Question bank- paper No.11

Drugs affecting metabolic hormones

I.Name the following:

1.A condition of jaw produced as a result of chronic imbalance of calcium and phosphorus in the diet.—("Rubber jaw")

2.A Condition in which excess production of parathormone occurs.-(parathyroid hypertrophy, "Rubber jaw", osteitis fibrosa cystica)

3.A disease in which glucocorticoid is used for curative treatment.-(Addisson's disease)

4.A 5HT receptor antagonist.-(cisapride)

5.A growth hormone receptor antagonist.-(pegvisomant)

6.A growth hormone releasing hormone analogue.-(sermorelin)

7.A hormone which stimulate blood glucose level.-(glucagon)

8.A hypolipidaemic agent that causes the release of vasopressin from hypothalamus.-(clofibrate)

9.A hormone antagonize the action of parathormone.—(calcitonin)

10.Aminoacid from which adrenaline is synthesised.-(Phenyl alanine/tyrosine)

11.Aminoacid from which histamine is synthesised .-(histidine)

12.Aminoacid used for the synthesis of melatonin.-(tryptophan)

13.An insulin like growth factor-1 analogue.-(mecasermin)

14.An anticonvulsant drug that stimulate release of vasopressin from hypothalamus.-(carbamazepin)

15.A non selective vasopressin receptor antagonist.-(conivaptan)

16.An imidazole derivative anti fungal antibiotic which inhibits steroid synthesis from cholesterol.-(Ketoconazole)

17.An insulin which have ultra long action.(40 hours). –(Insulindegludec)

18.An anti thyroid agent which inhibits the synthesis of T3 and T4.—(methimazole)

19.A recombinant human insulin analog which will act for 24 hours.—(insulin glargine)

20.A selective vasopressin V1A receptor antagonist .-(relcovaptan)

21.A selective vasopressin V1B receptor antagonist.-(nelivaptan)

22.A selective vasopressin V2 receptor antagonist .-(tolvaptan, mozavaptan)

23.A synthetic thyrotropin releasing hormone . –(protirelin)

24.A synthetic analogue of oxytocin .-(carbotoxin)

25.A sulphur containing imidazole derivative antithyroid drug. (methimazole,(thiamazole)

26.A vegetable having antithyroid activity.—(cabbage)

27.Disease caused by adrenal cortical hypofunction.-(Addison's disease)

28.Drugs which destroy thyroid tissue .-(radioactive iodine 131, 123, 125)

29.Drugs inhibiting thyroid hormone release .-(iodide of sodium and potassium, organic iodine)

30.First preparation used to treat diabetes ,other than insulin.-(tolbutamide)

31.Give two example for sulfonyl ureas —(glipizide, glyburide, glimepiride)

32.Hormones secreted by parathyroid.-(parathormone, calcitonine)

33.Most common naturally occurring corticosteroids.-(cortisole)

34.Most potent glucocorticoid.-(flumetasone)

35.Most potent mineralocorticoids.-(aldosterone)

36.One adrenal cortical steroid inhibitor.—mitotane)

37.One alpha glucosidase inhibitor.-(acarbose)

38.One short acting corticosteroid .-(hydrocortisone, cortisone)

39.One slow onset long duration oral glucocorticoids.-(dexamethazone, betamethazone,flumethazone)

40.One rapid onset short duration oral glucocorticoid.-(hydrocortisone, prednisone, methyl prednisone)

41.One rapid onset intermediate duration parenteral glucocorticoid.-(dexamethazone)

42.One slow onset long duration parenteral glucocorticoid.-(triamcinolone, methyl prednisolone, flumethazone)

43.One first generation sulfonyl ureas.-(tolbutamide)

44.One second generation sulfonyl urea .-(glibenclamide, glipizide)

45.One synthetic analogue of somatostatin ,-(octreotide, lanreotide)

46.One biguanide used for the treatment of diabetes.-(metformin)

47.One thiazolidine diones group of anti hyperglycemic agent . –(piglitazone, ciglitazone)

48.One antifungal agent which inhibits adrenal steroidogenesis.—(ketoconazole)

49.One synthetic drug which reduce insulin release from Beta cells.-(diazoxide)

50.One diuretic which increase blood glucose level .-(thiazides)

51.The most potent form of thyroxin used as injection –(liothyronin)

52.The hormone secreted by parafolicular C cells of thyroid which is used in treating hypocalcemia.-(Parathormone)

53.The mineralo corticoid by the adrenal cortex .-(aldosterone)

54.The gland which secrets parathormone.-(parathyroid)

55.The most potent form of thyroxin.-(Tri –iodo thyronine)

56.The most potent physiological anabolic agent-(Insulin)

57.Three adreno corticosteroid inhibitors.—(mitotane, ketoconazole, trilostane, selegiline)

58.Two anti thyroid thionamides-(thiourea, thiouracil)

59.Two anti thyroid ionic inhibitors.-(perchlorates and thiocynates)

60.Two vegetable which blocks iodine trapping by thyroid.-(cabbage, soyabean)

61.Two intermediary acting corticosteroid.-(prednisone, triamcinolone)

62.Two long acting corticosteroids. (betamethasone, flumethazone)

63.Two steroids used for the treatment of mineralo corticoid deficiency.-(desoxy corticosterone, fludrocortisone)

64.Two biguanides used in the treatment of diabetes mellitus.-(metformin and phenformin)

65.Two drugs used in the diagnosis of growth hormone deficiency.-(alpha 2 adrenoceptor agonist like clonidine, xylazine)

66.Two first generation sulphonyl ureas.-(chlorpropamide, tolazamide, carbutamide)

67.Two glucagon like peptide –I analogues.-(exenatide, liraglutide)

68.Two glucose transporter protein inhibitors.-(canagliflozin, dapagliflozin)

69.Two biguanides used in diabetes.-(metformin, phenformin)

70.Two drugs used to counteract the action of excess insulin.-(glucagon, octreotide)

71.Two alpha glucosidase inhibitors used as antidiabetes agent .-(acarbose, miglitol, voglibose)

72.Two natural glucocorticoid .—(hydrocortisone, deoxycorticosterone)

73.Two intermediary acting insulin preparation.-(globin zincinsulin, Isophan insulin.)

74.Two CRH -1 antagonist .-(antalarmin, pexacerfont)

75.Two drugs inhibiting iodine trapping .-(thiocynate, perchlorate)

76.Two dopamine agonist.-(bromocriptine, pergolide)or epinephrine)

77.Two long acting preparation of insulin .-(ultra lente insulin, protamin zinc insulin)

78.Two somatomedin .-(sonmatomedin C, IGF-1)

79. Drugs used to differentiate pseudo hypoparathyroidism from true hypoparathyroidism.-(teriparatide)

II. Fillup the blanks with most appropriate words:

1.After insulin therapy in diabetes patients hyper kalemia will ……..—(disappears /reduce)

2.Aldosterone is not available as a pharmacological agent because of its ……..of action .—(short duration)

3.Adrenocorticotropic hormone is secreted by ……..cells of anterior pituitary.-(Basophilic)

4.Adrenal cortical hypofunction causes.……disease.-(Addisons)

5.Among animals ,hypothyroidism is mostly seen in ……and horses.—(dogs)

6.Among animals hyperthyroidism is usually seen in ……—(cats)

7.Among short acting glucocorticoids .....is having high mineralo corticoid and glucocorticoid potency.—(fludrocortisone)

8.Arginin vasopressin is interacting primarily with ..... receptor.-(V2)

9.Biosynthetic insulin is manufactured originally with the help of a genetic engineered strain of ..... (E coli)

10.Biosynthesis and secretion of calcitonin is determind mainly by the plasma .....concentration.-(calcium)

11.Feline and ……insulin are similar in structure. –(bovine)

12.Globin zinc insulin will act for .....hrs.-(24)

13.Glucocorticoids increases the number of circulating erythrocyte and neutrophil and decreases the number of circulating……—(lymphocytes and eosinophils)

14.Glucocrticoid secretion is mainly controlled by ACTH and secretion of mineralo corticoid is controlled mainly by .....-(electrolytes in ECF and blood volume)

15.Glucocorticoids reduce protein stores in all body tissues except .....-(Liver)

16.Hair and skin growth is …… by glucocorticoids.—(inhibited)

17.Hyperfunction of adrenal cortex causes.....disease.-(cushings)

18.Hyperthyroidism is seen mostly in ……(animal) which will respond to treatment.—(cats)

