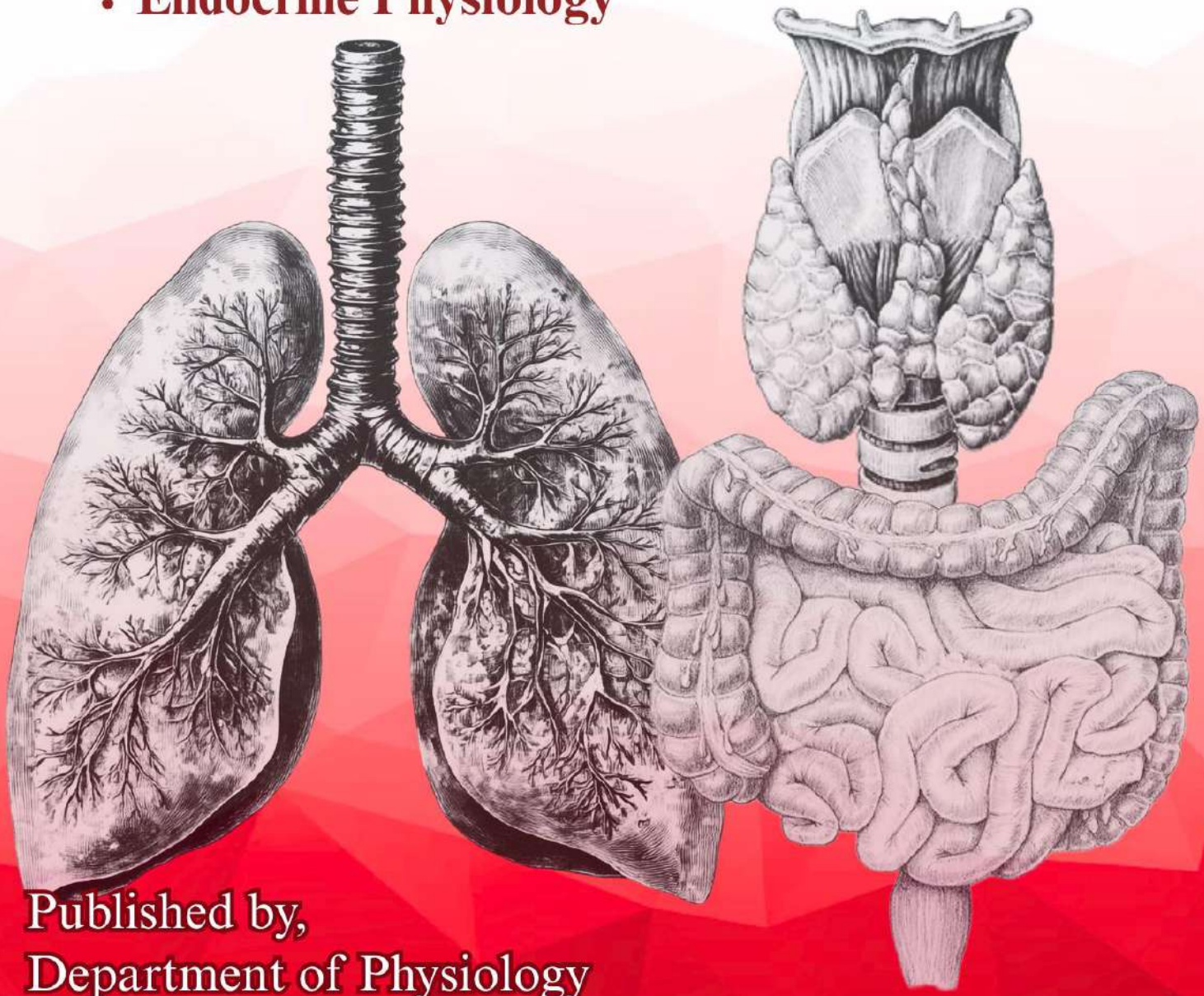
MASTERING PHYSIOLOGY
A Comprehensive Guide to MCQs, Essays, and OSPEs
Part II

Uva Wellassa University

Semester 2

- Respiratory Physiology
- Gastrointestinal Physiology
- Endocrine Physiology



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Authored by:
Department of Physiology
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Contributors to Excellence

A concept of: Snr. Prof. Muditha Vidanapathirana, Dean, Faculty of Medicine

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Authors:

Dr. Bandusiri Rathnayake
Consultant Physician,
MBBS (Ruhuna), MD (Medicine), FCCP (SL), FACP (USA)
Senior Lecturer (Grade I), Department of Physiology

Dr. K.P. Anuththara Sewwandi
MBBS (Peradeniya)
Lecturer (Probationary), Department of Physiology

Dr. Shamod Abeysekera
MBBS (Peradeniya)
Temporary Demonstrator, Department of Physiology

Dr. Kasun Dissanayake
MBBS (Sabaragamuwa)
Temporary Demonstrator, Department of Physiology

Snr. Prof. Muditha Vidanapathirana
Head of the Department of Forensic Medicine

Cover design by
Dr. Shamod Abeysekera, Temporary Demonstrator, Department of Physiology

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Message from the Vice Chancellor

Dear Students,

It is with immense pleasure that I offer my warm congratulations and genuine appreciation to the Faculty of Medicine, Uva Wellassa University of Sri Lanka, including Senior Professor Muditha Vidanapathirana, Dean of the Faculty of Medicine, for this outstanding initiative in publishing "Mastering Physiology – Part II: A Comprehensive Guide to MCQs, Essays & OSPEs" successfully.

This book stands as an invaluable resource, providing substantial benefits not only for MBBS undergraduates of Uva Wellassa University of Sri Lanka, but also for medical students nationwide overall.

I encourage you to fully utilize the excellent resources created by the Faculty of Medicine to strengthen your academic journey. Resources like this guide can make your studies more focused, efficient, and truly rewarding overall experience.

Wishing you all the very best in your academic and professional endeavors.

Senior Professor Kolitha B. Wijesekara
B.Sc. (Hon.) (Peradeniya), M.Phil. (Peradeniya),
Dr. Sc. Agr. (Gottingen, Germany), M.Biol.
Vice Chancellor
Uva Wellassa University of Sri Lanka
06.01.2026

PHYSIOLOGY MCQ (T/F & SBA) QUESTIONS

1. Respiratory Physiology

2. Gastrointestinal Physiology

3. Endocrine Physiology

Respiratory Physiology

Question (1)
T/F regarding the mechanism of breathing?
(A) During quiet inspiration, contraction of the diaphragm increases vertical diameter of the thoracic cavity.
(B) Intrapleural pressure becomes less negative during inspiration.
(C) External intercostal muscles help elevate the ribs during inspiration.
(D) Forced expiration is an active process involving abdominal and internal intercostal muscles.
(E) Transpulmonary pressure is defined as the difference between alveolar pressure and atmospheric pressure.

Question (2)
T/F regarding bronchial innervation?
(A) Parasympathetic stimulation causes bronchoconstriction and increased bronchial secretions.
(B) Sympathetic stimulation via β2 receptors leads to bronchodilation and reduced secretion.
(C) Substance P acts as a vasoconstrictor and inhibits bronchial secretions.
(D) α1 receptor stimulation decreases bronchial gland secretion.
(E) Vagal stimulation can cause reflex bronchoconstriction.

Question (3)
T/F regarding lung compliance?
(A) Lung compliance is defined as the change in lung volume per unit change in transpulmonary pressure.
(B) In pulmonary fibrosis, lung compliance is increased.
(C) Surfactant increases lung compliance by reducing alveolar surface tension.
(D) Lung compliance is highest at very high lung volumes.
(E) Emphysema is associated with increased lung compliance.

Question (4)
T/F regarding lung volumes and capacities?
(A) Functional Residual Capacity (FRC) is the volume of air remaining in the lungs after a normal tidal expiration.
(B) Inspiratory Reserve Volume (IRV) can be measured using a simple spirometer
(C) Residual Volume (RV) increases in obstructive lung diseases.
(D) Vital Capacity (VC) includes Residual Volume.
(E) Total Lung Capacity (TLC) is the sum of all lung volumes.

Question (5)
T/F regarding FEV_1/FVC ratio in restrictive and obstructive lung diseases?
(A) In obstructive lung diseases, the FEV_1/FVC ratio is decreased due to disproportionately reduced FEV_1.
(B) In restrictive lung diseases, both FEV_1 and FVC are reduced, but the ratio is usually normal or increased.
(C) A decreased FEV_1/FVC ratio is diagnostic of restrictive lung disease.

(D) COPD typically presents with a reduced FEV_1 and a relatively preserved FVC.
(E) Pulmonary fibrosis may present with a normal or increased FEV_1/FVC ratio despite reduced lung volumes.

Question (6)
T/F regarding spirometry findings of obstructive lung diseases?
(A) Asthma is characterized by reversible airway obstruction demonstrated by spirometry.
(B) COPD shows significant improvement in FEV_1 after bronchodilator use.
(C) Peak Expiratory Flow Rate (PEFR) is typically reduced during acute asthma exacerbations.
(D) FEV_1/FVC ratio is reduced in both asthma and COPD.
(E) Reversibility testing involves measuring FEV_1 response to corticosteroids.

Question (7)
T/F regarding alveolar and pulmonary ventilation?
(A) Pulmonary ventilation refers to the total volume of air entering and leaving the lungs per minute.
(B) Alveolar ventilation is always equal to pulmonary ventilation.
(C) An increase in anatomical dead space reduces alveolar ventilation if tidal volume remains constant.
(D) Physiological dead space includes both anatomical and alveolar dead space.
(E) Rapid shallow breathing decreases alveolar ventilation more than slow deep breathing.

Question (8)
T/F regarding respiratory regulation?
(A) Central chemoreceptors are primarily sensitive to changes in arterial PO_2.
(B) Peripheral chemoreceptors respond rapidly to hypoxemia.
(C) The pre-Bötzinger complex is involved in generating respiratory rhythm.
(D) Hypercapnia leads to increased firing of central chemoreceptors.
(E) Hering–Breuer inflation reflex plays a role in limiting tidal volume during quiet breathing.

Question (9)
T/F regarding non-chemical control of respiration, state whether the following statements?
(A) Proprioceptor activation in joints and muscles during exercise increases ventilation before measurable changes in PaO_2 or $PaCO_2$ occur.
(B) Baroreceptor stimulation in the carotid sinus and aortic arch during hypertension typically suppresses respiratory drive.
(C) Pulmonary stretch receptor activation during lung inflation initiates the Hering–Breuer inflation reflex.
(D) Sudden severe pain can transiently inhibit respiratory activity through higher brain centers.
(E) Cold receptor stimulation in the skin can reflexly increase respiratory rate via sympathetic activation.

Question (10)

T/F regarding the types of hypoxia and their examples?

(A) In hypoxic hypoxia, PaO_2 is reduced, as seen in high-altitude exposure.
(B) In anemic hypoxia, arterial oxygen content is reduced despite normal PaO_2, such as in carbon monoxide poisoning.
(C) Cyanide poisoning causes histotoxic hypoxia by blocking oxidative phosphorylation at the cytochrome oxidase level.
(D) Stagnant hypoxia occurs when oxygen delivery to tissues is reduced due to low cardiac output or circulatory obstruction.
(E) In histotoxic hypoxia, venous oxygen content is typically low due to increased tissue oxygen extraction.

Question (11)

A 62-year-old man with long-standing chronic obstructive pulmonary disease (COPD) presents for a routine follow-up. He appears stable and not in acute distress. His arterial blood gas (ABG) results are as follows:

pH: 7.36 (normal: 7.35–7.45)
$PaCO_2$: 55 mmHg (normal: 35–45 mmHg)
HCO_3^-: 30 mEq/L (normal: 22–26 mEq/L)
PaO_2: 68 mmHg (normal: 75–100 mmHg)

Which of the following is the most likely interpretation of his ABG findings?

(A) Metabolic alkalosis
(B) Uncompensated respiratory acidosis
(C) Compensated respiratory acidosis
(D) Acute respiratory alkalosis
(E) Combined metabolic and respiratory acidosis

Question (12)

A 25-year-old healthy man performs a spirometry as a part of a routine medical evaluation. Which of the following lung volumes cannot be measured accurately by spirometry?

(A) Expiratory reserve volume
(B) Inspiratory reserve volume
(C) Vital capacity
(D) Residual volume
(E) Inspiratory capacity

Question (13)

A 62-year-old man with long-standing COPD presents with worsening shortness of breath. His ABG shows PaO_2 of 48 mmHg. Chest X-ray reveals hyperinflated lungs and patchy areas of decreased ventilation. Which of the following mechanisms most likely contributes to the development of pulmonary hypertension in this patient?

A. Increased sympathetic stimulation leading to generalized pulmonary vasodilation.
B. Hypercapnia causing relaxation of pulmonary vascular smooth muscle.
C. Chronic hypoxia causing vasoconstriction of small pulmonary arteries.
D. Reduced red blood cell mass increasing blood viscosity.
E. Increased left atrial pressure causing reflex vasodilation in pulmonary vessels.

Question (14)

A 55-year-old man collapses at a shopping mall. Bystanders find him unresponsive and not breathing normally. You arrive first on the scene. Which of the following actions should you perform first?

(A) Deliver two rescue breaths.
(B) Begin chest compressions at a rate of 100–120 per minute.
(C) Check for a pulse for up to 10 seconds.
(D) Attach an automated external defibrillator (AED) immediately.
(E) Open the airway using the head-tilt–chin-lift maneuver.

Question (15)

A 68-year-old man with a history of chronic obstructive pulmonary disease (COPD) presents with increasing shortness of breath and confusion. On examination, he is using accessory muscles, has a respiratory rate of 32/min, SpO_2 82% on room air, and arterial blood gas shows:

pH: 7.25
$PaCO_2$: 65 mmHg
PaO_2: 55 mmHg
HCO_3^-: 28 mEq/L

Which of the following is the most appropriate next step in management?

(A) Start high-flow oxygen via non-rebreather mask at 15 L/min.
(B) Begin non-invasive positive pressure ventilation (NIPPV).
(C) Immediate endotracheal intubation and mechanical ventilation.
(D) Administer intravenous diuretics.
(E) Observe and repeat arterial blood gases in 2 hours.

Gastrointestinal Physiology

Question (16)
T/F regarding the physiology of gastric acid secretion?
(A) Gastric parietal cells secrete HCl through the action of the H^+/K^+ ATPase pump.
(B) Histamine stimulates acid secretion via H_2 receptors coupled to ↑ cAMP.
(C) Acetylcholine stimulates parietal cells through M_2 muscarinic receptors.
(D) Gastrin acts on CCK_2 receptors of enterochromaffin-like (ECL) cells, promoting histamine release.
(E) Somatostatin and prostaglandins inhibit gastric acid secretion.

Question (17)
T/F regarding the complications of total gastrectomy?
(A) Loss of intrinsic factor after total gastrectomy leads to vitamin B_{12} deficiency and megaloblastic anemia.
(B) Iron deficiency anemia is common due to loss of gastric acid, which normally facilitates iron absorption.
(C) Early dumping syndrome occurs due to rapid osmotic fluid shift into the intestine after a meal.
(D) Late dumping syndrome is related to reactive hypoglycemia from excessive insulin release.
(E) Gastric lipase deficiency has little effect on fat digestion, as pancreatic lipase compensates.

Question (18)
T/F regarding the exocrine pancreas and hormonal control of pancreatic secretion?
(A) Secretin stimulates secretion of bicarbonate-rich pancreatic juice from ductal cells.
(B) Cholecystokinin (CCK) primarily stimulates enzyme-rich secretion from pancreatic acinar cells.
(C) Acetylcholine acts on muscarinic receptors of pancreatic acinar cells to promote enzyme secretion.
(D) Secretin release is stimulated by acidic chyme entering the duodenum.
(E) CCK secretion is primarily triggered by carbohydrates in the duodenum.

Question (19)
T/F regarding the functions of the large intestine and related physiology?
(A) The large intestine plays a major role in absorption of nutrients like amino acids and glucose.
(B) The primary absorptive functions of the colon include water and electrolyte (Na^+, Cl^-) absorption.
(C) The gastrocolic reflex is mediated partly via parasympathetic pathways and is exaggerated in irritable bowel syndrome (IBS).
(D) Peyer's patches are prominent lymphoid aggregates found in the colon that play a key role in immune surveillance.
(E) Vitamin B12 supplementation is indicated in patients on long-term metformin therapy to prevent neuropathy and anemia.

Question (20)
T/F regarding the defecation reflex?
(A) The intrinsic defecation reflex is mediated entirely by the enteric nervous system.
(B) The parasympathetic defecation reflex arises from sacral spinal segments (S2–S4).
(C) Rectal distension relaxes the internal anal sphincter via the rectoanal inhibitory reflex.
(D) The external anal sphincter is under involuntary control through the autonomic nervous system.
(E) Voluntary contraction of abdominal muscles and relaxation of pelvic floor muscles facilitate defecation.

Question (21)
T/F regarding the vomiting reflex?
(A) The vomiting center is located in the medulla oblongata.
(B) The chemoreceptor trigger zone (CTZ) lies outside the blood–brain barrier.
(C) Vagus and sympathetic afferents from the gut can initiate vomiting.
(D) Glottis closes and soft palate is elevated during vomiting to prevent aspiration.
(E) The diaphragm and abdominal muscles contract to increase intra-abdominal pressure.

Question (22)
T/F regarding the development of peripheral stigmata in chronic alcoholic cirrhosis (CLCD)?
(A) Spider nevi result from dilated cutaneous capillaries due to impaired hepatic degradation of estrogens.
(B) Palmar erythema occurs due to hyperdynamic circulation and increased vasodilatory substances.
(C) Gynecomastia is due to decreased androgen metabolism and relative estrogen excess.
(D) Caput medusae develops from portosystemic collateral formation at the umbilicus due to portal hypertension.
(E) Testicular atrophy occurs from increased estrogen catabolism in the cirrhotic liver.

