SUMMER – 2023 EXAMINATION

Model Answer – Only for the Use of RAC Assessors

Subject Name: Pharmacotherapeutics **Subject Code:** 20224

Important Instructions to examiners:

1) The answers should be examined by key words and not as word-to-word as given in the model answer scheme.
2) The model answer and the answer written by the candidate may vary but the examiner may try to assess the understanding level of the candidate.
3) The language errors such as grammatical, spelling errors should not be given more Importance (Not applicable for subject English and Communication Skills.
4) While assessing figures, the examiner may give credit for principal components indicated in the figure. The figures drawn by candidate and model answer may vary. The examiner may give credit for any equivalent figure drawn.
5) Credits may be given step wise for numerical problems. In some cases, the assumed constant values may vary and there may be some difference in the candidate's answers and model answers.
6) In case of some questions credit may be given by judgement on part of the examiner of relevant answers based on the candidate's understanding.
7) For programming language papers, credit may be given to any other program based on an equivalent concept.
8) As per the policy decision of Maharashtra State Government, teaching in English/Marathi and Bilingual (English + Marathi) medium is introduced in the first year of AICTE diploma Programme from academic year 2021-2022. Hence if the students write answers in Marathi or bilingual language (English +Marathi), the Examiner shall consider the same and assess the answer based on matching of concepts with model answers.

Q. No.	Sub No.	Answers	Marking Scheme
1		**Answer any SIX of the following:**	**30M**
1	a	**Define pharmacotherapeutics and standard treatment guidelines along with advantages of STGs.** **Marking scheme-(each def 1 M, any 6 advantages 3 M)** **Answer:** **Pharmacotherapeutics** is the application of knowledge of drugs and diseases to prevent, treat, and diagnose the disease as well as alter normal functions (such as preventing pregnancy). **or** Pharmacotherapeutics is a branch of pharmacology that deals with the therapeutic applications and the effect of drugs. According to WHO, **Standard Treatment Guidelines** (STGs) are systematically developed statements to help practitioners make decisions about appropriate healthcare for specific clinical problems. **Advantages of STGs**: The use of STGs can benefit healthcare providers, healthcare officials, supply management personnel and patients in the following ways: **Healthcare providers**	**5M**

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1. Provides practitioners with standardised guidance.
2. Encourages high quality care by directing practitioners to the best medicines for specific conditions.
3. Encourages the best quality of care as patients are receiving optimal therapy.
4. Uses only formulary or essential medicines, so the healthcare system needs to provide only the medicines in the STGs.
5. Provides valuable assistance to all practitioners, especially to those with fewer skills.
6. Enables healthcare providers to concentrate on making the correct diagnosis because treatment options will be provided for them.

Healthcare officials

1. Provides a basis for evaluating the quality of care provided by healthcare professionals.
2. Provides the most effective therapy in terms of quality.
3. Provides a system for controlling costs by using funds more efficiently.
4. Provides practitioners with information about the institution's standards of care for patients.
5. Can serve as a vehicle for integrating special programs (e.g., diarrhoea disease control, acute respiratory infection (ARI), tuberculosis control, malaria) at the primary health care facilities through the use of a single set of guidelines

Supply management

1. Since the formulary or essential medicines is utilised, the healthcare system needs to provide only medicines in the STGs.
2. Provides forecasting and ordering information (because medicines and quantities for common diseases will be known).
3. Provides information for the purchase of prepackaged medicines.

Patients

1. Patients receive the best medical therapy.
2. Enables consistent and predictable treatment from all levels of providers and at all locations within the healthcare system.
3. Improves the availability of medicines because of more consistent use and ordering.
4. Provides improved outcomes because patients are receiving the best treatment regimens available.
5. Lowers the cost.

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1	b	**Write about etiopathogenesis of angina and its pharmacological management.** **Marking scheme:**(Etiopathogenesis 2.5 M and pharmacological management 2.5 M)	5M

Answer:

Etiopathogenesis:

- **Coronary artery disease:** This is a buildup of plaque and cholesterol inside your coronary arteries, which supply blood to your heart muscle. The buildup narrows your artery so much that the oxygen-rich blood your heart needs can't get through, and your heart muscle becomes starved for oxygen. This causes ischemia and angina. Atherosclerotic plaque causes 70% of fatal heart attacks.
- **Blood clot:** When plaque that forms in your narrow coronary artery breaks apart, it can attract a blood clot. When a blood clot settles in a coronary artery that's already narrow, it can cause a blockage (thrombosis).
- **Coronary artery spasm:** This happens when the coronary arteries spasm, which temporarily reduces or cuts off blood supply to your heart.
- **Coronary artery dissection:** This rare condition can keep blood from getting to your heart.
- Ischemic heart disease like angina and myocardial infarction result due to the effect on supply and demand of oxygen to myocardium.
- Coronary arteries attached to the aorta supply the oxygen to the heart muscle i.e. myocardium.
- Problems can arise when there is a restriction of blood flow through coronary arteries; this is commonly due to atherosclerosis, in which fatty material deposits in arteries and restricts the blood flow to myocardium.
- When the luminal diameter is more than 70% blocked, stable angina arises.
- When oxygen demand of myocardium increased due to stress, coronary arteries were unable to supply due to stenosis(narrowing).
- When coronary flow becomes zero this leads to ischemia and leads to angina and it depends on demands of oxygen by myocardium.

The risk factors associated with angina pectoris and myocardial infarction are dyslipidaemia with low high-density lipoprotein and elevated low-density lipoprotein forms of cholesterol, hypertriglyceridemia, family history, age, premature menopause in women, smoking, and disease conditions like hypertension, obesity, and diabetes mellitus.

Pharmacological management

The drugs show their action by either increasing the amount of blood flow to the cardiac muscles (oxygen supply) or decreasing the oxygen requirement (workload) of the heart.

The following classes of drugs are useful in the treatment of angina pectoris:

- **Organic nitrates** e.g., Glyceryl trinitrate, isosorbide dinitrate, pentaerythrityl tetranitrate
- Beta adrenergic blockers e.g., metoprolol, atenolol