19.Hypo adrenocorticism is otherwise known as .....—(Addisons disease)

20.Hypoparathyroidism is rarely seen in .....breeds of dogs.-(Terriers/ Schnauzers)

21.Hyposecretion of mineralocorticoid results in .....disease.-(Addison)

22.Hyper production of mineralocorticoids cause.....syndrome.-(Conns)

23.Hyper adrenal cortism is otherwise known as .....--(Cushings syndrome)

24.1, 25-(OH)2 D3 is called as .....—(Calcitriol)

25.In 1915 .....isolated the active principle from thyroid gland.-(Kendal)

26.Injectable water insoluble preparations of corticosteroids are in .....form or, pivalate , acetonide form.—(acetate)

27.In dogs and cats type ……diabetes is more .—(two)

28.In most animals GH is having anti insulin action,over production of GH after epiphyseal line closure results in .....(Acromegaly)

29.Insulin binds to its receptors and stimulates .....kinase.—(tyrosin)

30.Insulin secretion is inhibited by somatostatin and ……--(galanin)

31.Insulin was isolated by .....and.....in 1922.-(Banting and Best)

32.Intermediary acting corticosteroids have a biological half life of ……hours.—(12—36)

33.Isophane insulin (NPH) is an ……..(duration) acting insulin.—(intermediate)

34.Large dose of adrenaline may cause ……of the area of injection.-(necrosis)

35.Adrenaline causes conversion of ……in to glucose and energy.-(glycogen)

36.Levothyroxin contain ……..form of thyroxin.—(T4)

37.Liothyronin contain ……..form of thyroxin.—(T3)

38.Lysine vasopressin is interacting primarily with …… receptors.-(V1)

39.Long acting preparation of corticosteroids have a biological t ½ of ……..hours.—(36—72)

40.Mineralocorticoids got only …… duration of action in replacement therapy .-(very short)

41.Deoxycorticosterone and……are used for replacement therapy.(fludrocortisones)

42.Out of prednisone and prednisolone ,……..is the biologically active form.—(prednisolone)

43.Out of cortisone and cortisol ,……..is the biologically active form.—(cortisol)

44.Over production of GH before line epiphyseal closure results in ……(Gigantism)

45.Panhypopituitarism is otherwise known as ……(Simmonds disease)n.-(T)

46.Parathormone is not active by ……..route.-(oral)

47.Parathyroid hormone along with ……..and……regulate calcium homeostasis in the body .-(calcitonin and vitamin D)

48.Parathyroid glands of adult of many species contain mainly chief cells and moderate number of ……cells .-(oxyphil)

49.Parathormone secretion is regulated by circulating level ……..ions in blood .-(calcium)

50.Parathormone enhance tubular reabsorption of calcium and decrease tubular reabsorption of ……(.phosphate)

51.Protamine zinc insulin preparation consist of 90% bovine insulin and 10% ……insulin.—(porcine)

52.Protamine zinc insulin will act for ……hours.—(48)

53.Primary hypoadrenocorticism (Addisons disease) is usually due to auto immune disease and

54.Secondary hypoadrenocorticism is due to a……secretion.—(reduction in ACTH)

55.Primary hypo adrenocorticesm is mainly due to ……..disorders.—(auto immune)

56.Renin is produced by ……apparatus of kidney.-(Juxta glomerular)

57.Secondary hypo adrenocorticesm is due to reduction in ……secretion.—(ACTH)

58.Selegilin inhibits ACTH secretion by increasing ……concentration.—(Dopamine)

59.Semilente insulin will act for ……hrs.-(12 ---16)

60.Selegilin increase the dopamine concentration by inhibiting the metabolism by.....—(MAO-B)

61.Short acting preparation of corticosteroids have a biological t ½ of less than .....hours.—(12)

62.Since Calcitriol serves as a growth factor to promote differentiation of skin cells , it is recommended in the treatment of primary idiopathic.....in small animals.—(seborrhoea)

63.Steroid hormones are derivative of .....-(cholesterol)

64.Succinate and phosphate salt of corticosteroid preparations are.....soluble and hence can be given i/v.-(water)

65.The metabolites of selegiline .....and..... produce stimulation of CNS.-(methamphetamine , L amphetamine)

66.The growth hormone receptors are associated with Janus kinase (JAK) a cytosolic form of.....—(tyrosine kinase.)

67.The action of isophan insulin occurs in 2 hours and last for.....hours.—(24)

68.The physiologically active form of thiamine is .....-(Thiamine pyrophosphate)

69.The prodrug of thiamazole is .....(carbimazole)

70."The duration of action of lente insulin is .....hours.—(24)

71.The t ½ of Na 131 I is .....days.—(8)

72.Thyroxin is produced from the aminoacid .....-(Tyrosin)

73.Thyroxin is stored in thyroid gland as .....-(Thyro globulin)

74.Trilostane is not recommended in pregnant animals since it inhibits the .....synthesis.—(Progesterone)

75.Type one diabetes is otherwise known as .....diabetes.-(insulin dependent)

76.Under production of growth hormone in immature animals results in .....(Dwarfism)

77.Vetsulin contain .....insulin for use in dogs.—(lente porcine)

78.....is the preferred drug for the treatment of hypo adrenocorticism.—(flurocortisone)

79..... is used to treat pituitary dependent hyper adrenocortism in dogs.—(Selegilin)

80.....insulin is the fast acting one among globulin zinc insulin, isophane insulin, and crystalline insulin.-(crystalline)

III. STATE TRUE or FALSE

1.Acetyl choline inhibits insulin secretion.—(F)

2.Acetyl choline stimulate insulin secretion by activating M-3 receptors (coupled to Gq).-(T)

3.ACTH receptors are coupled to the Gs adenyl cyclase system—(T)

4.Adrenocorticotropic hormone is polypeptide in nature.-(T)

5.Administration of glucocorticoids causes ‘ moon face”,deposition of fat in cheeks.-(T)

6.Administration of glucocorticoids causes “ buffalo hump”- alteration of fat distribution in neck region.-(T)

7.Adrenocorticotropic hormone is not effective orally as it is digested in the GI tract.-(T)

8.Adrenocorticotropic hormone is effective only when a functional cortex is present.-(T)

9.Aldosterone secretion is stimulated by high plasma angiotensin , ACTH, or K+ concentration.—(T)

10.Aldosterone is recommended in chronic interstitial nephritis .-(T)

11.Aldosterone stimulate the mRNA synthesis of Na + channels, K+ channels and H+ ATPase (in the apical cell side).—(T)

12.All tissues can remove iodine from the thyroid hormone.-(T)

13.Aldosterone is available only for research purpose not for therapeutic use.-(T)

14.Aldosterone increase the synthesis of Na+ K+-ATPase and HCO_3 Cl- antiport in the distal renal tubule and intestine.-(T)

15.Alpha-2 adrenoceptor agonist can be used in the diagnosis of growth hormone deficiency.-(T)

16.Amylin , a peptide hormone from Beta cells contribute to glycaemic control. (T)

17.Amylin is deficient in diabetes patient ,which contribute to glycaemic control.-(T)

18.Antalarmin is a non peptide drug that reduces the release of ACTH.-(T)

19.Antalarmin is a corticotrophin releasing hormone receptor antagonist.-(T)

20.Anions such as nitrate, thiocyanate and perchlorate inhibits NaI uptake by competing with iodide for active transport.-(T)

21.Atosiban blocks the contractile effect of oxytocin on myometrium.-(T)

22.Atosiban is used mainly as a tocolytic agent.-(T)

23.Because of prolonged action of protamine zinc insulin it is convenient for use in veterinary practice.—(T)

24.Biguanides are not acting through Beta cells.-(T)

25.Biguanides are effective in unstabilized form of diabetes.-(T)

26.Biguanides will act through Beta cells.-(F)

27.Biguanides are insulin sensitizers.-(T)

28.Brain cells and adrenal medullary chromaffin cells require insulin for glucose transport.-(F)

29.Broccoli and cabbage is having antithyroid activity.-(T)

30.Bromocriptine can be used in case of excess growth hormone production.-(T)

31.Bromocriptine is used as a dopamine agonist and prolactin inhibitors.-(T)

32.Cabage has an antithyroid activity in rabbits.-(T)

33.Calcitriol is used in small animals to treat hypo calcaemia due to hypo parathyroidism or severe renal disease.-(T)

34.Calcitonin is released in response to hypercalcemia.-(T)

35.Calcitonin is secreted by the parafolicular C cells of parathyroid.-(F)

