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QUESTION NO: 1

Pancoast's tumor is not linked to which of the following statements?

A. Horner's syndrome

B. Anhidrosis

C. Cervical plexus involvement

D. Arthritis

ANSWER: C

Explanation:

Cervical plexus involvement is the best answer because it is not a characteristic anatomic association of a Pancoast tumor. A Pancoast tumor is a bronchogenic carcinoma arising at the pulmonary apex; its local extension produces the characteristic Pancoast syndrome through invasion or compression of structures in the thoracic inlet. The clinically important neural structures include the inferior brachial plexus—particularly its lower roots/trunk, which produces severe shoulder pain radiating down the medial arm—and the cervical sympathetic pathway, which can produce Horner syndrome. The cervical plexus is a distinct network formed by the anterior rami of C1–C4 and chiefly supplies sensation to the neck, shoulder region, and motor innervation to selected neck muscles. It is not the plexus classically affected by apical lung-tumor extension. Thus, identifying cervical plexus involvement as unrelated reflects the relevant anatomy and the expected pattern of neurologic involvement in Pancoast syndrome. For further review, see StatPearls: Pancoast Syndrome and the Merck Manual overview of pulmonary tumors.

QUESTION NO: 2

Which of the following syndromes corresponds to: can be caused by high doses of Tetracyclines?

A. Dubin-Johnson syndrome

B. Fanconi's syndrome

C. Edward's syndrome

D. Cri-du-chat syndrome

ANSWER: B

Explanation:

Fanconi's syndrome is correct because tetracycline-associated proximal tubular injury can produce a generalized defect in proximal renal tubular reabsorption. This is classically associated with degraded or outdated tetracycline preparations, whose breakdown products are nephrotoxic; exposure to tetracyclines can therefore be tested as an acquired cause of Fanconi syndrome. The proximal tubule normally reabsorbs filtered glucose, phosphate, bicarbonate, uric acid, amino acids, and other solutes. When this function is impaired, affected patients may develop normoglycemic glycosuria, phosphaturia with hypophosphatemia, aminoaciduria, uricosuria, and bicarbonate wasting that causes a proximal (type 2) renal tubular acidosis. Clinical consequences can include weakness, volume depletion, bone pain, or osteomalacia due to chronic phosphate loss. Identifying the medication exposure alongside this characteristic proximal-tubule transport defect supports the diagnosis of Fanconi's syndrome. Merck Manual lists outdated tetracycline among causes of acquired Fanconi syndrome: Merck Manual—Fanconi Syndrome. A clinical review of the disorder and its proximal tubular manifestations is also available from NCBI Bookshelf—Fanconi Syndrome.

QUESTION NO: 3

Which of the following syndromes corresponds to: A failure of neutrophils to generate an immune response and lab values indicate elevated IgE levels?

A. Job's syndrome

B. Wiskott-Aldrich syndrome

C. Carcinoid syndrome

D. Mallory-Weiss syndrome

ANSWER: A

Explanation:

Job's syndrome is the classic hyper-IgE syndrome and best matches markedly elevated serum IgE together with defective neutrophil-mediated host defense. The condition is most often caused by dominant-negative variants affecting *STAT3*, which disrupt signaling needed for normal immune-cell differentiation and function. A key clinical consequence is impaired neutrophil recruitment/chemotaxis to sites of infection, so patients develop recurrent bacterial skin and pulmonary infections, often with relatively little local inflammation. The characteristic clinical pattern includes recurrent “cold” staphylococcal abscesses, recurrent pneumonias that may lead to pneumatoceles, chronic eczematous dermatitis, and substantial eosinophilia and IgE elevation. Nonimmune findings can include retained primary teeth, characteristic facies, scoliosis, and bone-fracture susceptibility. Thus, the combination in the question of a neutrophil functional/recruitment defect and elevated IgE identifies Job's syndrome. The Merck Manual summarizes hyper-IgE syndrome as involving impaired chemotaxis and very high IgE levels: Merck Manual—Hyper-IgE Syndrome. Additional clinical and genetic details are available from GeneReviews—STAT3 Hyper-IgE Syndrome.

QUESTION NO: 4

A 45-year-old woman with systemic sclerosis (scleroderma) comes to the physician because of a 3-week history of progressive shortness of breath and nonproductive cough. Her temperature is

36.9°C (98.4°F), pulse is 82/min, respirations are 20/min, and blood pressure is 136/85 mmHg. Crackles are heard in both lower lung fields. Pulmonary function tests show total lung capacity is 80% of predicted, and diffusing capacity for carbon monoxide, corrected for alveolar volume, is 65% of predicted. Histologic examination of a lung biopsy specimen is most likely to show which of the following findings?

A. Diffuse interstitial fibrosis

B. Intra-alveolar exudates

C. Multiple thromboemboli

D. Necrotizing vasculitis

E. Non-necrotizing interstitial granulomas

ANSWER: A

Explanation:

Diffuse interstitial fibrosis is correct because this patient has systemic sclerosis with interstitial lung disease. Progressive exertional dyspnea, a dry cough, bibasilar crackles, reduced total lung capacity, and reduced diffusing capacity are classic manifestations of a restrictive fibrosing process involving the lung interstitium. In systemic sclerosis, immune-mediated injury causes inflammation and activation of fibroblasts, leading to excess collagen deposition, thickening of alveolar septa, and progressive architectural distortion of the pulmonary interstitium. Histopathology most often resembles a nonspecific interstitial pneumonia pattern, with relatively uniform interstitial inflammation and fibrosis; more advanced disease may produce extensive diffuse fibrosis. The thickened alveolar-capillary membrane accounts for impaired carbon monoxide diffusion, while stiff fibrotic lungs reduce compliance and lung volumes. Pulmonary involvement is a major cause of morbidity and mortality in systemic sclerosis, so pulmonary function testing with diffusing capacity measurement is used to identify and monitor disease. The Merck Manual describes interstitial lung disease in systemic sclerosis as fibrosis affecting the lung

interstitium, and the American Thoracic Society guideline addresses treatment of systemic sclerosis-associated interstitial lung disease. See Merck Manual: Systemic Sclerosis and American Thoracic Society clinical practice guideline.

QUESTION NO: 5

A 55-year-old hypertensive man develops sudden onset of excruciating pain beginning in the anterior chest, and then radiating to the back. Over the next 2 hours, the pain moves downward toward the abdomen. Which of the following is the most likely diagnosis?

A. Aortic dissection

B. Syphilitic aneurysm

C. Aortic valve stenosis

D. Atherosclerotic aneurysm

E. Myocardial infarction

ANSWER: A

Explanation:

Aortic dissection is the most likely diagnosis. This condition occurs when blood enters through a tear in the aortic intima and propagates within the aortic wall, creating a false lumen. Long-standing hypertension is a major predisposing factor because it promotes degenerative changes and increased shear stress in the aorta. The characteristic presentation is abrupt, severe, often described as tearing or ripping chest pain that radiates to the back. Importantly, pain may migrate as the dissection extends along the aorta; progression from the chest toward the abdomen over several hours strongly reflects distal propagation of the intimal dissection flap. Aortic dissection is a time-critical emergency because extension can compromise branch vessels, cause acute aortic regurgitation, produce pericardial tamponade, or rupture. Initial evaluation in a hemodynamically stable patient commonly uses CT angiography, while transesophageal echocardiography is particularly useful when rapid bedside imaging is needed or CT is unsuitable. Immediate management includes heart-rate and blood-pressure control with intravenous beta blockade to reduce aortic shear stress, followed by urgent surgical assessment when the ascending aorta is involved. See the Merck Manual overview of aortic dissection and the StatPearls review of aortic dissection.

QUESTION NO: 6

During an experiment, drug X is added to a muscle bath containing a strip of guinea pig intestinal smooth muscle. Agonists are added to the bath, and the resultant effects on muscle tension are shown in the table.

Agonist	Muscle Tension Before Drug X (g)	Muscle Tension After Drug X (g)
Vehicle	6.0	6.1
Acetylcholine	11.3	18.5
Norepinephrine	4.1	4.2

Which of the following types of drugs is most likely to produce effects most similar to those of drug X?

A. α1-Adrenergic antagonist

B. β-Adrenergic antagonist

C. Cholinesterase inhibitor

D. Monoamine oxidase inhibitor

E. Muscarinic antagonist

ANSWER: C

Explanation:

Cholinesterase inhibitor is correct because inhibition of acetylcholinesterase decreases the enzymatic breakdown of acetylcholine in the tissue bath. Acetylcholine therefore persists longer at muscarinic receptors on intestinal smooth muscle, producing an enhanced and/or prolonged contractile response. In guinea pig ileum, muscarinic receptor activation—particularly via the Gq-coupled M3 receptor subtype—increases intracellular calcium in smooth-muscle cells and raises muscle tension. Thus, a drug that potentiates the contractile effect attributable to acetylcholine, while acting indirectly by increasing its local availability rather than directly stimulating the receptor, has the characteristic functional effect of a cholinesterase inhibitor. This pharmacologic principle also explains why acetylcholinesterase inhibitors increase cholinergic activity at parasympathetic effector organs. The mechanism and clinical effects of this drug class are reviewed in StatPearls: Cholinergic Medications. The muscarinic signaling pathway responsible for smooth-muscle contraction is further summarized in StatPearls: Muscarinic Receptor.

QUESTION NO: 7

Which of the following findings is most likely in primary adrenal insufficiency?

A. Hyperpigmentation

B. Suppressed adrenocorticotropic hormone

C. Hypernatremia

D. Hypokalemia

ANSWER: A

Explanation:

Hyperpigmentation is a characteristic finding in primary adrenal insufficiency. Loss of adrenal cortisol production removes negative feedback on pituitary adrenocorticotropic hormone secretion. Increased adrenocorticotropic hormone production is accompanied by increased melanocyte-stimulating activity, which can cause diffuse skin and mucosal hyperpigmentation.

Primary adrenal insufficiency may also cause hypotension, hyponatremia, hyperkalemia, and hypoglycemia. In secondary adrenal insufficiency, adrenocorticotropic hormone levels are low or inappropriately normal, so hyperpigmentation and hyperkalemia are less typical. See StatPearls: Adrenal Insufficiency.

QUESTION NO: 8

Which of the following is not one of the key steps in the grief process?

A. Denial

B. Anger

C. Bargaining

D. Rejection

ANSWER: D

Explanation:

“Rejection” is correct because it is not included in the commonly taught Kübler-Ross model of responses associated with grief and loss. This model describes five stages: denial, anger, bargaining, depression, and acceptance. The framework originated from Elisabeth Kübler-Ross’s work with people facing terminal illness and has since been widely used as a way to discuss emotional responses to loss. Therefore, “rejection” does not name one of the model’s established stages.