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		• Calcium channel blockers e.g., verapamil, diltiazem, amlodipine • Antiplatelet drugs e.g., aspirin, clopidogrel • Potassium channel activators e g., nicorandil, minoxidil, diazoxide **Organic nitrates:** They are available in multiple dosage forms like sublingual tablets, sprays, ointments, transdermal patches, oral sustained release, and intravenous preparations. The nitrates act as vasodilators, mainly as venodilators. Due to the dilation of the veins, the venous return decreases, leading to a reduction in the preload. This helps by reducing the myocardial oxygen demand. Also, they cause coronary vasodilation, leading to increased blood supply and, in turn, oxygen supply to the myocardium, thus relieving angina. **Beta adrenergic blockers:** Beta blockers help in the treatment of angina pectoris by reducing the myocardial oxygen demand. **Calcium channel blockers:** These drugs inhibit the entry of calcium ions and cause vasodilation and decreased heart rate. **Antiplatelet drugs:** These drugs work by decreasing platelet aggregation and inhibiting thrombus formation. **Potassium channel activators:** These act as agonists on ATP-sensitive potassium channels. It shows vasodilation both in arterial and venous vascular beds, leading to a reduction in afterload and preload of the heart. It is used in patients with stable coronary artery disease.	
1	c	**Explain etiopathogenesis and pharmacological management of diabetes.** **Marking scheme:**Etiopathogenesis 2.5 M and pharmacological management 2.5 M) **Answer:** **Etiopathogenesis** **Etiology:** Type-I Diabetes Mellitus: (or IDDM (insulin dependent diabetes mellitus) • Genetic Factors: It involves multiple genes responsible for inheritance of diabetes mellitus. • Autoimmune Factors: Sometimes the beta cells act as autoantigens and activate CD4+ T lymphocytes, causing destruction of pancreatic beta cells. • Environmental Factors: Certain viral infections (e.g., Mumps, measles Coxsackie virus, cytomegalovirus), drugs (e.g., alloxan, pentamidine, streptozocine), geographic and seasonal variation can also induce type I diabetes mellitus. Type II Diabetes Mellitus: (or NIDDM (non-insulin dependent diabetes mellitus)	**5M**

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- Genetic Factors: Study of genetic factors for type II DM is more important than for type L. If parents are suffering from type II DM, then offsprings will be at 50% more risk to have type II DM.
- Constitutional Factors: It involves the environmental factors and other factors such as obesity, hypertension and low level of physical activity.
- Insulin Resistance: Most prominent feature of type II DM is lack of response to the insulin secreted (peripheral tissues like skeletal muscle, liver develop resistance to inulin. Obesity is strongly associated with insulin resistance.
- Impaired Insulin Secretion: At earlier stages of type II DM insulin secreted in large amounts to compensate for increased glucose levels. But later on, there is an occurrence of failure of beta cells to secrete adequate insulin. Means in this type also mild deficiency of insulin occurs.
- Increased Hepatic Glucose Synthesis: Insulin has a role to suppress gluconeogenesis in the liver. But in type II DM, the liver becomes resistant to insulin. So, gluconeogenesis i.e. synthesis of glucose cannot be suppressed by insulin.

Numerous factors are associated with it. Few of them are:

- Obesity
- Increasing age
- Emotional stress
- Autoimmune - cells destruction
- Endocrine diseases like hypothyroidism and acromegaly
- Drugs like glucocorticoids, thyroid hormones

Pathogenesis:

- In this condition the immune system attacks and destroys the insulin producing beta cells of the pancreas.
- There is beta cell deficiency leading to complete insulin deficiency.
- Thus it is termed an autoimmune disease where there are anti insulin or anti-islet cell antibodies present in blood.
- These cause lymphocytic infiltration and destruction of the pancreas islets.
- The destruction may take time but the onset of the disease is rapid and may occur over a few days to weeks.
- There may be other autoimmune conditions associated with type 1 diabetes including vitiligo and hypothyroidism.
- Type 1 diabetes always requires insulin therapy, and will not respond to insulin-stimulating oral drugs.

Pharmacological Management

Pharmacotherapy of diabetes includes insulin or oral hypoglycemic agents.

Insulin

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All type I patients require insulin for their survival. Insulin may be categorized based on its time activity profile as:

- Ultra-short acting
- Short acting
- Intermediate acting
- Long acting insulin includes Lantus (Insulin Glargine), Levemir (Insulin detemir), Tresiba (Insulin degludec) made by recombinant DNA technology.

Humalog (insulin lispro) and NovoLog are short acting insulins which is given in combination with a long acting insulin.
Oral Hypoglycemic Agents

Following categories of the drugs can be used orally for type 2 diabetes patients.

- Sulphonylureas e.g. tolbutamide, chlorpropamide (1st generation), glibenclamide, glipizide, glimepiride, glyburide (2nd generation)
- Biguanides e.g. metformin
- Alpha-glucosidase inhibitors e.g. Acarbose, voglibose
- Thiazolidinediones e.g. pioglitazone, rosiglitazone
- Meglitinides e.g. repaglinide, nateglinide
- Dipeptidyl peptidase - 4 (DPP-4) inhibitors e.g. sitagliptin, vildagliptin

Sulphonylureas: Sulphonylureas exert both pancreatic and extrapancreatic effects but are useful only in patients who have intact and functioning beta cells. It increases insulin secretion directly and decreases glucagon release. It also increases insulin receptor binding affinity thereby increasing post receptor action., e.g. tolbutamide, chlorpropamide, glibenclamide, glipizide, glimepiride, gliclazide

Biguanides: They reduce hepatic glucose production and glycogen metabolism in the liver and enhance insulin-mediated glucose uptake by skeletal muscles, e.g. metformin Metformin is the only biguanide widely used for diabetic patients. It is more useful in overweight patients as it does not cause weight gain. In the initial stage of treatment with metformin, adverse effects like abdominal bloating, nausea, intestinal cramping and diarrhoea are observed, but they subside with continued use.

Alpha-glucosidase Inhibitors: Alpha-glucosidase along with alpha-amylase causes hydrolysis of complex carbohydrates. Acarbose, which is a alpha-glucosidase and alpha-amylase inhibitor, reduces the rate of carbohydrate metabolism and subsequent glucose absorption, thereby lowering postprandial glucose excursion in diabetic patients.

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		Acarbose is particularly effective in patients with postprandial hyperglycemia. **Thiazolidinediones:** Thiazolidinediones also known as glitazones, are insulin sensitizers which lower glucose level by enhancing insulin action and reducing insulin resistance. It is mainly active on skeletal muscles, liver and heart. Common example is pioglitazone, but it is associated with weight gain and oedema. It should not be used in patients with preexisting heart failure. **Meglitinides:** Meglitinide, also known as a postprandial glucose regulator, is an insulin releasing agent. They have more rapid onset and short duration of action than sulphonylureas. Adverse effects of meglitinide include hypoglycemia, visual disturbances, abdominal pain, diarrhoea, constipation, nausea, vomiting and rarely hypersensitivity reactions, e.g. repaglinide, nateglinide. **Dipeptidyl peptidase - 4 (DPP-4) inhibitors:** These act on incretin hormones, mainly GLP-1 (glucagon-like peptide-1) and GIP (gastric inhibitory peptide), which maintain glucose homeostasis by increasing insulin secretion and decreasing glucagon secretion. e.g. sitagliptin, vildagliptin Combination Therapy: It has been observed that combination therapy is more beneficial in type 2 diabetic patients. Insulin requirement may be reduced when combined with oral agents like sulphonylureas, acarbose, metformin, troglitazone and repaglinide.	
1	d	**Explain epilepsy with the types and give the pharmacological management of epilepsy.** **Marking scheme** :(explanation of epilepsy 0.5 X 6 = 3 M and pharmacological management 0.5 X 4 = 2 M) **Answer:** Epilepsy is a chronic neurological disorder characterised by recurrent episodes of seizures. An epileptic seizure or convulsion is a transient abnormal event caused by a paroxysmal release of cerebral neurons that is visible to a person experiencing the seizures and/or a spectator. Seizures are termed “generalized” when both the hemispheres are activated at the same time. If a discharge starts in a localized area of the brain, the seizures are termed partial or focal.	5M

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1. **Generalized seizures:** These seizures affect both hemispheres of the brain at the same time.

