be told to separate the labia with one hand and keep them separated while washing and collecting the specimen to decrease the risk of contamination of the specimen. If the female patient is menstruating, this should be specified on the laboratory form. A tampon may be used to prevent contamination of the specimen. The uncircumcised male patient should be directed to retract the foreskin with one hand and keep it retracted while cleansing and voiding. At least 10 mL of urine should be collected. If a urinalysis is ordered for a patient with a urinary catheter, the nurse obtains the urine specimen. This specimen is considered sterile because it is coming directly from the bladder into the urinary catheter tubing. To obtain the specimen, wear clean gloves and use an alcohol swab to clean the sample port on the catheter tubing. Insert a blunt needle of a syringe (usually 10 mL) into the port and withdraw urine from the tubing into the syringe. Then empty the urine from the syringe into a collection container and safely dispose of the syringe. Composite urine specimens are collected over a period of time that may range from 2 to 24 hours. These specimens are usually used to examine the urine for specific components such as glucose, electrolytes, protein, 17-ketosteroids, catecholamines, creatinine, and minerals. These specimens may need refrigeration or may have preservatives added to the collection container. The patient is instructed to void and discard this specimen. The time is noted and is the start time of the test. All subsequent voiding is saved in the container for the designated time period. At the end of the time frame, the patient is asked to void and this is added to the container as the last amount to be added. Reminding the patient to save all of the urine is critical for accurate results. Incomplete collections do not result in accurate results.

2. **Urine culture**; it is conducted in cases of suspected infection or urine cytology, endoscopic, and radiographic studies when microscopic hematuria is present.

3. **Blood tests**; they are useful in selected cases of urinary disorders. The most common tests are those that evaluate renal function, e.g., serum blood urea nitrogen and creatinine, quantitative test for protein, creatinine clearance etc. In some select cases, more specific blood and urine testing may be performed, but these are usually dependent on patient history and physical as well as the results of simple tests.

4. **A voiding and intake diary or an intake and output record**; a record of intake and output provides a baseline to assess future treatment. A voiding and intake diary should include the time, type and amount of fluid intake, the time and amount of each void, and any associated symptoms such as incontinence, extreme urgency or pain. The diary should be done for a period of 24 h, and several days are preferred. The patient should also note how representative a particular 24-h period was of his or her normal symptoms. It is useful to describe the nature and quantify the severity of symptoms such as frequency, nocturia and incontinence.

5. **Measurement of post-void residual volume**; the post void residual volume (PVR) is defined as the volume of urine remaining in the bladder immediately following voiding. It provides information on the ability of the bladder to empty as well as its functional capacity (voided volume plus PVR). Normal lower urinary tract function is usually associated with a negligible PVR. Elevated PVR may be an indication of detrusor hypo-contractility or bladder outlet obstruction and may prompt further evaluation depending on the patient and the symptoms or condition being evaluated. PVR can be measured directly by in and out urethral catherizationordeterminednoninvasivelybyultrasonography.Portable bladder scanners based on ultrasound technology can be used to determine bladder volume.

6. **Uroflowmetry**; this is the determination of urinary flow rate over time it is a simple way to measure bladder emptying. In and of itself, auroflow is rarely able to determine the cause of voiding dysfunction; however, in conjunction with a careful history and physical examination, it can provide valuable information. In addition, it is extremely useful in selecting patients for more complex urodynamic testing. Uroflow is measured by a device called a uroflowmeter. Modern uroflowmeters consist of electronic collection equipment with graphic expression of the flow rate as a function of time.

Common parameters determined by uroflowmetry include **Voided volume**: Actual volume of urine voided. **Flow time**: Time during which measurable flow occurs. **Total voiding time**: The total time of void taking into account periods of no flow in the patient with an intermittent pattern. **Maximum flow rate (Qmax):** The highest flow rate achieved during the voiding episode. **Time to maximal flow**: The elapsed time from the beginning of voiding to the point of maximal flow. It is generally about one third of the total voided time. **Mean flow rate (Qave)**: Voided volume divided by flow time. Only interpretable if flow is continuous and uninterrupted. Urinary flow rate and Qmax varies as a function of patient age, sex, anxiety, and voided volume. Uroflow is more commonly utilized in men as opposed to women, probably because of the relatively high incidence of bladder outlet obstruction and decreased flow in elderly men

7. **Urodynamics**; Urodynamics is the study of the transport, storage, and evacuation of urine by the urinary tract. It is comprised of a number of tests that individually or collectively can be used to gain information about lower urinary tract function and can provide a precise diagnosis of the etiology of urinary disorders.

   Multichannel Urodynamics; The various components of the urodynamice valuation include monitoring of bladder pressure during filling (**cystometrogram**), monitoring of bladder pressure and simultaneous urinary flow rate during voiding (voiding pressure flow study), and monitoring of pelvic floor and external sphincter activity (**electromyography**). Usually abdominal pressure is also monitored during filling and voiding so that subtracted detrusor pressure can be determined. In select cases, the urethral pressure can also be assessed during storage and voiding (**urethral pressure profilometry**). It is when these components are combinedtogetheras“multichannel-urodynamics”thatamostsophisticated study of the lower urinary tract is obtained.

8. **Cystometrogram;** the cystometrogram (CMG) is a measure of the bladder’s response to being filled. Normally the bladder should store increasing volumes of urine at low pressure and without involuntary contractions. CMG determines the pressure-volume relationship within the bladder and also provides a subjective measure of bladder sensation with the cooperation of the patient. Ideally, the CMG

should mimic normal bladder filling and gives an accurate assessment of true bladder function. CMG is performed by filling the bladder at a constant rate (usually 10–100 mL/s) with fluid (normal saline or contrast media) or gas (such as carbon dioxide). Filling occurs via a catheter, which is inserted transure thrally or supra pubically. Usually there are two lumens on the catheter, one to measure pressure and one to fill the bladder. Most urodynamisists have abandoned gas cystometry because fluid is more physiologic and allows the determination more parameters.

9. **Renal Function Tests;** A number of blood and urine tests reflect kidney function. If then kidneys are not functioning adequately,these test results will be elevated. These tests are usefulbecause they provide information about the severity of apatient's kidney disease and also the patient's response toany treatments or medications being used. In this way, clinicalprogress can be monitored. Renal function tests maystill be within the normal range until the glomerular filtrationrate is less than 50% of normal. The most accurate wayto assess kidney function is to use several tests and analyzethe results together.

10. **Endoscopic Procedures;** Endoscopic procedures examine the inside of hollow organs with an endoscope. The endoscope is a device consisting of a tube and an optical system. The observations can be done through a natural body opening such as the urethra or a small incision through the skin. Cystourethros copy has been used in the evaluation of patients with lower urinary tract symptoms and voiding dysfunction in order to assess the bladder, the urethra, and prostate and look for extra urethral causes of incontinence. Cystourethroscopy has a more definitive role in men who have undergone surgical treatment of the prostatic for benign or malignant disease when anatomic causes of postoperative voiding dysfunction are suspected (e.g., bladder neck contracture or anastomotic stricture).Extra urethral Incontinence Endoscopy can be an invaluable tool in the diagnosis and treatment of extra urethral incontinence due to vesico vaginal fistula and ectopic ureter. Cystourethros copy can precisely localize a fistula site in the bladder and help plan surgical correction.

3.4 Urinary Tract Imaging

In certain cases of voiding dysfunction, imaging studies, including radiography, ultrasonography, magnetic resonance, and nuclear scanning,

are an important part of the evaluation. Specifically, when detrimental effects on the upper urinary tract or anatomical abnormalities of the upper and lower urinary tract are suspected, such studies can be useful. We will limit our discussion to imaging of the upper and lower urinary tract; however, there are cases where a urologic work- up of voiding dysfunction may prompt radiographic investigation of the nervous system or spine (e.g., in cases of suspected neurogenic voiding dysfunction).

Renal Ultrasound; Ultrasonography is a noninvasive study using sound waves passed into the body through a transducer to detect abnormalities. It is commonly used to examine the anatomy of the urinary tract. Ultrasound requires no contrast media and no preparation. There are also no contraindications to ultrasound.

Other investigations include;

1. **VOIDING PRESSURE FLOW STUDY/Cystometry;** assesses the bladder's response to filling, however, by itself tells nothing about the bladder's ability to empty. This can be determined by allowing a patient to void (voluntarily or involuntarily) during bladder pressure monitoring. When the simultaneous measurement of uroflow is added, i.e., voiding pressure-flow study, detrusor contractility as well as the resistance of the bladder outlet can be determined. In fact, detrusor pressure during voiding is actually determined by the amount of outlet resistance.
2. **ELECTROMYOGRAPHY**; The storage and emptying phases of the micturition cycleare affected by the perineal musculature including the striated external urethralsphincter.Sphincteractivitycanbemeasuredduringurodynamictesting either by surface electrodes (similar to those used for electrocar diogram) or by inserting needle electrodes directly into the sphincter muscle.
3. **VIDEOURODYNAMICS;** in certain cases, multichannel urodynamic testing is unable to provide a precise diagnosis. In such cases video urodynamics may be necessary. Video urodynamics refers to the simultaneous measurement and display of urodynamic parameters with radiographic visualization of the lower urinary tract .In these cases, the bladder is filled with radiographic contrast filling during urodynamics. Because all urodynamic parameters previously mentioned are visualized simultaneously with the radiographic appearance of the lower urinary tract, the clinician can better appreciate their interrelationships and recognize artifacts. Video urodynamics is the most precise way to evaluate lower urinary tract function and disturbances in micturition

NURSING RESPONSIBILITIES DURING THESE DIAGNOSTIC EVALUATIONS

1. Assess the patient for signs and symptoms of anxiety evidenced by verbalization, tenseness, tachycardia, elevated blood pressure, facial pallor, and self-focused behaviors. Anxiety may be manifested by increases in vital signs, pallor, or self-focused behaviors. A high level of fear may interfere with learning and cooperation.
2. Introduce staff who will be caring for the patient. As the Familiarity with staff will decrease anxiety.
3. Orient patient to the environment, equipment, and routines this decreases anxiety.
4. Involve family members or significant others in orientation and teaching sessions to encourage their support of the patient.
5. Assess the patient's understanding of the procedure to provide a baseline for teaching.
6. Explain all activities that will take place in the diagnostic area and afterward. This will reduce fear and promote cooperation during testing.
7. Provide information based on the patient's current needs at the level the patient can understand.
8. Reinforce physician's explanations and clarify misconceptions about the diagnostic test or procedure to help alleviate anxiety.
9. Encourage family members or significant others to provide support to patient without obvious anxiousness as anxious family members will convey those feelings to the patient.
10. Identify allergies the patient may have to contrast agents, or drugs prior to diagnostic testing. This will prevent allergic responses and provide for patient safety.
11. Provide information about injections or invasive procedures that may be done to the patient to reduce anxiety.
12. Explain any unfamiliar machines or equipment to the patient to reduce anxiety.
13. Explain that the patient may have to drink increased fluids after the test and that the patient's intake and output will be closely monitored. Increased fluids help rid the patient of the contrast media after the procedure.
14. Provide information about self-care following the procedure or diagnostic test. This will facilitate the patient in self-care at home.
15. Maintain a calm, supportive, and confident manner when interacting with the patient. This will reduce anxiety.
16. Respond to patient call signals as soon as possible to reduce anxiety.
17. Encourage the patient
18. Document all findings

UNIT 2 REVEIEW OF RELATED ANATOMY & PHYSIOLOGY OF THE URINARY SYSTEM

CONTENTS

1.0 INTRODUCTION

The urinary system consists of two kidneys, two ureters, the urinary bladder, and the urethra. The kidneys form urine, and the rest of the system eliminates urine. The purpose of urine formation is the removal of potentially toxic waste products from the blood. You are going to be learning more about this.

2.0 OBJECTIVES

At the end of this lesson, the learners will be able to

- discuss the function of the urinary system
- describe the anatomy of the urinary system
- describe the physiology of urinary formation.

3.0 MAIN CONTENT

3.1 The Urinary System

The urinary system consists of two kidneys, two ureters, the urinary bladder, and the urethra. The kidneys form urine, and the rest of the system

eliminates urine. The purpose of urine formation is the removal of potentially toxic waste products from the blood. Urine production and elimination are one of the most important mechanisms of body homeostasis all body systems are directly or indirectly affected by kidney function eg. composition of blood is determined more by kidney function than by diet main function of kidneys is to get rid of metabolic wastes !typically referred to as "excretory system" excretory wastes = metabolic wastes. chemicals & toxins produced by cells during metabolism

3.2 General Functions of Urinary System

1. removal of metabolic wastes & toxins but we have several organs that serve an excretory function other than kidneys: skin, sweat glands rid body of water, minerals, some nitrogenous wastes (ammonia), lungs rid body of CO2 from energy metabolism of cells, liver; liver excretes bile pigments, salts, calcium, some toxins.

2. elimination of excess nutrients & excess hormones, helps to regulate blood volume & pressure. blood pressure is directly affected by the volume of fluids retained or removed from body: eg. excessive salts promote water retention greater volume increases BP eg. dehydration lower volume decreases BP, regulation of electrolytes & body Ph

3. Regulates erythropoiesis; the kidneys produce hormone erythropoietin that regulates erythropoiesis. Hypoxic conditions secretes more erythropoietin excessive Oxygen inhibits hormone production. It also aids in calcium absorption; affects the absorption of Calcium from intestine by helping to activate Vitamin D circulating in blood.

   The general function of the kidneys is to clean and filter blood
   The ureters – are the tubes that take urine to bladder
   The bladder – it stores urine until eliminated
   The urethra – it removes urine from body

3.3 Description

1. **kidneys;** The two kidneys are located in the upper abdominal cavity behind the peritoneum on each side of the vertebral column. The upper portions of both kidneys rest on the lower surface of the diaphragm and are enclosed and protected by the lower rib cage. The kidneys are cushioned by surrounding adipose tissue, which is in

turn covered by a fibrous connective membrane called the renal fascia; both help hold the kidneys in place. On the medial side of each kidney is an indentation called the hilus, where the renal artery enters and the renal vein and ureter emerge. The renal artery is a branch of the abdominal aorta, and the renal vein returns blood to the inferior vena cava. The ureter carries urine from the kidney to the urinary bladder.

Internal Structure of the Kidney

A frontal section of the kidney shows three distinct areas; the outermost area is the renal cortex, which contains the parts of the nephrons called renal corpuscles and convoluted tubules. The middle area is the renal medulla, which contains loops of Henle and collecting tubules. The renal medulla consists of wedge-shaped pieces called renal pyramids; the apex, or papilla, of each pyramid points medially. The third area is a cavity called the renal pelvis; it is formed by the expansion of the ureter within the kidney at the hilus. Funnel-shaped extensions of the renal pelvis, called calyces, enclose the papillae of the renal pyramids. Urine flows from the pyramids into the calyces, then to the renal pelvis, and finally into the ureter.There are extensions of the cortex into renal columns which divides the medulla into 6-10 renal pyramids, the papilla of each pyramid nestled in cup shaped calyces the calyces converge to form **renal pelvis**.

2. Nephron

The nephron is the structural and functional unit of the kidney. Urine is formed in the approximately 1 million nephrons in each kidney. The two major parts of a nephron are the renal corpuscle and the renal tubule.

-A renal corpuscle consists of a glomerulus surrounded by a Bowman's capsule. The glomerulus is a capillary network that arises from an afferent arteriole and empties into an efferent arteriole. The diameter of the efferent arteriole is smaller than that of the afferent arteriole, which helps maintain a fairly high blood pressure in the glomerulus. Bowman's capsule is the expanded end of a renal tubule; it encloses the glomerulus. The inner layer of Bowman's capsule has pores and is highly permeable; the outer layer has no pores and is not permeable. The space between the inner and outer layers contains renal filtrate, the fluid that is formed from the blood in the glomerulus and that will eventually become urine.

