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NABP FPGEE

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Topic Break Down

Topic	No. of Questions
Topic 1, Basic Biomedical Sciences	115
Topic 2, Pharmaceutical Sciences	136
Topic 3, Social/Behavioral/Administrative Pharmacy Sciences	14
Topic 4, Clinical Sciences	255
Total	520

QUESTION NO: 1

An enzyme is:

A. an oligopeptide

B. a nucleic acid

C. unable to hydrolyse polysaccharides

D. a catalyst

E. changed chemically in a reaction

ANSWER: D

Explanation:

An enzyme is a catalyst: it increases the rate at which a chemical reaction reaches equilibrium without being consumed by the reaction. Most enzymes are proteins with specific three-dimensional structures that create active sites, where substrates bind and are converted to products. By binding substrates and stabilizing the transition state, an enzyme lowers the activation energy required for the reaction to proceed. This allows essential biochemical reactions to occur rapidly under physiological conditions, including digestion, energy production, biosynthesis, and drug metabolism.

Enzymes are not permanently chemically changed or used up during the reactions they catalyze; after product release, they can generally participate in additional catalytic cycles. Their activity can be influenced by factors such as temperature, pH, substrate concentration, cofactors, and inhibitors. Although a small number of catalytic RNA molecules exist, the fundamental definition tested here is based on function: an enzyme acts as a biological catalyst. The International Union of Biochemistry and Molecular Biology describes enzymes as catalysts of biochemical reactions, and a concise overview of enzyme catalysis is available from NCBI Bookshelf and the IUBMB Enzyme Nomenclature.

QUESTION NO: 2

Condition characterized by a reversible form of airflow obstruction is known as:

A. Aneurism

B. Emphesema

C. Embolism

D. Cirrhose

E. Jaundice

F. Asthma

ANSWER: F

Explanation:

Asthma is the condition characterized by variable, usually reversible airflow obstruction. It is a chronic inflammatory disease of the airways in which airway smooth-muscle constriction, mucosal edema, and mucus production can narrow the airways. The degree of obstruction often changes over time and may improve spontaneously or after treatment with a bronchodilator, such as an inhaled short-acting beta$_2$-agonist. This variability and reversibility are central features used in asthma diagnosis.

Objective testing commonly demonstrates an increase in forced expiratory volume in 1 second (FEV_1) after bronchodilator administration. In adults, an increase in FEV_1 of more than 200 mL and more than 12% from baseline supports variable expiratory airflow limitation. Symptoms also tend to vary, with episodic wheeze, shortness of breath, chest tightness, and cough occurring particularly at night, with exercise, or after exposure to triggers. The Global Initiative for Asthma describes asthma as having variable expiratory airflow limitation, and the Merck Manual likewise identifies variable and reversible airflow obstruction as characteristic of asthma. See the GINA reports and the Merck Manual overview of asthma.

QUESTION NO: 3

The only antibiotic available as chew able tablet includes:

A. Penicillin

B. Amoxicillin

C. Ampicillin

D. Erythromycin

E. Clarithromycin

ANSWER: B

Explanation:

Amoxicillin is correct because it has long been supplied in pediatric-friendly chewable-tablet formulations, including 125 mg and 250 mg strengths. Chewable tablets provide an alternative for patients, particularly children, who can chew a solid dosage form but may have difficulty swallowing conventional capsules or tablets. The product should be chewed thoroughly before swallowing; it may also be swallowed whole when appropriate. Amoxicillin is an aminopenicillin antibacterial used for infections caused by susceptible organisms, and dosage selection depends on the infection, patient age and weight, renal function, and applicable treatment guidance. The availability of a chewable formulation is a dosage-form characteristic and does not change the need to use the drug only when an antibacterial is clinically indicated. Current prescribing information for amoxicillin identifies chewable tablets among its oral dosage forms and provides administration and dosing information. See the FDA labeling resource at FDA amoxicillin prescribing information and the NIH DailyMed drug-label database at DailyMed: amoxicillin chewable tablets.

QUESTION NO: 4

Which of the following agents or combinations may be appropriated to treat severe intermittent asthma?

A. High doses of inhaled corticosteroid

B. Long acting β2-agonists

C. Short acting β2-agonists

D. Oral corticosteroids

E. Combination of all the above medication

ANSWER: C

Explanation:

Short acting β2-agonists are appropriate because they provide rapid bronchodilation for the prompt relief of episodic bronchospasm and asthma symptoms. In traditional stepwise asthma-management frameworks, intermittent asthma is managed with a quick-relief bronchodilator used as needed, rather than scheduled daily controller therapy. Agents such as albuterol act within minutes to relax bronchial smooth muscle, improving acute symptoms such as wheeze, chest tightness, cough, and shortness of breath. This makes a short acting β2-agonist the appropriate selection when symptoms occur intermittently and immediate symptom relief is needed. Excessive reliance on a reliever inhaler is clinically important because it signals inadequate asthma control and should prompt reassessment of the patient's regimen, inhaler technique, adherence, triggers, and need for anti-inflammatory controller treatment. Current asthma strategies also emphasize that patients should receive inhaled corticosteroid-containing treatment to reduce exacerbation risk; however, among the listed choices, the short acting β2-agonist is the medication specifically used for rapid, as-needed relief of intermittent symptoms. See the NHLBI Asthma Care Quick Reference and the Global Initiative for Asthma strategy report.

QUESTION NO: 5

Which of the following is the predominant intracellular element?

A. Sodium

B. Magnesium

C. Potassium

D. Calcium

E. Phosphorous

ANSWER: C

Explanation:

Potassium is the predominant intracellular electrolyte and the principal cation within body cells. Approximately 98% of total body potassium is located intracellularly, where its concentration is much higher than in extracellular fluid. This distribution is actively maintained by the sodium-potassium ATPase pump, which transports potassium into cells while moving sodium out. The resulting potassium gradient is essential for establishing the resting membrane potential of excitable tissues, including skeletal muscle, cardiac muscle, and neurons. Changes in serum potassium can therefore significantly affect neuromuscular function and cardiac electrical activity, even though serum potassium represents only a very small fraction of total body potassium. In pharmacy practice, this physiology is important when evaluating electrolyte laboratory results, recognizing risks from diuretics or renin-angiotensin-aldosterone system inhibitors, and monitoring potassium replacement therapy. Standard physiology references identify potassium as the major intracellular cation and sodium as the major extracellular cation. See the NCBI Bookshelf overview of potassium and the OpenStax discussion of electrolyte balance.

QUESTION NO: 6

Which of the following agents may cause pulmonary toxicity?

I Amiodarone

II Bleomycin

III Ramipril

A. I only

B. III only

C. I and II only

D. II and III only

E. I, II and III

ANSWER: C

Explanation:

I and II only is correct because both amiodarone and bleomycin are well-established causes of clinically significant drug-induced pulmonary toxicity. Amiodarone can accumulate extensively in lung tissue because of its pharmacologic properties and long half-life. Its pulmonary adverse effects include interstitial pneumonitis, organizing pneumonia, acute respiratory distress syndrome, and pulmonary fibrosis. Symptoms may include progressive dyspnea, nonproductive cough, fever, and new infiltrates on chest imaging; prompt recognition and discontinuation of the drug are important when toxicity is suspected.

Bleomycin is also classically associated with dose-related pulmonary injury. It may produce pneumonitis that can progress to irreversible pulmonary fibrosis. Risk rises with increasing cumulative exposure and may be influenced by factors such as older age, renal impairment, concomitant thoracic radiation, and high inspired oxygen concentrations. Pulmonary monitoring and assessment of new respiratory symptoms are therefore important during bleomycin treatment. These toxicities are emphasized in the prescribing information for both drugs. See the amiodarone prescribing information and the bleomycin prescribing information.

QUESTION NO: 7

Degradation of hemoglobin in mammal involves:

A. Formation of bile pigments

B. Formation of urobilinogen

C. Formation of biliverdin

D. Non-oxidative cleavage of the porphyrin ring

E. All are correct

F. Formation of bile pigments, biliverdin, and urobilinogen

ANSWER: F

Explanation:

“Formation of bile pigments, biliverdin, and urobilinogen” is correct because hemoglobin catabolism proceeds through each of these products in a connected physiologic pathway. Senescent erythrocytes are taken up primarily by macrophages of the reticuloendothelial system. Heme oxygenase then catalyzes oxidative opening of the heme porphyrin ring, releasing iron and carbon monoxide and producing biliverdin. Biliverdin reductase converts biliverdin to unconjugated bilirubin, a principal bile pigment. After hepatic uptake and conjugation, bilirubin is excreted in bile into the intestine. Intestinal bacteria metabolize it to urobilinogen. Some urobilinogen is reabsorbed and ultimately excreted in urine as urobilin, while most is converted to stercobilin and eliminated in feces. Thus, biliverdin formation, bile-pigment production, and urobilinogen formation are all involved in normal mammalian hemoglobin degradation. The key mechanistic feature is oxidative, rather than nonoxidative, cleavage of the porphyrin ring. See NCBI Bookshelf: Bilirubin and NCBI Bookshelf: Heme Degradation.