- **Grandmal epilepsy or Tonic-clonic Seizures:** The usual sequence is aura, epileptic cry, unconsciousness and sudden fall due to tonic convulsions followed by clonic jerking and then prolonged sleep and depression. The attack lasts for 1-2 min. The attack may be accompanied by tongue biting, frothing and urinary incontinence.
- **Petitmal epilepsy or Absence seizures:** This is prevalent in children and the episode lasts for a few seconds. No aura, no or only momentary loss of consciousness, no fall The patient appears to go blank for less than 30 seconds and staring in one direction. Absence seizures have two categories: typical and atypical. Atypical absence seizures are similar to typical seizures, except that they tend to begin more slowly, last longer up to a few minutes, and can include falling down. The patient may feel confused for a short time after regaining consciousness.
- **Tonic seizures:** In this seizure, the muscle tone is greatly increased: the body, arms,or legs become suddenly stiff or tense. They are short, usually less than 20 seconds.
- **Atonic seizures:** Patients may fall without accompanying tonic or clonic movements. There may be brief loss of consciousness with relaxation of all muscles due to excessive inhibitory discharges.
- **Myoclonic seizures:** This is a sudden, brief, repetitive contraction of muscles of a limb or the whole body. There may be violent fall without loss of consciousness.

2.Partial or focal seizures: These are limited to one hemisphere of the brain.

- **Simple partial seizures:** Localised jerking of a limb or the face, stiffness or twitching of one part of the body, numbness, or abnormal sensations may occur during a simple partial seizure. The consciousness is fully preserved.
- **Complex partial seizures:** These seizures comprise impaired consciousness with abnormal behaviours like lip-smacking, buttoning or unbuttoning of clothing, or wandering behaviour.

Secondarily generalised seizures: These are partial seizures in which the discharge spreads to the entire brain which leads to a convulsive attack with the same characteristics as a generalised tonic-clonic seizure.

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Antiepileptic drugs for different types of seizures

Seizure type	First-line treatment	Second-line treatment
Generalised seizures		
Tonic clonic	Sodium valproate	Lamotrigine
Absence	Ethosuximide, Sodium valproate	Clonazepam, Lamotrigine
Myoclonic	Sodium valproate, Clonazepam	Levetiracetam,Acetazolamide, Topiramate
Atonic	Clonazepam, Clobazam	Lamotrigine, Carbamazepine, Phenytoin, Acetazolamide, Topiramate
Partial seizures	Carbamazepine,Oxcarbazepine, Levetiracetam	Topiramate, Sodium valproate, Clobazam, , Zonisamide, Pregabalin, Phenytoin, Gabapentin, Lamotrigine, Lacosamide

1 **e** **Explain in detail about peptic ulcer and its management.**

Marking scheme:(peptic ulcer explanation 2 M and management 1.5X2 = 3 M)

Answer : Peptic ulcer disease are ulcers (sores) which occur in the lining of the stomach (gastric ulcer) &/or small intestine (duodenal ulcer) due to exposure to gastric acid.

The risk factors for peptic ulcer disease are Helicobacter pylori infection, stress and long-term use of NSAIDs. The less common risk factors are Zollinger Ellison syndrome, viral infections, radiation therapy and chemotherapy.

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		Clinical manifestations • Burning stomach pain • Feeling of fullness, bloating or belching • Intolerance to fatty foods • Heartburn • Nausea • Vomiting or vomiting blood — which may appear red or black • Dark blood in stools, or stools that are black or tarry • Trouble breathing • Feeling faint • Nausea or vomiting • Unexplained weight loss • Appetite changes **Non-pharmacological management** • Peptic ulcer disease patients should eliminate smoking • Stop the use of non-steroidal anti-inflammatory drugs including aspirin • Try to reduce psychological stress. • The patients should also avoid spicy food, caffeine and alcohol that cause dyspepsia and exacerbate the symptoms of ulcers. • Try taking antacids • New techniques of endoscopic procedures like use of mechanical clips and coagulation forceps are being utilized. **Pharmacological management** 1. Proton pump inhibitors e.g. omeprazole, esomeprazole, lansoprazole, rabeprazole and pantoprazole 2. H_2 receptor antagonists e.g. cimetidine, famotidine and ranitidine 3. Bismuth compounds e g bismuth subsalicylate and colloidal bismuth subcitrate 4. Sucralfate 5. Prostaglandins e.g. Misoprostol 6. Antacids e.g. Aluminium hydroxide and Magnesium hydroxide	
1	f	**State the etiopathogenesis of hepatitis according to its types.** **Marking scheme:**(5 types 5 M) **Etiopathogenesis**	5M

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		Hepatitis A - Hepatitis A virus, also known as HAV or infectious hepatitis, is usually self-limiting and is an acute viral infection of the liver. This is a non-enveloped RNA virus and affects people worldwide. The route of transmission is the faecal oral route. Sources of infection are poor personal hygiene, poor sanitation, contaminated food and water, and infected food handlers. **Hepatitis B -** Hepatitis B, also known as HBV, is highly infectious. This is a DNA virus which replicates within the liver. The main routes of transmission are mother to child and sexual transmission in the case of adults. HBV can be transmitted via blood transfusion or blood product transfusion or via intravenous drug use or needle sharing, tattooing and ear piercing with contaminated needles. Various factors like age, genetic factors of the host, and virus characteristics determine the HBV infection. **Hepatitis C -** Hepatitis C virus (HCV) is a single-stranded RNA virus which was earlier labelled as *'non-A non-B hepatitis'*. Over 170 million people are infected worldwide with HCV. The hepatitis C virus replicates very fast inside the hepatocytes, thereby posing a challenge to the immune system of the host. Transmission of HCV commonly occurs through intravenous drug use, sharing of contaminated needles, and contaminated blood and blood products. A small risk is associated with tattooing, ear piercing, acupuncture, sexual contact, and transmission from mother to child. **Hepatitis D -** The Hepatitis D or HDV or Delta hepatitis agent is a defective single strand RNA virus that can co-infect with other hepadnaviruses like HBV. This virus cannot replicate on its own and requires the helper function of hepadnaviruses for its replication. **Hepatitis E (HEV)** Hepatitis E virus, HEV, was earlier labelled as 'endemic' or '*enterically transmitted non-A non-B hepatitis'*. It is primarily seen in India, other Asian countries, Africa, and Latin America. It is an RNA virus that causes acute hepatitis and is transmitted enterically through contaminated food and water and poor sanitation.	
1	**g**	**Give the management of tuberculosis in accordance with DOT therapy.** **Marking scheme: (Explanation regarding DOTs 3 marks and drug treatment 2 marks)**	**5M**

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DOT stands for 'directly observed therapy'. This is undertaken for patients where the sputum test does not come negative even after two months of therapy. The patient is asked to take the anti-TB medication under the observation of a healthcare worker. The medication is administered to the patient under DOTS therapy three times a week on alternate days.