It is important to use the stages as a descriptive educational framework rather than a fixed sequence that every bereaved person must experience. Grief is individual, may involve many emotions, and does not necessarily unfold in a predictable

order. A person may move back and forth among emotions, experience some but not all of them, or have reactions not captured by a stage model. The National Cancer Institute notes that grief responses vary substantially among individuals, while the American Psychological Association similarly emphasizes that grief is not a linear process. See the National Cancer Institute's bereavement information and the American Psychological Association's grief resources.

QUESTION NO: 9

An S3 heart sound is often associated with?

A. CHF

B. COPD

C. Atrial fib.

D. Ventricular fib.

ANSWER: A

Explanation:

CHF is correct. An S3, also called a third heart sound or ventricular gallop, occurs shortly after S2 during rapid passive ventricular filling in early diastole. In adults, an S3 commonly reflects increased ventricular filling pressures and volume overload, particularly when ventricular systolic function is impaired. In congestive heart failure, a weakened and/or dilated ventricle has reduced compliance and is exposed to elevated atrial pressure; the rapid inflow of blood into that ventricle produces the low-frequency S3 sound. Clinically, an S3 is best heard with the bell of the stethoscope at the cardiac apex with the patient in the left lateral decubitus position for left-sided heart failure. It may be physiologic in children, young adults, and pregnancy, but in middle-aged or older adults it is an important finding that supports heart failure in the appropriate clinical setting. The sound can be remembered as a cadence of "Kentucky," with S1 and S2 followed by the extra diastolic sound. The American Heart Association describes a third heart sound as a possible sign of heart failure, and standard bedside examination references associate it with ventricular dysfunction and elevated filling pressures. See the American Heart Association's heart-failure warning signs and the NCBI StatPearls review of the third heart sound.

QUESTION NO: 10

Which of the following cranial nerves can be directly linked to respiratory and cardiac dysfunction?

A. IV

B. VII

C. X

D. XI

ANSWER: C

Explanation:

The vagus nerve (cranial nerve X) is directly linked to both cardiac and respiratory function because it is the principal parasympathetic nerve supplying thoracic viscera. Its cardiac branches influence the sinoatrial and atrioventricular nodes, allowing parasympathetic signaling to slow heart rate and affect cardiac conduction. Vagal dysfunction or excessive vagal stimulation can therefore produce clinically significant bradycardia or other disturbances of cardiac rhythm.

The vagus nerve also provides motor innervation to the pharynx and larynx through its branches, which is important for airway protection, phonation, and normal upper-airway function. In addition, its extensive sensory and autonomic connections to the airways participate in reflex control of bronchial tone and respiratory responses. Damage involving the vagus nerve or its central connections may consequently contribute to impaired airway protective reflexes, altered respiratory regulation, and cardiovascular instability. These broad parasympathetic and visceral functions make cranial nerve X the

cranial nerve most directly associated with combined respiratory and cardiac dysfunction. See StatPearls: Neuroanatomy, Cranial Nerve 10 (Vagus Nerve) and Merck Manual: Overview of the Cranial Nerves.

QUESTION NO: 11

Which of the following enzyme breaks down starches to maltose?

A. Amylase

B. Lipase

C. Trypsinogen

D. Pepsin

ANSWER: A

Explanation:

Amylase is correct because it catalyzes the hydrolysis of dietary starch, a polysaccharide, into smaller carbohydrate products, including maltose. Salivary amylase begins this process in the mouth, and pancreatic amylase continues starch digestion in the small intestine. Maltose is a disaccharide composed of two glucose molecules; it is subsequently hydrolyzed by maltase at the intestinal brush border to yield absorbable glucose. This role makes amylase the key enzyme associated with initial starch digestion and maltose production. The physiologic process is clinically relevant because carbohydrate digestion depends on pancreatic enzyme secretion and intact small-intestinal brush-border function. The National Institute of Diabetes and Digestive and Kidney Diseases describes pancreatic amylase as an enzyme that helps digest carbohydrates, and OpenStax details amylase-mediated starch digestion in the oral cavity and small intestine. See NIDDK: Pancreatitis—Definition & Facts and OpenStax: Chemical Digestion and Absorption.

QUESTION NO: 12

Which of the following terms matches the statement: to increase the fibrous element; to make hard as in the presence of cellulites?

A. Induration

B. Necrosis

C. Eschar

D. Maceration

ANSWER: A

Explanation:

Induration is the correct term because it describes abnormal hardening or firming of a tissue. Clinically, this palpable firmness may result from inflammation and edema within the skin and subcutaneous tissues, or from increased deposition of fibrous connective tissue. In cellulitis, inflammation of the dermis and subcutaneous tissue can produce a characteristic area of warmth, erythema, swelling, tenderness, and firmness; that firm quality on examination is termed induration. Thus, the wording “to increase the fibrous element” and “to make hard” corresponds directly to induration.

Induration is an examination finding rather than a diagnosis by itself. It is assessed by palpation and reflects tissue resistance or hardness compared with surrounding tissue. Depending on the setting, it can occur with acute inflammation, chronic fibrosis, infiltrative disease, or healing tissue. The National Cancer Institute defines induration as an abnormal hardening of tissue, a definition that captures the essential meaning tested here. Further clinical context on cellulitis and its inflammatory involvement of skin and subcutaneous tissue is available in the Merck Manual. See also the NCI Dictionary of Cancer Terms.

QUESTION NO: 13

A previously healthy 40-year-old woman is brought to the emergency department by her husband because of a 2-day history of fever, lethargy, and confusion. Her temperature is 38°C (100.4°F), pulse is 80/min, respirations are 18/min, and blood pressure is 140/90 mmHg. Physical examination shows scattered petechiae and ecchymoses over the lower extremities. Neurologic examination shows moderate generalized motor weakness. She is oriented to person but not to place or time. Laboratory studies show:

Hemoglobin	9 g/dL
Hematocrit	27%
Leukocyte count	8000/mm^3 with a normal differential
Platelet count	15,000/mm^3
Prothrombin time	12 sec (INR=1.1)
Partial thromboplastin time	30 sec
Serum	
Urea nitrogen	25 mg/dL
Lactate dehydrogenase	1000 U/L

A peripheral blood smear shows 3+ polychromasia and 3+ schistocytes. Urine and blood cultures grow no organisms. A chest x-ray shows no abnormalities. Which of the following is the most likely diagnosis?

A. Acute myeloid leukemia

B. Autoimmune hemolytic anemia

C. Thrombotic thrombocytopenic purpura

D. Toxic shock syndrome

E. von Willebrand disease

ANSWER: C

Explanation:

Thrombotic thrombocytopenic purpura is the most likely diagnosis because this presentation combines thrombocytopenia, microangiopathic hemolytic anemia, neurologic dysfunction, and fever. Petechiae and ecchymoses reflect the low platelet count. Schistocytes on the peripheral smear are fragmented erythrocytes produced when red cells are mechanically sheared while passing through platelet-rich microthrombi in small vessels. Marked polychromasia supports an appropriate marrow response to hemolysis through increased reticulocyte production. The acute confusion, disorientation, and generalized weakness are characteristic consequences of microvascular ischemia in the central nervous system. TTP is generally caused by severe deficiency of the von Willebrand factor–cleaving protease ADAMTS13, most often due to acquired autoantibodies in adults. This allows unusually large von Willebrand factor multimers to promote widespread platelet aggregation and microthrombus formation. The classic syndrome may also include renal injury, although renal findings need not be prominent for the diagnosis. TTP is a hematologic emergency; clinical suspicion warrants urgent plasma exchange and corticosteroid-based immunosuppression while ADAMTS13 testing is pending. Additional details are available from the National Heart, Lung, and Blood Institute and the Merck Manual Professional Edition.

QUESTION NO: 14

Which of the following is not considered an H2 blocker?

A. Ranitidine (Zantac)

B. Famotidine (Pepcid)

C. Cimetidine (Tagament)

D. Sucralfate (Carafate)

ANSWER: D

Explanation:

Sucralfate (Carafate) is correct because it is a locally acting gastrointestinal mucosal protectant rather than a histamine-2 receptor antagonist. In the acidic environment of the stomach, sucralfate polymerizes into a viscous, negatively charged paste that adheres to positively charged proteins at an ulcer base. This creates a protective barrier against acid, pepsin, and bile salts and supports mucosal healing. Its therapeutic benefit therefore comes primarily from topical protection of damaged mucosa, with minimal systemic absorption, not from suppressing gastric acid secretion through histamine receptor blockade. The FDA prescribing information describes sucralfate as an oral agent indicated for short-term treatment of active duodenal ulcer and explains its local protective mechanism. This distinction is clinically useful because sucralfate administration must be separated from several oral medications, as its binding action can reduce their absorption. See the FDA Carafate prescribing information and the NCBI StatPearls review of sucralfate.

QUESTION NO: 15

Wrist extensors are primarily controlled by what nerve?

A. Radial

B. Ulnar

C. Median

D. Tibial

ANSWER: A

Explanation:

Radial is correct. Extension at the wrist is produced principally by the extensor carpi radialis longus, extensor carpi radialis brevis, and extensor carpi ulnaris muscles. These muscles lie in the posterior (extensor) compartment of the forearm, whose motor innervation is supplied by the radial nerve and its deep branch, the posterior interosseous nerve. The radial nerve supplies extensor carpi radialis longus before dividing near the lateral epicondyle; its deep branch continues through the supinator as the posterior interosseous nerve and supplies most of the remaining forearm extensor muscles, including extensor carpi radialis brevis and extensor carpi ulnaris. Thus, a radial neuropathy can cause weakness of wrist extension; a high lesion classically produces wrist drop because the wrist extensors lose their effective motor input. This relationship is clinically useful when localizing upper-extremity nerve injury during a neurologic or musculoskeletal examination. The anatomic distribution of the radial nerve and its deep/posterior interosseous branch is described by StatPearls: Anatomy, Shoulder and Upper Limb, Radial Nerve. A concise clinical overview of radial-nerve motor deficits, including impaired wrist extension, is available in the Merck Manual Professional Edition.

QUESTION NO: 16

Insulin inhibits the release of _____.

A. Glucagon

B. ADH

C. Beta cells

D. Somatostatin

ANSWER: A

Explanation:

Glucagon is correct because insulin is an important paracrine regulator within the pancreatic islets. After a meal, rising blood glucose stimulates pancreatic beta cells to secrete insulin. In addition to promoting glucose uptake and storage in peripheral

tissues, insulin acts locally on neighboring alpha cells to suppress glucagon secretion. This coordinated response limits hepatic glucose production at a time when circulating glucose is already plentiful.

