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United States Medical Licensing Step 1

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QUESTION 1

A patient presents with fever (her core temperature equals 39°C, with normal core temperature equaling 3637.5°C) as well as an elevated white cell count. Which of the following statements is true regarding the patient\\'s elevated core body temperature?

A. Bacterial toxins act directly on skeletal muscle to increase muscle contractile activity, therebygenerating heat and elevating core temperature.

B. Core body temperature now exceeds the hypothalamic set point temperature.

C. Increased core body temperature is due to increased heat production by leukocytes.

D. Increased prostaglandins have raised the hypothalamic set point temperature.

E. The patient will be sweating in an effort to further elevate core temperature.

Correct Answer: D

Section: Physiology In fever, cytokines act to increase prostaglandin levels in the hypothalamus. By an undefined mechanism, prostaglandins increase the hypothalamic set point temperature. The body\\'s heat- generating mechanisms are activated to increase core body temperature until it approaches the new set point. Core temperature will approach but not exceed the set point temperature (choice B). Sweating lowers core temperature (choice E). Though bacterial toxins can be important in the etiology of fever, they do not act directly to increase muscle heat production (choice A). Leukocytes generate cytokines, which act centrally to influence the hypothalamus (choice C). The cellular activity that is most important in heat generation is muscle contraction.

QUESTION 2

A 7-year-old boy is examined by his pediatrician because of complaints of severe cramping pain in his legs whenever he rides his bike. He also expriences nausea and vomiting during these attacks. The child has noted that the severity of the cramps is most intense after dinners that include baked potatoes or pasta, and sometimes bread. Clinical studies undertaken following a treadmill test demonstrate myoglobinuria, hyperuricemia, and increased serum bilirubin. Which of the following enzyme deficiencies is associated with these clinical findings?

A. glucose-6-phosphatase

B. glycogen synthase

C. liver glycogen debranching enzyme

D. muscle phosphofructokinase

E. muscle phosphorylase

Correct Answer: D

Section: Biochemistry A deficiency in muscle phosphofructokinase results in glycogen storage disease type VII (Tarui disease). Clinically, the symptoms seen in Tarui disease are very similar to those seen in muscle phosphorylase deficiency (choice E), glycogen storage disease type V (McArdle disease) such as exercise-induced cramping and early fatigue. There are five clinical characteristics allowing distinction between Tarui and McArdle diseases: exercise intolerance is evident in childhood, is more severe, and is associated with nausea and vomiting; the intolerance is particularly acute following meals rich in carbohydrates; hyperuricemia is more severe; compensated hemolytic anemia

is evidenced by increased serum bilirubin and reticulocyte count, and lastly; an abnormal polysaccharide is present in muscle fibers. Deficiency in glucose-6-phosphatase (choice A) is one cause of glycogen storage disease type I (specifically type Ia, von Gierke disease). Classic symptoms of this deficiency include neonatal hypoglycemia and lactic acidosis. If symptoms do not appear until the third or fourth month they include hepatomegaly and hypoglycemic seizures. Liver glycogen synthase deficiency (choice B) presents with morning fatigue and ketotic hypoglycemia on fasting--both of which rapidly disappear on feeding. Symptoms can be rapidly relieved and chemical signs corrected by introducing frequent protein-rich meals and nighttime feedings of suspensions of uncooked corn starch. Deficiency in glycogen debranching enzyme (choice C) results in glycogen storage disease type III (Cori or Forbes disease). Symptoms of type III disease are short stature, variable skeletal myopathy, cardiomyopathy, hepatomegaly, and hypoglycemia.

QUESTION 3

A 38-year-old man has been treated for myasthenia gravis with pyridostigmine and propantheline for 8 years. He has had the "flu" for 10 days and his wife calls reporting that he now has serious muscle weakness. What should be administered after admitting this patient to the emergency department?