- DOT is particularly important for children and adolescents, for patients with HIV infection, psychiatric illness, or substance abuse, After treatment failure, relapse, or development of drug resistance .
- DOT helps patients finish TB therapy as quickly as possible, without unnecessary gaps.
- DOT helps prevent TB from spreading to others.
- DOT decreases the risk of drug-resistance resulting from erratic or incomplete treatment.
- DOT decreases the chances of treatment failure and relapse.

DOT includes:

- Delivering the prescribed medication
- Checking for side effects
- Watching the patient swallow the medication
- Documenting the visit
- Answering questions

DOT should be initiated when TB treatment starts. Do not allow the patient to try self-administering medications and missing doses before providing DOT. If the patient views DOT as a punitive measure, there is less chance of successfully completing therapy.

DOT involves treatment with a four drug regimen. First line drugs are isoniazid (INH), Rifampicin (Rif), Prazinamide (PZA) and Ethambutol (EMB) for 6-9 months.

Isoniazid

Also called nicotinic acid hydroxide, INH is the first-line most widely used drug against TB. The advantages of this drug are that it is bactericidal in nature, relatively non-toxic, economical, and well absorbed when given orally or parenterally. INH is available in various dosage forms like tablets, syrups, and parenterals given as intravenous or intramuscular injections.

The adverse reactions of isoniazid are hepatitis, peripheral neuritis, aplastic anaemia, GI effects, hypersensitivity of skin, and CNS toxicity like hallucinations and convulsions.

Rifamycins-

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		Rifamycins are a group of broad-spectrum antibiotics consisting of rifampicin, rifabutin, and rifapentine. Rifampicin is a bactericidal agent for *Mycobacterium tuberculosis* as it shows synergism with INH. Rifampicin is available in the form of capsules and an aqua solution as a parenteral. The adverse reactions of rifampicin are effects on the functioning of the liver, skin reactions, gastrointestinal reactions, thrombocytopenic purpurea, fever, and chills. It also imparts a reddish-orange colour to body fluids and urine. **Pyrazinamide** Pyrazinamide is a bactericidal drug against mycobacteria in an acidic pH. It is a synthetic pyrazine analogue of nicotinamide. Pyrazinamide has been added to the initial therapy of tuberculosis treatment along with INH and rifampicin. The adverse reactions are anorexia, nausea, flushing, hepatitis, vomiting, arthralgia, hyperuricemia, dysuria, and skin hypersensitivity. **Ethambutol** Ethambutol is a bacteriostatic anti-TB agent. This is a synthetic agent which interferes with the mycobacterial cell wall formation. It is usually targeted towards actively dividing tubercle bacillus. The adverse reactions of Ethambutol include optic neuritis, headache, gastrointestinal intolerance, hyperuricemia and arthralgia. Second-line drugs -Other antibiotics are active against TB and are used primarily when patients have drug-resistant TB (DR-TB) or do not tolerate one of the first-line drugs. The 2 most important classes are aminoglycosides (and the closely related polypeptide drug, capreomycin) and fluoroquinolones; aminoglycosides are available only for parenteral use.	
2		**Answer any TEN of the following:**	**30 M**
2	**a**	**Define hypertension. What lifestyle changes need to be taken in hypertension?** **Marking scheme-(Def. 1 M, Explanation 0.5 x 4 = 2 M)** **Answer:** Hypertension is defined as abnormally high blood pressure (more than 120/80 mmHg) in the arteries. Persistent increase in systemic arterial blood pressure is known as hypertension.	**3M**

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		The following lifestyle modification need to be taken in hypertension : **1) Physical activity:** Regular aerobic physical activity for about 30 min/day on maximum days of the week. **2)Dietary sodium Restrictions**: Reduce dietary sodium intake to not more than 2.4 gm sodium or 6gm sodium chloride per day. **3)Adopt a DASH eating plan:** [Dietary Approaches to Stop Hypertension]:A diet rich in fruits, vegetables, with low-fat dairy products with a reduced content of saturated and total fat. **4)Body Weight Reduction:** Attain normal body weight with a Target BMI-18.5 to 22.9 kg/mm. **5)Avoid smoking.** **6) Reduce alcohol consumption:** Alcohol intake maximum of 2 drinks per day for men and 1 drink per day for women and lightweight people. **7) Stress management:** Do meditation regularly.	
2	**b**	**Write etiopathogenesis and clinical manifestations of asthma.** **Marking scheme-(Etiopathogenesis 1.5 M and clinical manifestations 1.5 M)** **Answer:** **Etiology:** The exact causes of asthma is unknown. An asthma trigger is anything that irritates the airways and sets off asthma symptoms. **The trigger factor of asthma are as follows:** 1. Allergens exposure: Allergy to feathers, Animal dander, Dust, Mites, Pollens, etc. 2. Air pollutants: Exhaust fumes, Perfumes, Oxidants, Cigarette smoking. 3. Drugs NSAIDs, Beta-adrenergic blockers, food preservatives, sulphite-containing topical ophthalmic solutions. 4. Cold air. 5. Upper respiratory tract infection. 6. Exercise, emotional stress. 7. Wood and vegetable dust, industrial chemicals, and plastics. 8. Environmental factors.	**3M**

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		Pathophysiology: • Pathophysiology of asthma consists of three key abnormalities: bronchoconstriction, airway inflammation, and mucous impaction. • Asthma is usually caused by an allergic reaction to an allergen and is mediated by immunoglobulin E (IgE). IgE is formed in response to exposure to allergens such as pollen or animal dander. • Sensitization occurs at first exposure, which produces allergen-specific IgE antibodies that attach to the surface of mast cells. • Upon subsequent exposure, the allergen binds to the allergen-specific IgE antibodies present on the surface of mast cells, causing the release of inflammatory mediators such as leukotrienes, histamine and prostaglandins. • These inflammatory mediators cause bronchospasm, triggering an asthma attack. If an attack is left untreated, eosinophils, T helper cells and mast cells migrate into the airways. Excess mucus production caused by goblet cells plug the airway and, together with increased airway tone and airway hyperresponsiveness, this causes the airway to narrow and further exacerbates symptoms. **Clinical manifestation:** 1. Wheezing 2. Shortness of breath 3. Tachypnoea 4. Chest tightness 5. Cough that produces thick mucus 6. Tachycardia 7. Sweating 8. Coughing.	
2	**c**	**Give the management of parkinsonism.** **Marking scheme-(Non Pharmacological management 0.5 X 2 = 1 mark and Pharmacological management 0.5 X 4 = 2 marks)** **Answer:** **Non Pharmacological management of Parkinson's disease:** • Offer psychosocial help to patients and family. • Counsel patients and families on how to cope with the illness. • Counsel on the adaptations that may be needed to ensure productivity at work. • Fall Prevention-prevent falls by wearing leather-soled shoes. **Pharmacological management:**	**3M**