Glucagon normally raises plasma glucose by stimulating hepatic glycogenolysis and gluconeogenesis. Suppression of glucagon by insulin therefore helps prevent an inappropriate postprandial rise in hepatic glucose output. The reciprocal relationship is clinically important: when insulin secretion is absent or markedly reduced, such as in untreated type 1 diabetes, glucagon secretion may be relatively increased and contributes to hyperglycemia and ketone production. This is why normal glucose homeostasis depends not only on insulin's direct metabolic actions but also on its local inhibition of glucagon release. See the NCBI Bookshelf overview of pancreatic hormone physiology and the NCBI Bookshelf discussion of insulin physiology.

QUESTION NO: 17

Which of the following drugs is associated with the reaction of Stevens-Johnson syndrome?

A. Valproic acid

B. Quinidine

C. Isoniazid

D. Ethosuximide

ANSWER: D

Explanation:

Ethosuximide is associated with Stevens-Johnson syndrome, a rare but potentially life-threatening severe cutaneous adverse reaction. Ethosuximide is an antiseizure medication used primarily for absence seizures. Although it is generally well tolerated, its safety information includes serious dermatologic hypersensitivity reactions, including Stevens-Johnson syndrome and toxic epidermal necrolysis. These reactions can begin with nonspecific prodromal symptoms such as fever, malaise, sore throat, or ocular burning, followed by a painful rash, mucosal erosions, blistering, and epidermal detachment. Prompt recognition is essential because continued exposure to the causative medication can worsen the reaction. If a patient taking ethosuximide develops findings suggestive of Stevens-Johnson syndrome, the medication should be discontinued immediately and the patient should receive urgent medical evaluation and supportive care. This association is clinically important when counseling patients and caregivers to report rash or mucosal symptoms promptly, particularly early in therapy. The National Library of Medicine's Ethosuximide review describes Stevens-Johnson syndrome as a rare serious adverse effect. Additional clinical background on medication-triggered Stevens-Johnson syndrome is available from Merck Manual Professional.

QUESTION NO: 18

A 14-year-old girl is brought to the physician after her mother learned that she began having sexual intercourse with various partners 1 month ago. She does not use condoms or other contraception. The mother is concerned about her behavior. The patient's parents separated 3 months ago. She had been an honor student and excelled in sports and leadership positions at school before the separation. Since the separation, however, she has become sullen, defiant, and rebellious. She has begun smoking cigarettes, disobeying her curfew, and being truant from school. This patient is most likely using which of the following defense mechanisms?

A. Acting out

B. Displacement

C. Projection

D. Reaction formation

E. Sublimation

ANSWER: A

Explanation:

Acting out is correct because the patient is expressing the distress, anger, and loss of control associated with her parents' separation through impulsive, externally directed behaviors rather than identifying and verbalizing the underlying emotions. Her abrupt pattern of risky sexual activity, cigarette use, defiance of rules, and school truancy began after a major psychosocial stressor and represents behavioral enactment of an internal emotional conflict. This is particularly common in children and adolescents, who may have less-developed capacity to recognize, tolerate, and communicate painful feelings directly. In clinical terms, acting out is an immature defense mechanism in which unconscious emotional tension is discharged through actions, often actions that are disruptive, risky, or socially inappropriate. The temporal relationship between the family disruption and the marked behavioral change supports this interpretation: the behaviors function as an expression of her unprocessed emotional response to the separation. Recognizing this mechanism helps guide an empathic assessment that explores mood symptoms, family stress, safety, substance use, sexual health, and available supports while avoiding a purely punitive framing. For further descriptions of defense mechanisms and their clinical role, see StatPearls: Defense Mechanisms and the Merck Manual discussion of defense mechanisms.

QUESTION NO: 19

Which of the following matches the definition: abnormal placenta development covering the cervix?

A. Placenta Previa

B. Abruptio Placentae

C. Multigravida

D. Proliferative phase

ANSWER: A

Explanation:

Placenta previa is correct. It is an obstetric condition in which the placenta implants in the lower uterine segment so that it lies close to, partially covers, or completely covers the internal cervical os. The key feature in the definition is placental tissue covering the cervix, particularly the internal opening of the cervix into the uterus. As the lower uterine segment stretches later in pregnancy and the cervix begins to efface or dilate, this location can disrupt placental attachment and cause the classic presentation of painless, bright-red vaginal bleeding in the second half of pregnancy. Diagnosis is established with ultrasonography, especially transvaginal ultrasound, which is safe and more accurate for determining the placental relationship to the internal os. A digital vaginal examination should be avoided until placenta previa has been excluded in a patient with late-pregnancy bleeding because it can precipitate substantial hemorrhage. Management depends on bleeding severity, gestational age, fetal status, and whether the placenta continues to cover the os; persistent previa generally requires cesarean delivery. See the American College of Obstetricians and Gynecologists overview of bleeding during pregnancy and the Merck Manual Professional discussion of placenta previa.

QUESTION NO: 20

Which of the following waveforms is most commonly found with light sleepers?

A. Theta

B. Alpha

C. Beta

D. Zeta

ANSWER: A

Explanation:

Theta is the waveform most commonly associated with light sleep, particularly stage N1 of non-rapid eye movement sleep. N1 is the transition from relaxed wakefulness to sleep and is characterized on electroencephalography by low-amplitude, mixed-frequency activity in which theta waves, typically about 4–7 Hz, become prominent. People awakened during this stage often report that they were only lightly asleep or may even feel they had not yet fallen asleep. As sleep becomes deeper, the EEG pattern changes: N2 is identified by sleep spindles and K complexes, while N3 contains high-amplitude slow-wave (delta) activity. Thus, theta activity is the best match for a light sleeper rather than deep sleep. Sleep-stage scoring and the characteristic EEG findings are summarized in the American Academy of Sleep Medicine–based overview in StatPearls: Sleep Physiology. The National Institute of Neurological Disorders and Stroke also describes sleep as progressing through stages with distinct brain-wave patterns: Understanding Sleep.

QUESTION NO: 21

Which of the following is the primary activator of zymogen secretion?

A. Somatostatin

B. Secretin

C. Acetylcholine

D. Gastrin

E. Cholecystokinin

ANSWER: E

Explanation:

Cholecystokinin is the primary physiologic activator of pancreatic acinar-cell zymogen secretion. It is released from I cells in the duodenal and jejunal mucosa in response particularly to fatty acids and amino acids entering the small intestine. Cholecystokinin acts through receptors on pancreatic acinar cells and through vagovagal neural pathways to stimulate release of digestive enzyme precursors, including trypsinogen, chymotrypsinogen, procarboxypeptidases, and lipase. These zymogens are released into pancreatic ducts and subsequently reach the duodenum, where enterokinase (enteropeptidase) initiates their activation by converting trypsinogen to trypsin. This coordinated response delivers pancreatic digestive enzymes when nutrients requiring digestion are present in the intestinal lumen. Secretin has a complementary pancreatic role: it primarily promotes bicarbonate-rich fluid secretion from ductal cells, which helps neutralize gastric acid and provides an appropriate pH for enzyme activity. For a review of pancreatic exocrine regulation and the actions of cholecystokinin, see NCBI Bookshelf: Physiology, Pancreas and NCBI Bookshelf: Physiology, Cholecystokinin.

QUESTION NO: 22

A 14-year-old boy is brought to the physician because of a 2-day history of a sore throat and fever that peaks in the late afternoon. He also has a 1-week history of progressive fatigue. He recently began having unprotected sexual intercourse with one partner. He appears ill. His temperature is 39°C (102.2°F). Physical examination shows cervical lymphadenopathy and pharyngeal erythema with a creamy exudate. Which of the following is the most likely diagnosis?

A. Candidiasis

B. Herpahgina

C. Infectious mononucleosis

D. Mumps

E. Syphilis

ANSWER: C

Explanation:

Infectious mononucleosis is the most likely diagnosis. This syndrome is usually caused by primary Epstein-Barr virus infection and is especially common in adolescents and young adults. Its classic clinical pattern includes fever, prominent fatigue or malaise, pharyngitis that can be markedly erythematous and exudative, and cervical lymphadenopathy. The patient's progressive fatigue preceding the acute sore throat and fever is particularly characteristic of this systemic viral illness. Although Epstein-Barr virus is commonly transmitted through saliva, it may also be transmitted through sexual contact; therefore, the recent sexual history is compatible with the diagnosis. The presentation can initially resemble exudative bacterial pharyngitis, but the combination of substantial fatigue and cervical adenopathy strongly supports infectious mononucleosis. Evaluation is commonly supported by a heterophile antibody test in an appropriate clinical setting, while Epstein-Barr virus-specific serology can be useful when heterophile testing is negative early in disease or when the diagnosis remains uncertain. Patients should be counseled about rest, hydration, and avoiding contact sports until splenic enlargement and risk of splenic rupture have been appropriately considered. See the CDC overview of Epstein-Barr virus and the Merck Manual discussion of infectious mononucleosis.

QUESTION NO: 23

Which of the following is the rate-limiting enzyme of cholesterol synthesis?

A. HMG-CoA synthase

B. HMG-CoA reductase

C. Acetyl-CoA carboxylase

D. Acyl-CoA cholesterol acyltransferase

ANSWER: B

Explanation:

HMG-CoA reductase is the rate-limiting enzyme in cholesterol synthesis. It catalyzes the conversion of 3-hydroxy-3-methylglutaryl coenzyme A to mevalonate, an early committed step in the pathway. Statin medications lower cholesterol synthesis by competitively inhibiting this enzyme.

HMG-CoA synthase participates in formation of HMG-CoA, whereas acyl-CoA cholesterol acyltransferase esterifies cholesterol for storage. An overview of cholesterol metabolism is available from the NCBI Bookshelf.

QUESTION NO: 24

Which of the following is considered a bronchodilator?

A. Acetylcysteine

B. Guaifenesin

C. Theophylline

D. Epinephrine HCL

ANSWER: D

Explanation:

Epinephrine HCL is considered a bronchodilator because it is an adrenergic agonist that stimulates beta-adrenergic receptors in bronchial smooth muscle. This stimulation increases intracellular cyclic AMP, producing relaxation of bronchial smooth muscle and thereby widening the airways. The resulting reduction in airway resistance can improve airflow, particularly when bronchospasm is part of an acute allergic or anaphylactic reaction. Epinephrine also has alpha-adrenergic effects that reduce mucosal edema in the upper and lower airway, which may further support airway patency in an emergency setting. Its rapid bronchodilating action is one reason epinephrine is used as first-line treatment for anaphylaxis with respiratory involvement; it is administered intramuscularly for this indication rather than as routine long-term maintenance therapy for obstructive lung disease. The pharmacologic basis for epinephrine's airway effects is described in

the NCBI Bookshelf review of epinephrine. The CDC/NIOSH epinephrine drug profile likewise identifies bronchodilation among its physiologic respiratory effects.

QUESTION NO: 25

Which of the following is not included in the femoral triangle?

