LIVER

1. Hepatic encephalopathy is a result of:

    1. Liver damage.

    2. Terminal liver failure.

    3. Alcoholic fatty liver dystrophy.

    4. Diabetic steatosis.

    5. Thrombosis of the hepatic veins.

2. In hepatic encephalopathy the hyperammoniemia is mainly a result of:

    1.Increased desamination of aminoacids.

    2. Impaired renal ammoniogenesis.

    3. Suppresion of the ornithine cycle in the liver.

    4. Entering of ammonia in the organs is slowed down.

    5. Hyperglutaminemia.

3. The increased levels of fatty acids with short and middle sized chains in hepatic encephalopathy is due to:

    1. Increased production in the colon and fat tissue.

    2. Disturbed esterification in the liver and insufficient extraction from the blood.

    3. Increased mobilisation from the fat tissue.

    4. Increased fatty acid synthesis from carbohydrate sources.

    5. Primarily increased renal retention.

4. Increased GABA level in the blood in hepatic encephalopathy is due to:

    1. Increased production in the colon.

    2. Disturbed receptor binding and exiting the nervous system.

    3. Decreased metabolisation in extrahepatic structures.

    4. Disturbed esterification in the liver and insufficient extraction from the blood.

    5. Spontaneously increased production.

5. How does the “hyperammoniemic theory” explain the pathogenesis of hepatic encephalopathy:

    1. Decreased energy in the brain.
    2. Direct toxic effect.
    3. Synthesis of false neurotransmitters.
    4. Neuronal mutagenic effect.
    5. Disturbed interaction between neurons and glial cells.

6. The accumulation of the false neurotransmitter octopamine in the CNS is a direct effect of:

    1. Increased transportation of aromatic aminoacids from the blood to the brain.
    2. Large ammounts of phenylalanine in the brain.
    3. Accumulation of tyrosine in the brain cells.
    4. Disturbed transformation of DOPA into dopamine.
    5. Disturbed transormation of dopamine into catecholamines.

7. Why does tyrosine accumulate in the brain during hepatic encephalopathy?

    1. Inhibition of tyrosine hydroxylase.
    2. Increased concentration of phenylalanine in the brain.
    3. Activation of phenylalanine hydroxylase.
    4. Blocked transformation of noradrenaline into adrenaline.
    5. Increased levels of octopamine in the brain cells.

8. What is the role of octopamine in the pathogenesis of hepatic encephalopathy?

    1. Blocks the synthesis of catecholamines in the brain.
    2. Competitively displaces dopamine and noradrenaline in the respective synaptosomes.
    3. Has a direct toxic effect on the CNS.
    4. Takes the role of a real mediator in the brain.
    5. 1, 3.

9. If bilirubin cannot enter the hepatocytes the result would be what type of jaundice?

1. Prehepatic unconjugated.
2. Posthepatic conjugated.
3. Hepatic unconjugated.
4. Hepatic conjugated.
5. Posthepatic unconjugated.

10. Prehepatic jaundice is present in which of the following?
    1. Massive hemolysis.
    2. Liver disease.
    3. Obstructed bile ducts.
    4. Intrahepatic cholestasis.
11. In what condition bilirubin cannot enter the hepatocyte?
    1. Deficiency of the proteins Z and Y.
    2. Deficiency of a special membrane-linked protein.
    3. High levels od the complex albumin-bilirubin.
    4. Deficiency of glucuronic acid.
    5. Inhibited UDPGT.
12. The conjugation of bilirubin in the hepatocytes is mainly disturbed in what condition?
    1. Low levels of the proteins Z and Y.
    2. Deficiency of a special membrane-linked protein.
    3. Inhibited UDPGT.
    4. Deficiency of glucuronic acid.
    5. Massive hemolysis.
13. Dubin-Johnson syndrome is due to:
    1. Impaired conjugation of bilirubin in the hepatocytes.
    2. Deficiency of a special membrane-linked protein.

3. Deficiency of the transport proteins Z and Y.

4. Defective excretion of the conjugated bilirubin.

5. Obstructed bile ducts.

14. Cholestatic jaundice is due to:

1. Disturbed captation of bilirubin.

2. Disturbed bile transport.

3. Disturbed bilirubin conjugation.

4. Defective excretion of the conjugated bilirubin out of the hepatocyte.

5. Massive hemolysis.

15. What mechanisms are involved in the pathogenesis of drug hepatotoxicity?

1. Direct suppression of protein synthesis.

2. Binding to proteins and forming antigens.

3. Prominent chromozome anomalies.

4. Mitotic burst.

5. 1, 2.

6. 1, 3, 4.

16. Which of the following is the most common etiological factor damaging the liver?

1. Heart failure.

2. Hepatotropic viruses.

3. Bacteria.

4. Parasites.

5. Snake poisoning.

17. What mechanisms are involved in alcoholic liver damage?

1. NADH-2 – dependant liver lipogenesis.

2. Changes in the phenotype of the hepatocytes.

3. Impaired VLDL secretion.

4. Membrane damage of the hepatocytes.

5. 1, 2, 4.

6. 1, 3, 4.

18. What are the possible outcomes from an acute hepatitis?

1. Developing of cirrhosis.

2. Recovery or chronification.

3. Acute liver failure.

4. Malignisation.

5. 2, 3.

6. 1, 2, 3, 4.

19. What is the leading cause of hepatitis chronification?

1. Provocation of a genetically-determined liver remodelling.

2. Immune-dependant liver damage.

3. Direct viral lesion of the hepatocytes.

4. Systemic reaction of mono- and phagocytes.

5. 1, 3.

20. Which cells are mostly responsible for the fibrosis in cirrhosis?

1. Endothelial cells of the sinusoids.

2. Kupffer cells.

3. Immobilised monocytes.

4. Pseudolobated hepatocytes.

5. Ito cells - lipocytes.

21. Prominent portal hypertension is present in portal pressure above:

1. 5 cm H_2O.

2. 10 cm H2O.

3. 20 cm **H2O**.

4. 50 cm H2O.

5. 100 cm H2O.

22. What is the most common mechanism of portal hypertension in liver cirrhosis?

1. Intrahepatic presinusoidal block.

2. Posthepatic compressive block.

3. Prehepatic obstructive block.

4. Intrahepatic postsinusoidal block.

5. Combined pre- and posthepatic block.

23. What is the most important pathogenetic unit for ascites development in liver cirrhosis?

1. Portal hypertension.

2. Hypoalbuminemia.

3. Impaired lymphatic drainage.

4. Increased capillary permeability.

5. Inhibited secretion of natriuretic factors.

24. Which is the second most important factor for ascites development and directly participates in the stabilisation of the condition?

1. Increased membrane permeability.

2. Low oncotic pressure.

3. Impaired lymphatic drainage.

4. Lowered peritoneal antipressure.

5. Renal retention of water and salts.