# USMLE-STEP-1 Q&As

United States Medical Licensing Step 1

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**QUESTION 1**

A 55-year-old male diabetic has an accommodative power of the lens of 10 dioptres. His near point is located 5 cm (2 in), his far point 10 cm (3.9 in) in front of the eye. Which of the following statements are correct?

A. his corrective lenses are convex

B. his corrective lenses have a positive dioptric value

C. the patient has hyperopia

D. the patient is capable of driving a car without corrective glasses

E. the patient is functionally blind

Correct Answer: E

Section: Physiology Functionally blind means that a person has a visual impairment, that does not qualify as "legally blind" but results in substantial impediment. With a near point of 5 cm and a far point of 10 cm, the man has a severe case of myopia, not hyperopia as stated in choice C. The total convergence power of the relaxed eye with normal vision is approximately 60 dioptres, and the cornea accounts for more than two-thirds of that (40 dioptres). The accommodative power of the lens is about 20 dioptres in the very young, about 10 dioptres at age 25, and would be around 1 dioptre at the patient\\'s age, if he had normal vision. For young adults with normal vision, the near point is about 10 cm from the eye; the far point is at infinity. The corrective lenses for the myopic eye are concave, not convex (choice A). Concave lenses compensate for the excessive positive dioptres of the myopic eye. These lenses are thin in the middle and wide at the edges and have negative dioptric values, not positive ones (choice B). The patient definitely won\\'t be able to drive a car or perform other activities that require fast accommodation without corrective glasses (choice D).

**QUESTION 2**

A 12-year-old boy has suffered from chronic sinopulmonary disease including persistent infection of the airway with Pseudomonas aeruginosa. He has constant and chronic sputum production as a result of the airway infection. Additionally, he suffers from gastrointestinal (GI) and nutritional abnormalities that include biliary cirrhosis, meconium ileus, and pancreatic insufficency. The symptoms are classical for which of the following disorders?

A. congenital adrenal hyperplasia

B. CF

C. Renal Fanconi syndrome

D. sickle cell anemia

E. Tay-Sachs disease

Correct Answer: B

**QUESTION 3**

A 75-year-old woman, a life-long smoker with Type II diabetes, dies secondary to complications of alcoholic cirrhosis. At autopsy, a hard, infiltrative mass is observed in the head of the pancreas. Microscopic examination of the body and tail of the pancreas demonstrates acinar loss with marked fibrosis, duct dilation, some lymphocytic infiltration, and sparing of islets. Based upon the information given, which of the following would most likely represent the microscopic appearance of the pancreatic mass?

A. deposition of an acellular, eosinophilic material between parenchymal cells

B. diffuse interstitial fibrosis with intraand extracellular golden-brown pigment

C. moderately differentiated, mucussecreting cells in a dense, fibrotic stroma

D. necrosis of pancreatic parenchyma and adjacent fat with a neutrophil infiltrate E. plugging of ducts, atrophy of exocrine glands, and fibrosis

Correct Answer: C

Section: Pathology and Path physiology Little is known about the etiology of pancreatic adenocarcinoma although smoking, chronic pancreatitis, and diabetes mellitus (especially in women) have been associated with an increased risk for the disease. The microscopic description for the body and tail of the pancreas is consistent with chronic pancreatitis, thus, this woman had all three of these risk factors. The microscopic appearance of ductal pancreatic adenocarcinoma most usually (~70%) demonstrates moderately to well-differentiated cells producing mucus and surrounded by a dense fibrotic stroma. Deposition of an acellular, eosinophilic material (choice A) is a description of amyloidosis. Diffuse interstitial fibrosis with intra- and extracellular golden-brown pigment (choice B) describes hemochromatosis. Necrosis of pancreatic parenchyma and adjacent fat with inflammation (choice D) would be seen in acute pancreatitis. Plugging of ducts, atrophy of exocrine glands and fibrosis (choice E) may be seen in cystic fibrosis. In addition, choices A, B, D, and E would not be expected to be associated with a focal mass.