A. Femoral Artery

B. Femoral Nerve

C. Femoral Vein

D. Femoral Ligament

ANSWER: D

Explanation:

Femoral Ligament is the correct answer. The femoral triangle is a subfascial space in the proximal anterior thigh that serves as an important anatomic landmark for examination and vascular access. Its major contents are arranged from lateral to medial as the femoral nerve, femoral artery, femoral vein, and the femoral canal containing lymphatic vessels and a deep inguinal lymph node. Therefore, the femoral artery, femoral nerve, and femoral vein are all structures found within the triangle.

“Femoral ligament” is not a standard structure described as a content of the femoral triangle. The triangle’s superior boundary is the *inguinal ligament*, which is distinct from its contents. In contrast, the ligament of the head of the femur is an intra-articular hip structure and is not located in the femoral triangle. This distinction between a triangle’s boundaries and the neurovascular structures passing through it is especially useful when identifying the femoral pulse or planning femoral vascular cannulation. For a detailed review, see StatPearls: Anatomy, Abdomen and Pelvis, Femoral Triangle and TeachMeAnatomy: The Femoral Triangle.

QUESTION NO: 26

A study is designed to evaluate the feasibility of acupuncture in children with chronic headaches. Sixty children with chronic headaches are recruited for the study. In addition to their usual therapy, all children are treated with acupuncture three times a week for 2 months. Which of the following best describes this study design?

A. Case-control

B. Case series

C. Cross-sectional

D. Cross-sectional

E. Historical cohort

F. Randomized clinical trial

ANSWER: B

Explanation:

Case series is correct because the investigators enroll a group of patients with the same clinical condition and describe their experience after all receive the same intervention. Here, 60 children with chronic headaches are prospectively given acupuncture in addition to their usual therapy, with the stated goal of assessing feasibility. There is no separate comparison group receiving usual therapy alone, sham acupuncture, or another intervention, and participants are not assigned to different treatment groups. Thus, the study can characterize implementation issues such as acceptability, adherence, and practical delivery of acupuncture, and it may describe outcomes observed in the treated patients. This is appropriately classified as a prospective case series, an uncontrolled descriptive study design. Case series are commonly used as an

early step in evaluating a new or less-established clinical approach, especially when the immediate question is whether a larger comparative study is practical. A subsequent controlled trial could be designed if feasibility and preliminary observations support further evaluation. The National Library of Medicine describes case reports and case series as descriptive observational evidence in its evidence-based medicine resources: NCBI Bookshelf: Study Designs in Epidemiology. Additional discussion of clinical research design and the role of uncontrolled studies is available from NCBI Bookshelf: Introduction to Clinical Research.

QUESTION NO: 27

Which of the following conditions correlate with the following information:

High pH

Neutral HCO3

Neutral BE Low pCO2

A. Respiratory alkalosis

B. Respiratory acidosis

C. Metabolic acidosis

D. Metabolic alkalosis

ANSWER: A

Explanation:

Respiratory alkalosis is correct because the primary disturbance is a decrease in arterial carbon dioxide tension (pCO_2), which raises blood pH. Carbon dioxide behaves as an acid in the bicarbonate buffer system: when ventilation exceeds metabolic carbon dioxide production, excess CO_2 is eliminated and the concentration of carbonic acid falls. This shifts the equilibrium toward a more alkaline pH. A neutral bicarbonate concentration and neutral base excess indicate that there is no substantial primary metabolic process causing the alkalemia. This pattern is therefore most consistent with an acute or minimally compensated respiratory alkalosis. In acute respiratory alkalosis, renal compensation has not yet had time to appreciably lower bicarbonate; with more sustained hypocapnia, the kidneys excrete bicarbonate and bicarbonate and base excess become reduced. Arterial blood gas interpretation begins by identifying whether pH shows acidemia or alkalemia, then determining whether the direction of pCO_2 accounts for that pH change. Here, high pH together with low pCO_2 directly identifies a respiratory alkalotic process. See the Merck Manual overview of respiratory alkalosis and the NCBI review of arterial blood gas interpretation.

QUESTION NO: 28

A 51-year-old man comes to the office because of a 6-month history of a lump on his tongue that is interfering with his speech and eating; he also has had a 6.8-kg (15-lb) weight loss during this period. He has smoked 1pack of cigarettes daily and has consumed six 12-oz bottles of beer on weekend nights during the past 30 years. His vital signs are within normal limits. Physical examination shows a 1.5-cm mass on the apex of the tongue. Further evaluation of the mass confirms squamous cell carcinoma. It is most appropriate to evaluate which of the following lymph nodes first for evidence of metastasis in this patient?

A. Inferior deep cervical

B. Parotid

C. Retropharyngeal

D. Submental

E. Superior deep cervical

ANSWER: D

Explanation:

Submental is correct because lymphatic spread from a tongue carcinoma is predicted primarily by the anatomic site of the lesion. The apex (tip) of the tongue drains first through lymphatic vessels to the submental lymph nodes, which lie in the submental triangle beneath the chin. Therefore, these nodes are the initial regional nodal basin to assess for metastatic involvement in a patient with squamous cell carcinoma located at the tongue apex.

Lingual lymphatic drainage is clinically important because the tongue has abundant lymphatic channels and oral tongue squamous cell carcinoma can metastasize early to regional cervical nodes. The midline location of the tongue tip also permits drainage across the midline; accordingly, examination and imaging should ultimately assess both sides of the neck as clinically indicated. However, when asked for the first nodal group expected to receive metastatic cells from a lesion specifically at the apex, the submental nodes are the appropriate answer. Further nodal drainage from this region can proceed into the deep cervical lymph-node chain. See the anatomic overview in StatPearls: Anatomy, Head and Neck, Tongue and the National Cancer Institute's discussion of oral cavity cancer staging and regional lymph nodes at NCI Oral Cavity Cancer Treatment.

QUESTION NO: 29

A 45-year-old male who has a BMI of 42 kg/m² undergoes a myocardial perfusion scintigraphy due to frequent chest pain during strenuous activities. An angiogram had earlier revealed a 95% occlusion in the proximal segment of his left circumflex coronary artery. There were no changes in the other coronary arteries. The most likely other finding in this patient is

A. inducible myocardial ischemia in the anterior segment of the left ventricle

B. inducible myocardial ischemia in the anteroseptal segments of the left ventricle

C. inducible myocardial ischemia in the inferior segments of the right ventricle

D. inducible myocardial ischemia in the inferolateral segment of the left ventricle

E. inducible myocardial ischemia in the septal segments of the left ventricle

ANSWER: D

Explanation:

Inducible myocardial ischemia in the inferolateral segment of the left ventricle is the expected finding. A severe proximal stenosis of the left circumflex coronary artery restricts the increase in coronary blood flow needed during exertion. Thus, during stress myocardial perfusion imaging, the myocardial territory supplied by this vessel will demonstrate relatively reduced tracer uptake compared with resting images, indicating reversible, or inducible, ischemia.

The left circumflex artery courses in the left atrioventricular groove and commonly supplies the lateral and posterolateral portions of the left ventricle through obtuse marginal branches. On standard perfusion maps, this distribution is typically described as lateral or inferolateral. The exact extent of inferior-wall involvement can vary with coronary dominance, because in left-dominant circulation the circumflex artery also gives rise to the posterior descending artery. Nevertheless, with an isolated proximal circumflex lesion, an inferolateral left-ventricular perfusion defect is the most appropriate regional correlate.

Myocardial perfusion scintigraphy is specifically designed to identify these stress-induced regional perfusion abnormalities and relate their distribution to coronary territories. See the NCBI review of coronary artery anatomy and the NCBI review of myocardial perfusion imaging.

QUESTION NO: 30

Which of the following is not directly related to a drug toxicity of Ibuprofen?

A. Nausea

B. Renal dysfunction

C. Anemia

D. Muscle wasting

ANSWER: D

Explanation:

Muscle wasting is the finding not directly associated with ibuprofen toxicity. Ibuprofen is a nonsteroidal anti-inflammatory drug that inhibits cyclooxygenase enzymes and reduces prostaglandin synthesis. Its clinically important toxic effects consequently center on prostaglandin-dependent gastrointestinal and renal physiology. Gastrointestinal irritation can manifest as nausea, and more serious mucosal injury or bleeding can lead to clinically significant blood loss and anemia. In the kidneys, reduced prostaglandin production can decrease renal perfusion, particularly in patients with volume depletion, preexisting kidney disease, heart failure, or concomitant use of certain antihypertensive medicines; this may cause renal dysfunction or acute kidney injury. These recognized adverse-effect pathways do not produce muscle catabolism or progressive loss of skeletal-muscle mass. Muscle wasting instead suggests a separate chronic systemic process, such as severe malnutrition, malignancy, endocrine disease, prolonged immobility, or another cause of cachexia. Therefore, among the listed findings, muscle wasting is the best answer to the question asking for an effect not directly related to ibuprofen toxicity. The prescribing information describes gastrointestinal adverse effects, anemia related to gastrointestinal blood loss, and renal toxicity: DailyMed: Ibuprofen prescribing information. Additional safety information is available from the FDA NSAID safety information.

QUESTION NO: 31

Which of the following is caused by a Vitamin C deficiency?

A. Fever

B. Anemia

C. Headaches

D. Nausea

ANSWER: B

Explanation:

Anemia is the best answer. Vitamin C deficiency causes scurvy, in which defective collagen formation leads to fragile capillaries and tissues. This can produce gingival, skin, and gastrointestinal bleeding; chronic blood loss may contribute to anemia. Vitamin C also has an important nutritional role in enhancing absorption of nonheme iron, the form of iron found in plant foods and many fortified foods. Therefore, low vitamin C intake can reduce iron absorption and contribute to iron-deficiency anemia, particularly when dietary iron intake is marginal. In established scurvy, anemia may be multifactorial, reflecting bleeding, reduced iron absorption, and sometimes associated nutritional deficiencies. Clinical findings that commonly accompany this process include fatigue and weakness, which can be worsened by anemia. The NIH Office of Dietary Supplements notes that inadequate vitamin C can lead to scurvy and identifies iron absorption as a relevant function of vitamin C. The Merck Manual also lists anemia among potential manifestations of scurvy. See the NIH Vitamin C Health Professional Fact Sheet and the Merck Manual discussion of vitamin C deficiency.

QUESTION NO: 32

A 27-year-old man is admitted to the hospital 45 minutes after being involved in a motor vehicle collision. Physical examination shows a sluggish response to stimuli. Neurologic examination shows no other abnormalities. A skull x-ray shows a linear, nondepressed basal skull fracture. Two weeks later, the patient develops polyuria and polydipsia. Laboratory studies show a serum glucose concentration within the reference range, increased serum osmolality, and decreased urine osmolality. Following the administration of desmopressin, urine osmolality increases. The beneficial effect of this drug is most likely due to activation of which of the following?

