QUESTION BANK (VETERINARY PHARMACOLOGY & TOXICOLOGY)

PAPER No-24

HEAVY METALS

1.A chloride of mercury which is a violent poison.-(Mercuric chloride)

2.Agents used to precipitate antimony in the stomach.-(magnesium oxide, calcium hydroxide)

3.A heavy metal that binds to ceruloplasmin.-(Copper)

4.A heavy metal causing Bluish green feces in cattle.-(Copper)

5.A highly toxic mercury salt with powerful antiseptic action.-(Mercuric chloride)

6.A mercury salt with purgative property .-(Mercurous chloride)s

7.An enzyme which contain selenium .-(Glutathione peroxidase)

8.Animal which are comparatively resistant to the action of lead.—(pigs)

9.A pigment which contains mercury.-(Vermillin)

10.A plant which will accumulate copper.-(*Heliotropum europium*)

11.Arsenical compound carried as a dust with smoke .-(Arsenic trioxide)

12.A selenium containing enzyme.-(Glutathion peroxidase)

13.A selenocysteine containing enzyme.-(Glutathion peroxidase)

14.A wood preservative compound which contains copper .-(copper napthenate)

15.Chemical present in “BLUE STONE”.-(Copper sulphate)

16.Common name of cupric aceto arsenate--(parish green)

17.Heavy metal which cause blackberry jam spleen.-(Copper)

18.Highly toxic mercury salt which is a wood preservative.-(mercuric chloride)

19.One arsenical livestock dips.-(Lead arsenate)

20.One arsenical feed additive.-(Acetarsol, neoarsphenamen, salvarsan)

21.One arsenical rodenticide.-(Arsenic trioxide)

22.One arsenical antiparasitic injection.-(Sodium thiocetarsamide, Sodium arsenamide, Sodium acetarsenate

23.One heavy metal which volatilizes from liquid stage even at room temperature.-(Mercury)

24.One highly toxic heavy metal which is liquid in atmospheric condition).-(Mercury)

25.One inorganic arsenical compound useful as a medicine .-(Potassium arsenite, Fowlers solution)

26.One inorganic arsenical which will not show typical signs of arsenic poisoning.-(Arsine gas)

27.One iron compound in the body which will not binds with desferrioxamine.-(Cytochrome)

28.One organic mercurial used as an antiseptic on mucous membranes.-(Mercurochrome)

29.Other name for mercuric chloride.-(Corrosive sublimate)

30.Other name of Copper arsenite .-(Parish green)

31.Other name for mercurous chloride.-(Calomel)

32.Other name of Seliniferous plant .-(Facultative accumulator)

33.Other name for chronic form of selenium toxicity.-(Alkali disease/ Locoism)

34.Other name of obligatory accumulator plants.-(Indicator plants)

35.Subacute toxicity of selenium.-(Blind staggers)

36.The active ingredient of Grey powder.-(Mercury)

37.The characteristic lead lines on gums in chronic lead toxicity .-(Burtons line)

38.The common name of mercuric iodide.-(Red iodide/ bin iodide of mercury)

39.Trace element which compete with phosphorus to deposit in bones.-(molybdinum)

40.The common name of mercuric iodide.-(Red iodide/ bin iodide of mercury)

41.Three indicator plants .- (Astragalus, Oxytropis, Acacia cana)

42.Three important enzymes which are affected by mercury.-(Alkaline phosphatase, ATPase, Glucose -6 phosphatase)

43.Three seleniferous plants. (Aster, Atriplex, Grayia)

44.Two arsenical insecticides.-(Sodium arsenate, Arsenic trioxide)

45.Two arsenical herbicide.-(Monosodium methane arsonate(MSMA) Mono ammonium methane arsonate (MAMA), Disodium methane arsonate,(DSMA).

46.Two copper fungicides.-(copper sulphate, Bordeaux mixture, copper napthenate)

47.Two chloride of mercury used in veterinary practice.-(mercuric chloride, mercurous chloride)

48.Two inorganic mercury preparation used in veterinary practice .-(Mercuric chloride/ Mercurous chloride)

49.Two lead compound which are absorbed through intact skin.-(tetramethyl lead, tetraethyl lead)

50.Two organic form of lead compound which is absorbed through intact skin .-(tetramethyl lead and tetraethyl lead)

51.Two obligatory accumulators.-(Astragalus,xylorrhiza, oxitropis)

52.Two organic form of selenium.-(Selinite and Selinate, Selinide)

53.Two oxides of mercury used in veterinary practice.-(red oxide of mercury, yellow oxide of mercury)

54.Two sources of copper poisoning in veterinary practice.-(use as an emetic ,copper tar dressing, use to close oesophagial groove)

55.Two trivalent form of arsenic .-(Arsphenamine, Thiacetarsamide)

56.Two water soluble derivatives of BAL.-(Meso dimercapto succinic acid.(MDS), Dimercapto succinic acid. (DMSA).

II.STATE TRUE OR FALSE:

1.Absorption of lead compound via inhalation route is up to 90% .-(T)

2.Absorption of lead compound via oral route is below 10%. –(T)

3.Absorbed lead via oral route binds with metallothionin and gradually excreted in bile.-(T)

4.Absorbed copper binds to alpha globulins and ceruloplasmins.-(T)

5.Acetyl cysteine is the suggested treatment for selenium toxicity.-(T)

6.A competition is seen between Molybdenum and phosphorus for deposition in bone.-(T)

7.Accumulation of lead in bone is a detoxifying mechanism of the body.-(T)

8.Acute form of toxicity of selenium is common than chronic form.-(F)

9.Acute selenium toxicity by plants are rare because animals will avoid eating indicator plants.-(T)

10.Alkali disease is chronic selenium poisoning.-(T)

11.Alkyl mercurials crosses the blood brain barrier and placental barrier. –(T)

12.Alkyl mercury compounds are more toxic than aryl mercury compounds.—(T)

13.Alkyl mercurials (methyl or ethyl) are highly lipid soluble and enters in to all organs including brain that causes neurological symptoms.—(T)

14.Animals will not eat seliniferous plants.-(T)

15.Arsenical gas is an inorganic arsenic.-(T)

16.Arsenic tends to accumulate in soft tissues like liver, kidney, heart and lung.-(T)

17.Arsenic will deposit in hard tissue like bone and teeth.-(F)

18.Arsenical gas will not cause typical signs of inorganic arsenic.-(T)

19.Arsenic can be detected in urine , feces and milk for a period up to 10 days following ingestion of arsenic.-(T)

20.Aster and atriplex is an example for seleniferous plant.—(T)

21.Baby pigs are most susceptible to iron poisoning .—(T)

22.BAL is considered to be nontoxic.-(F)

23.BAL is more effective than unitiol in the treatment of mercury poisoning.-(F)

24.BAL and Vitamin –E is effective in the treatment for selenium poisoning.-(F)

25.BAL and Vitamin-E is contra indicated in case of selenium toxicity.-(T)

26.BAL is the drug of choice in copper toxicity.-(F)

27.Basophilic stipling of RBC is a characteristic feature in copper toxicity.-(T)

28.Blind stagger is acute selenium poisoning.-(T)

29.Bluish green faeces is the typical symptoms in copper toxicity in cattle.-(T)

30.By giving feed containing 10 – 20 ppm selenium for weeks , sub acute form of selenium toxicity can be induced.-(T)

31.Cadmium prevent the normal utilization of zinc.-(T)

32.Cadmium prevent the normal utilization of zinc as a component of many enzymes.—(T)

33.Calomel give ash grey colour to the mucous membrane of G.I. Tract.—(T)

34.Cattle is more susceptible to lead poisoning than birds.-(T)

35.Cattle is the most susceptible species to Molybdenum toxicity.-(T)

36.Copper toxicity is seen mostly in cattle.-(T)

37.Copper binds with SH group of vital enzymes.-(T)