Question (23)
T/F regarding esophageal motility?
(A) The upper esophageal sphincter (UES) is composed of skeletal muscle and is under voluntary control.
(B) Peristalsis in the esophagus is initiated by smooth muscle contraction in the distal esophagus only.
(C) The lower esophageal sphincter (LES) exhibits tonic contraction at rest to prevent gastroesophageal reflux.
(D) Primary peristalsis is initiated by swallowing and involves sequential contraction of striated and smooth muscle.
(E) The enteric nervous system alone is sufficient to coordinate esophageal peristalsis without input from the central nervous system.

Question (24)
T/F regarding jaundice?

(A) Jaundice becomes clinically visible when serum bilirubin exceeds approximately 2–3 mg/dL.
(B) Hemolytic jaundice is usually associated with elevated direct (conjugated) bilirubin.
(C) Obstructive (post-hepatic) jaundice typically presents with dark urine and pale stools.
(D) In hepatocellular jaundice, both unconjugated and conjugated bilirubin levels can be elevated.
(E) Physiologic jaundice of the newborn is usually due to increased conjugation of bilirubin.

Question (25)
T/F regarding saliva secretion?
(A) Primary saliva secreted by acinar cells is isotonic with plasma, and it becomes hypotonic as it passes through the ductal system due to reabsorption of sodium and chloride.
(B) Sympathetic stimulation increases the protein concentration of saliva, making it thicker and more viscous.
(C) The ductal modification of saliva, including potassium secretion and bicarbonate exchange, is independent of flow rate.
(D) Salivary secretion exhibits a circadian rhythm, with maximal production during the daytime and minimal production at night.
(E) In Sjögren's syndrome, autoimmune destruction of salivary glands leads primarily to a loss of acinar cell function, reducing both fluid and enzyme secretion.

Question (26)
A 56-year-old woman undergoes a total pancreatectomy because of the presence of a tumor. Which of the following outcomes would not be expected after she recovers from the operation?
(A) Steatorrhea
(B) Hyperglycemia
(C) Metabolic acidosis
(D) Weight gain
(E) Decreased absorption of amino acids

Question (27)
A 52-year-old man presents with jaundice and dark urine. Investigations reveal elevated total bilirubin with direct hyperbilirubinemia. Which of the following is the most likely cause of his jaundice?
(A) Hemolytic anemia
(B) Acute viral hepatitis
(C) Gallstone obstructing the common bile duct
(D) Gilbert syndrome
(E) Crigler-Najjar syndrome

Question (28)
A 38-year-old woman presents with chronic heartburn and regurgitation. She asks why this happens. Which of the following is the most common underlying cause of gastroesophageal reflux disease (GERD)?

(A) Hiatal hernia
(B) Impaired lower esophageal sphincter (LES) tone
(C) Delayed gastric emptying
(D) Esophageal motility disorders
(E) Increased acid production by the stomach

Question (29)
A 32-year-old woman presents with bloating, abdominal cramps, and diarrhea after consuming dairy products. Laboratory testing shows normal pancreatic function. Which of the following best explains the physiological mechanism causing her symptoms?
(A) Impaired starch digestion due to pancreatic amylase deficiency
(B) Inability to hydrolyze lactose in the small intestine due to lactase deficiency
(C) Malabsorption of glucose due to defective SGLT1 transporters
(D) Impaired digestion of sucrose due to sucrase deficiency
(E) Inhibition of salivary amylase by gastric acid

Question (30)
A 45-year-old man presents with a 2-year history of progressive dysphagia for both solids and liquids, regurgitation of undigested food, and occasional chest pain. Barium swallow shows a dilated esophagus with a narrowed gastroesophageal junction (bird-beak appearance). Which of the following is the most likely underlying pathophysiology?
(A) Lower esophageal sphincter (LES) hypertonicity due to loss of inhibitory neurons in the myenteric plexus
(B) Increased acid secretion leading to esophageal inflammation and strictures
(C) Autoimmune destruction of striated muscle in the proximal esophagus
(D) Transient relaxation of the LES due to vagal nerve hyperactivity
(E) External compression of the esophagus by a mediastinal mass

Endocrine Physiology

Question (31)
T/F regarding hormones?
(A) A hormone is a chemical substance released into the bloodstream to act on distant target tissues.
(B) Hormonal signaling usually has a more prolonged duration of action than neural signaling.
(C) Hormones act only on cells located far from the site of secretion.
(D) Hormones require specific receptors on target cells to exert their effects.
(E) Hormonal and neural systems often interact to regulate the same physiological processes.

Question (32)
T/F regarding insulin?
(A) Half-life is about 5 minutes in humans.
(B) C peptide level is a marker of insulin production.
(C) GLUT 3 receptors in muscle and adipose tissues are stimulated by insulin.
(D) Causes increased transport of sodium ions into insulin-sensitive cells.
(E) Type 1 diabetes occurs due to absolute deficiency of insulin.

Question (33)
T/F regarding glucocorticoids?
(A) They mobilize fatty acids from adipose tissues.
(B) They decrease protein synthesis in the live.
(C) They decrease peripheral glucose utilization.
(D) They suppress the immune system.
(E) They stimulate gluconeogenesis.

Question (34)
T/F regarding aldosterone?
(A) It is the primary regulator of salt balance.
(B) It is secreted in the zona reticularis.
(C) It simulates sodium reabsorption and potassium secretion.
(D) Conn syndrome is characterized by hypoaldosteronism.
(E) Renin concentration is used to diagnose primary hyperaldosteronism.

Question (35)
T/F regarding thyroid hormones?
(A) The majority of circulating T3 is produced directly by the thyroid gland.
(B) T4 is more potent than T3 at the nuclear receptor level.
(C) Most thyroid hormone circulates bound to thyroxine-binding globulin (TBG), with only the free fraction being biologically active.
(D) TSH stimulates both increased synthesis of thyroid hormones and hypertrophy of thyroid follicular cells.
(E) High levels of iodine acutely stimulate thyroid hormone release.

Question (36)
T/F regarding calcium & phosphate metabolism?
(A) Parathyroid hormone (PTH) increases renal phosphate reabsorption in the proximal tubule.
(B) Vitamin D (calcitriol) increases intestinal absorption of both calcium and phosphate.
(C) Calcitonin is essential for maintaining normal serum calcium levels.
(D) Calcitriol stimulates osteoclast activity indirectly through osteoblast signaling.
(E) Chronic kidney disease commonly leads to hyperphosphatemia.

Question (37)
T/F regarding renin-angiotensin-aldosterone system (RAAS)?
(A) Renin release from the juxtaglomerular cells increases when renal perfusion pressure decreases.
(B) Angiotensin II causes efferent arteriolar constriction more than afferent arteriolar constriction.
(C) Angiotensin-converting enzyme (ACE) is found predominantly in the lung endothelial cells.
(D) Increased sympathetic nerve activity (β_1 stimulation) enhances renin secretion.
(E) The macula densa decreases renin release when it detects low sodium chloride delivery.

Question (38)
T/F regarding the physiology of hypothalamus?
(A) The posterior pituitary synthesizes oxytocin and vasopressin (ADH).
(B) Dopamine from the hypothalamus stimulates prolactin secretion.
(C) Thyrotropin-releasing hormone (TRH) stimulates both TSH and prolactin release.
(D) Oxytocin release is inhibited by cervical stretch during labor.
(E) Continuous high levels of GnRH increase LH and FSH secretion.

Question (39)
T/F regarding adrenal medulla?
(A) The adrenal medulla is functionally a modified sympathetic ganglion.
(B) Chromaffin cells secrete mostly norepinephrine under normal physiological conditions.
(C) Catecholamines increase glycogenolysis and lipolysis during stress.
(D) Dopamine is the major circulating hormone released by the adrenal medulla.
(E) Phenylethanolamine N-methyltransferase (PNMT) converts norepinephrine to epinephrine.

Question (40)
T/F regarding abnormalities of pituitary hormone secretion?
(A) Hypersecretion of growth hormone in adults leads to acromegaly, while in children it causes gigantism.
(B) Prolactin deficiency is a common cause of infertility in women and is usually caused by pituitary adenomas.
(C) Secondary hypothyroidism results from insufficient TSH secretion by the pituitary gland.
(D) Hypersecretion of ACTH from the pituitary (Cushing disease) typically causes hypokalemia and metabolic alkalosis due to excess cortisol.

(E) Pan-hypopituitarism involves selective deficiency of one pituitary hormone while other hormones remain normal.

Question (41)

Vitamin D deficiency causes defective calcification in bone matrix and the disease called rickets in children. Impairment of which of the following physiological effects of vitamin D is the main reason for this condition?

(A) Increasing synthetic activity of osteoblasts
(B) Increasing absorption of dietary calcium
(C) Decreasing the resorption of bones by osteoclasts
(D) Increasing excretion of phosphate in the kidneys
(E) Increasing reabsorption of calcium in the kidneys

Question (42)

A 28-year-old woman presents with weight loss, palpitations, heat intolerance, and tremors over the past 3 months. On examination, she has tachycardia, diffuse goiter, and exophthalmos. Laboratory studies show elevated free T4 and suppressed TSH. Which of the following mechanisms best explains her hyperthyroidism?

(A) Autoantibodies stimulating the TSH receptor on thyroid follicular cells
(B) Excess secretion of TSH from the anterior pituitary
(C) Autonomous thyroid nodule producing thyroid hormones independently of TSH
(D) Increased dietary iodine intake leading to excessive thyroid hormone synthesis
(E) Autoimmune destruction of thyroid tissue causing release of preformed hormones

Question (43)

A 50-year-old man presents with polyuria, polydipsia, and unexplained weight loss over several months. Laboratory tests reveal fasting plasma glucose of 180 mg/dL, HbA1c of 8.2%, and positive autoantibodies against pancreatic beta cells. Which of the following best describes the underlying pathophysiology of his condition?

(A) Insulin resistance in peripheral tissues with compensatory hyperinsulinemia
(B) Autoimmune destruction of pancreatic beta cells leading to insulin deficiency
(C) Excess hepatic glucose production due to glucagon overactivity without insulin deficiency
(D) Impaired renal glucose reabsorption causing glucosuria and secondary hyperglycemia
(E) Defective incretin hormone secretion leading to reduced postprandial insulin release

Question (44)

A 35-year-old woman presents with central obesity, purple abdominal striae, easy bruising, and muscle weakness. Laboratory tests reveal elevated serum cortisol with suppressed ACTH. Which of the following is the most likely source of her condition?

(A) Primary adrenal adenoma producing cortisol
(B) ACTH-secreting pituitary adenoma
(C) Ectopic ACTH-producing tumor
(D) Exogenous glucocorticoid use
(E) Congenital adrenal hyperplasia due to 21-hydroxylase deficiency

Question (45)

A 52-year-old man presents with muscle weakness and low blood pressure. Laboratory tests show hyponatremia, hyperkalemia, low plasma aldosterone, and elevated plasma renin activity. Which of the following is the most likely diagnosis?

(A) Primary hyperaldosteronism
(B) Secondary hyperaldosteronism
(C) Hyporeninemic hypoaldosteronism
(D) Cushing syndrome
(E) 17α-hydroxylase deficiency

PHYSIOLOGY MCQ (T/F & SBA) ANSWERS

Question (1)

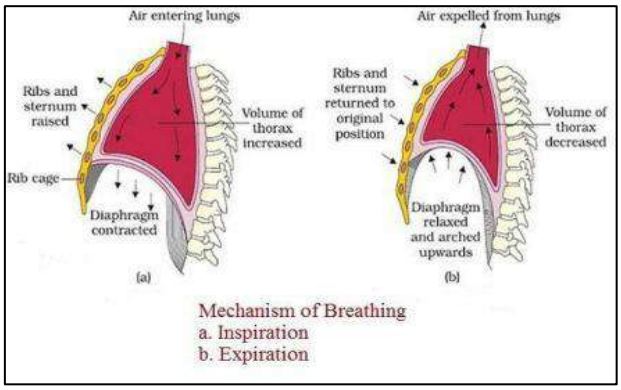
(A) **T** – Diaphragm contraction flattens its dome, increasing vertical thoracic space and lung volume.

(B) **F** – Intrapleural pressure becomes more negative during inspiration to draw air into the lungs.

(C) **T** – External intercostals elevate the ribs, increasing thoracic cavity dimensions.

(D) **T** – Forced expiration uses abdominal and internal intercostal muscles to push air out actively.

(E) **F** – Transpulmonary pressure = Alveolar pressure – Intrapleural pressure, not atmospheric pressure.



https://microbiologynotes.com/mechanism-of-breathing/

Question (2)

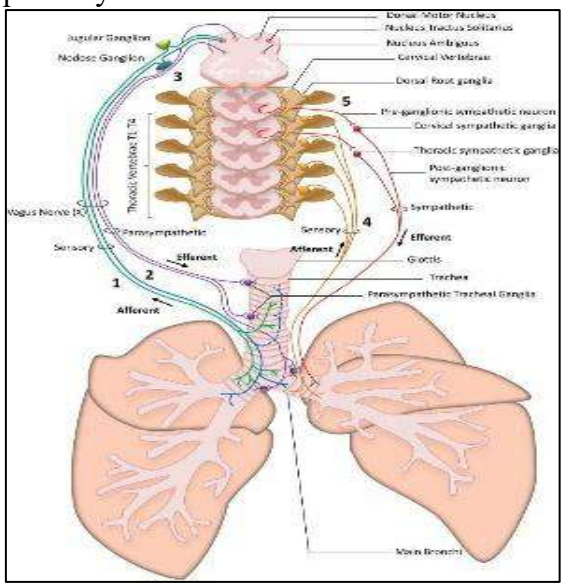
(A) **T** – Parasympathetic fibers (via vagus) release ACh, causing bronchoconstriction and stimulating mucus secretion.

(B) **T** – β2 activation relaxes smooth muscle and tends to reduce glandular secretion.

(C) **F** – Substance P is a neuropeptide that promotes secretion and can cause bronchoconstriction, not inhibition.

(D) **T** – α1 adrenergic receptor activation tends to reduce bronchial secretion through vasoconstriction of secretory glands.

(E) **T** – Vagal reflexes (e.g. due to irritants) trigger bronchoconstriction via parasympathetic pathways.



https://www.researchgate.net/figure/Schematic-representation-of-airway-innervation-The-airways-are-innervated-by-a-dual_fig1_235894786

Question (3)

(A) **T** – Compliance = ΔV/ΔP (volume change per unit pressure change across the lung).
(B) **F** – Fibrosis stiffens the lungs, decreasing compliance.
(C) **T** – Surfactant reduces surface tension, especially in smaller alveoli, thereby increasing compliance.
(D) **F** – At very high lung volumes, lungs become stiff and less distensible, so compliance is reduced.
(E) **T** – Loss of elastic tissue in emphysema makes lungs more distensible, increasing compliance.

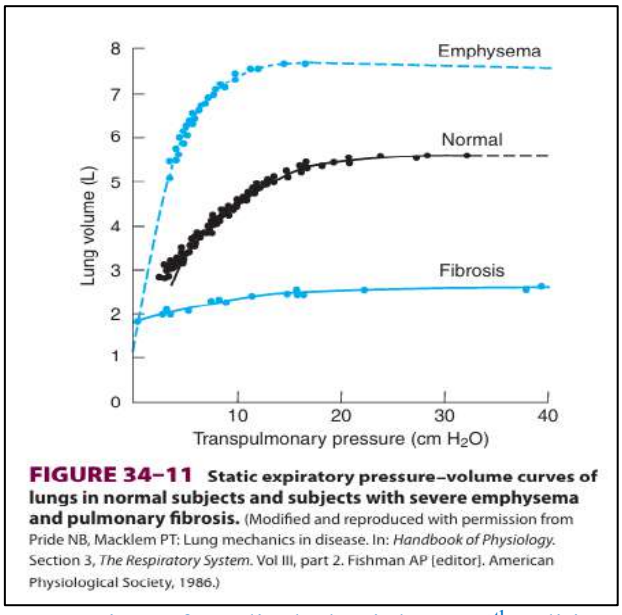


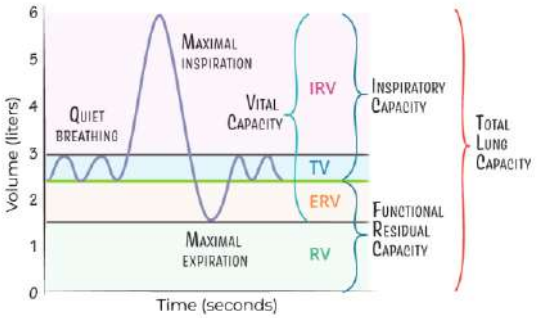
FIGURE 34–11 **Static expiratory pressure–volume curves of lungs in normal subjects and subjects with severe emphysema and pulmonary fibrosis.** (Modified and reproduced with permission from Pride NB, Macklem PT: Lung mechanics in disease. In: *Handbook of Physiology.* Section 3, *The Respiratory System.* Vol III, part 2. Fishman AP [editor]. American Physiological Society, 1986.)

Ganong's Review of Medical Physiology 24th Edition p. 631

Question (4)

(A) **T** – FRC = Expiratory Reserve Volume + Residual Volume; it remains after a normal expiration.
(B) **T** – IRV can be measured via spirometry during a maximal inspiratory effort.
(C) **T** – In obstructive diseases like emphysema, air trapping increases RV.
(D) **F** – VC = IRV + TV + ERV; it does not include RV.
(E) **T** – TLC = IRV + TV + ERV + RV; it is the total volume the lungs can hold.

Volumes and Capacities



Forced Expiratory Volume = Air (L) that can be forcibly expired after max. inspiration. FEV1 = FEV in 1 sec.

https://ditki.com/course/physiology/glossary/physiological-process/lung-volumes-capacities

Question (5)

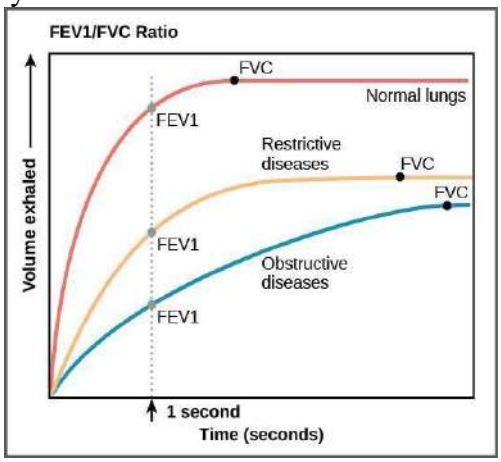
(A) **T** – Obstructive diseases (e.g., asthma, COPD) cause more reduction in FEV_1 than FVC, lowering the ratio.