QUESTION NO: 8

Mechanism of action of flunisolide is:

A. Inhibits DNA gyrase

B. Inhibits phosphodiesterase

C. Inhibits phospholipase A2

D. beta-2 agonist

E. Competitively inhibits H2 receptors

ANSWER: C

Explanation:

Inhibits phospholipase A2 is correct. Flunisolide is a synthetic glucocorticoid used primarily for its local anti-inflammatory effects in the airways and nasal mucosa. After entering cells, it binds intracellular glucocorticoid receptors; the resulting receptor–drug complex regulates gene transcription. One important consequence is increased production of anti-inflammatory proteins, including lipocortin (also called annexin A1), which inhibits phospholipase A2. Phospholipase A2 normally releases arachidonic acid from membrane phospholipids. By reducing this initial substrate-release step, flunisolide decreases formation of arachidonic-acid-derived inflammatory mediators, including prostaglandins and leukotrienes. It also suppresses inflammatory-cell activity and the expression of numerous proinflammatory mediators, contributing to reduced mucosal edema, airway inflammation, and allergic symptoms. Thus, inhibition of phospholipase A2 is the best answer among the listed mechanisms, although it occurs indirectly through glucocorticoid receptor-mediated changes in protein synthesis rather than through direct enzymatic blockade. The product labeling identifies flunisolide as a corticosteroid with potent anti-inflammatory activity, consistent with this mechanism. See the DailyMed flunisolide prescribing information and NCBI Bookshelf review of corticosteroids.

QUESTION NO: 9

The organophosphates commonly found in insecticides are thought to act by which of the following mechanisms?

A. Combining with acetylcholine

B. Potentiating the action of acetylcholinesterase

C. Forming a very stable complex with acetylcholinesterase

D. Reacting at the cholinergic receptor

E. Preventing the release of acetylcholine from the nerve ending

ANSWER: C

Explanation:

Organophosphates act by forming a very stable complex with acetylcholinesterase. These compounds phosphorylate the active-site serine residue of acetylcholinesterase, the enzyme responsible for rapidly hydrolyzing acetylcholine in synaptic junctions. Inhibition prevents normal acetylcholine breakdown, so acetylcholine accumulates and produces excessive stimulation of muscarinic, nicotinic, and central nervous system cholinergic pathways. This mechanism accounts for the characteristic cholinergic toxicity associated with organophosphate insecticide exposure.

The enzyme-inhibitor complex is initially potentially reversible with prompt treatment using an oxime such as pralidoxime. However, it can undergo “aging,” a chemical dealkylation process that makes the phosphorylated enzyme bond especially stable and effectively irreversible. Consequently, acetylcholinesterase activity may not recover until new enzyme is synthesized. This stable enzymatic complex, rather than a direct effect on cholinergic receptors or acetylcholine release, is the defining pharmacologic mechanism. For further detail, see StatPearls: Organophosphate Toxicity and the CDC/NIOSH organophosphate emergency response information.

QUESTION NO: 10

MT, a pharmacy technician, has worked in a community pharmacy for 5 years. He is well-trained and welcomes new learning opportunities. His manager, now wanting to delegate a new technical project to him, should:

I discuss suggested steps to accomplish the project.

II negotiate a time schedule for completion of the project.

III provide support and follow-up as required.

A. I only

B. III only

C. I and II only

D. II and III only

E. I, II and III

ANSWER: E

Explanation:

I, II and III is correct because effective delegation involves clearly defining the work, agreeing on expected results and timing, and maintaining appropriate managerial support. Even when a pharmacy technician is experienced, competent, and enthusiastic about learning, a new project may involve unfamiliar processes, competing workflow demands, or decisions that require direction from the pharmacist or manager. Discussing suggested steps establishes a shared understanding of the project scope, intended outcomes, available resources, and any boundaries of the technician’s authority. Negotiating a completion schedule creates a realistic, mutually understood deadline that accounts for the technician’s routine duties and the pharmacy’s operational priorities. Providing support and follow-up does not mean micromanaging; it means remaining

available for questions, monitoring progress at appropriate intervals, addressing barriers, and confirming that the completed work meets expectations. These practices help the manager retain accountability while giving the technician meaningful responsibility and an opportunity to develop professionally. NABP recognizes pharmacy technicians as important members of the pharmacy team working under pharmacist supervision; delegation should therefore include clear communication and oversight appropriate to the task. See the NABP examination programs and the ASHP Pharmacy Technician Resources.

QUESTION NO: 11

Mechanism of action of neomycin is:

A. Inhibits dihydropteroate synthetase

B. Blocks K channels

C. Blocks Na channels

D. Binds to 30S subunit of bacterial ribosome

E. Inhibits mycolic acid synthesis

ANSWER: D

Explanation:

Neomycin binds to the bacterial 30S ribosomal subunit. It is an aminoglycoside antibiotic, a class whose antibacterial effect results from disruption of bacterial protein synthesis. After entering susceptible bacteria through an oxygen-dependent transport process, neomycin binds tightly and essentially irreversibly to 16S ribosomal RNA and associated proteins in the 30S subunit. This interferes with formation of the initiation complex and causes misreading of messenger RNA during translation. Consequently, bacteria produce abnormal, nonfunctional proteins; aminoglycosides are therefore bactericidal rather than merely bacteriostatic. Neomycin has limited systemic absorption after oral or topical administration, so it is commonly used for local intestinal decontamination, such as preoperative bowel preparation in combination with other agents, and in selected topical products. The molecular target—bacterial ribosomal 30S subunit—is the key mechanism tested in this question. The FDA-approved prescribing information identifies neomycin as an aminoglycoside and describes its inhibition of bacterial protein synthesis through ribosomal binding. Additional pharmacology details for aminoglycosides are available in StatPearls: Aminoglycosides and the DailyMed neomycin sulfate label.

QUESTION NO: 12

The pharmacist fills a prescription for sumatriptan 100 mg tablets for a migraine patient.

Appropriate information to provide to the patient includes which of the following?

A. If the sumatriptan does not relieve the headache within four hours, ergotamine may be used.

B. If no relief is achieved in two hours, sumatriptan may be repeated.

C. If the headache is relieved but another headache occurs eight hours later, sumatriptan may be used for the second headache.

D. The maximum dosage in any 24 hour period is six tablets.

E. If relief is not achieved, no other medication can be used for 24 hours.

ANSWER: B

Explanation:

“If no relief is achieved in two hours, sumatriptan may be repeated.” is appropriate counseling because oral sumatriptan may be taken as a second dose when the initial dose does not adequately relieve the migraine. The repeat dose must be separated from the first dose by at least 2 hours. For a patient using 100 mg tablets, the labeled maximum oral dose is 200 mg in a 24-hour period, so this generally means no more than two 100 mg tablets during that interval. Counseling should

also clarify that sumatriptan is intended for acute treatment of a migraine attack rather than prevention. The patient should follow the prescribed directions and seek clinical advice if migraine attacks become frequent, change in character, or do not respond as expected. In addition, use with ergot-containing migraine products requires appropriate separation because of additive vasospastic risk; patient-specific counseling should account for all current medications and cardiovascular risk factors. The dosing interval and daily maximum are specified in the FDA-approved prescribing information for oral sumatriptan. See the DailyMed sumatriptan prescribing information and the FDA Imitrex tablets label.

QUESTION NO: 13

Which of the following vitamins has tetrahydrofolate coenzyme activity based on its pteridine ring?

A. Thiamine

B. Vitamin D

C. Vitamin K

D. Folic acid

E. Pyridoxine

ANSWER: D

Explanation:

Folic acid is correct because it is the synthetic, oxidized form of folate (vitamin B9), whose chemical structure includes a pteridine ring linked to para-aminobenzoic acid and glutamate. After absorption and cellular metabolism, folic acid is reduced to tetrahydrofolate (THF) and related reduced folate cofactors. THF is the biologically active coenzyme form that accepts and transfers one-carbon units at different oxidation states. These one-carbon transfer reactions are essential for purine and thymidylate synthesis, amino acid metabolism, and normal DNA synthesis and cell division.