-The renal tubule continues from Bowman's capsule and consists of the proximal convoluted tubule, the loop of Henle, and the distal convoluted tubule. The distal convoluted tubules from several nephrons empty into a collecting tubule. Several collecting tubules then unite to form a papillary duct that empties urine into a calyx of the renal pelvis. All the parts of the renal tubule are surrounded by the peritubular capillaries, which arise from the efferent arterioleand receive the materials reabsorbed by the renal tubules.

3. **Ureters**; the rest of urinary system is "plumbing". The renal pelvis funnels urine to paired ureters tubular extensions of renal pelvis peristalsis moves urine along to bladder

4. **Bladder**; its is small in size about the size of a walnut when empty, it can hold up to 800 ml, voluntarily up to 2000 ml when obstructed.Its wall consists of 4 layers (same as GI tract). The mucosa -innermost layer, secretes mucous for protection from corrosive effects of urine;submucosa -fibrous connective tissue; muscularis -several smooth muscle layers; serosa -visceral peritoneum. It has involuntary internal & voluntary external urethral sphincters, as bladder expands to hold urine, activates stretch receptors in wall that monitor volume when volume exceeds 200 ml the receptor signals enter our conscious perception causing a desire to urinate

5. **Urethra**; in the male it has dual function (rid body of urine & release of seminal fluid during orgasm) In the female it has a single function (rids body of urine). It is shorter more prone to UTI's

Blood Supply; kidneys are highly vascularized every minute, 1200 ml/min of blood flows through kidneys which is 1/5th of cardiac output. The pathway of blood flow through the kidney is an essential part of the process of urine formation. Blood from the abdominal aorta enters the renal artery, which branches extensively within the kidney into smaller arteries. The smallest arteries give rise to afferent arterioles in the renal cortex. From the afferent arterioles, blood flows into the glomeruli (capillaries), to efferent arterioles, to peritubular capillaries, to veins in the kidney, to the renal vein, and finally to the inferior vena cava. In this pathway are two sets of capillaries; that is, two sites of exchanges between the blood and the surrounding tissues (in this case, the parts of the nephrons). The exchanges that take place in the capillaries of the kidneys form urine from blood plasma.

More blood perfuses the kidney per weight than any other organ (much more than eg. brain, heart, liver, etc) within the kidney, blood flow is greatest in the cortex where glomeruli are located; flow decreases with depth in the medulla. The Renal Artery brings blood to kidney and it branches eventually into afferent arterioles. Afferent Arteriole bring blood to individual nephrons. Glomerulus dense capillary bed formed by afferent arteriole inside Bowman's capsule.

Bowman's Capsule + Glomerulus = Renal Corpuscle
Efferent Arteriole blood leaves glomerulus via efferent arteriole. Peritubular Capillaries efferent arteriole divides into another capillary bed surrounds the rest of the nephric tubule (PCT-LH-DCT-CT). Renal Vein returns blood to vena cava

THE PHYSIOLOGY OF URINE FORMATION

Urine formation in nephrons occurs by: the processes of filtration, reabsorption and secretion

1. **Filtration** ; it occurs in the renal corpuscle- Glomerulus, Bowmans Capsule, water, salts, small molecules and wastes are filtered out of blood capillaries of glomerulus via the fenestrated capillaries which have higher filtration pressure than other capillaries of body afferent arteriole is larger than efferent arteriole increases pressure in glomerulus pressure ~55mmHg (vs 35mmHg in most capillaries). The kidneys can maintain a fairly constant filtration rate changes in arterial pressure from 80 to 180 mmHg produce little change in blood flow and filtration rate in glomerulus. If blood pressure is reduced below this urine formation slows down, filtrate is essentially the same composition as plasma without formed elements or proteins solutes (filtrate) enter Bowmans capsule

2. **Tubular Reabsorption**; urine is not the same composition as this filtrate. Most of the filtrate is reabsorbed on the overall, 99% of glomerular filtrate gets reabsorbed and only 1% of original filtrate actually leaves the body as urine reabsorption is more selective. Needed nutrients are conserved wastes and toxins are eliminated blood levels of fluids, salts, acidity etc are actively regulated.

The main metabolic wastes removed by kidneys are "nitrogen wastes": urea, uric acid and creatinine

1. Urea; main nitrogen containing waste produced during metabolism formed in liver as result of protein breakdown concentration in urine mainly determined by dietary intake.

2. Uric acid; it is the end product of nucleic acid metabolism some is also secreted by PCT
3. Creatinine; it is the normal end product of muscle metabolism occurs all along nephric tubule.

Different substances are reabsorbed back into blood from different parts of tubule:

Proximal Convoluted Tubule; 80% of materials to be reabsorbed are reabsorbed in PCTcells lining PCT have microvilliall small proteins, glucose, amino acids are reabsorbedmost water, most salts are reabsorbed.

Loop of Henle; additional Chloride and sodium ions are reabsorbed by active transportunder the control of aldosterone (mineralocorticoids)secretion controlled by salt concentrations in tissue fluidsalso affects reabsorption of water (water follows salt).

Distal Convoluted Tubule & Collecting Tubule; additional water is reabsorbedunder control of ADH (antidiuretic hormone). Without ADH the tubules are practically impermeable to water

3. **Tubular secretion**; In tubular secretion, substances are actively secreted from the blood in the peritubular capillaries into the filtrate in the renal tubules. Waste products, such as ammonia and creatinine, excess water-soluble vitamins, and the metabolic products of medications may be secreted into the filtrate to be eliminated in urine. Hydrogen ions may be secreted by the tubule cells to help maintain the normal pH of the blood. Tubular reabsorption conserves useful materials, tubular secretion may add unwanted substances to the filtrate, and most waste products simply remain in the filtrate and are excreted in urine.

UNIT 3 CARING FOR PATIENTS WITHFLUID AND ELECTROLYTE DISORDERS IN RENAL DISORDERS

CONTENTS

1.0 INTRODUCTION

2.0 OBJECTIVES

At the end of this lesson, the learners will be able to

- Explain the importance of fluid and electrolyte to normal body physiology
- Describe the distribution and movement of body fluid and electrolyte
- Discuss the function of the kidney as the main regulator of body fluid and electrolyte.
- Describe the disturbances in fluid and acid base balance
- Discuss the nursing management of clients with fluid and electrolyte imbalance

3.0 MAIN CONTENT

3.1 Body Fluid and Electrolyte

In good health, a delicate balance of fluids, electrolytes, and acids and bases is maintained in the body. This balance, or physiologic homeostasis, depends on multiple physiologic processes that regulate fluid intake and output and the movement of water and the substances dissolved in it between the body compartments. Almost every illness has the potential to threaten this balance. Even in daily living, excessive temperatures or

vigorous activity can disturb the balance if adequate water and salt intake is not maintained. Therapeutic measures, such as the use of diuretics or nasogastric suction, can also disturb the body's homeostasis unless water and electrolytes are replaced.

The proportion of the human body composed of fluid is surprisingly large. Approximately 60% of the average healthy adult's weight is water, the primary body fluid. In good health this volume remains relatively constant and the person's weight varies by less than 0.2 kg in 24 hours, regardless of the amount of fluid ingested. Water is vital to health and normal cellular function, serving as a medium for metabolic reactions within cells, a transporter for nutrients, waste products, and other substances. A lubricant, an insulator and shock absorber.

Age, sex, and body fat affect total body water. Infants have the highest proportion of water, accounting for 70% to 80% of their body weight. The proportion of body water decreases with aging. In people older than 60 years of age, it represents only about 50% of the total body weight. Women also have a lower percentage of body water than men. Women and the elderly have reduced body water due to decreased muscle mass and a greater percentage of fat tissue. Fat tissue is essentially free of water, whereas lean tissue contains a significant amount of water. Water makes up a greater percentage of a lean person's body weight than an obese person's.

Distribution of Body Fluids

The body's fluid is divided into two major compartments, intracellular and extracellular. Intracellular fluid (ICF) is found within the cells of the body. It constitutes approximately two-thirds of the total body fluid in adults.

Extracellular fluid (ECF) is found outside the cells and accounts for about one-third of total body fluid. It is subdivided into compartments. The two main compartments of ECF are intravascular and interstitial. Intravascular fluid, or plasma, accounts for approximately 20% of the ECF and is found within the vascular system. Interstitial fluid, accounting for approximately 75% of the ECF, surrounds the cells. The other compartments of ECF are the lymph and transcellular fluids. Examples of transcellular fluid include cerebrospinal, pericardial, pancreatic, pleural, intraocular, biliary, peritoneal, and synovial fluids. Intracellular fluid is vital to normal cell functioning. It contains solutes such as oxygen, electrolytes, and glucose, and it provides a medium in which metabolic processes of the cell take place. Although extracellular fluid is in the smaller of the two compartments, it is the transport system that carries nutrients to and waste products from the cells. For example, plasma carries oxygen from the lungs

and glucose from the gastrointestinal tract to the capillaries of the vascular system. From there, the oxygen and glucose move across the capillary membranes into the interstitial spaces and then across the cellular membranes into the cells. The opposite route is taken for waste products, such as carbon dioxide going from the cells to the lungs and metabolic acid wastes going eventually to the kidneys. Interstitial fluid transports wastes from the cells by way of the lymph system as well as directly into the blood plasma through capillaries.

Composition of Body Fluids Extracellular and intracellular fluids contain oxygen from the lungs, dissolved nutrients from the gastrointestinal tract, excretory products of metabolism such as carbon dioxide, and charged particles called ions. Many salts dissociate in water, that is, break up into electrically charged ions. The salt sodium chloride breaks up into one ion of sodium and one ion of chloride. These charged particles are called **ELECTROLYTES** because they are capable of conducting electricity.

The number of ions that carry a positive charge, called **CATIONS**, and ions that carry a negative charge, called **ANIONS**, should be equal. Examples of cations are sodium, potassium, calcium, and magnesium. Examples of anions include chloride, bicarbonate, phosphate, and sulfate. Electrolytes generally are measured in milliequivalents per liter of water (mEq/L) or milligrams per 100 milliliters (mg/100 mL).

The composition of fluids varies from one body compartment to another. In extracellular fluid, the principal electrolytes are sodium, chloride, and bicarbonate. Other electrolytes such as potassium, calcium, and magnesium are also present but in much smaller quantities. Plasma and interstitial fluid, the two primary components of ECF, contain essentially the same electrolytes and solutes, with the exception of protein. Plasma is a protein-rich fluid, containing large amounts of albumin, but interstitial fluid contains little or no protein. The composition of intracellular fluid differs significantly from that of ECF. Potassium and magnesium are the primary cations present in ICF, with phosphate and sulfate the major anions. As in ECF, other electrolytes are present within the cell, but in much smaller concentrations. Maintaining a balance of fluid volumes and electrolyte compositions in the fluid compartments of the body is essential to health. Normal and unusual fluid and electrolyte losses must be replaced if homeostasis is to be maintained. Other body fluids such as gastric and intestinal secretions also contain electrolytes. This is of particular concern when these fluids are lost from the body (for example, in severe vomiting or diarrhea or when gastric suction removes the gastric secretions). Fluid

and electrolyte imbalances can result from excessive losses through these routes.

Movement of Body Fluids and Electrolytes

The body fluid compartments are separated from one another by cell membranes and the capillary membrane. While these membranes are completely permeable to water, they are considered to be selectively permeable to solutes as substances move across them with varying degrees of ease. Small particles such as ions, oxygen, and carbon dioxide easily move across these membranes, but larger molecules like glucose and proteins have more difficulty moving between fluid compartments. The methods by which electrolytes and other solutes move are osmosis, diffusion, filtration, and active transport.

In the body, water is the solvent; the solutes include electrolytes, oxygen and carbon dioxide, glucose, urea, amino acids, and proteins. Osmosis occurs when the concentration of solutes on one side of a selectively permeable membrane, such as the capillary membrane, is higher than on the other side. For example, a marathon runner loses a significant amount of water through perspiration, increasing the concentration of solutes in the plasma because of water loss. This higher solute concentration draws water from the interstitial space and cells into the vascular compartment to equalize the concentration of solutes in all fluid compartments. Osmosis is an important mechanism for maintaining homeostasis and fluid balance.

Sodium is by far the greatest determinant of serum osmolality, with glucose and urea also contributing. Potassium, glucose, and urea are the primary contributors to the osmolality of intracellular fluid. The term tonicity may be used to refer to the osmolality of a solution. Solutions may be termed isotonic, hypertonic, or hypo- tonic.

An isotonic solution has the same osmolality as body fluids. Normal saline, 0.9% sodium chloride, is an isotonic solution.

Hypertonic solutions have a higher osmolality than body fluids; 3% sodium chloride is a hypertonic solution.

Hypotonic solutions such as one-half normal saline (0.45% sodium chloride), by contrast, have a lower osmolality than body fluids.

Regulating Body Fluids In a healthy person, the volumes and chemical composition of the fluid compartments stay within narrow safe limits. Normally fluid intake and fluid loss are balanced. Illness can upset this balance so that the body has too little or too much fluid. Fluid Intake During periods of moderate activity at moderate temperature, the average adult drinks about 1,500 mL per day but needs 2,500 mL per day, an

additional 1,000 mL. This added volume is acquired from foods and from the oxidation of these foods during metabolic processes. Interestingly, the water content of food is relatively large, contributing about 750 mL per day. The water content of fresh vegetables is approximately 90%, of fresh fruits about 85%, and of lean meats around 60%.

Water as a by-product of food metabolism accounts for most of the remaining fluid volume required. This quantity is approximately 200 mL per day for the average adult. The thirst mechanism is the primary regulator of fluid intake. The thirst center is located in the hypothalamus of the brain. A number of stimuli trigger this center, including the osmotic pressure of body fluids, vascular volume, and angiotensin (a hormone released in response to decreased blood flow to the kidneys). For example, a long-distance runner loses significant amounts of water through perspiration and rapid breathing during a race, increasing the concentration of solutes and the osmotic pressure of body fluids. This increased osmotic pressure stimulates the thirst center, causing the runner to experience the sensation of thirst and the desire to drink to replace lost fluids. Thirst is normally relieved immediately after drinking a small amount of fluid, even before it is absorbed from the gastrointestinal tract. However, this relief is only temporary, and the thirst returns in about 15 minutes. The thirst is again temporarily relieved after the ingested fluid distends the upper gastrointestinal tract. These mechanisms protect the individual from drinking too much, because it takes from 30 minutes to 1 hour for the fluid to be absorbed and distributed throughout the body.

There are four routes of fluid output:

1. Urine
2. Insensible loss through the skin as perspiration and through the lungs as water vapor in the expired air
3. Noticeable loss through the skin
4. Loss through the intestines in feces

URINE.

Urine formed by the kidneys and excreted from the urinary bladder is the major avenue of fluid output. Normal urine output for an adult is 1,400 to 1,500 mLper 24 hours, or at least 0.5 mL per kilogram per hour. In healthy people, urine output may vary noticeably from day to day. Urine volume automatically increases as fluid intake increases. If fluid loss through perspiration is large, however, urine volume decreases to maintain fluid balance in the body.

INSENSIBLE LOSSES.

Insensible fluid loss occurs through the skin and lungs. It is called insensible because it is usually not noticeable and cannot be measured. Insensible fluid loss through the skin occurs in two ways. Water is lost through diffusion and through perspiration (which is noticeable but not measurable). Water losses through diffusion are not noticeable but normally account for 300 to 400 mLper day. This loss can be significantly increased if the protective layer of the skin is lost as with burns or large abrasions. Perspiration varies depending on factors such as environmental temperature and metabolic activity. Fever and exercise increase metabolic activity and heat production, thereby increasing fluid losses through the skin. Another type of insensible loss is the water in exhaled air. In an adult, this is normally 300 to 400 mLper day. When respiratory rate accelerates, for example, due to exercise or an elevated body temperature, this loss can increase.

FAECES.