(B) **T** – Restrictive diseases reduce both FEV_1 and FVC proportionally or FVC more, so ratio stays normal or increases.

(C) **F** – A decreased FEV_1/FVC ratio suggests obstructive, not restrictive, pathology.

(D) **T** – In COPD, FEV_1 drops significantly while FVC drops less, decreasing the ratio.

(E) **T** – In restrictive diseases like pulmonary fibrosis, lung volumes drop, but FEV_1/FVC ratio may remain normal or rise.



https://courses.lumenlearning.com/wm-biology2/chapter/the-work-of-breathing/

Question (6)

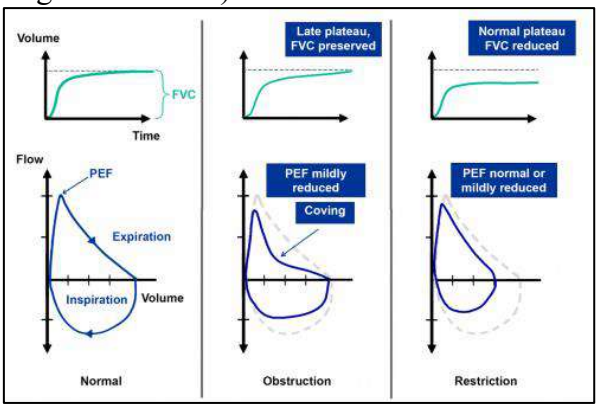
(A) **T** – Asthma shows reversible obstruction; post-bronchodilator FEV_1 increases ≥12% and 200 mL supports the diagnosis.

(B) **F** – COPD shows limited reversibility; improvement in FEV_1 is minimal or absent after bronchodilators.

(C) **T** – PEFR is commonly decreased in asthma, especially during exacerbations, and used for monitoring severity.

(D) **T** – Both conditions are obstructive, so FEV_1/FVC ratio is typically <70%

(E) **F** – Reversibility is tested using bronchodilators, not corticosteroids (though steroids assess long-term control).



https://www.medikro.com/understanding-your-spirometry-test-results

Question (7)

(A) **T** – Pulmonary ventilation = tidal volume × respiratory rate; it includes all air moved in and out of lungs.

(B) **F** – Alveolar ventilation = (tidal volume – dead space) × respiratory rate; it is less than pulmonary ventilation.
(C) **T** – With constant tidal volume, increasing dead space reduces effective alveolar ventilation.
(D) **T** – Physiological dead space includes non-gas-exchanging regions: anatomical + non-perfused alveoli.
(E) **T** – Rapid shallow breaths lead to more air lost in dead space, reducing alveolar gas exchange efficiency.

Question (8)

(A) **F** – Central chemoreceptors are primarily sensitive to changes in CO_2 (via pH changes in CSF), not directly to PO_2.
(B) **T** – Peripheral chemoreceptors (in carotid and aortic bodies) respond quickly to hypoxemia, especially when PO_2 falls below 60 mmHg.
(C) **T** – The pre-Bötzinger complex in the medulla is considered the primary respiratory rhythm generator.
(D) **T** – Hypercapnia (increased CO_2) leads to acidification of CSF, which stimulates central chemoreceptors, increasing ventilation.
(E) **F** – The Hering–Breuer inflation reflex is more active during large tidal volumes or deep breathing, not significant during quiet breathing in adults.

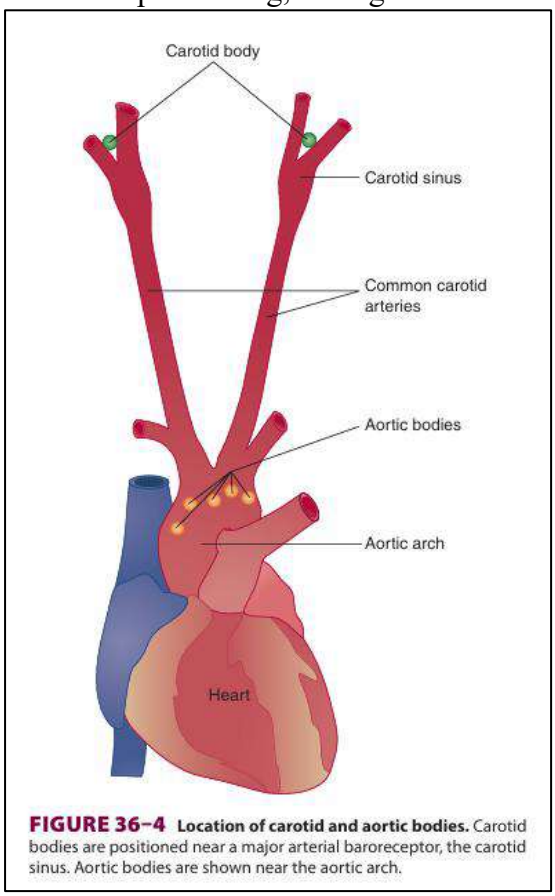


FIGURE 36–4 **Location of carotid and aortic bodies.** Carotid bodies are positioned near a major arterial baroreceptor, the carotid sinus. Aortic bodies are shown near the aortic arch.

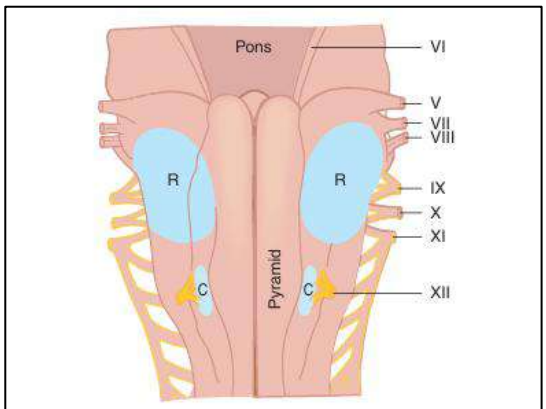


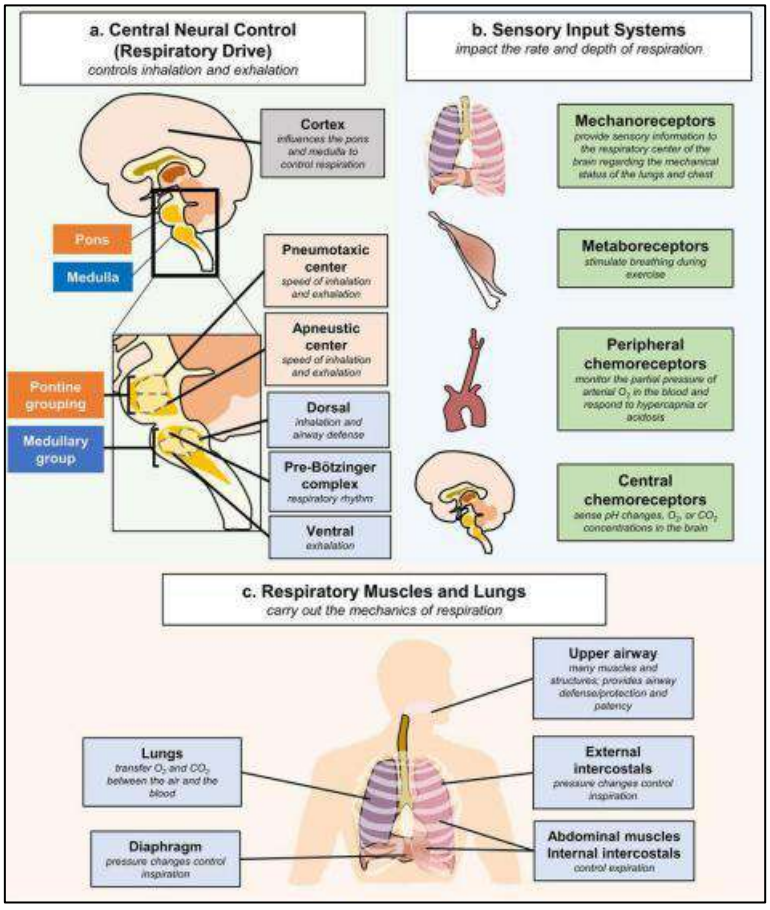
FIGURE 36–7 **Rostral (R) and caudal (C) chemosensitive areas on the ventral surface of the medulla.** Cranial nerves, pyramid and pons are labeled for reference.

Ganong's Review of Medical Physiology 24th Edition p. 660-661

Question (9)

(A) **T** – Proprioceptive input from limbs stimulates medullary respiratory centers early in exercise, increasing ventilation without blood gas changes.

(B) **T** – Baroreceptor activation inhibits medullary centers, causing slight suppression of breathing during acute hypertension.
(C) **T** – The Hering–Breuer reflex reduces inspiratory drive to prevent overinflation of the lungs.
(D) **T** – Pain signals can transiently halt breathing via cortical suppression and brainstem reflexes.
(E) **T** – Cold shock reflex includes tachypnea triggered by cutaneous cold receptor stimulation.

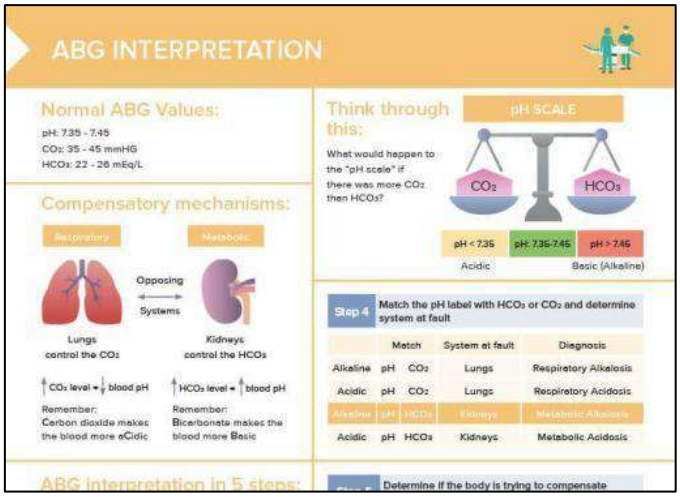


https://www.researchgate.net/figure/Control-of-respiration-a-The-respiratory-center-in-the-brain-controls-various-components_fig1_345322590

Question (10)
(A) **T** – Hypoxic hypoxia involves low PaO_2; high altitude decreases inspired PO_2
(B) **T** – In anemic hypoxia, hemoglobin content or function is impaired; CO binds Hb, lowering O_2 carrying capacity while PaO_2 stays normal.
(C) **T** – Cyanide inhibits cytochrome oxidase in the electron transport chain, preventing O_2 utilization → histotoxic hypoxia.
(D) **T** – Stagnant hypoxia is due to impaired blood flow (e.g., heart failure, shock).
(E) **F** – In histotoxic hypoxia, tissues cannot use O_2, so venous O_2 content remains high.

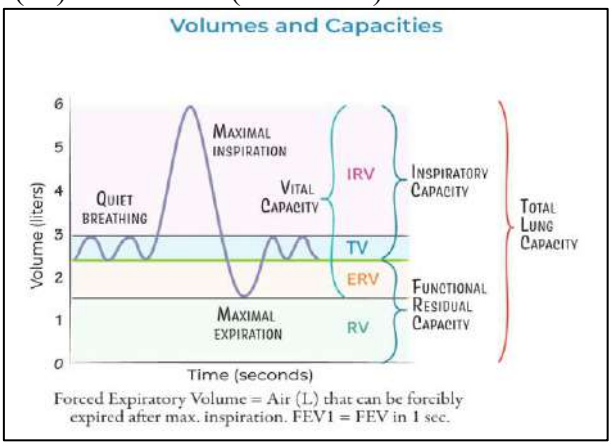
Question (11)
Correct Answer: C. Compensated respiratory acidosis
The pH is within normal limits but on the lower side (7.36), indicating a compensated state.

The $PaCO_2$ is elevated (55 mmHg) suggesting respiratory acidosis.
The HCO_3^- is also elevated (30 mEq/L), showing renal compensation.
This pattern—high CO_2, high HCO_3^-, normal pH—is consistent with chronic compensated respiratory acidosis, as commonly seen in COPD patients.



https://www.pinterest.com/pin/arterial-blood-gas-interpretation-guide--342062534215362846/

Question (12)
Correct answer: D. Residual volume
Spirometry only measures air that moves in and out of the lungs. Residual volume (RV) is the air left in the lungs after a maximal forced expiration, and it cannot be exhaled, so it cannot be measured directly by spirometry.
Other values in your list can be measured or derived from spirometry:
Expiratory reserve volume (ERV) – measured
Inspiratory reserve volume (IRV) – measured
Vital capacity (VC) – measured (IRV + TV + ERV)
Inspiratory capacity (IC) – measured (TV + IRV)



https://ditki.com/course/physiology/glossary/physiological-process/lung-volumes-capacities

Question (13)
Correct Answer: C. Chronic hypoxia causing vasoconstriction of small pulmonary arteries
In COPD, many alveoli are poorly ventilated.

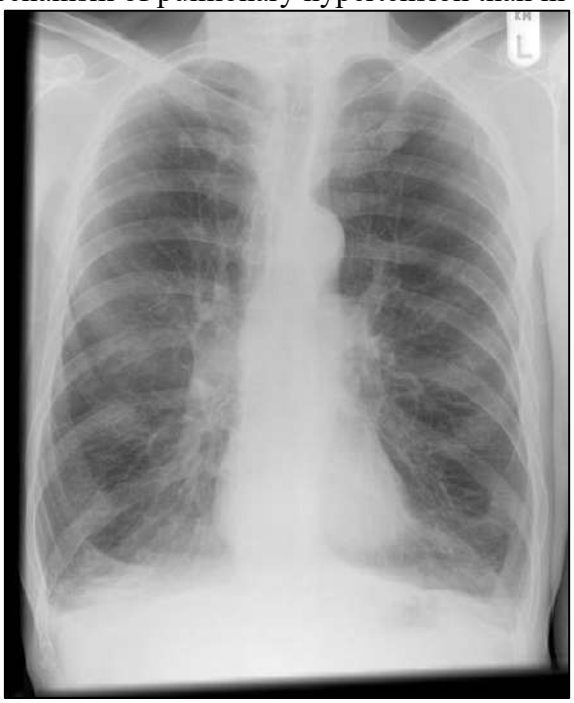
Alveolar hypoxia causes constriction of pulmonary arterioles supplying those regions → this is hypoxic pulmonary vasoconstriction (HPV).
When widespread (as in chronic lung disease), HPV becomes global, raising pulmonary vascular resistance.
This leads to pulmonary hypertension and eventually cor pulmonale.
This is a key difference from systemic circulation, which dilates in response to hypoxia.
Sympathetic activation causes vasoconstriction in systemic vessels, but the pulmonary vessels are:

- Low-resistance
- Weakly influenced by sympathetic tone

They do not dilate significantly with sympathetic stimulation, and they do not play a major role in COPD-related pulmonary hypertension.
CO_2 usually causes vasoconstriction in the pulmonary circulation, not relaxation.
So hypercapnia would worsen pulmonary hypertension, not relieve it.
Low RBC count reduces viscosity → decreases pulmonary vascular resistance.
This would not cause pulmonary hypertension.
COPD patients more commonly develop secondary polycythemia, which increases viscosity—opposite of the option.
Increased left atrial pressure (e.g., in mitral stenosis or heart failure) causes:

- Passive backup of pressure into pulmonary veins/arteries
- Pulmonary venous hypertension, not vasodilation

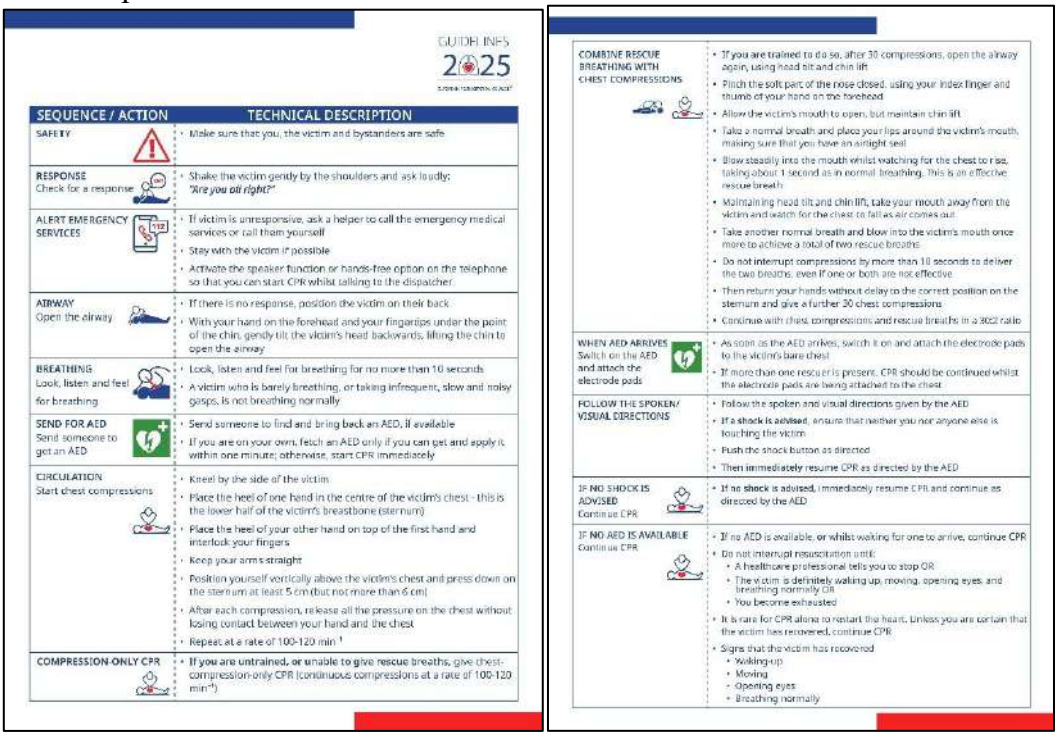
This is a different mechanism of pulmonary hypertension than in COPD.



