Q. 37. Describe the anatomy of Adrenal gland.

The adrenal gland is a compound structure with complex functions. It is situated on the upper border of each kidney, two in number and roughly triangular in shape. It has got outer cortex and inner medulla. The outer cortex of adrenal gland is essential for vertebrate life and its secretions are involved in the regulation of salt and water metabolism and in the maintenance of the integrity of living membranes. The inner medulla of adrenal gland influences carbohydrate metabolism and serves as a functional adjunct to the sympathetic division of the autonomic nervous system.

Anatomy : The adrenal glands are paired, convoluted, somewhat pyramidal in shape and each being placed on the upper pole of the corresponding kidney. Anatomically each adrenal gland is a single gland but functionally and developmentally, it is a combibination of two glands in one consisting of two functional units known as outer cortex and inner medulla. In some animals like some fishes, the cortex and the medulla occur separately. The two adrenal glands differ from each other in shape. The right adrenal gland is triangular and the left one is semilunar in shape. The outer cortex is approximately 80 percent by weight in the normal-adult which is firm and golden yellow in colour and the inner medulla is soft and reddish-brown in colour.

(a Capsules : Each adrenal gland has got two capsules known as true and false.

(b) Glands proper : Each adrenal gland measures about one to two inches in leng h, one inch in breadth and about one fourth inch in thickness. Each gland weighs about 5g. They are smaller in conditions associated with a deficiency of ACTH and becomes as much as four times larger in response to a chronic excess of ACTH (1) *Right adrenal gland :* It is triangular in shape and its apex directed upwards and the b se downwards. (2) *Left adrenal gland :* It is semlunar in shape and forms a broad and expanded base directed downwards and a taper ing apex directed upwards. The lower part of the anterior surface is in relation with the tail of the pancreas and the splenic vessels.

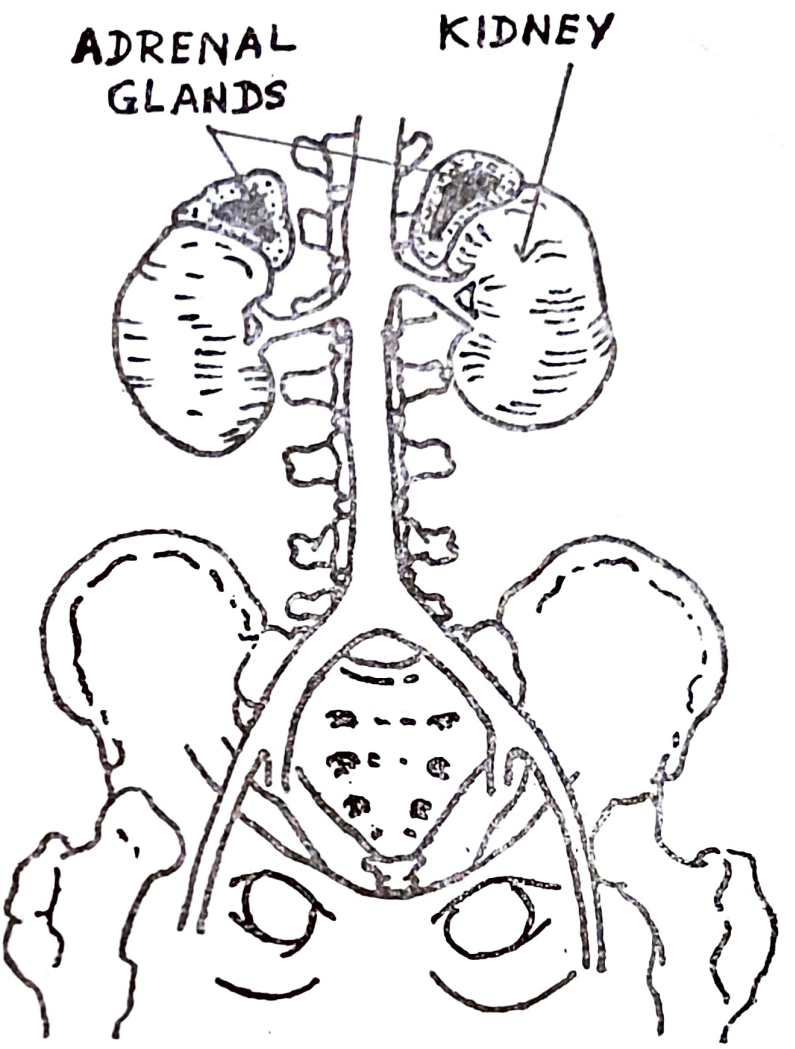


Fig. 82. Anatomical position of Human Adrenal g ands at superior pole of each kidney.