The chyme that passes from the small intestine into the large intestine contains water and electrolytes. The volume of chyme entering the large intestine in an adult is normally about 1,500 mLper day. Of this amount, all but about 100 mL is reabsorbed in the proximal half of the large intestine. Certain fluid losses are required to maintain normal body function. These are known as obligatory losses. Approximately 500 mL of fluid must be excreted through the kidneys of an adult each day to eliminate metabolic waste products from the body. Water lost through respirations, through the skin, and in feces also are obligatory losses, necessary for temperature regulation and elimination of waste products. The total of all these losses is approximately 1,300 mLper day.

Maintaining Homeostasis; The volume and composition of body fluids is regulated through several homeostatic mechanisms. A number of body systems contribute to this regulation, including the kidneys, the endocrine system, the cardiovascular system, the lungs, and the gastrointestinal system. Hormones such as antidiuretic hormone (ADH; also known as arginine vasopressin or AVP), the renin-angiotensin- aldosterone system, and atrial natriuretic factor are involved, as are mechanisms to monitor and maintain vascular volume.

THE KIDNEYS AS PRIMARY REGULATORS OF BODY FLUID AND ELECTROLYTE.

The kidneys are the primary regulator of body fluids and electrolyte balance. They regulate the volume and osmolality of extracellular fluids by regulating water and electrolyte excretion. The kidneys adjust the

reabsorption of water from plasma filtrate and ultimately the amount excreted as urine. Although 135 to 180 Lof plasma per day is normally filtered in an adult, only about 1.5 L of urine is excreted. Electrolyte balance is maintained by selective retention and excretion by the kidneys. The kidneys also play a significant role in acid base regulation, excreting hydrogen ion and retaining bicarbonate.

ANTIDIURETIC HORMONE

Antidiuretic hormone, which regulates water excretion from the kidney, is synthesized in the anterior portion of the hypothalamus and acts on the collecting ducts of the nephrons. When serum osmolality rises, ADH is produced, causing the collecting ducts to become more permeable to water. This increased permeability allows more water to be reabsorbed into the blood. As more water is reabsorbed, urine output falls and serum osmolality decreases because the water dilutes body fluids. Conversely, if serum osmolality decreases, ADH is sup- pressed, the collecting ducts become less permeable to water, and urine output increases. Excess water is excreted, and serum osmolality returns to normal. Other factors also affect the production and release of ADH, including blood volume, tempera- ture, pain, stress, and some drugs such as opiates, barbiturates, and nicotine.

RENIN-ANGIOTENSIN-ALDOSTERONE SYSTEM

Specialized receptors in the juxtaglomerular cells of the kidney nephrons respond to changes in renal perfusion. This initiates the renin- angiotensin-aldosterone system. If blood flow or pressure to the kidney decreases, renin is released. Renin causes the conversion of angiotensinogen to angiotensin I, which is then converted to angiotensin II by angiotensin-converting enzyme. Angiotensin II acts directly on the nephrons to promote sodium and water retention. In addition, it stimulates the release of aldosterone from the adrenal cortex. Aldosterone also promotes sodium retention in the distal nephron. The net effect of the renin-angiotensin- aldosterone system is to restore blood volume (and renal perfusion) through sodium and water retention.

ATRIAL NATRIURETIC FACTOR

Atrial natriuretic factor (ANF) is released from cells in the atrium of the heart in response to excess blood volume and stretching of the atrial walls. Acting on the nephrons, ANF promotes sodium wasting and acts as a potent diuretic, thus reducing vascular volume. ANF also inhibits thirst, reducing fluid intake.

Electrolytes, charged ions capable of conducting electricity, are present in all body fluids and fluid compartments. Just as maintaining the fluid

balance is vital to normal body function, so is maintaining electrolyte balance. Although the concentration of specific electrolytes differs between fluid compartments, a balance of cations (positively charged ions) and anions (negatively charged ions) always exists. Electrolytes are important for maintaining fluid balance, Contributing to acid–base regulation, facilitating enzyme reactions, transmitting neuromuscular reactions. Most electrolytes enter the body through dietary intake and are excreted in the urine. Some electrolytes, such as sodium and chloride, are not stored by the body and must be consumed daily to maintain normal levels. Potassium and calcium, on the other hand, are stored in the cells and bone, respectively. When serum levels drop, ions can shift out of the storage "pool" into the blood to maintain adequate serum levels for normal functioning. The regulatory mechanisms and functions of the major electrolytes are summarized below.

Sodium; Sodium is the most abundant cation in extracellular fluid and a major contributor to serum osmolality. Normal serum sodium levels are 135 to 145 mEq/L. Sodium functions largely in controlling and regulating water balance. When sodium is reabsorbed from the kidney tubules, chloride and water are reabsorbed with it, thus maintaining ECF volume. Sodium is found in many foods, such as bacon, ham, processed cheese, and table salt.

Potassium; Potassium is the major cation in intracellular fluids, with only a small amount found in plasma and interstitial fluid. ICF levels of potassium are usually 125 to 140 mEq/L while normal serum potassium levels are 3.5 to 5.0 mEq/L. The ratio of intracellular to extracellular potassium must be maintained for neuromuscular response to stimuli. Potassium is a vital electrolyte for skeletal, cardiac, and smooth muscle activity. It is involved in maintaining acid–base balance as well, and it contributes to intracellular enzyme reactions. Potassium must be ingested daily because the body can't conserve it. Many fruits and vegetables, meat, fish, and other foods contain potassium.

Calcium; The vast majority, 99%, of calcium in the body is in the skeletal system, with a relatively small amount in extracellular fluid. Although this calcium outside the bones and teeth amounts to only about 1% of the total calcium in the body, it is vital in regulating muscle contraction and relaxation, neuromuscular function, and cardiac function. ECF calcium is regulated by a complex interaction of parathyroid hormone, calcitonin, and calcitriol, a metabolite of vitamin D. When calcium levels in the ECF fall, parathyroid hormone and calcitriol cause calcium to be released from bones into ECF and increase the absorption of calcium in the intestines, thus

raising serum calcium levels. Conversely, calcitonin stimulates the deposition of calcium in bone, reducing the concentration of calcium ions in the blood. With aging, the intestines absorb calcium less effectively and more calcium is excreted via the kidneys. Calcium shifts out of the bone to replace these ECF losses, increasing the risk of osteoporosis and fractures of the wrists, vertebrae, and hips. Lack of weight-bearing exercise (which helps keep calcium in the bones) and a vitamin D deficiency because of inadequate expo- sure to sunlight contribute to this risk. Milk and milk products are the richest sources of calcium, with other foods such as dark green leafy vegetables and canned salmon containing smaller amounts. Many clients benefit from calcium supplements. Serum calcium levels are often reported in two ways, based uponthewayitiscirculatingintheplasma.Approximately50% of serum calcium circulates in a free, ionized, or unbound form. The other 50% circulates in the plasma bound to either plasma proteins or other non-protein ions. The normal total serum calcium levels, which range from 8.5 to 10.5 mg/dL, represent both bound and unbound calcium. The normal ionized serum calcium, which ranges from 4.0 to 5.0mg/dL, represents calcium circulating in the plasma in free, or unbound, form.

Magnesium; Magnesium is primarily found in the skeleton and in intracellular fluid. It is the second most abundant intracellular cation with normal serum levels of 1.5 to 2.5 mEq/L. It is important for intracellular metabolism, being particularly involved in the production and use of ATP. Magnesium also is necessary for protein and DNA synthesis within the cells. Only about 1% of the body's magnesium is in ECF; here it is involved in regulating neuromuscular and cardiac function. Maintaining and ensuring adequate magnesium levels is an important part of care of clients with cardiac disorders. Cereal grains, nuts, dried fruit, legumes, and green leafy vegetables are good sources of magnesium in the diet, as are dairy products, meat, and fish.

Chloride; Chloride is the major anion of ECF, and normal serum levels are 95 to 108 mEq/L. Chloride functions with sodium to regulate serum osmolality and blood volume. The concentration of chloride in ECF is regulated secondarily to sodium; when sodium is reabsorbed in the kidney, chloride usually follows. Chloride is a major component of gastric juice as hydrochloric acid (HCl) and is involved in regulating acid–base balance. It also acts as a buffer in the exchange of oxygen and carbon dioxide in RBCs. Chloride is found in the same foods as sodium.

Phosphate; Phosphate is the major anion of intracellular fluids. It also is found in ECF, bone, skeletal muscle, and nerve tissue. Normal serum levels

of phospate in adults range from 2.5 to 4.5 mg/dL. Children have much higher phosphate levels than adults, with that of a newborn nearly twice that of an adult. Higher levels of growth hormone and a faster rate of skeletal growth probably account for this difference. Phosphate is involved in many chemical actions of the cell; it is essential for functioning of muscles, nerves, and red blood cells. It is also involved in the metabolism of protein, fat, and carbohydrate. Phosphate is absorbed from the intestine and is found in many foods such as meat, fish, poultry, milk products, and legumes.

Bicarbonate; Bicarbonate is present in both intracellular and extracellular fluids. Its primary function is regulating acid–base balance as an essential component of the carbonic acid–bicarbonate buffering system. Extracellular bicarbonate levels are regulated by the kidneys: Bicarbonate is excreted when too much is present; if more is needed, the kidneys both regenerate and reabsorb bicarbonate ions. Unlike other electrolytes that must be consumed in the diet, adequate amounts of bicarbonate are produced through metabolic processes to meet the body's needs.

THE ACID–BASE BALANCE

An important part of regulating the chemical balance or homeostasis of body fluids is regulating their acidity or alkalinity. An acid is a substance that releases hydrogen ions in solution. Strong acids such as hydrochloric acid release all or nearly all their hydrogen ions; weak acids like carbonic acid release some hydrogen ions. Bases or alkalis have a low hydrogen ion con- centration and can accept hydrogen ions in solution. The relative acidity or alkalinity of a solution is measured as pH. The pH reflects the hydrogen ion concentration of the solution: The higher the hydrogen ion concentration (and the more acidic the solution), the lower the pH. Water has a pH of 7 and is neutral; that is, it is neither acidic in nature nor is it alkaline. Solutions with a pH lower than 7 are acidic; those with a pH higher than 7 are alkaline. The pH scale is logarithmic: A solution with a pH of 5 is 10 times more acidic than one with a pH of 6. Regulation of Acid–Base Balance Body fluids are maintained within a narrow range that is slightly alkaline. The normal pH of arterial blood is between 7.35 and 7.45. Acids are continually produced during metabolism. Several body systems, including buffers, the respiratory system, and the renal system, are actively involved in maintaining the narrow pH range necessary for optimal function. Buffers help maintain acid–base balance by neutralizing excess acids or bases. The lungs and the kidneys help maintain a normal pH by either excreting or retaining acids and bases.

Buffers prevent excessive changes in pH by removing or releasing hydrogen ions. If excess hydrogen ion is present in body fluids, buffers bind with the hydrogen ion, minimizing the change in pH. When body fluids become too alkaline, buffers can release hydrogen ion, again minimizing the change in pH. The action of a buffer is immediate, but limited in its capacity to maintain or restore normal acid–base balance. The major buffer system in extracellular fluids is the bicarbonate and carbonic acid system. When a strong acid such as hydrochloric acid is added, it combines with bi- carbonate and the pH drops only slightly. A strong base such as sodium hydroxide combines with carbonic acid, the weak acid of the buffer pair, and the pH remains within the narrow range of normal. The amounts of bicarbonate and carbonic acid in the body vary; however, as long as a ratio of 20 parts of bicarbonate to 1 part of carbonic acid is maintained, the pH remains within its normal range of 7.35 to 7.45. Adding a strong acid to ECF can change this ratio as bicarbonate is depleted in neutralizing the acid. When this happens, the pH drops, and the client has a condition called acidosis. The ratio can also be upset by adding a strong base to ECF, depleting carbonic acid as it combines with the base. In this case the pH rises and the client has alkalosis. In addition to the bicarbonate–carbonic acid buffer system, plasma proteins, hemoglobin, and phosphates also function as buffers in body fluids.

The lungs help regulate acid–base balance by eliminating or retaining carbon dioxide, a potential acid. Combined with water, carbon dioxide forms carbonic acid. This chemical reaction is reversible; carbonic acid breaks down into carbon dioxide and water. Working together with the bicarbonate–carbonic acid buffer system, the lungs regulate acid–base balance and pH by altering the rate and depth of respirations. The response of the respiratory system to changes in pH is rapid, occurring within minutes. Carbon dioxide is a powerful stimulator of the respiratory center. When blood levels of carbonic acid and carbon dioxide rise, the respiratory center is stimulated and the rate and depth of respirations increase. Carbon dioxide is exhaled, and carbonic acid levels fall. By contrast, when bicarbonate levels are excessive, the rate and depth of respirations are reduced. This causes carbon dioxide to be retained, carbonic acid levels to rise, and the excess bicarbonate to be neutralized. Carbon dioxide levels in the blood are measured as the PCO2, or partial pressure of the dissolved gas in the blood. PCO2 refers to the pressure of carbon dioxide in venous blood. PaCO2 refers to the pressure of carbon dioxide in arterial blood. The normal PaCO2 is 35 to 45 mm Hg.

Although buffers and the respiratory system can compensate for changes in pH, the kidneys are the ultimate long-term regulator of acid–base balance.

They are slower to respond to changes, requiring hours to days to correct imbalances, but their response is more permanent and selective than that of the other systems. The kidneys maintain acid–base balance by selectively excreting or conserving bicarbonate and hydrogen ions. When excess hydrogen ion is present and the pH falls (acidosis), the kidneys reabsorb and regenerate bicarbonate and excrete hydrogen ion. In the case of alkalosis and a high pH, excess bicarbonate is excreted and hydrogen ion is retained. The normal serum bicarbonate level is 22 to 26 mEq/L.

FACTORS AFFECTING BODY FLUID, ELECTROLYTES, AND ACID–BASE BALANCE

The ability of the body to adjust fluids, electrolytes, and acid–base balance is influenced by age, gender and body size, environmental temperature, and lifestyle. Age Infants and growing children have much greater fluid turnover than adults because their higher metabolic rate increases fluid loss. Infants lose more fluid through the kidneys because immature kidneys are less able to conserve water than adult kidneys. In addition, infants' respirations are more rapid and the body surface area is proportionately greater than that of adults, increasing insensible fluid losses. The more rapid turnover of fluid plus the losses produced by disease can create critical fluid imbalances in children much more rapidly than in adults. In elderly people, the normal aging process may affect fluid balance. The thirst response often is blunted. Antidiuretic hormone levels remain normal or may even be elevated, but the nephrons become less able to conserve water in response to ADH. Increased levels of atrial natriuretic factor seen in older adults may also contribute to this impaired ability to conserve water. These normal changes of aging increase the risk of dehydration. When combined with the increased likelihood of heart diseases, impaired renal function, and multiple drug regimens, the older adult's risk for fluid and electrolyte imbalance is significant. Additionally, it is important to consider that the older adult has thinner, more fragile skin and veins, which can make an intravenous insertion more difficult. Gender and Body Size Total body water also is affected by gender and body size. Because fat cells contain little or no water, and lean tissue has a high water content, people with a higher percentage of body fat have less body fluid. Women have proportionately more body fat and less body water than men. Water accounts for approximately 60% of an adult man's weight, but only 52% for an adult woman. In an obese individual this may be even less, with water responsible for only 30% to 40% of the person's weight. Environmental Temperature People with an illness and those participating in strenuous activity are at risk for fluid and electrolyte imbalances when the environmental temperature is high. Fluid losses through sweating are increased in hot environments as the body attempts to dissipate heat. These losses are even greater in people who have not been acclimatized to the

environment. Both salt and water are lost through sweating. When only water is replaced, salt depletion is a risk. The person who is salt depleted may experience fatigue, weakness, headache, and gastrointestinal symptoms such as anorexia and nausea. The risk of adverse effects is even greater if lost water is not replaced. Body temperature rises, and the person is at risk for heat exhaustion or heatstroke. Heatstroke may occur in older adults or ill people during prolonged periods of heat; it can also affect athletes and laborers when their heat production exceeds the body's ability to dissipate heat. Consuming adequate amounts of cool liquids, particularly during strenuous activity, reduces the risk of adverse effects from heat. Balanced electrolyte solutions and carbohydrate-electrolyte solutions such as sports drinks are recommended because they replace both water and electrolytes lost through sweat.