The pteridine-containing folate structure is central to this chemistry: reduction of the pteridine ring produces tetrahydrofolate, enabling the coenzyme to carry one-carbon groups. In nutrition and pharmacy contexts, “folic acid” commonly identifies the vitamin precursor supplied in fortified foods and many supplements, while “folate” includes the naturally occurring forms in foods and the metabolically active reduced derivatives. Thus, the vitamin associated with tetrahydrofolate coenzyme activity is folic acid. The NIH Office of Dietary Supplements describes folate’s role in one-carbon metabolism and identifies folic acid as a form of the vitamin: NIH Folate Fact Sheet for Health Professionals. Chemical information on folic acid is also available from PubChem.

QUESTION NO: 14

The formation of glucose from non-carbohydrates sources such as lactate and Kreb’s cycle metabolites is an anabolic process called:

A. Glycogenolysis

B. Glycolysis

C. Glycogenesis

D. Gluconeogenesis

E. None of the above

ANSWER: D

Explanation:

Gluconeogenesis is the anabolic metabolic pathway that synthesizes glucose from noncarbohydrate precursors. Important substrates include lactate, which is converted to pyruvate in the Cori cycle; glycerol derived from triacylglycerol breakdown; and glucogenic amino acids, whose carbon skeletons can enter central metabolism as pyruvate or citric acid-cycle

intermediates. The pathway occurs mainly in the liver, with the kidney cortex also contributing, especially during prolonged fasting. Gluconeogenesis is essential for maintaining blood glucose when dietary glucose is unavailable and hepatic glycogen stores have declined. It supplies glucose to tissues with an important glucose requirement, including erythrocytes and parts of the central nervous system. Because several glycolytic reactions are effectively irreversible, gluconeogenesis uses specific bypass enzymes, including pyruvate carboxylase, phosphoenolpyruvate carboxykinase, fructose-1,6-bisphosphatase, and glucose-6-phosphatase. The process requires energy, consistent with its anabolic role. A detailed overview of gluconeogenesis and its physiological regulation is available in StatPearls: Biochemistry, Gluconeogenesis and in the OpenStax Biology discussion of the citric acid cycle and metabolic intermediates.

QUESTION NO: 15

Structure-activity studies of cholinergic drugs indicate that:

I maximum activity is achieved when the quaternary nitrogen is three carbons removed from the ester oxo group.

II structures which display a quasi-ring conformation exhibit muscarinic activity.

III one methyl group of the trimethylammonium group can be replaced by an ethyl group with only slight loss of activity.

A. I only

B. III only

C. I and II only

D. II and III only

E. I, II and III

ANSWER: D

Explanation:

II and III only is correct. Structure–activity relationships for acetylcholine-like direct cholinergic agonists recognize that muscarinic receptor activation is favored by a folded, or quasi-ring, conformation. This three-dimensional arrangement appropriately positions the cationic quaternary ammonium center relative to the ester-containing portion of the molecule for productive muscarinic receptor binding. The conformational preference is therefore an important determinant of muscarinic versus nicotinic activity in this drug class.

The quaternary ammonium substituents also tolerate limited enlargement. Replacing one methyl substituent on the trimethylammonium group with an ethyl group produces only a relatively small decrease in agonist activity. This principle is illustrated by methacholine, an acetylcholine analog containing a β-methyl substituent that retains substantial muscarinic agonist activity and has increased resistance to hydrolysis by acetylcholinesterase. These relationships are useful in understanding why small, carefully placed structural modifications can preserve cholinergic activity while changing receptor selectivity, metabolism, or duration of action. See the discussion of direct-acting cholinergic agonists in StatPearls: Cholinergic Medications and the structural and pharmacologic information for methacholine at PubChem.

QUESTION NO: 16

Example of drug used in prophylaxis of malaria includes:

A. Cloroquine

B. Quinine

C. Primaquin

D. Quinine

E. Quinidine

ANSWER: A

Explanation:

Cloroquine (intended spelling: chloroquine) is an antimalarial that can be used for malaria chemoprophylaxis when travel is limited to areas where *Plasmodium* parasites remain chloroquine-sensitive. It prevents clinical malaria by suppressing blood-stage parasites after exposure from mosquito bites. Its suitability is determined by the malaria species and, especially, the geographic pattern of antimalarial resistance at the traveler's destination. Chloroquine prophylaxis is therefore a destination-specific choice rather than a universal regimen. When indicated, it is generally started before travel, continued weekly while in the malaria-risk area, and continued for a period after leaving the area to cover parasites that may emerge into the bloodstream after exposure. The Centers for Disease Control and Prevention identifies chloroquine as a recommended preventive medicine for destinations with chloroquine-sensitive malaria and provides current country-specific recommendations in its travelers' guidance. Pharmacists should verify the most current destination information, patient-specific contraindications, potential interactions, and the appropriate dosing schedule before recommending prophylaxis. See the CDC's Malaria Information and Prophylaxis by Country and Choosing a Drug to Prevent Malaria.

QUESTION NO: 17

Allopurinol, xanthine oxidase inhibitor, used in gout treatment may produce all the following side effects, EXCEPT:

A. Increase in protrombin time

B. Hypersensitivity rashes

C. GIT intolerance

D. Peripheral neuritis

E. Necrotizing vasculitis

ANSWER: A

Explanation:

"Increase in protrombin time" is the exception because it is not an intrinsic adverse effect expected from allopurinol therapy. A prolonged prothrombin time may occur when allopurinol is used with coumarin-type oral anticoagulants, because allopurinol can potentiate their anticoagulant effect in some patients. In that setting, the laboratory change reflects a clinically important drug interaction and may require closer monitoring and adjustment of the anticoagulant dose; it is not classified as a direct allopurinol toxicity.

Allopurinol reduces uric acid formation by inhibiting xanthine oxidase and is used for long-term management of hyperuricemia in gout. Its labeled safety information emphasizes the need to discontinue therapy promptly if a rash or other signs of hypersensitivity occur, reflecting the potential for severe allopurinol hypersensitivity reactions. Authoritative prescribing information also specifically advises reassessing prothrombin time in patients receiving dicumarol when allopurinol is added, supporting that altered coagulation testing is tied to concomitant anticoagulant treatment. See the DailyMed allopurinol prescribing information and NCBI Bookshelf review of allopurinol.

QUESTION NO: 18

Which is responsible for N in vivo?

A. Arginine

B. Tryptophan

C. Guanine

D. Thiamine

E. Leucine

ANSWER: A

Explanation:

Arginine is correct because the question is most plausibly referring to nitric oxide production in vivo. L-arginine is the physiological substrate for nitric oxide synthase enzymes. These enzymes oxidize L-arginine in a reaction requiring oxygen and reducing equivalents, producing nitric oxide together with L-citrulline. Nitric oxide is an important endogenous signaling molecule: endothelial nitric oxide synthase supports vascular tone and blood-flow regulation, neuronal nitric oxide synthase participates in neurotransmission, and inducible nitric oxide synthase contributes to host-defense and inflammatory responses. Thus, when a pharmacology or biochemistry question asks which compound is responsible for nitric oxide formation in the body, the expected precursor is arginine. The relevant substrate is specifically the naturally occurring L-isomer, although examination options commonly state simply “arginine.” This biochemical relationship is also clinically relevant because altered L-arginine availability or nitric oxide synthase activity can affect endothelial function and cardiovascular physiology. PubChem identifies L-arginine as an endogenous amino acid and provides its biochemical data at PubChem: L-Arginine. A detailed review of nitric oxide synthase biology and its conversion of L-arginine to nitric oxide and L-citrulline is available through the National Library of Medicine.

QUESTION NO: 19

Side effect of fluvastatin is:

A. Seizures

B. Skin rash

C. Nausea

D. Urinary tract complications

E. Myalgia

ANSWER: E

Explanation:

Myalgia is the best answer because muscle symptoms are a well-recognized adverse effect of fluvastatin and other HMG-CoA reductase inhibitors (statins). Statins reduce cholesterol synthesis by inhibiting HMG-CoA reductase, but in some patients they can also cause skeletal-muscle adverse effects that range from muscle aches, tenderness, cramps, or weakness (myalgia) to rare, severe myopathy and rhabdomyolysis. Muscle symptoms may occur with or without substantial creatine kinase elevation. Patients should be instructed to promptly report unexplained muscle pain, tenderness, or weakness, especially if it is associated with malaise or fever. Clinically important risk factors for statin-associated myopathy include higher statin exposure, interacting drugs that increase statin concentrations, older age, renal impairment, hypothyroidism, and certain genetic predispositions. Fluvastatin prescribing information specifically lists myopathy and rhabdomyolysis as warnings and advises discontinuation if markedly elevated creatine kinase occurs or if myopathy is diagnosed or suspected. Therefore, myalgia is the characteristic adverse effect tested in this question. See the DailyMed fluvastatin label and the NCBI StatPearls review of statin medications.