36.Carbamazepine , an antiepileptic drug and clofibrate an antihyperlipidemic drug are effective in some cases of central diabetes insipidus.-(T)

37.Canagliflozin and dapagliflozin are inhibitors of sodium glucose transport protein that are responsible for absorption of glucose from the intestine and reabsorption from renal tubule.-(T)

38.Cats , rabbits, rodents and birds do not have detectable level of thyroxine binding globulin.—(T)

39.Chronic imbalance in calcium phosphorus intake may cause secondary parathyroidism.-(T)

40.Corticotrophin releasing hormone receptors are coupled to Gq receptors.(T)

41.Corticosteroid is having anti inflammatory action.-(T)

42.Corticosteroids with or without antibiotics are contra indicated in the initial treatment of corneal ulcers.-(T)

43.Corticosteroids temporarily alleviate hypoglycaemia in ketotic cow.-(T)

44.Corticosteroids are produced from acetate and cholesterol.-(T)

45.Corticosteroids are readily absorbed from the GI tract and mucous membrane and skin.-(T)

46.Crystalline insulin is fast acting and long acting insulin.-(F)

47.Cyproheptadine is an antihistaminic with serotonin receptor agonist and calcium channel blocking properties.-(T)

48.Deoxycorticosterone is produced by the adrenal cortex in response to ACTH stimulation.—(T)

49.Dextro form of nor adrenaline is inactive.-(T)

50.Deficiency of thyroxin causes myxedema in young ones and cretinism in adults.-(F)

51.Deficiency of thyroxin causes cretinism in young ones and myxedema in adults.-(T)

52.Desoxy corticosterone acetate is a mineralocorticosteroids.-(T)

53.Desmopressin acetate is an ADH analog.-(T)

54.Dihydrotachysterol is used in small animals to treat hypocalcemia secondary to hypoparathyroidism or severe renal disease.-(T)

55.Dietary fibre and phytase stimulate the absorption of calcium from the small intestine.(F)

56.Dogs have more thyroxin binding globulin than humans.-(F)

57.Drugs that compete for protein binding site decrease free T3 and T4 level.-(F)

58.Elcatonin is a calcitonin derivative used as an anti parathyroid agent.-(T)

59.Epinephrine and nor epinephrine inhibit insulin secretion by activating Alpha-2 adrenergic receptors (coupled to Gi/o).-(T)

60. Erythropoietin,which promote erythropoisis is produced in the liver.-(F)

61.Exenatide is similar to glucagon like peptide -1 which is involved in pancreatic insulin secretion after ingestion of food.-(T)

62.Ergonovine is an ergot alkaloid causing prolonged contraction of smooth muscles including the myometrium and blood vessels.-(T)

63.5' Deiodinase enzyme require trace quantities of selenium for optimum activity.-(T)

64.Fine control of hyper glycemia is difficult with long acting preparation like lente insulin.—(T)

65.For feline use, PZI- vet insulin preparation is available. —(T)

66.For the control of summer cough in dogs steroids can be used.-(T)

67.Discontinuationof the prolonged treatment with corticosteroid must be tapered for 2-3 weeks.-(T)

68.Globin zinc insulin may produce hypersensitivity.-(T)

69.Globin zinc insulin is an intermediary acting preparation.-(T)

70.Glucagone causes break down of glycogen to glucose.-(T)

71.Glucagon release is stimulated during hypoglycaemia and inhibited during hyperglycaemia.-(T)

72.Glucocorticoids increases susceptibility to infection by inducing suppression of immunity. —(T)

73.Glucose enter the Beta cells through the glucose transporter (GLUT-2).-(T)

74.Glucocorticoids increase liver glycogen synthesis and storage and stimulate gluconeogenesis and hyperglycemia.-(T)

75.Glucocorticoids increases the number of lymphocytes in the system .-(F)

76.Glucocorticoids increase the expression of alpha adrenergic receptors in vascular smooth muscles and beta adrenergic receptors in the myocardium.-(T)

77.Glucocorticoids inhibit the virus induced interferon production.-(T)

78.Glucocorticoids stimulate the CNS leading to euphoria.—(T)

79.Glucocorticoids stimulate protein catabolism and cause a negative nitrogen balance-(T)

80.Glucocorticoids causes an inhibition of antibody production.--(T)

81.Glucocorticoids stabilizes lysosomes , prevent escape of enzymes and reduce inflammation.-(T)

82.Glucocorticoids are having anti allergic action.-(T)

83.Glucocorticoides causes bronchodilation by increasing the expression of Beta 2 receptors—(T)

84.Glococorticoid deficiency cause weakness of skeletal muscles and long term use may cause wasting of skeletal muscles-(T)

85.Glucocorticoids suppress immunity by decreasing cytokine production. —(T)

86.Growth hormone is a sulphur containing protein.-(T)

87.Growth hormone stimulate the growth of osceous tissue but not of soft tissue.-(T)

88.Growth hormone administration have the side effect of diabetes mellitus and ketosis.-(T)

89.Growth hormone has direct effects on lipid and carbohydrate metabolism its anabolic and growth promoting effects are mediated indirectly by somatomedins.-(T)

90.Growth hormone releasing hormone is also known as somatocrinin.-(T)

91.Growth hormone and placental lactogen can be used in deficiency of prolactin.-(T)

92.Hirsutism is one of the adverse effect associated with glucocorticoids.-(T)

93.High plasma thyroxin binding globulin level give long duration of action.-(T)

94.Hypoparathyroidism is seen mostly following parturition.-(T)

95.Hormones of polypeptide and glycoproteins in nature are well absorbed from the GI tract.-(T)

96.Hormones of steroid in nature are well absorbed from the gut.-(T)

97.Humulin is an NPH insulin .-(T)

98.Humen have four times as much of Tyroid binding globulin than dogs.-(T)

99.Human erythropoietin is produced in the kidney and promote erythropoiesis.-(T)

100.In diabetic patients hyperkalemia is seen.—(T)

101.In Hypothyroidism levothyroxine once daily is recommended because of its long half life.-(T)

102.In dogs diabetes mellitus is the major side effect of somatotropin administration.-(T)

103.In growing animals thyroxin deficiency causes permanent damage to nerves.-(T)

104.In adults the damage to nerves due to thyroxin deficiency is reversible.-(T)

105.In lactating animals calcitonin will inhibit lactation.-(T)

106.Isophan insulin is otherwise called as NPH insulin.-(T)

107.Insulin is a protein which will be digested if administered orally.-(T)

108.Insulin is necessary to facilitate transport of glucose across the plasma membrane of all cells in the body.-(F)

109.Insulin glargine and insulin detemir need to administer once daily.-(T)

110.Insulin reduces the potassium level of serum.-(T)

111.Insulin assists the protein synthesis.-(T)

112.Insulin is a powerful antiketogenic agent in cattle.-(T)

113.Increase secretion of aldosterone can cause rickets.-(T)

114.Increase secretion of aldosterone can cause obesity.-(T)

115.In acute lymphocytic leukemia corticosteroid is effective .-(T)

Induced

116. Parturition with steroids often accompanied by high incidence of retained placenta.-(T)

117.Insulin resistance is more seen in dogs than in cats.-(F)

118.Insulin resistance is more likely to develop in cats given human or porcine insulin preparation.-(T)

119. In the absence of thyroid stimulating hormone thyroid will not function at all .-(F)

120.In the presence of Alpha-2 receptors blockade epinephrine and norepinephrine stimulate

121.Insecticides like DDD (dichloro diphenyl dichloro ethane) destroy adrenal cortex .-(T)

122.Iodine deficiency causes endemic goitre.-(T)

123.Isophane insulin contain small amount of protamin .—(T)

124.Ipodate is an iodine containing compound that inhibits the conversion of T4 to T3 by binding to iodinases.—(T)

125.Immunosuppressive action of corticosteroid is more on cell mediated immunity than humoral immunity.-(T)

126.Ipodate is useful in feline hyperthyroidism eventhoug, primarily this is a radiographic agent.-(T)

127.Ketocinazole inhibits cytochrome P 450 that are involved in steroidogenesis in adrenal cortex.—(T)

128.Ketoconazole inhibit adrenal steroidogenesis.-(T)

129.Ketoconazole is not recommended in rats as they are deficient in phase I metabolic enzymes.-(T)

130.Ketoconazole causes reproductive disturbances as it inhibits the synthesis of all steroids.—(T)