Lifestyle related factors such as diet, exercise, and stress affect fluid, electrolyte, and acid–base balance. The intake of fluids and electrolytes is affected by the diet. People with anorexia nervosa or bulimia are at risk for severe fluid and electrolyte imbalances because of inadequate intake or purging regimens (e.g., induced vomiting, use of diuretics and laxatives). Seriously malnourished people have decreased serum albumin levels, and may develop edema because the osmotic draw of fluid into the vascular compartment is reduced. When calorie intake is not adequate to meet the body's needs, fat stores are broken down and fatty acids are released, increasing the risk of acidosis. Regular weight-bearing physical exercise such as walking, running, or bicycling has a beneficial effect on calcium balance. The rate of bone loss that occurs in postmenopausal women and older men is slowed with regular exercise, reducing the risk of osteoporosis. Stress can increase cellular metabolism, blood glucose concentration, and catecholamine levels. In addition, stress can in- crease production of ADH, which in turn decreases urine production. The overall response of the body to stress is to in- crease the blood volume. Other lifestyle factors can also affect fluid, electrolyte, and acid–base balance. Heavy alcohol consumption affects electrolyte balance, increasing the risk of low calcium, magnesium, and phosphate levels. The risk of acidosis associated with breakdown of fat tissue also is greater in the person who drinks large amounts of alcohol.

DISTURBANCES IN FLUID VOLUME, ELECTROLYTE, AND ACID–BASE BALANCES

A number of factors such as illness, trauma, surgery, and medications can affect the body's ability to maintain fluid, electrolyte, and acid–base balance. The kidneys play a major role in maintaining fluid, electrolyte, and acid–base balance, and renal disease is a significant cause of imbalance.

Clients who are con- fused or unable to communicate their needs are at risk for inadequate fluid intake. Vomiting, diarrhea, or nasogastric suction can cause significant fluid losses. Tissue trauma, such as burns, causes fluid and electrolytes to be lost from damaged cells. Decreased blood flow to the kidneys due to impaired cardiac function stimulates the renin-angiotensin-aldosterone system, causing sodium and water retention. Medications such as diuretics or corticosteroids can result in abnormal losses of electrolytes and fluid loss or retention. Diseases such as diabetes mellitus or chronic obstructive lung disease may affect acid–base balance. Diabetic ketoacidosis, cancer, and head injury may also lead to electrolyte imbalances.

Fluid imbalances are of two basic types: isotonic and osmolar.

Isotonic imbalances occur when water and electrolytes are lost or gained in equal proportions, so that the osmolality of body fluids remains constant. Osmolar imbalances involve the loss or gain of only water, so that the osmolality of the serum is altered. Thus four categories of fluid imbalances may occur:

(a) an isotonic loss of water and electrolytes,
(b) an isotonic gain of water and electrolytes,
(c) a hyperosmolar loss of only water, and
(d) a hypo-osmolar gain of only water.

These are referred to, respectively, as fluid volume deficit, fluid volume excess, dehydration (hyperosmolar imbalance), and overhydration (hypo-osmolar imbalance).

Fluid Volume Deficit Isotonic fluid volume deficit (FVD) occurs when the body loses both water and electrolytes from the ECF in similar proportions. Thus, the decreased volume of fluid remains isotonic. In FVD, fluid is initially lost from the intravascular compartment, so it often is called hypovolemia.

FVD generally occurs as a result of (a) abnormal losses through the skin, gastrointestinal tract, or kidney; (b) decreased intake of fluid (c)bleeding (d)movement of fluid into a third space.(In third space syndrome, fluid shifts from the vascular space into an area where it is not readily accessible as extracellular fluid. This fluid remains in the body but is essentially unavailable for use, causing an isotonic fluid volume deficit. Fluid may be sequestered in the bowel, in the interstitial space as edema, in inflamed tissue, or in potential spaces such as the peritoneal or pleural cavities. The client with third space syndrome has an isotonic fluid deficit but may not manifest apparent fluid loss or weight loss. Careful nursing assessment is vital to effectively identify and intervene for clients experiencing third-

spacing. Because the fluid shifts back into the vascular compartment after time, assessment for manifestations of fluid volume excess or hypervolemia is also vital).

Fluid Volume Excess Fluid volume excess (FVE) occurs when the body retains both water and sodium in similar proportions to normal EC. This is commonly referred to as hypervolemia (increased blood volume). FVE is always secondary to an increase in the total body sodium content, which leads to an increase in total body water. Because both water and sodium are retained, these sodium concentration remains essentially normal and the excess volume of fluid is isotonic.

Specific causes of FVE include (a) excessive intake of sodium chloride; (b) administering sodium-containing infusions too rapidly, particularly to clients with impaired regulatory mechanisms; and (c) disease processes that alter regulatory mechanisms, such as heart failure, renal failure, cirrhosis of the liver and Cushing's syndrome.

EDEMA

In fluid volume excess, both intravascular and interstitial spaces have an increased water and sodium content. Excess interstitial fluid is known as edema. Edema typically is most apparent in areas where the tissue pressure is low, such as around the eyes, and in dependent tissues (known as dependent edema), where hydrostatic capillary pressure is high. Edema can be caused by several different mechanisms. The three main mechanisms are increased capillary hydrostatic pressure, decreased plasma oncotic pressure, and increased capillary permeability. It may be due to FVE that increases capillary hydrostatic pressures, pushing fluid into the interstitial tissues. This type of edema is often seen in dependent tissues such as the feet, ankles, and sacrum because of the effects of gravity. Low levels of plasma proteins from malnutrition or liver or kidney diseases can reduce the plasma oncotic pressure so that fluid is not drawn into the capillaries from interstitial tissues, causing edema. With tissue trauma and some disorders such as allergic reactions, capillaries become more permeable, allowing fluid to escape into interstitial tissues. Obstructed lymph flow impairs the movement of fluid from interstitial tissues back into the vascular compartment, resulting in edema. Pitting edema is edema that leaves a small depression or pit after finger pressure is applied to the swollen area. The pit is caused by movement of fluid to adjacent tissue, away from the point of pressure. Within 10 to 30 seconds the pit normally disappears.

Dehydration

Or hyperosmolar imbalance, occurs when water is lost from the body leaving the client with excess sodium. Because water is lost while

electrolytes, particularly sodium, are retained, the serum osmolality and serum sodium levels increase. Water is drawn into the vascular compartment from the interstitial space and cells, resulting in cellular dehydration. Older adults are at particular risk for dehydration because of decreased thirst sensation. This type of water deficit also can affect clients who are hyperventilating or have prolonged fever or are in diabetic ketoacidosis and those receiving enteral feedings with insufficient water intake.

Overhydration; also known as hypo-osmolar imbalance or water excess, occurs when water is gained in excess of electrolytes, resulting in low serum osmolality and low serum sodium levels. Water is drawn into the cells, causing them to swell. In the brain this can lead to cerebral edema and impaired neurologic function. Water intoxication often occurs when both fluid and electrolytes are lost, for example, through excessive sweating, but only water is replaced. It can also result from the syndrome of inappropriate antidiuretic hormone (SIADH), a disorder that can occur with some malignant tumors, AIDS, head injury, or administration of certain drugs such as barbiturates or anesthetics.

ELECTROLYTE IMBALANCES

The most common and most significant electrolyte imbalances involve sodium, potassium, calcium, magnesium, chloride, and phosphate.

Sodium as the most abundant caution in the extracellular fluid, not only moves into and out of the body but also moves in careful balance among the three fluid compartments. It is found in most body secretions, for example, saliva, gastric and intestinal secretions, bile, and pancreatic fluid. Therefore, continuous excretion of any of these fluids, such as via intestinal suction, can result in a sodium deficit. Because of its role in regulating water balance, sodium imbalances usually are accompanied by water imbalance**. Hyponatremia** is a sodium deficit, or serum sodium level of less than 135 mEq/L, and is, in acute care settings, a common electrolyte imbalance. Because of sodium's role in determining the osmolality of ECF, hyponatremia typically results in a low serum osmolality. Water is drawn out of the vascular compartment into interstitial tissues and the cells, causing the clinical manifestations associated with this disorder. As sodium levels decrease, the brain and nervous system are affected by cellular edema. Severe hyponatremia, serum levels below 110 mEq/L, is a medical emergency and can lead to permanent neurological damage.

Hypernatremia is excess sodium in ECF, or a serum sodium of greater than 145 mEq/L. Because the osmotic pressure of extracellular fluid is

increased, fluid moves out of the cells into the ECF. As a result, the cells become dehydrated. Like hyponatremia, the primary manifestations of hypernatremia are neurological in nature. It is important to note that a person's thirst mechanism protects against hypernatremia. For example, when an individual becomes thirsty, the body is stimulated to drink water which helps correct the hypernatremia. Clients at risk for hypernatremia are those who are unable to access water (e.g., unconscious, unable to request fluids such as infants or elders with dementia, or ill clients with an impaired thirst mechanism).

Potassium; the amount of potassium in extracellular fluid is small, it is vital to normal neuromuscular and cardiac function. Normal renal function is important for maintenance of potassium balance as 80% of potassium is excreted by the kidneys. Potassium must be replaced daily to maintain its balance. Normally, potassium is replaced in food. **Hypokalemia** is a potassium deficit or a serum potassium level of less than 3.5 mEq/L. Gastrointestinal losses of potassium through vomiting and gastric suction are common causes of hypokalemia, as are the use of potassium-wasting diuretics, such as thiazide diuretics or loop diuretics (e.g., furosemide). Symptoms of hypokalemia are usually mild until the level drops below 3 mEq/L unless the decrease in potassium was rapid. When the decrease is gradual, the body compensates by shifting potassium from the intracellular environment into the serum. **Hyperkalemia** is a potassium excess or a serum potassium level greater than 5.0 mEq/L. Hyperkalemia is less common than hypokalemia and rarely occurs in clients with normal renal function. It is, however, more dangerous than hypokalemia and can lead to cardiac arrest. As with hypokalemia, symptoms are more severe and occur at lower levels when the increase in potassium is abrupt.

Calcium; Regulating levels of calcium in the body is more complex than the other major electrolytes so calcium balance can be affected by many factors. Imbalances of this electrolyte are relatively common. **Hypocalcemia** is a calcium deficit, or a total serum calcium level of less than 8.5 mg/dL or an ionized calcium level of less than 4.0 mg/dL. Severe depletion of calcium can cause tetany with muscle spasms and paresthesias (numbness and tingling) around the mouth and hands and feet) and can lead to convulsions. Two signs indicate hypocalcemia: The Chvostek's sign is contraction of the facial muscles that is produced by tapping the facial nerve in front of the ear. Trousseau's sign is a carpal spasm that occurs by inflating a blood pressure cuff on the upper arm to 20 mm Hg greater than the systolic pressure for 2 to 5 minutes. Clients at greatest risk for hypocalcemia are those whose parathyroid glands have been removed. This is frequently associated with total thyroidectomy or bilateral neck surgery

for cancer. Low serum magnesium levels (hypomagnesemia) and chronic alcoholism also increase the risk of hypocalcemia. **Hypercalcemia**, or total serum calcium levels greater than 10.5 mg/dL, or an ionized calcium level of greater than 5.0 mg/dL, most often occurs when calcium is mobilized from the bony skeleton. This may be due to malignancy or prolonged immobilization.

Magnesium; Magnesium imbalances are relatively common in hospitalized clients, although they may be unrecognized. **Hypomagnesemia** is a magnesium deficiency, or a total serum magnesium level of less than 1.5 mEq/L. It occurs more frequently than hypermagnesemia. Chronic alcoholism is the most common cause of hypomagnesemia. Magnesium deficiency also may aggravate the manifestations of alcohol withdrawal, such as delirium tremens (DTs). **Hypermagnesemia** is present when the serum magnesium level rises above 2.5 mEq/L. It is due to in- creased intake or decreased excretion. It is often iatrogenic, that is, a result of overzealous magnesium therapy.

Chloride Because of the relationship between sodium ions and chloride ions imbalances of chloride commonly occur in conjunction with sodium imbalances. **Hypochloremia** is a decreased serum chloride level, in adults a level below 95 mEq/L, and is usually related to excess losses of chloride ion through the GI tract, kidneys, or sweating. Hypochloremic clients are at risk for alkalosis and may experience muscle twitching, tremors, or tetany. Conditions that cause sodium retention also can lead to a high serum chloride level or hyperchloremia, in adults a level above 108 mEq/L. Excess replacement of sodium chloride or potassium chloride are additional risk factors for high serum chloride levels. The manifestations of hyperchloremia include acidosis, weak- ness, and lethargy, with a risk of dysrhythmias and coma.

Phosphate The phosphate anion is found in both intracellular and extracellular fluid. Most of the phosphorus in the body exists as Phosphate is critical for cellular metabolism because it is a major component of adenosine triphosphate (ATP). Phosphate imbalances frequently are related to therapeutic interventions for other disorders. Glucose and insulin administration and total parenteral nutrition can cause phosphate to shift into the cells from extracellular fluid compartments, leading to **hypophosphatemia**, defined in adults as a total serum phosphate level less than 2.5 mg/dL. Alcohol withdrawal, acid–base imbalances, and the use of antacids that bind with phosphate in the GI tract are other possible causes of low serum phosphate levels. Manifestations of hypophosphatemia include paresthesias, muscle weakness and pain, mental changes, and

possible seizures. Hyperphosphatemia, defined in adults as a total serum phosphate level greater than 4.5 mg/dL, occurs when phosphate shifts out of the cells into extracellular fluids (e.g., due to tissue trauma or chemotherapy for malignant tumors), in renal failure, or when excess phosphate is administered or ingested. Infants who are fed cow's milk are at risk for hyperphosphatemia, as are people using phosphate-containing enemas or laxatives. Clients who have high serum phosphate levels may experience numbness and tingling around the mouth and in the fingertips, muscle spasms, and tetany.

Acid–base imbalances generally are classified as respiratory or metabolic by the general or underlying cause of the disorder. Carbonic acid levels are normally regulated by the lungs through the retention or excretion of carbon dioxide, and problems of regulation lead to respiratory acidosis or alkalosis. Bicarbonate and hydrogen ion levels are regulated by the kidneys, and problems of regulation lead to metabolic acidosis or alkalosis. Healthy regulatory systems will attempt to correct acid–base imbalances, a process called compensation.

Respiratory Acidosis; Hypoventilation and carbon dioxide retention cause carbonic acid levels to increase and the pH to fall below 7.35, a condition known as respiratory acidosis. Serious lung diseases such as asthma and COPD are common causes of respiratory acidosis. Central nervous system depression due to anesthesia or a narcotic overdose can sufficiently slow the respiratory rate so that carbon dioxide is retained. When respiratory acidosis occurs, the kidneys retain bicarbonate to restore the normal carbonic acid to bicarbonate ratio. The kidneys are relatively slow to respond to changes in acid–base balance, so this compensatory response may require hours to days to restore the normal pH.

Respiratory Alkalosis; When a person hyperventilates, more carbon dioxide than normal is exhaled, carbonic acid levels fall, and the pH rises to greater than 7.45. This condition is termed respiratory alkalosis. Psychogenic or anxiety-related hyperventilation is a common cause of respiratory alkalosis. Other causes include fever and respiratory infections. In respiratory alkalosis, the kidneys will excrete bicarbonate to return the pH to within the normal range. Often, however, the cause of the hyperventilation is eliminated and the pH returns to normal before renal compensation occurs.