**QUESTION 4**

A 7-year-old boy experienced respiratory tract inflammation, sore throat, and fever. Labored breathing soon followed the development of a gray membrane in the tonsil area, and diphtheria was diagnosed. Which of the following represents the most immediate course of action by his physician?

A. acid-fast stain of a throat specimen

B. culture of a throat specimen on blood agar

C. injection of diphtheria antitoxin

D. oral administration of sulfonamides

E. performance of a spinal tap

Correct Answer: C

Section: Microbiology/Immunology A physician is justified in giving antitoxin on clinical evidence, or suspicion of diphtheria, without waiting for laboratory confirmation. The antitoxin dosage should be adjusted according to the weight of the patient and the severity of the infection. The antitoxin is given to neutralize free diphtheria exotoxin in the body fluids and timeliness is of extreme importance. Once the exotoxin has been bound by the body cells and exerted its influence, diphtheria antitoxin is of little value. C. diphtheria localizes in the throat, and thus spinal taps are useless (choice E). Tellurite agar, not blood agar, is used for the isolation of C. diphtheriae from throat swabs, because it is a selective medium for this germ, inhibiting the growth of other bacteria present in throat swabs (choice B). C. diphtheriae is not an acid-fast microbe. Methylene blue is used to stain smears for the bacteriological diagnosis of diphtheria (choice A). This initial treatment of choice for diphtheria is antitoxin. Treatment with penicillin G or erythromycin, but not

sulfonamides, may be used.

Penicillin G or erythromycin are not substitutes for diphtheria antitoxin (choice D).

**QUESTION 5**

Glucagon binding to liver cells induces an increase in intracellular cAMP concentration. The rate- limiting step in cholseterol biosynthesis is regulated as a consequence of this glucagon-mediated rise in cAMP. The effect of increased cAMP on the rate of cholesterol biosynthesis occurs because of which of the following?

A. AMP-activated protein kinase (AMPK) is activated and directly phosphorylates human menopausal gonadotropin (HMG)-CoA reductase, leading to an increase in the activity of the latter enzyme.

B. Cyclic AMP-dependent protein kinase (PKA) is activated and directly phosphorylates HMG-CoA reductase, reducing the activity of the latter enzyme.

C. PKA is activated and phosphorylates AMP-regulated kinase, which then phosphorylates and activates HMGCoA reductase.

D. Removal of phosphate from HMG-CoA reductase increases its activity. Activated PKA results in a reduced level of phosphate removal from HMG-CoA reductase so that the latter enzyme is kept less active.

E. The increased cAMP directly inhibits HMG-CoA reductase.

Correct Answer: D

Section: Biochemistry Regulation of HMG-CoAreductase (the ratelimiting enzyme of cholesterol biosynthesis) activity is complex and involves a number of distinct enzyme activities (below figure).