38.Copper causes lysosomal damage .-(T)

39.Copper causes lysis of RBC.-(T)

40.Copper arsenate is used in pigments.-(T)

41.Copper binds with molybdenum in the gut and absorbed.-(F)

42.Commercially penicillamine is produced by hydrolytic degradation of penicillin.-(T)

43.Cupric aceto arsenite is otherwise known as parish green.-(T)

44.CNS toxicity is more prominent with mercury in the vapour form.-(T)

45.Desferoxamine is the specific antidote of Iron.-(T)

46.Desferoxamine does not bind with iron in haemoglobin.-(F)

47.Desferrioxamine is a chelator of Iron and it is obtained from *Streptomyces pilosus*.—(T)

48.Desferrioxime is a chelator of iron, and it is synthesised in the laboratory.-(F)

49.Dimercaprol sodium sulfonate can be used in the treatment of mercury poisoning.-(T)

50.Disodium calcium EDTA can be used in the treatment of cadmium toxicity.—(T)

51.Divalent mercurials are more soluble and more toxic.-(T)

52.Dogs are more susceptible to lead poisoning than birds.-(T)

53.Donovan solution is effective in the treatment of black head in turkey.-(T)

54.D -penicillamine can be used for treatment of lead poisoning.-(T)

55.DTPA (diethyl triamine penta acetic acid) is a poly carboxylic acid chelator shows greater affinity for most heavy metals than EDTA-(T)

56.Excretion of desferrioxamine along with chelated iron will give a 'von rose' (reddish brown)urine.(T)

57.Even though poisoning with Zinc is rare ,above 72000ppm in the diet can cause chronic toxicity.-(T)

58.Excess molybdenum may interfere with hepatic storage of copper.-(T)

59.Facultative accumulator require selenium for their growth.-(F)

60.Fifty percent of the absorbed lead is in the free form.-(F)

61.Fowl and swine are rarely poisoned with arsenic.-(T)

62.Fossil fuels can cause volatile mercury toxicity .-(T)

63.Glutathione contains selenium.-(T)

64.Goats, swine and chicken are comparatively resistant to lead.-(T)

65.Grasses near busy high way may contain high level of lead.-(T)

66.Heliotropum europaeum contain high amount of copper.-(T)

67.High intake of phosphorus favors skeletal storage of lead.-(T)

68.High dietary sulphate increases molybdenum toxicity.-(T)

69.High dietary sulphate decreases the excretion of molybdenum.-(T)

70.High inorganic phosphorus hasten the excretion of molybdenum in the urine.-(T)

71.High protein in diet reduce gut absorption of selenium.-(T)

72.In acute oral poisoning with mercury gastric lavage with saturated soda bicarb is advisable.-(T)

73.In arsenic poisoning gut content may contain blood and shreds of epithelial cells.-(T)

74.In bones lead will produce toxicity only in chronic case.-(T)

75.In chicken cadmium toxicity causes the hatching out of chicks without beaks and with “Ropy feather”.-(F)

76.In chronic mercury poisoning the radio opacity of bone is not seen .-(T)

77.Incorporation of molybdenum in the ration give some protection against copper toxicosis.-(T)

78.In chronic cases of lead poisoning when bone is get saturated only it will produce toxicity.-(T)

79.In chicken selenium toxicity causes the hatching out of chicks without beaks and with “Ropy feather”.-(T)

80.In chronic lead poisoning the radio opacity of bone is seen .-(T)

81.Indicator plants does not require selenium for their growth .-(F)

82.Ingestion of copper increases selenium absorption.-(F)

83.In mercury poisoning stomach lavage with sodium formaldehyde sulfoxylate is advisable.-(T)

84.In selenium poisoning there will be increased concentration of oxidized glutathione.-(T)

85.In selenium poisoning there will be decreased concentration of reduced glutathione.-(T)

86.In subacute toxicity of selenium , paralysis of throat and tongue and wandering by animals are two characteristic symptoms.-(T)

87.In the rumen catecholes are stimulated by molybdenum.-(F)

88.In the treatment of arsenic poisoning with BAL, aging will not delay the regeneration of enzymes.-(F)

89.In textile industry dyes are one of the occupational source of mercury poisoning.-(T)

90.In lead poisoning basophilic stipling of RBC is is seen.-(T)

91.In lead poisoning animals will push against fences and wander in circle .-(T)

92.In zinc toxicity calcium disodium EDTA followed by D-penicillamine is the specific treatment.-(T)

93.Large amount of lead at a time is not toxic as it will take time to accumulate in tissue to produce toxicity.-(F)

94.Lead causes damage to the blood brain barrier and allow cytotoxic agents to get enter the brain.-(T)

95.Lead causes cerebral oedema but does not cause cerebral haemorrhage.-(F)

96.Lead toxicity causes rapture of lysosomes.-(T)

97.Lead interfere with Na+ K+ ATPAse.-(T)

98.Lead causes immuno suppression.-(T)

99.Lead interfere with the synthesis of haeme .-(T)

100.Lead inhibit delta amino levulinate dehydratase.-(T)

101.Leugols solution 1% can precipitate lead in stomach.—(T)

102.Lead sulphide is sold in powder form instead of Antimoney trisulphide as”Suruma”.-(T)

103.Lead acetate is less toxic than lead sulfide.-(F)

104.Lead affect the cellular respiration.-(T)

105.Lead interfere with zinc and iron to act as prosthetic group of enzymes.-(T)

106.Lemon juice stored in lead coated vessel is toxic.-(T)

107.Leugols iodine solution 1% can be used to precipitate lead, mercury, quinine, and strychnine.-(T)

108.Lewisite is an arsenical poison used in second world war.(T)

109.Liver remove lead by binding to cytoplasmic protein metallothionin.-(T)

110.Loss of hair from mane and tail are characteristic symptoms in sub acute selenium poisoning.-(F)

111.Loss of hair from mane and tail are characteristic symptoms in chronic selenium poisoning.-(T)

112.Magnesium hydroxide and iron preparations are contra indicated orally .—(T)

113.Meat from mercury poisoned animal is not toxic for consumption.-(T)

114.Mercuric sulphide is highly toxic.-(F)

115.Mercury vapour will not be absorbed through the lungs.-(F)

116.Mercury vapour can not cross the blood brain barrier.-(F)

117.Mercury binds with amino, carboxy and phosphate group.-(T)

118.Mercuric chloride is more toxic than mercurous chloride.-(T)

119.Mercury vapour absorbed via lung is oxidized to divalent form by catalase in the RBC.-(T)

120.Mercury and copper in the diet reduce the gut absorption of selenium.-(T)

121.Mercury combines with thiol group (SH)of proteins and inhibits the enzymatic activity.-(T)

122.Mercuric salt precipitate the mucous membrane of gastro intestinal tract.-(T)

123.Mercurochrome is an organic mercurial.-(T)

124.Methyl mercury causes more prominent CNS toxicity than elemental mercury.-(T)

125.Milk from lead poisoned animal is safe for consumption.-(F)

126.Milk from mercury poisoned animal is toxic for consumption.-(T)

127.Milk from arsenic poisoned cows are toxic and meat is safe for consumption.-(T)

128.Molybdinosis resulting from the contamination of forage from one industry is prevented by contamination of the same pasture with copper from another industry.-(T)

129.Monogastric animals are more susceptible to zinc intoxication than ruminants.—(F)

130.Monovalent mercurials are more soluble and more toxic.-(F)

131.Molybdenum will not be excreted in the milk.-(F)

132.Molybdenum toxicity cause change in colour of animals.-(T)

133.Monovalent mercurials are less toxic .-(T)

134.Molybdenum promote hepatic copper excretion.-(T)

135.Molybdenum deficiency cause Teart in cattle.-(F)

136.Orally ascorbic acid and desferrioxamine can enhance the excretion of iron.-(T)

137.Orally lead is less toxic than other route since it forms insoluble complex in the gut.-(T)