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		Antiparkinsonian drugs can only help to reduce the symptoms and improve the quality of life. Two categories which are used in the management of disease: 1. To enhance dopamine activity 2. To depress cholinergic overactivity. **1. Drugs affecting brain dopaminergic system:** • Dopamine precursor- Ex.Levodopa • Peripheral decarboxylase inhibitors- Ex.Carbidopa, Benserazide • Dopaminergic agonists- Ex.Bromocriptine, Ropinirole, Pramipexole • MAO-B inhibitors- Ex.Selegiline • COMT inhibitors-Ex. Entacapone, Tolcapone • Dopamine facilitator- Ex.Amantadine **2. Drugs affecting brain cholinergic system:** • Central anticholinergics- Ex.Trihexyphenidyl , Procyclidine, Biperiden • Antihistamines-Ex. Orphenadrine, Promethazine	
2	d	**Explain etiopathogenesis and clinical manifestation of GERD.** **Marking scheme-(etiopathogenesis 1.5M and clinical manifestations 1.5 M)** **Answer: Etiopathogenesis of GERD.:** • Smoking, eating large meals or eating late at night • Eating certain foods such as fatty or fried foods. • Drinking certain beverages, such as alcohol or coffee. • Taking certain medications such as aspirin. Typical symptoms of gastro-oesophageal reflux disease are heartburn and regurgitation but gastro-oesophageal reflux disease has also been related to extra-oesophageal manifestations, such as asthma, chronic cough and laryngitis. The pathogenesis of gastro-oesophageal reflux disease is multifactorial, involving transient lower esophageal sphincter relaxations and other lower esophageal sphincter pressure abnormalities. As a result, reflux of acid, bile, pepsin and pancreatic enzymes occur, leading to esophageal mucosal injury. The mechanisms involved in the pathogenesis of GERD are multiple and include: a) Motor abnormalities, such as impaired lower esophageal sphincter (LES) resting tone, transient LES relaxations (TLESR), impaired esophageal acid clearance and delayed gastric emptying, b) Anatomical factors, such as hiatal hernia. c) Visceral hypersensitivity.	**3M**

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d) Impaired mucosal resistance.

Clinical manifestations of GERD:

The most common symptoms of GERD include:

i)Heartburn (acid indigestion)

ii)Burning chest pain that starts behind the breastbone and moves upward to neck and throat

iii)Feeling like food is coming back into the mouth, leaving an acid or bitter taste.

iv)Pain in stomach, Nausea,

v)Bad breath, Trouble breathing,

vi)A hard time swallowing, Vomiting,

vii)Wearing away of tooth enamel,

viii)A lump in the throat,

ix)A lingering cough

x)Laryngitis,

xi)Sleep problems.

2	e	**Write about Management of Megaloblastic Anaemia.**	3M

Marking scheme-(Non Pharmacological management 1.5 M and Pharmacological management 1.5 M)

Answer: Pharmacological Management of Megaloblastic Anaemia:

A) Treatment of megaloblastic anaemia caused by Vitamin B12 deficiency:

Cobalamin Deficiency Treatment: Lifelong regular cobalamin injections are necessary for patients who have developed cobalamin deficiency. Hydroxycobalamin and Cyanocobalamin are the most common forms used for the treatment and Oral medications are also available for treatment.

Prophylactic dose: 3-10 µg/day orally in those at risk of developing deficiency.

B) Treatment of megaloblastic anaemia caused by folate deficiency:

Folate Deficiency Treatment: Oral doses of 5-15 mg of folic acid daily are sufficient. The duration of treatment depends on the underlying disease. It is usually necessary to treat for about 4 months. Patients who take anti- folate drugs are given folinic acid, a reduced form of folate.

Oral folic acid: therapeutic 2 to 5 mg/day, prophylactic 0.5 mg/day.

In acutely ill patients, therapy may be started with injection of folic acid 5 mg/day. Routine folate supplementation (1 mg/day) is recommended during pregnancy.

Non-Pharmacological Management of Megaloblastic Anaemia:

There are several ways to reduce your risk of megaloblastic anemia:

- Try to eat a balanced diet that emphasizes foods containing vitamin B12 and vitamin B9.
- Drink alcohol in moderation. Regular alcohol consumption may affect your digestive system and keep your body from absorbing vitamin B12.

Foods with vitamin B12

- Animal food products: Red meat, fish, poultry, eggs, milk and other dairy products all contain vitamin B12.
- Fortified foods: Fortified foods are foods that have certain vitamins and nutrients added to them that they don't naturally have. Fortified foods include certain breakfast cereals, nutritional yeast, plant milk and certain bread. Be sure to check the food label (nutritional facts) to confirm the food is fortified with vitamin B12.

Foods with vitamin B9:

- Vegetables: Dark green leafy vegetables, peas, beans and legumes.
- Fruits: Citrus fruits.
- Animal food products: Liver, seafood, meat and poultry.
- Eggs and dairy.

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		• Fortified foods: Certain breads, flour, pasta, rice and cereal contain folic acid. Be sure to check the food label (nutritional facts) to confirm the food is fortified with vitamin B9.	
2	f	**Write pharmacological and non pharmacological management of malaria.** **Marking scheme-(Pharmacological mag. 2M and non pharmacological mag.1 M)** **Answer: Pharmacological management of** malaria: These are the drugs which are used for Prophylaxis, treatment and prevention of relapse of Malaria. Antimalarial drugs are classified as follows: • Chloroquine phosphate: Chloroquine is the preferred treatment for any parasite that is sensitive to the drug. But in many parts of the world, parasites are resistant to chloroquine, and the drug is no longer an effective treatment. • Artemisinin-based combination therapies (ACTs): ACT is a combination of two or more drugs that work against the malaria parasite in different ways. This is usually the preferred treatment for chloroquine-resistant malaria. Examples include artemether-lumefantrine (Coartem) and artesunate-mefloquine. Other common antimalarial drugs include: • Atovaquone-proguanil (Malarone) • Quinine sulfate (Qualaquin) with doxycycline (Oracea, Vibramycin, others) • Primaquine phosphate **or** 1. Quinoline Derivatives: a. Cinchona Alkaloids:Ex. Quinine, Quinidine. b. 4-amino Quinoline:Ex. Chloroquine, Amodiaquine. c. 8-aminoQuinoline:Ex. Primaquine, Bulaquine. d. Quinoline Methanol: Ex.Mefloquine. 2. Biguanide: Ex.Proguanil, Chloroquine, Chlorproguanil. 3. Diaminopyrimidines or Pyrimidine Derivatives: Ex.Pyrimethamine, Trimethoprim.	3M