Metabolic Acidosis; when bicarbonate levels are low in relation to the amount of carbonic acid in the body, the pH falls and metabolic acidosis develops. This may develop because of renal failure and the in- ability of the kidneys to excrete hydrogen ion and produce bicarbonate. It also may

occur when too much acid is produced in the body, for example, in diabetic ketoacidosis or starvation when fat tissue is broken down for energy. Metabolic acidosis stimulates the respiratory center, and the rate and depth of respirations increase. Carbon dioxide is eliminated and carbonic acid levels fall, minimizing the change in pH. This respiratory compensation occurs within minutes of the pH imbalance.

In metabolic alkalosis, the amount of bicarbonate in the body exceeds the normal 20-to-1 ratio. Ingestion of bicarbonate of soda as an antacid is one cause of metabolic alkalosis. Another cause is prolonged vomiting with loss of hydrochloric acid from the stomach. The respiratory center is depressed in metabolic alkalosis, and respirations slow and become shallower. Carbon dioxide is retained and carbonic acid levels increase, helping balance the excess bicarbonate.

SIGNS AND SYMPTOMS OF FLUID AND ELCTROLYTE IMBALANCES

1. Muscular weakness
2. Constipation
3. Anorexia & vomiting
4. Polyuria, polydipsia and dehydration
5. Neuromuscular irritability
6. Tetany
7. Tachycardia etc

NURSING MANAGEMENT OF PATIENTS WITH FLUID AND ELECTROLYTE DISORDERS IN RENAL DISEASE.

ASSESSMENT; Three simple clinical measurements can be initiated without a primary care provider's order. They are daily weights, vital signs, and fluid intake and output.

DAILY WEIGHTS; Daily weight measurements provide a relatively accurate assessment of a client's fluid status. Significant changes in weight over a short time (e.g., more than 5kg in a week or less) are indicative of acute fluid changes. Each kilogram of weight gained or lost is equivalent to 1 L of fluid gained or lost. Such fluid gains or losses indicate changes in total body fluid volume rather than in any specific compartment, such as the intravascular compartment. Rapid losses or gains of 5% to 8% of total body weight indicate moderate to severe fluid volume deficits or excesses.

To obtain accurate weight measurements, the nurse should balance the scale before each use and weigh the client

(a) at the same time each day (e.g., before breakfast and after the first void)
(b) wearing the same or similar clothing, and
(c) on the same scale.

The type of scale (i.e., standing, bed, chair) should be documented. Regular assessment of weight is particularly important for clients in the community and extended care facilities who are at risk for fluid imbalance. For these clients, measuring intake and output may be impractical because of lifestyle or problems with incontinence. Regular weight measurement, either daily, every other day, or weekly, provides valuable information about the client's fluid volume status.

VITAL SIGNS; Changes in the vital signs may indicate, or in some cases precede, fluid, electrolyte, and acid–base imbalances. For example, elevated body temperature may be a result of dehydration or a cause of increased body fluid losses. Tachycardia is an early sign of hypovolemia. Pulse volume will decrease in FVD and increase in FVE. Irregular pulse rates may occur with electrolyte imbalances. Changes in respiratory rate and depth may cause respiratory acid–base imbalances or act as a compensatory mechanism in metabolic acidosis or alkalosis. Blood pressure, a sensitive measure to detect blood volume changes, may fall significantly with FVD and hypovolemia or increase with FVE. Postural, or orthostatic, hypotension may also occur with FVD and hypovolemia. To assess for orthostatic hypotension, measure the client's blood pressure and pulse in a supine position. Allow the client to remain in that position for 3 to 5 minutes, leaving the blood pressure cuff on the arm. Stand the client up and immediately reassess the blood pressure and pulse. A drop of 10 to 15 mm Hg in the systolic blood pressure with a corresponding drop in diastolic pressure and an increased pulse rate (by 10 or more beats per minute) is indicative of orthostatic or postural hypotension.

FLUID INTAKE AND OUTPUT.
The measurement and recording of all fluid intake and output (I & O) during a 24-hour period provides important data about the client's fluid and electrolyte balance. Generally, intake and output are measured for hospitalized at-risk clients. The unit used to measure intake and output is the milliliter (mL) or cubic centimeter (cc); these are equivalent metric units of measurement. In household measures, 30 mL is roughly equivalent to 1 fluid ounce, 500 mL is about 1 pint, and 1,000 mL is about 1 quart. To measure fluid intake, nurses convert house- hold measures such as a glass, cup, or soup bowl to metric units. Most agencies provide conversion tables, since the sizes of dishes vary from agency to agency. Such a table is often

provided on or with the bedside I & O record. It is important to inform clients, family members, and all caregivers that accurate measurements of the client's fluid intake and output are required, explaining why and emphasizing the need to use a bedpan, urinal, commode, or in-toilet collection device (unless a urinary drainage system is in place). Instruct the client not to put toilet tissue into the container with urine. Clients who wish to be involved in recording fluid intake measurements need to be taught how to compute the values and what foods are considered fluids.

LABORATORY INVESTIGATIONS; various diagnostic measures can be used to measure the complete electrolyte profile to be able to ascertain the exact electrolyte deficit or excess.

DIAGNOSIS; diagnosis id made based on the assessment conducted and a plan of action can be made.

NURSING INTERVENTION

1. Involve all members of the health team in planning and management.
2. A fluid challenge can be tried to assess the function of the kidney. Fluid can be replaced orally or parenterally. Various fluids that can be used as indicated.
3. A close monitoring of intake and output should be maintained.
4. Fluid and sodium restrictions may be indicated in some cases, diuretics and dialysis may be indicated in some cases.
5. Certain electrolytes may be replaced e.g. parenteral potassium replacement, intravenous infusion of glucose solution, bicarbonate replacement, magnesium replacement therapy e.t.c.

UNIT 4 CARING FOR PATIENTS WITH DYSFUNCTIONAL VOIDING PATTERNS; CONGENITALVOIDING DYSFUNCTION

CONTENTS

1.0 INTRODUCTION

Voiding dysfunction is described as a condition where there is lack of coordination between the bladder muscle, and the urethra. With normal urination, the urethra relaxes and opens when the bladder muscle contracts allowing urine to pass out of the body freely. In those with voiding dysfunction, the urethra does not relax when the bladder muscle contract, making it difficult for urine to pass.

Voiding dysfunction usually presents in one of two ways. The first is in the form of symptoms.

Symptoms related to voiding dysfunction are broadly referred to as lower urinary tract symptoms (LUTS).LUTS have classically been divided into; Obstructive symptoms such as difficulty initiating a stream, decreased force of urinary stream, need to push and strain to void (stranguria), hesitancy or intermittent urine flow, and irritative symptoms such as urinary frequency, urgency, and nocturia. In addition, symptoms of incontinence and lower abdominalor pelvic pain may exist.

The second way in which voiding dysfunction presents is in the form of urinary tract decompensation such as incomplete bladder emptying or urinary retention, renal insufficiency, and recurrent urinary tract infections. It is possible for patients who present with urinary tract decompensation to have little or no symptoms.

In the case of symptoms, evaluation and treatment are often driven by the degree of bother to the patient. In many cases, patients with mild LUTS of a

minimal bother will not even bring these to the attention of their physician. However, when urinary tract decompensation is diagnosed, a more aggressive diagnostic and treatment plan must be implemented.

There are also patients who have diseases known to effect the lower urinary tract and causes voiding dysfunction, yet do not have significant symptoms or obvious signs of decompensation. These include patients with a variety of neurological conditions such as spinal cord injuries or multiple sclerosis, or non-neurological conditions such as prior pelvic irradiation or extensive pelvic surgery. In many cases careful evaluation of the urinary tract will uncover underlying voiding dysfunction.

Thus the diagnostic valuation of voiding dysfunction will be influenced by the type and degree of bother of symptoms, the presence of urinary tract de compensation, and coexisting medical conditions that might affect the lower urinary tract or its treatment.

2.0 OBJECTIVES

At the end of this lesson, the learners will be able to

- Define voiding dysfunction
- List the classes of voiding dysfunction
- Discuss the management and nursing management of voiding dysfunctions.

CLASSIFICATION OF VOIDING DYSFUNCTION
Voiding dysfunction can be divided into three categories:

1. Failure to store urine.
2. Failure to empty urine.
3. Failure to store and empty.

The symptom of urinary frequency or incontinence is usually associated with dysfunction of the storage phase of micturition, whereas decreased force of stream or elevated postvoid residual are associated with dysfunction of the emptying phase.

Voiding dysfunction in simple anatomical terms can be classified thus:

1. Bladder dysfunction (overactive, underactive).
2. Bladder outlet dysfunction (overactive, underactive).
3. Combined bladder and outlet dysfunction.

These two concepts can be combined so that one can imagine that a patient could present with urinary incontinence (failure to store) secondary to bladder overactivity or bladder outlet underactivity. Similarly a patient with urinary retention (failure to empty) might have an underactive—or hypocontractile—bladder or an overactive—or obstructing—outlet. Failure to empty and failure to store as well as bladder and outlet dysfunction are not mutually exclusive conditions and can exist in multiple combinations.

MANAGEMENT OF DYSFUNCTIONS

The following concepts can be applied to all types of voiding dysfunction. Therefore when evaluating voiding dysfunction, from history and physical examination to simple and comprehensive testing, keeping these concepts in mind can greatly facilitate the process.

HISTORY;

The patient's history is the first step in directing the clinician toward the appropriate evaluation and treatment. It should provide a detailed account of the precise nature of the patient's symptoms. The history is only as accurate as the patient's ability to describe their symptoms, some skill is required by the physician to obtain this information. This is especially true for patients who have difficulty communicating or those who are anxious or embarrassed about their condition. The history begins with an assessment of a patient's symptoms and their onset. Each symptom should be characterized as to its onset, frequency, duration, severity.

It is important to note whether the onset of the symptom occurred after a specific event such as surgery, childbirth, menopause, or with the use of a new medication. Any prior treatments by other physicians for their symptoms and the resultant outcome should also be noted.

Specific questions about childhood and adolescent voiding troubles or problems with toilet training should be asked.

Patients will often present with one or more voiding symptoms that have been traditionally separated into irritative or obstructive in nature. Irritative voiding symptoms are common presenting complaints that may herald a number of different types of voiding dysfunction.

Urgency is defined as an intense desire to void secondary to an abrupt sensation of bladder discomfort or as a conditional response from the fear of urine leakage.

Frequency is defined as more than seven diurnal voids and may reflect excessive fluid in take, diureticuse, or excessive caffeine consumption.

Nocturia is nighttime frequency and may be secondary to detrusor overactivity, reduced bladder capacity, or excessive fluid/ caffeine intake prior to bedtime. Daytime frequency without nocturia may be suggestive of timing of diuretic medications or a psychogenic component to the voiding dysfunction.

Dysuria refers to the burning sensation that occurs during micturition and implies bladder, urethral, or prostatic inflammation. Obstructive voiding symptoms include decreased force of urinary stream, straining to void, hesitancy (the prolonged interval necessary to voluntarily initiate the urinary stream), and interruption of urinary stream. They may be present in men with bladder outlet obstruction secondary to benign prostatic enlargement or urethral stricture, or in women with pelvic organ prolapse.

Obstructive and irritative symptoms with symptoms of storage (e.g. frequency, urgency, incontinence) and symptoms of voiding (hesitancy, decreased force of stream, incomplete emptying).

Urinary in continence is simply defined as the involuntary loss of urine, however, this can be further characterized according to the information relayed by the patient:

1. Urge incontinence: The symptom is incontinence is associated with a sudden uncontrollable desire to void. This condition is usually due to involuntary detrusor contractions.
2. Stress incontinence: The symptom is incontinence that occurs during coughing, sneezing, physical exertion, changes in body position, or other action that causes an increase in abdominal pressure. This condition may be caused by sphincter abnormalities or bladder over activity provoked by physical activity.
3. Unconscious incontinence: The symptom of incontinence is unconscious and occurs without patient awareness of urges or stress or increases in abdominal pressure. This condition may be caused by bladder over activity, sphincter abnormalities, overflow, or extra urethral causes such as a fistula or ectopic ureter.
4. Continuous leakage: The symptom is a complaint of continuous loss of urine. This may be caused by sphincter abnormalities or extra urethral causes.

There are several aspects of a patient's history that may be intimately related to voiding function.

- Sexual and bowel dysfunction are often associated with voiding dysfunction. Therefore the review of symptoms should focus on these areas including defecation (constipation, diarrhea, fecal incontinence, changes in bowel movements), sexual function, dysparunia, and pelvic pain.
- Neurological problems are frequently associated with voiding dysfunction, a thorough neurological history is critical, including known neurologic disease as well as symptoms that could be related to occult neurological disease (back pain, radiculopathy, extremity numbness, tingling, or weakness, headaches, changes in eyesight, and so on).
- A focused history regarding LUTS and voiding dysfunction, a thorough urological history is important. This includes a history of hematuria, urinary tract infections, sexually transmitted diseases, urolilthias is, and urological malignancy and their treatment.
- The past medical history should provide information about concurrent medical diseases, obstetric and gynecologic history, past surgical history, and medication use. Many medications have profound effects on the lower urinary tract or can effect fluid mobilization and urine production and thus contribute to LUTS. Examples of medications that may be associated with voiding dysfunction include alpha-adrenergicagonists, such as pseudoephedrine, diuretics, antidepressants, and anticholinergics.
- A detailed history of known neurological diseases (e.g., stroke, Parkinson's disease, spinal cord injury, multiple sclerosis, myelodysplasia, and so on) is important because these diseaseshave the potential to affect bladder and sphincteric function.
- A history of medical diseases such as diabetes or congestive heart failure can cause LUTS by their effects on the lower urinary tract or fluid mobilization. For women with voiding dysfunction, obstetrical and gynecological history is extremely important. Pregnancy and childbirth, particularly vaginal delivery, are associated with voiding dysfunction, especially in continence and pelvicprolapse. Thus, number of pregnancies, deliveries (including method, i.e., vaginal vs cesarean), and the onset of the symptoms in relation to these events is important.
- A women's hormone status (pre-, peri, or postmenopausal) and the onset of symptoms with changes in status should be noted.

- Prior surgery may have effects on lower urinary tract function. This includes surgery on the lower urinary tract (e.g., prostate surgery in men or incontinence surgery in women). Other pelvic surgery such as gynecological surgery or lower intestinal surgery also may affect the bladder directly or indirectly through damage to the nerve supply to the bladder or sphincter. History of pelvic radiation for treatment of pelvic malignancy (urological, gynecological, or rectal) is important as this can have a marked effect on lower urinary tract function and LUTS.

PHYSICAL EXAMINATION

A complete physical exam is important; however, certain aspects of the exam need to be emphasized. A focused physical examination should be performed to:

1. Assess the bladder for masses and fullness
2. Assess the external genitalia
3. Assess the pelvic floor, including anal sphincter tone, and thoroughly examine for support defects, prolapse, and other pelvic conditions in women
4. Assess the prostate in men
5. Demonstrate incontinence in patients with that symptom
6. Detect neurologic abnormalities that may contribute to voiding dysfunction. The abdominal exam, which includes examination of the flanks, begins with inspection for scars, masses, or hernias. Examination of the back should be performed to check for scars and scolios is which may be an indication of potential spine abnormalities that may contribute to voiding dysfunction.

LABORATORY TESTING

Urine analysis is part of the standard evaluation of the patient with LUTS and voiding dysfunction. Urinalysis can screen for pyuria, bacturia hematuria, and the presence of glucosuria or proteinuria. Voiding dysfunction and LUTS can be associated with infection, malignancy, or medical illness such as diabetes, which can be discovered as a result of an abnormal urine analysis. When abnormalities are found on urine analysis, further testing may be warranted such as urine culturein cases of suspected infection or urine cytology, endoscopic, and radiographic studies when microscopic hematuria is present. Blood tests are useful in select cases of voiding dysfunction.