Q 38. Describe the structure and active principle of Adrenal cortex.

The adren l cortex is the outer ost part of the adrenal gland. It is approxim tely 80 percent by weight in the normal adult which is gol en yellow in colour and firm in consi tency. It is a very vital organ of all animals and man. It regulates salt, water, carbohydrate, pr tein and fat metabolism.

Histological stru ture : The cells of the adrenal cor ex are polyh dr l in shape with well defi ed nuclei, G lgi apparatus, typical mitoch.ondria with high lipid content in t eir protoplasm. The adrenal c rtex is cove ed with thick, collogenous capsule from which a fibrous trabeculae pass into the substance o the gland carrying blood vessels and dividing the cortex into cell columns. In the embryo, the adren l cort x is made up of adult outer zone and foetal inner zone. After birth the classical adult z one forms the adult cortex fr m which glomerulosa, fasciculata and r ticularis develop, The cortical part is rich in ascorbic acid and cholesterol esters and arra ged into three z ones from outside inwards :

(a) Zona glomerulosa (Outer zone) : It is the outer layer of the cortex situated immediately beneath the fibrous capsule and their columnar cells lie with their long axis parallel to the surface. They

are arranged in ovoid or circular fashion. This layer consists of small polyhedral cells arranged in rounded groups or curved columns. The cells having deeply staining nuclei and scanty basophilic cytoplasm in which a few lipid droplets may be present. Their lipids contents are low and cytoplasm contains acidophil granules. The columnar cells are comparatively small and thickly set. The cells are rounded and arranged in groups with smooth endoplasmic reticulum, elongated mitochondria with transverse shelf-like infoldings of the inner mitochondrial membrane and lamellar cristae. The large amounts of smooth endoplasmic reticulum seems to be involved in the steroid forming process. The steroid biosynthesis occurs in the mitochondria. Zona glomerulosa secretes mainly aldosterone and a small amount of glucocorticoids and mineralocortico-steroid hormones.

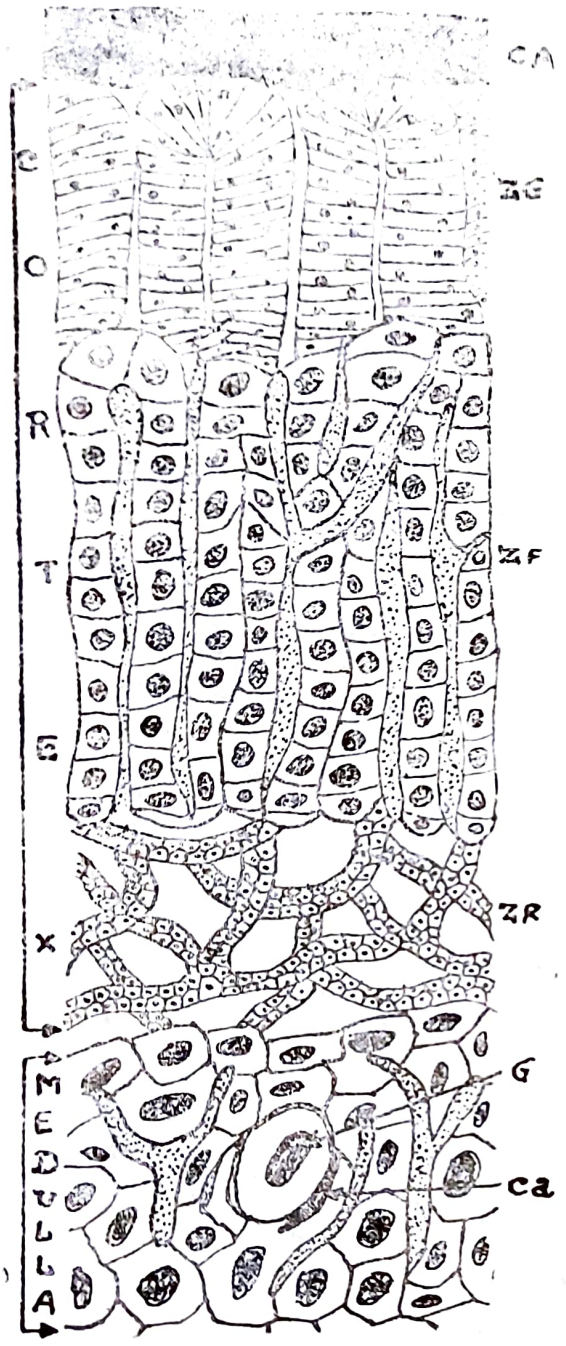


Fig. 83. Section of Adrenal gland. CA-Capsule, ZG-Zona Glomerulosa, ZF Zona Fasciculata, ZR-Zona Reticularis. G-Ganglion cell, Ca-Capsule cell.

