COURSE GUIDE

NSC 213
HUMAN ANATOMY II

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INTRODUCTION

Congratulation on the successful completion of the first-year courses. Welcome to the second-year courses. NSC 213 – Human Anatomy II. This is a second-year course and runs at the same time as first semester courses with Human Anatomy I (NSC 205). This part will cover the gross anatomy, embryology and histology of the kidney, ureter, urinary bladder and male and female urethra. The gross anatomy and clinical relevance of endocrine organs such as pituitary, thyroid, parathyroid, pancreas, gonads and adrenal glands. Caring for patients always requires a sound understanding of the normal structure of the body organs as to know what could be wrong and how such manifests. Basic assessments done before planning general and nursing care usually consider the various organs that function within systems and as interrelated systems. You will be required to be able to describe these organs and discuss their clinical correlates to the knowledge of the body parts. You will enjoy drawing and labelling, as well as seeing some of these organs in real life. You will also see the variations in normal and diseased organs as you are encouraged to participate in all laboratory assignments.

COURSE AIM

The aim of this course is to further your understanding of the structural makeup of two (2) of the life-supporting systems as such prepares you to apply your knowledge in planning to meet the care needs of your body and that of your clients as such may relate to normal and abnormal changes in the various organs that make up the systems.

COURSE OBJECTIVES

By the end of this course, you should be able to:

- discuss the structure and relations, in the urinary system
- explain embryology and histology of the organs in the endocrine system.

WORKING THROUGH THIS COURSE

The course will be delivered adopting the blended learning mode, 70% of online but interactive sessions and 30% of face-to-face during laboratory sessions. You are expected to register for this course online before you can have access to all the materials and have access to the class sessions online. You will have the hard and soft copies of course materials, you will also have online interactive sessions, face-to-face sessions 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 to have meaningful contributions to all sessions and to complete 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 MATERIALS

Course Guide
Course Text in Study Units
Textbooks (Hard and electronic)
Book of Laboratory Practical
Assignment File/Portfolio

STUDY UNITS

This course comprises 2 Modules and 10 units. They are structured as presented below:

Module 1 Urinary System

Unit 1 The Anatomy of the Kidneys
Unit 2 The Anatomy of the Ureters
Unit 3 The Anatomy of the Bladder
Unit 4 The Anatomy of the Urethra

Module 2 Endocrine System

Unit 1 Functions of the Endocrine System
Unit 2 Hormones
Unit 3 Pituitary Gland
Unit 4 Thyroid and Parathyroid Gland
Unit 5 Adrenal Gland
Unit 6 Pancreas

REFERENCE TEXTBOOKS

1. Sadler T.W (2004). *Langman's Medical Embryology*. (9th ed.).
2. Philip Tate (2012). *Seeley's Principles of Anatomy and Physiology* (2nd ed.).

3. Katherine M. A. Rogers & William N. S. (2011) *Nurses! Test yourself in anatomy and physiology*
4. Kent M. Van De Graff, R.Ward Rhees, Sidney Palmer (2010). *Schaum's Outline of Human Anatomy and Physiology*. (3rd ed.).
5. Kathryn A. B. & Terri. D. Wyman (2008). *Anatomy, Physiology, and Pathophysiology for Allied Health*
6. Keith, L. M. & Persuade T.V.N. (2006). *The Developing Human Clinically Oriented Embryology*. (8th ed.). Lippincott Williams & Wilkins.

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 logbook, submission of reports from all laboratory practical sessions and attendance of the final course examination. You are also expected to:

1. Be versatile in basic computer skills.
2. Participate in all laboratory practical up to 90% of the time
3. Submit personal reports from laboratory practical sessions on schedule.
4. Log in to the class online discussion board at least once a week and contribute to ongoing discussions.
5. Contribute actively to group seminar presentations.

EQUIPMENT AND SOFTWARE NEEDED TO ACCESS COURSE

You will be expected to have the following tools:

1. A computer (laptop or desktop or a tablet)
2. Internet access, preferably broadband rather than dial-up access
3. MS Office software – Word PROCESSOR, Powerpoint, Spreadsheet
4. Browser – Preferably Internet Explorer, Mozilla 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 as will be moderated by your facilitator. Your participation links 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 is the series of activities, assignments and end of the 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:**

The in-course examination will come up in the middle of the semester. These would come in form of a Computer Marked Assignment. This will be in addition to one compulsory Tutor Marked Assignment (TMA‘s) and three Computer Marked Assignment that comes after the modules.

- **Laboratory practical**

Attendance, records of participation and other assignments will be graded and added to the other scores from 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%	30%
Discussion Topic participation	5%	
Laboratory practical	10%	
End of Course examination	70%	

GRADING SCALE

A = 70-100
B = 60 - 69
C= 50 - 59
F = < 49

SCHEDULE OF ASSIGNMENTS WITH DATES

Every Unit has an activity that must be done by you as spelt out in your course materials. In addition to this, a specific assignment will also be provided for each module by the facilitator.

SPECIFIC READING ASSIGNMENTS

To be provided in each module.

COURSE OVERVIEW

Human Anatomy (II)

Human Anatomy (II) is the second of the three courses that cover some of the major organs that are responsible for life. In this course, two systems that are responsible for the maintenance of the body will be covered. The structures and locations of the various organs that make each of the systems will be studied. These are the urinary and endocrine systems. The course has theory and laboratory components 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 learning objectives that help 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 the venue, date and title of the practical session.

HOW TO GET THE MOST FROM THIS COURSE

1. Read and understand the context of this course by reading through this course guide paying attention to details. You must know the requirements before you will do well.
2. Develop a study plan for yourself.
3. Follow instructions about registration and master expectations in terms of reading, participation in the discussion forum, end of unit and module assignments, laboratory practical and other directives given by the course coordinator, facilitators and tutors.
4. Read your course texts and other reference textbooks.
5. Listen to audio files, watch the video clips and consult websites when given.
6. Participate actively in the online discussion forum and make sure you are in touch with your study group and your course coordinator.
7. Submit your assignments as at when due.
8. Work ahead of the interactive sessions.
9. Work through your assignments when returned to you and do not wait until when the examination is approaching before resolving any challenge you have with any unit or any topic.
10. Keep in touch with your study centre, the NOUN, School of Health Sciences websites as information will be provided continuously on these sites.
11. Be optimistic about doing well.

MAIN COURSE

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MODULE 1 URINARY SYSTEM

INTRODUCTION

The urinary system consists of the paired kidneys and ureters and the unpaired bladder and urethra. This system contributes to the maintenance of homeostasis by a complex process that involves **filtration, active absorption, passive absorption,** and **secretion.** The result is the production of urine, in which various metabolic waste products are eliminated.

Unit 1 The Anatomy of the Kidneys
Unit 2 The Anatomy of the Ureters
Unit 3 The Anatomy of the Bladder
Unit 4 The Anatomy of the Urethra

UNIT 1 THE ANATOMY OF THE KIDNEYS

CONTENTS

1.0 INTRODUCTION

The kidneys make up the body‘s main purification system. They control the composition of blood by removing waste products, many of which are toxic, and conserving useful substances. The kidneys help control blood volume and consequently play a role in regulating blood pressure. The kidneys also play an essential role in regulating blood pH. Approximately one-third of one kidney is all that is needed to maintain homeostasis. Even after extensive damage, the kidneys can still perform their life-sustaining functions. If the kidneys are damaged further, however, death results unless specialised medical treatment is administered.

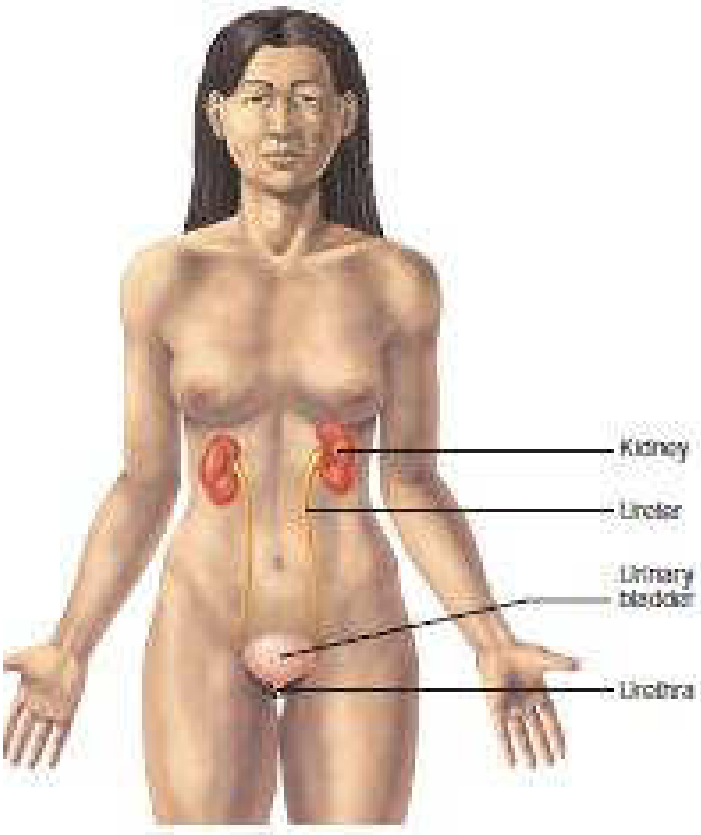


Fig. 1.1: The Urinary System

2.0 LEARNING OBJECTIVES

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

- discuss the functions of the kidneys
- describe the embryology of the kidneys
- describe the anatomy of the kidneys
- explain some clinical conditions related to the kidneys.

3.0 MAIN CONTENT

3.1 Developmental Anatomy of the Kidneys

Knowledge of the development of the urinary tract will enable you to understand how abnormalities can easily occur while the foetus is growing and why young babies have difficulties with fluid challenges. The system develops from the intermediate mesoderm on either side of the dorsal (back) body wall, which gives rise to three successive nephric structures (filtering units) of increasingly advanced design. The kidney changes three times before it is completed! The first kidneys are transitory, non-functional segmental nephrotomes in the cranial region which regress in the fourth week on day 24 to 25. After this, an elongated pair of mesonephros appear in the thoracic and lumbar region on either side of the vertebral column. These structures are functional, as they have complete nephrons and drain caudally via the Wolffian ducts to the urogenital sinus.

By week five the ureteric buds sprout from the Wolffian ducts and develop into the definitive kidneys that will serve the child for life. The bladder expands from the superior urogenic sinus and the inferior section gives rise to the urethra in both sexes. Ureters are then emplaced on the bladder wall. This articulation can give rise to multiple ureters forming or joining with the bladder ineffectively. At week six, germ cells migrating from the yolk sac induce the mesonephros to differentiate into Sertoli cells in the male and follicle cells in the female. At the same time, a new Müllerian duct develops parallel to the mesonephric duct. It is in week six, when the Y chromosome exerts its effect, that a development cascade then sees the forming of the male or female external genitalia and the kidneys ascending to their lumbar site in the abdomen, the right being lower than the left due to the presence of the liver.

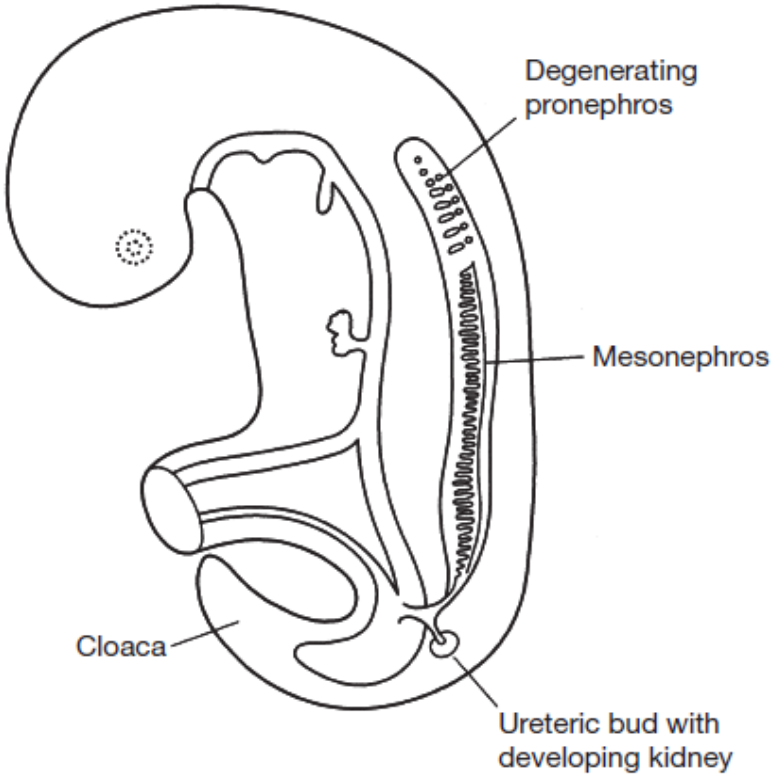


Fig. 1.2: The Kidney Bud Position

By the tenth week, the foetal kidney is functional and commences urine production. Foetal urine is important, not to get rid of waste products from the blood as the placenta regulates fluid and electrolyte homeostasis, but to supplement the production of amniotic fluid. Amniotic fluid is vital to foetal development as it contains proteins, carbohydrates, lipids and phospholipids, urea and electrolytes. It is a clear slightly yellow liquid around the foetus and increases during the pregnancy to 800 ml at thirty-four weeks. It is constantly circulated by the foetus as it swallows and "inhales" the fluid, replacing it with "exhalation" and urination. The amniotic fluid protects the foetus by cushioning it from outside crushing, allows it to move and develop its muscular-skeletal system, keeps it at an even temperature and allows the lungs and gut to mature.

The kidney and urine production at birth

The neonate has an immature kidney function at birth which makes it vulnerable to water loss and fluid gain, such as losing fluid through rapid breathing or failure to feed. The neonate‘s kidneys weigh about 23 g but have their full complement of filtering units (nephrons); this weight will double in six months and treble by the end of the first year eventually growing to its adult size by puberty which shows a ten-fold increase from birth. The growth of the kidney depends on its work; if one kidney is removed the other will double in size and take on the function of both. When the infant is born the loss of placenta flow, followed by a rapid increase in the infant‘s own renal blood flow, causes a high vascular resistance in the neonate's kidney. This results in a temporarily reduced renal blood flow and filtration through the filtering units to produce urine; however, as the infant starts to feed and the load presented to the kidney increases, 95 per cent of infants will pass urine in the first twenty-four hours after birth. The neonate will pass 20–35 ml of urine four times a day while the intake is low and milk production establishes in the mother, but this soon rises to 100–200 ml ten times a day by the tenth day of life. The urine that is first produced shows reduced urea excretion because of the overall tissue growth rate in the infant that uses the protein rather than allowing it to be broken down in the liver.

Also, in the first few days, urea is deposited in the kidney medulla to create the concentration gradient for the Loop of Henle function in adjusting water and sodium in the blood. Growth is thus sometimes referred to as the _third kidney‘. The kidney capillary network resistance reduces over the first few weeks of life, which allows increasing filtration ability by the glomeruli, however, the newborn kidney glomeruli capsules are formed of cuboid epithelium and are not

fully replaced by thin pavement epithelium and are fully functional until after the first year.

3.2 The Gross Anatomy of the Kidneys

The kidneys excrete the end products of metabolism and excess water (regulate the fluid and electrolyte balance of the body). The kidneys also have endocrine functions producing and releasing erythropoietin which affects red blood cell formation, renin which influences blood pressure, 1,25-dihydroxycholecalciferol, which is involved in the control of calcium metabolism.