A. Adenylyl cyclase

B. Ca2+ channels

C. Janus kinase

D. Serine kinase

E. Tyrosine kinase

ANSWER: A

Explanation:

Adenylyl cyclase is correct. The delayed onset of polyuria, polydipsia, elevated serum osmolality, and inappropriately dilute urine after head trauma indicates central diabetes insipidus caused by deficient secretion of antidiuretic hormone (ADH, or vasopressin). The rise in urine osmolality after desmopressin confirms that the renal collecting ducts can respond appropriately to an ADH analog, supporting a central rather than nephrogenic process.

Desmopressin produces its antidiuretic effect primarily by binding vasopressin V2 receptors on principal cells of the renal collecting duct. V2 receptors are Gs protein-coupled receptors. Their activation stimulates adenylyl cyclase, increasing intracellular cyclic AMP and activating protein kinase A. This signaling promotes insertion of aquaporin-2 water channels into the apical membrane, allowing water reabsorption down the medullary osmotic gradient. As more water is reabsorbed, urine volume falls and urine osmolality rises. This is the specific renal mechanism responsible for the beneficial response described. See NCBI Bookshelf: Physiology, Vasopressin and NCBI Bookshelf: Central Diabetes Insipidus.

QUESTION NO: 33

The thymus is located with the _____.

A. Mediastinum

B. Peristinum

C. Epistinum

D. Endostinum

ANSWER: A

Explanation:

The thymus is located in the **mediastinum**, the central compartment of the thoracic cavity between the lungs. More specifically, the thymus is most prominent in the superior mediastinum and extends into the anterior portion of the inferior mediastinum, lying posterior to the sternum and anterior to the great vessels and pericardium. It is a primary lymphoid organ where T lymphocytes mature and undergo selection, an essential process for development of adaptive cellular immunity. The thymus is relatively large and active during infancy and childhood; after puberty, it gradually involutes and is increasingly replaced by fatty tissue, although residual thymic function can persist. Knowing its mediastinal location is clinically important when interpreting chest imaging and considering the differential diagnosis of an anterior mediastinal mass. Anatomical descriptions of the mediastinum and thymus are available from StatPearls: Anatomy, Thorax, Mediastinum and StatPearls: Anatomy, Thorax, Thymus.

QUESTION NO: 34

Which of the following syndromes corresponds to: presence of ipsilateral motor loss and contralateral spinothalamic tract damage?

A. Brown-Sequard syndrome

B. Thoracic outlet syndrome

C. Angelman's syndrome

D. Goodpasture's syndrome

ANSWER: A

Explanation:

Brown-Sequard syndrome is the characteristic clinical pattern caused by hemisection, or functional hemisection, of one side of the spinal cord. Injury to the lateral corticospinal tract produces upper motor neuron weakness below the lesion on the same side because these fibers have already crossed at the pyramidal decussation in the caudal medulla before descending in the spinal cord. Thus, a unilateral spinal-cord lesion causes ipsilateral motor loss below its level.

The spinothalamic system carries pain and temperature sensation. Primary sensory fibers entering the cord synapse and then cross through the anterior white commissure, usually within one to two spinal segments of entry, before ascending contralaterally. Consequently, damage to one side of the cord interrupts spinothalamic fibers that originated from the opposite side of the body, causing contralateral loss of pain and temperature sensation beginning slightly below the lesion. The classic syndrome may also include ipsilateral loss of vibration, proprioception, and fine touch from dorsal-column involvement. This combination of ipsilateral motor findings with contralateral spinothalamic sensory loss identifies Brown-Sequard syndrome. See StatPearls: Brown-Séquard Syndrome and StatPearls: Neuroanatomy, Spinothalamic Tract.

QUESTION NO: 35

The primary effect of cocaine on the nervous system is that cocaine blocks the re-uptake of ____.

A. Monoamines

B. Trans amines

C. Catecholamine

D. Monoamine oxidase

ANSWER: A

Explanation:

Monoamines is correct because cocaine inhibits presynaptic monoamine transporters, reducing removal of neurotransmitter from the synaptic cleft. Its clinically important action is blockade of the dopamine transporter, which increases synaptic dopamine and contributes to euphoria, reinforcement, and the drug's high abuse potential. Cocaine also inhibits norepinephrine and serotonin reuptake transporters, increasing signaling through those monoamine pathways. Thus, "monoamines" is the best inclusive term for the primary neurotransmitter reuptake effect of cocaine. The increased norepinephrine activity also helps account for prominent sympathetic effects such as tachycardia, hypertension, and vasoconstriction. This mechanism is transporter blockade rather than stimulation of neurotransmitter synthesis or direct release from presynaptic terminals. The National Institute on Drug Abuse describes cocaine's elevation of dopamine signaling by preventing dopamine recycling into the neuron that released it. Standard pharmacology references likewise identify inhibition of dopamine, norepinephrine, and serotonin reuptake as cocaine's central nervous system mechanism. See the National Institute on Drug Abuse cocaine overview and NCBI Bookshelf review of cocaine toxicity.

QUESTION NO: 36

A 25-day-old male is brought to the physician due to seizures, rigidity and frequent viral infections. Physical examination reveals a neonate with cyanosis in the lower extremities. The most likely cause of these findings is a

A. defect in the IL-2R gamma chain

B. defect in the lysosomal trafficking regulation gene

C. defect in the tyrosinase kinase gene

D. deletion in chromosome 22q11.2

E. mutation in the STAT3 gene

ANSWER: D

Explanation:

Deletion in chromosome 22q11.2 is correct because this presentation is characteristic of DiGeorge syndrome (22q11.2 deletion syndrome). The deletion disrupts normal development of the third and fourth pharyngeal pouches. Failed thymic development produces deficient T-lymphocyte maturation and therefore impaired cellular immunity, which can manifest early as recurrent or unusually frequent viral infections. Failed parathyroid development causes low parathyroid hormone levels and hypocalcemia; in a neonate, hypocalcemia can produce neuromuscular irritability, rigidity/tetany, and seizures. The cyanosis suggests an associated conotruncal congenital cardiac defect, such as tetralogy of Fallot or truncus arteriosus, which is a classic cardiac association of this syndrome. Thus, the combination of neonatal hypocalcemic seizures, T-cell immunodeficiency, and cyanotic congenital heart disease localizes to the developmental consequences of a 22q11.2 deletion. The diagnosis can be supported by lymphocyte subset testing showing low T-cell numbers, serum testing showing hypocalcemia with inappropriately low parathyroid hormone, and genetic testing to identify the deletion. Authoritative overviews of the syndrome and its characteristic immune, endocrine, and cardiac manifestations are available from GeneReviews: 22q11.2 Deletion Syndrome and the MedlinePlus Genetics review of 22q11.2 deletion syndrome.

QUESTION NO: 37

Which of the following is an effect of a diuretic?

A. Decreased Cardiac Output

B. Increased fluid volume

C. Increased sodium re-absorption

D. Increased chloride ion re-absorption

ANSWER: A

Explanation:

Decreased Cardiac Output is an effect that can result from diuretic therapy. Diuretics promote renal excretion of sodium and water, producing natriuresis and diuresis. The resulting reduction in total body fluid and intravascular plasma volume lowers venous return to the heart (preload). Because cardiac output is influenced by preload and stroke volume, a sufficiently reduced circulating volume can decrease stroke volume and cardiac output. This volume-reducing effect is clinically useful in conditions characterized by fluid overload, such as heart failure, pulmonary edema, edema due to renal or hepatic disease, and hypertension. In heart failure, reducing preload can relieve congestion and decrease the work the heart must perform; the degree of cardiac-output reduction depends on the patient's baseline volume status, cardiac function, diuretic dose, and response to treatment. Diuretics therefore require monitoring of blood pressure, weight, renal function, electrolytes, and symptoms of excessive volume depletion. The pharmacologic basis for this effect is increased urinary sodium excretion, with water following sodium osmotically, rather than retention of sodium, chloride, or fluid. See StatPearls: Diuretic Medications and Merck Manual: Drugs for Heart Failure.

QUESTION NO: 38

Which of the following is a disease characterized by the presence of hives?

A. Keloid

B. Seborrhea

C. Eczema

D. Urticaria

ANSWER: D

Explanation:

Urticaria is the medical term for hives, a condition characterized by transient, pruritic wheals (raised, erythematous or skin-colored swellings) that can occur anywhere on the body. Individual lesions commonly change shape, migrate, and resolve within 24 hours, although new wheals may continue to appear. The underlying process is activation of cutaneous mast cells and release of mediators, particularly histamine, which increases vascular permeability and produces localized superficial edema. Urticaria may be acute or chronic and can be triggered by infections, medications, foods, physical stimuli, or autoimmune mechanisms; in many cases, particularly chronic cases, no specific trigger is identified. Angioedema may occur at the same time, causing deeper swelling of tissues such as the lips, eyelids, tongue, or extremities. Recognition of urticaria is clinically important because hives accompanied by respiratory symptoms, hypotension, or significant mucosal swelling can indicate anaphylaxis and require urgent treatment. The American Academy of Dermatology describes hives as itchy, raised welts on the skin, and the Merck Manual identifies urticaria as the characteristic wheal-and-flare eruption. See American Academy of Dermatology: Hives overview and Merck Manual: Urticaria.

QUESTION NO: 39

Which of the following is caused by a B6 deficiency?

A. Excessive irritability

B. Nonproductive cough

C. Dry mouth

D. Depression

ANSWER: A

Explanation:

Excessive irritability is a recognized neuropsychiatric manifestation of vitamin B6 (pyridoxine) deficiency. Vitamin B6 is converted to pyridoxal 5′-phosphate, a coenzyme required for amino-acid metabolism and for synthesis of several neurotransmitters, including gamma-aminobutyric acid, serotonin, dopamine, and norepinephrine. When pyridoxal 5′-phosphate availability is inadequate, altered neurotransmitter production can cause central nervous system symptoms, including irritability and, in severe cases, confusion or seizures. This association is especially important in infants and in patients with increased risk of deficiency, such as those with malabsorption, alcohol use disorder, kidney disease, or exposure to medications that interfere with pyridoxine activity, including isoniazid. The clinical findings of deficiency can also include dermatitis, cheilosis, glossitis, and microcytic or sideroblastic anemia, reflecting B6's wider metabolic roles. The National Institutes of Health describes neurologic and neuropsychiatric effects among the manifestations of severe B6 deficiency, and Merck Manual notes irritability among the possible clinical features. See the NIH Vitamin B6 Health Professional Fact Sheet and the Merck Manual discussion of vitamin B6 deficiency.

QUESTION NO: 40

Which of the following is not a type of embolus?