QUESTION NO: 20

Half-life of tolbutamide is:

A. 12-24 hours

B. 3 hours

C. 6-12 hours

D. 10 hours

E. 8 hours

F. 4.5–6.5 hours

ANSWER: F

Explanation:

The correct answer is **4.5–6.5 hours**. Tolbutamide is a first-generation sulfonylurea with a relatively short plasma elimination half-life. After oral administration, it is extensively metabolized in the liver, primarily to inactive metabolites that are eliminated in urine. Its reported plasma half-life is approximately 4.5 to 6.5 hours in individuals with usual hepatic and renal function. This pharmacokinetic property supports its classification as a shorter-acting first-generation sulfonylurea, although its glucose-lowering effect can persist beyond the measured plasma half-life because insulin release and pharmacodynamic effects continue after drug concentrations begin to decline.

It is important to distinguish elimination half-life from duration of action. A range such as 6–12 hours is commonly associated with the clinical duration of hypoglycemic effect, not the usual elimination half-life. Factors such as advanced age, hepatic impairment, renal impairment, drug interactions, and reduced caloric intake may increase susceptibility to prolonged hypoglycemia, even with a drug that normally has a relatively short half-life. Drug information and pharmacokinetic data for tolbutamide are available through DailyMed and PubChem.

QUESTION NO: 21

Contraindication of indacrinone is:

A. Pregnancy

B. Hyperlipidemia

C. Diabetes

D. Diabetes mellitus

E. Hearing impairment

ANSWER: E

Explanation:

Hearing impairment is the correct answer. Indacrinone is an ethacrynic-acid–related loop diuretic. A clinically important toxicity of this class is ototoxicity, which can manifest as tinnitus, vertigo, or sensorineural hearing loss. This effect is particularly relevant in a patient who already has impaired hearing, because further auditory injury may be difficult to detect promptly and may be irreversible. The risk is associated with high systemic exposure, rapid parenteral administration where applicable, renal dysfunction, and concomitant use of other ototoxic medicines. Therefore, pre-existing hearing impairment is treated as a contraindication or a strong reason to avoid indacrinone in examination-oriented pharmacology references. The pharmacologic basis is the loop-diuretic effect on ion transport in the inner ear, in addition to the intended inhibition of salt reabsorption in the thick ascending limb of the loop of Henle. Clinical references on loop diuretics emphasize ototoxicity as a significant class adverse effect and recommend careful auditory-risk assessment. See the NCBI Bookshelf review of loop diuretics and the PubChem indacrinone record.

QUESTION NO: 22

Protein synthesis inhibitors may bind to:

A. 30s ribosomal subunit only

B. 50s ribosomal subunit only

C. 70s ribosomal subunit only

D. 30s and 50s and 70s ribosomal subunits

E. 30s and 70s ribosomal subunits

F. 30S and 50S ribosomal subunits

ANSWER: F

Explanation:

Protein synthesis inhibitors may bind to the bacterial 30S or 50S ribosomal subunits. Bacterial ribosomes are composed of a small 30S subunit and a large 50S subunit; together, these form the functional 70S ribosome. Different antimicrobial classes act at distinct sites within these subunits to interrupt translation. For example, tetracyclines bind the 30S subunit and prevent aminoacyl-tRNA attachment, while macrolides bind the 50S subunit and interfere with peptide-chain elongation. Thus, the appropriate inclusive answer is “30S and 50S ribosomal subunits.” The term 70S describes the complete assembled bacterial ribosome rather than an individual ribosomal subunit. This distinction is clinically useful because the selective targeting of bacterial ribosomes underlies the antibacterial activity of these agents. The National Center for Biotechnology Information describes bacterial ribosomes as 70S particles consisting of 30S and 50S subunits and reviews antimicrobial interference with translation in its Antimicrobial Pharmacology reference. Drug-specific mechanisms are also available in FDA labeling through the DailyMed drug-label database.

QUESTION NO: 23

Which of the following statement is/are correct regarding Gray Babies syndrome?

I- Occurs in premature and new born infants when choramphenicol is administrated during the first few days of life

II- Due to inability of the infant to metabolize the drug because of glucoronyl transferase deficiency

III- Glucoronyl transferase is required to detoxify cloramphenicol

A. I only

B. III only

C. I and II only

D. II and III only

E. All are correct

ANSWER: E

Explanation:

All are correct is the correct selection. Gray baby syndrome is a serious toxicity associated with chloramphenicol exposure in neonates, particularly premature infants and infants in the first days of life. At this age, hepatic drug-metabolizing capacity is immature. Chloramphenicol is normally inactivated primarily through hepatic glucuronidation, a conjugation process involving UDP-glucuronosyltransferase (often called glucuronyl transferase in this context). The resulting glucuronide metabolite is substantially less active and can be eliminated from the body.

Because newborns have low glucuronidation capacity, chloramphenicol may accumulate rapidly when conventional doses are used. Immature renal excretion can further contribute to accumulation. High circulating concentrations can lead to the characteristic syndrome, including abdominal distention, vomiting, cyanosis, vasomotor collapse, and a gray or ashen skin color. Therefore, the age-related occurrence, impaired metabolism from deficient glucuronyl-transferase activity, and the role of glucuronidation in chloramphenicol detoxification are all clinically and pharmacologically accurate. Chloramphenicol product labeling specifically warns of this potentially fatal neonatal toxicity and emphasizes appropriate dosing and monitoring. See DailyMed chloramphenicol prescribing information and the NIH LiverTox monograph on chloramphenicol.

QUESTION NO: 24

Mechanism of action of carbenicillin is:

A. Inhibits transpeptidase

B. Blocks Na channels

C. Activates heparin cofactor II which inhibits thrombin (at high concentrations)

D. Blocks sustained repetitive neuronal firing

E. GM-CSF

ANSWER: A

Explanation:

Inhibits transpeptidase is correct. Carbenicillin is a carboxypenicillin, a semisynthetic β-lactam antibiotic. Like other penicillins, it binds to penicillin-binding proteins, particularly transpeptidase enzymes involved in the final stages of bacterial cell-wall synthesis. Transpeptidases catalyze cross-linking of peptidoglycan strands, a process that provides the cell wall with the rigidity needed to resist osmotic pressure. By acylating and inactivating these enzymes, carbenicillin prevents peptidoglycan cross-linking and weakens the bacterial cell wall. This promotes cell-wall disruption and bacterial lysis, particularly in actively growing, susceptible organisms. Its antibacterial effect is therefore bactericidal and depends on maintaining drug concentrations for sufficient time relative to the organism's minimum inhibitory concentration. Carbenicillin historically had activity against certain Gram-negative organisms, including *Pseudomonas aeruginosa*, although its clinical use has become limited and susceptibility should guide therapy. The FDA label identifies carbenicillin as a penicillin antibacterial agent, and standard pharmacology references describe β-lactam activity as inhibition of penicillin-binding proteins responsible for peptidoglycan cross-linking. See the DailyMed carbenicillin label and NCBI StatPearls review of penicillin.

QUESTION NO: 25

Antibiotics that often cause colitis-acute inflammation of colon as side effect include:

I- Broad-spectrum penicillins

II- Cephalosporins

III- Clindamycin

A. I only

B. III only

C. I and II only

D. II and III only

E. All are correct

ANSWER: E

Explanation:

All are correct. Broad-spectrum penicillins, cephalosporins, and clindamycin can disrupt the normal protective intestinal microbiota. This loss of colonization resistance permits overgrowth of *Clostridioides difficile*, whose toxins can cause antibiotic-associated diarrhea and colitis, ranging from mild illness to severe pseudomembranous colitis. Clindamycin is a classic, strongly recognized precipitating antimicrobial, but the risk is not limited to that drug. Broad-spectrum beta-lactam antibiotics, including broad-spectrum penicillins and cephalosporins, are also important exposures because of their substantial effects on gut flora. The likelihood of antibiotic-associated colitis varies with the specific agent, duration of therapy, cumulative antimicrobial exposure, patient age, hospitalization, and other host factors, but each listed antibiotic class is clinically associated with this adverse effect. The Centers for Disease Control and Prevention identifies antibiotic exposure as the principal risk factor for *C. difficile* infection and emphasizes that antibiotics disturb normal gut flora. The Infectious Diseases Society of America and Society for Healthcare Epidemiology of America guideline likewise recognizes exposure to high-risk antibiotics, including clindamycin, cephalosporins, and broad-spectrum penicillins, in the prevention and management context. See the CDC C. difficile risk-factor information and the IDSA/SHEA clinical practice guideline.

QUESTION NO: 26

Which is the correct start dose of methothrexate in the treatment of rheumatoid arthritis?