Hyperexpansion and coarse bronchovascular markings consistent with COPD.
https://radiopaedia.org/cases/chronic-obstructive-pulmonary-disease-3

Question (14)
Correct answer: B. Begin chest compressions at a rate of 100–120 per minute
According to current CPR guidelines (AHA 2020), unresponsive adults who are not breathing normally should receive immediate chest compressions. Early initiation of high-quality compressions improves survival. Pulse checks can delay compressions,

and rescue breaths are secondary if you are alone and untrained. AED use should follow compressions as soon as it is available.



GUIDELINES 2025

SEQUENCE / ACTION	TECHNICAL DESCRIPTION
SAFETY	• Make sure that you, the victim and bystanders are safe
RESPONSE Check for a response	• Shake the victim gently by the shoulders and ask loudly: *"Are you all right?"*
ALERT EMERGENCY SERVICES	• If victim is unresponsive, ask a helper to call the emergency medical services or call them yourself • Stay with the victim if possible • Activate the speaker function or hands-free option on the telephone so that you can start CPR whilst talking to the dispatcher
AIRWAY Open the airway	• If there is no response, position the victim on their back • With your hand on the forehead and your fingertips under the point of the chin, gently tilt the victim's head backwards, lifting the chin to open the airway
BREATHING Look, listen and feel for breathing	• Look, listen and feel for breathing for no more than 10 seconds • A victim who is barely breathing, or taking infrequent, slow and noisy gasps, is not breathing normally
SEND FOR AED Send someone to get an AED	• Send someone to find and bring back an AED, if available • If you are on your own, fetch an AED only if you can get and apply it within one minute; otherwise, start CPR immediately
CIRCULATION Start chest compressions	• Kneel by the side of the victim • Place the heel of one hand in the centre of the victim's chest - this is the lower half of the victim's breastbone (sternum) • Place the heel of your other hand on top of the first hand and interlock your fingers • Keep your arms straight • Position yourself vertically above the victim's chest and press down on the sternum at least 5 cm (but not more than 6 cm) • After each compression, release all the pressure on the chest without losing contact between your hand and the chest • Repeat at a rate of 100-120 min⁻¹
COMPRESSION-ONLY CPR	• If you are untrained, or unable to give rescue breaths, give chest-compression-only CPR (continuous compressions at a rate of 100-120 min⁻¹)

COMBINE RESCUE BREATHING WITH CHEST COMPRESSIONS	• If you are trained to do so, after 30 compressions, open the airway again, using head tilt and chin lift • Pinch the soft part of the nose closed, using your index finger and thumb of your hand on the forehead • Allow the victim's mouth to open, but maintain chin lift • Take a normal breath and place your lips around the victim's mouth, making sure that you have an airtight seal • Blow steadily into the mouth whilst watching for the chest to rise, taking about 1 second as in normal breathing. This is an effective rescue breath • Maintaining head tilt and chin lift, take your mouth away from the victim and watch for the chest to fall as air comes out • Take another normal breath and blow into the victim's mouth once more to achieve a total of two rescue breaths • Do not interrupt compressions by more than 10 seconds to deliver the two breaths, even if one or both are not effective • Then return your hands without delay to the correct position on the sternum and give a further 30 chest compressions • Continue with chest compressions and rescue breaths in a 30:2 ratio
WHEN AED ARRIVES Switch on the AED and attach the electrode pads	• As soon as the AED arrives, switch it on and attach the electrode pads to the victim's bare chest • If more than one rescuer is present, CPR should be continued whilst the electrode pads are being attached to the chest
FOLLOW THE SPOKEN/ VISUAL DIRECTIONS	• Follow the spoken and visual directions given by the AED • If a shock is advised, ensure that neither you nor anyone else is touching the victim • Push the shock button as directed • Then immediately resume CPR as directed by the AED
IF NO SHOCK IS ADVISED Continue CPR	• If no shock is advised, immediately resume CPR and continue as directed by the AED

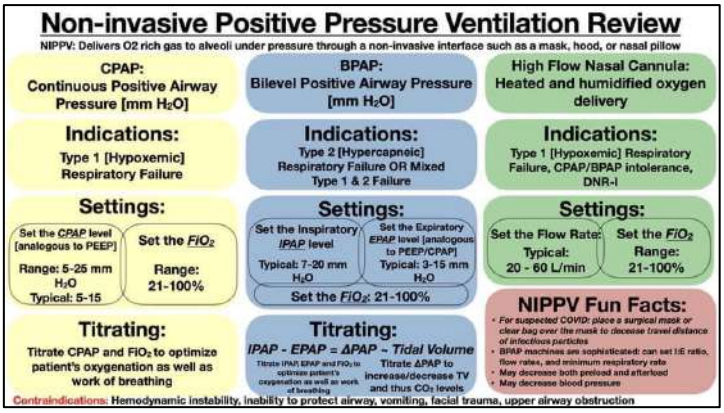
IF NO AED IS AVAILABLE
Continue CPR
	• If no AED is available, or whilst waiting for one to arrive, continue CPR
	• Do not interrupt resuscitation until:
	  ◦ A healthcare professional tells you to stop CPR
	  ◦ The victim is definitely waking up, moving, opening eyes, and breathing normally OR
	  ◦ You become exhausted
	• It is rare for CPR alone to restart the heart. Unless you are certain that the victim has recovered, continue CPR
	• Signs that the victim has recovered
	  ◦ Waking up
	  ◦ Moving
	  ◦ Opening eyes
	  ◦ Breathing normally

https://www.resuscitationjournal.com/article/S0300-9572(25)00283-7/fulltext

Question (15)

Correct answer: B. Begin non-invasive positive pressure ventilation (NIPPV)

This patient has acute-on-chronic hypercapnic respiratory failure (type 2) due to COPD exacerbation, indicated by elevated $PaCO_2$, low pH, and hypoxemia. NIPPV is first-line therapy in such patients, as it improves gas exchange, reduces the work of breathing, and can prevent intubation. High-flow oxygen must be used cautiously in COPD due to the risk of worsening CO_2 retention. Immediate intubation is reserved for patients who fail NIPPV or have severe acidosis, shock, or altered consciousness.



Non-invasive Positive Pressure Ventilation Review

NIPPV: Delivers O2 rich gas to alveoli under pressure through a non-invasive interface such as a mask, hood, or nasal pillow

CPAP: Continuous Positive Airway Pressure [mm H_2O]	BPAP: Bilevel Positive Airway Pressure [mm H_2O]	High Flow Nasal Cannula: Heated and humidified oxygen delivery
Indications: Type 1 [Hypoxemic] Respiratory Failure	**Indications:** Type 2 [Hypercapneic] Respiratory Failure OR Mixed Type 1 & 2 Failure	**Indications:** Type 1 [Hypoxemic] Respiratory Failure, CPAP/BPAP intolerance, DNR-I
Settings: Set the *CPAP* level [analogous to PEEP] Range: 5-25 mm H_2O Typical: 5-15; Set the *FiO_2* Range: 21-100%	**Settings:** Set the Inspiratory *IPAP* level Typical: 7-20 mm H_2O; Set the Expiratory *EPAP* level [analogous to PEEP/CPAP] Typical: 3-15 mm H_2O; Set the *FiO_2*: 21-100%	**Settings:** Set the Flow Rate: Typical: 20 - 60 L/min; Set the *FiO_2* Range: 21-100%
Titrating: Titrate CPAP and FiO_2 to optimize patient's oxygenation as well as work of breathing	**Titrating:** *IPAP - EPAP = ΔPAP ~ Tidal Volume* Titrate IPAP, EPAP and FiO_2 to optimize patient's oxygenation as well as work of breathing; Titrate ΔPAP to increase/decrease TV and thus CO_2 levels	**NIPPV Fun Facts:** • For suspected COVID: place a surgical mask or clear bag over the mask to decrease travel distance of infectious particles • BPAP machines are sophisticated: can set I:E ratio, flow rates, and minimum respiratory rate • May decrease both preload and afterload • May decrease blood pressure

Contraindications: Hemodynamic instability, inability to protect airway, vomiting, facial trauma, upper airway obstruction

https://www.facebook.com/photo/?fbid=831473282352734&set=a.467834225383310

Question (16)

(A) **T** – Proton pump (H^+/K^+ ATPase) is final common pathway for H^+ secretion into lumen.

(B) **T** – Histamine binds to H_2 receptors → ↑ adenylate cyclase → ↑ cAMP → ↑ acid.

(C) **F** – ACh acts mainly via M_3 receptors on parietal cells → ↑ Ca^{2+} → H^+ secretion.

(D) **T** – Gastrin primarily stimulates ECL cells (via CCK_2 receptor) → histamine release → parietal cell activation.
(E) **T** – Somatostatin (paracrine) & prostaglandins (via ↓ cAMP) inhibit acid secretion.

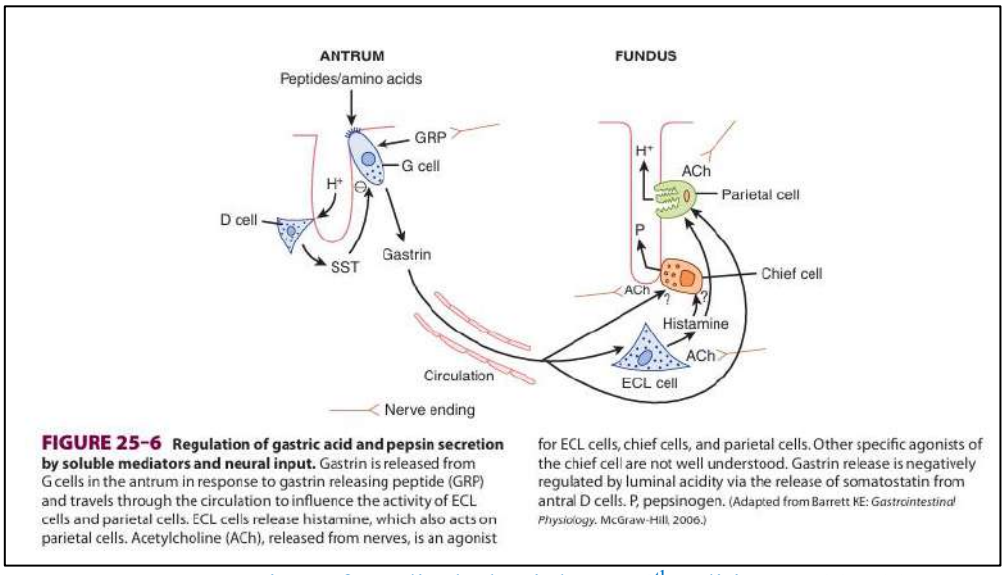
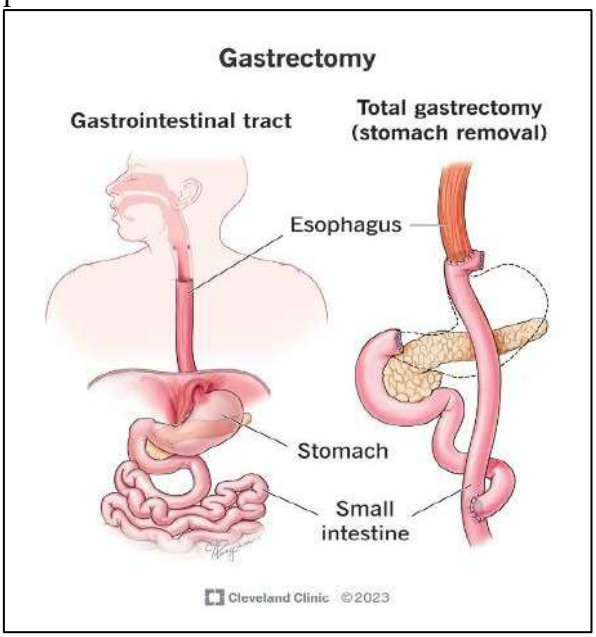


FIGURE 25–6 Regulation of gastric acid and pepsin secretion by soluble mediators and neural input. Gastrin is released from G cells in the antrum in response to gastrin releasing peptide (GRP) and travels through the circulation to influence the activity of ECL cells and parietal cells. ECL cells release histamine, which also acts on parietal cells. Acetylcholine (ACh), released from nerves, is an agonist for ECL cells, chief cells, and parietal cells. Other specific agonists of the chief cell are not well understood. Gastrin release is negatively regulated by luminal acidity via the release of somatostatin from antral D cells. P, pepsinogen. (Adapted from Barrett KE: *Gastrointestinal Physiology*. McGraw-Hill, 2006.)

Ganong's Review of Medical Physiology 24th Edition p. 660-661

Question (17)

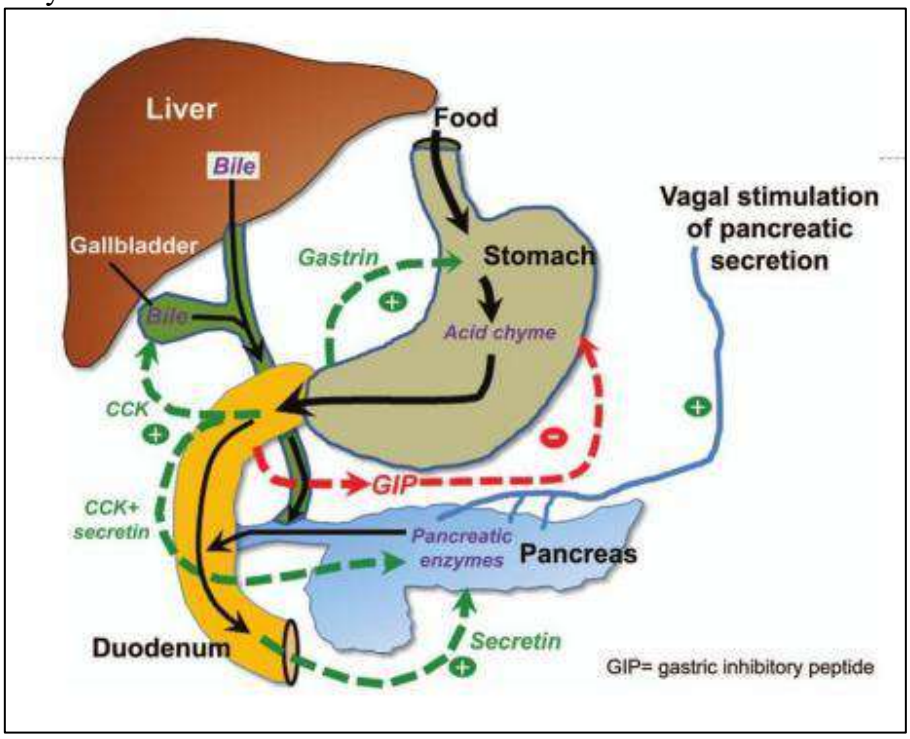
(A) **T** – Intrinsic factor from parietal cells is essential for B_{12} absorption in the ileum → deficiency causes megaloblastic anemia.
(B) **T** – Gastric acid helps convert dietary ferric (Fe^{3+}) to ferrous (Fe^{2+}) form; deficiency impairs iron absorption → microcytic anemia.
(C) **T** – Rapid emptying of hyperosmolar chyme draws fluid into lumen → hypovolemia, abdominal cramps, diarrhea.
(D) **T** – Rapid glucose absorption → hyperglycemia → exaggerated insulin response → hypoglycemia (late dumping).
(E) **T** – Gastric lipase is minor; pancreatic lipase is the major enzyme for fat digestion, so impact is minimal.



https://my.clevelandclinic.org/health/procedures/gastrectomy

Question (18)

(A) **T** – Secretin acts on ductal cells → ↑ HCO_3^- secretion to neutralize gastric acid.
(B) **T** – CCK stimulates acinar cells → ↑ digestive enzyme secretion.
(C) **T** – ACh (via vagus) → muscarinic receptors → potentiates acinar enzyme secretion.
(D) **T** – Low duodenal pH (<4.5) is the main stimulus for secretin release.
(E) **F** – CCK release is triggered mainly by fats and partially by amino acids, not carbohydrates.



https://journals.sagepub.com/doi/10.1177/0192623313508479

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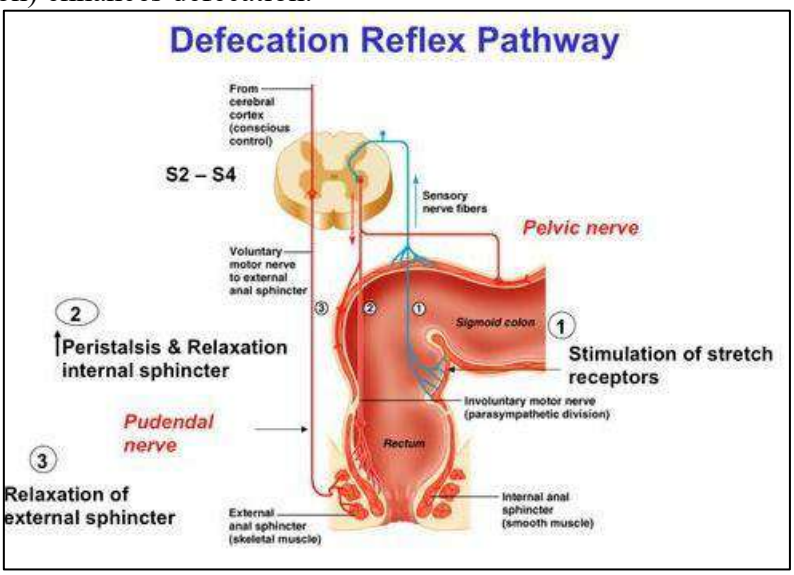
Question (19)

(A) **F** – Small intestine handles nutrient absorption; large intestine mainly reabsorbs water and electrolytes.
(B) **T** – Main colon function → water & Na^+, Cl^- absorption → stool consolidation.
(C) **T** – Gastrocolic reflex: gastric distension stimulates colonic motility via vagal/enteric pathways; exaggerated in IBS.
(D) **F** – Peyer's patches are found in the ileum of the small intestine, not the colon; function = immune surveillance of gut.
(E) **T** – Long-term metformin → risk of B12 deficiency → supplementation prevents neuropathy & megaloblastic anemia.