A. Air

B. Bacteria

C. Tumor

D. Viral

ANSWER: D

Explanation:

Viral is correct because an embolus is a detached intravascular mass that travels through the bloodstream and lodges at a distant site, potentially obstructing blood flow. Emboli are classified by the physical material that is transported, such as solid material, liquid material, or gas. A virus is a microscopic infectious particle that infects host cells and may circulate in blood during viremia, but it is not itself a macroscopic intravascular mass capable of mechanically lodging in a vessel in the

pathologic sense of an embolus. Viral disease can contribute to inflammation, endothelial injury, or coagulation abnormalities in some clinical settings, yet those effects do not make a virus an embolic material. The defining mechanism of embolism is physical vascular occlusion by transported material, rather than dissemination of an infectious agent at the cellular or molecular level. This distinction is central to pathology definitions of embolism and to clinical descriptions of embolic vascular disease. See the StatPearls review of acute pulmonary embolism and the Merck Manual overview of embolic disorders for the clinical definition and mechanism of embolic obstruction.

QUESTION NO: 41

Which of the following is not an adverse effect of glucagon?

A. Allergic reaction

B. Vomiting

C. Nausea

D. Fever

ANSWER: D

Explanation:

Fever is the best answer because it is not a recognized expected adverse effect of glucagon when it is used to treat severe hypoglycemia. Glucagon rapidly raises plasma glucose by promoting hepatic glycogen breakdown and gluconeogenesis. Its adverse-effect profile is chiefly related to gastrointestinal symptoms, particularly as glucose levels recover and oral intake resumes, as well as occasional hypersensitivity reactions in susceptible individuals. In contrast, fever is not an expected pharmacologic consequence of glucagon administration and is not included among the typical adverse reactions in product prescribing information. A patient who develops fever after receiving glucagon should therefore be assessed for another clinical cause, such as an underlying infection or illness that may also have contributed to the hypoglycemic episode, rather than attributing the fever to glucagon itself. The FDA prescribing information for glucagon products provides the most appropriate reference for expected adverse reactions and safety considerations. See the FDA prescribing information for Gvoke (glucagon) and the DailyMed glucagon labeling database.

QUESTION NO: 42

Which of the following is not associated with Wilson's disease?

A. Asterixis

B. Basal ganglia

C. Cirrhosis

D. Pancreatitis

ANSWER: D

Explanation:

Pancreatitis is the best answer because it is not a characteristic or routinely recognized clinical manifestation of Wilson disease. Wilson disease is an inherited ATP7B-related disorder of copper transport that causes pathologic copper accumulation, with clinically important effects centered on hepatic and neurologic systems. Standard descriptions therefore emphasize liver disease ranging from biochemical abnormalities to hepatitis, cirrhosis, or acute liver failure, as well as neurologic disease caused by involvement of the basal ganglia and related brain structures. Advanced hepatic dysfunction may also produce manifestations of hepatic encephalopathy. In contrast, pancreatic inflammation is not part of the usual diagnostic phenotype, screening evaluation, or core organ-involvement pattern used to recognize Wilson disease. Although an individual with Wilson disease could independently develop pancreatitis for common reasons, that coexistence would not establish pancreatitis as a typical disease association. This distinction is important in exam questions: choose the finding outside the established hepatic, neuropsychiatric, ophthalmologic, hematologic, and renal manifestations of impaired copper

excretion. Authoritative clinical summaries of the recognized manifestations and evaluation are available in GeneReviews: Wilson Disease and the Merck Manual Professional Edition.

QUESTION NO: 43

Which of the following syndromes corresponds to: increased pulmonary permeability and fluid entering the lung space?

A. Acute coronary syndrome

B. ARDS

C. Budd-Chiari syndrome

D. DiGeorge's syndrome

ANSWER: B

Explanation:

ARDS is correct because acute respiratory distress syndrome is a form of noncardiogenic pulmonary edema caused by diffuse inflammatory injury to the alveolar-capillary membrane. This injury increases pulmonary capillary permeability, allowing protein-rich fluid to leak from the intravascular space into the pulmonary interstitium and alveoli. Alveolar flooding disrupts gas exchange, producing severe hypoxemia and decreased lung compliance. Clinically, ARDS often develops within 1 week of a major insult such as sepsis, pneumonia, aspiration of gastric contents, trauma, pancreatitis, or multiple transfusions. The Berlin definition characterizes ARDS by acute onset, bilateral pulmonary opacities, respiratory failure not fully explained by cardiac failure or fluid overload, and impaired oxygenation. The key concept in the question is permeability-related fluid entry into the lungs rather than increased hydrostatic pressure from left-sided heart failure. This pathophysiology directly identifies ARDS. For a detailed overview, see the NCBI Bookshelf review of acute respiratory distress syndrome and the Berlin definition of ARDS.

QUESTION NO: 44

A 35-year-old male comes to the physician due to fatigue. He recently returned from a trip to Japan. Laboratory results are as follows:

Hemoglobin – 9 g/dL

Mean corpuscular volume – 108 fl

MCHC – 33 g/dL

Mean corpuscular hemoglobin – 35 pg

A peripheral blood smear is obtained which reveals enlarged red blood cells and hypersegmented neutrophils. These findings were most likely caused by

A. Diphyllobothrium latum

B. Echinococcus granulosus

C. Onchocerca volvulus

D. Schistosoma haematobium

E. Taenia solium

ANSWER: A

Explanation:

Diphyllobothrium latum is correct because infection with this fish tapeworm can produce vitamin B12 deficiency, leading to megaloblastic anemia. The low hemoglobin explains the patient's fatigue, while the increased mean corpuscular volume and smear findings of macrocytes (enlarged red cells) and hypersegmented neutrophils are classic hematologic manifestations of impaired DNA synthesis due to B12 deficiency. Humans acquire the parasite by eating raw or inadequately cooked freshwater fish containing larval forms. This exposure is particularly relevant after travel to areas where raw fish dishes are consumed, including Japan. Once present in the intestine, the adult tapeworm competes with the host for dietary vitamin B12 and can substantially reduce B12 availability. Deficient B12 disrupts thymidine synthesis in rapidly dividing bone marrow precursors, producing ineffective erythropoiesis and the characteristic megaloblastic morphology seen on the peripheral smear. The CDC describes diphyllobothriid tapeworm infection as a fish-borne intestinal cestode infection and notes that it may cause B12 deficiency and anemia. Treatment generally includes praziquantel, along with assessment and replacement of vitamin B12 when deficiency is present. See the CDC DPDx overview of diphyllobothriasis and the Merck Manual discussion of diphyllobothriasis.

QUESTION NO: 45

Which of the following syndromes corresponds to: right sided valvular disease and diarrhea?

A. Job's syndrome

B. Wiskott-Aldrich syndrome

C. Carcinoid syndrome

D. Mallory-Weiss syndrome

ANSWER: C

Explanation:

Carcinoid syndrome is correct because vasoactive substances released by a neuroendocrine tumor, most importantly serotonin, produce the characteristic combination of secretory diarrhea and right-sided valvular heart disease. These tumors commonly arise in the gastrointestinal tract. When tumor products reach the systemic circulation—typically after hepatic metastasis or from a primary tumor that bypasses hepatic metabolism—they can cause the systemic manifestations of carcinoid syndrome. Serotonin increases intestinal motility and secretion, resulting in recurrent watery diarrhea. It also promotes fibrous plaque formation on the endocardial surfaces of the right heart, especially the tricuspid and pulmonic valves. This fibrosis can lead to tricuspid regurgitation and pulmonic stenosis. The left-sided valves are usually spared because the lungs metabolize circulating serotonin before it reaches the left side of the heart; left-sided involvement may occur in unusual settings such as a bronchial carcinoid or a right-to-left cardiac shunt. Evaluation can include measurement of 24-hour urinary 5-hydroxyindoleacetic acid, a serotonin metabolite, and assessment of cardiac involvement with echocardiography. Additional clinical features may include episodic flushing and bronchospasm. See the NCBI Bookshelf review of carcinoid syndrome and the Merck Manual overview of carcinoid syndrome.

QUESTION NO: 46

A couple comes for preconceptional genetic counseling because they both have a family history of α-thalassemia. The woman has a minimally decreased hemoglobin concentration. Genetic studies show a single gene deletion. The man has microcytic anemia and a two-gene deletion. If the two-gene deletion is in trans (one deletion on the maternal gene and one deletion on the paternal gene), which of the following percentages of their offspring will have a two-gene deletion?

A. 0%

B. 25%

C. 50%

D. 75%

E. 100%

ANSWER: C

Explanation:

50% is correct. Each person normally has four alpha-globin genes, arranged as two genes on each chromosome 16. A single-gene deletion in the woman is represented as −α/αα, so she produces two types of gametes in equal proportions: one carrying a chromosome with one deleted alpha-globin gene (−α) and one carrying a normal two-gene chromosome (αα).

A trans two-gene deletion in the man is represented as −α/−α: each homologous chromosome has one deleted alpha-globin gene. Consequently, all of his gametes carry −α. When a paternal −α gamete combines with a maternal −α gamete, the child has −α/−α, a two-gene deletion in trans. This occurs for one-half of conceptions. When the paternal −α combines with the woman's normal αα gamete, the child has a single-gene deletion instead. Thus, Mendelian segregation predicts that 50% of offspring will inherit a two-gene alpha-globin deletion. This genotype commonly produces alpha-thalassemia trait with microcytosis and mild anemia. See GeneReviews: Alpha-Thalassemia and MedlinePlus Genetics: Alpha thalassemia.

QUESTION NO: 47

Which of the following does not require the pre-cursor progesterone?

A. Cortisol

B. Testosterone

C. ACTH

D. Aldosterone

ANSWER: C

Explanation:

ACTH is correct because it is a peptide hormone, not a steroid hormone synthesized from cholesterol through progesterone-containing steroidogenic pathways. ACTH is produced by corticotroph cells of the anterior pituitary as part of the larger precursor protein proopiomelanocortin (POMC). After POMC is translated, it undergoes enzymatic cleavage to generate ACTH, which is then released into the circulation. Its principal role is to stimulate the adrenal cortex, especially the zona fasciculata and zona reticularis, to increase steroid hormone synthesis.

Progesterone is an intermediate in adrenal and gonadal steroidogenesis. In contrast, ACTH synthesis depends on gene transcription, peptide translation, post-translational processing, and regulated secretion rather than enzymatic conversion of progesterone. This distinction reflects the broad biochemical difference between peptide hormones, which are encoded as proteins and processed from precursor polypeptides, and steroid hormones, which are derived from cholesterol. The Endotext review of adrenal steroidogenesis describes cholesterol conversion to pregnenolone and progesterone in steroid production, while its discussion of ACTH describes POMC as the peptide precursor for ACTH. See Endotext: Adrenal Steroidogenesis and Endotext: The Hypothalamic-Pituitary-Adrenal Axis.