A. 7.5mg once a week

B. 15mg once a week

C. 7.5mg daily

D. 15mg daily

E. 22mg once a week

ANSWER: A

Explanation:

For rheumatoid arthritis, methotrexate is initiated as a **once-weekly** regimen, and 7.5 mg once weekly is the classic labeled starting dose. The dose may subsequently be increased gradually according to clinical response, tolerability, blood-count monitoring, liver-function results, renal function, and concomitant medicines. Methotrexate has a narrow safety margin, and its anti-inflammatory effect in rheumatoid arthritis is achieved with low-dose intermittent administration rather than continuous daily dosing. Clear weekly dosing instructions are especially important because inadvertent daily administration of low-dose methotrexate can cause severe, potentially fatal toxicity. The U.S. prescribing information for methotrexate tablets describes 7.5 mg orally once weekly as the recommended starting dose for adult rheumatoid arthritis, with dose adjustment when appropriate. Contemporary rheumatology guidance also supports prompt optimization of methotrexate toward an effective weekly dose after initiation, individualized to the patient's response and tolerability. See the DailyMed methotrexate prescribing information and the 2021 American College of Rheumatology guideline for rheumatoid arthritis.

QUESTION NO: 27

Which of the following is the only disaccharide NOT synthesized during human metabolism?

A. Lactose

B. Maltase

C. Fructose

D. Glucose

E. Cellulose

F. Sucrose

ANSWER: F

Explanation:

Sucrose is the appropriate answer because it is a disaccharide composed of glucose and fructose that humans do not synthesize as part of normal human metabolism. It is primarily made by plants, where it serves as a major transport and storage carbohydrate, and humans obtain it from dietary sources such as sugar cane, sugar beets, fruits, and processed foods. Although human digestive enzymes can hydrolyze ingested sucrose into its monosaccharide components through intestinal sucrase activity, hydrolysis is digestion rather than synthesis. Human carbohydrate metabolism can synthesize and interconvert monosaccharides and can produce certain carbohydrate-containing compounds, but it lacks the biosynthetic pathway required to form sucrose. This distinction is important: the presence of an enzyme that digests a carbohydrate does not indicate that the body makes that carbohydrate. For background on sucrose's plant origin and its glucose-fructose composition, see Encyclopaedia Britannica: Sucrose. The role of sucrase in intestinal digestion of dietary sucrose is described by NCBI Bookshelf: Physiology, Carbohydrates.

QUESTION NO: 28

Indication of epinephrine is:

A. Post myocardial infarction

B. Asthma

C. Tremor

D. Trigeminal neuralgia

E. Neurosurgical anesthesia

ANSWER: B

Explanation:

Asthma is the best answer among the listed choices because epinephrine has potent beta-2 adrenergic activity that relaxes bronchial smooth muscle, producing rapid bronchodilation. Its alpha-adrenergic vasoconstrictor effect can also reduce mucosal edema in acute upper-airway reactions. Historically, epinephrine was used to treat acute bronchospasm associated with asthma, particularly when rapid parenteral bronchodilator action was needed. In current clinical practice, inhaled selective beta-2 agonists are generally preferred for routine asthma exacerbations because they provide targeted bronchodilation with fewer systemic effects. Nevertheless, epinephrine remains clinically important when bronchospasm occurs as part of anaphylaxis; it is the first-line treatment for anaphylaxis and addresses airway edema, bronchoconstriction, and hypotension simultaneously. Thus, in a single-best-answer pharmacology question asking for an indication of epinephrine from these choices, asthma is the applicable indication. The FDA prescribing information for epinephrine injection includes emergency treatment of allergic reactions, including bronchospasm, and the National Heart, Lung, and Blood Institute describes bronchodilator therapy as central to management of acute asthma symptoms. See FDA epinephrine injection prescribing information and NHLBI asthma treatment guidance.

QUESTION NO: 29

Intra-articular means:

A. In the joint space

B. In the tissues space

C. In the spinal fluid

D. In the spinal column

E. In the dermis

ANSWER: A

Explanation:

“In the joint space” is correct because *intra-articular* describes a route of administration or location within a joint cavity. The prefix *intra-* means “within,” and *articular* pertains to a joint. In clinical practice, an intra-articular injection delivers a medication directly into the synovial joint space, where it can produce a local therapeutic effect. Examples include corticosteroid injections used to reduce inflammation and pain in conditions such as osteoarthritis or inflammatory arthritis, as well as hyaluronic acid products used in selected patients with knee osteoarthritis. This route is distinct from administration into tissue surrounding a joint and from injection into the spinal canal or skin layers. Correct identification of route terminology is important in pharmacy because the route affects product selection, sterility requirements, administration technique, expected onset and distribution, and patient counseling. The National Cancer Institute’s Dictionary of Cancer Terms defines intra-articular as “within a joint,” and MedlinePlus describes joint injections as injections placed into a joint to treat pain and inflammation. See the National Cancer Institute definition of intra-articular and MedlinePlus information on joint injection.

QUESTION NO: 30

Pernicious anemia is:

A. due to dietary deficiency of vitamin B12.

B. prevented by oral administration of a vitamin B12 supplement.

C. treated by parenteral administration of folic acid.

D. treated by parenteral administration of vitamin B12.

E. caused by dietary iron deficiency.

ANSWER: D

Explanation:

Pernicious anemia is a megaloblastic anemia caused by vitamin B12 deficiency resulting from autoimmune destruction of gastric parietal cells and/or antibodies against intrinsic factor. Intrinsic factor is necessary for normal active absorption of vitamin B12 in the terminal ileum. Because this underlying absorption mechanism is impaired, vitamin B12 replacement is required, and parenteral administration has traditionally been the standard, reliable treatment. Intramuscular cyanocobalamin or hydroxocobalamin bypasses the gastrointestinal absorption defect and replenishes body stores, allowing normal DNA synthesis and effective red-cell production to resume.

Folic acid should not be used in place of vitamin B12 for this condition because it does not correct the underlying cobalamin deficiency. Treatment also requires ongoing replacement because the autoimmune loss of intrinsic-factor-mediated absorption generally persists. Clinical references describe parenteral vitamin B12 as appropriate therapy for pernicious anemia, particularly during initial repletion or when dependable absorption is needed. See the MSD Manual's guidance on vitamin B12 deficiency and the NIH Office of Dietary Supplements vitamin B12 fact sheet.

QUESTION NO: 31

The side effects of isoniazid are potentialized by deficiency of which vitamin?

A. Thiamine

B. Pyridoxine

C. Folic acid

D. Niacin

E. Cyanocobalamin

ANSWER: B

Explanation:

Pyridoxine is correct because isoniazid can interfere with vitamin B6 metabolism and reduce the availability of active pyridoxine. Vitamin B6 is an essential cofactor in neurotransmitter synthesis and other metabolic pathways; depletion during isoniazid therapy can therefore increase the risk of adverse effects, particularly peripheral neuropathy. Patients at increased risk of pyridoxine deficiency include those with malnutrition, diabetes, HIV infection, alcohol use disorder, chronic renal failure, pregnancy, seizure disorders, or preexisting neuropathy. For prevention, pyridoxine is commonly coadministered with isoniazid, with the dose tailored to the patient's risk and treatment context. The U.S. prescribing information for isoniazid specifically identifies pyridoxine deficiency as a potential cause of peripheral neuritis and notes that pyridoxine administration can prevent or treat this effect. This drug–nutrient relationship is clinically important in tuberculosis treatment because supplementation helps patients tolerate therapy while continuing an effective antitubercular regimen. See the DailyMed isoniazid prescribing information and the CDC guidance on treatment for tuberculosis disease.

QUESTION NO: 32

Carotene is the precursor of:

A. retinol

B. thiamine

C. calciferol

D. riboflavin

E. retinoic acid

ANSWER: A

Explanation:

Retinol is correct because dietary carotenoids—particularly β-carotene—serve as provitamin A compounds. After absorption, β-carotene can be enzymatically cleaved in the intestinal mucosa and other tissues to form retinal. Retinal is then reduced to retinol, the principal alcohol form of vitamin A. Retinol can be transported in blood bound to retinol-binding protein, stored mainly in the liver as retinyl esters, and converted as needed to biologically active vitamin A derivatives. This precursor relationship is clinically important because vitamin A supports vision, epithelial-cell differentiation, immune function, growth, and reproduction. The conversion of β-carotene to vitamin A is regulated and varies with dietary fat intake, nutritional status, food preparation, and the individual's genetic and physiologic factors. Therefore, carotene is accurately identified as a precursor of retinol (vitamin A). Authoritative nutrition references identify β-carotene and related provitamin A carotenoids as dietary sources that the body converts to vitamin A: NIH Office of Dietary Supplements: Vitamin A Fact Sheet for Health Professionals and NCBI Bookshelf: Vitamin A.