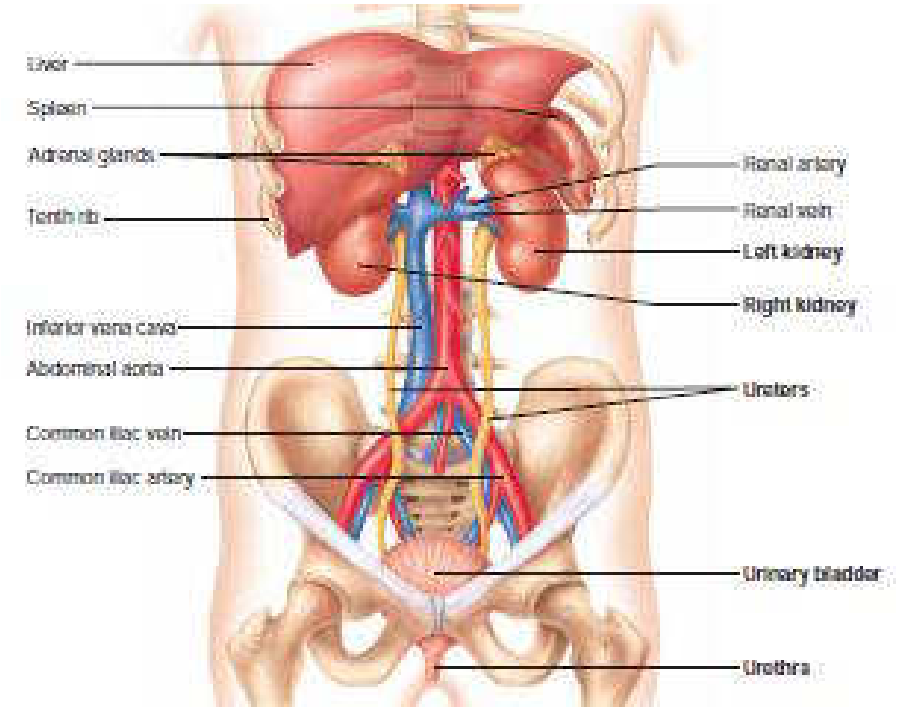


Fig. 1.3: Gross Anatomy of the Kidneys

The two kidneys are reddish-brown, bean-shaped organs. They lie retroperitoneally on the posterior abdominal wall, within the paravertebral gutters resting on the muscles of the posterior abdominal wall. They are largely under cover of the costal margin. They lie craniocaudally at the level of the T12 - L3 vertebrae. The hilum is about 5 cm from the midline at the level of L1.

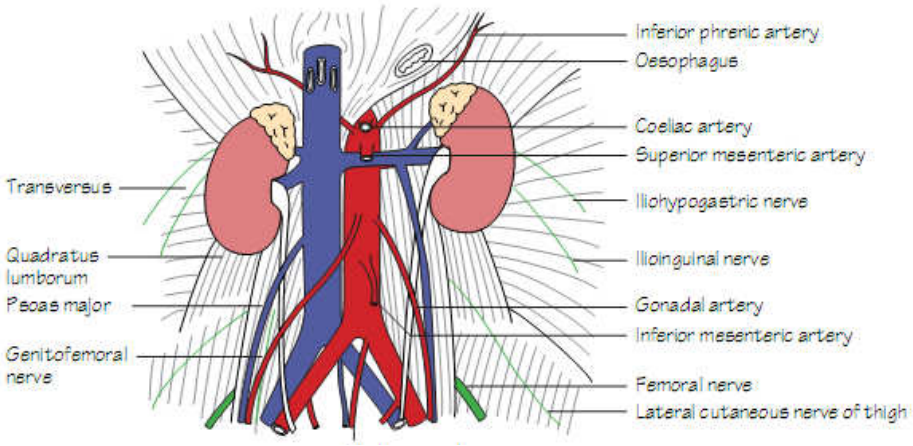


Fig.1.4: The Kidneys

Dimension and Weight

A normal kidney measures approximately 10 - 12 cm in length, 5 - 6 cm in width, and 2.5 - 3 cm in thickness (AP dimension). The average weight in adult males is about 150 g while in females it is about 135g

External Topography

Each kidney has 2 borders (medial and lateral) 2 surfaces (anterior and posterior) and 2 poles (superior and inferior).
The upper pole of the right kidney usually lies slightly (about 2.5 cm) inferior to that of the left kidney, probably owing to its relationship to the bulk of the right lobe of the liver.

Relations

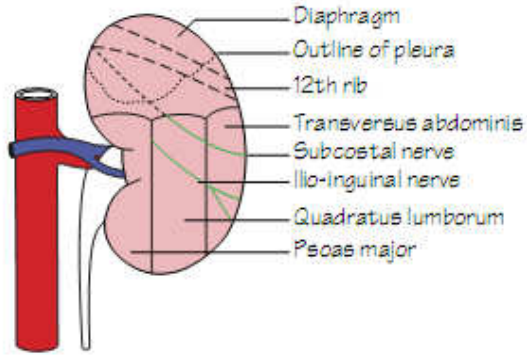
Relations of the kidney could be described in respect of its anterior and posterior surfaces

Posterior Relations

Posteriorly, the right and left kidneys are related to similar structures with a little exception. They include: the diaphragm, psoas major, quadratuslumborum, aponeurosis of the transversus abdominis muscles, 12th rib, transverse process of L1 vertebra, subcostal vessels (artery and vein), Subcostal iliohypogastric and ilioinguinal nerves. The posterior surface of the left kidney is related to the eleventh rib.

Anterior Relations of the right and left kidney Right

Right	Left
right suprarenal gland • liver and is separated from it by hepatorenal recess • the descending part of the duodenum • right colic flexure	left suprarenal gland • stomach • spleen • body and tail of the pancreas • left colic flexure and the beginning of the descending colon, • Proximal parts of the jejunum.



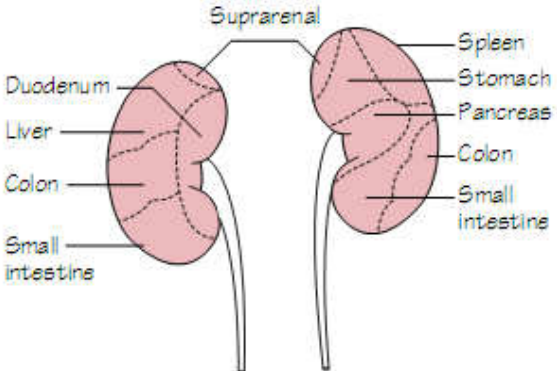


Fig. 1.5a: Posterior Relation of the Kidney Fig.1.5b: Anterior Relation of the right and left Kidney

Renal hilum and renal pelvis

The renal hilum is a deep vertical opening on the medial margin of each kidney through which renal vessels, lymphatics, nerves and ureter enter

and leave the substance of the kidney. Internally, the hilum is continuous with the renal sinus. Perinephric fat continues into the hilum and sinus and surrounds all structures.

The Renal pelvis is the funnel-shaped commencement of the ureter. It is normally the most posterior of the three main structures in the hilum. The capacity of the average pelvis is less than 5 mL.

Arterial supply: the arterial supply to the kidneys is from the renal arteries which arise as a lateral branch of the abdominal aorta at the level of L2. Each renal artery divides into five *segmental arteries* at the hilum which, in turn, divide sequentially into *lobar*, *interlobar*, *arcuate* and *cortical radial branches*. The cortical radial branches give rise to the afferent arterioles which supply the glomeruli and go on to become efferent arterioles. The differential pressures between afferent and efferent arterioles lead to the production of an ultrafiltrate which then passes through, and is modified by, the nephron to produce urine.
Veinous Drainage: The kidneys are drained by the right and left renal veins. Each renal vein drains into the IVC.

Lymphatic Drainage is to the para-aortic lymph nodes.

Innervation: The innervations of the kidney is from the renal plexus which have sympathetic and parasympathetic parts. The sympathetic contribution is from the least splanchnic nerve and lumbar splanchnic nerve while the parasympathetic supply is from the vagal trunk.

The general structure of the kidneys

Each kidney consists of an outer renal cortex and an inner renal medulla. The renal cortex lies beneath the renal capsule. It is a continuous band of pale tissue that completely surrounds the renal medulla. Extensions of the renal cortex called the renal columns (of Bertin) project into the inner aspect of the kidney; they divide the renal medulla into discontinuous aggregations of striated triangular-shaped tissue called the renal pyramids. The bases of the renal pyramids are directed outward, towards the renal cortex, while the apex of each renal pyramid projects inward, towards the renal sinus as renal papilla. The renal papilla is surrounded by a minor calyx. The minor calices receive urine and represent the proximal parts of the tube that will eventually form the ureter. In the renal sinus, several minor calices unite to form a major calyx, and two or three major calices unite to form the renal pelvis, which is the funnel-shaped superior end of the ureters.

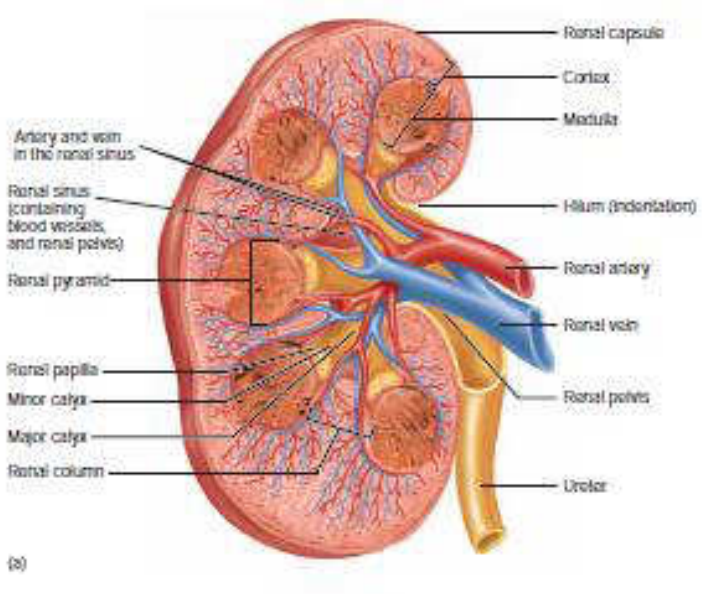


Fig. 1.6: The general structure of the kidney

4.0 CONCLUSION

The kidneys make up the body‘s main purification system. They control the composition of blood by removing waste products, many of which are toxic, and conserving useful substances. The kidneys help control blood volume and consequently play a role in regulating blood pressure. The kidneys also play an essential role in regulating blood pH.

5.0 SUMMARY

In this unit, you have learnt that:

i. The urinary system consists of the paired kidneys and ureters and the unpaired bladder and urethra.
ii. The functions of the urinary system include excretion, regulation of blood volume and pressure, regulation of concentration of solutes in the blood, vitamin D synthesis and regulation of red blood cell synthesis.
iii. The anatomy and histology of the kidneys
iv. Clinical conditions associated with kidney malfunctions.

Clinical correlates

Kidney Stones

i. **Kidney stones** are hard objects usually found in the renal pelvis of the kidney. They are normally 2–3 mm in diameter, with a smooth or a jagged surface. About 1% of all autopsies reveal

kidney stones, and many of the stones occur without causing symptoms. The symptoms associated with kidney stones occur when a stone passes into the ureter, resulting in intense referred pain down the back, side, and groin area. The ureter contracts around the stone, causing the stone to irritate the epithelium and produce bleeding. Kidney stones can also block the ureter, cause ulceration in the ureter, and increase the probability of bacterial infections. About 65% of all kidney stones are composed of calcium oxylate mixed with calcium phosphate, 15% are magnesium ammonium phosphate, and 10% are uric acid or cystine. The cause of kidney stones is usually obscure. Predisposing conditions include concentrated urine and an abnormally high calcium concentration in the urine, although the cause of the high calcium concentration is usually unknown.

ii. **Renal failure** can result from any condition that interferes with kidney function.

Acute renal failure occurs when kidney damage is extensive and leads to the accumulation of urea in the blood and to acidosis. In complete renal failure, death can occur in 1–2 weeks. Acute renal failure can result from acute glomerular nephritis, or it can be caused by damage to or blockage of the renal tubules. Some poisons, such as mercuric ions or carbon tetrachloride, which are common to certain industrial processes, cause necrosis of the nephron epithelium. If the damage does not interrupt the basement membrane surrounding the nephrons, extensive regeneration can occur within 2–3 weeks. Severe ischemia associated with circulatory shock resulting from sympathetic vasoconstriction of the renal blood vessels can cause necrosis of the epithelial cells of the nephron.

Chronic renal failure results when so many nephrons are permanently damaged that the nephrons that remain functional cannot adequately compensate.

iii. **Diabetic nephropathy** is a disease of the kidney associated with diabetes mellitus, and it is the principal cause of chronic renal failure. It damages renal glomeruli and ultimately results in the destruction of functional nephrons through progressive scar tissue formation, which is mediated in part by an inflammatory response.

iv. Other causes of renal failure can include: chronic glomerular nephritis, trauma to the kidneys, the absence of kidney tissue caused by congenital abnormalities, tumours, urinary tract

obstruction by kidney stones, damage resulting from pyelonephritis (inflammation of the renal pelvis).

6.0 TUTOR-MARKED ASSIGNMENT

6.1 Activity

Some patients with hypertension are kept on a low-salt (low-sodium) diet. Propose an explanation for this therapy.

6.2 Please answer the following questions:

1. Describe the functions of the kidneys
2. Describe the embryology of the kidneys
3. Describe the anatomy of the kidneys
4. Understand some clinical conditions related to the kidneys

7.0 REFERENCES/FURTHER READING

Hutchinson, M., Mallat, J., Marieb, E.N. & Wilhelm P.B. (2007). *A Brief Atlas of the Human Body*. San Franscisco: Pearson Education Inc.

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UNIT 2 ANATOMY OF URETER

CONTENTS

1.0 INTRODUCTION

The ureters are muscular tubes whose peristaltic contractions convey urine from the kidneys to the urinary bladder. Each measures 25 cm in length and comprise the renal pelvis, abdominal, pelvic and intravesical portions. Its luminal diameter is 3 mm but is slightly less at three areas of constriction including: the pelvic-ureteric (**uretero-pelvic**) junction, where it crosses the common iliac vessels at the pelvic brim and where it runs within the wall of the urinary bladder, which is its narrowest part.

2.0 OBJECTIVES

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

- describe the course of the ureters
- explain in detail the relations of the ureters
- discuss the vasculature of the ureters.

3.0 MAIN CONTENT

3.1 Gross Anatomy of the Ureters

Each descends slightly medially anterior to the psoas major and enters the pelvic cavity where it curves laterally, then medially as it runs down to open into the base of the urinary bladder.

Relations

Abdominal ureter

In the abdomen, both ureters courses anterior to the psoas major, the tip of lumbar process, genitofemoral nerve and posterior to gonadal (testicular and ovarian) vessels. The right ureter courses lateral to the inferior vena cava and posterior to the descending part of the duodenum, right colic and ileocolic vessels, the lower part of the mesentery and terminal ileum. The left ureter courses posterior to the left colic vessels, loops of jejunum, sigmoid colon and mesentery, medial to the aorta and lateral to the inferior mesenteric vessels. At the pelvic brim the ureter courses anterior to the bifurcation of the common iliac artery and the sacroiliac joint

Pelvic ureter

The ureter in both sexes runs anterior to the lateral wall of the lesser pelvis, internal iliac artery (IIA), the commencement of anterior trunk of IIA and anteromedial to the umbilical artery, inferior vesical artery and middle rectal artery. In males the ureter courses inferior to the vas deferens and anterosuperior to the seminal vesicle; while in females it courses posterior to the ovary, posteroinferior (and later lateral) to the uterine artery, lateral uterus and then anterior vagina

3.2 Vasculature of the Ureters

Arterial supply

The ureters receive arterial branches from adjacent vessels as they pass towards the bladder. These vessels include ureteric branches of the renal arteries; abdominal aorta, the testicular or ovarian arteries, and the common iliac arteries; the internal iliac arteries (i.e. superior vesical and uterine arteries). In all cases, arteries reaching the ureters divide into ascending and descending branches, which form longitudinal anastomoses.