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		4. Sulfonamides and Sulfone:Ex.Sulfadoxine, Dapsone, Sulfamethpyrazine. 5. Antibiotics or Tetracyclines:Ex.Tetracyclines, Doxycycline. 6. Artemisinin Derivatives:Ex.Artesunate, Artemether, Arteether. 7. Amino alcohols: Ex.Halofantrine, Lumefantrine. 8. Naphthoquinone: Ex.Atovaquone. 9. Mannich base:Ex.Pyronaridine. **Non-pharmacological Management of malaria:** • i)Administer Bark of Cinchona tree in any form as it contains Quinine. • ii)Early diagnosis through examination of blood smears. • iii) Give immediate notification to health authorities. • iv) Take hygienic measures like good drainage. • v)Prevent stagnation of water where mosquitoes breed. • vi) Destruction of mosquito can be done by spraying DDT or Kerosene. • vii) Prevent mosquito bite by rubbing mosquito repellents or using Mosquito nets. • viii) Health Education in Society is a must. • ix) Wear full sleeves, socks etc. • x) Fit wire gauze in windows.	
2	**g**	**Write etiopathogenesis and clinical manifestation of osteoarthritis.** **Marking scheme-(etiopathogenesis (etiology + pathophysiology =2 marks and clinical manifestation = 1 mark)** Answer: Etiology Of osteoarthritis: 1. Old age: The risk of developing osteoarthritis increases. 2. Gender: Woman are more likely to develop osteoarthritis 3. Joint injuries: Such as those which occur while playing sports or have got from an accident can increase a person's risk to develop osteoarthritis. Some injuries which a person has got long back and have healed apparently, can also increase his risk of developing osteoarthritis later in life. 4. Activities which cause repeated Stress on the joint: If your job demands you to perform activities putting stress on joints again and again or if you are into a sport	**3M**

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which places repetitive stress on a joint, there is every possibility that the concerned joint may eventually develop osteoarthritis.

5. Obesity: obesity or increased weight adds stress on the weight bearing joints such as hips and knees. Further, fat tissue produces proteins that can cause harmful inflammation in and around the joints. More you weigh the greater is your like to develop osteoarthritis.
6. Metabolic disorders: Some metabolic disorder such as diabetes mellitus and hemochromatosis
7. Bone Deformities: Some people are born with malformed joints or defective cartilage.

Pathophysiology of osteoarthritis :

- Osteoarthritis (OA) is primarily a disease of cartilage commonly begins with damage to articular (joint) cartilage.
- There is a failure of the chondrocyte to maintain proper balance between cartilage formation and destruction.
- In osteoarthritis, the cartilage in a joint becomes rigid, loses its elasticity and begins to break down.
- Over time, the cartilage may lose its ability to act as a shock absorber.
- If the condition worsens, the bones get exposed and could rub against each other.
- Further, bones may break down and develop growths called spurs. With the changes in cartilage and subchondral bone (bone underneath cartilage in a joint) local inflammatory changes and pathologic changes occur in the joint capsule and synovium.
- Pain in osteoarthritis may result from distension of the synovial capsule by increased joint fluid, microfracture, periosteal irritation, or damage to ligaments, synovium (synovial membrane), or the meniscus
- Common causes for osteoarthritis include inflammation, mechanical forces, and bio proinflammatory mediators and proteases.

Clinical manifestation of osteoarthritis: Osteoarthritis symptoms often develop slowly and worsen over time. Symptoms of Osteoarthritis include:

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		1. Joint Pain: Affected joints might hurt during or after movement. 2. Stiffness: Joint stiffness might be most noticeable upon awakening or after being inactive. 3. Tenderness: The joint might feel tender when applying light pressure to or near it. 4. Loss of Flexibility: The person is not able to move the joint through its full range of motion. 5. Grating Sensation: Grating sensation and hear popping or crackling in joints. 6. Bone Spurs: Hard lumps occur around the affected joint. 7. Swelling: Soft tissue inflammation around the joint.	
2	h	**Explain in detail about clinical manifestation of anxiety.** **Marking scheme-(0.5 X 6 = 3 M)** **Answer: Clinical manifestation:** Common anxiety symptoms includes: Physical symptoms: • Cold or sweaty hands. • Dry mouth. • Heart palpitations. • Nausea. • Numbness or tingling in hands or feet. • Muscle tension. • Shortness of breath. Mental symptoms: • Feeling panic, fear and uneasiness. • Nightmares. • Repeated thoughts or flashbacks of traumatic experiences. • Uncontrollable, obsessive thoughts. Behavioral symptoms: • Inability to be still and calm. • Ritualistic behaviors, such as washing hands repeatedly. • Trouble sleeping.	3M

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OR

Clinical manifestations

Apart from the psychological symptoms of apprehension and fear, individuals complain of somatic symptoms like headaches, palpitations, shortness of breath, dysphagia, gastrointestinal disturbances, feeling nervous, sweating, feeling weak or tired, trouble concentrating, trouble sleeping, and tremors.

Generalized anxiety disorder (GAD): The main feature is excessive and unrealistic worry about several life situations that have been present for 6 months or longer. This fear is accompanied by symptoms like restlessness, fatigue, difficulty concentrating, irritability, muscle tension, and disturbed sleep. Many patients also experience the somatic symptoms discussed above.

Panic disorder (PD): In this condition, the patient has recurrent, unexpected attacks of overwhelming anxiety accompanied by severe physical symptoms like hyperventilation and sympathetic nervous system activity. Many patients with PD also develop symptoms of agoraphobia (fear of having a panic attack where help may be unavailable), for example, being in places away from home, being in crowds, being on bridges, and travelling in a bus, train, or car.

Obsessive-compulsive disorder (OCD): It is a condition characterized by recurrent obsessions and compulsions that cause significant distress and interfere with normal social occupational functioning. Obsessions are persistent ideas, thoughts, or images that are distressing and senseless. Examples of obsessions are recurrent thoughts of harming a loved one, or recurrent thoughts of contamination. Compulsions are repetitive, intentional behaviours performed in response to an obsession. Examples of compulsions are repetitive hand washing, counting, checking, etc.

Post-traumatic stress disorder (PTSD): PTSD may develop in response to a stressful event or situation of an exceptionally threatening nature. Causes include natural or human disasters, war, serious accidents, witnessing the violent deaths of others, being the victim of sexual abuse, rape, torture, terror, or being kidnapped. Such individuals experience flashbacks, insomnia, avoidance of activities that remind them of the incident, and intense anxiety.