QUESTION NO: 33

All of the following are narrow spectrum protein synthesis inhibitors, EXCEPT:

A. Erythromycin

B. Azithromycin

C. Clarithromycin

D. Tetracycline

E. Clindamycin

ANSWER: D

Explanation:

Tetracycline is the exception because it is classically categorized as a broad-spectrum antibacterial protein-synthesis inhibitor. Tetracyclines enter susceptible bacterial cells and reversibly bind the 30S ribosomal subunit. This prevents aminoacyl-tRNA from attaching to the ribosomal acceptor site, thereby inhibiting addition of amino acids to the growing peptide chain. Their antibacterial activity extends across a relatively wide range of organisms, including many gram-positive and gram-negative bacteria, as well as atypical pathogens such as *Chlamydia*, *Mycoplasma*, and *Rickettsia*. This broad coverage is the key classification point tested by the question, regardless of current resistance patterns or clinical restrictions that may limit use against particular organisms. The Merck Manual describes tetracyclines as broad-spectrum antibiotics and summarizes their 30S-mediated mechanism of action. A detailed pharmacology review is also available through Merck Manual: Tetracyclines and NCBI Bookshelf: Tetracyclines. Therefore, tetracycline is appropriately identified as the drug that is not narrow spectrum.

QUESTION NO: 34

Enzymes that catalyze the transfer of one group to other group is called:

A. Oxidoreductase enzymes

B. Transferase enzymes

C. Hydrolyses enzymes

D. Isomerasis enzymes

E. Lyasis enzymes

ANSWER: B

Explanation:

Transferase enzymes is the correct answer. Transferases are classified as enzymes that catalyze the movement of a functional group from one molecule, called the donor, to another molecule, called the acceptor. Their general reaction pattern is donor–group + acceptor → donor + acceptor–group. Commonly transferred groups include phosphate, amino, methyl, glycosyl, and acyl groups. For example, kinases are transferases that transfer a phosphate group, usually from ATP, to a substrate; aminotransferases transfer amino groups between amino acids and keto acids. This activity is central to many biochemical pathways relevant to pharmacy, including drug metabolism, synthesis of endogenous compounds, and cellular signaling. In the Enzyme Commission system maintained under the International Union of Biochemistry and Molecular Biology nomenclature, transferases are assigned enzyme class 2 (EC 2). The class definition specifically identifies these enzymes as catalyzing the transfer of groups other than hydrogen between substrates. See the IUBMB Enzyme Nomenclature resource and the BRENDA EC 2 transferase classification for enzyme-class details.

QUESTION NO: 35

The metabolic degradation of hemoglobin takes place principally in:

A. RBCs

B. Reticuloendothelial system

C. Liver

D. Kidney

E. Pancreas

ANSWER: B

Explanation:

Reticuloendothelial system is correct because senescent erythrocytes are removed from the circulation primarily by macrophages of the mononuclear phagocyte system, historically called the reticuloendothelial system. This process occurs especially in the spleen, with additional important activity in hepatic Kupffer cells and bone-marrow macrophages. Within these macrophages, hemoglobin is separated into globin and heme. Globin is hydrolyzed to amino acids for reutilization. Heme is converted by heme oxygenase to biliverdin, releasing iron and carbon monoxide; biliverdin is then reduced to unconjugated bilirubin. The iron is conserved and transported or stored for future hemoglobin synthesis, whereas unconjugated bilirubin travels bound to albumin to the liver for conjugation and subsequent biliary excretion. Thus, although the liver has a major downstream role in bilirubin conjugation and excretion, the principal initial metabolic breakdown of hemoglobin occurs in reticuloendothelial macrophages. This distinction is central to understanding normal bilirubin production and the handling of iron following erythrocyte turnover. See NCBI Bookshelf: Physiology, Bilirubin and NCBI Bookshelf: Biochemistry of Hemoglobin.

QUESTION NO: 36

Dopamine is useful in the treatment of cardiogenic shock because it:

I selectively dilates renal and mesenteric vascular beds.

II does not induce peripheral vasoconstriction.

III decreases the force of myocardial contraction.

A. I only

B. III only

C. I and II only

D. II and III only

E. I, II and III

ANSWER: A

Explanation:

I only is correct. Dopamine has dose-dependent adrenergic and dopaminergic effects that can support a patient with cardiogenic shock when pharmacologic circulatory support is appropriate. At low infusion rates, dopamine predominantly activates dopaminergic receptors, particularly in renal and mesenteric vascular beds. This receptor activity produces vasodilation in those beds and can increase renal blood flow and urinary output. The selective vasodilatory effect described in the statement is therefore the relevant pharmacologic property.

As the infusion rate increases, dopamine also stimulates cardiac beta-1 receptors, increasing myocardial contractility and cardiac output. At still higher doses, alpha-adrenergic stimulation can increase systemic vascular resistance. Thus, its clinical effects must be anticipated from the dose used and the patient's hemodynamic status. The FDA prescribing information describes dopamine's low-dose dopaminergic vasodilatory activity in the renal and mesenteric circulation and its progressively greater beta-1 and alpha-adrenergic effects at higher infusion rates. See the DailyMed dopamine hydrochloride injection labeling and the NCBI StatPearls review of dopamine.

QUESTION NO: 37

In the treatment of rheumatoid arthritis the salicylates:

A. Stop and often reverse the progressive joint involvement

B. Specifically reverse the cause of the disease

C. Provide symptomatic analgesic and anti-inflammatory effects without preventing progressive joint damage

D. Are effective because they are uricosuric agents

E. Are more effective when given with allopurinol

ANSWER: C

Explanation:

Provide symptomatic analgesic and anti-inflammatory effects without preventing progressive joint damage is correct. Salicylates, such as aspirin, inhibit cyclooxygenase activity and thereby reduce prostaglandin-mediated pain, tenderness, swelling, and stiffness. In rheumatoid arthritis, these actions can improve comfort and functional ability, particularly while a patient is awaiting benefit from longer-acting therapy or needs additional symptomatic relief. Their role is therefore symptomatic: they can reduce manifestations of active inflammation but do not modify the underlying autoimmune process or reliably prevent the structural cartilage and bone damage associated with persistent rheumatoid arthritis. Modern rheumatoid arthritis management emphasizes early treatment with disease-modifying antirheumatic drugs because controlling inflammation with a disease-modifying strategy is necessary to reduce progression and preserve joint function. The American College of Rheumatology's rheumatoid arthritis guidance discusses treatment approaches centered on disease-modifying therapy: American College of Rheumatology—Rheumatoid Arthritis Clinical Practice Guidelines. Additional clinical background on the disease course and treatment goals is available from the Merck Manual Professional Edition.

QUESTION NO: 38

Which of the following is considered a class IA Sodium Channel blocker?

A. Propafenone

B. Disopyramide

C. Aminodarone

D. Quinidine

ANSWER: D

Explanation:

Quinidine is a class IA antiarrhythmic sodium-channel blocker. Class IA agents bind to and inhibit fast voltage-gated sodium channels in working atrial and ventricular myocardium, reducing the rate of phase 0 depolarization and slowing impulse conduction. In addition to sodium-channel blockade, quinidine blocks potassium currents, which prolongs the action-potential duration and effective refractory period. This combined electrophysiologic effect produces QT-interval prolongation on the ECG, a characteristic feature of class IA antiarrhythmic activity. Quinidine has historically been used for suppression or conversion of certain atrial and ventricular arrhythmias, although its use is limited by adverse effects and proarrhythmic potential, including torsades de pointes. The FDA prescribing information identifies quinidine as a class IA antiarrhythmic drug and describes its effects on cardiac conduction and repolarization. For the pharmacologic classification and clinical information, see the DailyMed quinidine drug-label search and the NCBI review of antiarrhythmic medications.

QUESTION NO: 39

Which of the following is considered as the best treatment of asthma?

A. 2-agonists

B. -Blockers

C. -agonists

D. Anticholinergic drugs

E. Cholinergic drugs

F. Inhaled corticosteroid-containing therapy

ANSWER: F

Explanation:

Inhaled corticosteroid-containing therapy is the best answer because asthma is a chronic inflammatory airway disease, and treatment should address the underlying airway inflammation as well as symptoms. Inhaled corticosteroids reduce airway inflammation and hyperresponsiveness, decrease the frequency and severity of exacerbations, improve lung function and symptom control, and reduce asthma-related hospitalization and death. They are the foundation of long-term asthma management across the spectrum of persistent asthma. Current Global Initiative for Asthma guidance recommends that adults and adolescents receive an inhaled corticosteroid-containing regimen rather than treatment with a short-acting bronchodilator alone. Depending on symptom frequency and severity, this may be as-needed low-dose inhaled corticosteroid-formoterol or a daily inhaled corticosteroid-containing controller regimen. The selected regimen should be individualized, with inhaler technique, adherence, trigger reduction, and a written asthma action plan reviewed regularly. This answer is necessarily broader and more current than a bronchodilator-only choice, because prompt symptom relief does not by itself provide the anti-inflammatory control needed to prevent future attacks. See the Global Initiative for Asthma Strategy Report and the National Heart, Lung, and Blood Institute asthma guidance.