QUESTION NO: 48

Which of the following best describes the inheritance pattern of hemophilia A?

A. Autosomal dominant

B. Autosomal recessive

C. X-linked dominant

D. X-linked recessive

ANSWER: D

Explanation:

X-linked recessive is the inheritance pattern of hemophilia A. Hemophilia A results from pathogenic variants affecting factor VIII. Because the responsible gene is located on the X chromosome, males with the pathogenic variant are generally affected, whereas heterozygous females are usually carriers but can have variable bleeding symptoms.

An affected male transmits his altered X chromosome to all daughters and none of his sons. A carrier female has a 50% chance of transmitting the altered allele to each child. See GeneReviews: Hemophilia A.

QUESTION NO: 49

Which of the following types of immunoglobulins is located on the surface of most B Lymphocytes?

A. IgA

B. IgM

C. IgD

D. IgE

ANSWER: C

Explanation:

IgD is correct because it is a membrane-bound immunoglobulin characteristically expressed on the surface of mature, naïve B lymphocytes. Surface immunoglobulin serves as the antigen-recognition component of the B-cell receptor complex. When antigen binds and cross-links these receptors, it initiates signaling events that contribute to lymphocyte activation, antigen uptake, processing, and presentation to helper T cells. IgD is coexpressed with membrane IgM on many mature naïve cells through alternative RNA processing of the same heavy-chain gene transcript, but IgD is the immunoglobulin class classically identified as being located on the surface of most B lymphocytes in questions testing immunoglobulin distribution and function. Its presence is therefore a useful marker of mature naïve B-cell status before antigen-driven activation and class-switch recombination alter the antibody isotype produced by descendant plasma cells. Surface expression is distinct from secreted antibody: the membrane form includes a hydrophobic segment that anchors it in the cell membrane and associates with signaling proteins needed for receptor function. See the NCBI Bookshelf overview of B-cell receptors and the Merck Manual discussion of humoral immunity.

QUESTION NO: 50

A 2-year-old boy has a CT scan of the head performed after a pediatrician notices a disproportionate growth in his head circumference compared with the rest of the body. The scan demonstrates a large choroid plexus papilloma involving the body of the right lateral ventricle. Which of the following brain structures might be affected by direct extension of this tumor?

A. Caudate nucleus

B. Pons

C. Cerebellum

D. Hippocampus

E. Hypothalamus

ANSWER: A

Explanation:

Caudate nucleus is correct because it lies immediately adjacent to the body of the lateral ventricle. The caudate is a C-shaped basal ganglia structure that follows the contour of the lateral ventricle; its body contributes to the lateral ventricular wall and is closely related to the ventricular body. Therefore, a large choroid plexus papilloma arising within the body of the right lateral ventricle can directly compress or extend into the caudate nucleus. In young children, choroid plexus papillomas most often arise in the lateral ventricles and may grow sufficiently to produce increased intracranial pressure, macrocephaly, or hydrocephalus through excess cerebrospinal fluid production and/or obstruction of ventricular flow. The question

specifically asks about direct extension, so the key is the local anatomy of the ventricular body rather than structures related to other portions of the ventricular system. Detailed relationships of the lateral ventricles and caudate nucleus are reviewed in StatPearls: Neuroanatomy, Lateral Ventricles. The typical location and clinical behavior of these tumors in children are summarized in StatPearls: Choroid Plexus Papilloma.

QUESTION NO: 51

Which of the following is not directly related to a drug toxicity of Nitroglycerin?

A. Headaches

B. Tachycardia

C. Dizziness

D. Projectile vomiting

ANSWER: D

Explanation:

Projectile vomiting is not a characteristic direct manifestation of nitroglycerin toxicity. Nitroglycerin increases nitric oxide signaling and causes vascular smooth-muscle relaxation, with prominent venodilation and, at higher exposure, arterial dilation. Its clinically important toxic effects therefore stem from excessive vasodilation and compensatory autonomic responses, as well as the potential for methemoglobinemia with substantial overdose. Projectile vomiting is instead a descriptive pattern of forceful emesis classically associated with increased intracranial pressure or certain gastrointestinal obstructive processes; it is not a standard finding used to identify nitrate toxicity. Although nonspecific nausea or vomiting can occur in many illnesses and may occasionally accompany hypotension, the specific finding of projectile vomiting does not follow directly from nitroglycerin's pharmacologic mechanism and is not listed among the expected hallmark adverse or toxic effects. This makes projectile vomiting the best answer to a question asking for a finding not directly related to nitroglycerin toxicity. For prescribing safety information and recognized adverse effects, see the DailyMed nitroglycerin label and the NCBI StatPearls review of nitroglycerin.

QUESTION NO: 52

A 6-year-old male is brought to the physician due to a sore throat, fever and malaise. Physical examination reveals an erythematous tongue and a sandpaper-like body rash. This most likely sequelae of this infection includes all of these except

A. hematuria

B. joint pain

C. muscular spasm

D. myocarditis

E. nodules under the skin

ANSWER: C

Explanation:

The presentation is classic for scarlet fever caused by group A streptococcal pharyngitis: fever and sore throat accompanied by a diffuse rough, "sandpaper" rash and an erythematous tongue. Important nonsuppurative sequelae of group A streptococcal infection are acute rheumatic fever and poststreptococcal glomerulonephritis. Acute rheumatic fever results from an immune-mediated response after pharyngitis, with molecular mimicry involving streptococcal antigens and host tissues. Its major manifestations are described by the Jones criteria and include carditis, migratory polyarthritis, Sydenham chorea, erythema marginatum, and subcutaneous nodules. Although Sydenham chorea causes involuntary, irregular movements due to central nervous system involvement, "muscular spasm" is not a recognized characteristic sequela or Jones criterion. Therefore, *muscular spasm* is the best exception. Appropriate antibiotic treatment of confirmed group A

streptococcal pharyngitis is important because it prevents acute rheumatic fever, even though it does not reliably prevent poststreptococcal glomerulonephritis. See the CDC clinical guidance on acute rheumatic fever and the CDC overview of group A Streptococcus.

QUESTION NO: 53

A newborn infant who was apparently healthy at birth develops aspiration pneumonia in the first 2 days of life. All attempts to feed the infant cause it to cough and choke. Which of the following abnormalities is the most likely cause of the infant's difficulties?

A. Bronchogenic cysts

B. Congenital pulmonary cysts

C. Posterior deviation of the tracheoesophageal septum

D. Pulmonary immaturity

E. Pulmonary sequestration

ANSWER: C

Explanation:

Posterior deviation of the tracheoesophageal septum is correct because it can produce esophageal atresia with a distal tracheoesophageal fistula, the most common form of this congenital anomaly. During normal embryologic development, the tracheoesophageal septum separates the primitive foregut into a ventral respiratory tube and a dorsal esophagus. Posterior displacement disrupts this partitioning, leaving the upper esophagus as a blind-ending pouch and commonly maintaining an abnormal connection between the distal esophagus and trachea.

The clinical presentation is classic: an infant may initially appear well but develops coughing, choking, cyanotic episodes, and respiratory distress when oral feeding begins. Milk and oral secretions cannot pass normally through the atretic esophagus and may enter the airway, causing recurrent aspiration and early aspiration pneumonia. A distal fistula can additionally permit gastric contents to reach the tracheobronchial tree. This diagnosis requires prompt stabilization, avoidance of oral feeding, suctioning of the proximal pouch, and pediatric surgical evaluation. It is also important to assess for associated congenital anomalies, particularly the VACTERL spectrum. Further embryologic and clinical discussion is available from StatPearls: Tracheoesophageal Fistula and Merck Manual: Esophageal Atresia and Tracheoesophageal Fistula.

QUESTION NO: 54

Which of the following is caused by a Vitamin D deficiency?

A. Edema

B. Anemia

C. Lupus

D. Rickets

ANSWER: D

Explanation:

Rickets is correct because vitamin D is essential for maintaining calcium and phosphate homeostasis and for normal mineralization of growing bone. Vitamin D increases intestinal absorption of calcium and phosphate; when deficiency occurs in children, inadequate mineral availability prevents proper mineralization at the growth plates. This produces the characteristic skeletal disorder called rickets. Clinical manifestations can include impaired linear growth, bone pain, delayed motor development or muscle weakness, widening of the wrists and ankles, and deformities of weight-bearing bones such as bowing of the legs. The risk is greatest during periods of rapid growth and when vitamin D intake, sun exposure, or intestinal

absorption is insufficient. The diagnosis is supported by the clinical setting and biochemical findings related to disturbed mineral metabolism, with radiographic changes at the metaphyses in established disease. Timely vitamin D repletion, together with adequate dietary calcium when needed, restores mineralization and helps prevent persistent skeletal deformity. For further review, see the NCBI StatPearls review of rickets and the NIH Office of Dietary Supplements vitamin D fact sheet.

QUESTION NO: 55

Another name for the (Billroth I) procedure is a _____.

A. Gastrojejunostomy

B. Gastroduodenostomy

C. Cholangiogram

D. Cholecystogram

ANSWER: B

Explanation:

Billroth I is correctly termed a **gastroduodenostomy**. It is a reconstructive operation usually performed after distal gastrectomy, in which the remaining portion of the stomach is directly anastomosed to the duodenum. This restores continuity of the gastrointestinal tract while maintaining passage of food through the duodenum. The procedure is classically associated with treatment of peptic ulcer disease and may also be used as reconstruction following gastric resection for selected distal gastric lesions, provided that the duodenum can be safely mobilized and joined to the gastric remnant without excessive tension. The name describes the anatomy of the anastomosis: “gastro-” refers to the stomach and “duodeno-” to the duodenum. Accurate identification of this terminology is important because postoperative anatomy affects expected physiology, nutritional consequences, and interpretation of imaging or endoscopic findings. A surgical review from StatPearls: Gastric Resection describes Billroth I reconstruction as an anastomosis between the stomach and duodenum. The National Cancer Institute Dictionary of Cancer Terms likewise defines gastroduodenostomy as a surgical connection between the stomach and duodenum.

QUESTION NO: 56

Which of the following is not a gram-negative bug?

A. Clostridium perfringens

B. Vibrio cholerae

C. Escherichia coli

D. Bordetella pertussis

ANSWER: A

Explanation:

Clostridium perfringens is the correct answer because it is a gram-positive bacterium, not a gram-negative organism. It is a large, boxcar-shaped, spore-forming anaerobic rod in the genus *Clostridium* (now commonly classified within *Clostridium* sensu stricto). Its thick peptidoglycan cell wall retains crystal violet during Gram staining, producing the characteristic gram-positive appearance. Clinically, *C. perfringens* is important because its toxins can cause rapidly progressive clostridial myonecrosis (gas gangrene), food poisoning, and soft-tissue infection. In tissue infection, it can produce gas and extensive muscle necrosis, making prompt recognition and management essential. Although organisms may occasionally stain variably in older cultures or clinical specimens, its fundamental cell-wall classification is gram-positive. This makes *Clostridium perfringens* the organism that does not belong among gram-negative bacteria in this question. Further review is available in the NCBI Bookshelf review of Clostridium perfringens infection and the Merck Manual discussion of clostridial soft-tissue infections.