(b) Zona fasciculata (Middle zone) : It is the broadest layer with proportionately larger cells containing browish-yellow pigment granules. This layer consists of columnar cells and lie vertical to the surface. The cells are usually cuboidal, light staining and binucleate and in columns or cord like fashion. The cells contain much lipoid material known as *liposomes*, large amounts of phosphol pids, fats, fatty acids and cholesterol, few vesicular mitochondria with tubular villi and basophil granules in their cytoplasm. The cells are arranged in radiating columns and perpendicular to the surface; The cells are rich in cholesterol and vitamin C. These are stained by Sudan III and hence they are also called *sudanophilic granules.* The mitochondria are more nearly spherical and the cristae appear as short tubular invaginations of inner membrane. This layer contains large amounts of smooth surfaced endoplasmic reticulum which are supposed to be related to the synthesis of steroid hormones. Zona fasciculata secretes predominantly glucocorticoids, cortisol and corticosteroids.

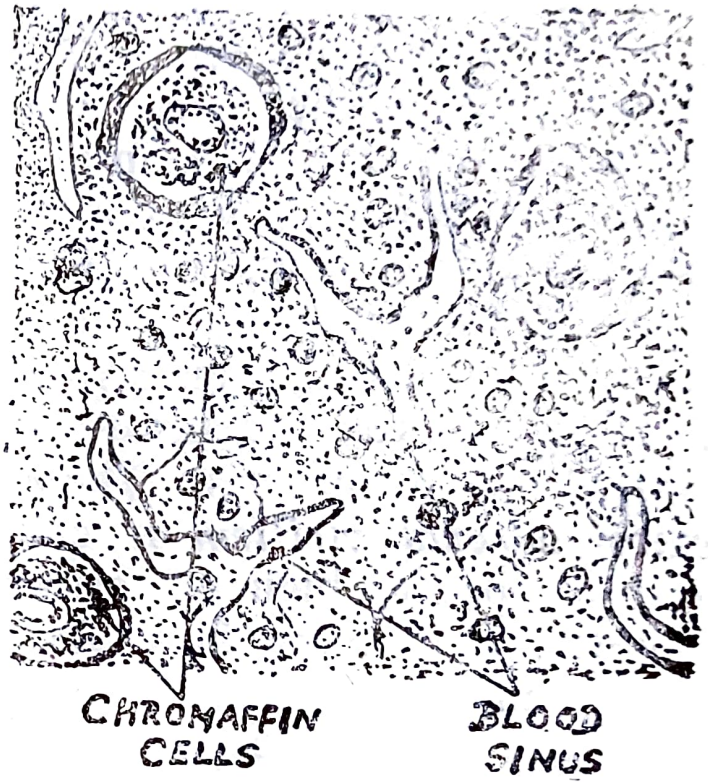
(c) Zona reticularis (Inner zone) : This layer is in contact with the medulla and consists of cells that are interblocked with each other to form a reticular arrangement leaving wide blood spaces. The cytoplasm contains much agranular endoplasmic reticulum, numerous lysosomes and some degenerated pigment bodies. Zona reticularis secretes sex corticoids producing sex hormones and a small amount of glucorticocoids but no aldosterone. The dominant secretions of this layer are the adrenal androgens like dehydroepiandrosterone (DHEA) and androstenedione.

Q. 43. Describe the structure of Adrenal medulla and mechanism of actions of Catecholamines.

Adrenal medulla represents a modified sympathetic ganglion and is regulated by preganglionic sympathetic nerves. A small groups of cells known as paraganglia which resembles adrenal medulla. They are found near the thoracic and abdominal sympathetic ganglia.