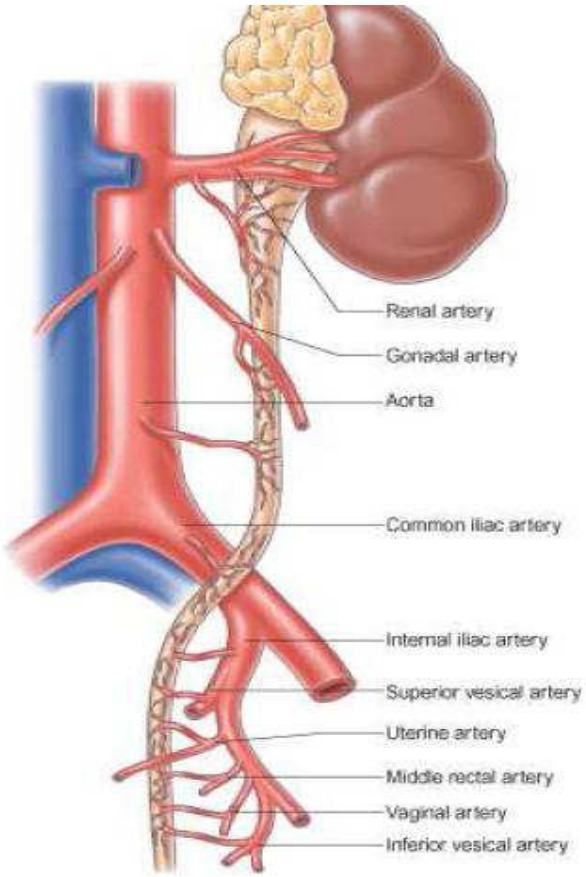

Fig. 2.1:

Venous drainage

Veins draining the abdominal part drain into the renal and gonadal (testicular or ovarian) veins. Veins draining the pelvic part drain into the internal iliac veins

Lymphatic drainage

Lymphatic drainage of the ureters follows a pattern similar to that of the arterial supply. Lymph from the upper part of each ureter drains to the lumbar nodes; those from the middle part of each ureter drains to lymph nodes associated with the common iliac vessels; while lymph from the inferior part of each ureter drains to lymph nodes associated with the external and internal iliac vessels.

Innervation

The innervations are autonomic having sympathetic and parasympathetic contributions from T10 – L1 segment of the spinal cord and pelvic splanchnic nerve (S2 – S4) respectively.

3.3 Histology of the Ureters

The wall of the ureter is composed of an external adventitia, a smooth muscle layer and an inner mucosal layer. The mucosal layer consists of the urothelium (transitional epithelium) and an underlying connective tissue lamina propria. It has no muscularis mucosae. The muscle bundles are so arranged that morphologically distinct longitudinal and circular layers cannot be clearly distinguished.

5.0 SUMMARY

In this unit, you have learnt that:
i. The ureters are muscular tubes whose peristaltic contractions convey urine from the kidneys to the urinary bladder. Each measures 25 cm in length and comprise the renal pelvis, abdominal, pelvic and intravesical portions.
ii. The ureters receive arterial branches from adjacent vessels as they pass towards the bladder.
iii. Lymphatic drainage of the ureters follows a pattern similar to that of the arterial supply
iv. The venous drainage depends on the part of the ureter been drained.

Clinical correlates

Ureteric stones:

Ureteric stones are kidney stones that gradually move down the urinary system from the kidneys to the bladder.

6.0 TUTOR-MARKED ASSIGNMENT

1. Describe the course of the ureters.
2. Eplain in detail the relations of the ureters.
3. Discuss the vasculature of the ureters.
4. Describe the phenomenon ―water under the bridge.

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UNIT 3 URINARY BLADDER

CONTENTS

1.0 INTRODUCTION

Infants are expected to be incontinent, but the ability to control voiding of urine depends on a complete and functioning renal system, maturation of the nervous supply, opportunity/support given to the child to void and cultural expectations. Children can become anxious and regress if expectations are beyond their ability and control. The maturation of control mechanisms usually takes up to five years for healthy children to be dry in the day and overnight. The urinary bladder is a complex organ made of specialised muscle layers and enervated by a reflex arc to the spine and central coordination in the brain. Remember that if the child does not want to void, for whatever reason, they can override the messages to their brain from their distending bladder.

2.0 OBJECTIVES

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

- discuss the gross anatomy of the urinary bladder
- explain vasculature of the urinary bladder
- examine histology of the urinary bladder.

3.0 MAIN CONTENT

3.1 Gross Anatomy of the Urinary Bladder

The urinary bladder is a hollow viscus with a strong muscular wall characterised by its distensibility. It is a temporary reservoir for urine. It varies in size, shape, position and relations, according to its content and the state of neighbouring viscera. When empty, the adult urinary bladder lies entirely in the lesser pelvis posterior to the pubic bones but as it

distends it expands anterosuperiorly into the abdominal cavity and may ascend to the level of the umbilicus when fully distended. In infants and children, the urinary bladder lies in the abdomen even when empty. The bladder usually enters the greater pelvis by 6 years of age; It enters the lesser pelvis after puberty. When empty, it is pyramidal in shape and has an apex, a base, two inferolateral surfaces, a superior surface and the neck as shown in the diagram below.

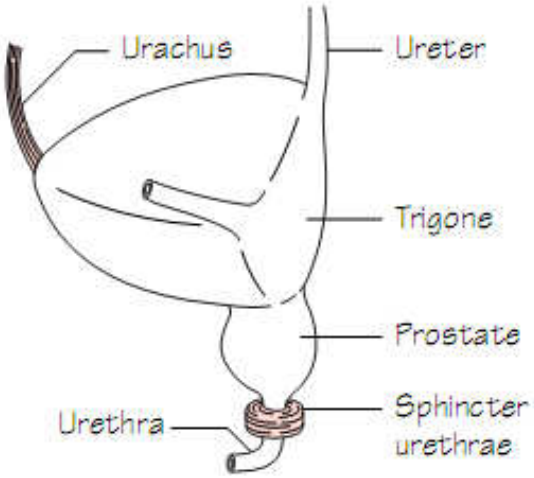

Fig. 3.1: Urinary Bladder

The ***apex*** of the pyramid points forwards and from it, a fibrous cord, the *urachus*, passes upwards to the umbilicus as the *median umbilical ligament*. The ***base*** **(posterior surface, fundus)** is triangular. In the male, the seminal vesicles lie on the outer posterior surface of the bladder and are separated by the vas deferens. The rectum lies behind the seminal vesicle. In the female, the vagina intervenes between the bladder and rectum.

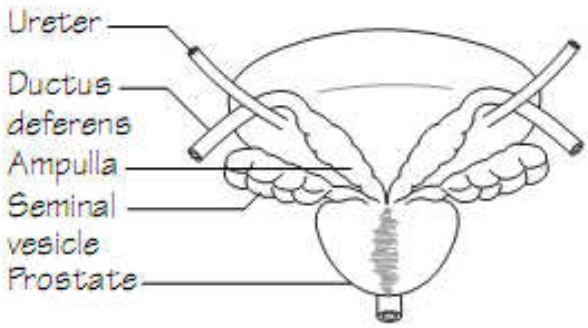

Fig. 3.2: Inferolateral Surfaces

The ***inferolateral*** **surfaces** are related inferiorly to the pelvic floor and anteriorly to the retropubic fat pad and pubic bones.

The **superior surface** is triangular. In males it is completely covered by the peritoneum which posteriorly reflects on to the rectum as the rectovesical pouch while in females, it is largely covered by the peritoneum, which is reflected posteriorly onto the uterus at the level of the internal os to form the vesicouterine pouch.

The **bladder *neck*** fuses with the prostate in the male whereas it lies directly on the pelvic fascia (which surrounds the upper urethra) in the female. The pelvic fascia is thickened in the form of the *puboprostatic ligaments* (male) and *pubovesical ligaments* to hold the bladder neck in position.

Bladder Interior

The mucous membrane of the bladder is thrown into folds called rugae when the bladder is empty with the exception of the membrane overlying the *trigone* which is smooth.

Trigone is a triangular area on the interior of the base of the bladder. The superior angles of the trigone mark the openings of the ureteric orifices while its inferior angle corresponds to the *internal urethral meatus*.

3.2 Vasculature of the Urinary Bladder

Arterial supply: superior and inferior vesical arteries (branches of the internal iliac artery.

Veinous drainage: The vesical veins coalesce around the bladder to form a plexus that drains into the internal iliac vein.

Lymph drainage: lymphatic vessels from the superolateral aspects of the bladder pass to the external iliac lymph nodes whereas those from the fundus and neck pass to the internal iliac lymph nodes.

Nerve supply (Vesical Plexus): Sympathetic fibres are conveyed from inferior thoracic and upper lumbar spinal cord levels to the vesical plexuses primarily through the hypogastric plexuses and nerves, parasympathetic fibres from sacral spinal cord levels are conveyed by the pelvic splanchnic nerves and the inferior hypogastric plexus. Motor input to the detrusor muscle is from efferent parasympathetic fibres from S2–4. Fibres from the same source convey inhibitory fibres to the internal sphincter so that coordinated micturition can occur. Conversely, sympathetic efferent fibres inhibit the detrusor and stimulate the sphincter.

Histology of the urinary bladder

The wall of the urinary bladder consists of three layers: an outer adventitial layer of soft connective tissue (which in some regions possesses a serosal covering of peritoneum); a smooth muscle coat composed of a triple layer of trabeculated smooth muscle known as the *detrusor* muscle; and an inner mucosal layer which lines the interior of the bladder with transitional epithelium. The detrusor is thickened at the bladder neck to form the internal urethra sphincter.

5.0 SUMMARY

In this unit, you have learnt that:

- The urinary bladder is a muscular organ that serves as a reservoir for the urine and the interior is made up of mucous membrane thrown into folds called rugae when the bladder is empty with the exception of the membrane overlying the *trigone* which is smooth.
- The Arterial supply to the bladder is the superior and inferior vesical arteries (branches of the internal iliac artery).
- The veinous drainage includes The vesical veins that coalesce around the bladder to form a plexus that drains into the internal iliac vein.
- Lymph drainage includes the lymphatic vessels from the superolateral aspects of the bladder pass to the external iliac lymph nodes whereas those from the fundus and neck pass to the internal iliac lymph nodes.

Clinical correlates

Urinary Bladder Cancer

In the United States, urinary bladder cancer affects more than 60,000 new patients each year and is among the 10 most common cancers in men and women. Half the diagnosed cases of urinary bladder cancer can be attributed to cigarette smoking, even 10 years or more after cessation of smoking. When bladder cancer is detected early (the cancer is confined to the bladder), the survival rate is 94%, whereas, if it is detected late (after it has spread to other areas), the survival rate is 6%. Unfortunately, early detection of urinary bladder cancer is especially challenging due to its rapid growth rate. Frequently, blood in the urine is a symptom but, because this symptom is also associated with other, less serious problems, it tends to be ignored. What is the statistics of bladder cancer in Nigeria?

6.0 TUTOR-MARKED ASSIGNMENT

1. Discuss the Gross anatomy of the urinary bladder.
2. Explain Vasculature of the urinary bladder.
3. Explain Histology of the urinary bladder.
4. Describe the concept of urinary incompetence.

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UNIT 4 THE URETHRAS

CONTENTS

1.0 INTRODUCTION

The **urethra** is a tube that exits the urinary bladder inferiorly and anteriorly. It carries urine to the outside of the body. In males, the urethra extends to the end of the penis. In females, it opens into the vestibule anterior to the vaginal opening. The female urethra is approximately 4 cm in length, whereas the male urethra is approximately 20 cm.

2.0 OBJECTIVES

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

- discuss the anatomy and the functions of the urethra
- explain the differences between the male and female urethra.

3.0 MAIN CONTENT

3.1 Anatomy of the Male Urethra

The male urethra is approximately 20 cm long. It is considered in three parts:

Prostatic urethra (3 cm): bears a longitudinal elevation (*urethral crest*) on its posterior wall. On either side of the crest a shallow depression, the *prostatic sinus*, marks the drainage point for 15–20 prostatic ducts. The *prostatic utricle* is a 5 mm blind-ending tract which opens into eminence in the middle of the crest at the *verumontanum*. The ejaculatory ducts open on either side of the utricle.

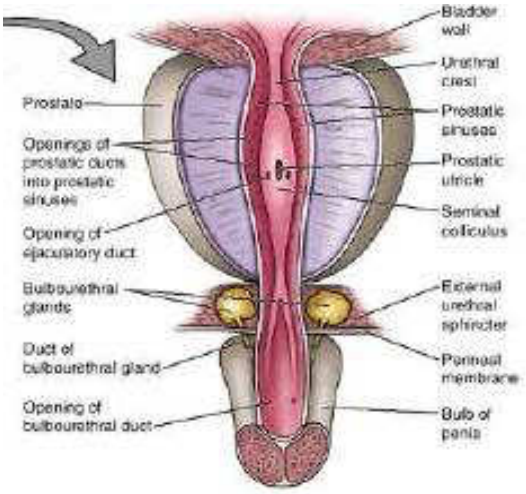

Fig. 4.1:

Membranous urethra (2 cm): lies in the urogenital diaphragm and is surrounded by the external urethral sphincter (*sphincter urethrae*).

Penile urethra (15 cm): traverses the corpus spongiosum of the penis to the external urethral meatus.

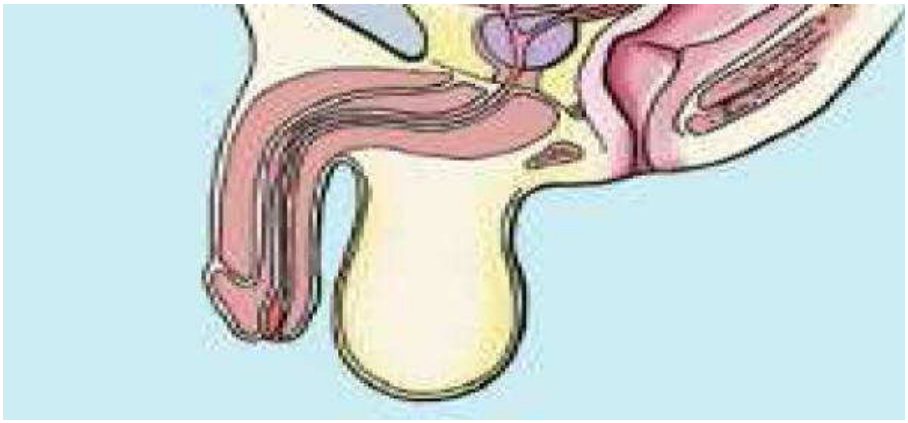

Fig.4.2:

Vasculature of the male urethra

Arterial supply:

The blood supply is from the vessels of the prostate, sphincter urethra and the corpus spongiosum as it passes through them. They include branches of the inferior vesical artery, middle rectal artery and internal pudendal artery.

Innervation:

The mucous membrane of the penile part receives a branch from the perineal nerve, while the more proximal parts are innervated by the inferior hypogastric plexus having a sympathetic contribution from the sacral sympathetic trunk and parasympathetic fibres from the pelvic splanchnic nerve S2-S4.

Lymphatics

Lymphatic drainage is into the internal and external iliac lymph nodes.

3.2 Anatomy of the Female Urethra

The female urethra is a narrow membranous canal, about 4cm long, extending from the internal urethra orifice at the lower angle of the trigone of the bladder to the external urethra orifice. It is placed behind the symphysis pubis, embedded in the anterior wall of the vagina and its direction is obliquely downward and forward. Its diameter when undilated is about 6mm. It perforates the fascia of the urogenital diaphragm and its external orifice is situated directly in front of the vagina opening and about 2.5cm behind the clitoris.

Vasculature of the female urethra

Blood Supply

The upper part of the female urethra is supplied by the vagina arteries while the lower end receives contribution from the internal pudendal artery.

The veins drain into the vesical plexus and the internal pudendal veins.

Lymphatics

Lymph vessels pass mainly into the internal iliac lymph nodes but some reach the external iliac groups of nodes.

Nerve Supply

The nerve supply is from the inferior hypogastric plexus and the perineal branch of the pudendal nerve.

4.3 Histology of the Male and Female Urethra

The urethra is composed of the mucous membrane, supported by sub-mucous tissue which connects it with the various structures through which it passes.