Social phobia: It is characterised by a persistent fear of social or performance situations in which embarrassment may occur. Such individuals, when exposed to social or performance situations, experience an immediate anxiety response (palpitations, tremors,

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		sweating, gastrointestinal discomfort, diarrhoea, muscle tension, blushing, and confusion). **Some other phobias** include agarophobia (fear of open spaces), claustrophobia (fear of enclosed spaces), and arachnophobia (fear of spiders). Children are phobic about the dark and ghosts.	
2	i	**What is conjunctivitis? Write about its management.** **Marking scheme-(What is conjunctivities 0.5 M, pharmacological management 1.5M and non pharmacological management 1M)** **Answer: Conjunctivitis** is an inflammation or infection of the transparent membrane (conjunctiva) that lines the eyelid and covers the white part of your eyeball. When small blood vessels in the conjunctiva become inflamed, they're more visible. This is what causes the white part of eyes to appear reddish or pink. It is also known as "Pink eye". **Pharmacological Treatment of conjunctivitis:** **1) Viral Conjunctivitis:** Most cases of viral conjunctivitis are mild. The infection will usually clear up in 7 to 14 days without treatment and without any long-term consequences. However, in some cases, viral conjunctivitis can take 2 to 3 weeks or more to clear up. A doctor can prescribe antiviral medication to treat more serious forms of conjunctivitis. For example, conjunctivitis caused by herpes simplex virus or varicella-zoster virus. Antibiotics will not improve viral conjunctivitis, these drugs are not effective against viruses. Topical antivirals such as 1% trifluorothymidine can be given 5 times a day for 7 to 10 days. **2) Bacterial Conjunctivitis:** Antibiotics may help shorten the length of infection, reduce complications, and reduce the spread to others. Antibiotics may be necessary in the following cases:-With discharge (pus) -When conjunctivitis occurs in people whose immune system is compromised -When certain bacteria are suspected • Fluoroquinolones: • 2nd generation: Ciprofloxacin 0.3% drops or ointment, or Ofloxacin 0.3% drops	3M

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- 3rd generation: Levofloxacin 0.5% drops
- 4th generation: Moxifloxacin 0.5% drops, Gatifloxacin 0.5% drops, or Besifloxacin 0.6% drops

- Aminoglycosides:
  - Tobramycin 0.3% drops
  - Gentamicin 0.3% drops
- Macrolides:
  - Erythromycin 0.5% ointment
  - Azithromycin 1% solution
- Other
  - Bacitracin ointment
  - Bacitracin/Polymixin B ointment
  - Neomycin/Polymixin B/Bacitracin
  - Neomycin/Polymixin B/gramicidin
  - Polymixin B/Trimethoprim
  - Sulfacetamide
  - Chloramphenicol.

3) Allergic Conjunctivitis: If the irritation is allergic conjunctivitis, your doctor may prescribe one of many different types of eyedrops for people with allergies. These may include medications that help control allergic reactions, such as antihistamines and mast cell stabilizers, or drugs that help control inflammation, such as decongestants, steroids and anti-inflammatory drops. Over-the-counter eye drops that contain antihistamines and anti- inflammatory medications also may be effective. e.g., levocabastine, emedastine difumarate

Non-Pharmacological Treatment of conjunctivitis: Non-pharmacological treatment can be done by lifestyle modifications and home remedies:

1. Apply a compress to the eyes.
2. Try eye drops.
3. Stop wearing contact lenses.
4. Reduce exposure to light.
5. Prevent rubbing on the eyes.

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		6. Clean the eyes by using sterile water and cotton swabs from inner canthus to outer canthus. 7. Use of dark sunglasses is advised, in presence of photophobia.	
2	j	**Explain various mechanisms involved in resistance to antimicrobial agent.** **(Marking scheme- any 3 mechanisms 3M)** **Answer:** Antimicrobial resistance mechanisms fall into four main categories: i) Limiting uptake of a drug ii) Modifying a drug target iii) Inactivating a drug iv) Active drug efflux. Intrinsic resistance may make use of limiting uptake, drug inactivation, and drug efflux. Acquired resistance mechanisms used may be drug target modification, drug inactivation and drug efflux. Because of differences in structure, there is variation in the types of mechanisms used by gram-negative bacteria versus gram-positive bacteria. Gram-negative bacteria make use of all four main mechanisms, whereas gram-positive bacteria less commonly use limiting the uptake of a drug (don't have an LPS outer membrane), and don't have the capacity for certain types of drug efflux mechanisms. **i)Limiting Drug Uptake:** There is a natural difference in the ability of bacteria to limit the uptake of antimicrobial agents. The structure and functions of the LPS layer in gram-negative bacteria provide a barrier to certain types of molecules. This gives those bacteria innate resistance to certain groups of large antimicrobial agents. The mycobacteria have an outer membrane that has a high lipid content, so hydrophobic drugs such as rifampicin and fluoroquinolones have easier access to the cell, but hydrophilic drugs have limited access **ii) Modification of Drug Targets:** There are multiple components in the bacterial cell that may be targets of antimicrobial agents, and there are just as many targets that may be modified by the bacteria to enable resistance to those drugs. One mechanism of resistance to the B-lactam drugs used almost exclusively by gram- positive bacteria is via alterations in the structure and/or number of PBPs (penicillin-binding proteins).	3M

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		iii) Drug Inactivation: There are two main ways in which bacteria inactivate drugs, by actual degradation of the drug or by transfer of a chemical group to the drug. The ß-lactamases are a very large group of drug hydrolyzing enzymes. Another drug that can be inactivated by hydrolyzation is tetracycline, via the tetX gene. Drug inactivation by transfer of a chemical group to the drug most commonly uses the transfer of acetyl, phosphoryl and adenyl groups. There are a large number of transferases that have been identified. Acetylation is the most diversely used mechanism and is known to be used against aminoglycosides, chloramphenicol, streptogramins and fluoroquinolones. Phosphorylation and adenylation are known to be used primarily against aminoglycosides.. **iv) Drug Efflux:** Bacteria possess chromosomally encoded genes for efflux pumps. Some are expressed constitutively, and others are induced or overexpressed (high-level resistance is usually via a mutation that modifies the transport channel) under certain environmental stimuli or when a suitable substrate is present. The efflux pumps function primarily to rid the bacterial cell of toxic substances, and many of these pumps will transport a large variety of compounds (multi-drug [MDR] efflux pumps). The resistance capability of many of these pumps is influenced by what carbon source is available.	
2	**k**	**Write etiopathogenesis and clinical manifestation of dysmenorrhea.** **(etiopathogenesis etiology + pathophysiology = 2 Marks and clinical manifestations 1 mark)** **Answer:** **Etiology of Dysmenorrhoea:** **1. Causes of Primary Dysmenorrhea:** During the normal menstrual cycle, the uterus contracts to help expel its lining. Prostaglandin involved in pain and inflammation trigger the uterine muscle contractions. Increased prostaglandins production by the endometrium in an ovulatory cycle causes contraction of the uterus that leads to more-severe menstrual cramps. **2. Causes of Secondary Dysmenorrhea:** Menstrual cramps can be caused by:	**3M**

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1. Endometriosis: It is a disorder in which the tissue that normally lines the uterus grows outside the uterus, most commonly on fallopian tubes, ovaries or the tissue lining the pelvis.