131.Lysosomal membranes are stabilized by glucocorticoids.—(T)

132.Liothyronin is more active than levothyroxin.-(T)

133.Liraglutide , another glucagon like peptide used to treat type-2diabetes.-(T)

134.Lanreotide is primarily used for the treatment of acromegaly it can inhibit the release of thyroid releasing hormone, insulin and glucagon.-(T)

135.Lente insulin is addition of zinc in acetate buffered insulin.—(T)

136.Levo form of noradrenalin is more active than dextro form .-(T)

137.Liver cells depend on insulin for glucose transport to hepatic cells.-(F)

138.Long therapy with corticosteroid causes atrophy of adrenal cortex.-(T)

139.Loss of hair is a symptom of hypervitaminosis A .-(T)

140.Meglitinide is a non sulfonyl urea secretagogues used to treat type-1 diabetes.-(F)

141.Metformin is a hypoglycaemic drug.—(F)

142.Metergoline is an ergot derivative with dopamine agonist action.-(T)

143.Methimazole and carbimazole block the thyroxine synthesis and secretion.-(T)

144.Metformin is an antihyperglycemic agent.-(T)

145.Metformin stimulate insulin secretion.-(T)

146.Metformin reduce hepatic glucose output and improve peripheral sensitivity to insulin.-(T)

147.Methimazole can be used to treat hyperthyroid cats.-(T)

148.Most compound with antithyroid activity are inhibitors of thyroid peroxidise enzyme.-(T)

149.Natural glucocorticoids are short acting compound because of its rapid metabolism by liver.—(T)

150.Natural steroids are rapidly metabolised by liver enzymes.—(T)

151.Octreotide is a drug used in excess growth hormone production and is approved for the treatment of acromegaly.-(T)

152.Pancreatitis is one of the adverse effect associated with glucocorticoids.-(T)

153.Parathyroid hypertrophy condition is referred as "Rubber jaw"-(T)

154.Para amino benzoic acid deficiency cause greying of hairs.-(T)

155.Parathyroid hormone receptors are located mainly on the cell surface of osteoblats and renal epithelial cells.-(T)

156.Primary hyperparathyroidism is mainly due to abnormality of parathyroid.-(T)

157.Pregnancy toxaemia is not well respond to steroid.-(T)

158.Protamin zinc insulin will act for 36-48 hours.-(T)

159.Prolactin receptors are also present in T lymphocytes and has been shown to modulate immune response.-(T)

160.Porcine and canine insulin molecules are identical and are similar to human insulin.-(T)

161.Rape seed contain glucosinolate which is metabolised to thicyanate , an inhibitor of thyroid iodide uptake.-(T)

162.Radioactive iodine (Na 131 I) can be used to destroy thyroid follicle.—(T)

163.Replacement therapy with steroid is only recommended in addisons disease.-(T)

164.Repaglinide is a hypoglycaemic drug used to treat type2 diabetes.-(T)

165.Rubber jaw is a condition due to hypertrophy of parathyroid.-(T)

166.Rise in plasma calcium increase the synthesis and secretion of calcitonin.-(T)

167.Ruminants are more depended on insulin glucagon mechanism for energy production. –(F)

168.Ruminants can synthesise their requirement of vitamin B12 if cobalt is included in their diet.-(T)

169.Secondary hyperparathyroidism is mainly nutritional in origin.-(F)

170.Secondary hyper parathyroidism is due to chronic renal disease or chronic imbalance in calcium phosphorus intake.-(T)

171.Somatomedins are otherwise called as insulin like growth factor.-(T)

172.Steroid therapy increases the milk yield in cattle.-(F)

173.Steroid therapy causes suppression of infectious diseases.-(F)

174.Steroid therapy causes flaring up of infectious diseases.-(T)

175.Sugar is more sweeter than saccharin.-(F)

176.Sulfonyl ureas stimulate insulin secretion from Beta cells.-(T)

177.Sulphonamide has an anti thyroid activity in rats.-(T)

178.Sulfonyl ureas has no effect on insulin release from Beta cells.-(F)

179.Sulfonyl ureas stimulate the pancreas to secrete insulin –(T)

180.Sulphonyl ureas increase the insulin synthesis.-(F)

181.Sulfonyl ureas will be effective only when functional Beta cells are present ._(T)

182.Sulphonyl ureas are very effective in type I diabetes.-(T)

183.Swine can synthesise their own requirement of cyanocobalamine.-(F)

184.Synthetic steroids can be effectively administered orally.—(T)

185.Teriparatide is used in the treatment of osteoporosis.-(T)

186.Tetracosactide isa synthetic peptide with pharmacological action similar to ACTH.-(T)

187.The oxyphil cells are absent in parathyroid glands of chicken and rats.-(T)

188.The release of aldosterone is regulated by renin-angiotensin system.-(T)

189.The glucose transport across plasma membrane of brain cell is not depended on insulin.-(T)

190.Thyroid hormone is well absorbed from the gut.—(T)

191.Thyroid hormone increase the number or sensitivity of Beta adrenoceptors in myocardium .-(T)

192.Thyroid gland release tri and tetra form of thyroxin in to blood.-(T)

193.Thyroxin increases the size and number of mitochondria in the cells.-(T)

194.Thyroxin is tetra iodothyronine.-(T)

195.Thioureylene drugs such as methimazole inhibits oxidation and coupling process and prevent thyroid hormone secretion.-(T)

196.Thiouracil interfere with the iodine trapping by thyroid gland.-(T)

197.Thiocyanates inhibit the synthesis of thyroxin.-(T)

198.Toxicity of STH causes diabetes mellitus.-(T)

199.The output of ADH is directly related to the degree of hydration of body.-(F)

200.The pressar effect of vasopressin is so much less than its effect as a diuretic.-(T)

201.T3 is 3—5 times more active than T4.—(T)

202.T4 is the prothyroid hormone.-(T)

203.Tolbutamide is effective in juvenile diabetes .-(F)

204.Tolbutamide is a first generation sulphonyl ureas.-(T)

205.Triamcinolone is used to treat bovine ketosis.-(T)

206.Trilostane is a synthetic steroid analogue inhibiting corticosteroid synthesis.-(T)

207.Trilostane competitively inhibit 3-Beta hydroxysteroid dehydroginase.-(T)

208.Trilostane is used to treat hyper adrenocorticism.-(T)

209.Type two diabetes is otherwise known as noninsulin dependent diabetes.-(T)

210.Vasopressin V2 receptors activation results in antidiuresis.-(T)

211.V1- receptors of ADH are found in renal tubule to mediate anti diuretic effect.-(F)

212.V2- receptors of ADH are found in renal tubule to mediate anti diuretic effect.-(T)

213.V1-receptors of ADH are found in vascular smooth muscles.-(T)

214.Zinc content is more in lente insulin than ultra-lente insulin.-(F)

IV.Choose the correct answers from the given ones.

1.A drug which inhibits the release of histamine is a) betazole b) cimetidine c) dimenhydrinate d) cromolyn sodium –(d)

2.ACTH is used as substitution therapy in a) primary adrenal deficiency b) secondary adrenal deficiency c) primary thyroid deficiency d) secondary thyroid deficiency .-(b)

3.ACTH is most effective in stimulating the secretion of : a) glucocorticoids b) adrenal androgenic steroids c) aldosterone d) nor adrenaline.-(a)

4.Cortisone is a) short acting b) intermediary acting c) long acting compound.—(a)
Corticotrophin is not active if given : a)intravenously b) intramuscular c) oral d) slow IV drip.-(c)

5.Corticosteroid a) increase gluconeogenesis b) reduce peripheral utilisation of glucose c) antagonise insulin d) hyperglycemia e) all the above.-(e)

6.Diabetes insipidus may respond to one of the following drugs a) Amiloride b) aminophylline c) chlorothiazide d) acetazolamide- (c)

7.Different hormones are chemically belong to the following groups a) Polypeptide and protein b) glycoproteins c) steroids d) all the above.—(d)

8.Following are some of the signal transduction mechanism through which many hormones act. a) Tyrosine kinase –receptors with tyrosine kinase activity –cytosolictyrosinekinase b) Guanyl cyclase c) GTP binding protein in the plasma membrane Gs, Gq, Gi/o. d) Intracellular receptors. e) all the above.—(e)

9.Diabetes insipidus can be a) due to absolute deficiency of ADH b) due to insensitivity of renal tubule to ADH c) both are true.-(c)