The mucous membrane is lined by transitional epithelium (typical of the urinary tract) except at the navicular fossa (in males) and external urethra orifice where the mucosa is lined by a non-keratinised stratified squamous epithelium. The urethra mucosa has numerous mucous urethra glands (of Littre).

5.0 SUMMARY

In this unit, you have learnt that:

- The **urethra** as a tube that exits the urinary bladder inferiorly and anteriorly. In males, the urethra extends to the end of the penis. In females, it opens into the vestibule anterior to the vaginal opening.
- The male urethra has 3 parts - prostatic, membranous and penile urethra.

6.0 TUTOR-MARKED ASSIGNMENT

1. Discuss the anatomy and the functions of the urethra
2. Describe with the aid of a diagram the difference between the male urethra and the female urethra.
3. From your experience what are the implications of the different structures of the urethra for males and females in clinical care?

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MODULE 2 THE ENDOCRINE SYSTEM

UNIT 1 HORMONES

CONTENTS

1.0 INTRODUCTION

The **endocrine system** is composed of **endocrine glands,** which are ductless glands secreting chemical messengers into the circulatory system. In contrast, exocrine glands have ducts that carry their secretions to surfaces. The term *endocrine* is derived from the Greek words *endo*, meaning within, and *krin*o, to separate. The term implies that cells of endocrine glands produce chemical messengers within the glands that influence tissues separated from the glands by some distance. The chemical messengers secreted by endocrine glands are called hormones, a term derived from the Greek word *hormon,* meaning to set into motion. Thus, hormones stimulate responses from cells.

2.0 OBJECTIVES

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

- describe the functions of the endocrine system
- define the terms endocrine gland and hormone
- explain how the regulation of hormone secretion is achieved

- describe how hormones are transported and excreted.

3.1 Functions of the Endocrine System

The main regulatory functions of the endocrine system are the following:

1. *Metabolism and tissue maturation.* The endocrine system regulates the rate of metabolism and influences the maturation of tissues, such as those of the nervous system.
2. *Ion regulation.* The endocrine system helps regulate blood pH, as well as Na+, K+, and Ca2+ concentrations in the blood.
3. *Water balance*. The endocrine system regulates water balance by controlling the solute concentration of the blood.
4. *Immune system regulation*. The endocrine system helps control the production of immune cells.
5. *Heart rate and blood pressure regulation.* The endocrine system helps regulate the heart rate and blood pressure and helps prepare the body for physical activity.
6. *Control of blood glucose and other nutrients.* The endocrine system regulates blood glucose levels and other nutrient levels in the blood.
7. *Control of reproductive functions.* The endocrine system controls the development and functions of the reproductive systems in males and females.
8. *Uterine contractions and milk release*. The endocrine system regulates uterine contractions during delivery and stimulates milk release from the breasts in lactating females.

3.2 Transport of Hormones in the Blood

Hormones can be defined as chemicals secreted by a cell that affect the functions of other cells. Once released, most hormones enter the bloodstream where they are carried to their target cells. The target cells of a hormone are the cells that contain the receptors for the hormone. A hormone cannot affect a cell unless the cell has receptors for it. Many hormones in the body are derived from steroids. Steroids are soluble in lipids and can therefore cross cell membranes very easily. Once a **steroid hormone** is inside a cell, it binds to its receptor, which is common in the nucleus of the cell. The hormone-receptor complex turns a gene on or off. When new genes are turned on or off, the cell begins to carry out new functions, and this is ultimately how steroid hormones affect their target cells. Examples of steroid hormones are **estrogen, progesterone, testosterone,** and **cortisol. Non -steroid hormones** are those that are made of amino acids or proteins. Proteins cannot cross the cell membrane easily. Therefore, these hormones bind to receptors on

the surface of the cell. The hormone-receptor complex in the membrane usually activates a **G-protein.** The G-protein causes enzymes inside the cell to be turned on. Different chemical reactions then begin inside the cell. The cell now takes on new functions. **Prostaglandins** are local hormones. They are derived from lipid molecules and typically do not travel in the bloodstream to find their target cells. Instead, their target cells are located close by. They have the same effects as other hormones and are produced by many body organs, including the kidneys, stomach, uterus, heart, and brain.

Transport of Hormones in the Blood

Water-soluble hormones (peptides and catecholamines) are dissolved in the plasma and transported from their sites of synthesis to target tissues, where they diffuse out of the capillaries, into the interstitial fluid, and ultimately to target cells. *Steroid and thyroid hormones,* in contrast, circulate in the blood mainly bound to plasma proteins. Usually, less than 10 per cent of steroid or thyroid hormones in the plasma exist free in solution. For example, more than 99 per cent of the thyroxine in the blood is bound to plasma proteins. However, protein-bound hormones cannot easily diffuse across the capillaries and gain access to their target cells and are therefore biologically inactive until they dissociate from plasma proteins. The relatively large amounts of hormones bound to proteins serve as reservoirs, replenishing the concentration of free hormones when they are bound to target receptors or lost from the circulation. The binding of hormones to plasma proteins greatly slows their clearance from the plasma.

3.3 Interaction of Hormones with their Target Tissues

Hormones bind to proteins or glycoproteins called receptors. The portion of each protein or glycoprotein molecule where a hormone binds is called a receptor site, or binding site. The shape and chemical characteristics of each receptor site allow only a specific type of chemical messenger to bind to it. The tendency for each type of chemical messenger to bind to a specific type of receptor, and not to others, is called specificity. Insulin, therefore, binds to insulin receptors but not to receptors for growth hormone. Some hormones, however, can bind to a number of different receptors that are closely related. For example, epinephrine can bind to more than one type of epinephrine receptor. Hormone receptors have a high affinity for the hormones that bind to them, so only a small concentration of a given hormone results in a significant number of receptors with hormones bound to them. Hormones are secreted and distributed throughout the body by the circulatory system, but the presence or absence of specific receptor

molecules in cells determines which cells will or will not respond to each hormone.

For example, there are receptors for thyroid-stimulating hormone (TSH) in cells of the thyroid gland, but there are no such receptors in most other cells of the body. Consequently, cells of the thyroid gland produce a response when exposed to TSH, but cells without receptor molecules do not respond to it. In general, the number of functional receptors affects the amplitude of a cell's response to a hormone. More receptors produce a larger response than fewer receptors. The number of functional receptors can be regulated. In down-regulation, the number of functional receptors is reduced by temporary or permanent removal of receptors from the plasma membrane, inactivation of receptors, or decreased synthesis of replacement receptors. In up-regulation, the number of functional receptors is increased through increased receptor synthesis or availability. Drugs with structures similar to specific hormones may compete with those hormones for their receptors. A drug that binds to a hormone receptor and activates it is called an agonist for that hormone. A drug that binds to a hormone receptor and inhibits its action is called an antagonist for that hormone. For example, drugs exist that compete with epinephrine for its receptor. Epinephrine agonists activate epinephrine receptors, whereas epinephrine antagonists inhibit them.

3.4 Clinical Correlates

Lipid- and Water-Soluble Hormones in Medicine

Specific hormones are given as treatments for certain illnesses. Hormones that are soluble in lipids, such as steroids, can be taken orally because they can diffuse across the wall of the stomach and intestine into the circulatory system. Examples include the synthetic estrogen and progesterone-like hormones in birth control pills and steroids that reduce the severity of inflammation, such as prednisone. In contrast to lipid-soluble hormones, protein hormones cannot diffuse across the wall of the intestine because they are not lipid-soluble. Furthermore, protein hormones are not transported across the wall of the intestine because they are broken down to individual amino acids by the digestive system. The normal structure of a protein hormone is therefore destroyed, and its physiological activity is lost. Consequently, protein hormones must be injected rather than taken orally. The most commonly administered protein hormone is insulin, which is prescribed for the treatment of diabetes mellitus.

4.0 CONCLUSION

The **endocrine system** is composed of **endocrine glands,** which are ductless glands secreting chemical messengers into the circulatory system. In contrast, exocrine glands have ducts that carry their secretions to surfaces.

5.0 SUMMARY

- The main regulatory functions include water balance, uterine contractions and milk release, metabolism and tissue maturation, ion regulation, heart rate and blood pressure regulation, control of blood glucose and other nutrients, immune system regulation, and control of reproductive functions
- Endocrine glands produce hormones that are released into the interstitial fluid and diffuse into the blood. Hormones act on target tissues, producing specific responses. The protein group of hormones includes hormones that are proteins, glycoproteins, polypeptides, and amino acid derivatives. The lipid group of hormones includes hormones that are steroids and fatty acid derivatives.
- Generalisations about the differences between the endocrine and nervous systems include the following: (a) The endocrine system is amplitude-modulated, whereas the nervous system is frequency modulated, and (b) the response of target tissues to hormones is usually slower and of longer duration than their response to neurons.
- Water-soluble hormones, such as proteins, epinephrine, and norepinephrine, are rapidly removed from the blood. These hormones regulate activities that have a rapid onset and a short duration. Lipid-soluble hormones and thyroid hormones are not quickly removed from the blood. They produce a prolonged effect.

6.0 TUTOR-MARKED ASSIGNMENT

1. Which of these regulates the secretion of a hormone from an endocrine tissue?
   a. other hormones
   b. negative-feedback mechanisms
   c. nonhormone substance in the blood
   d. the nervous system
   e. all of the above

2. Hormones are released into the blood
   a. at relatively constant levels.
   b. in large amounts in response to a stimulus.
   c. in a cyclic fashion.
   d. all of the above.
3. Given these observations:
   1. A hormone affects only a specific tissue (not all tissues).
   2. A tissue can respond to more than one hormone.
   3. Some tissues respond rapidly to a hormone, whereas others take many hours to respond.

   Which of these observations can be explained by the characteristics of hormone receptors?
   a. 1
   b. 1,2
   c. 2,3
   d. 1,3
   e. 1,2,3
4. A hormone
   a. can function as an enzyme.
   b. is also a G protein.
   c. can bind to a receptor.
   d. is an intracellular mediator.
   e. all of the above.

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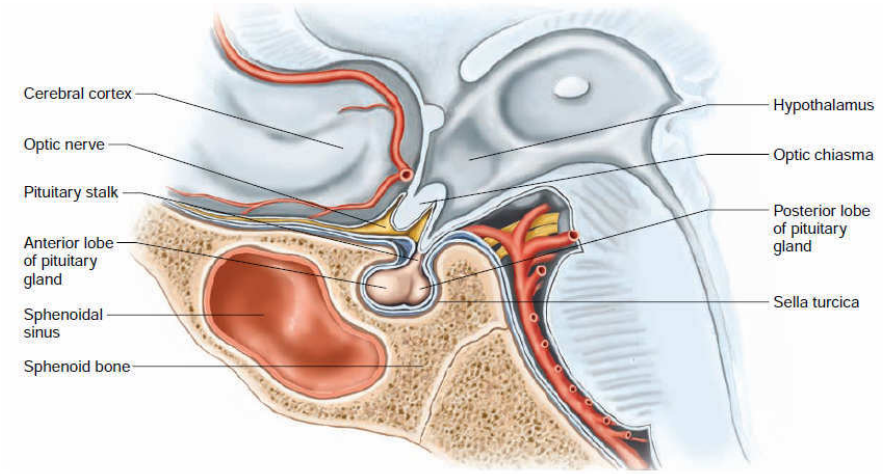
UNIT 2 THE PITUITARY GLAND AND HYPOTHALAMUS

CONTENTS

1.0 INTRODUCTION

The pituitary gland is located at the base of the brain and is controlled by the hypothalamus. This gland is well protected by a bony structure called the **sella turcica.** Just superior to the gland is the **optic chiasm,** which carries visual information to the brain for interpretation. The pituitary is divided into two lobes—the anterior and the posterior.
The location of the pituitary gland



The **pituitary gland,** or **hypophysis,** secretes nine major hormones that regulate numerous body functions and the secretory activity of several other endocrine glands. The **hypothalamus** of the brain and the pituitary gland are major sites where the nervous and endocrine systems interact. The hypothalamus regulates the secretory activity of the pituitary gland. Hormones, sensory information that enters the central nervous system, and emotions, in turn, influence the activity of the hypothalamus.

2.0 OBJECTIVES

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

- describe the structure of the pituitary gland
- discuss the hormones of the pituitary gland

3.1 Structure of the pituitary gland

The pituitary gland is roughly 1 cm in diameter, weighs 0.5–1.0 g, and rests in the sella turcica of the sphenoid bone. It is located inferior to the hypothalamus and is connected to it by a stalk of tissue called the **infundibulum.** The **posterior pituitary** or **neurohypophysis** is continuous with the brain. It is formed during embryonic development from an outgrowth of the inferior part of the brain in the area of the hypothalamus. The outgrowth forms the infundibulum, and the distal end of the infundibulum enlarges to form the posterior pituitary.

Relationship of the pituitary gland to the brain

Portal vessels are blood vessels that begin in a primary capillary network, extend some distance, and end in a secondary capillary network. The **hypothalamohypophyseal portal system** is one of two major portal systems. The other is the hepatic portal system. The hypothalamohypophyseal portal system extends from the hypothalamus to the anterior pituitary. The primary capillary network in the hypothalamus is supplied with blood from arteries that deliver blood to the hypothalamus. From the primary capillary network, the hypothalamohypophyseal portal vessels carry blood to a secondary capillary network in the anterior pituitary. Veins from the secondary capillary network eventually merge with the general circulation.

Hormones, produced and secreted by neurons of the hypothalamus, enter the primary capillary network and are carried to the secondary capillary network. There the hormones leave the blood and act on cells of the anterior pituitary. They act either as **releasing hormones,** increasing the secretion of anterior pituitary hormones or as **inhibiting hormones,** decreasing the secretion of anterior pituitary hormones. Each releasing hormone stimulates and each inhibiting hormone inhibits the production and secretion of a specific hormone by the anterior pituitary. In response to the releasing hormones, anterior pituitary cells secrete hormones that enter the secondary capillary network and are carried by the general circulation to their target tissues. Thus, the hypothalamohypophyseal portal system provides a means by which the hypothalamus, using hormones as chemical messengers, regulates the secretory activity of the anterior pituitary.

There is no portal system to carry hypothalamic hormones to the posterior pituitary. Hormones released from the posterior pituitary are produced by neurosecretory cells with their cell bodies located in the hypothalamus. The axons of these cells extend from the hypothalamus through the infundibulum into the posterior pituitary and form a nerve tract called the **hypothalamohypophyseal tract**. Hormones produced in the hypothalamus pass down these axons in tiny vesicles and are stored in secretory vesicles in the enlarged ends of the axons. Action potentials originating in the neuron cell bodies in the hypothalamus are propagated along the axons to the axon terminals in the posterior pituitary. The action potentials cause the release of hormones from the axon terminals, and they enter the circulatory system.

Hormones of the pituitary gland

Posterior Pituitary Hormones

The posterior pituitary stores and secretes two polypeptide hormones called antidiuretic hormone and oxytocin. A separate population of cells secretes each hormone. **Antidiuretic hormone (ADH)** is so named because it prevents the output of large amounts of urine (*diuresis*). ADH binds to membrane-bound receptors and increases water reabsorption by kidney tubules. This results in less water loss from the blood into the urine, and urine volume decreases. ADH can also cause blood vessels to constrict when released in large amounts. Consequently, it is sometimes called **vasopressin**. **Oxytocin** binds to membrane-bound receptors and causes contraction of the smooth muscle cells of the uterus and milk ejection, or milk "let-down" from the breasts in lactating women. Oxytocin plays an important role in the expulsion of the fetus from the uterus during delivery by stimulating uterine smooth muscle contraction. Commercial preparations of oxytocin are given under certain conditions to assist in childbirth and to constrict uterine blood vessels following childbirth.