Histology : The adrenal medulla is made up of scattered, irregularly disposed polyhedral cells or sometimes neurogleal cells, richly supplied with blood vessels and is controlled by sympathetic nervous system. It consists of interlacing cords of densely inner-

vated granule-containing cells surrounded by blood sinuses. Morphologically, two types of cells can be distinguished which are an epinephrine-secreting type that has larger, less dense granules and a non-epinephrine-secreting type in which the smaller, very dense granules fail to fill the vesicles in which they are contained. The cells are large ovoid or columnar in type containing granules which are stained by chromic acid and its salts. The cells are stained brown due to oxidation of catecholamines by chromic acid. These cells are described as the chromaffin cells. The cells of adrenal medulla are described now-a-days as pheochromocytes. They can also be stained with ferric chloride which turn the granules blue and osmic acid turns them black. Electron microscopic study have revealed that these granules are surrounded by a smooth-surfaced membrane in secretory vesicle, the diameter varying from 0·1 to 0·5μ. The packaging process of the granuless take place in Golgi apparatus. The membrane-enclosed granules move towards the cell surface during discharge. The medullary cells do not have many rough-surfaced vesicles. The moderate content of free ribosomes is also present. The non-epinephrine secreting granules are released by exocytosis and the granule contents enter the blood stream.



Fig, 86. Histological Structure of Adrenal Medulla.

I. Functions of Glucocorticoids : The glucocorticoid hormones have distinct effects on carbohydrate metabolism including promotion of gluconeogenesis, promotion of liver glycogen deposition and elevation of blood glucose concentrations. The glucocorticoids posses the following biologic properties and some of which are of considerable clinical importance.

(a) Effects on different types of Stress : The variety of conditions like trauma, haemorrhage, infections, sensitizing antigens and chemicals always demand additional glucocorticoids. Any stress particularly haemorrhagic shock produces alarmed reaction which stimulates the hypothalamus to release corticotrophin releasing hormone (CRH) that acts on corticotrophs of adenohypophysis to secrete ACTH. The glucocorticoids cause rapid mobilization of amino acids and fats from their cellular stores which make available both for energy and for synthesis of other compounds. The amino acids are used to synthesize certain essential intracellular substances like purines, pyrimidines and creatine phosphate which are necessary for maintenance of cellular life.

(b) Effects on ACTH suppression : All glucocorticoids suppress the synthesis and secretion of ACTH. The action of steroids is partially exerted at the level of pituitary itself but it is possible that glucocorticoids also act to suppress CRF.

(c) Effects on Inflammation and Allergy (Anti-inflammatory activity) : Glucocorticoids inhibit inflammatory and allergic reactions in supraphysiologic amounts. Inflammation is characterized by Rubor (redness), Calor (warmth), Tumor (swelling) and Dolor (pain). Their reactions are due to liberation of chemical materials by bacteria or bacteria-host reaction with defensive force of body

causing migration of leucocytes, rupture of lysosomal membrane and production of immune bodies. These are necessary for natural diffuse mechanism of the body.

(d) **Effects on Haemopoietic System :** Cortisol increases red blood cell count, increases neutrophil but decreases lymphocytes and eosinophils. When excess cortisol is secreted by the adrenal glands, polycythemia results and on the other hand, when the adrenal glands secrete no cortisol and anaemia often results. A finding of lymphocytopenia or eosinopenia is an important diagnostic feature for overproduction of cortisol by adrenal gland.

(f) **Effects on Cardiovascular Sytem :** Cortisol maintains normal blood volume with the help of the maintenance role on extracellular fluid composition. During excess administration of glucocorticoids, blood volume is raised with oedema and hypertension. Cortisol raises blood pressure by enhancing the action of catecholamine and at the same time, cortisol reduces capillary permeability. The rise of blood pressure is possibly due to increased angiotensinogen formation which in turn produces excess angiotensin that leads to excess aldosterone secretion.

(g) **Effects on Respiratory System :** It causes maturation of foetal lung with manufacture of surfactant. It facilitates proper ventilation in asthma.

(h) **Effects on G. I System :** (1) Cortisol increases hydrochloric acid secretion from the oxyntic cell of the stomach and leads to hyperchlorhydria. The continued administration of corticoids will diminish mucosal barrier which may lead to peptic ulcer leading many complications. (2) It also stimulates trypsinogen secretion from pancreastosome extent and pepsinogen secretion to a lesser extent. (3) It inhibits absorption of calcium from small intestine.

(i) **Effects on Skin :** Skin will be very thin with appearance of purplish striae in hypercortisolaemia.

(j) **Effects on Muscular System :** (1) Cortisol causes loss of muscle power and muscular weakness. 2 During adrenocortical insufficiency, if cortisol is administered, it will restore muscular weakness and loss of muscle power to some extent. (3) On excess cortisol administration, muscular wasting and weakness are localised due to muscle proteins.

(k) **Effects on Skeletal System :** Excess cortisol administration leads to : (1) Inhibition of formation of cartilaginous bone. (2) Thinning of epiphyseal cartilaginous plate. (3) Defect in the formation of bone matrix and calcification due to lack of blood calcium. (4) The inhibition of absorption of calcium from the small intestine, osteoporosis develops that may precipitate fracture. (5) It antagonises the action of vitamin D.