10.Following are the basic reason for insulin resistance. a) insulin antibody b) stress c) insulin receptors desensitization(down regulation) d) all the above.—(d)

11.Following are some of the functions of glucocorticoids a) stimulate liver glycogen synthesis and storage b) gluconeogenesis c) lypolysis d) redistribution of lipids e) all the above.—(e)

12.Following drugs are second generation sulfonyl ureas a) glibenclamide b) gliburide c) glipizide d) all the above.-(d)

13.Following agents suppress adrenal cortex a) thiourea b) amphenon-B, c) metyrapone, d)aminoglutethamide e) all the above.-(e)

14.Glucocorticoids a) reduce antibody production b) delays healing process c) reduce inflammation d) all the above.-(d)

15.Glucocorticoid chronic administration may cause a) micronodullar cirrhosis b) gastric ulceration c) colonic perforation d) all the above.-(d)

16.Glucagon stimulate a) glycogenolysis b) gluconeogenesis c) glycogen synthesis in the liver cells d) lypolysis in adipose tissue e) all the above .-(e)

17.Vasopressin V1 receptors activation results in a) vasoconstriction b) platelet aggregation c) glycogenolysis d) ACTH release and growth of vascular smooth muscles cells. e) all the above.-(e)

18.Growth hormone have the following pharmacological effect a) promote growth of bone, muscles, mammary glands b) it stimulate the uptake of amino acids in to cells and promote lypolysis c) it increase the milk production. d) promote the growth of epiphyseal plate. e) all the above.—(e)

19.Insulin secretion is increased by the following a) glucose b) n-butyric acid in ruminants c) gastrin d) glucagon e) all the above are correct.—(e)

20.Insulin secretion is inhibited by a) somatostatin b) galanin c) epinephrine d) nor epinephtrine e) all the above are correct.—(e)

21."Incretins" include a) gut glucagon b) secretins c) gastrin d) gastric inhibitory polypeptide e) all the above. –(e)

22.Insulin: a) it is a protein composed of21 amino acid in A chain and 30 amino acid in B chain b) facilitate the penetration of amino acids through the cell membrane. c) fructose does not require insulin for its metabolism d) all the above are correct.-(d)

23.In dogs corticosteroids are recommended in the treatment of a) arthritis b) summer cough c) pastular dermatitis d) all the above.-(d)

24.In horses corticosteroids are used in the treatment of a) laminitis b) gout c) ulcerative colitis d) all the above.-(d)

25.Inadequate iodine uptake causes: a) greater secretion of thyrotropin b) an enlarged thyroid c) increased vascularity d) all the above.-(d)

26.Metformin exert the action by a) decreasing glucose absorption from the G I tract b) decreasing glucose output from liver c) increasing insulin receptor sensitivity d) all the above.-(d)

27.One of the following is an intermediary acting glucocorticoid a) betamethazone b) fludrocortisone c) triamcinolone.—(c)

28.One of the following drug is antidiuretic in diabetes insipidus a) mersalyl b) theophylline c) chlorothiazide d) furosemide.-(c)

29.One of the following is not a long acting glucocorticoid a) fludrocortisone b)dexamethasone c) flumethasone d)betamethasone. —(a)

30.One of the following is a short acting corticosteroid a) hydrocortisone b) prednisone c) betamethasone.-(a)

31.Paramethasone is.....acting steroid compound, a) an intermediary b) short c) long .—(c)

32.Parathormone a) increase calcium re absorption from the bone b) increase the calcium re absorption from the kidney c) reduce the reabsorption of phosphorus d) all the above.—(d)

33.Parathormone causes a) hyperphosphaturia b) hypophosphatemia c) hyper calcemia d) hypo calcuria e) all the above.-(e)

34.Parathormone can be recommended in a) hypocalcemic tetany b) fluoride toxicity c) oxalate toxicity d) lead toxicity e) all the above .—(e)

35.Somatostatin (growth hormone releasing inhibitory hormone) producing neurons are seen a) within the CNS b) outside CNS in epithelial cells (D cells) of the gastric mucosa and small intestine c) Islets of Langerhans d) all the above.-(d)

36.The main actions of insulin are given below a) reduce the blood glucose level b) accelerate the hexokinase c) accelerate the conversion of glucose to glycogen d) inhibit glycogenolysis and gluconeogenesis e) all the above.-(e)

37.The parathormone secretion by parathyroid is regulated mainly by a) level of circulating calcium b) pituitary c) iodine in circulation d) all the above.-(d)

38.Glucagon stimulate a) glycogenolysis b)gluconeogenesis c) glycogen synthesis in the liver d) lypolysis in adipose tissue e) all the bove.-(e)

39.The essential function of vitamin A is a) maintain the integrity of epithelial cells b) role in retinal function c) labilise lysosomes d) all the above.-(d)

40.The following drugs are sulfonyl ureas group of oral hypoglycaemic agent a) tolbutamide, b) chlorpropamide c) acetohexamid e d) tolazamide e) all the above.-(e)

41.The important mode of action of antithyroid drugs are given below a) inhibition of iodine trapping like thiocyanate b) inhibition of iodination of thyroglobulin eg. Thionamide c) inhibition of coupling reaction eg. Carbimazole d) destruction of thyroid tissue by 131 I.-e) all the above.-(e)

42.The release of aldosterone is regulated by a) renin-angiotensin system b) sodium level in the body c) thyroxin level in the body -(a & b)

43.The main function of aldosterone is a) increase gluconeogenesis b) increase excretion of potassium, calcium, phosphorus c) increase retention of sodium, chloride, water.-(b &c)

44.Thyroid hormones exert specific effects upon: a) growth b) metamorphic development c) embryonic development d) all the above.-(d)

45.Which of the following agent is important hormonal antagonists of insulin in the body: a) glucagon b) growth hormone c) adrenaline d) all the above.-(a)

V.Choose the correct answer and give your explanation:

1. Chronic administration of glucocorticoid may A)induce anemia B) increase the amount of adipose tissue in the body by decreasing lipolysis. C) induce osteoporosis D) induce lymphocytosis.

Answer is C. Glucocorticoid may create a negative calcium balance by inhibiting calcium absorption from gastrointestinal tract and reabsorption from the kidney. Glucocorticoids induce polycythemia, lipolysis and lymphocytopenia.

2. All the following statement concerning agents for treatment of hypocalcemia are true except
A)Only a small amount of calcium administered is excreted in the urine. B) Calcium gluconate is also used for the treatment of cardiac arrhythmias induced by hyperkalemia. C) Dihydrotachysterol like parathyroid hormone elevates serum calcium concentration and lowers serum phosphate concentration. D) dihydrotachysterol is metabolised in the liver to a 25(OH) metabolite which is the active form of the drug.
Answer is C: Dihydrotachysterol (DHT)elevates both serum calcium and phosphate concentrations. Like parathyroid hormone increase serum calcium concentration by promoting calcium and phosphorus absorption from the GI tract and by decreasing calcium excretion from the kidney. However unlike parathyroid hormone DHT decreases phosphorus excretion from the kidney.

3. Use of a glucocorticoid is contraindicated in all of the following conditions except A)diabetes mellitus B) corneal ulceration C) anaphylactic shock D) infection uncontrolled by antibiotics E) burns.
Answer is C: glucocorticoids are not contraindicated in anaphylactic shock. They should not be used in animals with corneal ulcers, diabetes mellitus, infections uncontrolled by antibiotics, or burns.

4.Which of the following steroids is used to treat anemia? A) deoxycorticosterone B) estradiol C) medoxyprogesterone D) boldenone

The answer is D. Androgen increase erythropoiesis by promoting erythropoietin synthesis. Therefore, androgens such as boldenone can be used to treat anemia. Deoxycorticosterone, estradiol, and medoxyprogesterone are not androgens, and thus do not promote erythropoiesis.

5.Administration of megestrol acetate in queens as an oral contraceptive may cause A) masculinisation. B) diabetesmellitus C) aplastic anemia. D) hepatopathy.

The answer is B. megestrol, a progestin,may cause diabetes mellitus, particularly in a diabetes–prone animals, this is due to progestins glucocorticoid activity. Megestrol is not an androgen , so it does not cause masculinisation or hepatopathy . It is not an estrogen , so it does not cause aplastic anemia

6. All of the following statements concerning antidiabetic agents are true except A) commercial insulin preparations are mostly of human origin. B) hepatocytes, erythrocytes, leukocytes, adrenal medullary chromaffin cells , and brain cells do not require insulin to transport glucose. C) dogs are

more likely than cats to develop insulin antibodies to porcine or human insulin preparations. D) glipizide , a sulfonylurea, has been used with some success in treating cats with moderate diabetes.