138.Organic mercurials can not cross the blood brain barrier.-(F)

139.Organic form of lead is less absorbed.-(F)

140.Organic form of lead is water soluble.-(F)

141.Organic form of lead is lipid soluble.-(T)

142.Organic form of lead is absorbed even through intact skin.-(T)

143.Organic form of lead is some what volatile.-(T)

144.Organic mercurials can cause teratogenic effect.-(T)

145.Parish green is a copper salt used in coloured wall paper.-(F)

146.Passive accumulators require minute amount of selenium for growth.-(F)

147.Passive accumulators are otherwise called as indicator plants.-(F)

148.Peat scour is the direct toxicity of molybdenum.-(F)

149.Peat scour is due to the excess growth of bacteria in the rumen.-(T)

150.Pentavalent arsenical will produce nervous signs.-(T)

151.Pentavalent form of arsenic is more toxic than trivalent form.-(F)

152.Pentavalent form of arsenic is converted to less toxic trivalent form.(F)

153.Penicillamine is available as CUPRIMINE capsules.-(T)

154.Penicillamine is contra indicated in pregnancy and previous history of penicillin induced agranulocytosis.-(T)

155.Prussian blue helps in the elimination of thalium from the gut.—(T)

156.Pyruvate dehydrogenase system is highly sensitive to arsenic.-(T)

157.Rapid administration of desferrioxamine may cause cardiac arrhythmia.-(T)

158.Rechargeable dry cell can act as a source of cadmium contamination.-(T)

159.Since mercury is not eliminated in milk it is safe for consumption.-(F)

160.Selinite is more toxic than selinate .-(T)

161.Selinate is more toxic than selinite.-(F)

162.Selenium is present in glutathione peroxide.-(T)

163.Selenium and vitamin E in the diet reduce toxicity of organic mercurials.—(T)

164.Selenium binds with SH groups of enzymes.-(T)

165.Seleniferous plants have a pleasant odor.-(F)

166.Selenium is an essential trace element.-(T)

167.Since calcium and cadmium are antagonist calcium can be used in cadmium toxicity.—(T)

168.Sodiumcarbonate is an antidote of zinc orally.—(T)

169.Sodium formaldehyde sulfoxylate convert mercury ion to less soluble metallic mercury.-(T)

170.Some amount of tolerance may develops towards lead toxicity if there was previous exposures.-(T)

171.Some arsenicals are used in rumenotoric preparation.-(T)

172.Some arsenicals will be absorbed through intact skin.-(T)

173.Sulphates reduce molybdenum excretion in the urine.-(T)

174.Tartar emetic is Antimony potassium tartrate.(T)

175.TCA cycle is not affected by arsenic.-(F)

176.Tetraethyl lead can penetrate intact skin.-(T)

177.Thalium inhibit sodium potassium ATPase.—(T)

178.There is competition between molybdenum and phosphorus for deposition in bones.-(T)

179.The aryl (phenyl mercury) compounds are readily excreted via kidney and less likely to accumulate in brain and muscles –(T)

180. The toxicity of molybdenum is increased by zinc.-(T)

181.The toxic dose of copper in Lambs is 20mg/kg.-(T)

182.Thallium binds with SH group of enzymes.-(T)

183.The biological half life of arsenic for most species is only a few days.-(T)

184.The biotransformation of organic mercurial is very fast.-(F)

185.Thioctic acid can be used as a remedy in arsenic toxicity.-(T)

186.Toxicosis due to iron can be prevented by increasing Vitamin-E and Selenium in the feed._(T)

187.Tolerance level of consumption of mercury is 0.3 mg/week.-(T)

188.Trivalent arsenicals will bind to lipoic acid.-(T)

189.Trivalent arsenic is more toxic.-(T)

190.Vitamin E is contra indicated in selenium poisoning.(T)

191.Vitamine E deficiency enhances iron toxicity.—(T)

192.Volcanic eruptions can cause mercury poisoning .-(T)

193.White lead is Zinc oxide.-(T)

194.Antimony potassium tartrate is otherwise known as tartar emetic.—(T)

195.Antimony trichloride is caustic.—(T)

196.In lead poisoning plasma may fluoresce under u/v light.—(T)

197.Iron is precipitated by milk of magnesia.—(T)

198.Iron toxicity can be prevented by increasing vitamin E and selenium in the feed.—(T)

199.Zinc toxicity can be treated with calcium disodium EDTA and Penicillamine.—(T)

200.Zinc oxide is otherwise known as “white lead”.—(T)

201.Zinc chloride is otherwise known as “ butter of zinc.—(T)

202.Tissues which require high oxidation energy are first affected by arsenic poison.-(T)

III. FILL UP THE BLANKS WITH MOST APPROPRIATE WORDS:

1.Abnormal hoof growth is a typical symptom in ……..poisoning-(selenium)

2.Acsute symptoms of selenium toxicity resembles sub acute ……poisoning.-(lead)

3.Alkali disease is due to chronic ……..toxicity.—(Selenium)

4.Among different breeds of Dogs ……breed is highly sensitive to copper toxicity.-(Bedlington terrier)

5.Among animals ……..are highly sensitive to mercury toxicity.-(Ruminants)

6.Antimony potassium tartrate is commonly known as ……--(Tartar emetic)

7.A pigment vermillin containing ……is non toxic.-(Mercury)

8.Arsenic combines with …….. groups of enzymes,-.-(SH group)

9.Basophilic stipling of RBC is seen in ……poisoning.-(lead)

10.Black berry jam spleen in sheep is characteristic in ……toxicity.-(Copper)

11.Boiled linseed oil can cause .....poisoning.-(Lead)

12.Bone can serve as a reservoir for compounds such as …….-(Lead, Fluorine , Tetracyclins)

13.Bordeaux mixture can cause .....toxicity.-(Copper)

14.British anti lewisite is a ……containing chelating agent.-(dithiol)

15.British anti lewisite is the specific antidote for……..-(Arsenic)

16. 'Burtons line ' is seen in gum margins, it is due to deposition of .....in gums.—(Lead)

17.Characteristic diarrhea in arsenic poisoning is called as……diarrhea.-(Rice gruel)

18.Chronic selenium poisoning is otherwise known as ……-(Locoism/Alkali disease)

19. Symptoms of chronic selenium toxicity resembles …….. and symptoms of acute poisoning resembles subacute lead poisoning.-(Hypovitaminosis –A)

20.Copper napthenate is used as a ……and animals licking those wood will get copper toxicity .-(wood preservative)

21.Dark green stools may be passed by animals in.....poisoning-(Copper)

22.Depigmentation of hairs occurs in toxicity due to .....—(molybdenum)

23.Depigmentation of hairs around eyes gives a spectacle appearance in …….poisoning.-(Molybdinum)

24.Desferioxamine is the specific antidote of ……..(Iron)

25.Dietary molybdenum of ……ppm can cause toxicity regardless of copper intake.-(10ppm)

26.Discarded automobile batteries can act as a source of .....—(lead contamination)

27.Donovan solution was used against ……in turkeys .-(Histomoniasis, Black head)

28.D-B-B-dimethyl cysteine is otherwise known as ……..—(Penicillamine)

29.Eventhough, penicillamine is primarily having chelating property it is having anti…….action also.-(rheumatoid)

30.Excess molybdenum interfere with ……..storage in the liver and causes deficiency.-(copper)

31.Fowlers solution contain ……(potassium arsenite)

32.Gunmetal kidney is seen in …….poisoning.-(Copper)

33.High blood value of lead indicate ……..time exposure.-(much back)

34.High blood and feces value of lead indicate relatively ……exposure.-(recent)

35.High intake of phosphorus favors skeletal storage of ……..—(lead)

36.Highest concentration of mercury is accumulated in ……..tissue.-(kidney)

37.In arsenic poisoning the most satisfactory clinical material for lab .examination is …….-(Urine)