(l) **Effects on Nervous System :** (1) Initially there is euphoria and after cortisol administration, it is followed by sense of depression and sluggish conduction of nerve impulse takes place through nerve fibres. (2) Excess cortisol leads to reduction in threshold of electrical stimulation of brain which may precipitate epileptiform convulsions.

(m) **Effects on Carbohydrate Metabolism :** Cortisol and corticosterone help carbohydrate metabolism by increasing neoglucogenesis and reducing peripheral utilization of sugar. (1) *Decreased glucose utilization by the cells :* Cortisol causes a moderate decrease in the rate of glucose utilization by the cells which is based on depressed activity of the oxidation of NADH. So, glucocorticoids slightly depress glucose transport into the cells which depress cellular glucose utilization. (2) *Raised blood glucose concentration :* The blood glucose concentration is raised due to increased rate of gluconeogenesis and the moderate reduction in rate of glucose utilization by the cells. The enzyme glucose-6-phosphatase catalyzes the dephosphorylation of liver glucose which promotes its transport into the blood and the blood glucose concentration is further increased.

(n) **Effects on Protein Metabolism :** The metabolism of protein is influenced by cortisol, corticosterone and aldosterone in the decreasing order. (1) *Increased liver protein and plasma protein :* Cortisol reduces protein synthesis in many tissue like muscles, skin and bone matrix but stimulates protein synthesis in the liver. (2) *Protein-wasting activity :* Glucocorticoids accelerate the breakdown of proteins like albumin and inhibit amino acid uptake and protein synthesis by many extrahepatic tissues. (3) *Diminished transport of amino acids :* Cortisol depresses amino acids transport into muscle cells and other extrahepatic cells. (4) *Increased plasma amino acid concentration and transport of amino acid :* Cortisol helps to increase the plasma concentration of amino acids and enhances transport of amino acid into the hepatic cells.

(o) **Effects on Fat Metabolism :** The metabolism of fat is also influenced by cortisol and corticosterone. (1) *Stimulation of Lipid synthesis :* Cortisol increases lipolysis in the adipose tissue due to its inducing action on adipose tissue lipase which may raise the blood free fatty acid level. It increases peripheral lipogenesis possibly due to an increased insulin secretion. It also accelerates the synthesis of triglycerides in the liver. So, cortisol may stimulate lipid synthesis in the liver. (2) *Mobilization of fatty acids :* Cortisol promotes mobilization of fatty acids from adipose tissue and thereby it increases the concentration of free fatty acids in the plasma which also increases their utilization for energy.

II Functions of Mineralocorticoids : The mineralocorticoids are selectively effective in maintenance of electrolyte and water balance in the body. The basic actions of mineralocorticoids show increased tubular reabsorption of sodium but they have got many additional secondary effects.

(a Effects on Kidney : Aldosterone serves tubular reabsorption of sodium and chloride ions, tubular secretion of potassium and alkalosis. (1) *Tubular reabsorption of Sodium* : Aldosterone and other mineralocorticoids help to increase the rate of tubular reabsorption of sodium from the renal tubules.

Increased aldosterone secretion causes increased tubular reabsorption of sodium which tends to increase the quantity of sodium in the extracellular fluids and there is no significant hypernatremia. (2) *Tubular secretion of Potassium* : Aldosterone enhances secretion of potassium into the distal tubules and collecting tubules of the kidneys and thereby the extracellular fluid potassium ion concentration is controlled. The excessive secretion of potassium ions under aldosterone activity decreases the potassium concentration in the extracellular fluids causing hypokalemia. Decreased potassium ion concentration develops muscle paralysis or severe muscle weakness caused by hyperpolarization of the nerve and muscle fibre membranes which prevents transmission of action potentials. (3) *Alkalosis* : The increased secretion of aldosterone promotes alkalosis while decreased secretion promotes acidosis. The changes in body fluid pH can be compensated by the normal acid-base regulatory mechanisms. (4) *Tubular reabsorption of chloride ions* : Aldosterone causes greatly enhanced reabsorption of chloride ions The chloride ions are negatively charged which are repelled from the electronegative tubular fluid and are attracted toward the extracellular fluid.