QUESTION NO: 57

Multiple sclerosis is a disease that attacks the _____ of neurons in the CNS.

A. Myelin sheaths

B. Axon terminals

C. Sodium channels

D. Nicotinic receptors

ANSWER: A

Explanation:

Multiple sclerosis is an immune-mediated disorder in which inflammatory processes within the central nervous system damage myelin, the insulating sheath that surrounds many axons. In the CNS, myelin is produced by oligodendrocytes and enables rapid, efficient saltatory conduction of electrical impulses along nerve fibers. When myelin is damaged or lost, nerve-signal transmission becomes slowed, blocked, or poorly coordinated. This disruption accounts for the variable neurologic manifestations of multiple sclerosis, which can include visual disturbances, sensory symptoms, weakness, impaired coordination, and cognitive changes depending on the affected CNS pathways. Therefore, **Myelin sheaths** correctly completes the statement because demyelination is the defining pathologic feature of multiple sclerosis. Axons may also sustain injury as the disease progresses, but the primary characteristic target is CNS myelin and the oligodendrocytes responsible for maintaining it. The National Institute of Neurological Disorders and Stroke describes MS as a disease in which the immune system attacks myelin in the brain and spinal cord. Further clinical and pathophysiologic information is available from the National Institute of Neurological Disorders and Stroke and the National Multiple Sclerosis Society.

QUESTION NO: 58

Which of the following is not true concerning Brown-Sequard syndrome?

A. Contralateral spinothalamic deficits

B. Ipsilateral spinothalamic deficits

C. Ipsilateral dorsal column deficits

D. Ipsilateral pyramidal tract deficits

ANSWER: B

Explanation:

Ipsilateral spinothalamic deficits is the statement that is not true in the classic pattern of Brown-Séquard syndrome. This syndrome results from hemisection or major unilateral damage to the spinal cord. Pain and temperature sensation travel in the spinothalamic tract, whose primary sensory neurons enter the cord and typically ascend or descend briefly in Lissauer tract before crossing through the anterior white commissure to the opposite side. Consequently, a unilateral cord lesion interrupts spinothalamic fibers that have already crossed from the contralateral side. The characteristic pain-and-temperature loss therefore begins approximately one to two spinal segments below the lesion and occurs on the side opposite the cord lesion, rather than ipsilaterally.

This crossing pattern is central to localizing a hemicord lesion clinically. The sensory finding is usually paired with same-side corticospinal tract dysfunction and loss of dorsal-column modalities below the lesion, producing the recognizable Brown-Séquard pattern. A small, segmental same-side pain or temperature deficit can occur at the precise level of injury if dorsal-root entry fibers or the dorsal horn are affected, but that does not alter the standard description of the long-tract deficit tested here. See StatPearls: Brown-Séquard Syndrome and StatPearls: Neuroanatomy, Spinothalamic Tract.

QUESTION NO: 59

Which of the following hormones causes increased atrial pressure and decreases sodium reabsorption in the kidneys?

A. Atrial natriuretic peptide

B. PTH

C. Aldosterone

D. Vasopressin

ANSWER: A

Explanation:

Atrial natriuretic peptide is the intended correct answer because it is a cardiac hormone released by atrial myocytes when atrial stretch rises, such as during increased intravascular volume and elevated atrial pressure. Its physiologic purpose is to reduce effective circulating volume and blood pressure by promoting natriuresis, the renal excretion of sodium. ANP increases glomerular filtration and reduces tubular sodium reabsorption, particularly through actions that oppose the renin-angiotensin-aldosterone system. It also suppresses renin and aldosterone secretion, further limiting renal sodium retention. Water follows the excreted sodium, so this response helps lower extracellular-fluid volume and relieve atrial distension. Strictly stated, increased atrial pressure stimulates ANP release rather than ANP causing increased atrial pressure; however, the association of atrial stretch with reduced renal sodium reabsorption identifies atrial natriuretic peptide. This role is summarized by the NCBI Bookshelf review of natriuretic peptides and the NCBI Bookshelf overview of renal sodium handling.

QUESTION NO: 60

Which of the following is the source cell for the secretion Pepsinogen?

A. Chief cell

B. Plasma cell

C. G cell

D. Parietal cell

ANSWER: A

Explanation:

Chief cell is correct. Gastric chief cells, also called zymogenic or peptic cells, are located predominantly in the base of the fundic (oxyntic) glands of the stomach body and fundus. They synthesize and secrete pepsinogen, an inactive zymogen that protects the secreting cell and gastric mucosa from proteolytic injury. After secretion into the gastric lumen, the acidic environment converts pepsinogen to pepsin. This conversion is initiated by gastric hydrochloric acid and can then be amplified by pepsin-mediated activation of additional pepsinogen molecules. Pepsin functions best at low gastric pH and begins digestion of dietary proteins by cleaving peptide bonds. Chief cells also produce gastric lipase, but their classic histologic and physiologic association in USMLE-style questions is secretion of pepsinogen. Their basal location within gastric glands and abundant rough endoplasmic reticulum reflect their specialized role in protein synthesis and secretion. For additional review, see the NCBI Bookshelf overview of gastric acid secretion and physiology and NCBI Bookshelf review of peptic ulcer disease.

QUESTION NO: 61

The Tzanck test is not used on which of the following viruses?

A. VZV

B. HSV-2

C. HHV-8

D. HSV-1

ANSWER: C

Explanation:

HHV-8 is the correct answer. A Tzanck smear is a rapid cytologic examination of cells scraped from the base of a fresh vesicle or ulcer. Its classic finding is multinucleated giant cells with viral cytopathic changes, a pattern associated with infections caused by herpes simplex viruses and varicella-zoster virus. HHV-8, also called Kaposi sarcoma-associated herpesvirus, has a different clinical and diagnostic context. It is linked to Kaposi sarcoma and certain lymphoproliferative disorders rather than the typical acute vesicular lesions from which a useful Tzanck preparation is obtained. Evaluation for HHV-8-related disease generally relies on histopathology of affected tissue and demonstration of viral infection with methods such as immunohistochemistry for the latent nuclear antigen or molecular testing, as appropriate to the clinical setting. Thus, the Tzanck test does not serve as a diagnostic test for HHV-8 infection. For further discussion of herpesvirus diagnostic methods, see the CDC overview of herpes and the NCBI StatPearls review of Kaposi sarcoma.

QUESTION NO: 62

A genotypic male (XY) is born with feminized external genitalia. The testes are retained within the abdominal cavity, and the internal reproductive tracts exhibit the normal male phenotype. Which of the following could account for this abnormal development?

A. Complete androgen resistance

B. 5α-reductase deficiency

C. 17α-hydroxylase deficiency

D. Sertoli-only syndrome

E. Testicular dysgenesis

ANSWER: B

Explanation:

5α-reductase deficiency accounts for an XY individual with testes, preserved male internal reproductive structures, and feminized or undervirilized external genitalia. Fetal Sertoli cells produce anti-Müllerian hormone, which suppresses Müllerian structures, while fetal Leydig cells produce testosterone. Testosterone acting through androgen receptors supports differentiation and maintenance of the Wolffian ducts into male internal structures, including the epididymis, vas deferens, seminal vesicles, and ejaculatory ducts. Thus, the normal male internal phenotype establishes that testosterone production and androgen-receptor signaling are sufficient for Wolffian-duct development. In contrast, masculinization of the external genitalia, prostate, and urogenital sinus derivatives depends predominantly on dihydrotestosterone, which is formed locally from testosterone by 5α-reductase. Deficiency of this enzyme therefore leaves internal male differentiation intact but prevents typical virilization of the external genitalia. Testes may remain undescended because androgen-dependent genital development is disrupted. At puberty, rising testosterone concentrations can cause variable virilization despite impaired dihydrotestosterone formation. This developmental distinction between testosterone-dependent Wolffian structures and dihydrotestosterone-dependent external genital development is a core feature of 5α-reductase deficiency. See StatPearls: 5-Alpha-Reductase Deficiency and Merck Manual: Ambiguous Genitalia.

QUESTION NO: 63

Which of the following matches the definition: The maximum volume of air that can be exhaled after taking the deepest breath possible?

A. Expiratory reserve volume

B. Inspiratory capacity

C. Inspiratory reserve volume

D. Vital capacity

ANSWER: D

Explanation:

Vital capacity is the maximum volume of air a person can voluntarily exhale after first inhaling as deeply as possible. It represents the full usable volume moved between maximal inspiration and maximal expiration. Physiologically, vital capacity equals the sum of inspiratory reserve volume, tidal volume, and expiratory reserve volume. This measurement is commonly obtained with spirometry and is useful for describing overall ventilatory capacity. In a healthy person, vital capacity varies with factors such as height, sex, age, and physical conditioning. The definition in the question specifically begins at the point of deepest possible inhalation and asks for the greatest possible subsequent exhalation, which is precisely the maneuver used to determine vital capacity. This differs from individual reserve volumes, which describe only portions of the air available beyond a normal tidal breath, rather than the total volume exhaled from maximal inspiration. For further review of lung volumes and capacities, see the NCBI StatPearls overview of physiology and lung volumes and the MSD Manual discussion of pulmonary function testing.

QUESTION NO: 64

Which of the following is the antidote for the toxin Lead?

A. Naloxone

B. Nitrite

C. CaEDTA

D. Dialysis

ANSWER: C

Explanation:

CaEDTA is correct because calcium disodium ethylenediaminetetraacetate (calcium disodium EDTA; $CaNa_2EDTA$) is a chelating agent used to enhance elimination of lead from the body. Its EDTA component binds lead in the circulation and tissues, forming a water-soluble complex that is excreted in urine. Giving the calcium-containing formulation is important: it provides calcium already bound to EDTA and thereby avoids the marked hypocalcemia that can occur if disodium EDTA is used instead. In clinically significant lead poisoning, chelation choice is guided by blood lead concentration, symptoms, and patient factors. Calcium disodium EDTA is particularly important in severe poisoning and may be used with dimercaprol for lead encephalopathy; it is administered parenterally and requires monitoring for renal toxicity and adequate urine output. Thus, when asked for the classic antidotal chelator for lead among these choices, CaEDTA identifies the appropriate treatment. The ATSDR clinical guidance describes calcium disodium EDTA as an FDA-approved chelator for lead poisoning, and the FDA label provides its indication and safety information. See the ATSDR Lead Toxicity treatment guidance and the FDA label for calcium disodium EDTA.