38.If arsenic level in the liver tissue exceed …….ppm it can be considered as toxicity.-(10 to 15)

39.In acute mercury poisoning the content in tissues may be ……ppm.(above 15 ppm)

40.Injection of iron is available in complex with carbohydrate in the form of ……-(Iron dextran)

41.In copper toxicity the peculiar nature of spleen is known as …… which may be confused with anthrax.-('Black berry jam spleen")

42.Incorporation of zinc in the ration give some protection against……..toxicosis.-(copper)

43.Indicator plants may contain ……ppm of selenium.-(more than 15000)

44.Indicator plants have ……in them to self protect from enemies.-(Selenium)
Indicator plants accumulate ……in them.-(Selenium)

45.In horse laryngeal hemiplagia and roaring is seen in chronic ……poisoning.-(Lead)

46.In swine 'sitting dog posture' is a characteristic symptoms of …….valent arsenical toxicity.-(Penta)

47.In the bone 90—98 % of the lead is stored in ……(region) where the bone is growing.-(Extremities)

48.In the body lead is permanently deposited in …….tissue.-(Bone)

49.Itai itai disease is due to …….toxicity.-(Cadmium)

50.Iron toxicity is more in ……and ……..(species of animals)-(Dogs , Pigs)

51.Iron injection may cause ……reactions .-(anaphylactic)

52.Low level molybdenum and sulphate in food increases the toxicity of another heavy metal…….-(Copper)

53.Mercuric chloride is commonly known as ……..sublimate.-(corrosive)

54.“Minamata” disease is ……..mercury poisoning.-(methyl)

55.Molybdinosis in animals shows ……..deficiency.—(copper}

56.Molybdenum has an ……relation with copper.-(inverse)

57.Molybdenum is a component of ……enzyme which convert xanthine to uric acid.-(xanthine oxidase)

58.Organic form of lead is added to petrol to increase its …….number-(Octane)

59.Organic form of arsenic exists in trivalent and …….valent form.-(penta)

60.Passive accumulators contain ……ppm of selenium.-(1 to 25)

61.Peat scour in cattle is other wise known as ……..-(Teart)

62.Perchloride of mercury is commonly known as ……..-(corrosive sublimate)

63.Pentavalent arsenicals will produce ……of nerve fibers and axonal degeneration.-(De-myelination)

64.Pentavalent arsenicals will……. oxidative phosphorylation forming unstable arsenite ester.- (uncouple)

65.Plants requiring selenium for growth is collectively called as…….-(Indicator plants/ Obligatory accumulator)

66.Plants/ feeds containing more than …….ppm of selenium is dangerous to consume.-(five)

67.Selenium prevent exudative diathesis in ……..-(chicks)

68.Selenium containing shampoos are used against ……in dogs.-(dermatosis)

69.Selenium toxicity interfere with circulation of …….. of legs and tail.-(extremities)

70.Selenium will accumulate permanently in …….tissues.-(Hoof and Hairs)

71.Selenium is an intracellular antioxidant and it prevent the accumulation of …….in the cells.-(Peroxide)

72.Some toxins like ……..is accumulated at site other than their site of action.--(lead)

73.Sodium arsanilate contain ……..valent and arsphenamine contain ……valent arsenicals.(penta, tri)

74.Sodium diethyl dithio carbamate is used in the treatment of ……. toxicity.—(Thalium)

75.Sodium arsanilate is a ……..valent arsenical.-(penta)

76.Subacute toxicity of selenium is otherwise called as……-(Blind staggers)

77.The active ingredient of CUPRIMINE is ……-(Penicillamine)

78.The active ingredient of “sindur” is……..tetroxide.-(Lead)

79.The Burtons line on gum margin is due to the deposition of ……-(Lead sulphide)

80.The commonly called ” sugar of lead” is chemically lead……..-(acetate)

81.The commonly called ”Salt of Saturn” is chemically lead……..(acetae)

82.The function of selenium in tissue is as an ……..-(antioxidant)

83.The level of arsenic in the hair of normal animal is……ppm.-(5 to 10)

84.The maximum permissible limit of mercury in drinking water is…….ppm.-(0.05)

85.The normal concentration of mercury in animal tissue is less than ……ppm(1. 0)

86.The permissible level of arsenic in drinking water is s below ……ppm.-(0.05)

87.The prominent symptoms in chronic ……toxicity is Toxaemic jaundice.-(Copper)

88.The specific treatment for arsenic toxicity is……therapy.-(BAL/ Dimercaprol)

89.The specific antidote of iron is ……-(Desferrioxamine)

The specific antidote of antimony is ……..—(BAL)

90.The specific antidote of mercury is ……..-(N-acetyl D-penicillamine)

91.Toxins like Lead is selectively deposited in ……tissue.-(Bone)

92.Ten ppm of mercury mean ……..mg of mercury / 100kg.—(10)

93.White vitriol is chemically ……(zinc sulphate)

94…….(animal) are most susceptible to copper poisoning.-(Sheeps)

95……% copper sulphate can be used as an emetic.-(one %)

96……acid dehydratase is the most important enzyme blocked by Lead.-(Delta amino levulinic)

97……..(heavy metal)toxicity may cause laryngeal hemiplagia.-(lead)

98……gas is liberated by the action of water on its pyretic ores.-(Arsine)

99……form of arsenic will not produce typical signs of arsenic poisoning.-(Gaseous)

100……..is otherwise known as Locoism/Alkali disease.-(Chronic Selenium poisoning)

101…….(metal).has a complex inter relationship with copper.-(Molybdinum)

102……..plants which contain selenium has a strong odor to self protect from animals.-(Indicator)

103…….(animal) are most susceptible to copper poisoning.-(Sheeps)

104……(enzyme)is the sensitive indicator for lead poisoning.-(δ-ALA)

105……., a trace element prevent white muscle disease in cattle.-(Selenium)

106…….can be used in the treatment of copper toxicity.-(D-Penicillamine)

107…….is the specific antidote of copper toxicity.—(D-Penicillamine)

108……..is called as parish green.—(Copper arsenite)

109. ……mg. is the toxic dose of arsenic in human.—(100 mg)

110.Cadmium toxicity is otherwise called as……disease.—('Itai Itai")

Rechargeable dry cell can cause toxicity of ……..and ……-(Nickel and cadmium)

IV. CHOOSE THE CORRECT ANSWERS FROM THE GIVEN ONES.

1.Animals comparatively resistant to the action of lead among the following is a) pigs b) dogs c) cattle d) goat e) sheep—(A)

2.Arsenicals will permanently deposited in the following tissues a) Keratin b) Bone c) Muscles d) none of the above. --(A)

3.Arsenic will bind with the following groups of enzymes. a) SH b) OH c) CO d) none of the above.-(A)

4.Arsenic will produce the following type of poisoning. a) per acute b) acute c) chronic d) all the above.-(D)

5.Arsenic can be detected in the milk after ingestion for a period up to a) 30 days b) 50 days c) 5 to 10 days.-(C)

6.Arsenic will bind with lipoic acid resulting in the inhibition of synthesis of a) Acetyl Co-enzyme-A b) Prpionyl Co-A c) Succinyl Co-A d) all the above.-(D)

7.Arsenic is deposited permanently in a) liver b)kidney c) hair d) none of the above._(C)

8.Depigmentation of hair occur in poisoning due to a) molybdenum b) lead c) arsenic d) copper e) none of the above.—(A)

9.Discarded automobile batteries can act a source of following poisoning. a) arsenic b) lead c) mercury d) copper.—(B)

10.Discarded broken CFL can act as a source of following contamination a) mercury b) lead c) arsenic d) copper-(A)

11..Donovan solution contains one of the following metal as a therapeutic agent a) Arsenic b) mercury c) lead d) copper.-(A)

12.Galvanised vessels can act as a source of poisoning with one of the following metal. a) Lead b) Tin c) Zinc d) Copper—(C)