The answer is C. Following administration of a human or porcine insulin preparation, cats are more likely than dogs to develop insulin antibodies. Most commercial products are developed using recombinant DNA techniques and are of human origin. Hepatocytes, erythrocytes, leucocytes, adrenal medullary chromaffin cells, and brain cells do not require insulin for glucose transport. Glipizidea sulfonylurea , has been used with some success to treat feline diabetes.

7. which of the following drug is preferred drug for the treatment of the adrenal cortical disease in ferrets? A) Ketoconazole B) Trilostane C)Leuprolide D) Mitotane.

The answer is C. Leuprolide is a partial agonist of Gn RH. The adrenl cortical disease (ACD) of ferrets is due to an increase in Gn RH secretion from the hypothalamus in neutered ferrets. Gn RH evokes hyper adrenocorticism in these animals with a great increase in sex steroids. Other inhibitors of the corticosteroid synthesis may be used in conjunction with leuprolide treatment.

8.Frequent administration of which one of the following sex steroids in animals would most likely inhibit growth of the adrenal cortex and adrenocorticotropic hormone(ACTH) secretion? A) boldenone B) estradiol C) megestrol D) stanozolol

The answer is C. progestin such as megestrol have glucocorticoid-like activities that can inhibit adrenocorticotropic hormone secretion, inducing iatrogenic hypoadrenocorticism. Estrogens and androgens do not have significant glucocorticoids activity.

VI. Match each one in A to all the matching ones in B

A	B
1. Vitamin A	Gizzard ulceration in chicks--6
2. Calciferol D2	Beriberi--7
3. Cholecalciferol-D3	Cobalt--10
4. Tocopherol	Pellagra--8
5. Coagulation vitamin	Calcium stones in the kidney.--3
6. Vitamin U	Avidin--9
7. Aneurine	Dehydrocholesterol, --3
8. Niacin	Ergosterol--2
9. Biotin	vitamin K--5
10.Vitamin B12.	Labiliselysosomes—1
	Rickets
	White muscle disease—4
	Xerophthalmia—1
	Epistaxis.—5
	Yellow fat disease in horse—4
	Coumarin toxicity.-5

A	**B**
1.Mineralo corticoids	cretinism-14

2.Justaglomerular apparatus	metformin-11
3.Glucocorticoid therapy	Rubber jaw-7
4.Aldosterone	uncoupling of oxidative phosphorylation-15
5.Hyperfunction of adrenal cortex	tapering dose-3
6.Calcitonin	alpha cells islets of langerhans-13
7.Hyper parathyroidism	acarbose--12
8.Thiocyanate	Addison's disease-4
9.Thiourea	fludrocortisone-1
10. Sulfonyl ureas	interfere thyroxin synthesis-9
11. Biguanides	parafolicular C cells-6
12. Alpha glucosidase inhibitors	cushings disease-5
13. Glucagon	tolbutamide-10
14.Thyroxin deficiency	block concentration of iodine-8
15.Dinitrophenol.	rennin-2

A	B
1. Nandrolone	"slow feed back inhibition"--(3)
2 .Glucocorticoids	glipizide—(11)
3. ACTH	Thyroxine-
4. Addison's disease	troglitazone—(10)
5. Spironolactone	insulin antagonism.—(9)
6. Glucocorticoid receptors	aldosterone antagonist-(5)
7. Mitotane	Acarbose.—(12)
8. L-Deprenyl	"Heat shock protein"—(2)
9. Glucagon	"Zinc fingers"-(6)
10. Thiazolidine diones	excess mineralo corticoids—(4)
11. Sulfonyl ureas	Cushing's disease-(7), (8)
12. Alpha glucosidase inhibitor	anabolic steroid-(1)

VII. Critically comment on the following :

1.A single dose of corticosteroid is given to an animal suffering from chronic bacterial infection.---In chronic bacterial infection corticosteroids are contra indicated .However a single dose is not at all contraindicated .hence the treatment is on the right way.

2. A diabetic dog undergoing a course of steroid therapy to heal a deep corneal ulcer has given antirabic vaccination.----- The treatment is fully unethical. For healing deep corneal ulcers steroids are not recommended that too in a diabetic dog. Steroids will aggravate diabetes .Anti rabbic vaccination will not be effective during steroid therapy since the immune response is suppressed due to steroid.

3. Parathormone is administered orally in a cow suffering from milk fever . parathormone can be given in milk fever but not oral therapy because this is a polypeptide and digested in the GI tract hence the treatment will not be effective.

4. Corticotropin lyophilized powder is administered orally after reconstitution in a dog suffering from corticoid deficiency due to adrenopathy. This treatment is not rationale because corticotrophin is a polypeptide which will be digested in GI tract ,further corticotrophin will work only when functional adrenal gland is present.

VIII. Explain the mechanism of action of the following (in 1-2sentence)

1.Augmentation of insulin secretion by glucose .--- Beta cells have glucose receptors- activated by glucose-increase intracellular cyclic AMP-increase calcium availability-causes release of insulin.

2. Glipizide on insulin release.--- is a sulfonyl urea inhibits ATP dependent potassium channels in the plasma membrane- result in depolarisation and release of insulin.

3. Sitagliptin as antidiabetic agent.---is an oral antidiabetes drug which inhibits dipeptidyl peptidase -4which is involved in the biotransformation of glucagon like peptide-1 (Incretin)which enhances the secretion of insulin.

4.Bbiguanides as antidiabetic agent.----- they enhance the binding of insulin to its receptors and stimulate insulin mediated glucose disposal, it inhibits intestinal absorption of glucose, stimulate peripheral utilisation of glucose, suppress hepatic gluconeogenesis .

5. Acarbose on carbohydrate metabolism.----it is an alpha glucosidase inhibitor acts by competitively inhibiting the action of intestinal brush border enzymes alpha glucosidase which digest carbohydrates and release glucose from larger carbohydrates.

IX. Answer the following

1.What is insulin shock? high amount of insulin causes sudden hypoglycaemia –early signs include tachycardia, hunger result from epinephrine release-worst sequel is characterised by CNS disturbances including convulsion, coma- (treat with Glucose I/V immediately otherwise fatal).

2. What are the main symptoms of diabetes mellitus? Hypoglycemia, glycosurea, polydipsia, polyphagia, debility, ketosis, polyurea.

3. What are the two types of hypoadrenocorticism (Addisons disease) how it happens?1) primary adrenocorticism occure due to defective functioning of the adrenal corte 2) secondary adrenocorticism is due to deficiency of ACTH.

4. Mechanism of action of thyroxin: T3 is the most active form. It stimulate the mRNA synthesis for a number of protein that are important for thyroid hormone functions-- it activate its mitochondrial receptors to increase mRNA synthesis in the organelle —thyroid hormone metabolite s may activate G- protein coupled receptors to mediate its acute and nongenomic effect.

5. In replacement therapy T4 is preferred than T3 Why? because T4 can maintain high thyroid hormone activity in brain than T3.

6. What are the molecular mechanism of action of hormones: many hormones affect signal transduction through the following mechanism. A) GTP binding protein in the plasma membrane. B) receptor with tyrosine kinase activity, cystosolic tyrosine kinase C) Guanylyl cyclise D) intracellular receptors.

7. What are the uses of adrenaline? a) epitaxis 1: 30000 to 1: 100000 to reduce secondary haemorrhage b) 1: 10000 to 1: 50000 along with local anaesthetic to induce vaso constriction c) In allergic condition 1: 1000 solution 4ml in horse .d) laminitis in horse 1: 2000 sol.2ml. e)In cardiac arrest.

8.What are the Contra indications of corticosteroids: Diabetes mellitus, hyper tension, deep corneal ulcers, renal insufficiency, late pregnancy, surgery, chronic bacterial infection, muscle wasting in thin animals. Reduce wool growth in sheep, inhibition of growth in young animals.

9. What is the mechanism of action of growth hormone: It activates Janus kinase (JAK2) signal transduction and activation of transcription (STAT) pathway which leads to an increase in transcription and protein synthesis . Growth hormone also stimulate the secretion of insulin like growth factor-1 (IGF-1, somatomedin) from the liver which in turn participate in some of the effect of G.H.

10.How selegilin inhibits ACTH secretion? Selegilin increase the dopamine concentration by inhibiting the metabolism by monoamine oxidase B and decreasing the reuptake of dopamine which inhibits ACTH secretion.