Question (20)

(A) **T** – Intrinsic reflex is weak, mediated by myenteric plexus; produces peristalsis in colon/rectum.
(B) **T** – Parasympathetic reflex (S2–S4, pelvic nerves) greatly strengthens the intrinsic reflex.
(C) **T** – Rectal distension → reflex relaxation of internal sphincter (rectoanal inhibitory reflex).
(D) **F** – External anal sphincter is under voluntary control via pudendal nerve (somatic).

(E) **T** – Voluntary straining (Valsalva + abdominal muscle contraction + pelvic floor relaxation) enhances defecation.



https://quizlet.com/515728469/gi-motility-flash-cards/

Question (21)

(A) **T** – Vomiting center in medulla coordinates the reflex.
(B) **T** – CTZ is in area postrema, sensitive to drugs, toxins; outside BBB.
(C) **T** – Vagal and splanchnic afferents carry signals from GI tract.
(D) **T** – Protective mechanism against aspiration/regurgitation into nasopharynx.
(E) **T** – Forceful contraction of diaphragm + abdominal muscles generates expulsion.

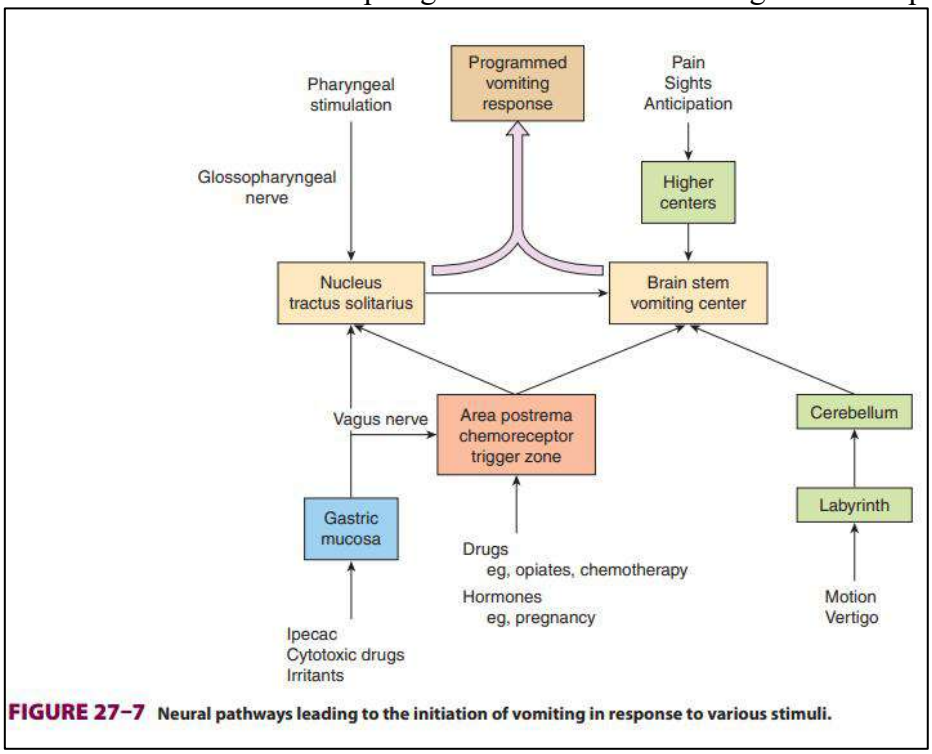
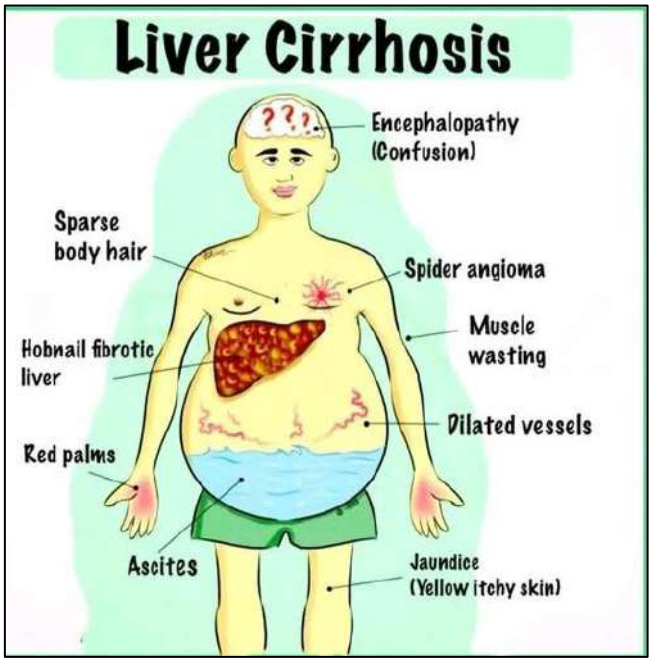


FIGURE 27–7 Neural pathways leading to the initiation of vomiting in response to various stimuli.

Ganong's Review of Medical Physiology 24th Edition p. 503

Question (22)

(A) **T** – Estrogen accumulation → dilated superficial cutaneous vessels → spider nevi.
(B) **T** – Vasodilators + estrogen excess → palmar erythema.
(C) **T** – Impaired androgen metabolism + estrogen excess → gynecomastia.
(D) **T** – Portal hypertension → dilated paraumbilical veins → caput medusae.
(E) **F** – Testicular atrophy is due to decreased androgen production, not increased estrogen catabolism.



https://www.facebook.com/groups/3278314818931717/posts/9453551908074613/

Question (23)

(A) **T** – UES is skeletal muscle, controlled voluntarily and by medullary swallowing center.
(B) **F** – Peristalsis begins in the proximal esophagus and is sequential along the length.
(C) **T** – LES maintains tonic contraction to prevent reflux.
(D) **T** – Primary peristalsis is swallowing-initiated, involving both striated (proximal) and smooth (distal) muscle.
(E) **F** – Coordinated peristalsis requires both enteric and central (vagal) input, especially for primary peristalsis.

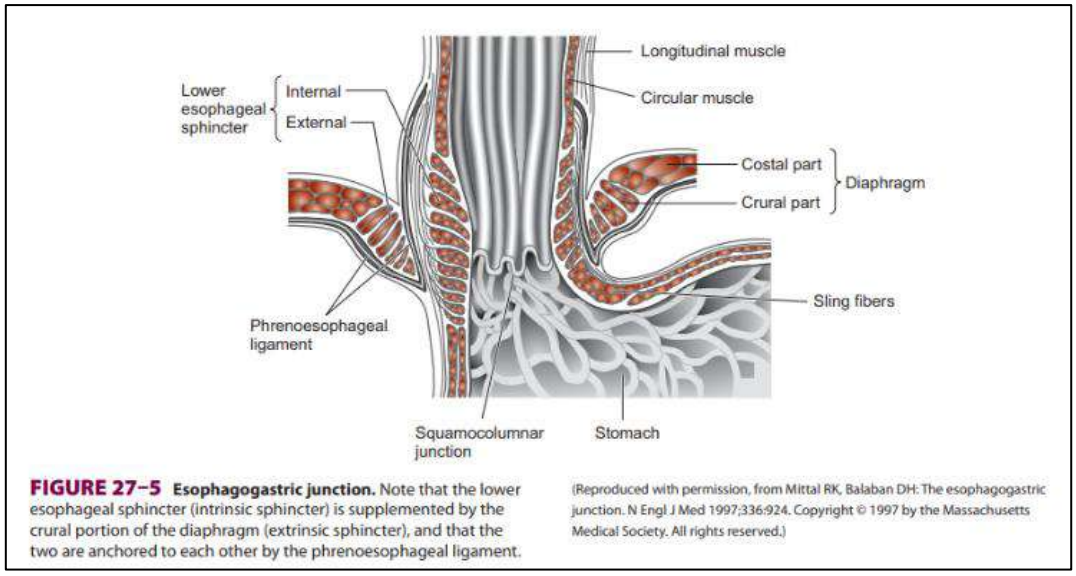
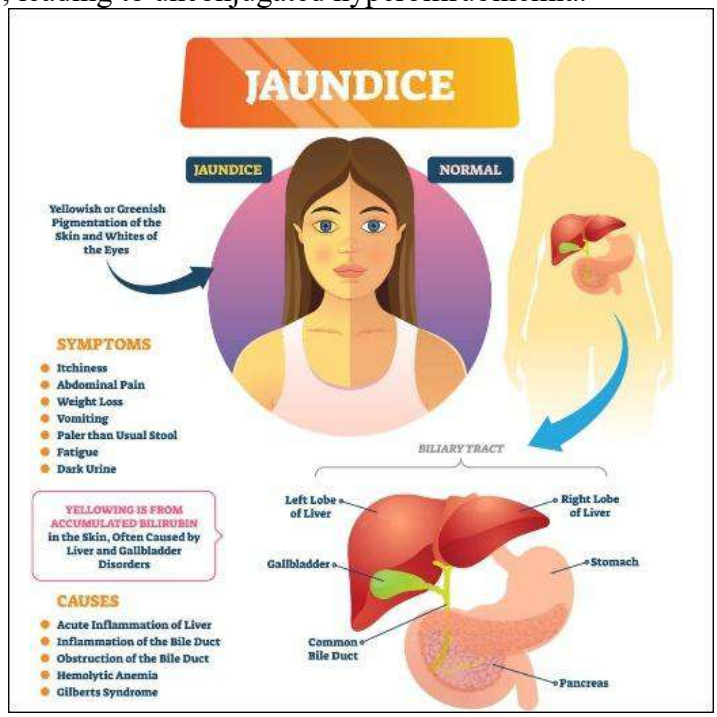


FIGURE 27–5 **Esophagogastric junction.** Note that the lower esophageal sphincter (intrinsic sphincter) is supplemented by the crural portion of the diaphragm (extrinsic sphincter), and that the two are anchored to each other by the phrenoesophageal ligament.

Ganong's Review of Medical Physiology 24th Edition p. 501

Question (24)

(A) **T** – Yellowing of skin and sclera appears around 2–3 mg/dL.
(B) **F** – Hemolytic jaundice primarily raises indirect (unconjugated) bilirubin.
(C) **T** – Obstruction prevents bilirubin excretion into the gut, causing pale stools; conjugated bilirubin is excreted in urine, darkening it.
(D) **T** – Hepatocellular injury impairs uptake, conjugation, and excretion, causing mixed hyperbilirubinemia.
(E) **F** – Neonatal physiologic jaundice is due to immature UDP-glucuronyl transferase, leading to unconjugated hyperbilirubinemia.



https://continentalhospitals.com/diseases/jaundice/

Question (25)

(A) **T** – Primary saliva is isotonic; ductal reabsorption of Na^+ and Cl^- makes final saliva hypotonic at low flow rates.
(B) **T** – Sympathetic activation produces smaller volumes of saliva that are protein-rich and more viscous.
(C) **F** – Ductal modification is flow-dependent; at high flow rates, saliva is less modified and closer to isotonic.
(D) **T** – Salivary output follows circadian rhythm, peaking during the day and decreasing at night.
(E) **T** – Sjögren's syndrome destroys acinar cells, causing xerostomia and reduced enzymatic activity.

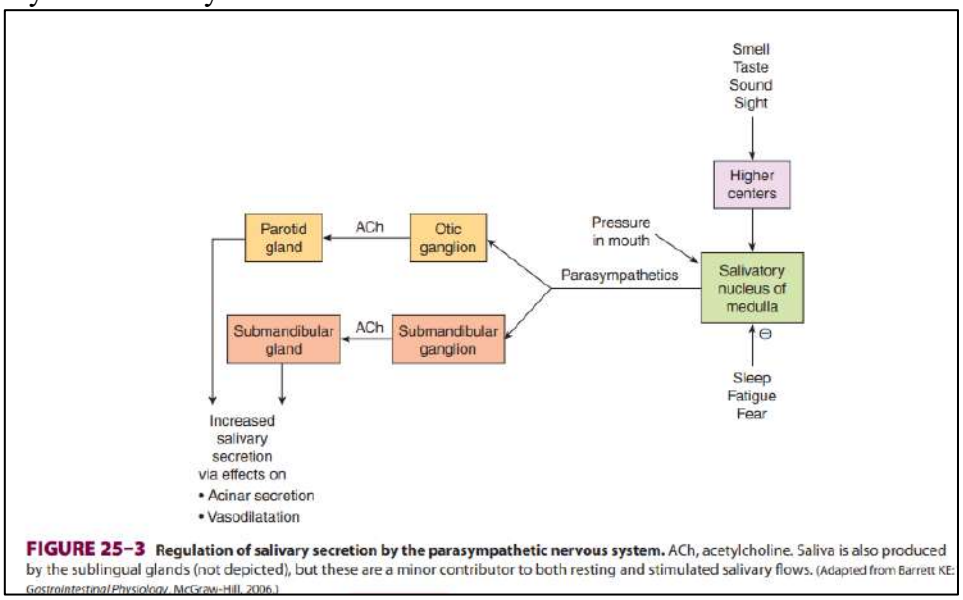


FIGURE 25–3 **Regulation of salivary secretion by the parasympathetic nervous system.** ACh, acetylcholine. Saliva is also produced by the sublingual glands (not depicted), but these are a minor contributor to both resting and stimulated salivary flows. (Adapted from Barrett KE: *Gastrointestinal Physiology*. McGraw-Hill, 2006.)

Ganong's Review of Medical Physiology 24th Edition p. 458

Question (26)
Correct answer: D. Weight gain
Pancreatectomy is the surgical removal of the whole pancreas. When the pancreas is removed, both the exocrine and the endocrine functions of the pancreas cease. As pancreas secretes lipase, which is important for the absorption of lipids, lack of lipase causes malabsorption of lipids which causes excess lipids to be excreted with feces resulting in oily, pale, hard-to-flush stools known as steatorrhea. As insulin is deficient, hyperglycemia occurs. When insulin is deficient, glucose cannot enter insulin-stimulated cells (GLUT4 containing cells in muscle and adipose tissue). As this creates a sort of "starving" situation, counter-regulatory hormones such as glucagon, epinephrine, cortisol and growth hormone initiate regulatory mechanisms to increase the blood glucose level. This causes increased lipolysis. Paired with the lack of inhibition of lipolysis due to lack of insulin, this leads to excess fatty acid formation. These fatty acids are then used to form ketone bodies causing diabetic ketoacidosis resulting in metabolic acidosis. Pancreas also secretes proteolytic enzymes which are responsible for protein absorption. Lack of these enzymes result in decreased amino acid absorption. The malabsorption of nutrients along with breaking down of fat and muscle in diabetes results in weight loss, not gain.

TABLE 1.

Digestive Enzymes Secreted by the Pancreas

Enzyme	Target
Amylolytic enzymes	
Amylase	alpha-1,4-glycosidic bonds in starch
Lipolytic enzymes	
Lipase	Triglyceride, producing fatty acids and 2-monoglycerides
Phospholipase A2	Phosphotidylcholine, producing a free fatty acid and lysophosphotidylcholine
Carboxylesterase	Cholesterol esters, lipid-soluble vitamin esters, and glycerides (tri-, di-, or monoglycerides)
Proteolytic enzymes	
Trypsin	Interior peptide bonds involving basic amino acids
Chymotrypsin	Interior peptide bonds involving aromatic amino acids
Carboxypeptidase A and B	External peptide bonds involving aromatic and neutral aliphatic amino acids (A) and basic amino acids (B) at the carboxy-terminal end
Elastase	Interior peptide bonds involving neutral aliphatic amino acids
Nucleases	
Deoxyribonuclease (DNAse)	Endonuclease that splits phosphodiester linkages adjacent to pyrimidine nucleotide
Ribonuclease (RNAse)	Catalyzes the breakdown of RNA

Adapted from Henderson.[1]

https://derangedphysiology.com/main/cicm-primary-exam/gastrointestinal-system/Chapter-111/exocrine-functions-pancreas

Question (27)

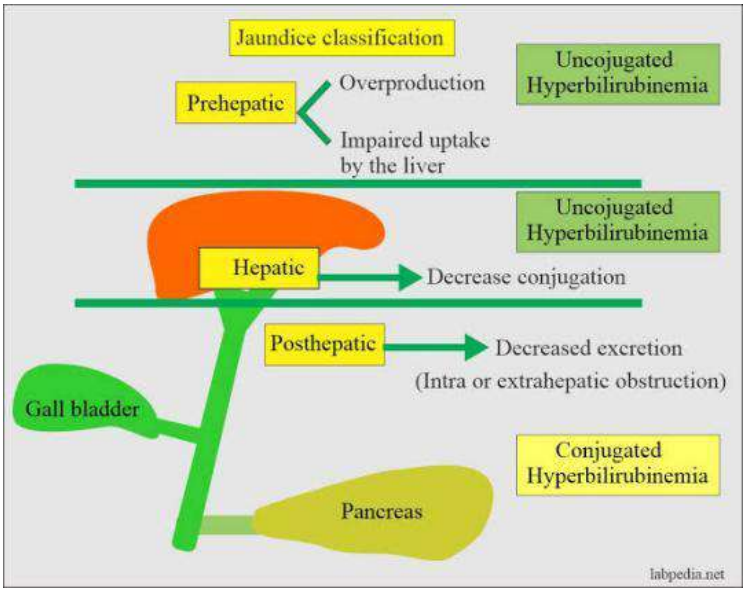
Correct answer: C. Gallstone obstructing the common bile duct

This patient is presenting with jaundice and dark urine and investigations show direct hyperbilirubinemia. This pattern is characteristic of obstructive (post-hepatic) jaundice. The conjugated bilirubin cannot be excreted through the intestine. So, it is excreted in the urine. The only post-hepatic cause given here is gallstone obstructing the common bile duct.