Oxytocin also causes the contraction of uterine smooth muscle in non - pregnant women during menses, which helps expel the uterine epithelium and a small amount of blood. Oxytocin also promotes the movement of sperm cells through the uterus and uterine tubes. Oxytocin has been called the great facilitator of life. In addition to its role in reproduction and lactation, oxytocin produced in the limbic system and other parts of the brain influences a variety of social and non-social behaviours in females and males. In many species, oxytocin promotes pair bonding, sexual behaviour, and parental care. In humans, oxytocin promotes social interactions, feelings of attachment, and maternal behaviour. Oxytocin also inhibits memory, decreases the stress

response, reduces feelings of anxiety, suppresses appetite, and raises the pain threshold.

Anterior Pituitary Hormones

Releasing and inhibiting hormones that pass from the hypothalamus through the hypothalamohypophyseal portal system to the anterior pituitary influence anterior pituitary secretions. The hormones secreted are proteins, glycoproteins, or polypeptides. They are transported in the circulatory system and bind to membrane-bound receptor molecules on their target cells. For the most part, each hormone is secreted by a separate cell type. The major hormones of the anterior pituitary, their target tissues, and their effects on target tissues are listed below. Anterior pituitary hormones include growth hormone, thyroid-stimulating hormone, adrenocorticotropic hormone and related substances, luteinising hormone, follicle-stimulating hormone, and prolactin.

Hormones of the pituitary gland

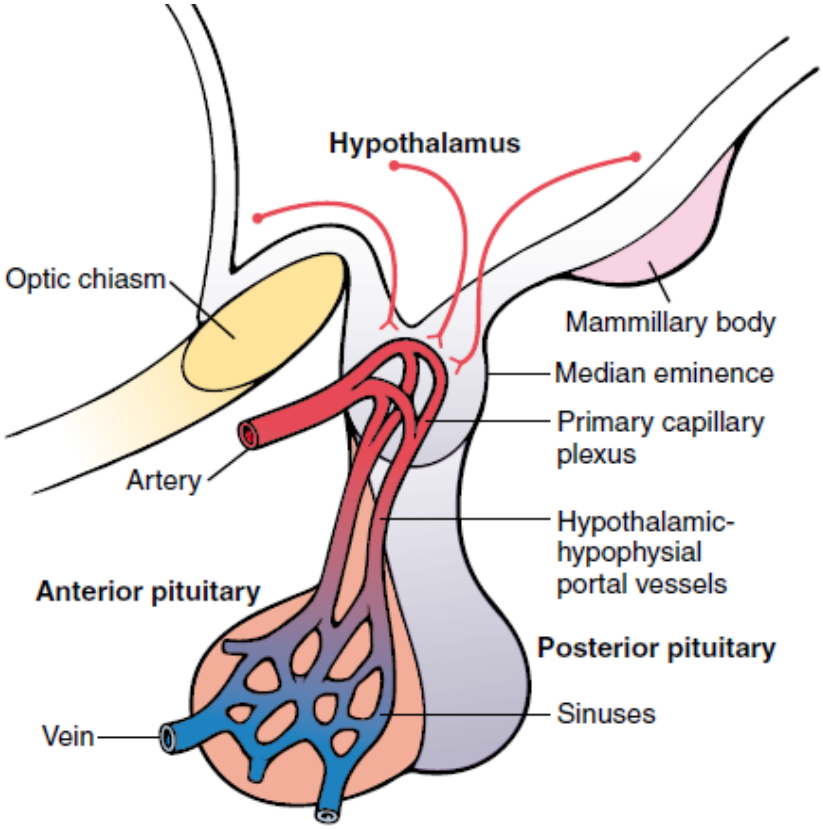
Hormones	Structure	Target Tissue	Response
Posterior Pituitary			
Antidiuretic hormone (ADH)	Small peptide	Kidney	Increased water reabsorption (less water is lost in the form of urine)
Oxytocin	Small peptide	Uterus; mammary glands	Increased uterine contractions; increased milk expulsion from mammary glands; unclear function in males
Anterior Pituitary			
Growth hormone (GH), or somatotropin	Protein	Most tissues	Increased growth in tissues; increased amino acid uptake and protein synthesis; increased breakdown of lipids and release of fatty acids from cells; increased glycogen synthesis and increased blood glucose levels; increased somatomedin production
Thyroid-stimulating hormone (TSH)	Glycoprotein	Thyroid gland	Increased thyroid hormone secretion
Adrenocorticotropic hormone (ACTH)	Peptide	Adrenal cortex	Increased glucocorticoid hormone secretion
Melanocyte-stimulating hormone (MSH)	Peptide	Melanocytes in the skin	Increased melanin production in melanocytes to make the skin darker in color
Luteinizing hormone (LH)	Glycoprotein	Ovaries in females; testes in males	Ovulation and progesterone production in ovaries; testosterone synthesis and support for sperm cell production in testes
Follicle-stimulating hormone (FSH)	Glycoprotein	Follicles in ovaries in females; seminiferous tubes in males	Follicle maturation and estrogen secretion in ovaries; sperm cell production in testes
Prolactin	Protein	Ovaries and mammary glands in females	Milk production in lactating women; increased response of follicle to LH and FSH; unclear function in males

Mechanism by which hypothalamus controls pituitary secretion

Almost all secretion by the pituitary is controlled by either hormonal or nervous signals from the hypothalamus. Indeed, when the pituitary gland is removed from its normal position beneath the hypothalamus and transplanted to some other part of the body, its rates of secretion of the different hormones (except for prolactin) fall to very low levels. Secretion from the posterior pituitary is controlled by nerve signals that originate in the hypothalamus and terminate in the posterior pituitary. In contrast, secretion by the anterior pituitary is controlled by hormones

called *hypothalamic releasing* and *hypothalamic inhibitory hormones* (or *factors*) secreted within the hypothalamus and then conducted to the anterior pituitary through minute blood vessels called *hypothalamichypophysial portal vessels.* In the anterior pituitary, these releasing and inhibitory hormones act on the glandular cells to control their secretion.

The hypothalamus receives signals from many sources in the nervous system. Thus, when a person is exposed to pain, a portion of the pain signal is transmitted into the hypothalamus. Likewise, when a person experiences some powerful depressing or exciting thought, a portion of the signal is transmitted into the hypothalamus. Olfactory stimuli denoting pleasant or unpleasant smells transmit strong signal components directly and through the amygdaloid nuclei into the hypothalamus. Even the concentrations of nutrients, electrolytes, water, and various hormones in the blood excite or inhibit various portions of the hypothalamus. Thus, the hypothalamus is a collection centre for information concerning the internal well-being of the body, and much of this information is used to control secretions of the many globally important pituitary hormones.



Hypothalamic- hypophyseal system

Growth Hormone

Growth hormone (GH) stimulates the growth of most tissues and, through its effect on the epiphyseal plates of bones, GH plays a role in determining how tall a person becomes. GH promotes the protein synthesis necessary for growth by increasing the movement of amino acids into cells and promoting their incorporation into proteins. It also decreases the breakdown of proteins.

GH plays an important role in regulating blood nutrient levels between meals and during periods of fasting. GH increases lipolysis, the breakdown of lipids. Fatty acids released from fat cells into the blood circulate to other tissues and are used as an energy source. The use of fatty acids as an energy source "spares" the use of blood glucose, helping maintain blood sugar levels. In addition, GH increases glucose synthesis by the liver, which releases glucose into the blood. Thus, through its effects on adipose tissue and the liver, GH maintains or increases blood sugar levels. GH has indirect effects on some tissues by stimulating the production of polypeptides called **somatomedins**, primarily by the liver but also by skeletal muscle and other tissues. Somatomedins circulate in the blood, stimulating growth in cartilage and bone and increasing the synthesis of protein in skeletal muscles.

The best-known somatomedins are two polypeptide hormones produced by the liver called **insulin-like growth factor I** and **II** because of the similarity of their structure to insulin. Two hormones released from the hypothalamus regulate the secretion of GH. Growth hormone-releasing hormone (GHRH) stimulates the secretion of GH, whereas growth hormone inhibiting hormone (GHIH) inhibits the secretion of GH. Stimuli that influence GH secretion act on the hypothalamus to increase or decrease the secretion of the releasing and inhibiting hormones. Low blood glucose levels and stress stimulate the secretion of GH, and high blood glucose levels inhibit the secretion of GH. An increase in certain amino acids stimulates increased GH secretion. Most people have a rhythm of growth hormone secretion, with daily peak levels occurring during deep sleep. Growth hormone secretion also increases during periods of fasting and prolonged exercise.

Blood growth hormone levels do not become greatly elevated during periods of rapid growth, although children tend to have somewhat higher blood levels of growth hormone than do adults. In addition to growth hormone, genetics, nutrition, and sex hormones influence growth.

Clinical correlates

1. **Gigantism** is a condition of abnormally increased height that usually results from excessive cartilage and bone formation at the epiphyseal plates of long bones. The most common type of gigantism, **pituitary gigantism,** results from excess secretion of GH. The large stature of some individuals, however, can result from genetic factors rather than from abnormal levels of GH.

2. **Acromegaly** is caused by excess GH secretion in adults, and many pituitary giants develop acromegaly later in life. The GH stimulates the growth of connective tissue, including bones. Bones in adults can increase in diameter and thickness, but not in length because the epiphyseal plates have ossified. The effects of acromegaly are most apparent in the face and hands. Hypersecretion of GH can also cause elevated blood glucose levels and may eventually lead to diabetes mellitus.

3. **Dwarfism**, the condition in which a person is abnormally short, is the opposite of gigantism. Pituitary dwarfism results when abnormally low levels of GH affect the whole body, thus producing a small person who is normally proportioned.

Achondroplasia, or achondroplastic dwarfism, is the most common type of dwarfism; it produces a person with a nearly normal-sized trunk and head but shorter-than-normal limbs. Achondroplasia is a genetic disorder, not a hormonal disorder.

Modern genetic engineering has provided a source of human GH for people who produce inadequate quantities. Human genes for GH have been successfully introduced into bacteria using genetic engineering techniques. The gene in the bacteria causes GH synthesis, and the GH can be extracted from the medium in which the bacteria are grown.

4.0 CONCLUSION

The **pituitary gland,** or **hypophysis,** secretes nine major hormones that regulate numerous body functions and the secretory activity of several other endocrine glands. The **hypothalamus** of the brain and the pituitary gland are major sites where the nervous and endocrine systems interact. The hypothalamus regulates the secretory activity of the pituitary gland. Hormones, sensory information that enters the central nervous system, and emotions, in turn, influence the activity of the hypothalamus.

5.0 SUMMARY

- The pituitary gland secretes at least nine hormones that regulate numerous body functions and other endocrine glands. The hypothalamus regulates pituitary gland activity through hormones and action potentials.
- The posterior pituitary develops from the floor of the brain and connects to the hypothalamus by the infundibulum. The anterior pituitary develops from the roof of the mouth.
- The hypothalamohypophyseal portal system connects the hypothalamus and the anterior pituitary. Through the portal system, the hormones inhibit or stimulate hormone production in the anterior pituitary. The hypothalamohypophyseal tract connects the hypothalamus and the posterior pituitary. Hormones are produced in hypothalamic neurons. The hormones move down the axons of the tract and are secreted from the posterior pituitary.

• Antidiuretic hormone (ADH) promotes water retention by the kidneys. Oxytocin promotes uterine contractions during delivery and causes milk ejection in lactating women. Growth hormone (GH) stimulates growth in most tissues and is a regulator of metabolism. GH stimulates the uptake of amino acids and their conversion into proteins and stimulates the breakdown of fats and the synthesis of glucose.

Activity

Examine the histological structure of the pituitary gland ad distinguish between the anterior and posterior pituitary glands.

6.0 TUTOR-MARKED ASSIGNMENT

1. The pituitary gland
   a. Develops from the floor of the brain.
   b. Develops from the roof of the mouth.
   c. Is stimulated by hormones produced in the midbrain.
   d. Secretes only three major hormones.
   e. Both a and b.
2. The hypothalamohypophyseal portal system
   a. Contains one capillary bed.
   b. Carries hormones from the anterior pituitary to the body.
   c. Carries hormones from the posterior pituitary to the body.
   d. Carries hormones from the hypothalamus to the anterior pituitary.
   e. Carries hormones from the hypothalamus to the posterior pituitary.
3. Hormones secreted from the posterior pituitary

a. Are produced in the anterior pituitary.
b. Are transported to the posterior pituitary within axons.
c. Include GH and TSH.
d. Are steroids.
e. All of the above.

4. Oxytocin is responsible for
   a. Preventing milk release from the mammary glands.
   b. Preventing goiter.
   c. Causing contraction of the uterus.
   d. Maintaining normal calcium levels.
   e. Increasing metabolic rate.
5. Growth hormone
   a. Increases the usage of glucose.
   b. Increases the breakdown of lipids.
   c. Decreases the synthesis of proteins.
   d. Decreases the synthesis of glycogen.
   e. All of the above.

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UNIT 3 THE THYROID AND PARATHYROID GLANDS

CONTENTS

1.0 INTRODUCTION

The **thyroid gland** consists of two lobes connected by a narrow band called the **isthmus**. The lobes are located on each side of the trachea, just inferior to the larynx. The thyroid gland is one of the largest endocrine glands, with a weight of approximately20 g. It is highly vascular and appears redder than its surrounding tissues.

2.0 OBJECTIVES

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

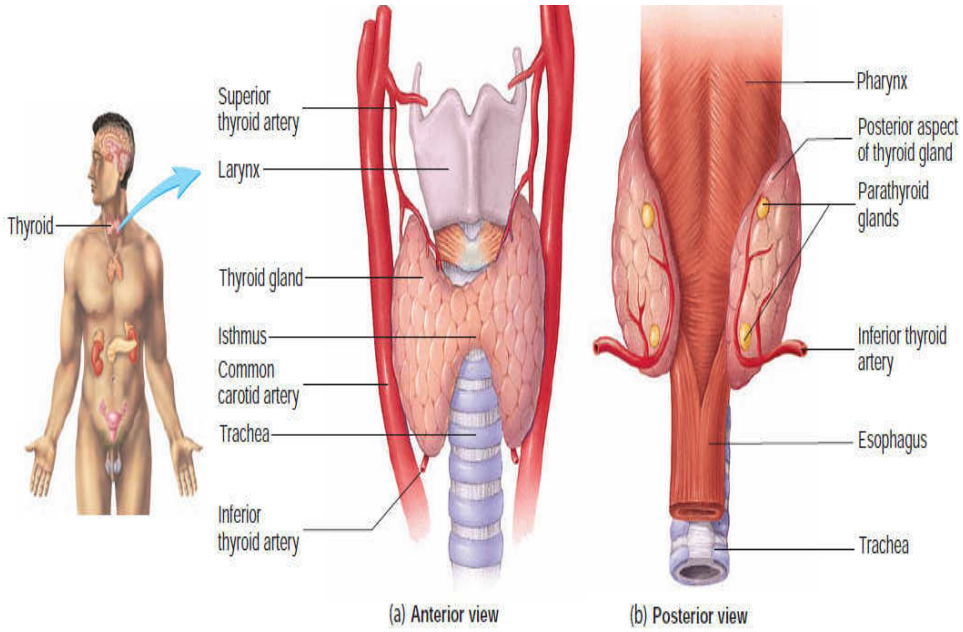
• describe the anatomy of the thyroid and parathyroid gland
• discuss the thyroid hormones and parathyroid hormones.

3.0 MAIN CONTENT

3.1 Structure of the thyroid gland

The thyroid gland contains numerous **follicles,** which are small spheres whose walls are composed of a single layer of cuboidal epithelial cells. Each thyroid follicle is filled with proteins, called **thyroglobulin** which is synthesised and secreted by the cells of the thyroid follicles. Large amounts of thyroid hormones are stored in the thyroid follicles as part of the thyroglobulin molecules. Between the follicles, a delicate network of loose connective tissue contains scattered **parafollicular cells** or **C cells.**

Calcitonin is secreted from the parafollicular cells and plays a role in reducing the concentration of Ca2+ in the body fluids when Ca2+ levels become elevated.