QUESTION NO: 40

Which of the following is NOT an essential amino acid?

A. Methionine

B. Arginine

C. Leucine

D. Tryptophan

E. Glycine

ANSWER: E

Explanation:

Glycine is the correct answer because it is classified as a nonessential amino acid in healthy humans. “Nonessential” does not mean that glycine lacks physiologic importance; rather, it means that the body can synthesize an adequate amount under usual conditions, so a dietary supply is not obligatorily required to prevent deficiency. Glycine is produced endogenously primarily from serine through the enzyme serine hydroxymethyltransferase, using tetrahydrofolate-dependent one-carbon metabolism. It also participates broadly in protein synthesis and serves as a precursor for important molecules, including glutathione, purines, heme, creatine, and bile salts. Nutrition references commonly list glycine among the amino acids that can be made by the body, in contrast to amino acids that must be obtained from food because human synthesis is absent or insufficient. This distinction is central when evaluating protein quality and dietary amino-acid requirements. For an overview of amino-acid metabolism and the synthesis of glycine from serine, see NCBI Bookshelf: Amino Acid Metabolism. The National Academies’ dietary reference material also discusses indispensable amino-acid requirements and protein nutrition: Dietary Reference Intakes.

QUESTION NO: 41

Indication of phenoxybenzamine is:

A. Pain

B. Movement disorders

C. Thyrotoxicosis - preparation for radioactive iodine therapy

D. Anaphylactic shock

E. Acute hypertension

F. Pheochromocytoma, including preoperative management of hypertension and sweating

ANSWER: F

Explanation:

Phenoxybenzamine is indicated for treatment of pheochromocytoma, a catecholamine-secreting tumor that can cause episodic or sustained severe hypertension, diaphoresis, palpitations, and headache. It is used to prevent hypertensive episodes and excessive sweating in affected patients and is particularly important before surgical tumor removal. By producing long-lasting, noncompetitive blockade of peripheral adrenergic receptors, phenoxybenzamine reduces the vasoconstrictive effects of excess norepinephrine and epinephrine. Preoperative receptor blockade helps stabilize blood pressure and decreases the risk of dangerous hypertensive crises during anesthesia and manipulation of the tumor. Dosing is individualized and titrated clinically, with restoration of intravascular volume commonly addressed during preparation for surgery. The FDA-approved prescribing information specifically identifies pheochromocytoma treatment, prevention of hypertensive episodes and sweating, and preoperative management as its labeled uses. See the DailyMed phenoxybenzamine prescribing information and the NCBI StatPearls review of phenoxybenzamine for clinical pharmacology and use in pheochromocytoma.

QUESTION NO: 42

Half-life of streptomycin is:

A. 10-50 hours

B. 8 hours

C. 1 hour

D. 16 hours

E. 3-4 hours

ANSWER: E

Explanation:

The correct answer is **3-4 hours**. Streptomycin is an aminoglycoside antibiotic that is administered parenterally and is eliminated primarily as unchanged drug through glomerular filtration. In adults with normal renal function, its usual elimination half-life is approximately 2 to 3 hours; pharmacokinetic references may describe a range that extends to about 3 to 4 hours depending on the population studied, assay method, and clinical conditions. Therefore, 3-4 hours is the best available answer.

This time course is clinically important because aminoglycoside concentrations and toxicity risk are strongly affected by renal clearance. When kidney function is impaired, streptomycin elimination is substantially prolonged, requiring individualized dose adjustment and, when appropriate, therapeutic drug monitoring. The product labeling describes renal excretion as the principal route of elimination and notes the importance of renal function in determining drug accumulation. For prescribing and pharmacokinetic information, consult the DailyMed streptomycin drug-label search and the NCBI Bookshelf aminoglycosides review.

QUESTION NO: 43

Pannus is an inflammatory exudates characterized by:

I- Synovial cell proliferation inside of a joint

II- Normally occurs in patients with rheumatoid arthritis

III- An immunologic complication

A. I only

B. III only

C. I and II only

D. II and III only

E. All are correct

ANSWER: E

Explanation:

All are correct. In rheumatoid arthritis, persistent autoimmune synovitis causes hyperplasia and proliferation of synovial lining cells along with infiltration by immune and inflammatory cells. This chronically inflamed synovium develops into pannus: invasive vascular granulation-like tissue that extends over articular cartilage and into adjacent bone. Thus, synovial-cell proliferation within the joint is a key feature of pannus formation.

Pannus is classically associated with rheumatoid arthritis, in which immune dysregulation drives chronic synovial inflammation. Activated T cells, B cells, macrophages, cytokines such as tumor necrosis factor and interleukin-6, and synovial fibroblasts contribute to the destructive tissue response. The pannus can release inflammatory mediators and matrix-degrading enzymes, promoting cartilage loss and marginal bone erosions. Therefore, it is appropriately understood as an immunologically mediated manifestation of rheumatoid arthritis rather than merely a nonspecific joint effusion.

These features connect the microscopic synovial proliferation with the clinically important joint destruction characteristic of established rheumatoid arthritis. See the Merck Manual Professional discussion of rheumatoid arthritis and the NCBI Bookshelf review of rheumatoid arthritis.

QUESTION NO: 44

Importance of Vitamin D in osteoporosis:

I- Increase the absorption of calcium from the small intestine

II- The best vitamin D analog to treat osteoporosis is calcitriol

III- Antacids containing aluminum and magnesium may interfere with proper activity ofvitamin D.

A. I only

B. III only

C. I and II only

D. II and III only

E. All are correct

F. I and III only

ANSWER: F

Explanation:

I and III only is correct. Vitamin D is important in osteoporosis because it promotes intestinal absorption of calcium, helping maintain an adequate calcium supply for bone mineralization and for suppression of secondary hyperparathyroidism. In people with osteoporosis or at risk for fracture, vitamin D sufficiency is a foundational component of bone health management, particularly when calcium intake is inadequate. The National Institutes of Health notes that vitamin D promotes calcium absorption in the gut and is necessary for normal bone growth and mineralization.

Aluminum- and magnesium-containing antacids also warrant attention during vitamin D therapy, especially with active vitamin D products such as calcitriol and in patients with impaired renal function. Product labeling cautions that magnesium-containing antacids can contribute to hypermagnesemia in susceptible patients, while aluminum exposure can be clinically important because vitamin D therapy may increase aluminum absorption. These mineral-related interactions can compromise the safe and appropriate use of vitamin D therapy and should be avoided or managed carefully. See the NIH Vitamin D Fact Sheet for Health Professionals and the DailyMed calcitriol prescribing information.

QUESTION NO: 45

What is correct regarding the formation of proteins?

A. Formed by condensation reactions that create peptide bonds

B. Geometric sequence of amino acids

C. The formation occurs by complexation of amino acids

D. Proteins are formed from carbohydrates

E. None of the above is right

ANSWER: A

Explanation:

Proteins are formed when amino acids are linked in a specific linear order by **peptide bonds**. A peptide bond is an amide linkage formed between the carboxyl group of one amino acid and the amino group of the next amino acid. Its formation is a condensation (dehydration) reaction because the reaction results in the loss of the elements of water. Repetition of this reaction produces a polypeptide chain, which subsequently folds into a functional protein; some proteins also assemble with additional polypeptide subunits. In cells, protein synthesis occurs on ribosomes during translation: transfer RNAs bring amino acids specified by the messenger-RNA sequence, and the ribosome catalyzes peptide-bond formation as the chain elongates. Thus, describing proteins as formed by condensation reactions that create peptide bonds accurately identifies the fundamental chemical process responsible for their primary structure. Further detail on amino-acid polymerization and peptide bonds is available from NCBI Bookshelf: Molecular Biology of the Cell and OpenStax Biology: Translation.

QUESTION NO: 46

Correct statements regarding mutation may include:

I- Cell division

II- Change in genotype

III- Change in DNA replication altering the gene

A. I only

B. III only

C. I and II only

D. II and III only

E. All are correct

ANSWER: D

Explanation:

“II and III only” is correct because a mutation is a heritable alteration in an organism’s genetic material, usually involving a change in the DNA nucleotide sequence. Since genotype describes an individual’s genetic constitution, an alteration in a gene or other DNA sequence represents a change in genotype. This is the central feature of a mutation, whether the change is a single-base substitution, insertion, deletion, or a larger rearrangement of DNA.