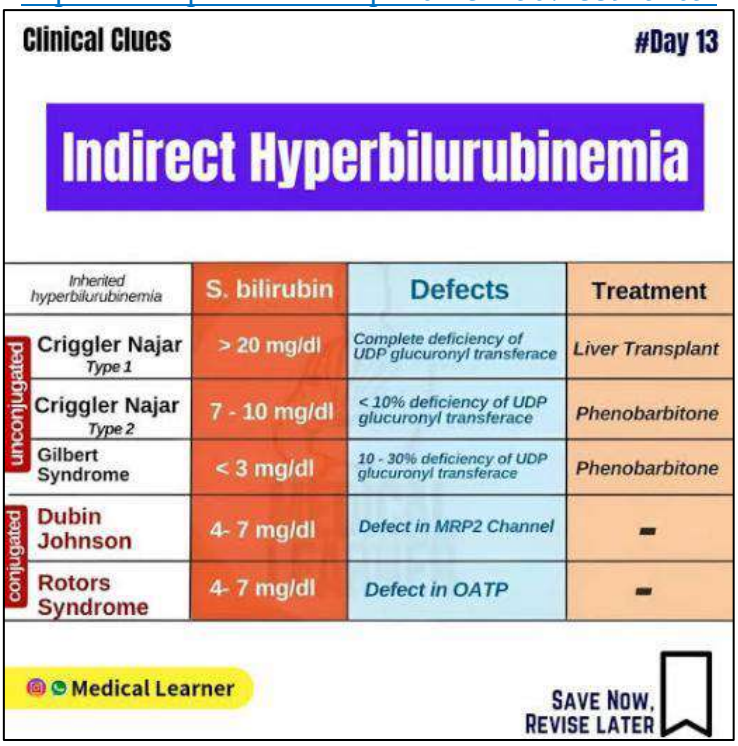
In hemolytic anemia, excessive breakdown of hemoglobin causes indirect hyperbilirubinemia. As unconjugated bilirubin is not water soluble, it is not excreted in urine.

In acute viral hepatitis, initially there is unconjugated hyperbilirubinemia due to hepatocellular damage.

Gilbert syndrome and Criggler Najjar syndrome both causes unconjugated hyperbilirubinemia.



https://www.pinterest.com/pin/624311567133915165/

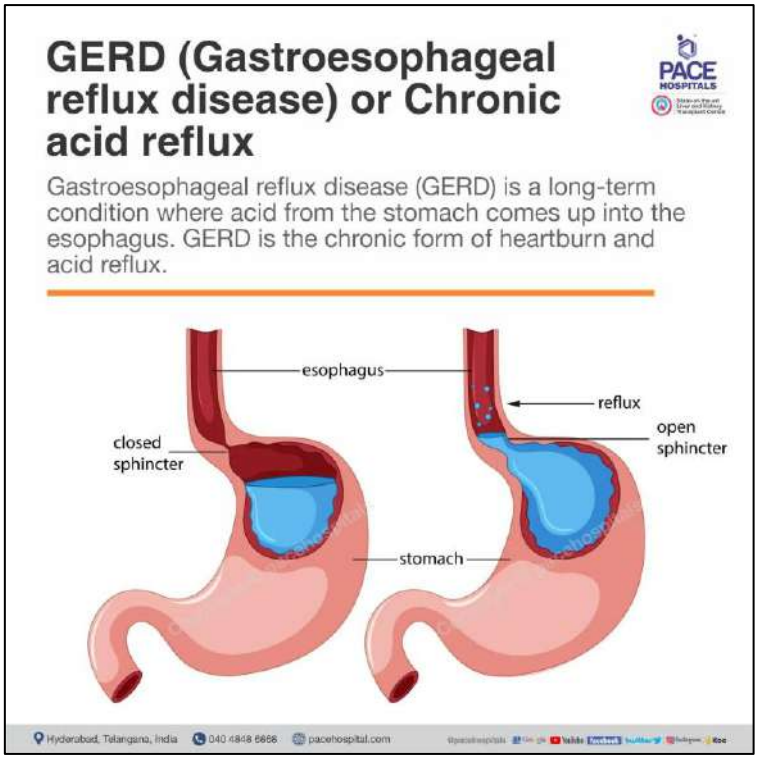


https://www.instagram.com/p/DAqa2oWzn_3/

Question (28)

Correct answer: B. Impaired lower esophageal sphincter (LES) tone

The primary mechanism in most cases of GERD is transient or chronically reduced LES pressure, allowing gastric contents to reflux into the esophagus. Hiatal hernia can contribute by impairing the LES and esophageal barrier function but is less common as the sole cause. Delayed gastric emptying and esophageal motility disorders may exacerbate reflux but are secondary contributors. Increased acid production alone is not typically the primary cause of GERD.

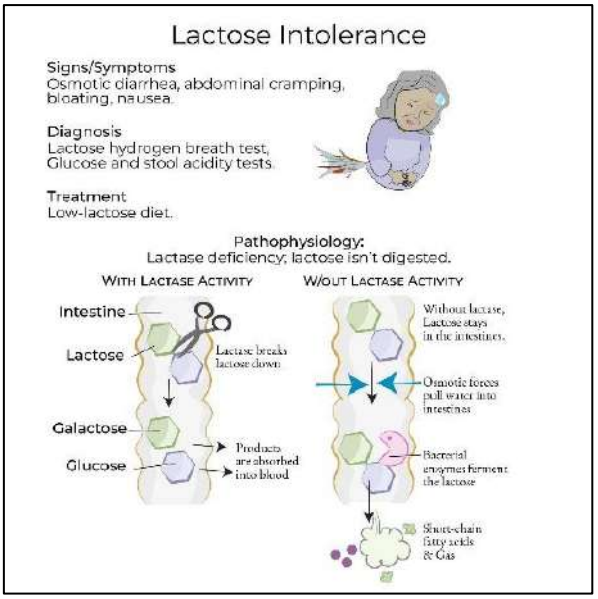


https://www.pacehospital.com/gastroesophageal-reflux-disease-gerd-symptoms-causes-diagnosis-and-treatment

Question (29)

Correct answer: B. Inability to hydrolyze lactose in the small intestine due to lactase deficiency

The patient's symptoms are classic for lactose intolerance, caused by deficiency of lactase, the brush-border enzyme that hydrolyzes lactose into glucose and galactose. Undigested lactose reaches the colon, where it is fermented by bacteria, producing gas and osmotic diarrhea. Pancreatic amylase deficiency (choice A) affects starch digestion, not lactose. SGLT1 defects (choice C) would cause glucose/galactose malabsorption, which is rare. Sucrase deficiency (choice D) leads to problems with sucrose, not lactose. Salivary amylase inactivation (choice E) is normal and not clinically significant in this scenario.

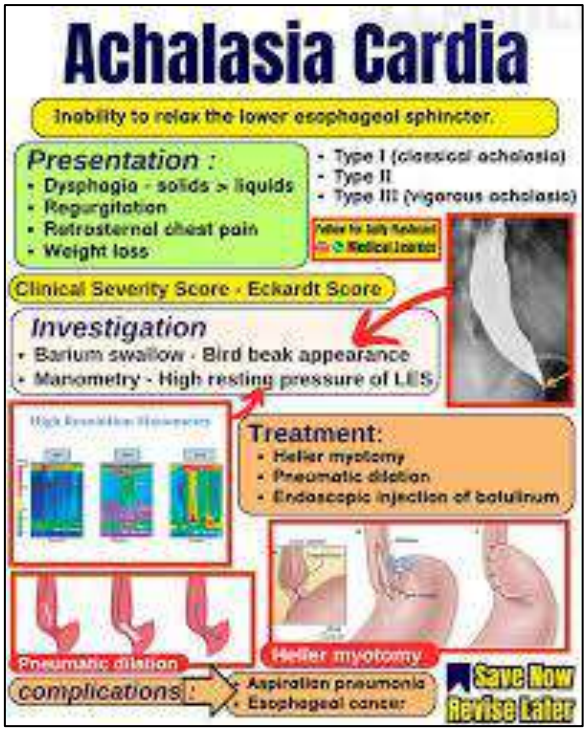


https://ditki.com/course/pathology/glossary/pathophysiologic-disorder/lactose-intolerance

Question (30)

Correct answer: A. Lower esophageal sphincter (LES) hypertonicity due to loss of inhibitory neurons in the myenteric plexus

Achalasia is caused by degeneration of inhibitory neurons (nitric oxide–producing) in the myenteric plexus, leading to failure of LES relaxation and absent peristalsis. Barium swallow typically shows a dilated esophagus with a tapered "bird-beak" distal segment. It is not due to acid hypersecretion, autoimmune striated muscle destruction, transient LES relaxation, or extrinsic compression, although secondary achalasia (pseudoachalasia) can result from tumors.



https://www.instagram.com/p/DFxp9sUs0mK/

Question (31)

(A) **T** - A hormone is defined as a chemical messenger released into the blood to act on distant target organs (endocrine action).
(B) **T** - Hormones remain in circulation longer → more sustained effects.
(C) **F** - Hormones can act locally (paracrine) or even on the same cell (autocrine), not only at distant sites.
(D) **T** - Target-cell receptors are essential; without them, hormones have no effect.
(E) **T** - Many functions—stress response, reproduction, metabolism—involve neuroendocrine integration (e.g., hypothalamus).

Question (32)

(A) **T**
(B) **T** - Insulin is secreted as a preprohormone. Preproinsulin originated from the ER. It contains A and B chains which are "connected" together by C-peptide. Disulfide bonds are formed between A and B chains which form proinsulin. In the golgi bodies, proinsulin is cleaved into insulin and C-peptide. So, an equal number of C-peptides are present to the number of insulin molecules formed, thereby acting as a marker of insulin production.
(C) **F** - GLUT 3 are glucose transporters which are located in the brain, placenta, kidney and some other organs. It is insulin independent. GLUT 4 however is stimulated by insulin. It is located in muscle and adipose tissue. When insulin binds with insulin receptors of the cells in these tissues, vesicles containing GLUT 4 move rapidly to the cell membrane and fuse with it, inserting the transporters into the cell membrane. This facilitates insulin uptake into these cells. When insulin action ceases, these transporters containing membrane patches are endocytosed and the vesicles are ready for the next insulin exposure.
(D) **F** - It causes increased transport of potassium ions into insulin-sensitive cells.
(E) **T**

TABLE 24–1 Principal actions of insulin.

Rapid (seconds)
Increased transport of glucose, amino acids, and K^+ into insulin-sensitive cells
Intermediate (minutes)
Stimulation of protein synthesis
Inhibition of protein degradation
Activation of glycolytic enzymes and glycogen synthase
Inhibition of phosphorylase and gluconeogenic enzymes
Delayed (hours)
Increase in mRNAs for lipogenic and other enzymes

Courtesy of ID Goldfine.

TABLE 24–3 Glucose transporters in mammals.

	Function	K_m (mM)*	Major Sites of Expression
Secondary active transport (Na^+-glucose cotransport)			
SGLT 1	Absorption of glucose	0.1–1.0	Small intestine, renal tubules
SGLT 2	Absorption of glucose	1.6	Renal tubules
Facilitated diffusion			
GLUT 1	Basal glucose uptake	1–2	Placenta, blood-brain barrier, brain, red cells, kidneys, colon, many other organs
GLUT 2	B-cell glucose sensor; transport out of intestinal and renal epithelial cells	12–20	B cells of islets, liver, epithelial cells of small intestine, kidneys
GLUT 3	Basal glucose uptake	<1	Brain, placenta, kidneys, many other organs
GLUT 4	Insulin-stimulated glucose uptake	5	Skeletal and cardiac muscle, adipose tissue, other tissues
GLUT 5	Fructose transport	1–2	Jejunum, sperm
GLUT 6	Unknown	—	Brain, spleen and leukocytes
GLUT 7	Glucose 6-phosphate transporter in endoplasmic reticulum	—	Liver

*The K_m is the glucose concentration at which transport is half-maximal.

Data from Stephens JM, Pilch PF: The metabolic regulation and vesicular transport of GLUT 4, the major insulin-responsive glucose transporter. Endocr Rev 1995;16:529.

TABLE 24–2 Effects of insulin on various tissues.

Adipose tissue
Increased glucose entry
Increased fatty acid synthesis
Increased glycerol phosphate synthesis
Increased triglyceride deposition
Activation of lipoprotein lipase
Inhibition of hormone-sensitive lipase
Increased K^+ uptake
Muscle
Increased glucose entry
Increased glycogen synthesis
Increased amino acid uptake
Increased protein synthesis in ribosomes
Decreased protein catabolism
Decreased release of gluconeogenic amino acids
Increased ketone uptake
Increased K^+ uptake
Liver
Decreased ketogenesis
Increased protein synthesis
Increased lipid synthesis
Decreased glucose output due to decreased gluconeogenesis, increased glycogen synthesis, and increased glycolysis
General
Increased cell growth

Ganong's Review of Medical Physiology 24th Edition p. 434-435

Question (33)

(A) **T** - Glucocorticoids stimulate lipolysis, releasing free fatty acids which are used to provide energy instead of glucose in times of stress and starvation.

(B) **F** - Glucocorticoids do reduce protein stores in all body cells except in the liver. In the liver, protein synthesis is increased due to amino acid transport into liver cells and enhancement of liver enzymes.

(C) **T** - Glucocorticoids also stimulate gluconeogenesis. Paired with decreased glucose utilization, hyperglycemia occurs.

(D) **T** - High doses of glucocorticoids depresses the immune response by inhibiting production of interleukins and T lymphocytes. Therefore, glucocorticoids are prescribed for organ transplant recipients.

(E) **T** - It does so by increasing formation of glucose from proteins and other substances by increasing enzymes required to convert amino acids to glucose in the liver, and mobilization of amino acids from extrahepatic tissues.

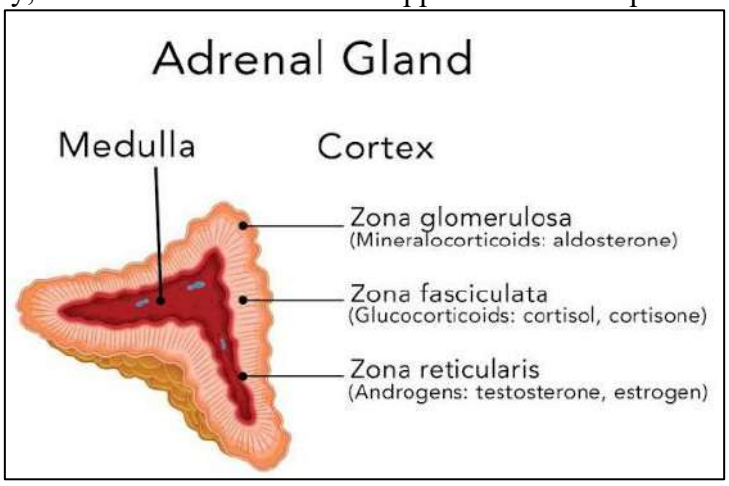
Question (34)

(A) **T** - as well as the regulator of extracellular volume

(B) **F** - there are three layers of the adrenal cortex which secretes hormones (1) zona glomerulosa which secretes aldosterone (2) zona fasciculata which secretes cortisol (3) zona reticularis which secretes adrenal androgens.

(C) **T** - it also has a similar action on salt and water transport in the colon, salivary glands, and sweat glands.

(D) **F** - in patients with adenomas of the glomerulosa cell, there is primary hyperaldosteronism (not hypoaldosteronism) which is called Conn syndrome. Hypertension and hypokalemia frequently develop in these patients.
(E) **T** - typically, the renin concentration is suppressed in these patients.



https://www.adrenalinsufficiency.org/what-is-adrenal-insufficiency/anatomy-physiology/

Question (35)

(A) **F** – The thyroid mainly secretes T4; most T3 is formed peripherally by deiodination.
(B) **F** – T3 is more potent than T4 at nuclear receptors.
(C) **T** – TBG binds most circulating hormone; free hormone is active.
(D) **T** – TSH stimulates hormone synthesis, release, and gland growth.
(E) **F** – High iodine inhibits organification and hormone release (Wolff–Chaikoff), not stimulates it.

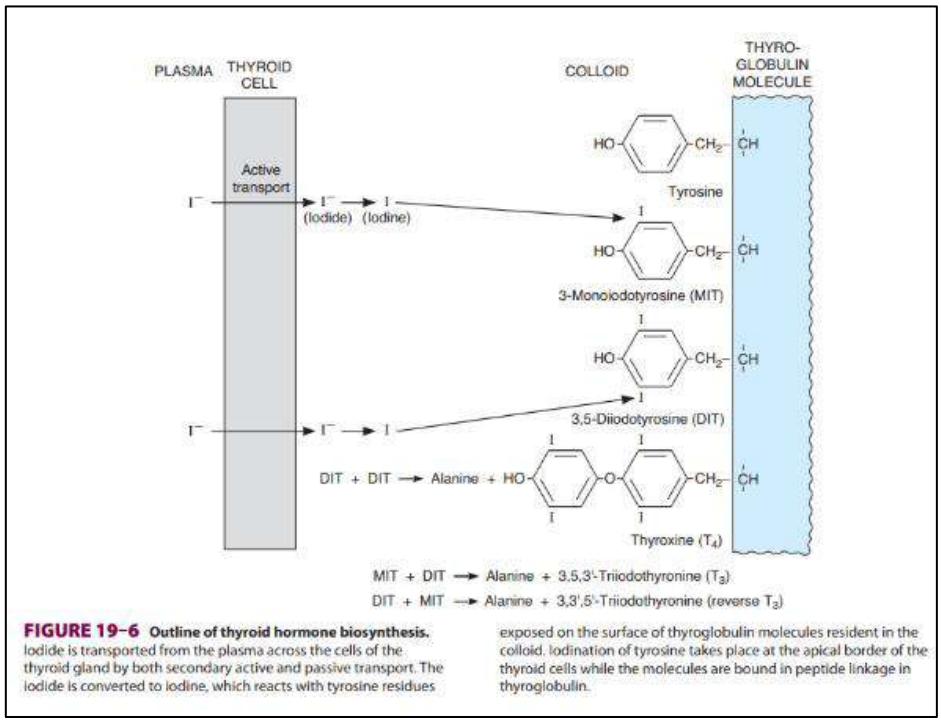


FIGURE 19–6 **Outline of thyroid hormone biosynthesis.** Iodide is transported from the plasma across the cells of the thyroid gland by both secondary active and passive transport. The iodide is converted to iodine, which reacts with tyrosine residues exposed on the surface of thyroglobulin molecules resident in the colloid. Iodination of tyrosine takes place at the apical border of the thyroid cells while the molecules are bound in peptide linkage in thyroglobulin.