Thyroid and parathyroid glands

Thyroid hormones

The thyroid hormones are **triiodothyronine (T3)** and **tetraiodothyronine (T4).** Another name for T4 is **thyroxine.** T3 constitutes 10% of thyroid gland secretions and T4 90%. Although calcitonin is secreted by the parafollicular cells of the thyroid gland, T3 and T4 are considered to be the thyroid hormones because they are more clinically important and because they are secreted from the thyroid follicles.

T3 and T4 Synthesis

Thyroid-stimulating hormone (TSH) from the anterior pituitary stimulates thyroid hormone synthesis and secretion. TSH causes an increase in the synthesis of T3 and T4, which are then stored inside the thyroid follicles as part of thyroglobulin. TSH also causes T3 and T4 to be released from thyroglobulin and to enter the circulatory system. An adequate amount of iodine in the diet is required for thyroid hormone synthesis because iodine is a component of T3 and T4. The following events in the thyroid follicles result in T3 and T4 synthesis and secretion:

1. Iodide ions (I−) are taken up by thyroid follicle cells by secondary active transport (symport). The movement of the I− is against a concentration gradient of approximately 30-fold in healthy individuals. TSH promotes the uptake of I−.

2. Iodide is transported into the follicle lumen and converted into iodine (Io).

3. Thyroglobulin molecules, which contain numerous tyrosine amino acids, are synthesized, packaged into secretory vesicles, and secreted into the lumen of the follicle.

4. Iodine atoms are bound to a few of the tyrosine amino acids of thyroglobulin, producing monoiodotyrosine, which has one iodine atom, or diiodotyrosine, which has two iodine atoms. After the tyrosines are iodinated, two diiodotyrosine combine to form tetraiodothyronine (T4), which has four iodine atoms. Also, one monoiodotyrosine and one diiodotyrosine combine to form triiodothyronine (T3), which has three iodine atoms. A 2–4 months reserve supply of T3 and T4 is stored within the thyroid follicles as part of thyroglobulin.

5. Thyroglobulin is taken into the thyroid follicle cells by endocytosis.

6. Lysosomes fuse with the endocytic vesicles, and proteolytic enzymes break down thyroglobulin to release T3, T4, and amino acids.

7. T3 and T4 are lipid-soluble and diffuse through the plasma membranes of the follicle cells into the interstitial fluid and finally into the blood. The remaining amino acids of thyroglobulin are used again to synthesize more thyroglobulin.

Effects of Thyroid Hormones

Thyroid hormones interact with their target tissues in a fashion similar to that of the steroid hormones. They readily diffuse through plasma membranes into the cytoplasm of cells. Within cells, they bind to receptor molecules in the nuclei. Thyroid hormones combined with their receptor molecules interact with DNA in the nuclei to influence genes and initiate new protein synthesis. The newly synthesised proteins within the target cells mediate the cells' response to thyroid hormones. It takes up to a week after the administration of thyroid hormones for a maximal response to develop, and new protein synthesis occupies much of that time. Thyroid hormones affect nearly every tissue in the body,

but not all tissues respond identically. Metabolism is primarily affected in some tissues, and growth and maturation are influenced in others.

The normal rate of metabolism for an individual depends on an adequate supply of thyroid hormone, which increases the rate at which glucose, fat, and protein are metabolised. The increased rate of metabolism produces heat. Thyroid hormones increase the activity of N+–K+ pumps, which helps increase the body temperature as ATP molecules are broken down. Thyroid hormones also alter the number and activity of mitochondria, resulting in greater ATP synthesis and heat production. The metabolic rate can increase 60%–100% when blood thyroid hormones are elevated. Low levels of thyroid hormones lead to the opposite effect. Maintaining normal body temperature depends on an adequate amount of thyroid hormones.

Normal growth and maturation of organs also depend on thyroid hormones. For example, bone, hair, teeth, connective tissue, and nervous tissue require thyroid hormones for normal growth and development. Both normal growth and normal maturation of the brain require thyroid hormones.

Regulation of Thyroid Hormone Secretion

Thyroid hormone secretion is regulated by hormones produced in the hypothalamus and anterior pituitary. Thyrotropin-releasing hormone (TRH) is produced in the hypothalamus. Chronic exposure to cold increases TRH secretion, whereas stress, such as starvation, injury, and infections, decreases TRH secretion. TRH stimulates TSH secretion from the anterior pituitary. Small fluctuations in blood levels of TSH occur on a daily basis, with a small nocturnal increase. TSH stimulates the secretion of thyroid hormones from the thyroid gland. TSH also increases the synthesis of thyroid hormones, as well as causing an increase in thyroid gland cell size and number. Decreased blood levels of TSH lead to decreased secretion of thyroid hormones and thyroid gland atrophy. Thyroid hormones have a negative feedback effect on the hypothalamus and anterior pituitary gland. As thyroid hormone levels increase in the circulatory system, they inhibit TRH and TSH secretion. Also, if the thyroid gland is removed or if the secretion of thyroid hormones declines, TSH levels in the blood increase dramatically.

Calcitonin

In addition to secreting thyroid hormones, the thyroid gland secretes a hormone called **calcitonin** produced by the parafollicular cells. Calcitonin secretion is directly regulated by blood Ca2+ levels.. As blood Ca2+ concentration increases, calcitonin secretion increases, and,

as blood Ca2+ concentration decreases, calcitonin secretion decreases. Calcitonin binds to membrane-bound receptors of osteoclasts and inhibits them, which reduces the rate of bone matrix breakdown and the release of Ca2+ from bone into the blood. Calcitonin may prevent blood Ca2+ levels from becoming overly elevated following a meal that contains a high concentration of Ca2+. The role of calcitonin in humans is unclear. It may be important in slowing bone turnover during periods of rapid growth. Calcitonin helps prevent elevated blood Ca2+ levels, but a lack of calcitonin secretion does not result in a prolonged increase in blood Ca2+ levels. Other mechanisms controlling blood Ca2+ levels, such as parathyroid hormone and vitamin D, are able to compensate for the lack of calcitonin secretion.

Parathyroid glands

The **parathyroid glands** are usually embedded in the posterior part of each lobe of the thyroid gland. Usually, four parathyroid glands are present, with their cells organized in densely packed masses or cords rather than in follicles. The parathyroid glands secrete a polypeptide hormone called **parathyroid hormone (PTH),** which is essential for the regulation of blood Ca2+ levels. PTH is much more important than calcitonin in regulating blood Ca2+ levels. PTH regulates blood Ca2+ levels by affecting Ca2+ release from bones, Ca2+ excretion by the kidneys, and vitamin D formation by the kidneys, which promotes Ca2+ absorption by the small intestine. PTH increases the release of Ca2+ from bones into the blood by increasing the number of osteoclasts in bone, which results in increased bone breakdown. PTH promotes an increase in osteoclast numbers by stimulating stem cells in the red bone marrow to differentiate and become osteoclasts. The effect of PTH on osteoclast formation, however, is indirect. PTH binds to its receptors on osteoblasts, stimulating them. The osteoblasts, through surface molecules and released chemicals, stimulate osteoclast stem cells to become osteoclasts.

In the kidneys, PTH increases Ca2+ reabsorption from the urine into the blood so that less calcium leaves the body in urine. PTH also increases the formation of active vitamin D in the kidneys. Vitamin D is carried by the blood to epithelial cells of the small intestine, where it promotes the synthesis of Ca2+ transport proteins. PTH increases blood Ca2+ levels by increasing the rate of active vitamin D formation, which in turn increases the rate of Ca2+ absorption in the intestine. PTH secretion is directly regulated by blood Ca2+ levels. As blood Ca2+ concentration increases, PTH secretion decreases; as blood Ca2+ concentration decreases, PTH secretion increases. This regulation keeps blood Ca2+ levels fluctuating within a normal range of values.

Clinical correlates

1. **Hypothyroidism** is reduced or has no secretion of thyroid hormones. It can be caused by inadequate TSH stimulation of the thyroid gland, an inability of the thyroid gland to produce thyroid hormones or the surgical removal or destruction of the thyroid gland for various reasons. Damage to the pituitary gland can result in decreased TSH secretion. Tumours and inadequate blood delivery to the pituitary because of blood loss during childbirth are causes of pituitary insufficiency. Lack of iodine in the diet can result in decreased thyroid hormone levels because iodine is necessary for the synthesis of thyroid hormones. Damage to the thyroid gland by drugs, chemicals, or an autoimmune disease (Hashimoto disease) can also reduce thyroid hormone production. Hyposecretion of thyroid hormones decreases the rate of metabolism. Low body temperature, weight gain, reduced appetite, reduced heart rate, reduced blood pressure, decreased muscle tone, constipation, drowsiness, and apathy are major symptoms.

2. **Hyperthyroidism** is an abnormally increased secretion of thyroid hormones. After diabetes mellitus, the most common endocrine disorder is a type of hyperthyroidism called **Graves disease.** It is an autoimmune disorder that produces a specific immunoglobulin, called **thyroid-stimulating immunoglobulin (TSI).**

3. **Goiter** is a chronic enlargement of the thyroid gland not due to a tumour. Goitre eventually develops with chronic hypersecretion of thyroid hormones. TSI in Graves disease or elevated TSH produced by pituitary tumours results in continual overstimulation of the thyroid gland. Thyroid hormone synthesis increases and thyroid gland cells increase in size and number, producing goitre. Hypothyroidism caused by an iodine deficiency in the diet can also cause goitre. Without adequate iodine to synthesize thyroid hormones, blood levels of thyroid hormones decrease. The reduced negative feedback of thyroid hormones on the anterior pituitary and hypothalamus results in elevated TSH secretion. TSH causes increased thyroid gland cell size and number and increased thyroglobulin synthesis, even though there is not enough iodine to synthesise thyroid hormones. Historically, goitres were common in people from areas where the soil was depleted of iodine. Consequently, plants grown in these areas, called goitre belts, had little iodine in them and caused iodine-deficient diets. Iodised salt has nearly eliminated iodine-

deficiency goitres. However, it remains a problem in some developing countries

4.0 CONCLUSION

The thyroid gland contains numerous **follicles,** which are small spheres whose walls are composed of a single layer of cuboidal epithelial cells. Each thyroid follicle is filled with proteins, called **thyroglobulin** which is synthesised and secreted by the cells of the thyroid follicles. Large amounts of thyroid hormones are stored in the thyroid follicles as part of the thyroglobulin molecules. Between the follicles, a delicate network of loose connective tissue contains scattered **parafollicular cells** or **C cells.**

5.0 SUMMARY

- The thyroid gland is just inferior to the larynx. The thyroid gland is composed of small, hollow balls of cells called follicles, which contain thyroglobulin. Parafollicular cells are scattered throughout the thyroid gland.
- Thyroid hormone (T3 and T4) synthesis occurs in thyroid follicles. Iodide ions are taken into the follicles by secondary active transport (symport), transported to the follicle lumen, and converted to iodine. Thyroglobulin is secreted into the follicle lumen. Tyrosine molecules with iodine combine to form T3 and T4 within thyroglobulin. Thyroglobulin is taken into follicle cells and is broken down; T3 and T4 diff use from the follicles to the blood.
- Thyroid hormones are transported in the blood. Thyroid hormones bind to thyroxine-binding globulin and other plasma proteins. The plasma proteins prolong the time that thyroid hormones remain in the blood. T3 and T4 bind with nuclear receptor molecules and initiate new protein synthesis.T3 and T4 affect nearly every tissue in the body. T3 and T4 increase the rate of glucose, fat, and protein metabolism in many tissues, thus increasing body temperature. Normal growth of many tissues is dependent on T3 and T4.
- Thyrotropin-releasing hormone (TRH) and thyroid-stimulating hormone (TSH) regulate T3 and T4 secretion. TRH from the hypothalamus increases TSH secretion. TRH increases as a result of chronic exposure to cold and decreases as a result of food deprivation, injury, and infections. Increased TSH from the anterior pituitary increases T3 and T4 secretion.

- The parafollicular cells secrete calcitonin. An increase in blood calcium levels stimulates calcitonin secretion. Calcitonin decreases blood Ca2+ levels by inhibiting osteoclasts.
- The parathyroid glands are embedded in the thyroid gland. Parathyroid hormone (PTH) increases blood Ca2+ levels. PTH stimulates an increase in osteoclast numbers, resulting in an increased breakdown of bone. PTH promotes Ca2+ reabsorption by the kidneys and the formation of active vitamin D by the kidneys. Active vitamin D increases calcium absorption by the intestine. A decrease in blood Ca2+ levels stimulates PTH secretion.

Activity

Examine the slides of the thyroid and parathyroid glands under the microscope in the histology laboratory

SELF – ASSESSMENT EXERCISE

i.. Which of these conditions most likely occurs if a healthy person receives an injection of T3 and T4?
   a. The secretion rate of TSH declines.
   b. The person develops symptoms of hypothyroidism.
   c. The person develops hypercalcemia.
   d. The person secretes more TRH.

ii. Which of these occurs as a response to a thyroidectomy (removal of the thyroid gland)?
   a. increased calcitonin secretion c. decreased TRH secretion
   b. increased T3 and T4 secretion d. increased TSH secretion

iii. Calcitonin
   a. is secreted by the parathyroid glands.
   b. levels increase when blood calcium levels decrease.
   c. causes blood calcium levels to decrease.
   d. insufficiency results in weak bones and tetany.

iv. If parathyroid hormone levels increase, which of these conditions is expected?
   a. Osteoclast numbers are increased.
   b. Calcium absorption from the small intestine is inhibited.
   c. Calcium reabsorption from the urine is inhibited.
   d. Less active vitamin D is formed in the kidneys.

6.0 TUTOR-MARKED ASSIGNMENT

1. Describe events in the thyroid follicles that result in the synthesis and secretion of thyroid hormones.
2. Describe the actions of thyroid hormones T3 and T4.

3. What are some common disorders associated with thyroid dysfunction?
4. What two cell types are found in the parathyroid glands?

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UNIT 4 ADRENAL GLANDS

CONTENTS

1.0 INTRODUCTION

An adrenal gland sits on top of each kidney. It is divided into two portions—the adrenal medulla and the adrenal cortex. The adrenal medulla is the central portion of the gland and secretes **epinephrine** and **norepinephrine.** These hormones produce the same effects that the sympathetic nervous system produces. They increase heart rate, breathing rate, blood pressure, and all the other actions that prepare the body for stressful situations.

2.0 OBJECTIVES

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

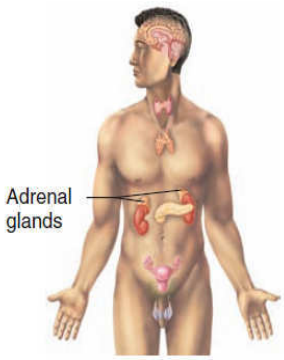
- describe the anatomy of the adrenal gland
- list the hormones released by the adrenal glands and give the functions of each.

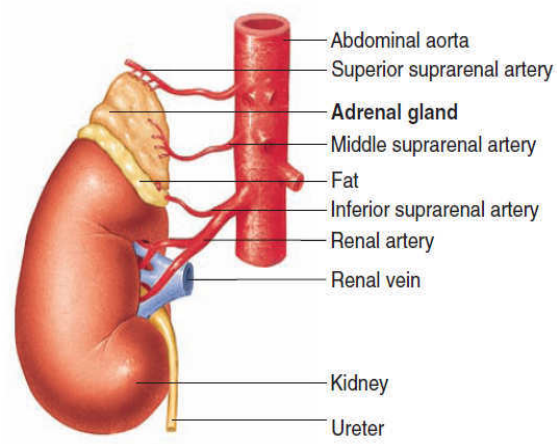
3.0 MAIN CONTENT

3.1 Anatomy of the Adrenal Glands

The adrenal glands, or suprarenal (above the kidney) glands, are two small glands that are located superior to each kidney. Each adrenal gland has an inner part, called the adrenal medulla (marrow or middle), and an outer part, called the adrenal cortex (bark or outer). The cortex has three indistinct layers: the zona glomerulosa, the zona fasciculata, and the zona reticularis.