13.Gun metal kidney is observed in the post mortem finding in poisoning with a) Mercury b) Copper c) Arsenic d) lead--(B)

14.In cattle storage of copper in liver is reduced mostly by a) molybdenum b) lead c) selenium.-(A)

15.If the level of arsenic in the liver of exposed animal is above the following it can be considered as a case of arsenic poisoning. a) 1 to 3ppm b) 5 ppm c) 10-15 ppm d) none of the above.-(C)

16.In the body molybdenum storage is greater in a) Kidney b) kidney and bone c) Liver and bone --(B)

17.In animal practice copper sulphate is used as a a) Molluscicide b) emetic c) closure of oesophagial groove d) parasitecide e) all the above.-(E)

18.In cattle occurrence of selenium poisoning is mainly due to a) contamination of feed b) contamination of air c) plants c) none of the above ,-(C)

19.Lead lines in gums is a symptom seen in lead poisoning it is seen in a) dogs b) cattle c) Human beings d) none of the above.-(C)

20.One of the characteristic symptoms of arsenic toxicity is a) Projectile watery diarrhoea with blood and shreds of epithelium b) Vomition c) Mottled teeth d) none of the above.-(A)

21.One of the following is not recommended in arsenic treatment a) Meso dimercapto succinic acid b) Penicillamine c) BAL .-(B)

22.One of the following is an arsenical insecticide a) arsenic trioxide b) Acetarsol c) Sulpharsen d) none of the above.-(A)

23.Presence of “long rocker shaped hoof “ is characteristic of toxicity due to a) Copper b) Selenium c) Arsenic d) Lead ,--(B)

24.Sodium chloride poisoning is seen in a) Pigs b) Poultry c) Fishes d) Cattle .—(B)

25.The compound which is used for chelation of Iron is a)Desferrioxamine b) Dimercaprol c) EDTA.-(A)

26.The following tissue of arsenic poisoned animal is safe for consumption. a) Milk b) Meat c) Kidney d) none of the above.-(B)

27.The most susceptible species to sodium chloride poisoning is a) Cattle b)Horse c)Dogs d)Poultry.-(D)

28. Sub acute selenium toxicity is known as a) Blind staggers b)Pine c) Limberneck d) none of the above -(A)

29.The toxic principle present in corrosive sublimate is a) Mercury b) Antimony c) Lead d) Arsenic,--(A)

30.The toxicity of BAL include a) vomiting b) tremor c) convulsion d) all the above.-(D)

31.Watery faeces with unpleasant odor and full of gas bubbles(peat scour) is seen in toxicity with a)Zinc b) Molybdenum c) Lead d) mercury.-(B)

32.Zinc ions inhibit the following enzymes a) Catalase b) Pepsin c) trypsin d) acid phosphatase e) acetyl choline esterase f) all the above.-(F)

V.MATCH EACH ONE IN A TO ALL THE MATCHING ONES IN B

A	B
1. Feed additives	Minamata disease-(6)
2. Potassium arsenite	Peat scour-(11)
3. Cupric aceto arsenite	Sulpharsen.—(1)
4. Arsanilic acid	Sugar of lead.—(7)
5. Arsenolysis.	Gun metal kidney-(10)
6. Methyl mercury	Black berry jam spleen-(10)
7. Lead acetate	Astragalus-(8)
8. Obligatory accumulator	Salt of Saturn-(7)
9. Seliniferous plants	Fowlers solution-(2)
10. Copper	Delta amino levulinate synthatase-(7)
11. Molybdenum	Pentavalent arsenical-(5) (4)
	Parish green –(3)
	Aster-(9)
	Heliotropum europium-(10)
	Disodium calcium EDTA(7)

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

A	B
1. Arsenic	Sodium diethyl di thiocarbamate.-(10)
2. Mercury	Basophilic stipling-(3)
3. Lead	Des ferrioxamine-(9) (7)(8)
4. Selenium	Teart-(6)
5. Copper sulphate	Haemosiderin-(7)
6. Molybdenum	Closure of oesophageal groove-(5)
7. Iron	D-penicillamine-(2)
8. Streptomyces pilosus	Burtons lines on gums margin-(3)
9. 'Von rose" urine	BAL-(1)
10. Thaliium	Disodium calcium EDTA-(3)
	Blind staggers-(4)
	Bordeaux mixture-(5)
	Glutathion peroxidase-(4)
	Spectacle appearance-(6)
	Alkai disease-(4)
	Imferon-(7)
	Ceruloplasmin-(5)
	Transferrinn (7)

A	**B**
1. Sulphuric acid	Donovan solution-5
2.Sodium hydroxide	Tartar emetic—4
3. Ammonia	British anti lewisite-4,5,9,7,10
4.Antimony	Rice gruel diarrhea-5
5. Arsenic	Burtons line-9
6. Copper	Vinegar-2
7.Chelating agent	Defoliant-1,5
8. Hydrogen cyanide	Gape worm-4
9. Lead	Inorganic acid-1

10. Minamata disease	Leukemia-5
11. Selenium	Basophilic stipling-9
12. Fluoroacetate	Calcium sodium EDTA-9,7
13. Coenzyme –A	Methyl mercury-10
14. Cadmium	Penicillamine-7,6,9
15. Reserpine	Japan-10
	Accumulators-11

Match each one in A to matching ones in B and C

A	B	C
1. Selenium	Nettle gas-(8)	Vermicide.-(7)
2. Blue vitriol	Astragalas-(1)	Rechargeable cells-(3)
3. Cadmium	Seldan-(1)	Bedlington terrier-(2)
4. Molybdenum	Copper sulphate-(2)	Indicator plants-(1)
5. Fluorine	Plastic industries-(3)	Black berry jam spleen-(2)
6. Mercury	Gun metal kidney-(2)	Dichloroformoxime-(8)
7. Tartar emetic	Molasicide-(2)	Closure of oesophageal groove(2)
8. Harrasing agent	Leishmaniasis-(7)	Intra cellular anti oxident--(1)

VI.ODD ONE OUT

1.Lead arsenate, arsenic trioxide, Donovan solution, cupric aceto arsenite.-----(Donovan solution)

2.Arsenic, Mercury, Lead, BAL----(BAL)

3.Per chloride of mercury, Bin iodide of mercury, Mercuric nitrate, Mercurous chloride.---(mercurous chloride)

4.Lead, Lead shots, Lead paints, lead acetate.----(lead acetate)

5.Basophilic stippling, radio opacity of long bones, Burtons lines, rice gruel diarrhea.-(Rice gruel diarrhea)

6.Astragalus, Aster, Oxytropis*, Heliotropum europaeum.---(Heliotropum europium)*

VII.UNDERLINE THE WRONG ANSWER FROM THE GIVEN ONES:

Animals comparatively resistant to the action of lead is pig / cattle.

BAL has no therapeutic use in copper/ arsenic toxicity.

Dark green coloured urine is seen in phenol/ lead poisoning.

Depigmentation of hair occurs in toxicity due to copper / molybdenum.

Discarded batteries can act as a source of mercury / lead .

In sodium chloride poisoning death is due to liver damage / disturbance in water balance.

In phenothiazine toxicity the colour of the urine will be yellow/red .

Penicillamine is an antibiotic / a chelating agent.

Rice gruel diarrhea is seen in toxicity with arsenic/ mercury.

Sodium bicarbonate is highly toxic in cattle/poultry.

The blood will be dark red in colour in toxicity due to hydrogen cyanide/ hydrogen sulphide.

The odour of breath will be bitter almond in toxicity due to phosphorus/ tapioca leaf.

The sugar of lead is lead sulphide / lead acetate.

The urine will be deep yellow in colour in picric acid /acetic acid poisoning.