The most common tests are those that evaluate renal function, e.g., serum blood urea nitrogen and creatinine, in cases where renal insufficiency is known or suspected. In select cases, more specific blood and urine testing

may be performed, but these are usually dependent on patient history and physical as well as the results of simple tests.

SIMPLE TESTS FOR EVALUATING VOIDING DYSFUNCTION;
When history and physical exam alone are insufficient to make a diagnosis or institute treatment, or when more objective information is desired, the clinician may start with simple tests to evaluate lower urinary tract function. These are noninvasive or minimally invasive (placement of a urethral catheter) tests that can provide information that may influence treatment or further diagnostic evaluation.

The most basic of these include;
- a voiding and intake diary
- measurement of post void residual volume
- uroflowmetry, and pad testing.
- Bedside urodynamics

NURSING MANAGEMENT
1. Pelvic floor therapy; a variety of techniques can be used to correct the nerves and muscles that may be responsible for the dysfunction.
2. Intermittent cathetherization of the bladder to avoid any form of urinary retention
3. Muscle relaxants can be administered
4. A bladder pacemaker can be inserted beneath the skin to help the nerves that control the bladder.
5. Other nursing care are accorded based on individual patients need.

MODULE 3 CARING FOR PATIENTS WITH INFLAMMATORY CONDITIONS

UNIT 1 CARING FOR PATIENTS WITH INFECTION/INFLAMMATORY CONDITIONS: ORBITAL AND OCULAR TRAUMA, DRY EYE SYNDROME, CONJUNCTIVITIS; UVEITIS; ORBITAL CELLULITIS

CONTENTS

1.0 INTRODUCTION

All ocular structures are vulnerable to injury, but the site often depends on the cause and mechanism of ocular injury. The anterior segment of the eye which consists of the cornea, conjunctiva, trabecular meshwork, anterior chamber, iris, and crystalline lens is vulnerable to direct trauma. Posterior ocular structures include the retina, choroid and optic nerve. You will now learn more about this.

2.0 OBJECTIVE

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

explain what is involved in caring for patients with eye infection(inflammatory condition).

3.0 MAIN CONTENT

3.1 Ocular Injury

All ocular structures are vulnerable to injury, but the site often depends on the cause and mechanism of ocular injury. The anterior segment of the eye which consists of the cornea, conjunctiva, trabecular meshwork, anterior chamber, iris, and crystalline lens is vulnerable to direct trauma. Posterior ocular structures include the retina, choroid and optic nerve. The worst outcome is often seen in the combined anterior and posterior segment injuries with the possibility of losing all useful vision.

Ocular injuries are divided into open globe and closed globe injuries, however, there may be an overlap in their classification based on the causative agent or inflicting object involved.

An open globe injury (an injury penetrating into the globe) involves a full thickness wound of the corneoscleral wall which may result from penetrating or blunt eye trauma. Open globe injuries include lacerations which are further divided into penetrating injuries, perforating injuries and intraocular foreign bodies.

Closed globe injuries are commonly due to blunt trauma whereby the corneoscleral wall of the globe remains intact (a partial thickness corneal wound) however, intraocular damage may be present. They are divided into burns, blunt trauma/contusions and lamellar lacerations. Ruptures are caused by blunt objects with the actual wound being produced by an inside-out mechanism. If the inflicting object is blunt, it can result in either a contusion or a rupture (open globe).

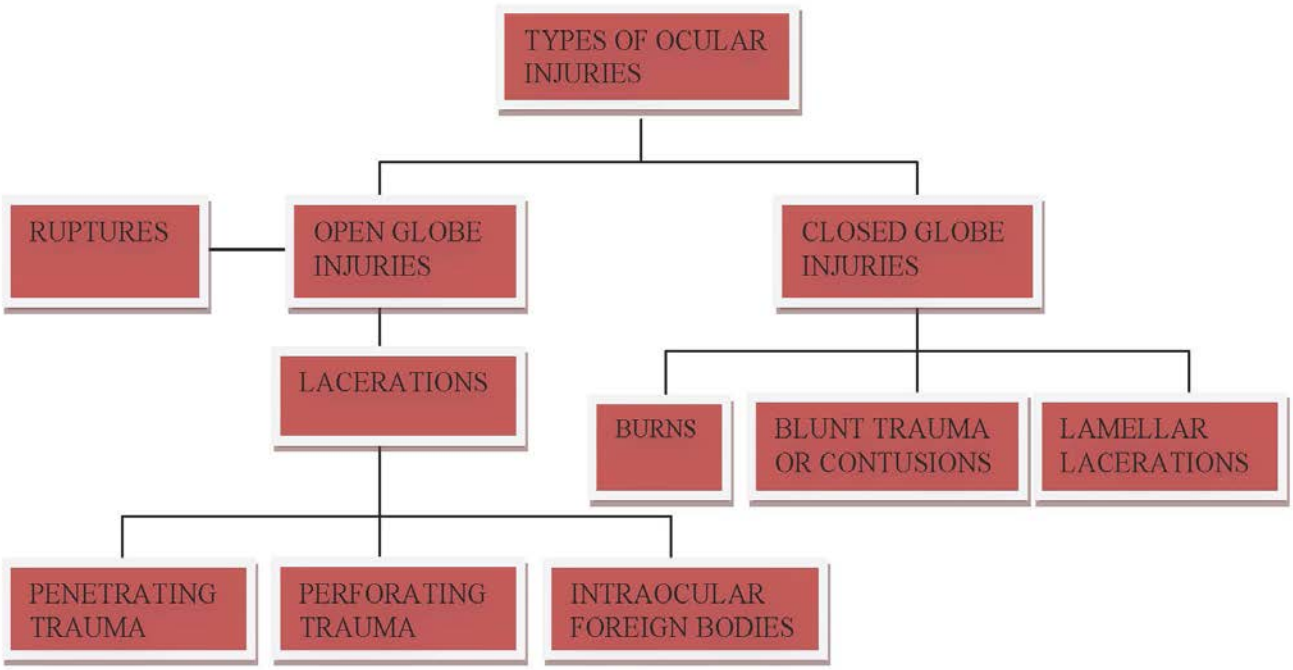

Fig. 1.1: **Standardized classification of ocular trauma using the Birmingham Eye Trauma Terminology (BETT) classification**

Open and closed globe injuries are further described in terms of grades, elaborating on which structure of the eye the wound involves and to what extent it is. For open globe injuries a grade I wound involves the cornea, a grade II wound extends into the anterior 5 mm of the sclera and a grade III wound involves the sclera extending more than 5 mm from limbus. In the case of closed globe injuries, a grade I wound involves only the conjunctiva, sclera or cornea, a grade II injury includes the anterior chamber including the lens and zonules and a grade III injury involves posterior structures including the vitreous, retina, optic nerve, choroid and ciliary body

Lacerations

"A laceration is a full thickness wound of the eye wall, usually caused by a sharp object. The wound occurs at the impact site by an outside-in mechanism. The classification is based on whether an intra-ocular foreign body or an exit wound is also present". Occasionally, an exit wound may be created by the object while remaining partially intraocular.

Lacerations to the eyelids and the conjunctiva commonly occur from sharp objects but can also occur from a fall. Most corneoscleral lacerations are

caused by glass from shattered spectacles and broken windows and associated with blunt trauma of flying objects. Lacerations may occur in one of two ways: i) lacerations without prolapse of tissue when the eyeball has been penetrated anteriorly but without prolapse of the intra-ocular contents; and ii) lacerations with prolapse when a small portion of iris prolapses through a wound, or uveal tissue has been injured. Corneal lacerations can involve the iris and crystalline lens forming a cataract whereby management depends on the duration and extent of the incarceration. Corneal lacerations frequently result in prolapse of the iris with distortion of the pupil. Hyphaema is often present reducing vision in the affected eye.

Ocular lacerations are treated in different ways depending on whether or not there is tissue prolapse. However, the surgeon has to explore the extent of the wound first and then determine the status of the crystalline lens whether to remove it or not with the aid of a slit lamp. A crystalline lens can only be removed following a water tight closure of the laceration. If the wound is extensive and loss of intra-ocular contents has been great enough and the prognosis for useful function is hopeless, enucleation/evisceration is indicated as a primary surgical procedure. However, when the wound is clean without tissue prolapse and free from contamination, it can usually be repaired by direct interrupted sutures and can often heal spontaneously with the aid of an eye pad, contact lenses, or cyanoacrylate adhesives while administering a topical antibiotic and controlling the patient's pain with oral analgesics. A precaution must be taken for a self-sealing wound because when an edematous cornea subsides a wound leak may develop. Therefore, Seidel test is indicated for evaluation of a corneal wound leak to determine whether aqueous is being emitted or not. The patient needs to be referred to the nearest ophthalmologist for surgical repair as soon as possible to restore the anatomy or structural integrity of the globe irrespective of the extent of the injury and the initial visual acuity. If a delay in specialist care is anticipated, a systemic oral antibiotic and tetanus prophylaxis should be administered to avoid development of endophthalmitis. However, it is advisable to wait until repair of the laceration has been completed before adding medications because these could be toxic to the retina.

PREVENTION OF OCULAR INJURY

Public enlightenment
Safety practices at work places
Safety measures at home
Avoid corporal punishment to children
Ensure supervised children play

Obey the traffic rules
Provision of prompt and effective first aids

3.2 Dry Eyes

Dry eye per se is not a disease entity, but a symptom complex occurring as a sequelae to deficiency or abnormalities of the tear film. When considering the tear film disorders, the ocular surface microenvironment of the eyelids, conjunctiva, cornea and tear films need to be evaluated. Any alteration in this environment has the potential to cause a tear film disturbance.

Dry eye can be divided into two categories:

1. aqueous tear-deficient dry eye; its causes include
i. Sjogren's syndrome (Primary Keratoconjunctivitis sicca)
ii. Non Sjogren's Keratoconjunctivitis sicca and
2. evaporative dry eye; its causes include
i. Meibomian gland dysfunction
ii. Lagophthalmos
iii. Defective blinking rate as seen in prolong computer users
iv. Vitamin A deficiency and other factors affecting ocular surface e.g. topical drugs, contact lens wear, preservatives, scaring disorders

SIGNS AND SYMPTOMS

Patients with dry eyes can present with an assortment of symptoms, the most common of which are foreign body sensation, burning, itching, light sensitivity, irritation, and transient blurry vision.
Signs of dry eyes are as follows:

Tear film signs: it may show presence of stingy mucous and particulate matter. Marginal tear strip is reduced or absent (normal height is 1mm)

Conjunctival signs; it becomes lusterless, mildly congested, conjunctival xerosis and keratinization may occur.

Corneal signs; it may show punctate epithelial erosions, filaments and mucous plaques. Cornea may lose luster.
Signs of causative disease such as posterior blepharitis, conjunctival scarring diseases (trachoma, Stevens Johnson syndrome, chemical burns, ocular pemphigoid) and lagophthalmos may be depicted.

DIAGNOSIS
Dry eyes can be detected using tear film tests which include; tear film break-up time (BUT), Schirmer-I test, vital staining with Rose Bengal.

MANAGEMENT
Medical management for dry eyes includes ocular surface lubrication in the form of drops, gels, and ointments. These should be used on a frequent basis for them to be therapeutic.

The following treatment modalities can be employed, namely; use of artificial tears, preservation of the existing tears by reducing evaporation and decreasing drainage, and treatment of causative disease of dry eye.

Surgical treatment is in the form of temporary or permanent punctual occlusion to assist with tear preservation.

Dry eye is a chronic condition that can often be controlled but not cured. Commonly management of these patients is psychological. Nurses need to spend time counseling these patients with careful explanation of the problem to assist in reducing patient's complaints, frustrations and fears.

3.3 Conjunctivitis

Conjunctivitis can be defined as the inflammation of th conjunctiva. It is evidenced by redness of the conjunctiva associated with discharges which may be watery, mucoid, mucopurulent or purulent.

CLASSIFICATION OF CONJUNCTIVITIS
Conjunctivitis can be classified as either based on the cause or on the clinical findings:

AETIOLOGICAL CLASSIFICATION
1. Infective conjunctivitis; bacterial, chlamydial, viral, fungal, rickettsial, spirochaetal, protozoal, parasitic
2. Irritative conjunctivitis
3. Allergic conjunctivitis
4. Keratoconjunctivitis, which is associated with disease of skin and mucous membrane
5. Traumatic conjunctivitis

CLINICAL CLASSIFICATION
1. Acute catarrhal or mucopurulent conjunctivitis

2. Acute purulent conjunctivitis
3. Serous conjunctivitis
4. Chronic simple conjunctivitis
5. Angular conjunctivitis
6. Membranous conjunctivitis
7. Pseudomembranous conjunctivitis
8. Papillary conjunctivitis
9. Follicular conjunctivitis
10. Ophthalmia neonatorum
11. Granulomatous conjunctivitis
12. Ulcerative conjunctivitis
13. Cicatrizing conjunctivitis

3.3.1 Bacterial Conjunctivitis

This is inflammation of the conjunctiva with diffuse injection of the superficial episcleral vessels, bulbar conjunctiva and occasionally papillae of the palpebral conjunctiva of the upper and lower lid. The condition is common in children and normally starts off in one eye before transmitting itself to the other eye thereby making unilateral bacterial conjunctivitis uncommon. Bacterial conjunctivitis is highly contagious and commonly occurs as an epidermics.

PREDISPOSING FACTORS

Flies
Poor hygienic conditions
Hot dry climate
Poor sanitation
Dirty habits

CAUSATIVE ORGANISMS

The main causative organisms are Streptococcus pneumonia, Staphylococcus aureus, Haemophilus influenza, Pseudomonas aeruginosa and gonococci

SIGNS AND SYMPTOMS

Red eye
Profuse and purulent discharge
Foreign body sensation in the eye
Crusty lid margin and lashes especially on waking up
Eye lids may be oedematous
Mild photophobia
Chemosis i.e. swelling of the conjunctiva

MANAGEMENT

Although acute bacterial conjunctivitis is usually self-limiting and does not cause any serious damage, patients presenting with it should be treated in order to shorten the course of the disease, reduce the spread of the disease, and reduce the risk of more extraocular disease.

i. Topical antibiotics to control the infection e.g. chloramphenicol 1%, gentamycin 0.3%, tobramycin 0.3%, ciprofloxacin 0.3%, ofloxacin 0.3% eye drops 3-4 hourly in the day and ointment used at night
ii. Irrigation of the conjunctival sac with sterile warm saline once or twice a day
iii. Use of dark goggles to prevent photophobia
iv. Do not pad the eye
v. Do not use steroids
vi. Analgesics and anti-inflammatory (e.g. paracetamol, ibuprofen) orally for 2 – 3 days

NURSING CARE

i. Health education information particularly on how to control the spread of infection
ii. Personal and environmental hygiene
iii. Regular hand washing before and after instillation of eye medications
iv. Encourage the use of dark glasses and never to pad the eye
v. The use of disposable tissue to wipe the eye instead of handkerchief
vi. Disallow sharing of face cloths, towels, handkerchief etc
vii. Discard the use of make-up
viii. Discontinue the use of contact lens
ix. Follow up visits to detect changes in visual acuity, development of new symptoms

3.3.2 Ophthalmia Neonatorum

It is the term used for bilateral inflammation of the conjunctiva occurring during the first 28-30 days of life. It is also known as conjunctivitis of the newborn. It is a preventable disease usually occurring as a result of carelessness at the time of birth.