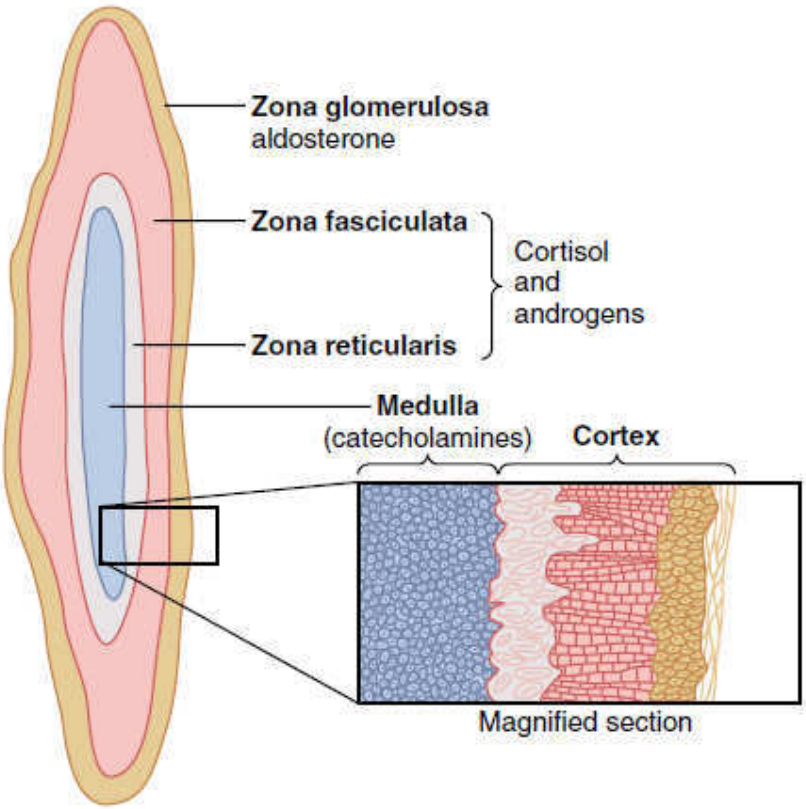
The adrenal glands





The medulla and the three layers of the cortex are structurally and functionally specialized. In many ways, an adrenal gland is four glands in one.

1. The *zona glomerulosa*, a thin layer of cells that lies just underneath the capsule, constitutes about 15 per cent of the adrenal cortex. These cells are the only ones in the adrenal gland capable of secreting significant amounts of *aldosterone* because they contain the enzyme *aldosterone synthase*, which is necessary for synthesis of aldosterone. The secretion of these cells is controlled mainly by the extracellular fluid concentrations of *angiotensin II* and *potassium*, both of which stimulate aldosterone secretion.
2. The *zona fasciculata*, the middle and widest layer, constitutes about 75 per cent of the adrenal cortex and secretes the glucocorticoids *cortisol* and *corticosterone*, as well as small amounts of *adrenal androgens* and *estrogens*. The secretion of these cells is controlled in large part by the hypothalamic-pituitary axis via *adrenocorticotropic hormone* (ACTH).
3. The *zona reticularis*, the deep layer of the cortex, secretes the adrenal androgens *dehydroepiandrosterone* (DHEA) and *androstenedione*, as well as small amounts of estrogens and some glucocorticoids. ACTH also regulates the secretion of these cells, although other factors such as *cortical androgen-stimulating hormone*, released from the pituitary, may also be involved. The mechanisms for controlling adrenal androgen production, however, are not nearly as well understood as those for glucocorticoids and mineralocorticoids.



Secretion of adrenocortical hormones by the different zones of the adrenal cortex and secretion of catecholamines by the adrenal medulla.

Hormones of the adrenal medulla

Approximately 80% of the hormone released from the adrenal medulla is epinephrine or adrenaline. The remaining 20% is norepinephrine. The adrenal medulla consists of cells derived from the same cells that give rise to postganglionic sympathetic neurons, which secrete norepinephrine. Epinephrine is derived from norepinephrine. The adrenal medulla and the sympathetic division function together to prepare the body for physical activity, producing the "fight-or-flight" response. Some of the major effects of the hormones released from the adrenal medulla are the following:

1. Increased breakdown of glycogen to glucose in the liver, the release of the glucose into the blood, and the release of fatty acids from fat cells. The glucose and fatty acids are used as energy sources to maintain the body's increased rate of metabolism.
2. Increased heart rate, which increases blood pressure and blood delivery to tissues.

3. Increased vasodilation of blood vessels of the heart and skeletal muscle, which increases blood flow to the organs responsible for increased physical activity. The hormones increase vasoconstriction of blood vessels to the internal organs and skin, which decreases blood flow to organs not directly involved in physical activity.
4. Increased metabolic rate of several tissues, especially skeletal muscle, cardiac muscle, and nervous tissue. The release of adrenal medullary hormones primarily occurs in response to stimulation by sympathetic neurons because the adrenal medulla is a specialized part of the autonomic nervous system. Several conditions, including exercise, emotional excitement, injury, stress, and low blood glucose levels, lead to the release of adrenal medullary hormones.

Hormones of the adrenal cortex

The adrenal cortex secretes three hormone types: mineralocorticoids, glucocorticoids, and androgens. All are similar in structure in that they are steroids, highly specialized lipids that are derived from cholesterol. Because they are lipid-soluble, they are not stored in the adrenal gland cells but diffuse from the cells as they are synthesised. Adrenal cortical hormones are transported in the blood in combination with specific plasma proteins; they are metabolized in the liver and excreted in the bile and urine. The hormones of the adrenal cortex bind to nuclear receptors and stimulate the synthesis of specific proteins that are responsible for producing the cell's responses.

Mineralocorticoids

The major secretory products of the zona glomerulosa are mineralocorticoids. The mineralocorticoids are so named because they are steroids, produced by the adrenal cortex, that affect the "minerals" Na^+, K^+, and H^+. **Aldosterone** is produced in the greatest amounts, although other, closely related mineralocorticoids are also secreted.

Aldosterone increases the rate of Na^+ reabsorption by the kidneys, thereby increasing blood Na^+ levels. Sodium reabsorption can result in increased water reabsorption by the kidneys and an increase in blood volume, providing ADH is also secreted. Increased blood volume can increase blood pressure. Aldosterone increases K^+ and H^+ excretion into the urine by the kidneys, thereby decreasing blood levels of K^+ and H^+. When aldosterone is secreted in high concentrations, it can result in abnormally low blood levels of K^+ and H^+. The reduction in H^+ can cause alkalosis, an abnormally elevated pH of body fluids.

Glucocorticoids

The zona fasciculata of the adrenal cortex primarily secretes glucocorticoids. The glucocorticoids are so named because they are steroids produced by the adrenal cortex that affect glucose metabolism. The major glucocorticoid is **cortisol**. The target tissues and responses to the glucocorticoids are numerous. The two major types of responses to glucocorticoids are classified as metabolic and anti-inflammatory. Cortisol increases the breakdown of protein and fat and increases their conversion to forms that can be used as energy sources by the body. For example, cortisol causes proteins in skeletal muscles to be broken down to amino acids, which are then released into the circulatory system. Cortisol acts on the liver, causing it to convert amino acids to glucose, which is released into the blood or stored as glycogen. Thus, cortisol increases blood sugar levels. Cortisol also acts on adipose tissue, causing fat stored in fat cells to be broken down to fatty acids, which are released into the circulation. The glucose and fatty acids released into the circulatory system are taken up by tissues and used as sources of energy.

Glucocorticoids decrease the intensity of the inflammatory and immune responses by decreasing both the number of white blood cells and the secretion of inflammatory chemicals from tissues. **Cortisone**, a steroid closely related to cortisol, is often given as a medication to reduce inflammation that occurs in response to injuries. It is also given to reduce the immune and inflammatory responses that occur as a result of allergic reactions or diseases resulting from abnormal immune responses, such as rheumatoid arthritis or asthma. In response to stressful conditions, cortisol is secreted in larger than normal amounts. Cortisol aids the body in responding to stressful conditions by providing energy sources for tissues. If stressful conditions are prolonged, however, immunity can be suppressed enough to make the body susceptible to infections. Cortisol secretion is regulated through the hypothalamus and anterior pituitary gland. Stress and low blood glucose levels stimulate increased **corticotropin-releasing hormone (CRH)** from the hypothalamus. CRH stimulates increased **adrenocorticotropic hormone (ACTH)** secretion from the anterior pituitary gland. ACTH stimulates increased cortisol secretion.

Clinical correlates

I. Adrenal Tumours

The two major disorders of the adrenal medulla are both tumours. Pheochromocytoma is a benign tumour; neuroblastoma is a malignant tumour.

Symptoms, resulting from the release of large amounts of epinephrine and norepinephrine, include hypertension (high blood pressure), sweating, nervousness, pallor, and tachycardia (rapid heart rate). High blood pressure results from the effect of these hormones on the heart and blood vessels and is correlated with an increased chance of heart disease and stroke.

II. **Chronic adrenocortical insufficiency,** often called **Addison disease,** results from abnormally low levels of aldosterone and cortisol in the blood. The cause of many cases of chronic adrenocortical insufficiency is unknown, but it frequently results from an autoimmune disease in which the body's defense mechanisms inappropriately destroy the adrenal cortex. Other causes are infections and tumours that damage the adrenal cortex.

I. **Aldosteronism** is caused by an excess production of aldosterone. Primary aldosteronism results from an adrenal cortex tumour, and secondary aldosteronism occurs when an extraneous factor, such as an overproduction of renin, a substance produced by the kidneys, increases aldosterone secretion. Major symptoms of aldosteronism are hypernatremia (elevated blood Na+), hypokalemia (decreased K+), alkalosis (decreased H+), and high blood pressure due to the retention of water and Na+ by the kidneys.

IV. **Cushing syndrome** is a disorder characterized by the hypersecretion of cortisol and androgens and possibly by excess aldosterone production. Most cases are caused by excess ACTH production by nonpituitary tumours, which usually result from a type of lung cancer. Some cases of increased ACTH secretion do result from pituitary tumours. Sometimes adrenal tumours or unidentified causes can be responsible for hypersecretion of the adrenal cortex without increases in ACTH secretion.

4.0 CONCLUSION

An adrenal gland sits on top of each kidney. It is divided into two portions—the adrenal medulla and the adrenal cortex. The adrenal medulla is the central portion of the gland and secretes **epinephrine** and **norepinephrine.**

5.0 SUMMARY

- The adrenal glands are near the superior poles of the kidneys. The adrenal medulla arises from the same cells that give rise to postganglionic sympathetic neurons.

- The adrenal cortex is divided into three layers: the zona glomerulosa, the zona fasciculata, and the zona reticularis.
- Epinephrine accounts for 80% and norepinephrine for 20% of the adrenal medulla hormones. The adrenal medulla hormones prepare the body for physical activity.
- The zona glomerulosa secretes the mineralocorticoids, especially aldosterone. Aldosterone acts on the kidneys to increase Na+ and to decrease K+ and H+ levels in the blood.
- The zona fasciculata secretes glucocorticoids, especially cortisol. Cortisol increases fat and protein breakdown, increases glucose synthesis from amino acids, decreases the inflammatory response.
- The zona reticularis secretes androgens. In females, androgens stimulate axillary and pubic hair growth and sexual drive

SELF – ASSESSMENT EXERCISE

1. What controls glucocorticoid secretion?
2. The adrenal medulla
   a. produces steroids.
   b. has cortisol as its major secretory product.
   c. decreases its secretions during exercise.
   d. is formed from a modified portion of the sympathetic division of the ANS.
   e. all of the above.
3. If aldosterone secretions increase,
   a. blood potassium levels increase.
   b. blood hydrogen levels increase.
   c. acidosis results.
   d. blood sodium levels decrease.
   e. blood volume increases.
4. Glucocorticoids (cortisol)
   a. increase the breakdown of fats.
   b. increase the breakdown of proteins.
   c. increase blood glucose levels.
   d. decrease inflammation.
   e. all of the above
5. What controls the secretion of aldosterone (a mineralocorticoid)?

6.0 TUTOR-MARKED ASSIGNMENT

1. Examine microscopic slides of the adrenal gland in the histology laboratory
2. Distinguish between the darker cortex with vertically arranged rows of cells and the lighter medulla.

7.0 REFERENCES/FURTHER READING

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UNIT 5 PANCREAS

CONTENTS

1.0 INTRODUCTION

The pancreas is located behind the stomach. It is an endocrine gland as well as an exocrine gland. It is considered an exocrine gland because it secretes digestive enzymes into a duct that leads to the small intestine. It is considered an endocrine gland because it contains structures known as islets of Langerhans that secrete hormones into the bloodstream. The islets of Langerhans secrete two hormones—insulin and glucagon.

2.0 OBJECTIVES

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

- describe the anatomy of the pancreas
- identify the pancreatic hormones
- explain their physiological effects.

3.0 MAIN CONTENT

3.1 Anatomy of the Pancreas

The pancreas lies retroperitoneally in roughly the transpyloric plane. For descriptive purposes, it is divided into head, neck, body and tail. The head lies in the C-curve of the duodenum and sends out the *uncinate process* which hooks posteriorly to the superior mesenteric vessels as these travel from behind the pancreas into the root of the mesentery. Posteriorly lie the inferior vena cava, the commencement of the portal vein, aorta, superior mesenteric vessels, the crura of the diaphragm, coeliac plexus, the left kidney and suprarenal gland. The tortuous splenic artery runs along the upper border of the pancreas. The splenic vein runs behind the gland, receives the inferior mesenteric vein and joins the superior mesenteric to form the portal vein behind the pancreatic neck.

To complete this list of important posterior relationships, the common bile duct lies either in a groove in the right extremity of the gland or embedded in its substance, as it passes to open into the second part of the duodenum. Anteriorly lies the stomach separated by the lesser sac. To the left, the pancreatic tail lies against the hilum of the spleen. Blood is supplied from the splenic and the pancreaticoduodenal arteries; the corresponding veins drain into the portal system.

Pancreatic hormones

Insulin promotes the uptake of glucose by cells. It, therefore, reduces glucose concentrations in the bloodstream. It also promotes the transport of amino acids into cells and increases protein synthesis. Glucagon increases glucose concentrations in the bloodstream and slows down protein synthesis.

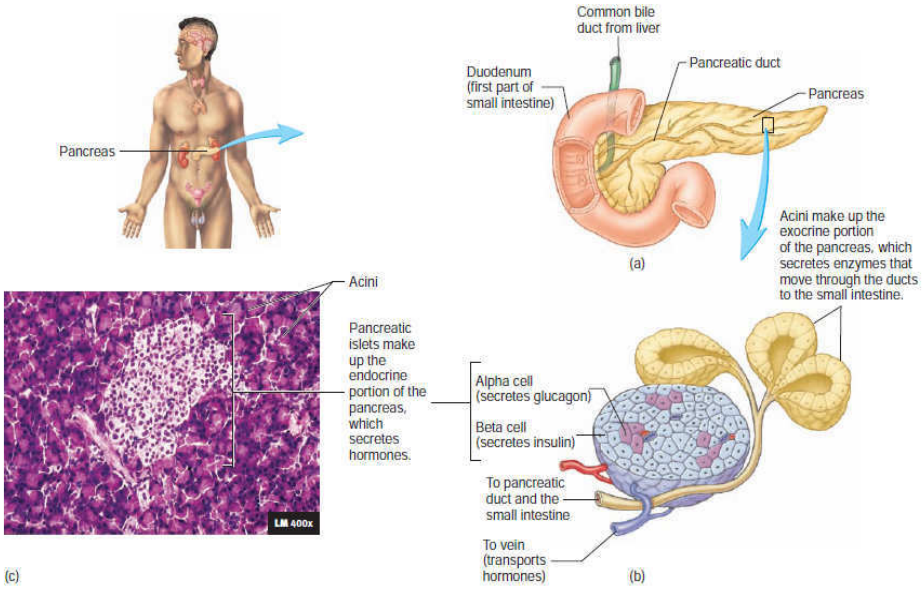

Figure 15.24 Pancreas AP|R
(*a*) Anterior view of the pancreas. (*b*) A pancreatic islet consists of clusters of specialized cells among the acini of the exocrine portion of the pancreas. (*c*) Light micrograph of pancreatic tissue. The stain used for this slide does not distinguish between alpha and beta cells.