VIII. ANSWER THE FOLLOWING.

1.Animals suffering from acute or subacute toxicosis with lead may not respond much to calcium disodium EDTA ,Why? – Calcium disodium EDTA will chelate only lead in the bones , not lead in soft tissues .In acute or sub acute toxicity there is only less lead in bones that is why less action.

2.Arsenic is accumulated in keratin tissues. Why?- Keratin tissues contain more of SH group ,arsenic is having more attraction towards SH group. that is why it is accumulated in keratin tissue

3.Boiled lin seed oil contain toxic amount of lead in it ,How? Lead is added while boiling the oil to increase its setting property. (used in paint industry)

4.Both high and low dietary sulphate causes molybdenum toxicity, How?--High dietary sulphate increase molybdenum toxicity by reduce copper absorption on the other hand low dietary sulphate reduce molybdenum excretion in the urine and causes toxicity.

5.Copper causes lysis of RBC ,How?—The Glutathion (which give protective anti oxidant effect) Concentration in the RBC is reduced leads to alteration in cell membrane structure and function results in lysis of RBC.

6.Elemental mercury is not toxic when ingested .Why? because of its very low absorption.

7.Fruit juice is not advisable to take in lead coated vessels ,Why?-mostly Fruit juices are acidic and that dissolve some amount of lead in it from the vessels-may cause toxicity.

8.Grasses near busy highway may contain high amount of lead .Why? lead is added to petrol to increase its octane number .Automobiles which burn this will exhaust in to the environment of highway. This is deposited in the road and nearby soil. From the road this is washed away by rain to nearby soil-accumulated by the grasses near by to give large amount of lead.

9.In lead toxicity an interval of 2 day is given between treatments. Why? drug will bind with lead in the blood only. To allow redistribution of lead in the body 2 days break is given—otherwise drug toxicity may happen later.

10.In pigs copper toxicity causes microcytic hypochromic anemia similar to piglet anemia, How?—Copper which accumulate in liver inhibits iron absorption from G.I. tract and cause microcytic hypochromic anemia.

11. It has a complex inter relation ship with Molybdenum and Sulphur. What?-(Copper has the inter relation ship with molybdenum and sulphur)

12. It is difficult to diagnose poisoning with arsenic gas Why? It will not produce typical signs of arsenic poisoning .

13.It was isolated in 1953 from urine of patients receiving penicillin, discovery of its chelating property leads to its use in heavy metal toxicosis. What was this chemical agent.---(Penicillamine)

14.Lemon juice is not advisable to take in lead coated vessels. Why? Lemon juice is acidic in nature which dissolve lead in it from vessels–while consuming this can cause lead toxicity.

15.What are the common causes of poisoning give eg. A) Naturally occurring hazard eg. Chronic Copper poisoning. B) Man made hazard.eg. contamination of pasture with fluorine from industries and poisoning due to grassing on the land, pesticide and herbicide contamination, Drug overdosing etc.

16. Molybdenum and copper has inverse relationship, How? Thiomolybdate produced in the rumen by molybdates and sulphides reduce the availability of dietary copper when absorbed impede the metabolism of tissue copper. Molybdenum promote hepatic copper excretion by forming copper molybdate complex that is excreted by kidney causing copper deficiency.

17.Molybdenum cause froathy diarrhea ,Why?-Molybdenum blocks the normal antibacterial substance catechols in the rumen which cause growth of bacteria and gas production.

18.Since copper is having inverse relationship with this element , in toxicity with this element copper glycinate injection can be given .In what toxicity?.-Molybdesnum

19.Their growth indicates high concentration of selenium in the soil ,What?—(Indicator plants eg. Astragalas)

20.What are the clinical signs of mercury toxicity; ? Elemental mercury-nausea, vomiting, cough, pneumonia, interstitial emphysema, pneumothorax . Chronic exposure –neurological disturbances-depression, irritability, vasomotor distrurbances hydrothorax, hydropericardium, haemorrhage in to epi and myo cardium. Inorganic – corrosion of the mucous membrane of gastro intestinal tract, intense pain, vomiting, bloody diarrhea, stomatitis, ulcer, colitis, Alopacia, shedding of teeth. Organic mercurials- Symptoms develops slowly 7-21 days, CNS disturbances, ataxia, paresis, erythema of skin, muscle tremors, hearing loss, conjunctivitis, lacrimation, blindness, stomatitis.

21.What are the occurrences of mercury in the environment: Metalic mercury-grey powder, Mercury vapour. Oxides of mercury-yellow and red oxide ointment of mercury used as medicine. Chloride of mercury-mercurous chloride used as toxic purgative, mercuric chloride used as antiseptic, wood preservative and anatomical specimen preservation. Iodides of mercury-Red iodide or bin iodide used for the preparation of blistering ointment. Mercuric sulphide-pigment vermillin.

22.What are the sources of copper toxicity? Accidental administration of excess amount of copper salt in mineral mixture, copper from sprayed foliage or pasture grass with Bordeaux mixture, used as foot bath fungicides, anthelmintics in vety. Practice, consumption of water from contaminated ponds, contamination of forage and Pasteur or soil in the vicinity of mines and smelters may cause poisoning.

23.What are the sources of lead toxicity? Accidental ingestion of lead objects such as batteries, gunshots, lead solders. Licking of lead based paints (freshly painted area, dry pealing paint, lead primer, putty). Grasses near busy highways-get lead from auto exhausts – now a days reduced because of use of lead free petrol. Pasteur contaminated with atmospheric fallout from smelter and mining operation, vapour and fumes from lead based industries. Use of fungicides and herbicides, parasiticides. Use of drinking water passing through lead pipes.

24.What are the sources of mercury toxicity: Ingestion of organic or inorganic mercury salt, Occupational exposure in industries, electrical equipment, thermometer, mirror, chloralkali, CFL. Volatile mercury from fossil fuel. volcanic ash. Contamination of forage /food with sewage sludge containing industrial waste.

25. What are seliniferous plants (Facultative accumulators) they do not require selenium for growth, since it is present in soil they accumulate (1/10 con of obligatory) Aster, Atriplex.

26.What is the protective mechanism in the body against lead toxicity? It binds with a cytoplasmic protein called metallothionin and gradually excreted in the bile.

27.What will happen if more disodium calcium EDTA is administered in lead toxicity?—when too much disodium calcium EDTA is administered much lead is mobilized from bone to blood which could not be handled by the kidney .hence blood level of lead will increase and acute toxicity occurs.

28. What are the symptoms of lead toxicity ?The symptoms may not seen till 2-3 days, Nervous symptoms are-blindness, rolling of eye ball, pushing against fences, wandering in circles. Grinding of teeth, frothy mouth, muscular trembling. \, weak pulse, diarrhea, colic, anaemia. Basophilic stippling of

RBC, stimulate SGOT, SGPT. In chronic case anorexia, wasting, muscular weakness, grinding of teeth, wandering in circles. Lead lines on gums, opisthotonus, clonic and tonic convulsion, in horse laryngeal hemiplagia- roaring.

29.Why EDTA sodium salt as such is not advisable in the treatment of lead toxicity?—If lead toxicity is treated with EDTA sodium salt alone it will chelate the blood calcium only not with lead , hypocalcemia may set in .Disodium calcium EDTA is having more affinity to lead and it wont bind with blood calcium to produce hypocalcemia.

30.Write in the order of increasing toxicity selinide, selinite, selinate.—-(Selinide—Selinate—Selinite.)

IX.WRITE SHORT NOTES ON:

1.Blind staggers: (sub acute selenium toxicity is called blind staggers) develops In large animals and swine consuming moderate amount of 20-25 ppm-obligate or facultative indicator plants or sodium selinite in diet over a period of weeks or months. Animal tends to wander or walk in to objects in its path , vision is impaired, wandering increase front legs become weak and vision is more impaired. Throat and lung become paralysed, temperature is subnormal, death follows from respiratory failure.