A. atropine to reverse the effects of an overdose

B. a test dose of edrophonium

C. pralidoxime on the assumption that he inadvertently overdosed

D. the daily dose of pyridostigmine on the assumption that he forgot to take his medication

E. tubocurarine

Correct Answer: B

Section: Pharmacology Myasthenia gravis is an autoimmune disease attributable to an impairment of nicotinic receptor function at the neuromuscular junction by antireceptor antibodies. Treatment consists of increasing the junctional concentration of acetylcholine with a carbamate anticholinesterase such as pyridostigmine. Infections such as influenza may change the anticholinesterase dose requirement in myasthenia either up or down. As in this patient, the problem is to distinguish whether the muscle weakness is attributable to myasthenic crisis (too little medication) or cholinergic crisis (too much medication); both conditions cause muscle weakness. The safest definitive method is to administer a small dose of the short-acting anticholinesterase edrophonium (choice B). If the patient is in myasthenic crisis, an immediate improvement in muscle function should be evident. If the patient is in cholinergic crisis, the patient\\'s condition may worsen, but because the duration of action for edrophonium is only 510 min, this test provides less risk than other alternatives. Parenteral atropine should be available to treat excess parasympathetic activity. Atropine for symptoms of parasympathetic overdose (choice A) is inappropriate because this patient is being treated with the muscarinic antagonist propantheline and atropine will have no effect on his muscle weakness. Administration of pralidoxime (choice C) is incorrect because pralidoxime is useful for reactivation of cholinesterase only in the case of recent organophosphate intoxication. Administration of pyridostigmine (choice D) is unwise because a patient in cholinergic crisis is put at risk of exacerbation and extension of the toxic episode for a significant period of time. Administration of a test dose of a long-acting agent such as tubocurarine (choice E) to elicit muscle weakness is a provocative test that is dangerous and does not provide definitive evidence for diagnosis.

QUESTION 4

A 60-year-old woman is admitted to the hospital with a fever and severe diarrhea for the last 24 hours. Cultures of blood, cerebrospinal fluid, urine, and stool are all negative for pathogens. The profile of gut hormones reveals elevated levels of VIP. An analogue of which of the following would most likely lower her VIP levels?

A. erythromycin

B. histamine

C. motilin

D. somatostatin

E. trypsin

Correct Answer: D

Section: Physiology VIP is a neurotransmitter in the brain and in the parasympathetic nerves of the digestive tract. It also acts as a hormone. VIP has a secretin-like effect on the pancreas. It increases the volume of water and bicarbonate output and affects GI blood flow and motility. All this contributes to severe secretory diarrhea in the case of VIP overproduction. Somatostatin is the best choice because it has a broad range of inhibitory effects, inhibiting GI secretions, slowing GI motility, and reducing splanchnic blood flow. An analogue of somatostatin, called octreotide, was found useful for treatment of syndromes related with VIP overproduction. Octreotide has a longer half-life than physiologic somatostatin. Erythromycin (choice A) is known to have antibiotic features. This is not the best choice, since no pathogens were found. Histamine (choice B) is a physiologic stimulant of gastric hydrochloric acid secretion, potentiating the effects of acetylcholine and gastrin. Motilin (choice C) stimulates gastric motility, which is contraindicated for diarrhea. Trypsin (choice E) is a protease released by pancreatic cells, breaking peptide bonds as well as converting many other proteolytic enzymes to their active form. All of the negative choices will not affect, and in fact may even worsen the VIPrelated symptoms.

QUESTION 5

The mechanism of anesthetic action for lidocaine involves which of the following?

A. blockade of axonal voltage-dependent calcium channels

B. blockade of axonal voltage-dependent sodium channels

C. hyperpolarization of axons via enhanced chloride influx

D. hyperpolarization of axons via enhanced potassium efflux

E. inhibition of nerve terminal pain receptors

Correct Answer: B

Section: Pharmacology Local anesthetic agents such as lidocaine inhibit nerve conduction through state- and use-dependent blockade of voltage-dependent fast sodium channels. As a result, the threshold of excitability of the nerve is increased, and the ability of the nerve to propagate an action potential is decreased. Ultimately, transmission of sensory stimuli to the CNS is suppressed, and motor function involving small fibers in the vicinity of the injection is also lost. Inhibition of nerve conduction via blockade of calcium channels (choice A) is not a mechanism for local anesthetic agents. Hyperpolarization of neurons via enhanced chloride influx (choice C) is not a mechanism for local anesthetic agents, but is the mechanism for inhibition of repetitive neuronal firing by the benzodiazepines, barbiturates, and other GABA-mimetic and -enhancing agents that act on the GABAA receptor-chloride channel of neurons. Enhanced potassium efflux (choice D) does not occur with local anesthetics. Inhibition of pain receptors (choice E) is not a mechanism for currently available analgesic agents, although antagonists at the neurokinin receptors involved in nociception (pain perception) are an area of intense research.

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