(b) Effects on Fluid volumes and Cardiovascular system : Aldosterone regulates and controls extracellular fluid volume, blood volume, cardiac output and arterial blood pressure. (1) *Extracellular fluid volume* : Mineralocorticoids greatly increase the quantities of sodium, chloride and bicarbonate ions in the extracellular fluids. (2) *Polydipsia and Polyuria* : Aldosterone causes a person to become thirsty due to the stimulation of thirst center and as a result the person drinks excessive amounts of water known as *polyuria.* (3) *Blood volume* : Aldosterone helps to moderate increase in blood volume because plasma volume increases almost proportionally with extracellular fluid volume. (4) *Cardiac output* : Lack of aldosterone secretion has a profound effect on cardiac output. Excessive secretion of aldosterone causes resultant increase in extracellular fluid volume and blood volume increases the cardiac output. (5) *Arterial blood pressure* : Excess aldosterone cause increase in blood volume and thereby arterial blood pressure is raised.

(c) Effects on Cellular mechanism : The sequence of events caused by the basic action of aldosterone lead to increase sodium reabsorption are the following : (1) Aldosterone diffuses lipid solubility in the cellular membranes to the interior of the tubular epithelial cells. (2) Aldosterone combines with a highly specific receptor protein which has a stereomolecular configuration in the cytoplasm of the tubular cells and it will allow aldosterone only or extremely similar compounds to combine.

III. Functions of Sexocorticoids : Several moderately active male sex hormones called adrenal androgens are continuously secreted by the adrenal cortex. Progesterone and oestrogen are female sex hormones and they have been extracted from the adrenal cortex

III. Functions of Sexocorticoids : Several moderately active male sex hormones called adrenal androgens are continuously secreted by the adrenal cortex. Progesterone and oestrogen are female sex hormones and they have been extracted from the adrenal cortex

in minute quantities. The multiple effects of sexocorticoids are due to an action on the genetic mechanism controlling protein synthesis, the sex hormones like androgen, oestrogen and progesterone, act by stimulating DNA-dependent synthesis of certain mRNA's in the nuclei of their target cells. Oestrogen and androgen play an extremely important role on the different action of sex in the foetus, development and growth of sex organs, sex glands and secondary sex character in the childhood.

(*a*) *Effects of Androgenic Sexocorticoids* : These may influence the growth and differtiation of secondary sex organs and characters including pubic and axillary hair in male. The adrenal androgens also exert mild effects in the female, not only before puberty but also throughout life. In children of either sex, an excess of adrenal androgen can cause phallic hypertrophy, increased muscularity, rapid somatic growth and precocious development of pubic, axillary and facial hair. The adult individual can develop acne vulgaris, coarsening of the voice and recession of scalp hair. These hormones may be responsible for libido in women.

(b) *Effects of Oestrogenic Sexocorticoids* : These may cause clitorial hypertrophy, hirsutism, balding, coarsening of the voice, amenorrhoea, infertility and breast atrophy in women. In men and children, it leads to enlargement of breasts and maturation of uterus and vagina in young girls.

Q. 44 What is Adrenaline? Describe the physiological actions of Epinephine.

Epinephrine is one of the active principles of adrenal medulla. The content of epinephrine of the resting gland is about 0·1 mg%. The total store of epinephirne in both glands is about 10 mg in man.

Physiological Actions of Epinephrine : Epinephrine liberates from the adrenal medulla which acts on different tissues and systems of the body which are briefly summarised below :

I. On Eye : (1) There will be contraction of meridional fibres of the iris which produces mydriasis i.e. dilatation of the pupil and by contraction, the dilator muscle of iris acting through β_1-receptor. (2) Secretion of tear from the lacrimal gland. (3) Retraction of eyeball due to the contraction of the muscles of Muller.

II. On Cardio-vascular System : It includes blood, heart and blood vessels.

(a) On Blood : (1) There will be increased count of red blood cells in the peripheral blood due to the contraction of the spleen producing a condition known as polycythaemia. (2) The eosinophil count in the blood will be diminished. This reduction occurs indirectly through the liberation of more ACTH and thereby liberation of cortical hormones. (3) Epinephrine hastens the process of blood coagulation by producing constriction of the blood vessels and by liberating more thromboplastin from disintegrated platelets. (4) There will be raised white blood cell count, platelet count, percentage of haemoglobin and increased blood volume due to contraction of spleen.

(b) On Heart : (1) Epinephrine increases the rate and force of the heart beats, rhythmicity and contractility of the heart and the cardiac output is increased. (2) Dilatation of the coronary blood vessels. (3) There will be increased excitability of myocardium of the heart. (4) There is raised conductivity of the bundle of His of the special junctional tissue of the heart.