2.British anti lewisite: BAL (2-3 Dimercaprol propanol) clear colourless viscous oily liquid with pungent odor. Peanut oil is the vehicle . It is a dithiol containing chelating agent form nontoxic easily excretable complex with arsenic . Relatively ineffective unless given at early phase because aging will delay regeneration. If arsenic is present in large amount BAL can not work. BAL is toxic (its signs include vomiting tremor, convulsion) For large animal 3mg/kg i/v route(5% solution in 10% of benzyl benzoate in peanut oil) repeat every four hours for first two days , every 6 hours for next 3 days, every 12 hours for next 10 days or until recovery. Small animals 2.5 mg/kg 10% solution in same dose interval.

3.Desferrioxime :It is an iron chelate from *Streptomyces pilosus*- high affinity to ferric iron- orally less active-some are allergic to it. in- vitro binds with or remove iron from haemosiderin, ferritin, transferrin and haemoglobin but not from cytochromes. Desferioxamine mesylate “Desferal mesylate) -- contra indicated in pregnancy

4.D-penicillamine: It is otherwise known as COUPRIMINE. First isolated in 1953 from urine of patients receiving penicillin . Discovery of its chelating propery leads to its use in heavy metal toxicosis. It is a good chelator of copper, mercury, zinc, lead and promote their excretion in the urine. Commercialy prepared by hydrolytic degradation of penicillin. There is no antibacterial activity. Active orally. It has anti rheumatic action also. Toxicity: chronic- urticaria, nausea, vomition, diarrhea, renal toxicity. Contra indicated in pregnancy, previous history of penicillin induced a granulocytosis.

5.Inter relation ship of Molybdenum with other elements which influence the toxicity: It is having inverse relation with copper. Excess molybdenum interfere with copper storage in liver causing copper deficiency and subsequent symptoms. High dietary sulphate increae the molybdenum toxicity by reducing copper absorption. But on the other hand low dietary sulphate reduces the molybdenum

excretion in the urine and this increases its toxicity. High inorganic phosphorus hasten the excretion of molybdenum in urine. The toxicity of molybdenum is increased by zinc.

6.Indicator plants: (obligatory accumulator) Require selenium for their growth. Their growth indicate high concentration of selenium in soil. These plants may contain 15000 ppm . when these plants decay selenium will be in top soil and this contamination become a source for other plants . Astragalus , oxytropis, *Acacia cana*. They have strong odor hence animals will not take it.

7.Iron toxicity: absorbed ferrous iron is oxidised to ferric form and bound to transferring .Excess iron absorbed over a period of time is stored as haemosiderin or ferritin. Iron directly damage the G.I.mucous membranes. Excess iron circulate in the plasma as free iron (not sufficient transferrin to handle this) It is a strong oxidant and corrosive. Liver damage , increase capillary permeability, metabolic acidosis. By injection it may cause anaphylactic reactions.

8.Mechanism of action of selenium toxicity: There are three oxidative state-selinate, selinite.selinide. Selenite is more toxic, absorb via G.I.tract , accumulate in kidney, liver, and higher amount in hoof and hair. It has some essential function-act as an intracellular anti oxidant prevent peroxide accumulation. It reduce peroxide by glutathions in the presence of selenocystein containing enzymes Glutathion peroxidase.

9.Mechanism of Arsenic toxicity:-Two form of arsenic is there Trivalent and pentavalent –pentavalent as such is less toxic but converted to trivalent form which is more toxic. Trivalent primarily binds to SH group of compounds especially lipoic acid and alpha keto oxidase - inhibits the activity of many enzymes -Pyruvate dehydrogenase system is highly sensitive-Lipoic acid is a co factor for decarboxylation of keto acids results in inhibition of formation of acetyl , succinyl, propionyl co-enzymes A. It also inhibits a group of oxidative enzymes. Glycolysis,TCA, energy production is reduced. Highly dividing cells like intestinal epithelium, kidney, liver, skin, and lungs are more affected. Loss of capillary integrity and dilation of it in GI tract, hypotension, shock,. Pentavalent form insane uncoupler of oxidative phosphorylation-arsenolysis. De-mylination and axonal degeneration. Arsine gas cause haemolysis, pulmonary oedema,

10.Mechanism of copper toxicity. Accumulate in liver and affect the integrity of plasma membranes, damages the lysosomal membranes, hepatocyte damage, inhibit essential metabolic enzymes like dihydrolipoyl dehydrogenase leading to reduction of pyruvate and alpha keto glutarate, gastro intestinal irritation and erosion of GI lining. Excess copper in blood oxidize erythrocyte membrane and increase their fragility result in lysis , excess haemoglobin clog the renal tubules result in tubular and glomerular necrosis and produce darkened gun metal grey kidney. In swine copper accumulation inhibit the absorption of iron from G.I, tract leading to iron deficiency anaemia. oxidative phosphorylation- damage to cellular

11.Mechanism of iron toxicity: it is corrosive to the GI tract- cellular toxicity- hepatotoxicity- mitochondrial poisoning, impair oxidative phosphorylation- damages the cellular structures like lipids, nucleic acids, proteins and carbohydrate. Increase the capillary permeability ,hypotension and cardiac

collapse. Anaphylactic reactions especially parenteral injections of iron. Calciphylaxis can occur with several days of iron injection.

12.Mechanism of action of mercury toxicity: interact with sulphydryl group of enzymes and other proteins , form covalent bonds with sulphur. Block several biochemical reactions and metabolic process. One important enzyme affected is choline acetyl transferase which is involved in acetyl choline synthesis, affect motor dysfunctions. Corrosive irritant action. Inorganic mercury cause tissue necrosis and renal tubular damage, corrosive action on GI tract, hepato toxicity action. In addition to SH group also affect CONH2, NH2, COOH and PO4 group and block the action of several enzymes like glucose-6 phosphatase, alkaline phosphatase, ATP ase etc. It disturb immune function, disrupt ion exchange across voltage gated and ligand gated channels. Methyl mercury increase the production of reactive oxygen species.

13.Mechanism of lead toxicity: Lead decreases the concentration of essential trace element such as zinc, copper, and iron and interfere with these metals to act as prosthetic group of enzymes on mitochondria- affecting cellular respiration. Damage the Blood brain barrier- cytotoxic agents enters the brain capillaries –endothelial damage- cerebral oedema and haemorrhage. Causes segmental demylination of motor nerves, affects action of several neurotransmitters like GABA, and dopamine in CNS, inhibits NMDA receptors. Causes rapture of lysosomes – inhibits brain adenyl cyclase- leads to encephalo malacia. Interfere with flux of ions in and out of brain inhibits Na+ K+ ATPase . Immuno suppression-reduce production of antibodies in certain viral and bacterial diseases. Very low concentration of lead to inhibits the synthesis of heme at several steps. Inhibits delta amino levulinate dehydratase. It blocks the conversion of coproporphyrinogen III to protoporphyrin Ix and then to heme by inhibiting ferrochelatase. inhibits pyrimidine 5 nucleotidase which is responsible for basophilic stippling.

14.Mechanism of selenium toxicity : It replace sulphur in amino acids- result in abnormal structural and enzymatic protein-explain hoof and hair defect in chronic poisoning. Causes depletion of glutathione- stimulate liver and erythrocyte glutathione peroxide activity. Catalise the activity of superoxide anion, hydrogen peroxide and other reactive metabolite. Depress ATP formation and tissue ascorbic acid.

15. Minamata disease is a disease condition caused mercury—Minamata is a small town in Japan. Major industries around it empty its effluents directly in to Minamata bay. Microbes convert inorganic mercury in the effluent in to methyl mercury. The compound is then taken up rapidly by plankton algae and then by fishes. It is concentrated in fish about 30000 fold via food chain. Resident of that area who consumed the fish were first to be poisoned over 100 people died. Many others affected by the neurological damage .