CAUSATIVE AGENTS

Chemical agents e.g. drugs
Neisseria gonococcus
Chlamydia trachomatis
Staphylococcus aureus

Streptococcus haemolyticus
Streptococcus pneumoniae
Herpes simplex virus

SIGNS AND SYMPTOMS
Pain and tenderness
Lid oedema
Purulent conjunctival discharge (much more profuse in gonococcal conjunctivitis)
Conjunctival chemosis

DIAGNOSIS
Microscopic culture and sensitivity of eye swab

MANAGEMENT
Prophylactic management involves antenatal measures (treatment of genital infections), peri-natal measures (hygienic environment and instruments), and post-natal measures (Crede's prophylaxis).
Curative management entails;
Saline lavage hourly till the discharge is eliminated.
Antibiotics therapy both systemic and topical e.g. ceftriaxone 75-100mg/kg/day, ciprofloxacin 10-20mg/kg/day

NURSING CARE
Clear and careful explanation to both parents of the condition, source and mode of infection
Educate the parent on the need to get screened and examined for genital infection
Educate and demonstrate to the career on how to clean the eyes using clean cotton wool and warm water
Educate and demonstrate on the correct method of instilling eye medications
Stress the importance of follow-up in the clinic

3.3.3 Allergic Conjunctivitis

This is a recurrent bilateral inflammation of the conjunctiva. It is periodical and seasonal depending on the cause. it is commoner in Nigeria between March and September during the corn season due to allergy from corn pollen. It is a self-limiting condition and the disease does not persist to adult life except in very few cases.

CAUSES
The cause is non-confirmatory but allergen like pollen, animal, fur, dust is suggestive.
Family history of hay fever, asthma or eczema
Commoner in temperate than warm climate
It is very severe in summer than in winter

SIGNS AND SYMPTOMS
Severe itching
Brownish conjunctiva
Ropy discharge
Mild photophobia
Foreign body sensation
Tearing
Severe chemosis during the acute attack

MANAGEMENT
Educate the patient and relations on the course of illness
Eye examination and visual acuity
Management is usually palliative
Educate on personal hygiene so as to prevent cross infection
Topical drugs commonly used are; Gutt spersallerge qds, or guttt alomide qds instilled into the affected eye
Tab piriton 2-4mg nocte

3.4 Uveitis

Uveitis refers to inflammation of the uveal tract and is the third most common cause of blindness in developed countries, generally affecting people aged 20-50 years with children making up about 5% of cases. Uveitis may be classified in many ways but a simple classification is on the basis of anatomy, clinical features and aetiology (Kanski, 2003).

A: ANATOMICAL CLASSIFICATION
It can be classified as follows:

a) Anterior uveitis: inflammation of the iris (iritis), inflammation of the ciliary body especially the pars plicata (cyclitis) and inflammation of the iris, ciliary body and anterior vitreous (iridocyclitis)
b) Intermediate uveitis: chronic inflammation of the pars plana, the extreme periphery of the retina and the choroid. It is also called pars planitis.

c) Posterior uveitis: inflammation of the choroid (choroiditis) and when the retina is involved (chorioretinitis)
d) Panuveitis: diffuse uveitis involving both anterior and posterior ocular structures (i.e. inflammation of the whole uvea)

B: CLINICAL CLASSIFICATION

Uveitis can be classified as;

a) Acute- occurring with a sudden, symptomatic onset lasting for about 6 weeks up to 3 months
b) Chronic- persisting longer than 3 months with a frequently insidious and asymptomatic onset.
c) Recurrent uveitis- characterized by repeated episodes with inactive periods of ≥ 3 months of treatment.

C: AETIOLOGICAL (DUKE ELDER'S) CLASSIFICATION

a) Infective uveitis
b) Immune-related uveitis
c) Toxic uveitis
d) Traumatic uveitis
e) Uveitis associated with non-infective systemic diseases
f) Idiopathic uveitis

3.4.1 Anterior Uveitis

It is inflammation of the uveal tissue from iris up to the pars plicata of the ciliary body. It may present clinically as either acute or chronic anterior uveitis.

Main symptoms of acute anterior uveitis are pain, photophobia, redness, lacrimation, and decreased vision.
In chronic uveitis, the eye may be white with minimal symptoms even in the presence of signs of severe inflammation

INVESTIGATIONS

Eye examination
Visual acuity
Haematological investigations (e.g. blood sugar level, serological tests, ESR, Rh factors etc.)
Urine and stool examination
Radiological investigations (e.g. x-rays, CT scan, MRI scan)
Skin tests (e.g. tuberculin test, toxoplasmin test)

COMPLICATIONS
Glaucoma
Cataract
Retinal detachment
Phthisis bulbi

3.4.2 Intermediate Uveitis

It refers to the inflammation of the pars plana ciliaris, peripheral retina, choroid and vitreous base. It accounts for 10% of all cases of uveitis.

CAUSES
Its cause is majorly idiopathic. Known causes (15%) include tuberculosis, syphilis, sarcoidosis, and Lyme disease.

SIGNS AND SYMPTOMS
Asymptomatic in many cases

COMPLICATIONS
Secondary glaucoma
Complicated cataract
Vitreous haemorrhage

3.4.3 Posterior Uveitis

This refers to inflammation of the choroid (choroiditis). Since the outer layers of retina are in close contact with the choroid and also depend on it for the nourishment, the choroidal inflammation almost always involves the adjoining retina leading to chorioretinitis.

SIGNS AND SYMPTOMS
Choroiditis is a painless condition, usually characterized by visual symptoms due to associated vitreous haze and involvement of the retina.
Defective vision
Black spots floating in front of the eyes
Metamorphosia
Positive scotoma
Vitreous opacity

COMPLICATIONS
Extension of the inflammation to anterior uvea
Complicated cataract
Vitreous degeneration
Macular oedema

Retinal detachment

MANAGEMENT OF UVEITIS

Aims:

i. To alleviate acute symptoms and suppress inflammation
ii. To preserve vision and prevent complications
iii. To treat the cause of inflammatory process if known

1. Non-specific treatment

A. Local therapy

a. Cycloplegic drugs- they are very useful and most effective during acute phase of iridocyclitis. Examples are 1% atropine eye drops/ ointment, 2% homatropine or 1% cyclopentolate eye drops. Also a subconjunctival injection of 0.25ml mydriacain (a mixture of atropine, adrenaline and procaine) should be given for more powerful cycloplegic effect.
b. Corticosteroids help to reduce the inflammation. It can be in forms of eye drops, eye ointment or anterior sub-tenon injection
c. Broad spectrum antibiotic eye drops

B. Systemic therapy

a. Corticosteroids- it provides potent effect especially in antigen-antibody reaction. Also in intractable anterior uveitis resistant to topical therapy. Examples are 60-100mg of Prednisolone or equivalent quantities of other steroids like dexamethasone or betamethasone.
b. Non-steroidal anti-inflammatory drugs- aspirin can be used where steroids are contraindicated, naproxen in patients with ankylosing spondilitis, phenylbutazone and oxyphenbutazone in uveitis associated with rheumatoid disease.
c. Immunosuppressive drugs- these should be used only in desperate and extremely serious cases of uveitis. Examples are cyclo-phosphamide, chlorambucil, azathioprine and methotrexate
d. Azithromycin or tetracycline or erythromycin to treat patients with chlamydial infection

C. Physical measures

Hot formentation is very soothing, reduces pain and increases circulation.
The use of dark goggles gives a feeling of comfort by reducing photophobia, lacrimation, blepharospasm.

2. Specific treatment

As effective as the non-specific treatment is, in most of the cases, it does not cure the disease, resulting in relapse. Therefore, efforts should be made to find out and treat the underlying cause.

3.5 Orbital Cellulitis

Orbital cellulitis is a bacterial infection of the soft tissue behind the orbital septum and is caused by the same organisms that cause acute sinusitis, i.e. pneumococci, streptococci or staphylococci. These enter the orbit from the infected frontal, maxillary, ethmoidal or sphenoidal sinuses.

Orbital cellulitis is a potentially life-threatening and vision-threatening condition. It is more common in children than adults.

CAUSES

The modes of infection can be;

1. Exogenous infection: it may result from penetrating injury especially when associated with retention of intraorbital foreign body, and

following operations like evisceration, enucleation, dacryocystectomy and orbitotomy.

2. Extension of infection from neighbouring structures: it is the most common mode of orbital infections. These include paranasal sinuses, teeth, face, lids, intracranial cavity and intraorbital structures
3. Endogenous infection: it may rarely develop as metastatic infection from breast abscess, puerperal sepsis, thrombophlebitis of legs and septicaemia

SIGNS AND SYMPTOMS

Orbital cellulitis is almost always unilateral, with a sudden onset. Its clinical features include;
Severe pain,
Fever,
proptosis, and
Restriction of ocular movements (extraocular muscles).
Others are swelling of lids, chemosis of conjunctiva, nausea and vomiting, redness of the eyelids.

INVESTIGATIONS

Eye examination
Visual acuity
Bacterial cultures of nasal and conjunctival swabs and blood samples
X-ray of the orbit and paranasal sinuses
CT scan and MRI are useful in detecting subperiosteal abscesses, orbital abscesses, intracranial extension etc.

MANAGEMENT

The patient should be admitted to hospital and assessed frequently because orbital cellulitis can be vision threatening or even life threatening. However, almost all cases respond well to large doses of systemic antibiotics.

Intensive Antibiotic therapy: Once the causative organism is identified, treat aggressively to overcome the infection. Intravenous antibiotics should be administered e.g. ceftriaxone

Analgesic and anti-inflammatory drugs are helpful in controlling pain and fever e.g. diclofenac

Surgical intervention is indicated when the patient is non-responsive to antibiotics, decreasing vision and presence of an orbital or subperiosteal abscess. Incision and drainage is instituted.

Patients need to be nursed in a quiet, dimly lit room. Cold compresses are normally found to be soothing and can relieve some ocular discomfort. Moistened eye pads can be utilized, using clean pads each time, and safely discarding the used pads.

Physical care should include care related to rest and sleep, adequate oral fluid and nutrition intake, personal and oral hygiene, temperature regulation and elimination needs

COMPLICATIONS

Ocular complications including exposure keratopathy, optic neuritis and central retinal artery occlusion

Orbital complications including subperiosteal abscess, orbital abscess

Intracranial complications include cavernous sinus thrombosis, meningitis and brain abscess

Temporal or parotid abscess

General septicaemia or pyaemia

UNIT 2 CARING FOR PATIENTS WITH ORBITAL TUMORS AND ORBITAL SURGERIES/ ENUCLEATION

CONTENTS

1.0 INTRODUCTION

In this unit, you will learn about caring for patients with orbital tumors.

2.0 OBJECTIVES

At the end of this unit, you should be able to:
explain the process of caring for patients with orbital tumors.

3.0 MAIN CONTENT

3.1 Orbital Tumors

RETINOBLASTOMA

It is a congenital malignant tumour arising from the neurosensory retina in one or both eyes. It is the most common childhood intraocular malignancy which appears before the age of 3 years.

SIGNS AND SYMPTOMS

Leukocoria: Is the most common presentation. Presenting as yellowish-white papillary reflex (also known as amaurotic cat's eye appearance)
Strabismus
Painful red eye with hypopyon
Nystagmus

STAGES OF RETINOBLASTOMA

It may be divided into four stages:

I Quiescent stage, lasting for about 6 Months to 1 year
II Glaucomatous/inflammatory stage
III Stage of extraocular extension
IV Stage of distant metastasis

DIAGNOSIS

Examination under anaesthesia
Plain x-rays of the orbit
Raised lactic dehydrogenase (LDH) level in the aqueous humour
Ultrasonography and CT scanning

MANAGEMENT

Treatment depends on the stage of the disease
When the tumour is diagnosed at an early stage (stage I), it may be treated conservatively
Conservative management entails:

i. Chemotherapy (use of drugs like vincristine, carboplatin, 5-flouracil)
ii. Local therapy: depending upon the location and size of the tumour, therapy can be chosen from the following modalities;
    - Crotherapy
    - Laser photocoagulation
    - Thermotherapy
    - Plaque radiotherapy
    - External beam radiotherapy (EBR)

But when the tumour is more than half of the retina, or the optic nerve is involved, or glaucoma is present, then enucleation is the treatment of choice
Postoperatively, radiotherapy (5000 rads) should be applied to the orbital apex and chemotherapy instituted.

NURSING CARE

A diagnosis of retinoblastoma is devastating for the parents of the child and it is essential that education and support be provided, particularly as there are frequent ocular examinations and treatments required. The nurses must ensure that they are cognizant with all the procedures and processes in their care.
The two parents must be involved in the care. Educate them on the nutritional care, family support and never to abandon the child.
Educate the parents on the need for follow up care.

3.2 Enucleation

Enucleation is the excision of the eyeball. It can be performed under local anaesthesia in adults and under general anaesthesia in children. Postoperatively, a conformer is used so that the conjunctival fornices are retained deep. A proper sized prosthetic eye can be inserted 6 weeks when healing of the enucleated socket is complete for good cosmetic appearance.

INDICATIONS

1. Absolute indications- retinoblastoma, malignant melanoma
2. Relative indications- painful blind eye, mutilating ocular injuries, anterior staphyloma, and phthisis bulbi
3. Indication for eye donation from cadaver is presently the mist common indication.

EVISCERATION: is the removal of the content of the eyeball. It is the scooping out of content of the eye ball, the cornea is cut off leaving behind the sclera coat and the optic nerve

ENUCLEATION: this is the removal of the whole globe. This is the removal of the whole content of the eye ball leaving only part of the optic nerve.

EXENTERATION: this is the removal of the eye ball, the fibrous and fatty tissue of the eye, and part of the orbital bone.

UNIT 3 CARING FOR PATIENTS WITH OCULAR CONSEQUENCES OF SYSTEMIC DISEASE: DIABETIC RETINOPATHY; CYTOMEGALOVIRUS; RETINITIS; HYPERTENSION-RELATED EYE CHANGES

CONTENTS

1.0 INTRODUCTION

DR refers to retinal changes seen in patients with diabetes mellitus. It is the leading cause of blindness in the Western countries. You are going to learn more about this shortly.

2.0 OBJECTIVES

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

- discuss caring for patients with ocular consequences of systemic disease**.**

3.0 MAIN CONTENT

3.1 Diabetic Retinopathy (Dr)

DR refers to retinal changes seen in patients with diabetes mellitus. It is the leading cause of blindness in the Western countries. The incidence of diabetic retinopathy has been on the increase in recent times owing to the increase in the life expectancy of diabetics.

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RISK FACTORS

Duration of diabetes is the most determining factor

Gender: more in females than males (4:3)
Heredity
Hypertension
Poor metabolic control, though less important than the duration
Others are smoking, obesity, anaemia and hyperlipidemia

CLASSIFICATION

DR is classified as follows:

1. Non-proliferative diabetic retinopathy (NPDR)
    - Mild NPDR
    - Moderate NPDR
    - Severe NPDR
    - Very severe NPDR
2. Proliferative diabetic retinopathy (PDR)
3. Diabetic maculopathy
4. Advanced diabetic eye disease (ADED)

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DIAGNOSIS

Urine examination
Blood sugar estimation
24 hour urinary protein
Renal function test
Lipid profile
Glycosylated haemoglobin
Fundus fluorescein angiography
Optical coherence tomography (OCT)

MANAGEMENT

i. Screening for diabetic retinopathy: a periodic fundus examination and follow-up is very important to provide timely intervention and to prevent visual loss occurring from diabetic retinopathy
ii. Medical treatment: this plays an essential role and can be discussed as
    1. Control of systemic risk factors which influence the occurrence, progression and effect of laser on DR. risk factors like hypertension, raised blood sugar, anaemia, hypoproteinemia
    2. Role of pharmacological modulation such as anti-vascular endothelial growth factors (Anti-VEGF), antioxidants e.g. vitamin E
    3. Role of intravitreal steroids in reducing diabetic macular oedema such as flucinolone acetonide intravitreal implant, and intravitreal injection of 2-4 mg triamcinolone

i. Photocoagulation: this remains the mainstay in the treatment of diabetic retinopathy and maculopathy. Double frequency YAG laser 532nm is preferred.
ii. Surgical treatment: vitrectomy is required in advanced cases of PDR

NURSING CARE
Nursing interventions for diabetic retinopathy focus on preventive eye care. Patients are taught the importance of yearly comprehensive eye examinations.
Assist patients to keep their diabetes under control, thus helps to reduce the onset of this condition.
For patient with visual loss, nursing care is focused on assisting the individual with home and health maintenance.