(c) On Blood Vessels : (1) Epinephrine will cause constriction of all the blood vessels of the body with the exception of coronary vessels and blood vessels of the skeletal muscles which will be dilated. (2) There will be dilatation of the coronary vessels, vessels of the skeletal muscles and hepatic blood vessels. (3) The venomotor tone is increased which helps in venous return.

III. On Respiratory System : It includes the action of bronchial musculature and process of respiration.

(a) On Bronchial Musculature : (1) Epinephrine will cause relaxation of bronchial musculature. (2) If mild dose of epinephrine is administered (1 ml of 1 in 1000), there will be dilatation of the bronchial passage leading to increased ventilation.

(b) On Respiration : (1) Sudden intravenous administration of epinephrine in anaesthesized cat causes temporary cessation of breathing due to rise of blood pressure and it is called *adrenaline*

apnoea. (2) It facilitates pulmonary ventilation by relaxation of the bronchi. (3) Respiratory quotient rises and therefore oxygen consumption rises by 20 to 40 % and carbon dioxide production by 30 to 50%.

IV. On Gastro intestinal System : Epinephrine act on the glands of gastro-intestinal tract like salvary glands, glands of stomach and intestine, liver, gall bladder and spleen.

(a) On Salivary Gland : Epinephrine inhibits secretion of the digestive glands and it will stimulate the secretion of the salivary gland. The secretion obtained from salivary glands is thick and scanty. It will stimulate profuse watery secretion and total volume of salivary secretion is increased.

(b) On Stomach and Intestine : Epinephrine inhibits the gastric and intestinal glands and there will be no secretion of gastric and intestinal juice. This hormone inhibits the peristaltic movement of stomach and intestine and increases the tone of the sphincters.

(c) On Liver : Epinephrine stimulates the process of glycogenolysis and thereby liver glycogen is converted into glucose which will circulate in the blood to produce hyperglycaemia and glycosuria. The content of liver glycogen is diminished.

(d) On Gall bladder : Epinephrine produces relaxation of the plain muscles of the gall bladder with construction of sphincter of Oddi. The present conception is that the hormone produces contraction of the gall bladder to help in the expulsion of the bile from the gall bladder.

V. On Spleen : Epinephrine produces contraction of the plain muscles lining the trabeculae of the spleen and the stored blood comes out of the spleen and circulates in the blood stream.

VI. On Excretory System : Epinephrine effects on kidney, urinary bladder, skin and sweat glands.

(a) On Kidneys : (1) The small dose of epinephrine produces constriction of the efferent renal arterioles but no effects on afferent renal arteriole and as a result, urinary output is increased. (2) Large doses of epinephrine produce constriction of both afferent and efferent renal arterioles, filtration through the glomerulus is greatly diminished and urinary output is much reduced.

(b) On Urinary bladder : Epinephrine produces relaxation of the detrusor muscles of the urinary bladder with the constriction of the internal urethral sphincher.

(c) On Skin : Epinephrine will cause eonstruction of the arrector pili muscles and as a result there will be erection of the hairs.

(d) On Sweat glands : The sweat glands in the axilla and groin are activated by epinephrine content of plasma. The sweat secretion of the horse depends on catecholamine concentration of plasma. The apocrine glands secrete thick, odoriferous secretion as a result of sympathetic stimulation and they are controlled by

adrenergic fibres rather than by cholinergic fibres and are controlled by sympathetic centres of the central nervous system rather than by the parasympathetic centres.

VII. On Metabolism : Epinephrine has got some effect on general metabolism. carbohydrate and fat metabolism.

(a) On General Metabolism : (1) The basal metabolic rate is increased about 20% by moderate doses of epinephrine and large doses cause a fall of basal metabolic rate. (2) Respiratory quotient is raised due to increased cardiac output upto 30 to 40%. Oxygen consumption is increased upto 20 to 40% of the basal level and carbon dioxide production is increased by 30 to 50%. (3) Epinephrine is calorigenic and heat production is immediate but comes to normal level quickly.

(b) On Carbohydrate Metabolism : (1) Epinephrine or norepinephrine raises the blood sugar level. Blood glucose is converted to muscle glycogen which is converted to lactic acid. Blood lactic acid is converted to liver glycogen. (2) Epinephrine augments glycogenolysis, glycogenesis and glycolysis. Epinephrine to some extent also causes neoglucogenesis. (3) It mobilizes liver glycogen and provides sugar for the active tissues. (4) Epinephrine stimulates the process of glycogenolysis. The blood sugar level will rise and there may be glycosuria when the blood sugar level exceeds the renal threshold value of glucose. (5) Epinephrine may stimulate hepatic gluconeogenesis by acting as an inducer for key gluconeogenic enzymes like pyruvate carboxylase, FDP-ase and glucose-6-phosphatase.