Effect of Insulin and Glucagon on Their Target Tissues
Insulin

Insulin increases the uptake of glucose and amino acids by target cells. Once insulin binds to its receptors, the receptors cause specific proteins in the membrane to become phosphorylated. Part of the cell's response to insulin is to increase the number of transport proteins in the membrane of cells for glucose and amino acids. The major target tissues of insulin are the liver, adipose tissue, the skeletal muscles, and the satiety centre within the hypothalamus of the brain. The **satiety centre** is a collection of neurons in the hypothalamus that controls appetite.

Unlike the satiety centre, most of the nervous system does not depend on insulin for the uptake of glucose. Insulin is very important for the normal functioning of the nervous system, however, because insulin regulates blood glucose levels. If blood glucose levels are not maintained within a normal range, the brain malfunctions because glucose is its primary energy source. When insulin levels increase, the movement of glucose and amino acids into cells increases. Glucose molecules that are not immediately used as an energy source are stored as glycogen in the liver, skeletal muscle, and other tissues, or they are converted to fat in adipose tissue. Amino acids are used to synthesize proteins.

When insulin levels decrease, the opposite effects are observed. The movement of glucose and amino acids into tissues slows. Glycogen is broken down to glucose, which is released from the liver, but not from skeletal muscle. Adipose tissue releases fatty acids, and proteins are broken down into amino acids, especially in skeletal muscle. The amino acids are released into the blood, taken up by the liver, and used to synthesize glucose, which is released into the blood. When insulin levels decrease, the liver uses fatty acids to make **acetoacetic acid,** which is converted to **acetone** and **_-hydroxybutyric acid.** These three substances collectively are referred to as **ketone bodies.** The liver releases the ketone bodies into the blood, from which other tissues take them up and use them as a source of energy. The ketone bodies are smaller, more readily used “packets” of energy than are fatty acids. Ketone bodies, however, are acids that can adversely affect blood pH if too many of them are produced. In addition, when insulin levels are low, the liver releases cholesterol and triglycerides into the blood.

Glucagon

Glucagon increases blood sugar and ketone levels. Glucagon primarily influences the liver, although it has some effect on skeletal muscle and adipose tissue. The pancreas secretes glucagon into the hepatic portal system, which carries blood to the liver from the pancreas and intestines. Glucagon binds to membrane-bound receptors, activates G proteins, and increases cAMP synthesis. In general, glucagon causes the breakdown of glycogen to glucose and increases glucose synthesis from amino acids. The release of glucose from the liver increases blood glucose levels.

Regulation of pancreatic hormones

Insulin

Blood levels of nutrients, neural stimulation, and hormones control the secretion of insulin. Elevated blood levels of glucose directly affect the β cells and stimulate insulin secretion. Low blood levels of glucose directly inhibit insulin secretion. Thus, blood glucose levels play a major role in the regulation of insulin secretion. Certain amino acids also stimulate insulin secretion by acting directly on the _ cells. After a meal, when glucose and amino acid levels increase in the circulatory system, insulin secretion increases. During periods of fasting, when blood glucose levels are low, the rate of insulin secretion declines. The autonomic nervous system also controls insulin secretion. The parasympathetic stimulation of digestive system organs is associated with food intake. Parasympathetic stimulation of the pancreas increases its secretion of insulin and digestive enzymes. Sympathetic stimulation inhibits insulin secretion and helps prevent a rapid fall in blood glucose levels during periods of physical activity or excitement. This response is important for maintaining the normal functioning of the nervous system. Gastrointestinal hormones involved with the regulation of digestion, such as gastrin, secretin, and cholecystokinin, increase insulin secretion.

Glucagon

Low blood glucose levels stimulate glucagon secretion, and high blood glucose levels inhibit it. Certain amino acids and sympathetic stimulation also increase glucagon secretion. After a high-protein meal, amino acids increase both insulin and glucagon secretion. Insulin causes target tissues to accept the amino acids for protein synthesis, and glucagon increases the process of glucose synthesis from amino acids in the liver.

Clinical correlates

Diabetes mellitus results from the inadequate secretion of insulin or the inability of tissues to respond to insulin. As a result, blood sugar levels increase. The two major types of diabetes are type 1 and type 2 diabetes. Type 1 diabetes mellitus, also called insulin-dependent diabetes mellitus (IDDM), results from diminished or absent insulin secretion. It affects approximately 5%–10% of people with diabetes mellitus and most commonly occurs in young people. Type 1 diabetes mellitus develops as a result of autoimmune destruction of the pancreatic islets, and symptoms appear after approximately 90% of the islets have been destroyed. Heredity plays a role in the condition, although the initiation of pancreatic islet destruction may involve a viral infection of the

pancreas. Type 2 diabetes mellitus, also called noninsulin-dependent diabetes mellitus (NIDDM), results from insulin resistance, the inability of tissues to respond normally to insulin. It affects approximately 90%–95% of people who have diabetes mellitus and usually develops in people older than 40–45 years of age, although the age of onset varies considerably. People with type 2 diabetes mellitus have a reduced number of functional receptors for insulin, or one or more of the enzymes activated by the insulin receptor are defective. Heredity influences the likelihood of developing type 2 diabetes, but it is not as important a risk factor as for type 1 diabetes.

Three potentially life-threatening conditions are associated with untreated diabetes mellitus: diabetic ketoacidosis, hyperglycaemic hyperosmolar state, and insulin shock.

- **Diabetic ketoacidosis (DKA)** is the triad of hyperglycemia, ketosis, and acidosis. **Ketosis** is the presence of excess ketone bodies in the blood.
- **Hyperglycemic hyperosmolar state (HHS)** consists of very elevated blood sugar levels. It is most likely to develop in type 2 diabetics who have enough insulin to prevent ketosis, but not enough insulin to prevent hyperglycemia.
- **Insulin shock** occurs when there is too much insulin relative to the amount of blood glucose. Too much insulin, too little food intake after an injection of insulin, or increased metabolism of glucose due to excess exercise by a diabetic patient can cause insulin shock. The high levels of insulin cause target tissues to take up glucose at a very high rate. As a result, blood glucose levels rapidly fall to a low level.

4.0 CONCLUSION

The pancreas is located behind the stomach. It is an endocrine gland as well as an exocrine gland. It is considered an exocrine gland because it secretes digestive enzymes into a duct that leads to the small intestine.

5.0 SUMMARY

- The exocrine portion of the pancreas consists of a complex duct system, which ends in small sacs, called acini, which produce pancreatic digestive juices.
- The endocrine portion consists of the pancreatic islets. Each islet is composed of alpha cells, which secrete glucagon, and beta cells, which secrete insulin.
- Insulin’s target tissues are the liver, adipose tissue, muscle, and the satiety center in the hypothalamus. The nervous system is not

a target tissue, but it does rely on blood glucose levels maintained by insulin.

- Insulin increases the uptake of glucose and amino acids by cells. Glucose is used for energy, stored as glycogen, or converted into fats. Amino acids are used to synthesize proteins. Low levels of insulin promote the formation of ketone bodies by the liver.
- Glucagon's target tissue is mainly the liver. Glucagon causes the breakdown of glycogen to glucose and the synthesis of glucose from amino acids. The liver releases glucose into the blood.

- Insulin secretion increases because of elevated blood glucose levels, an increase in some amino acids, parasympathetic stimulation, and gastrointestinal hormones. Sympathetic stimulation decreases insulin secretion.
- Glucagon secretion is stimulated by low blood glucose levels, certain amino acids, and sympathetic stimulation. Somatostatin inhibits insulin and glucagon secretion**.**

SELF – ASSESSMENT EXERCISE

1. What are the physiological effects of the pancreatic hormones?
2. What are causes of diabetes mellitus (insulin deficiency)?
3. Within the pancreas, the pancreatic islets produce
   a. insulin
   b. glucagon.
   c. digestive enzymes.
   d. both a and b.
   e. all of the above.
4. Insulin increases
   a. the uptake of glucose by its target tissues.
   b. the breakdown of protein.
   c. the breakdown of fats.
   d. glycogen breakdown in the liver.
   e. all of the above.
5. Which of these tissues is least affected by insulin?
   a. adipose tissue
   b. heart
   c. skeletal muscle
   d. brain
   e. liver

6.0 TUTOR-MARKED ASSIGNMENT

1. Examine the microscopic slides of the pancreatic tissue in the histology laboratory and note the pancreatic islets which are the endocrine portion of the pancreas.
2. In the gross anatomy laboratory, study the anatomy of the pancreas and its relations.

7.0 REFERENCES/FURTHER READING

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UNIT 6 OTHER ENDOCRINE GLANDS

CONTENTS

1.0 INTRODUCTION

There are other hormone-producing organs which most people are not aware of or consider as parts of the endocrine system. They will be discussed in great detail in this chapter, they include the pineal gland, thymus and the hormone-like substances. We will discuss the testis and ovaries as well.

2.0 OBJECTIVES

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

- discuss the anatomy and functions of other glands with endocrine functions.
- define autocrine and paracrine agents
- discuss the age-related changes that occur in the endocrine system.

3.0 MAIN CONTENT

3.1 Pineal Body

The pineal gland is a small, pine cone-shaped structure located superior and posterior to the thalamus of the brain. The pineal gland produces a hormone called melatonin, which inhibits the functions of the reproductive system in some animals. Melatonin helps to regulate your circadian rhythms, which is your biological clock. Your biological clock helps you decide when you should be awake or asleep. Melatonin is also thought to play a role in the onset of puberty. Tumours that destroy the pineal gland correlate with early sexual development, and tumours that result in pineal hormone secretion correlate with retarded development

of the reproductive system. It is not clear, however, if the pineal gland controls the onset of puberty.

The amount of light detected by the eyes regulates the rate of melatonin secretion. The axons of some neurons in the retina pass from the optic chiasm to the suprachiasmatic nucleus in the hypothalamus, which influences the pineal gland through sympathetic neurons. Increased light exposure inhibits melatonin secretion, whereas darkness allows melatonin secretion. Melatonin is sometimes called the "hormone of darkness" because its production increases in the dark. In many animals, longer day length (shorter nights) causes a decrease in melatonin secretion, whereas shorter day length (longer nights) causes an increase in melatonin secretion. For example, in animals that breed in the spring, increased day length results in decreased melatonin secretion. With decreased inhibition of the hypothalamus by melatonin, sex hormone production increases, which promotes the development of reproductive structures and behaviour. In the fall, decreased day length results in increased melatonin secretion, decreased sex hormone production, atrophy of reproductive

Thymus gland

The thymus is in the neck and superior to the heart in the thorax; it secretes a hormone called **thymosin.** Both the thymus and thymosin play an important role in the development and maturation of the immune system Thymosin promotes the production of certain lymphocytes.

Other hormone-producing organs include:

Cholecystokinin is released from the gastrointestinal tract. They regulate digestive functions by influencing the activity of the stomach, intestines, liver, and pancreas. The kidneys secrete a hormone in response to reduced oxygen levels in the kidney. The hormone is called erythropoietin. It acts on the red bone marrow to increase the production of red blood cells. In pregnant women, the placenta is an important source of hormones that maintain pregnancy and stimulate breast development. These hormones include estrogen, progesterone, and human chorionic gonadotropin, which is similar in structure and function to LH. These hormones are essential to the maintenance of pregnancy

Hormone-like Substances

Autocrine chemical messengers are chemicals released by a cell that affect the cell producing it or affect nearby cells of the same cell type. Examples of autocrine chemical messengers include a group of related

chemical mediators called eicosanoids, which are derived from fatty acids. The eicosanoids include prostaglandins, thromboxanes, prostacyclins, and leukotrienes.

Paracrine chemical messengers are chemicals released by a cell that affect nearby cells of a different cell type. Examples of paracrine chemical messengers include growth factors, clotting factors, and histamine. Autocrine and paracrine chemical messengers differ from hormones in that they are not secreted from discrete endocrine glands, they have local effects rather than systemic effects, or they have functions that are not understood adequately to explain their role in the body. The schemes used to classify chemicals on the basis of their functions are useful, but they do not indicate that a specific molecule always performs as the same type of chemical messenger in every place it is found. Some chemical messengers, such as prostaglandins, have both autocrine and paracrine functions. Furthermore, some of these chemicals can also act as hormones. Testosterone produced in the testes has a paracrine effect on the development of sperm cells, but it is released into the blood and has an endocrine effect on skeletal muscle development.

Effects of ageing on the endocrine system

Age-related changes to the endocrine system include a gradual decrease in the secretion of some, but not all, endocrine glands. Some of the decreases in secretion may be due to a decrease in physical activity as people age. GH secretion decreases as people age, and the decrease is greatest in people who do not exercise. It may not occur in older people who exercise regularly. Decreasing GH levels may explain some of the gradual decreases in bone and muscle mass and some of the increase in adipose tissue in many elderly people.

Administering GH to slow or prevent the consequences of ageing has not been established to be effective, however, and unwanted side effects are possible. A decrease in melatonin secretion may influence age-related changes in sleep patterns. The secretion of thyroid hormones decreases slightly with age. Age-related damage to the thyroid gland by the immune system can occur, and this happens in women more than in men. Approximately 10% of elderly women have some reduction in thyroid hormone secretion.

Parathyroid hormone secretion does not appear to decrease with age. Blood levels of Ca^{2+} may decrease slightly because of reduced dietary calcium intake and vitamin D levels. The greatest risk is a loss of bone matrix as parathyroid hormone increases to maintain blood levels of Ca^{2+} within their normal range.

Reproductive hormone secretion gradually declines in elderly men, and women experience menopause.

There are no age-related decreases in the ability to regulate blood glucose levels. There is an age-related tendency to develop type 2 diabetes mellitus in those who have a familial tendency to do so, and it is correlated with age-related increases in body weight. Thymosin from the thymus decreases with age. Fewer immature lymphocytes are able to mature and become functional, and the immune system becomes less effective in protecting the body. There is an increased susceptibility to infection and to cancer.

4.0 CONCLUSION

There are other hormone-producing organs which most people are not aware of or consider as parts of the endocrine system. These are pineal gland and thymus gland.

5.0 SUMMARY

- The pineal gland produces melatonin, which can inhibit reproductive maturation and may regulate sleep-wake cycles.
- The thymus produces thymosin, which is involved in the development of the immune system.
- The kidneys produce erythropoietin, which stimulates red blood cell production.
- Autocrine chemical messengers are chemicals that locally affect cells producing them or affect cells of the same type.
- There is a gradual decrease in the secretion rate of most, but not all, hormones. Some decreases are secondary to gradual decreases in physical activity**.**

SELF – ASSESSMENT EXERCISE

1. Melatonin
    a. is produced by the posterior pituitary.
    b. production increases as day length increases.
    c. inhibits the development of the reproductive system.
    d. increases GnRH secretion from the hypothalamus.
    e. decreases the tendency to sleep
2. The hormone secretin
    a. plays an important role in the development and maturation of the immune system.
    b. is released from the gastrointestinal tract.

c. acts on red bone marrow to increase the production of red blood cells.
d. is essential to the maintenance of pregnancy.

3. Which of the following statements about autocrine and paracrine agents is true?
   a. They usually have a local eff ect, but sometimes can have systemic effects.
   b. They typically are not produced in discrete endocrine glands.
   c. Paracrine agents affect different cell types from which the paracrine agent is released.
   d. Eicosanoids and prostaglandins are examples of autocrine agents.
   e. All of the above.
4. The production of which hormone does not decrease with age?
   a. growth hormone
   b. melatonin
   c. thyroid hormones
   d. parathyroid hormones
   e. reproductive hormones

6.0 TUTOR-MARKED ASSIGNMENT

1. Discuss the anatomy and functions of other glands with endocrine functions.
2. Define autocrine and paracrine agents.
3. Discuss the age-related changes that occur in the endocrine system.

7.0 REFERENCES/FURTHER READING

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