3.2 Cytomegalovirus (Cmv) Retinitis

CMV retinitis usually occurs in immune-compromised patients e.g. those suffering from AIDS, on cytotoxic chemotherapy, or long term immunosuppresion following renal transplantation. It is the most frequent (40% prevalence) ocular opportunistic infection in patients with AIDS. However, there has been a dramatic reduction since the advent of Highly Active Antiretroviral Therapy (HAART).

SIGNS AND SYMPTOMS
Often asymptomatic, but some may present with decreased vision and/or floaters in one or both eyes

MANAGEMENT
HAART is recommended to reduce retroviral load and to increase CD4+ count in patients with AIDS
Specific anti-CMV treatment includes; valaganciclovir, ganciclovir, foscarnet, and intravenous injections of cidofovir individually or in combination.

COMPLICATIONS
Retinal detachment
Retinal atrophy
Optic nerve disease

3.3 Retinitis

This is an inflammatory disorder of the retina. It occurs in 2 forms;

NON-SPECIFIC RETINITIS- it is caused by pyogenic organisms and may be either acute or sub-acute.

1. Acute purulent retinitis: it occurs in patients with pyaemia as metastatic infection. It readily metastasizes to endophthalmitis or even panophthalmitis.
2. Sub-acute retinitis of Roth: it typically occurs in patients suffering from sub-acute bacterial endocarditis. It is characterized by multiple superficial retinal haemorrhages, involving posterior part of the fundus. Most of the haemorrhages have a white spot in the center known as Roth's spots

SPECIFIC RETINITIS- it may be bacterial (tuberculosis, leprosy, syphilis), viral (rubella, herpes zoster), mycotic, rickettsia or parasitic in origin.

MANAGEMENT
Identification of the primary cause and treat
Retinitis is managed through argon laser, photocoagulation and cryotherapy.

3.4 Hypertensive Retinopathy

It refers to fundus changes occurring in patients suffering from systemic hypertension. It occurs secondary to an elevated systemic blood pressure, resulting in changes in the vasculature of the retina and choroid. Sustained hypertension causes disruption of the blood-retina barrier with the resultant increase in vascular permeability. Depending on the severity of hypertension, the retinal arterioles respond with narrowing of the lumen (vasoconstriction). This later leads to thickening of the vessel wall (arteriosclerotic changes), hypoxia which results in increased vascular permeability. According to Keith and Wegner (1939) hypertensive retinopathy changes occurs in 4 grades:

Grade I (Mild Retinopathy): it consists of mild generalized arteriolar attenuation, particularly of small branches, with broadening of the arteriolar light flex and vein concealment

Grade II (Moderate retinopathy): it comprises marked generalized narrowing and focal attenuation of arterioles associated with deflection of veins, cotton-wool spots

Grade III (Severe Retinopathy): it consists of grade II changes plus copper-wiring of arterioles, flame-shaped haemorrhages, cotton-wool spots, hard exudates

Grade IV (Malignant retinopathy): it consists of changes in Grade III plus silver-wiring of arterioles and papilloedema.

SIGNS AND SYMPTOMS

This depends on the severity of the retinopathy
It is generally asymptomatic in mild retinopathy
Blurred or distorted vision in moderate-severe retinopathy
Complains of headache in moderate-severe retinopathy
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MANAGEMENT

Control of hypertension is the key to controlling the retinopathy

UNIT 4 CONCEPT IN OCULAR MEDICATION ADMINISTRATION

CONTENTS

1.0 INTRODUCTION

Medications are administered in ophthalmic setting through various methods. These are:

2.0 OBJECTIVE

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

- discuss ocular medication administration**.**

3.0 MAIN CONTENT

3.1 Topical Instillation

This is the most commonly employed mode of administration for ocular therapeutics. The drugs can be administered topically in the form of eye drops, ointments, gels, ocuserts and with the help of soft contact lenses.

(a) Eye drops (gutta): This is the simplest and most convenient method of topical application, especially for daytime use. Eye drops may be in the form of aqueous solutions (drug totally dissolved) or aqueous suspensions (drug is present as small particles kept suspended in the aqueous medium) or oily solutions. Application in the form of eye drops makes the drug available for immediate action but it is quickly diluted by tears within about a minute.

(b) **Eye ointment (oculenta):** Topical application in the form of an eye ointment increases the bioavailability of the drug by increasing tissue contact time and by preventing dilution and quick absorption. However, the drug is not available for immediate use and ointments blur the vision. These are best for bedtime application or when ocular bandage is to be applied.

(c) **Gels:** These have prolonged contact time like ointments and do not cause much blurring of vision. However, they are costly and difficult to prepare.

(d) **Ocuserts:** These form a system of drug delivery through a membrane. These can be placed in the upper or lower fornix up to a week and allow a drug to be released at a relatively constant rate. Pilocarpine ocuserts have been found very useful in patients with primary open-angle glaucoma; by efficiently controlling intraocular pressure with comparatively fewer side-effects.

(e) **Soft contact lenses:** These are very good for delivering higher concentrations of drugs in emergency treatment. A pre-soaked soft contact lens in 1% pilocarpine has been found as effective as 4% pilocarpine eye drops in patients with acute angle closure glaucoma. Soft contact lenses are also used to deliver antibiotics and antiviral drugs in patients with corneal ulcers.

3.2 Periocular Injections

These are not infrequently employed to deliver drugs. These include subconjunctival, sub-tenon, retrobulbar and peribulbar injections.

(a) **Sub-conjunctival injections:** Drug penetration is by local diffusion through the tissues. The advantage is to achieve high local concentration and high tissue concentration of drug. Further, the drugs which cannot penetrate the cornea owing to large-sized molecules can easily pass through the sclera.

(b) **Sub-tenon injections:** These are preferred over subconjunctival injection. Anterior sub-tenon injections are used mainly to administer steroids in the treatment of severe or resistant anterior uveitis. Posterior sub-tenon injections are indicated in patients with intermediate and posterior uveitis.

(c) **Retrobulbar injections:** These are used to deliver drugs for optic neuritis, papillitis, posterior uveitis and also for administering retrobulbar block anaesthesia.

(d) **Peribulbar injections:** These are now frequently used for injecting anaesthetic agents. Peribulbar anaesthesia has almost replaced retrobulbar and facial block anaesthesia.

3.3 Intraocular Injections

Such injections are made in desperate cases (e.g., endophthalmitis) to deliver the drugs in maximum concentration at the target tissue. These include: intracameral injection (into the anterior chamber), and intravitreal injection (into the vitreous cavity).

3.4 Systemic Administration

The systemic routes include oral intake and intramuscular and intravenous injections. The intraocular penetration of systemically administered drugs mainly depends upon the blood-aqueous barrier. The passage through blood-aqueous barrier in turn is influenced by the molecular weight and the lipid solubility of the drug. Only low molecular weight drugs can cross this blood-aqueous barrier. No passage is allowed to large sized molecules, such as penicillin. Out of the borderline molecular weight drugs, those with high lipid solubility can pass easily e.g., sulphonamides have the same molecular weight as sucrose but are 16 times more permeable due to their lipid solubility. Similarly, chloramphenicol being lipid soluble also enters the eye easily.

CLASSIFICATION OF OPHTHALMIC DRUGS

Drugs acting on the eye may be classified under a variety of headings, but is classified here as thus;

- Antimicrobial agents, including antibiotics, antiviral and antifungal agents
- Anti-inflammatory agents, including steroids and antihistamines
- Drugs affecting the autonomic nervous system
- Drugs used in the treatment of glaucoma
- Ocular lubricants
- Local anaesthetic agents
- Diagnostic agents

1. ANTIMICROBIAL AGENTS

Antibiotics: These can be classified as either bactericidal or bacteriostatic.

Bactericidal is when the drug destroys the bacteria during active multiplication while bacteriostatic is when the drug diminishes the rate of multiplication.

Antibiotics may act by one or more bacteriostatic means:

- Interference with the synthesis of the cell wall of the bacterium, e.g. penicillin
- Prevention of protein synthesis inside the micro-organism, e.g. erythromycin
- Disturbance of cell wall permeability so that the bacteria dies, e.g. polymyxin B
- Inhibiting the enzyme responsible for supercoiling of the DNA helix (DNA gyrase), e.g. ofloxacin

Antibiotics can have a narrow or broad spectrum of activity; those with a narrow spectrum are effective against a specific type of bacterium (such as Gram positive); those that have a broad spectrum are effective against a wider range of bacteria. It should be noted that resistance to antibiotics can develop quite quickly and therefore antibiotics should be used only when an accurate diagnosis has been made and ideally after antibiotic sensitivity has been determined.

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Antivirals: Viruses proliferate inside cells, so antiviral agents must be able to penetrate the cells to prevent viral multiplication. There are several viruses that may affect the eye but effective treatment is available for only a few.

Examples are acyclovir, trifluorothymidine (F_3T); effective against the herpes simplex keratitis

Ganciclovir, fomivirsen are used in the treatment of cytomegalovirus (CMV) retinitis

Antifungals: These are rarely required in ocular disease. Fungal infections occur mostly after agricultural injuries, especially in hot and humid climates. Many different fungi are capable of producing ocular infection and they should be identified by appropriate laboratory procedures. Antifungal drugs that have been used in ocular infections include amphotericin and miconazole.

2. ANTI-INFLAMMATORY DRUGS

Corticosteroids: Steroids are substances that are normally produced in the cortex of the adrenal gland. Hydrocortisone has many physiological effects but the one with which we are concerned here is its anti-inflammatory effect. Local and systemic steroids are used in the treatment of eye disease. They are administered as eye drops, eye ointments or subconjunctival injection. They are important in the treatment of anterior segment inflammation and also post-operatively.

Examples are betamethasone, neomycin, dexamethasone, fluorometholone etc.

Non-steroidal anti-inflammatory drugs: these are drugs that block the effects of prostaglandins, which are found in almost all tissues including the eye. Prostaglandins are released in inflammatory reactions and are said to be mediators in the process.

Examples are diclofenac, flurbiprofen, ketorolac

Drugs for the treatment of allergy: Allergy is a common cause of conjunctivitis and the cause of signs and symptoms is principally the release of histamine. Treatment can be offered in two ways:

- Using drops that block histamine receptors, e.g. emedastine (antihistamine)
- Using drops that prevent the release of histamine, e.g. iodoxamide

3. DRUGS AFFECTING THE AUTONOMIC NERVOUS SYSTEM

Drugs affecting the sympathetic nervous system: Sympathomimetic agents mimic the actions of the transmitter thus producing some or all of the following effects:

- Dilatation of the pupil
- Reduction in the rate of production of aqueous humour
- Increase in outflow through the trabecular meshwork by lowering outflow resistance
- Constriction of the conjunctival vessels

Examples are phenylephrine, guanethidine

Drugs affecting the parasympathetic nervous system: The parasympathomimetics (miotics) may work either:

- Directly, e.g. pilocapine which causes miosis i.e. increased outflow of aqueous by opening up the inefficient drainage channels in the trabecular meshwork.
- Indirectly, e.g. by acting on enzymes that normally metabolize acetylcholine and therefore potentiate its action

The parasympatholytic agents work by blocking acetylcholine. Hence, they cause dilatation and varying degrees of cyclopegia. They vary in potency and duration of action.

Examples are atropine, tropicamide

4. ANTIGLAUCOMA DRUGS

Glaucoma is usually (but not always) with an abnormally high IOP.

The rise in pressure is almost always the result of reduced outflow of aqueous humour, the inflow remaining constant. Treatment is aimed at reducing IOP. Examples of the drugs based on their method of reducing the IOP are:

i. Drugs which increase trabecular outflow

Miotics (e.g., pilocarpine)
Epinephrine, Dipivefrine
Bimatoprost

ii. Drugs which increase uveoscleral outflow

Prostaglandins (latanoprost)
Epinephrine, Dipivefrine
Brimonidine
Apraclonidine

iii. Drugs which decrease aqueous production

Carbonic anhydrase inhibitors (e.g., acetazolamide, dorzolamide)
Alpha receptor stimulators in ciliary process (e.g., epinephrine, dipivefrine, clonidine, brimonidine, apraclonidine.
Beta blockers (e.g., timolol, betaxolol, levobunolol)

iv. Hyperosmotic agents (e.g., glycerol, mannitol, urea)

5. OCULAR LUBRICANTS

The precorneal tear film is made up of lipid, aqueous and mucin components. The outer lipid layer helps to decrease evaporative loss, while the inner mucin layer provides a hydrophilic surface, allowing the aqueous layer to maintain contact with the cornea. Disturbance in any of the layers affects the function of others, leading to a dry eye condition. Some of the available preparations are:

Hypromellose: traditional choice. It may need to be instilled frequently. Useful when aqueous is deficient

Polyvinyl alcohol: hydrophilic, mucomimetic

Carbomers: it improves tear film stability and prolongs tear break-up time
Paraffin eye ointments: it decreases evaporative loss. It is useful in corneal erosion

Systane: it is a gell and lubricating polymer system.

6. LOCAL ANAESTHETIC AGENTS

The topical anaesthetic agents are useful for examination and treatment of simple procedures such as removal of foreign bodies from the cornea, but they should never be used for the management of ocular symptoms.
Examples are tetracaine (amethocaine), lidocaine (lignocaine)

1. DIAGNOSTIC AGENTS (DYES)

Fluorescein sodium: This is the most commonly used diagnostic agent and is available in a number of different forms;

- 1% or 2% solution for topical use
- Dry paper impregnated with 1mg fluorescein
- In combination with lidocaine for tonometry
- An intravenous form that can be obtained in various strengths

Rose Bengal: this is more efficient for the diagnosis of conjunctival epithelial damage than fluorescein sodium, but it stings excessively (more so in those patients with dry eyes) unless a local anaesthetic is instilled beforehand.

HOW TO APPLY EYE DROPS

1. Wash hands.
2. Tilt the head back as if looking at the ceiling.
3. Gently pull the lower eyelid down until it forms a small pocket, or pouch.
4. Squeeze the bottle or the dropper to release a single drop into the eye. The nozzle of the bottle should not the eye or eyelid.
5. Release the lower lid and close the eyelid for 30seconds. Dab excess with cotton wool.

HOW TO APPLY EYE OINTMENT

1. Wash hands.
2. Tilt the head back and look at the ceiling.
3. Gently pull the lower eyelid down until it forms a small pocket or pouch.
4. Hold the tube parallel to the eye to avoid injury. Squeeze a line of approximately 1/2cm (1/4inch) of ointment into the pouch without touching the eye, lid or lashes.
5. Release the lower lid and allow to blink. Wipe away the excess ointment with cotton wool.

SPECIAL PRECAUTIONS

1. In order to keep the eye drops clean;
   i. The nozzle should not be allowed to touch the eye or anything else.
   ii. The eye drops is not transferable, so others should not use it.
   iii. The cap of the eye drops should be immediately replaced.
   iv. Discard the eye drops 4weeks after first opening.

2. If you are using more than one kind of eye drop in the same eye, wait 5minutes between each drop.

3. If you are using both drops and ointment in the same eye, the drops should always be used first and wait for 5miutes before applying the ointment

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4. If you wear contact lens, you should stop wearing them for the duration of the treatment unless otherwise directed by the physician.

6.0 REFERENCES/FURTHER READING

1. Anne Waugh and Allison Grant (2010). Ross and Wilson Anatomy and Physiology in Health and Illness. 11th Edition. UK: Elsevier Limited

2. Janet Marsden (2007). Ophthalmic Care. England: Whurr Publishers Limited

3. Khurana A.K. (2007). Comprehensive Ophthalmology. New Delhi: New Age International Limited

4. Suzanne C.S., Brenda G.B., Janice, L.H., Kerry H.C. Brunner & Suddath's Textbook of Medical Surgical Nursing. Twelfth Edition. Wotters Kluwer Health/Lippincott Williams & Wilkins.