(c) On Fat Metabolism : (1) Catecholamines activate the hormone-sensitive lipase in the adipose tissuse and increases lipolysis thereby β-effect and activate CAMP mechanism. This raises the blood free acid level whose oxidation increases energy production in cardiac and skeletal muscles during stress. (2) It mobilizes of fat from depot by enhancing lipase action. (3) It increases fatty acids and glycerol level in blood. Both epinephrine and norepinephrine are equally potent in the mobilization of FFA from the adipose tissue.

VIII. On Endocrine Glands : Epinephrine has got effect on anterior pituitary and pars intermedia. (1) Epinephrine stimulates anterior pituitary to secrete more ACTH which again helps in the release of glucocorticoid hormones after stimulating the supra-renal cortex. Glucocorticoids increase blood sugar level through gluconeogenesis process. (2) This hormone acts on melanophores. The effects of MSH on the dispersion of melanin granules within melanophores are antagonised by catecholamines through adrenergic α-receptors. It causes contraction of melanophores of toad and frog.

IX. On Nervous System : (1) Large dose of epinephrine has got direct depressant action on the spinal cord and muscle tone and the reflexes will be diminished. (2) It causes increase or decrease of conductivity of nerve impulse. (3) Epinephrine stimulates

ascending reticular activating system (ARAS) causing constant wakefulness, irritability and anxiety. (4) Injection of epinephrine into the ventricles of brain causes depression, coma with loss of reflexes. Increased alertness is the usual response. During an emergency which provokes a generalised sympathetic response, the usefulness of increased mental alertness for fight or flight is obvious.

Different Systems	*Epinephrine*	*Norepinephrine*
I. Cardiovascular System :		
(a) *Blood* ;		
Eosinophil count.	Increased.	No effect.
(b) *Heart* :		
(1) Heart rate.	Increased.	Decreased.
(2) Cardiac output.	Increased.	Decreased.
(3) Pulse rate.	Increased.	Decreared.
(4) Blood pressure :		
(i) Systolic Pressure.	Increased.	Increased.
(ii) Diastolic Pressure.	No effect	Increased.
(iii) Mean Arterial Pressure.	Decreased.	Raised.
(c) *Blood vessels* :		
(1) In denervated limb.	Vasodilatation.	Vasoconstriction.
(2) Coronary vessels.	Vasodilatation.	Vasodilatation.
(3) Net peripheral vascular effect.	Vasodilatation.	Overall vasoconstriction.
(4) Peripheral resistance.	Decreased.	Increased.
(d) *Blood flow through organs* :		
(1) Skeletal muscle.	100% increase.	Unaltered or decreased.
(2) Liver.	100% increase.	No material effect.
(3) Kidney.	40% decrease.	20% decrease.
(4) Brain.	20% increase.	Slightly decrease.

Different Systems	*Epinephrine*	*Norepinephrine*
II. Respiratory System :		
(1) Process of respiration.	Stimulated.	Stimulated.
(2) Bronchial muscle.	Inhibition.	Inhibition.
III. Metabolism :		
(1) Oxygen consumption.	Increased.	Increased.
(2) Blood sugar level.	Increased.	Increased.
(3) Free fatty acid release.	Increased.	Increased.
(4) Glycogenolysis.	Decreased.	Increased.
IV. Intestine :		
(1) Small Intestine.	Inhibition.	Inhibition.
(2) Large Intestine.	Inhibition.	Inhibition.
V. Central Nervous System :	Increased mental anxiety.	No effect.
VI. Eye : Dilator pupillae.	Excitation.	Excitation.
VII. Reproductive System :		
Non-pregnant uterus in rat or cat.	Inhibition	Inhibition.

Noradrenaline or Norepinephrine

1. Noradrenaline is secreted by ***adrenal medulla.***
2. Chemically, it is a ***catecholamine*** and is closely related to ***tyrosine*** and ***phenylalanine.***
3. It is the immediate precursor of adrenaline.
4. Like adrenaline, noradrenaline is also secreted under emergency condition. Hence, it is called an ***emergency hormone.***
5. Most of its actions are similar to those of adrenaline. Some actions are just the reverse of adrenaline.
6. It slightly increases the heart beat and the cardiac output is not increased.
7. It increases blood pressure.
8. It stimulates the constriction of blood vessels.
9. Respiratory rate is increased.
10. Metabolic rate is increased.
11. Blood sugar level is raised.