11.How Trilostane inhibits corticosteroid synthesis ? Trilostane is a competitive inhibitor of 3-Beta hydroxyl steroid dehydrogenase and this inhibits corticosteroid synthesis.

12. How T3 decreases plasma LDL ? T3 decrease plasma concentration of low density lipoprotein by increasing the expression of LDL transport receptors in the liver , thereby increasing utilization of LDL by the liver.

13.How ketoconazole inhibits adrenal steroidogenesis? Ketoconazole inhibits the cytochrome P-450 enzymes that are involved in steroid synthesis there by steroidogenesis in inhibited.

14.How gonadotropins acts? All gonadotropin receptors are coupled to Gs which activate adenyl cyclase to increase cyclic AMP formation which in turn activate protein kinase A which phosphorylates the cellular constituents for the action of the hormone.

15.What is the mechanism actions of biguanides. It enhance binding of insulin to receptors- inhibits intestinal absorption of glucose-suppress hepatic gluconeogenesis-promote peripheral utilization of glucose.

16.What are the actions of thyroxin: it increases the oxygen utilization of every cells- increase the size and number of mitochondria - Stimulate protein and fat utilization. Stimulate CNS functions.

17.What are the uses of thyroxin: used to prevent goitre, to treat thyroid deficiency, to reduce obesity, stimulate fertility of breeding bulls, to treat urinary incontinence, to treat certain types of eczema, bilateral alopecia, to treat loss of wire in wire haired breeds.

18. What are Soaks? Soaks are a form of hydrotherapy containing medications usually keratolytic agents. Animals are either allowed to sit in the medicated solution or the solution is applied with sponge and allowed to remain for several minutes for drying.

19.What are the preparations of thyroxin: a) desiccated thyroid from slaughter house b) Iodinated casein-protamon c) Thyroxin sodium –Eltroxin d) Sodium levo thyroxin-synthroid e) Tri-iodothyronine –cytobin the most potent form.

20.What is thyroid escape phenomenon ? The effect of iodide in hyperthyroidism is drastic beneficial effects are seen in 24 hours and maximum in 10 to 15 days of continuous iodide therapy. the symptoms are improved , reduction in vascularity and swelling of gland is reduced. However , after a few weeks of daily therapy thyroid ceases to respond to iodide and hyperthyroidism may return to initialintensity or even worse-this pheniminon is called as thyroid escape.

21.This toxic effect of DDD (dichloro diphenyl dichloro ethane) is utilised therapeutically in Cushings syndrome, What effect? DDD destroy adrenal cortex which is a toxic action of this – excess activity of adrenal cortex is seen in cushings syndrome.

22.ACTH is used mainly as a diagnostic tool for distinguishing the 2 types of adrenal insufficiency How? Two types of insufficiency are there , Primary and secondary- Primary is due to defunctioning of adrenal cortex due to various reasons, secondary is due to deficiency of ACTH. . When ACTH is administered in insufficiency if glucocorticoids are produced it is secondary insufficiency ,otherwise primary (even if ACTH is given no glucocorticoid production because of defunctional cortex)

X..Write short notes on.

1.Mechanism of anti inflammatory and immunosuppressive effect of glucocorticoids:
Different mechanisms are involved- 1) by stabilizing plasma and lysosomal membrane decrease release of histamine and proteolytic enzymes resulting in reduce vascular permeability and tissue destruction.2) by induction of lipocortin in macrophage , endothelium and fibroblast inhibits phospholipase A2 causing reduce production of prostaglandins , leukotriens and platelet activating factor. 3) by inhibiting cytokins in macrophages, endothelial cells and lymphocytes decreases the production of interleukins , TNF alpha, GM-CSF and gama interferons suppress T lymphocyte function and chemotactic factor. 4) by reducing the production of ECAM and ICAM-1 in endothelial cells , adhesion and localisation of leucocyte is interfered. 5)decrease acute phase reaction from macrophage and endothelial cells , complement fixation is interfered. 6) inhibition of IgE mediated histamine and LTC4 release from macrophages and endothelial cells inhibit antigen antibody mediated effect 7) decrease production of collagenase and stromelysin –prevent tissue destruction 8) inhibits inducible form of nitric oxide synthesis and reduce vasodilatation.

2. Actions of insulin:

Blood glucose level is reduced- pyruvate and lactate level is increased- inorganic phosphate and potassium level is reduced-accelerate hexokonase- accelerate rate at which glucose is converted to glycogen by liver. Inhibit glycogenolysis and gluconeogenesis-protein synthesis is assisted- regulate glucose utilization- assist for the transport of glucose across cell membrane-

3. Actions of parathormone on blood calcium level.

It act on the bone, small intestine and kidney to transfer calcium to the ECF -- stimulate the absorption of calcium from the intestine (vitamin D dependent process) -- stimulate the rate of resorption of calcium and phosphorus from bone-- stimulate renal tubular reabsorption of calcium and excretion of phosphate .

4. Action of glucocorticoids on immune system:

Increase susceptibility to infection, suppress immunity and organ rejection. Anti-inflammatory and anti allergic effect- leucocyte migration and functions are suppressed , lysosomal membrane is stabilised- synthesis of PG and leukotrien is suppressed . fibroblast activity, collagen synthesis and tissue repair are reduced. it increases the number of erythrocytes, neutrophils, monocytes, platelets. It decreases the number of lymphocyte, eosinophils, and basophils-- Reduces the size of lymph nodes and thymus. Production of interleukins by macrophage and T-Lymphocyte is inhibited.

5. Classsify anti thyroid drugs with examples.

a) thyroxin synthesis inhibitors- thionamides eg. thiourea and thiouracil b) Ionic inhibitors- block the iodine traping by thyroid eg. Thiocyanates and perchlorates c) Iodine itself eg. Potassium iodide and sodium iodide d) radio active iodine eg. Iodine 131. E) miscellaneous eg. Para amino salicylic acid, sulfathiozole.

6. Aadverse effects of Glucocorticoids high dose long term therapy.

Iatrogenic hypoadrenocorticism (may follow withdrawal of steroid) due to decrease in ACTH secretion. Decrease wound healing, increase susceptibility to infection, fluid and electrolyte imbalance, myopathy. Osteoporosis-decrease calcium absorption from GI tract and reabsorption from the kidney. (decrease plasma c++ conc. Send signals to parathyroid –increase parathormone-promote bone resorption), odema (increase Na+ retension). Congestive heart failure in cats. Thrombosis(increase platelets in blood) –hepatotoxicity in dogs, GI. ulceration, diabetes mellitus, abortion in late pregnancy, laminitis in horse.

7. Addisons disease:

caused due to hypo function of adrenal cortex- due to atrophy, destruction by infarction, disease, tumors ,or excess corticosteroids. It may be chronic with symptoms like vomition, muscular weakness, anorexia , depression, diarrhoea, dehydration, coma and death.

8.Aldosterone:

Mineralo corticoid secreted by adrenal cortex - regulated by renin angiotensin system and Sodium level in the system . Causes retention of sodium , chloride and water, increase the excretion of potassium, calcium and phosphorus. Underproduction causes addisons disease and over production

cause conn's syndrome, Eg. Desoxy corticosterone acetate-can be used to treat chronic interstitial nephritis

9. Classify drugs used in insulin disorders:

In insulin deficiency A) insulin and analogues are used: short acting semi lente insulin, intermediary –isophane insulin, long acting- protamine zinc insulin, ultra long action-insulin degludee.B) Insulin secretagogues: Sulphonyl ureas- glipizide, nonsulphonyl ureas- mitiglinide and exenatide, C) Insulin sensitizers: Biguanides- metformin, Thiazolidine diones-pioglitazone. D) Miscellaneous: acarbose, canagliflozin. In excess of insulin -Glucagon, diazoxide are used.

10. General principles of treatment with corticosteroids :

Dosage must be fixed individually. High dose in acute case and low dose in prolonged case. Sudden termination cause acute hypocortical state, hence terminate the treatment with tapering dose . In infectious disease special caution is necessary because it may flare up. Get only symptomatic relief in systemic diseases. In addisons disease steroids cause cure. If local application is enough give only local not systemic. Use in corneal ulcers and high dose for pregnant cow special caution is necessary.