16.Occurrence of Arsenic as a source of poisoning in animals: a) Arsenical pyrites- on roasting of metallic ores arsenic trioxide ,arsenic, inorganic arsenic may contaminate. b) Insecticides- Sodium , potassium and lead arsenate ,monosodium methane arsonate. c) Defolient/Herbicides- sodium potassium arsenite. e) Livestock dips- Lead arsenate. f) Rodenticides-Arsenic trioxide. g)Anti parasitic injection-Sodium thiocetarsamide. h) feed additives-acetarsol, Neoarsphenamen. i) Medicine-

Potassium arsenite. j)Arsine gas. K) wood preserved with arsenical compounds. Over dose-accidental ingestion –ingestion of forage, water contaminated with any of the above arsenical compound can cause toxicity.

17.Peat scour/ Teart (Molybdinosis): Peat scour the toxicity due to Molybdinum- mostly seen in cattle-they are more susceptible-Noraml bacterial growth in the rumen is controlled by catechols which are like other phenols having bacteriostatic activity. Molybdenum binds with this catechols and hence over activity of these bacteria cause diarrhea in 8-10 days, watery faeces with unpleasant odor and gas bubbles-called as peat scours or teart in cattle. Locomotor difficultys- stiffness of joints- hair coat dull-de pigmentation especially around eyes giving spectable appearance.

18.Sources of Arsenic poisoning: Animals gaining access to arsenical dips, contamination of herbage by weed killer/dips, contaminated water and herbage near metal smelting work, ingestion of Arsenical rat poison, Animal licking wood preserved with arsenic or contaminated pasture with ash of such wood, indiscriminate use of arsenical medicine, over dosage organic arsenic feed additives, calf taking milk from arsenic poisoned cow, Malicious poisoning with arsenic trioxide.

19.Sources of mercury poisoning: Ingestion of grains and seeds treated with organic mercurial fungicide eg. Ethyl mercuric chloride. Occupational exposures in electrical equipment industries. Antifoul paints, small batteries, thermometer, mirrors, semiconductor cells, fluorescent and mercury lamps, infrared detectors, chloralkali, plastics, dental amalgam. Ingestion of fish contaminated with organic mercurials such as methyl mercury. Mercury vapours from fossile fuels dissolve while raining and contaminate water and Pasteur. Indiscriminate use of mercury containing drugs, use of thiomerasol, organic mercurials as preservative. Contamination of water with sewage sludge containing industrial waste.

20.Symptoms of arsenic poisoning.: Per acute case is very rare- animal found dead. Acute case is very severe –gastro-intestinal symptoms-gastro enteritis, colitis, salivation, regurgitation, staggering gait, projectile watery diarrhea, blood in the faeces. Hind limb paralysis, sub normal temperature, death in 1 to 3 days. Skin contamination causes massive skin necrosis. In sub acute case animal may live for several days. In chronic case : it is rare- wasting , poor condition, brick red mucous membrane, weak irregular pulse, pentavalent causes nervous signs.

21.Symptoms of selenium toxicity: There is acute, subacute, and chronic forms . Acute- dark watery diarrhea, bloat, colic, cyanosis, rapid pulse, dyspnoea ,labored breathing, blood tinged frothing from nostril in 1—2 days, prostration, coma, death due to respiratory failure. usually rare because animals avoid taking it . Subacute –(Blind staggers)-giving feed containing 10—20ppm of selenium for weeks or months, salivation, lacrimation, blindness, wander in circles, and head pressing, vision impaired, throat and tongue become paralysed. Chronic (Alkali disease) locoism- alopecia, loss of hair from mane and tail, rough coat, lameness, abnormal hoof growth, sloughing , teratogenic, interfere with circulation at extrimities of legs and tail

22.Treatment of copper poisoning: copper excretion may be increased by use of copper chelators like penicillamine, ascorbic acid reduce the absorption of copper in dogs, ammonium molybdate and sodium sulphate reduce copper absorption , ammonium tetrathiomolybdate stimulate binding and excretion, oral zinc reduce absorption of copper. Supportive therapy-antioxident. Alkalinization of urine, supplementation with molybdenum.

23.Treatment of lead poisoning: Di sodium calcium EDTA - for chelation1-2% solution in 5% dextrose 110mg/kg i/m or s/c BID for 2 days give a break for 2 days to allow the redistribution of lead to bone -(chelate only bone lead and not lead in soft tissue) then repeat the treatment for 2 days again give a break continue for 10 to 14 days- calcium is exchanged for heavy metal ion- form soluble chelate and increase urinary excretion .Animals suffering from acute or subacute toxicosis may not respond much (as there is less lead in bones) Dog 1% sol. In 5% dextrose-25 mg/kg 4 times a day for 5 days s/c. It should not exceed 2g/day . When inject more much lead which could not be handled by the kidney is mobilized. Blood level increase acute symptoms may develop. EDTA itself can not be given because it chelate with calcium in the blood. d-penicillamin-Dog 110 mg /kg daily for 2 weeks. Supportive therapy-Anti convulsants like diazepam, phenobarbitone, osmotic cathartics, enema, glucose saline, parenteral fluids, broad spectrum antibacterial agents thiamine, steroids, osmotic diuretics, zinc suppliments, diazepam.

24.Symptoms of iron toxicity: Haemorrhagic gastro enteritis, diarrhea, vomiting, cyanosis, dehydration, shock, acidosis, haemolytic anemia. By injection it can damage the muscles around the site, muscle tremor, convulsion, shock and death in 60 min to 4 days. Acute form mostly affect the piglets , less acute form affect body defense mechanism, E coli enteritis and death. In another form massive mobilization of calcium following injection of iron –death after few days

25.Toxicity with Pentavalent arsenicals : It produce de- mylination and axonal degeneration – interfere with B vitamins- nervous signs- organic arsenicals liberate arsenic slowly and excreted faster- It uncouple oxidative phosphorylation forming unstable arsenite ester that are rapidly hydrolysed (instead phosphate ester which are stable) known as arsenolysis.

26. Treatment of Arsenic poisoning: Specific therapy is Dimercaprol/ British anti lewisite(BAL). It form a non toxic easily excretable complex with arsenic, aging will delay the regeneration. If large amount is present BAL is not effective and if administered it will produce toxicity. For large animal 3 mg/kg i/v repeat at every 4 hours for first 2 days then reduce to every six hour for next 10 days until recovery. Small animals 2.5 mg/kg. Thioctic acid 50mg/kg i/m tid as 20 % solution. Sodium thiosulphate react with arsenic and immobilize it . Mesodimercaptosuccinic acid, Dimercaptosuccinic acid are also effective. Supportive therapy –emetics, activated charcoal, gastric lavage and other symptomatic therapy.

27.Write on treatment of mercury toxicity: Row egg white beaten, gastric lavage with saturated sodium bi carbonate. Activated charcoal, magnesium sulphate. Dimercaprol sodium sulphonate (Unitiol) 3mg /kg i/m 4 hourly for first2 days, then six hourly for next 3 days, followed by 12 hourly for next 10 days or until recovery. N-acetyl D-penicillamine 15.5 mg/kg daily. Sodium thio sulphate 20% 10 ml/45 kg, i/v 4 dose at 8 hour intervals.

X.WRITE ESSAYS ON:

1.Explain the chances , pathogenesis, symptoms and treatment of Mercury poisoning in animals

2. Explain the occurrence, pathogenesis, symptoms and treatment of Lead poisoning in animals.

3.What are the mechanism of Lead toxicity in animals?

4.Lead as a toxic metal –chances of toxicity, mechanism of action, symptoms and treatment

5..Explain the occurrence ,pathogenesis , symptoms and treatment of Lead poisoning

6..Pentavalent arsenical toxicity in animals.

COURTESY

Dr. A . M. C. Nair & N.G. Kumar

Professors pharmacology (Rtd)

COVAS, Mannuthy, Thrissur, kerala

Suggestions and criticism to <amcnair50@ gmail.com>