Model Answer of Nursy exam 2016

I. Enumerate: (14 Marks)

1. Sulfate Containing GAGs:

1- Chondroitin sulfate
2- Dermatan sulfate
3- Keratan sulfate
4- Heparin:
5- Heparan sulfate:

2.Gluconeogenic substrates.

1. Lactate. 2. Glucogenic amino acids.
3. Glycerol. 4. Odd chain fatty acids:

3.Steriod hormones:

Sex hormones: estrogen, progesterone and androgen
Corticoids: mineralocorticoids and glucocorticoids

4.Management of ketosis:

1-Glucose intravenous in fasting or starvation.
2-Glucose and insulin in diabetes mellitus.
3-Bicarbonate to correct acidosis.
4-K^+ in hypokalemia (especially in diabetics) and fluids in dehydration.

5.Essential amino acids:

- Valine - Isoleucine- Threonine - Arginine - Leucine.

- Lysine- Tryptophan- Methionine - Phenylalanine. - Histidine

6.Causes of hemolytic jaundice (hemolytic anemia):

1. Abnormal hemoglobin e.g. sickle cell anemia and thalassemia.
2. Congenital spherocytosis.
3. Erythroblastosis fetalis: Rh -ve mother has Rh +ve fetus (due to Rh +ve father).
4. Favism, due to deficiency of G6PD.
5. Incompatible blood transfusion.
6. Some diseases e.g. malaria and black water fever.

7.Importance of Krebs (TCA) cycle regarding energy production:

3 NADH (through ETC) → 7.5 ATP.
1 $FADH_2$ (through ETC) → 1.5 ATP.
Substrate level (by succinate thiokinase) → 1 ATP.
Total gain =10 ATP

II. Give an account of: (35 Marks)

1. Buffers:

- Solutions that resist changes in their pH when moderate amounts of acids or bases are added.

Composition and Types:

- A weak acid and its salt with strong base:
  - Carbonic acid/Na-bicarbonate mixture (H_2CO_3 / $NaHCO_3$).
- A weak base and its salt with strong acid:
  - Ammonium hydroxide/ammonium chloride mixture.

Mechanism of Action:

1. Addition of a strong acid as HCl to carbonic/bicarbonate system:

$NaHCO_3 + HCl \rightarrow NaCl + H_2CO_3$.

2. Addition of a strong base as NaOH to carbonic/bicarbonate system:

$H_2CO_3 + NaOH \rightarrow NaHCO_3 + H_2O$.

- **Addition of excess amounts** of acids or bases to a buffer system may cause its depletion with marked change in the pH of the solution.

2.Importance of hexose monophosphate pathway (HMP) in red cells:

HMP produces NADPH providing reduced glutathione for removal of H2O2.

Favism is due to genetic deficiency of glucose-6-phosphate dehydrogenase (G6PD), where the red cell capacity to protect itself from oxidative damage is markedly decreased (due to decreased concentration of NADPH). Administration of drugs (premaquine, aspirin or sulfonamides), which stimulate the production of H2O2 or eating fava beans produce hemolysis of the fragile red cells.

3.Causes and types of hypoglycemia:

I. Fasting Hypoglycemia:

1. Hyper-insulinism.
2. Hyposecretion of anti-insulin hormones.
3- Liver diseases. 4- Chronic renal diseases.
5- Hereditary metabolic disorders:
a-Von Gierk's disease.
b- Genetic defects that produce impairment of FA oxidation.

II. Postprandial hypoglycemia:

Temporary drop of blood glucose that occurs about 2-5 hours after a carbohydrate meal.

Its causes include the following:

1- Alimentary postprandial hypoglycemia: after gastrectomy
2- Reactive hypoglycemia: if prolonged it indicates exagerated insulin response or decreased activity of anti-insulin hormones
3- Hereditary metabolic disorders:
a)Hereditary fructose intolerance. b) Galactosemia.

4.Causes of hypercholesterolemia:

1. Diet rich in CHO, saturated FA and cholesterol.
2. Diabetes Mellitus.
3. Hypothyroidism as thyroxin stimulates conversion of cholesterol to bile acids.
4. Obstructive jaundice due to ↓ excretion of cholesterol and bile acids.
5. Obesity
6. Coffee drinking and cigarette smoking.
7. Familial hypercholesterolemia.

5.Metabolic changes in Diabetes Mellitus (DM):

- There is ↓ in insulin/anti-insulin ratio, producing:

I. Changes in Carbohydrate Metabolism:

-↓ G uptake and oxidation by tissues, which ↓ ATP, leading to muscle weakness.
↓ glucose utilization (glycogenesis and lipogenesis).
↑ glucose formation (glycogenolysis and gluconeogenesis).
The net result is hyperglycemia.

II. Changes In Lipid Metabolism:

- ↓ lipogenesis and ↑ lipolysis in adipose tissues produce ***loss of weight*** and ↑ FFA.
- Fatty liver, hypercholesterolemia and hypertriacylglycerolemia.
- ↑ FFA oxidation *increases ketogenesis* in liver which may lead to *ketosis* in sever cases of DM and *acidosis* (***diabetic ketoacidosis***).

III. Changes in Protein Metabolism:

- ↑ *protein catabolism* and ↓ protein synthesis (*negative nitrogen balance*).
- ↓ protein synthesis leads to ↑ *sensitivity to infection* and *delayed healing of wounds*.

IV. Other Changes:

- Glucosuria produces ***polyurea***, ***dehydration*** and ***polydepsia***.
- ***Polyphagia*** due to decreased glucose utilization by brain centers.
- ***Polyurea*** leads to loss of electrolytes (Na^+ and K^+).
- Proteinuria in kidney damage.
- Atherosclerosis, hypertension, kidney failure, myocardial infarction, blindness and neuropathy, due to damage of the vascular system.
- ***Diabetic cataract, retinopathy, nephropathy*** and ***neuropathy***.

6.Liver function tests related to protein metabolism:

1. Plasma Albumin/Globulin Ratio:

It decreases in all liver diseases, due to ↓ albumin synthesis.

2. Prothrombin Time and Concentration:

Liver diseases produce hypoprothrombinemia & prolonged prothrombin time.

3. Plasma Ammonia Level: ↑ in liver failure or hepatic coma.
4. Serum ALT & AST: ↑ in hepatocellular damage.
5. Serum Alpha Fetoprotein (AFP): ↑ in liver cancer.

7.The difference between hypoglycemic and hyperglycemic coma:

Hyperglycemic Coma	Hypoglycemic Coma
Due to ketoacidosis.	Due to overdose of insulin or oral hypoglycemic drugs.
Urine contains excess amounts of glucose and ketone bodies.	Urine is free from glucose and acetone.
Treated by intravenous insulin with glucose and correction of acid base balance and disturbed electrolytes.	The only treatment is glucose administration.

III. Problem solving: (11 Marks)

a) Mention the normal level of ammonia. (3 marks)

Normal plasma level of ammonia is 0.05 to 0.1 mg/dL.

b) What is the cause of this condition. (2 marks)

Hyperammonemia is due to genetic defects of urea cycle enzymes.

c) On biochemical base explain: (6 marks)

1. **The cause of brain toxicity:**

   -High levels of serum ammonia are toxic to the brain and can cause brain damage.

   -Ammonia toxicity may be explained by withdrawal of α-KG from citric acid cycle to form glutamate and glutamine.

   -Encephalopathies produce convulsions and if not diagnosed early, it may produce mental retardation.

2. **The cause of respiratory alkalosis:**

- In cases of hyperventilation, with increased loss of CO_2 and decreased H_2CO_3 in blood as in fevers and high altitudes.