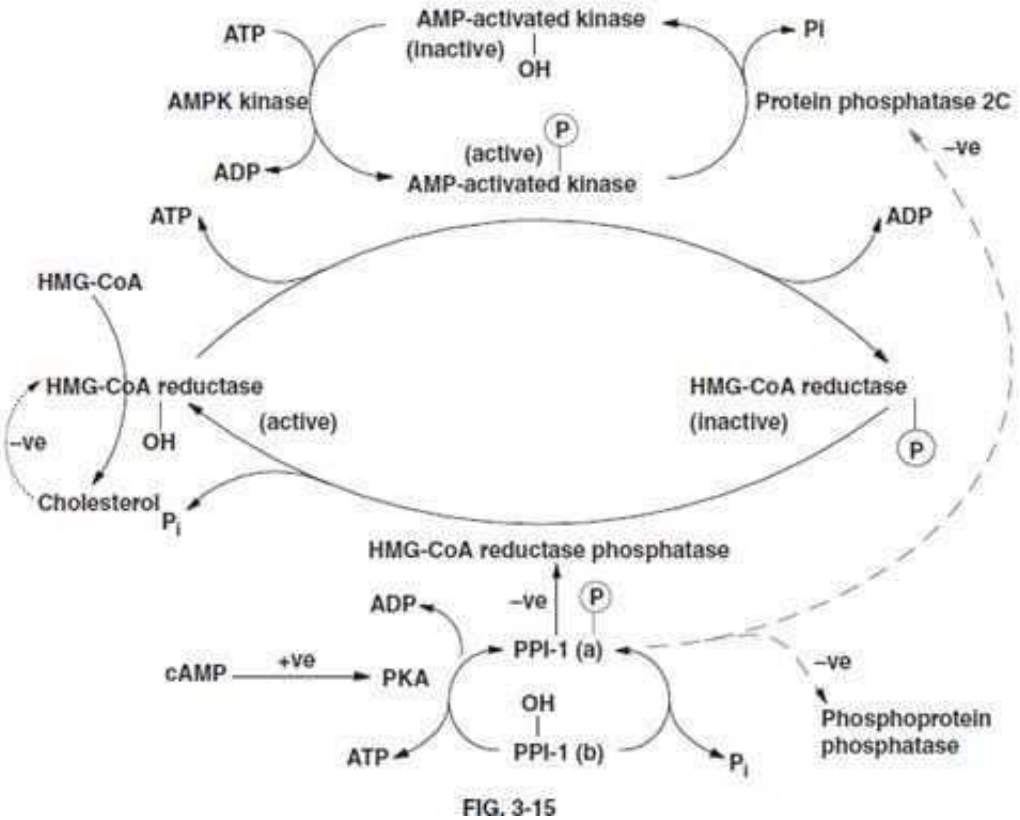

FIG. 3-15

Since cholesterol biosynthesis consumes large amounts of energy, it needs to be regulated in response to energy demands, particularly in hepatocytes. The principal site for the action of the hypoglycemic response hormone, glucagon, is the liver. Glucagon binding to hepatocytes triggers the liver to stop catabolizing carbohydrate and to divert carbon atoms into the gluconeogenesis pathway. This change in hepatic metabolism, in response to glucagon, comes about through a change in the phosphorylation state of numerous enzymes. One of these enzymes is HMG-CoA reductase. HMG-CoA reductase is most active when in the nonphosphorylated state. The enzyme is phosphorylated and rendered less active through the action of AMP-regulated kinase. AMP levels rise as the energy charge in the cell declines and thus the cell is able to recognize this change, at the level of HMG-CoA reductase activity, by inducing its phosphorylation and inhibition. In order to reverse the effect of HMG-CoA phosphorylation the phosphates must be removed. This occurs through the action of HMG-CoA reductase phosphatase. HMGCoAreductase phosphatase is regulated through the action of protein phosphatase inhibitor-1 (PPI1). In turn the activity of PPI-1 is regulated by its state of phosphorylation; it is more active when phosphorylated. When glucagon binds to hepatocytes, the result is a glucagon receptormediated activation of adenylate cyclase, which in turn produces cAMP from ATP. The effect of cAMP is to activate cAMPdependent protein kinase which then phosphorylates a number of substrates. With respect to glucagonmediated regulation of cholesterol metabolism, PKA phosphorylates PPI-1. Phosphorylated PPI-1 is more active at inhibiting HMG-CoA reductase phosphatase so that the removal of phosphate from HMG-CoA reductase is inhibited. This keeps HMG-CoA reductase in the phosphorylated and less active state. AMPK is not directly affected by glucagon-mediated increases in the levels of cAMP (choice A). Although glucogon action leads to an increase in PKA activity, the latter enzyme does not itself phosphorylate HMG-CoA reductase (choice B). The activity of AMP-regulated kinase is affected by its level of phosphorylation but it is not phosphorylated by PKA(choice C). Cyclic AMPitself does not have a direct effect on HMG-CoAreductase (choice E).