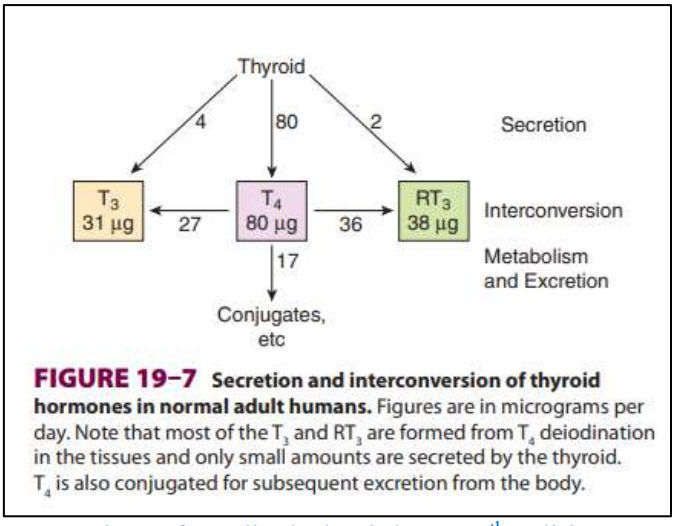


FIGURE 19–7 Secretion and interconversion of thyroid hormones in normal adult humans. Figures are in micrograms per day. Note that most of the T_3 and RT_3 are formed from T_4 deiodination in the tissues and only small amounts are secreted by the thyroid. T_4 is also conjugated for subsequent excretion from the body.

Ganong's Review of Medical Physiology 24th Edition p. 342-343

Question (36)

(A) **F** - PTH decreases renal phosphate reabsorption (phosphaturia).
→ "PTH = Phosphate Trashing Hormone."

(B) **T** - Calcitriol increases intestinal absorption of both calcium and phosphate via increased expression of transport proteins.

(C) **F** - Calcitonin plays a minor role in calcium homeostasis; it is not essential for life.

(D) **T** - Calcitriol stimulates osteoblasts to release RANKL → activates osteoclasts → promotes bone resorption when needed.

(E) **T** - CKD causes phosphate retention → hyperphosphatemia → ↑ PTH (secondary hyperparathyroidism).

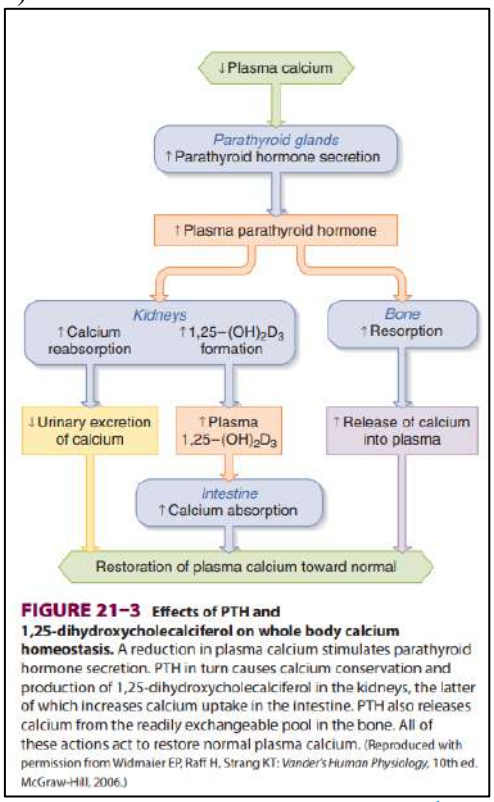


FIGURE 21–3 Effects of PTH and 1,25-dihydroxycholecalciferol on whole body calcium homeostasis. A reduction in plasma calcium stimulates parathyroid hormone secretion. PTH in turn causes calcium conservation and production of 1,25-dihydroxycholecalciferol in the kidneys, the latter of which increases calcium uptake in the intestine. PTH also releases calcium from the readily exchangeable pool in the bone. All of these actions act to restore normal plasma calcium. (Reproduced with permission from Widmaier EP, Raff H, Strang KT: *Vander's Human Physiology*, 10th ed. McGraw-Hill, 2006.)

Ganong's Review of Medical Physiology 24th Edition p. 381

Question (37)

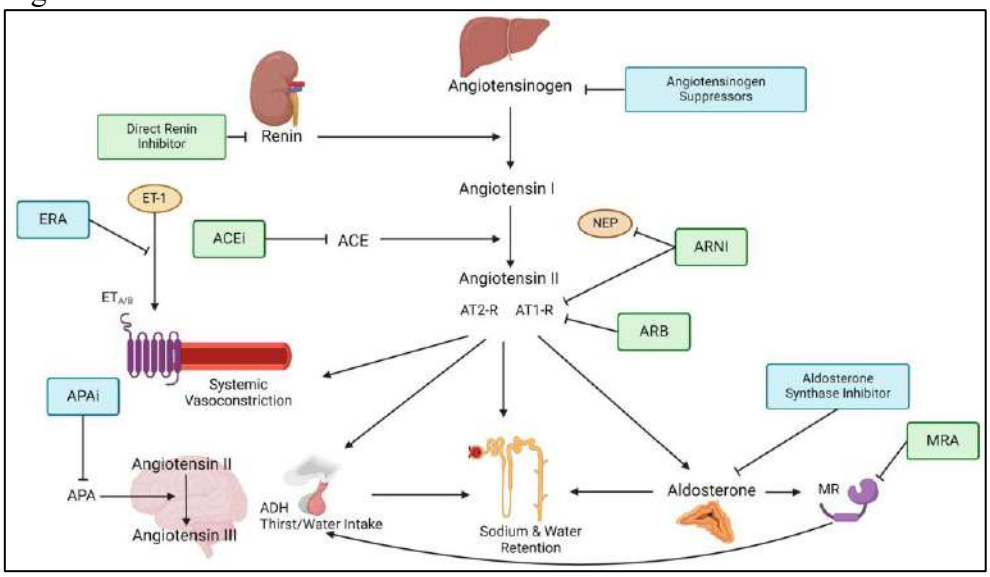
(A) **T** - Low renal perfusion pressure → stretch receptors in JG cells → ↑ renin release.

(B) **T** - Ang II preferentially constricts the efferent arteriole, helping maintain GFR during low renal perfusion.

(C) **T** - ACE is abundant on pulmonary endothelium, making the lungs a major site for angiotensin II generation.

(D) **T** - β_1 receptor activation on JG cells → increased renin release.

(E) **F** - Low NaCl at macula densa → increases renin.
High NaCl → decreases renin



https://www.mdpi.com/1422-0067/25/7/4035

Question (38)

(A) **T** - Ang II preferentially constricts the efferent arteriole, helping maintain GFR during low renal perfusion.

(B) **T** - Aldosterone increases Na^+ reabsorption and K^+ secretion via ENaC and ROMK channels.

(C) **T** - Ang II stimulates thirst, ADH secretion, and vasoconstriction → supports blood pressure and volume.

(D) **F** - Cervical stretch stimulates oxytocin release (Ferguson reflex).

(E) **F** - Continuous GnRH suppresses LH and FSH → used therapeutically in prostate cancer, endometriosis.

TABLE 17–1 Summary of principal hypothalamic regulatory mechanisms.

Function	Afferents from	Integrating Areas
Temperature regulation	Temperature receptors in the skin, deep tissues, spinal cord, hypothalamus, and other parts of the brain	Anterior hypothalamus, response to heat; posterior hypothalamus, response to cold
Neuroendocrine control of:		
Catecholamines	Limbic areas concerned with emotion	Dorsal and posterior hypothalamus
Vasopressin	Osmoreceptors, "volume receptors," others	Supraoptic and paraventricular nuclei
Oxytocin	Touch receptors in breast, uterus, genitalia	Supraoptic and paraventricular nuclei
Thyroid-stimulating hormone (thyrotropin, TSH) via TRH	Temperature receptors in infants, perhaps others	Paraventricular nuclei and neighboring areas
Adrenocorticotropic hormone (ACTH) and β-lipotropin (β-LPH) via CRH	Limbic system (emotional stimuli); reticular formation ("systemic" stimuli); hypothalamic and anterior pituitary cells sensitive to circulating blood cortisol level; suprachiasmatic nuclei (diurnal rhythm)	Paraventricular nuclei
Follicle-stimulating hormone (FSH) and luteinizing hormone (LH) via GnRH	Hypothalamic cells sensitive to estrogens, eyes, touch receptors in skin and genitalia of reflex ovulating species	Preoptic area; other areas
Prolactin via PIH and PRH	Touch receptors in breasts, other unknown receptors	Arcuate nucleus; other areas (hypothalamus inhibits secretion)
Growth hormone via somatostatin and GRH	Unknown receptors	Periventricular nucleus, arcuate nucleus
"Appetitive" behavior:		
Thirst	Osmoreceptors, probably located in the organum vasculosum of the lamina terminalis; angiotensin II uptake in the subfornical organ	Lateral superior hypothalamus
Hunger	Glucostat cells sensitive to rate of glucose utilization; leptin receptors; receptors for other polypeptides	Ventromedial, arcuate, and paraventricular nuclei; lateral hypothalamus
Sexual behavior	Cells sensitive to circulating estrogen and androgen, others	Anterior ventral hypothalamus plus, in the male, piriform cortex
Defensive reactions (fear, rage)	Sense organs and neocortex, paths unknown	Diffuse, in limbic system and hypothalamus
Control of body rhythms	Retina via retinohypothalamic fibers	Suprachiasmatic nuclei

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Question (39)

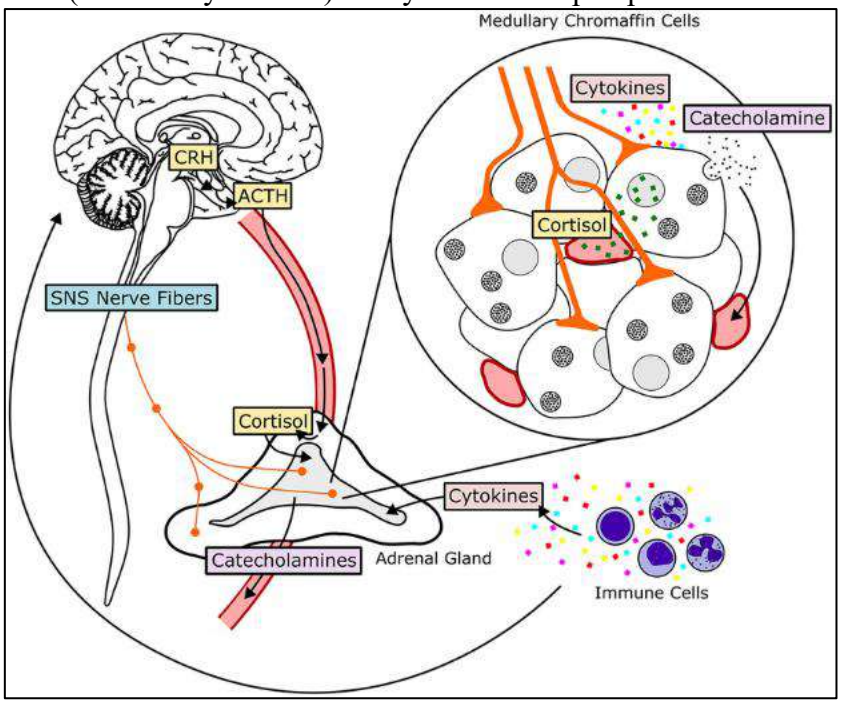
(A) **T** - The adrenal medulla develops from neural crest cells and functions like a sympathetic ganglion, releasing catecholamines into the blood instead of synapses.

(B) **F** - The adrenal medulla secretes ~80% epinephrine and ~20% norepinephrine.

(C) **T** - Catecholamines increase glycogenolysis, lipolysis, and gluconeogenesis, supporting energy needs during stress.

(D) **F** - Dopamine is only a small intermediate in catecholamine synthesis; the major circulating hormones are epinephrine and norepinephrine.

(E) **T** - PNMT (induced by cortisol) catalyzes NE → epinephrine.



https://www.researchgate.net/figure/Hormonal-and-neural-mechanisms-regulating-adrenal-medullary-chromaffin-cells-The_fig3_326038890

Question (40)

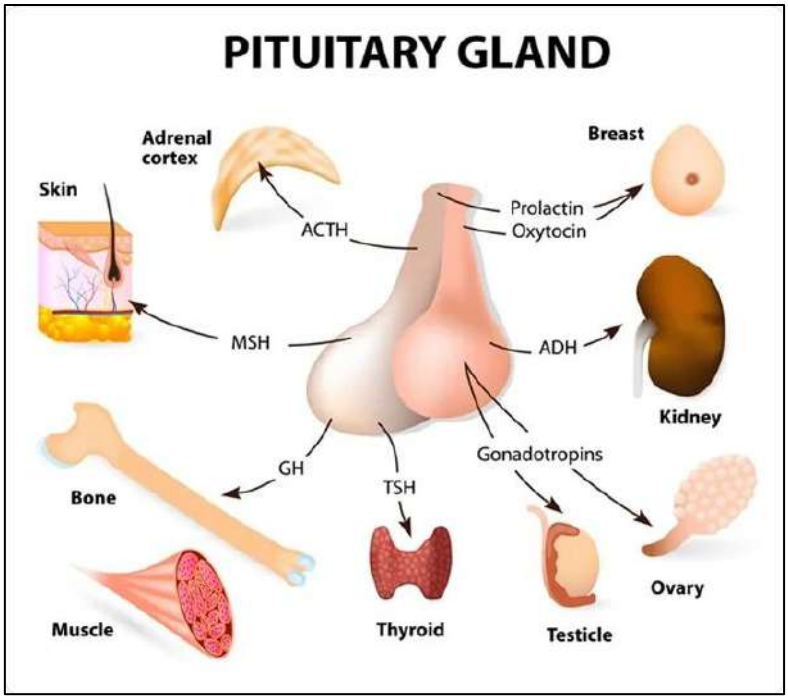
(A) **T** – GH excess before epiphyseal closure → gigantism; after closure → acromegaly.

(B) **F** – Prolactin deficiency is rare; hyperprolactinemia (excess) is a more common cause of infertility, usually due to prolactinomas.

(C) **T** – Secondary hypothyroidism arises from decreased TSH secretion by the pituitary.

(D) **T** – ACTH excess → cortisol excess → mineralocorticoid-like effects, causing hypokalemia and metabolic alkalosis.

(E) **F** – Pan-hypopituitarism is a deficiency of all or most pituitary hormones, not selective.

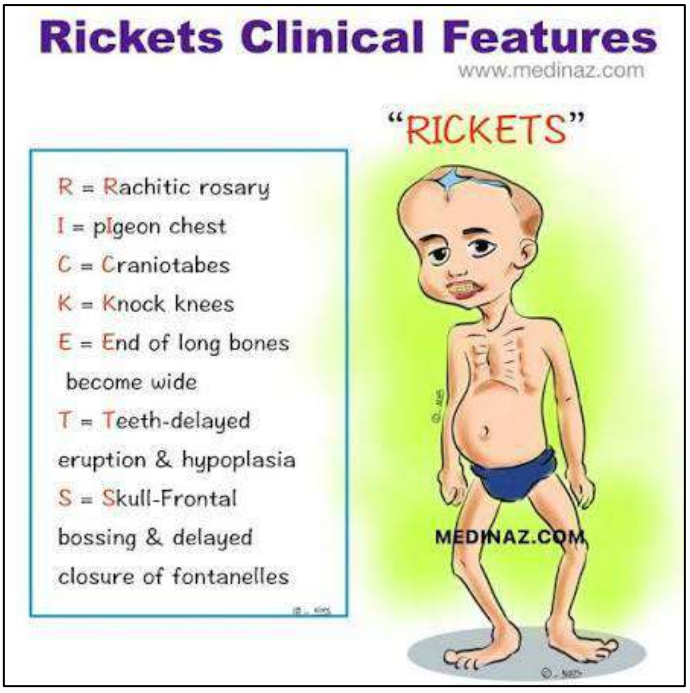


https://www.news-medical.net/health/Pituitary-Gland-Hormones-and-Functions.aspx

Question (41)

Correct answer: B. Increasing absorption of dietary calcium

Vitamin D (1,25 Dihydroxycholecalciferol) is involved in calcium homeostasis in the following ways (1) it stimulates the absorption of dietary calcium ion (2) it facilitates the calcium reabsorption in the kidneys (3) it increases the synthetic activity of osteoblasts (this causes secondary increase in the activity of osteoclasts). When vitamin D is deficient, it causes defective calcification of bone matrix and causes the diseases rickets in children and osteomalacia in adults. Although all three of the above processes are impaired, what affects the bone mineralization the most is the failure to deliver adequate amounts of calcium and phosphate to the sites of mineralization as the dietary absorption of calcium is impaired. Vitamin D does not increase the secretion of phosphate from the kidneys.



https://www.facebook.com/100057931396551/posts/rickets-clinical-features-/1232280505379651/

Question (42)

Correct answer: A. Autoantibodies stimulating the TSH receptor on thyroid follicular cells

Graves' disease is caused by thyroid-stimulating immunoglobulins (TSI) that mimic TSH, leading to diffuse thyroid hyperplasia and excess thyroid hormone production. Pituitary TSH overproduction would cause secondary hyperthyroidism, which is rare. Autonomous nodules and excessive iodine are causes of toxic nodular goiter or iodine-induced hyperthyroidism, not Graves' disease. Autoimmune destruction occurs in thyroiditis, causing transient hyperthyroidism due to hormone leakage, not TSI stimulation.

TABLE 19–1 Binding of thyroid hormones to plasma proteins in normal adult humans.