2. Uterine Fibroids: These are noncancerous cells that grow in the wall of the uterus and can cause pain. It can develop during a woman's childbearing years.

3. Adenomyosis: It occurs when the tissue that normally lines the uterus begins to grow into the muscular wall of the uterus.

4. Pelvic Inflammatory Disease: This infection of the female reproductive organs is usually caused by sexually transmitted bacteria.

5. Cervical Stenosis: In some women, the opening of the cervix is small enough to impede menstrual flow, causing a painful increase of pressure within the uterus.

6. Other Risk Factors: There are some other risk factors that can cause menstrual cramps.

These are:

1. Young age
2. Early puberty at age 11 or younger
3. Heavy bleeding during periods (menorrhagia)
4. Irregular menstrual bleeding (metrorrhagia)
5. Family history of menstrual cramps (dysmenorrhea)
6. Smoking
7. Congenital uterine anomaly
8. Intrauterine devices

Pathophysiology of Dysmenorrhea:

- Dysmenorrhea is attributed to excessive endometrial prostaglandin production.
- Prostaglandin is made within the lining of the uterus or endometrium. On the first day of the menstrual period, the levels are very high. Women with dysmenorrhea

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may have up-regulated cyclo-oxygenase (COX) enzyme activity, which. contributes to increased synthesis of prostaglandins.

- Women with painful periods produce 10 times as much prostaglandin F (PGF2a), a potent myometrial stimulant and vasoconstrictor, as asymptomatic women.
- Elevated levels of prostaglandins (especially PGF2 and PGE2) cause uterine hypercontractility, decreased blood flow to the uterus, and increased nerve hypersensitivity, thus resulting in pain.
- The pain often radiates into the groin and may be accompanied by backache, anorexia, vomiting, diarrhea, syncope, and headache; these symptoms are caused by the entrance of prostaglandins and prostaglandin metabolites into the systemic circulation.
- Prostaglandins are primarily released during the first 48 hours of menstruation, when symptoms are the most intense; but the pain usually persists for the first 1 to 3 days of the menstrual flow.
- As menstruation continues and the lining of the uterus is shed, the levels decrease. Pain usually decreases as the levels of prostaglandins decrease.

Clinical manifestation of Dysmenorrhea:

1. Pain in the lower abdomen that can spread to the lower back and legs.
2. Pain that is gripping or experienced as a constant ache, or a combination of both.
3. The pain starts when the period starts, or earlier.
4. The first 24 hours may be the most painful.
5. Clots may be passed in the menstrual blood.
6. Nausea and Vomiting.
7. Headaches.
8. Digestive problems, such as Diarrhoea or Constipation.
9. Fainting.
10. Premenstrual symptoms, such as tender breasts and a swollen abdomen, which may

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		continue throughout the period. 11. Pain continues after the first 24 hours (this tends to subside after two or three days).	
3		**Attempt the following**	12 M
3	a	**Define essential medicines** **Answer :** Essential medicines are those that satisfy the priority health care needs of the majority of the population and therefore should be available at all the times in adequate amounts and appropriate dosage forms. OR Essential medicines, as defined by the World Health Organization (WHO), are those that satisfy the priority healthcare needs of the population.	1M
3	b	**Define Hypertension** **Answer :**Hypertension is defined as abnormally high blood pressure (more than 120/80 mmHg) in the arteries. Persistent increase in systemic arterial blood pressure is known as hypertension.	1M
3	c	**The condition in which there is inadequate supply of oxygen is called as** **Answer:** Ischemia	1M
3	d	**Identify the chronic inflammatory disease related to the respiratory system.** **Answer:**iv) Asthma	1M
3	e	**What is the full form of COPD** **Answer :**Chronic obstructive pulmonary disease.	1M
3	f	**Diabetes Mellitus caused during pregnancy is called .....diabetes.** **Answer:** Gestational Diabetes	1M
3	g	**Define Parkinson's disease.** **Answer :**It is a chronic, progressive, motor disorder due to degeneration of dopaminergic neurons in the substantia nigra which result in dopamine deficiency and is characterized by rigidity, tremor of resting muscles, slowness of movement and postural instability.	1M

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3	h	**What is Alzheimer's disease?** **Answer :**Alzheimer's disease is an irreversible progressive neurodegenerative brain disorder that results in progressive memory loss, impaired thinking ,disorientation and changes in personality and mood.	1M
3	i	**Name 2 drugs used in treatment of inflammatory Bowel diseases.** **Answer:** Any 2 drugs • **Anti-inflammatory drugs** - corticosteroids -Prednisolone ,Budesonide Aminosalicylates, such as mesalamine, sulfasalazine.,osalazine etc • **Immune system suppressors** - azathioprine, mercaptopurine and methotrexate. • **Biologics** - infliximab, adalimumab, • **Antibiotics** -ciprofloxacin and metronidazole.	1M
3	j	**In which condition gastric content flows back into the esophagus?** **Answer:**iii) GERD	1M
3	k	**The most common cause of megaloblastic anemia is Vitamin B-9 and ..... deficiency.** **Answer: Vitamin B12**	1M
3	l	**Which organism causes gonorrhoea?** **Answer:** Neisseria gonorrhea	1M
3	m	**What is the most severe stage of HIV infection called?** **Answer:** AIDS (acquired immunodeficiency syndrome)	1M
3	n	**n) What is the definition of Eczema?** **Answer :** Eczema is a very common skin condition that causes itchy, red, dry, irritated and inflamed skin and leads to swelling, cracking or scaliness". **OR** Eczema is an inflammatory condition of the skin characterized by redness, itching, and oozing vesicular lesions which become scaly, crusted, or hardened.	1M
3	o	**o) Name the causative organism of scabies.** **Answer :** Sarcoptes scabiei	1M
3	p	**p) Auspitz sign refers to** **Answer :**iv) Psoriasis	1M

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3	q	**Name 2 classes of drugs used in the therapy of depression.** **Answer :** Any 2 drugs • **Selective Serotonin reuptake inhibitors (SSRI):**Fluoxetine, Fluvoxamine , Sertraline . • **Serotonin and norepinephrine reuptake inhibitors (SNRIs)** Venlafazine, Desvenlafazine • **Norepinephrine-dopamine reuptake inhibitors (NDRIs)** Bupropion (Wellbutrin) • **MAO inhibitors:** Phenelzine and Tranylcypromine, • **Tricyclic antidepressants (TCAs):**Imipramine, Amitryptiline,	1M
3	r	**Claustrophobia fear of** **Answer :** Fear of tight or crowded spaces	1M
3	s	**Hyperandrogenism is associated with** **Answer:** ii) Polycystic Ovary Syndrome	1M
3	t	**t) Combined oral contraceptives preparation contain progestin and.....** **Answer :**Estrogen	1M