Mutations commonly originate when an error occurs during DNA replication. Normally, DNA polymerases copy genetic information with high fidelity and proofreading mechanisms correct many errors. However, an uncorrected replication error can become fixed in the DNA sequence after subsequent replication, thereby altering a gene. Such changes may be inherited by daughter cells and, when present in germ cells, can potentially be transmitted to offspring. The National Human Genome Research Institute defines mutation as a change in a DNA sequence, and its educational resources describe replication errors as an important source of these changes. See NHGRI: Mutation and NCBI Bookshelf: DNA Replication, Repair, and Recombination.

QUESTION NO: 47

Which of the following is the most important vitamin in pregnancy?

A. Cyanocobalamin

B. Folic acid

C. Pantothenic acid

D. Pyridoxine

E. Niacin

ANSWER: B

Explanation:

Folic acid is the key vitamin to emphasize in pregnancy because adequate intake before conception and during early gestation substantially reduces the risk of neural tube defects, including spina bifida and anencephaly. The neural tube develops and closes very early—often before a person realizes that they are pregnant—so supplementation is most effective when it begins at least 1 month before conception and continues through the first 12 weeks of pregnancy. For people who could become pregnant, public-health guidance recommends 400 micrograms of folic acid daily from fortified foods, supplements, or both. Prenatal vitamins commonly provide this amount, although patients with a prior neural tube defect–affected pregnancy or certain high-risk conditions may require a higher dose under clinician supervision. Folate is needed for one-carbon transfer reactions that support DNA synthesis, cell division, and normal fetal tissue development, making it especially important during the rapid growth of early embryonic development. The CDC provides preconception and pregnancy-focused folic-acid guidance at CDC: About Folic Acid. The U.S. Preventive Services Task Force likewise recommends a daily supplement containing 0.4 to 0.8 mg of folic acid for persons planning to or capable of pregnancy; see USPSTF folic acid recommendation.

QUESTION NO: 48

Which of the following is considered a class IA Sodium Channel blocker?

A. Mexiletine

B. Amiodarone

C. Quinidine

D. Procainamide

ANSWER: C

Explanation:

Quinidine is a class IA antiarrhythmic medication and a sodium-channel blocker. Class IA agents bind cardiac fast sodium channels with intermediate kinetics, reducing the rate of phase 0 depolarization in fast-response myocardial tissue. This slows conduction velocity and widens the QRS complex. In addition to sodium-channel blockade, quinidine blocks potassium currents, prolonging the action-potential duration and effective refractory period; this is reflected clinically by QT-interval prolongation. These electrophysiologic effects make quinidine a prototypical class IA drug in the Vaughan Williams antiarrhythmic classification system. Its pharmacologic profile is important because the prolonged repolarization can predispose susceptible patients to QT-related proarrhythmia, so appropriate ECG monitoring and clinical assessment are central to its safe use. The National Library of Medicine's overview of antiarrhythmic drugs describes class IA sodium-channel blockade and its effects on conduction and repolarization. Additional prescribing and safety information for quinidine is available through the NIH DailyMed database.

References: NCBI Bookshelf: Antiarrhythmic Medications; DailyMed: Quinidine drug-label search.

QUESTION NO: 49

Correct statements regarding glossitis include which of the following?

I- Inflammation of the tongue

II- Characterized by presence of pinkish-red central lesions on the dorsal surface of thetongue

III- It can be a chronic condition of the tongue seen in pernicious anemia.

A. I only

B. III only

C. I and II only

D. II and III only

E. All are correct

ANSWER: E

Explanation:

All are correct. Glossitis is, by definition, inflammation of the tongue. Clinical findings vary with the underlying cause and the specific type of glossitis, but affected areas may become red or pink, smooth because of papillary atrophy, sore, and swollen. A centrally located pink-to-red lesion on the dorsal tongue is a recognized appearance in localized forms such as median rhomboid glossitis; more broadly, inflammatory and atrophic tongue disorders can produce erythematous depapillated areas on the dorsal surface.

Glossitis may also be chronic when it reflects an ongoing nutritional or hematologic disorder. Pernicious anemia causes vitamin B12 deficiency through impaired intrinsic-factor-mediated vitamin B12 absorption. Vitamin B12 deficiency can impair epithelial cell turnover and produce atrophic glossitis, classically described as a smooth, erythematous, painful or burning tongue. Therefore, tongue inflammation, pinkish-red dorsal lesions in characteristic glossitis presentations, and chronic glossitis associated with pernicious anemia are all clinically consistent statements. The MSD Manual discusses glossitis and associated nutritional deficiencies, while StatPearls reviews its clinical patterns and causes, including vitamin B12 deficiency: MSD Manual: Glossitis; NCBI Bookshelf: Glossitis.

QUESTION NO: 50

In an adequately powered, randomized controlled trial conducted over 3 years, a specific serious side effect (i.e. reduction in leukocytes) with conventional therapy is seen in 0.5% of the study sample. In patients who receive a newly discovered drug, only 0.45% experience the same side effect. Based on these results, the minimum number of patients that would have to receive the new drug for 3 years to statistically demonstrate the prevention of one episode of this side effect in at least one patient is:

A. 15.

B. 20.

C. 150.

D. 200.

E. 2000.

ANSWER: E

Explanation:

2000 is correct because the relevant measure is the number needed to treat (NNT), calculated from the absolute risk reduction. Over 3 years, the risk of leukocyte reduction is 0.5% with conventional therapy and 0.45% with the new drug. The absolute risk reduction is therefore 0.05 percentage points: 0.50% − 0.45% = 0.05%. Expressed as a proportion, 0.05% equals 0.0005. The NNT is the reciprocal of the absolute risk reduction: 1 ÷ 0.0005 = 2000. Thus, treating 2,000 patients with the new drug for the stated 3-year period would be expected to prevent one additional episode of leukocyte reduction compared with conventional therapy. NNT is conventionally reported as a whole number and reflects the treatment duration and outcome definition used in the underlying comparison. This calculation describes the expected absolute clinical benefit derived from the observed event rates; an adequately powered trial and appropriate statistical analysis are separately needed to establish that the observed difference is statistically reliable. See the Centre for Evidence-Based Medicine NNT resource and NCBI guidance on absolute risk reduction and NNT.

QUESTION NO: 51

Colitis associated with antibiotic use can best be treated by:

A. Cephalosporins

B. Macrolides

C. Aminoglycosides

D. Oral vancomycin

E. Penicillin

ANSWER: D

Explanation:

Oral vancomycin is an appropriate treatment for antibiotic-associated colitis caused by *Clostridioides difficile* (CDI), the clinically important cause of diarrhea and colitis that develops during or after antibacterial exposure. Treatment should also include stopping the precipitating antibiotic when it is no longer necessary, providing fluid and electrolyte support, and assessing disease severity. Vancomycin must be administered orally for CDI because its minimal gastrointestinal absorption allows high drug concentrations to remain in the intestinal lumen, where the organism is located. Oral vancomycin is a recommended regimen for an initial CDI episode and is particularly important in fulminant CDI, in which higher-dose oral or nasogastric administration may be used with additional therapy as clinically indicated. Although current guidelines generally prefer fidaxomicin when it is available and feasible for an initial episode, it is not among the listed choices; therefore, oral vancomycin is the best available answer. See the IDSA/SHEA focused CDI guideline update and the CDC's clinical overview of C. difficile.

QUESTION NO: 52

Subclass of lomafloxacin is:

A. Fluoroquinolones

B. IA

C. Calciferols

D. Beta-lactamase resistant penicillins

E. Aldosterone antagonists

ANSWER: A

Explanation:

Lomafloxacin is a fluoroquinolone antibacterial agent. Fluoroquinolones are a subclass of quinolone antibiotics characterized by fluorine substitution on the quinolone nucleus; this structural feature contributes to their antibacterial activity and pharmacologic properties. Lomafloxacin acts by inhibiting bacterial DNA gyrase and topoisomerase IV, enzymes required for DNA replication, transcription, and repair. Inhibition of these enzymes produces bactericidal activity against susceptible organisms. The official prescribing information identifies lomafloxacin as a synthetic broad-spectrum fluoroquinolone antimicrobial, supporting classification as Fluoroquinolones. This classification is clinically meaningful because medicines in this group share important pharmacologic characteristics and class safety considerations, including potentially serious adverse effects involving tendons, peripheral nerves, and the central nervous system. Although lomafloxacin has historically been used for selected bacterial infections, its regulatory labeling and availability may vary by jurisdiction and over time. The relevant drug class is determined by its quinolone chemical scaffold and antibacterial mechanism, rather than by its therapeutic indication alone. See the FDA-approved labeling for Maxaquin (lomafloxacin) prescribing information and the NCBI overview of fluoroquinolones.