11. Treatment of hyper thyroidism:

Excess amount of thyroid hormone in circulation –associated with a number of disease state including graves disease, toxic adenoma, thyroiditis- treatment 1) removal of a part of thyroid by surgery /Beta particle by radio active iodine(131 I). 2) inhibition of hormone synthesis by using propyl thiouracil, methimazole-no effect on already synthesised hormone so delayed clinical effect.3) propranolol, a Beta blocker block the sympathetic stimulation that occurs in hyper thyroidism 4) release of hormone can be blocked by Iodine itself – inhibit iodination of thyrosine-thus decrease the supply of stored thyroglobulin. Iodine block hormone release also –used in potentially fatal thyrotoxic crisis (thyroid storm) –not good for long term treatment.

12. Mechanism of action of sulfonyl ureas:

They block ATP sensitive K+ channels which decrease K+ exit from Beta cells. Retension of intracellular K+ leads to depolarisation of plasma membranes , which activate the voltage dependet Ca++ channels.(VDCCS) . Opening of VDCCS promote calcium entry to elevate Ca++ . which evokes exocytosis, resulting in insulin release. ATP can block ATP sensitive K+ channels as well . ATP is generated from glucose metabolism (This also explain how glucose stimulates insulin secretion)

13.Mechanism of action of Insulin:

It reduce blood glucose concentration by increasing glycogen synthesis and reduce glycogenolysis, reduce gluconeogenesis, increasing glucose transport in to skeletal muscle cells and adipocytes through activation of type 4 glucose transporter (GLUT 4) . Increase lipid synthesis and reduce lypolysis. Increase the uptake of amino acid and protein synthesis, increase uptake of potassium in to skeletal muscles and fat cells by providing ATP to activate Na+ K+ -ATPase.

14.Metabolic effect of corticosteroid:

On carbohydrate metabolism –stimulate gluconeogenesis,reduce peripheral utilisation of glucose. Antagonise insulin-hyperglycemia , glucosuria, temporarly alleviate hypoglycemia in ketotic cow,

stimulate protein catabolism, block anabolism-negative nitrogen balance ,growth ceases in young ones, inhibit antibody production ,muscular wasting and weakness. Catabolise fat, shift body storage of fat ,prolonged use cause moonface and buffalo hump , loss of fat from extrimities and deposit in cheeks and neck regions , muscular weakness, osteoporosis, fracture. Reduce inflammation, reduce antibody production, delay healing, antiallergic action.

15.Pharmacological and physiological effect of Thyroxin:

T3 is the active form it promote growth and maturation, growth and development of bone and nervous tissue, necessary for the oxidative metabolism increase the utilisation of oxygen by every cell, stimulate carbohydrate and fat utilisation, stimulate anabolic reaction. (but excess amount is catabolic). (increase GH secretion) It stimulate basal metabolic rate-activate uncoupling oxidative phosphorylation in brown fat cells. It is a cardio vascular stimulant –increase cardiac output (increase the expression of Alpha and Beta adrenergic receptors) –enhances the carbohydrate utilisation and lypolysis- reduces the plasma concentration of LDL by increasing utilisation of LDL by the liver.

16. Pharmacologicl and physiological action of Glucocorticoids on intermediary metabolism and cardio vascular system?

it increase liver glycogen synthesis and storage, gluconeogenesis, lypolysis and redistribution of lipids. Increase glucose level trigger insulin level and bring down plasma level (not happens in diabetes patients.) Chronic administration damages the beta cells and increases the protein breakdown. On CNS it stimulate and may leads to euphoria. On C.V. system glucocorticoids increase vasomotor response - increases the epinephrine synthesis –increase expression of alpha adrenergic receptors in vascular muscles and Beta receptors in myocardium. It facilitate the angiotensin system , promote breakdown of bradykinin , decrease capillary permeability.

17.Parathormon:

secreted by parathyroid (4 small glands at the poles of thyroid) polypeptide not active orally-secretion regulated by circulating calcium level in blood . It regulate calcium metabolism stimulate bone resorption of calcium. In kidney stimulate calcium resorption and reduce phosphorus reabsorption. Excess production causes hyper parathyroidism – rarely seen in dogs. Differs from hypocalcemia caused by parturition. In hyperparathyroidism- abnormal parathyroid-demineralisation of bone- surgical treatment can be adopted. Secondary hyperparathyroidism due to chronic renal disease or chronic imbalance in calcium phosphorus intake causes demineralisation of bone. Hypertrophy condition referred as " Rubber jaw" .used in hypocalcemic tetany of skeletal muscles, fluorine ,oxalate lead and other heavy metal toxicity.

18. Preparations of insulin?

I. Short acting preparation; soluble crystalline form of zinc insulin act for 8 hours. II.Intermediary acting a)Globin zinc insulin act for 24 hours. b) Isophane insulin –modified form of protamine insulin in neutral solution(NPH) c) Lente insulin. III. Long acting compound: ultralente insulin –large particle size act for 36 hours.

19. Uses of glucocorticoids:

As anti inflammatory agent used in skin disease, Eye condition, arthritis, graft regection, joints and tendon enlargement. Used in stress it stimulate the ability of the body to resist stress due to trauma, disease, fatigue, reduce oedema formation, allow better penetration of antibiotics.Used in pregnancy toxaemia in sheep, bovine ketosis to treat acute lymphocytic leukemia, Used in mastitis along with antibiotics. For induction of parturition in ruminents (single injection of dexamethasone).

20. Uses of short, intermediary and long acting preparation of glucocorticoids:

Short acting preparations (hydrocortisone, cortisone, fludrocortisone) for topical use to treat pruritus and inflammation associated with allergy . Intermediary acting compounds (prednisone, Triamcinolone, Prednisolone) used for long term control of allergy, chronic inflammation (arthritis) and immunosuppression. Orally used in alternate day therapy. Long acting compounds (betamethasone, sexamethasone, Flumethasone), Mometasone , flucinolone) used for the immediate relief of hypersensitivity and shock (haemorrhagic and septic shock) long term control of allergy in cats. Topically used to control pruritus and inflammation associated with allergy. May be used to induce parturition , Isoflupredone is used treat ketosis in cow.

21. Treatment of adrenal hormone disorders:

In deficiency of adrenal hormones: Glucocorticoids-short acting hydrocortisone, intermediary acting-prednisolone, long acting-dexamethasone, topically acting-beclomethasone. Mineralocorticoids –fludrocortisone.

In excess of adrenal hormones: inhibitors of corticosteroid biosynthesis-mitotane, inhibitors of glucocorticoid receptors –mifepristone, inhibitors of aldosterone receptors-spironolactone, inhibitors of ACTH biosynthesis and release –bromocriptin.

22. Indications of glucocorticoids:

In the disorders of the following systems-Gastro intestinal, Respiratory, Skin. a) Gastro intestinal colitis, ulcerative colitis, inflammatory bowel disease, non suppurative cholangiohepatitis, feline gingivitis/ stomatitis, lymphangiectasia. b) Respiratory system- chronic obstructive pulmonary disease, feline asthma syndrome,chronic allergic bronchitis, inhalation injury, pulmonary oedema from drowning, aspiration pneumonia, heart worm sensitivity. C) Skin- flea allergic dermatitis, parasitic dermatitis, drug reaction, immune-mediated skin disease, allergic dermatitis, non specific eczema.

23. Mechanismof action of Growthhormone:

The growth hormone receptors has single subunits that contain both GH binding and signaling domains . These receptors are associated with Janus kinase (JAK) a cytosolic form of tyrosine kinase . Upon activation and dimerization of GH receptors 2 JAKs undergo reciprocal phosphorylation which further attracts signal transduction and activation of transcription 2(STAT-2) protein. The homodimer of STAT 2 binds to DNA and stimulates gene transcription. GH also stimulate the secretion of insulin like growth factor-1 (IGf-1, somatomedin) from the liver which in turn participate in some of the effects of GH (eg. Growth, cartilage, protein metabolism)

XI.Write Essays.

1.Describe the pharmacological action of Metabolic hormones.

2.Describe the pharmacological effects of catecholamines.
3.Explain the physiological and pharmacological action of corticosteroids on different systems in the body.
4. Pharmacology of hormones secreted by Islets of Langerhans.
5.What are catecholamines ? explain their action on cardiovascular, metabolism and respiratory system.
6.Expain the synthetic anti-hyperglycemic agents .
7.Explain the action of glucocorticoids on different body system and its therapeutic applications.
8.Explain corticosteroids give emphasis to its regulation of secretion, action use and adverse effects.
9.Explain regulation of thyroxin secretion, metabolic action, source and use.