Protein	Plasma Concentration (mg/dL)	Amount of Circulating Hormone Bound (%)	
		T_4	T_3
Thyroxine-binding globulin (TBG)	2	67	46
Transthyretin (thyroxine-binding prealbumin, TBPA)	15	20	1
Albumin	3500	13	53

Ganong's Review of Medical Physiology 24th Edition p. 343

Question (43)

Correct answer: B. Autoimmune destruction of pancreatic beta cells leading to insulin deficiency

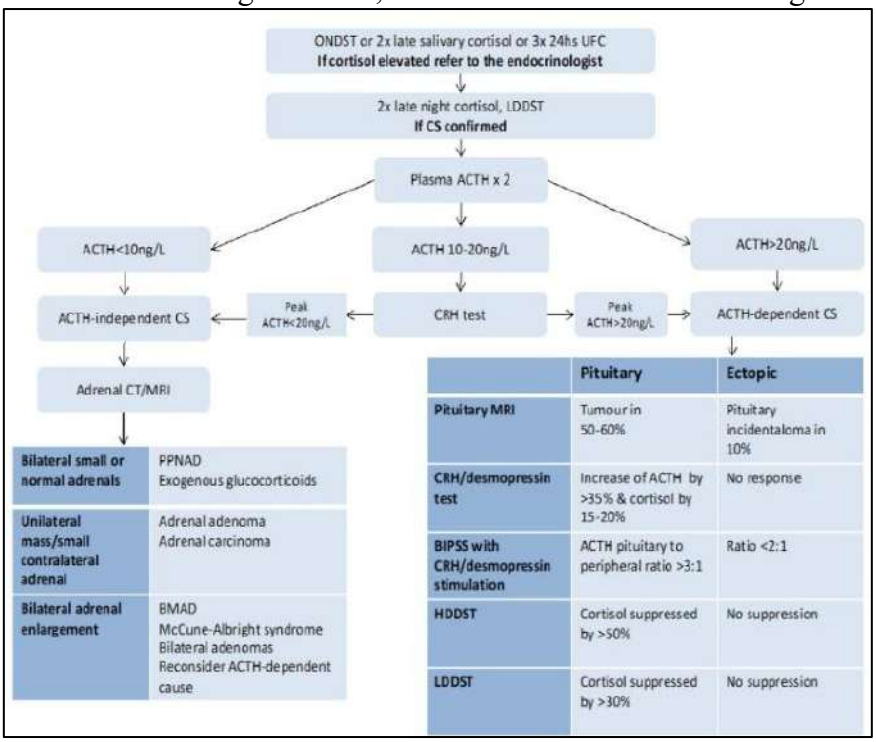
The patient's presentation and positive beta-cell autoantibodies are consistent with type 1 diabetes mellitus, characterized by absolute insulin deficiency due to

autoimmune destruction of pancreatic beta cells. Type 2 diabetes is primarily due to insulin resistance, often with obesity and hyperinsulinemia. Excess hepatic glucose contributes to hyperglycemia in diabetes but is secondary to insulin deficiency/resistance. Renal glucose reabsorption defects are rare and do not cause diabetes. Incretin defects are more relevant in type 2 diabetes and play a minor role in type 1.

Question (44)

Correct answer: A. Primary adrenal adenoma producing cortisol

The patient has Cushing syndrome, evidenced by features of cortisol excess. Suppressed ACTH indicates the source is primary adrenal (adrenal adenoma or carcinoma) producing cortisol autonomously. Pituitary adenoma (Cushing disease) or ectopic ACTH would cause elevated ACTH. Exogenous glucocorticoid use (choice D) also suppresses ACTH but must be confirmed via history. Congenital adrenal hyperplasia usually presents in childhood with enzyme deficiencies affecting cortisol synthesis and adrenal androgen excess, not isolated adult-onset Cushingoid features.



https://www.ncbi.nlm.nih.gov/books/NBK279088/figure/cushings-disease.F3/

Question (45)

Correct Answer: C. Hyporeninemic hypoaldosteronism

The patient presents with muscle weakness, hypotension, hyponatremia, and hyperkalemia, which are classic signs of aldosterone deficiency. Laboratory findings:

- Low plasma aldosterone → confirms aldosterone deficiency.
- Elevated plasma renin activity → indicates that the kidneys are trying to compensate for low aldosterone.

Hyporeninemic hypoaldosteronism is typically caused by impaired renin release or reduced adrenal responsiveness, often seen in diabetic nephropathy or chronic kidney disease. This leads to:

- Reduced sodium reabsorption → hyponatremia and hypotension
- Reduced potassium excretion → hyperkalemia

PHYSIOLOGY SGD & TUTE QUESTIONS & ANSWERS

Mechanics of Breathing

(1) Describe the changes in the intrapleural pressure and alveolar pressure during a normal respiratory cycle. (50 marks)

- Respiratory cycle is the sequence of events that occurs from the beginning of one inspiration
- to the beginning of the next inspiration.
- Therefore, a single respiratory cycle includes inspiration and expiration stages.
- Lungs inflates during inspiration and deflates during the expiration stages.
- Normally lungs are covered by pleura which has got 2 layers.
- The visceral layer lines the outer surface of the lungs. The parietal layer lines the inner
- surface of the chest wall.
- There is a potential space between the 2 layers as the intrapleural space.
- The pressure in this space is called intrapleural pressure.
- The pressure inside the alveoli is called alveoli pressure.
- The intrapleural pressure remains sub atmospheric throughout the respiratory cycle in
- normal healthy individuals.
- However, these pressures subjects to various changes in their values throughout the
- respiratory cycles.
- During the inspiration thoracic cavity expands as a result of the contraction of diaphragm
- And external intercostal muscles.
- This pulls the parietal pleura that of the chest wall along with the thoracic wall thus
- increasing the negativity of the intrapleural pressure.
- During inspiration the intrapleural space at the lung base is normally 2.5mmHg at the start
- and gradually the negativity increases up to the –6mmHg during quite inspiration.
- This creates a pressure gradient.
- As a result, lungs also expand thus alveoli pressure also slightly becomes negative during
- inspiration.
- When air enters to the alveoli from outside along the pressure gradient that created.
- When the air fills the negativity of the alveolar pressure reduces.
- When the alveolar pressure becomes equal to the atmospheric pressure inspiration stops
- and when it becomes slightly positive expiration starts.
- The alveolar pressure fluctuates between –1mmHg to +1mmHg throughout the respiratory
- cycle and becomes zero; equals to atmospheric pressure at the end of expiration and
- inspiration.

- During expiration lungs recoils thus increasing the alveolar pressure. This pulls the chest wall
- back into the expiratory position.
- As a result, the intrapleural pressure as the lung base returns to –2.5mmHg and the
- expiration stops when the alveolar pressure becomes equal to the atmospheric pressure.

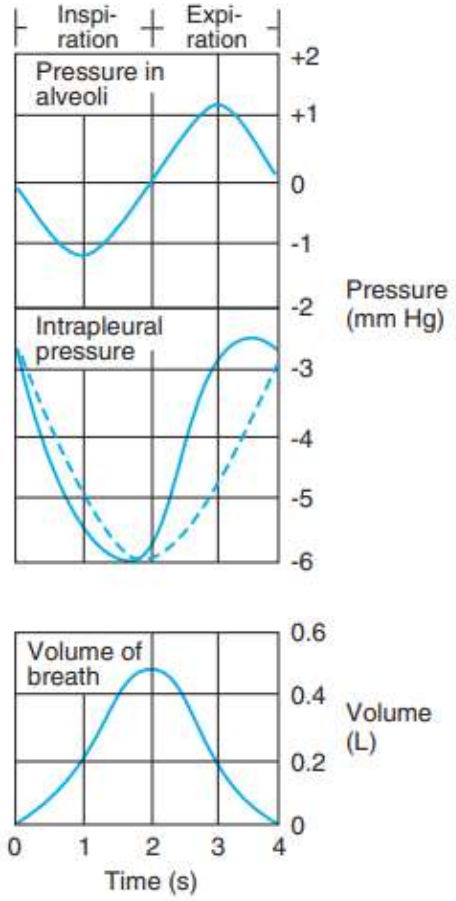


FIGURE 34–7 Pressure in the alveoli and the pleural space relative to atmospheric pressure during inspiration and expiration. The dashed line indicates what the intrapleural pressure would be in the absence of airway and tissue resistance; the actual curve (solid line) is skewed to the left by the resistance. Volume of breath during inspiration/expiration is graphed for comparison.

Ganong's Review of Medical Physiology 24th Edition p. 628

Gas Exchange & Ventilation

(1) Write short notes on the following
(1.1) CO_2 transport in blood
(1.2) Diffusing capacity of lung
(1.3) Tense and relaxed configuration of hemoglobin

(1)

(1.1) The ways of CO_2 transport in blood. (40)

- CO_2 is transported from tissues to lungs via blood mainly in 3 ways
- 10% of CO_2 is transported in dissolved state in plasma
- Its solubility is 20 times that of O_2
- 25% of CO_2 is transported as carbamino compounds
- Here CO_2 binds to amino groups of hemoglobin and other plasma proteins
- 65% of CO_2 is transported as HCO_3^- after hydration
- (state most CO2 is transported as HCO_3^- or percentage)
- In tissues CO_2 tension is greater than that of blood
- Therefore, CO_2 enters blood
- CO_2 that enters RBC is readily hydrated to H_2CO_3 by carbonic anhydrase enzyme
- H_2CO_3 is disassociated to H^+ and HCO_3^- , H^+ buffered by hemoglobin
- In the lungs tension of O_2 is high and CO_2 is low
- Binding of O_2 to Hemoglobin reduces its affinity to CO_2 (Haldane Effect)
- So, CO_2 is discharged to alveoli and removed via expiration

TABLE 35–2 Fate of CO_2 in blood.

In plasma
1. Dissolved
2. Formation of carbamino compounds with plasma protein
3. Hydration, H^+ buffered, HCO_3^- in plasma
In red blood cells
1. Dissolved
2. Formation of carbamino-Hb
3. Hydration, H^+ buffered, 70% of HCO_3^- enters the plasma
4. Cl^- shifts into cells; mOsm in cells increases

Ganong's Review of Medical Physiology 24th Edition p. 645

(1.2) Briefly explain the diffusing capacity. (30)

- Gases defuse from alveoli to the blood in the pulmonary capillaries or vice versa.
- Across the thin alveocapillary membrane made up of the
  - Pulmonary Epithelium
  - Capillary endothelium and
  - their fused basement membranes.

- The diffusing capacity of the lung for a given gas is directly proportionate to the surface area of the alveocapillary membrane and
- Inversely proportionate to its thickness
- The diffusing capacity for CO [DLco] is used as an index of diffusing capacity because its uptake is diffusion limited
- DLco is proportionate to the amount of CO entering blood [Vco] divided by
- Partial pressure of CO in blood entering pulmonary capillaries.
- Therefore, diffusing capacity of a gas can be defined as
  - Amount of gas entering capillary from alveoli, per unit difference of partial pressure of that gas in alveoli and capillary.
- In measuring DLco , partial pressure of CO in capillary can be ignored,
- Except in habitual smokers.
- Therefore DLco = Vco/PAco
- DLco increases up to threshold in exercise due to
  - Capillaries dialution and
  - Increase in number of active capillaries
- Diffusing capacity of CO is about 20 times of O_2
- In lung fibrosis diffusing capacity is decreased however
- Even in the cases CO_2 retentron is not a problem because of the high diffusing capacity.

(1.3) Explain about tense and relaxed configurations of haemoglobin. (15)

- Deoxyhaemoglobin has tense configuration
- And it has less affinity to oxygen after binding one oxygen molecule
- Its affinity to other oxygen molecules increased
- And this is called relaxed configuration of haemoglobin
- Cyanosis is a dusky, bluish discolouration of
  - Skin and
  - Mucous membranes due to
  - Circulation deoxygenated haemoglobin concentration of capillary blood
  - More than 5 g/dl
- This depends on 3 main factors
  - State of capillary circulation
  - Total haemoglobin concentration
  - Degree of unsaturation
- Cyanosis is of two types
  - Central
  - Peripheral
- Discolouration of tongue is due to central cyanosis
- [Hb] is within the normal range
- So, the most likely the cause of cyanosis is increased degree of unsaturation
- When Po2 in arterial blood falls saturation of Hb reduces
- This is a consequence of
- Hypoxic hypoxia which can be due to Reduction in alveolar ventilation
- When it exceeds a certain degree
- Deoxygenated Hb level reaches 5 g/dl
- This causes central cyanosis which is reflected as discolouration of tongue

(2) Describe the physiological basis for the variations of V/Q ratio in different parts of the lung.

- For the whole lung V/Q ratio is around 0.8 this refers to the
- Alveolar ventilation/right ventricular outflow
- However, there are marked regional differences in the V/Q ratio of a healthy upright person due to the effect of gravity
- At the apex the intrapleural pressure is increased due to gravity
- This causes an increased intrapleural intraalveolar pressure difference
- Which causes the expansion of the alveoli to become increased at the apex
- Due to this the expansion of the alveoli per unit pressure or compliance is decreased
- This causes ventilation at the apex to become reduced
- Since the apices of the lungs are situated above the level of the heart
- The blood pressure within the pulmonary vessels is low
- If these pulmonary vessels have a lower pressure than that of the alveoli then
- They may also collapse during certain parts of the cardiac cycle
- This causes perfusion to be reduced at the apex
- At the bases of the lungs the intrapleural pressure is decreased due to gravity
- This causes a decreased intrapleural intraalveolar pressure difference
- Which causes the expansion of the alveoli at the base to become reduced
- Resulting in smaller alveoli
- Due to this expansion of alveoli per unit change in pressure (compliance) is increased
- This resulting in an increase in ventilation
- Since the bases of the lungs are at a level lower than the heart
- The blood pressure within the pulmonary capillaries is increased
- The pulmonary arterial pressure is greater than the alveolar air pressure at all times of the cardiac cycle
- Thus, causing blood to flow throughout the cycle
- Therefore, perfusion is also increased at the base
- Therefore, both the ventilation and perfusion increase from the bottom to the top of the lung
- However, the perfusion increases rapidly than the ventilation
- Causing a lower V/Q ratio at the base and
- A higher V/Q ratio at the apex

(3) An increased physiological dead space leads to an abnormal V/Q ratio. (30)

- The V/Q ratio determines gas exchange in any single lung unit.
- It indicates matching of ventilation with blood flow.
- Normal V/Q is nearly 0.8.
- Physiological dead space is the total volume of air not equilibrating with blood.
- Physiological dead space = Anatomical dead space + Volume of alveoli which are ventilated but not perfused.
- Increased physiological dead space can be caused by
- Increased amount of ventilated but not perfused alveoli.
- So, it results in increased V/Q ratio.
- Abnormal V/Q ratio is the most common cause for hypoxic hypoxia.

Neonatal Respiratory Distress Syndrome

(1) A preterm baby is born at 32 weeks of gestation by normal vaginal delivery. The baby weighs 1.2kg. Few minutes after birth the baby has a respiratory rate of 70 (normal respiratory rate for a new born baby is 40-60), is grunting with intercostal recessions. Baby is also cyanosed. During auscultation the baby shows reduced air entry bilaterally. Baby is transferred to Neonatal Intensive Care Unit (NICU) where X Ray is taken. Baby is connected to a ventilator. After considering the history, examination findings and investigation results, the pediatrician decides to start surfactant therapy for the baby.

(1.1) What is the most probable diagnosis and give reasons for the diagnosis?

(1.2) Explain the physiological basis of,

a. Increased respiratory rate
b. Grunting and intercostal recessions
c. Cyanosis
d. Bilateral reduced air entry

(1.3) Describe the composition of surfactants.

(1.4) Describe the physiological functions of surfactants.

(1.5) Describe the histological features of type II alveolar cells and explain their role in surfactant production. How does the structure of these cells facilitate their function?

(1.6) Explain how the lung maturity of the fetus can be gauged by determining the ratio of DPCC to sphingomyelin.

Following day, after receiving surfactant therapy, the baby showed a marked improvement in respiratory parameters. It was decided to wean off the ventilatory support. By the end of the second day baby was put on CPAP.

(1.7) Discuss the different types of ventilatory support available in the hospital for patients with impaired ventilation.

(1.1) What is the most probable diagnosis and give reasons for the diagnosis?

Neonatal respiratory distress syndrome

When babies are born prematurely, they have insufficient surfactants produced in their lungs. This causes high surface tension in the alveoli, leading to alveolar collapse and reduced functional capacity of the lung. Therefore, these babies show signs of respiratory distress after birth which is improved with surfactant therapy.

(1.2) Explain the physiological basis of,

a. Increased respiratory rate

Due to low lung compliance and inadequate oxygenation, the infant increases respiratory rate to improve oxygen intake and eliminate carbon dioxide.

b. Grunting and intercostal recessions

Grunting: This is an expiratory sound made by the baby which shows respiratory distress due to lung collapse

Intercostal recessions: Occur as the infant's accessory muscles work harder to expand the poorly compliant lungs, leading to visible retractions.

c. Cyanosis

Cyanosis results from inadequate oxygenation of the blood, caused by impaired gas exchange in the atelectatic lungs.

d. Bilateral reduced air entry

Reflects collapsed alveoli and poor ventilation distribution, a consequence of surfactant deficiency

(1.3) Describe the composition of surfactants.

Surfactant is a complex mixture of lipids (90%) and proteins (10%), with DPPC (dipalmitoyl - phosphatidylcholine (DPPC, or dipalmitoyl lecithin) being the major component for reducing surface tension. In DPPC, positions 1 and 2 on the glycerol are occupied by palmitate.

DPPC, made and secreted by Type II pneumocytes, is the major lipid component of lung surfactant—the extracellular fluid layer lining the alveoli. Surfactant serves to decrease the surface tension of this fluid layer, reducing the pressure needed to reinflate alveoli, thereby preventing alveolar collapse (atelectasis).

(1.4) Describe the physiological functions of surfactants.

Reduction of Surface Tension:

Surfactant significantly lowers the surface tension at the air-liquid interface within alveoli, preventing alveolar collapse during expiration. This reduction in tension minimizes the work required for lung inflation, thus improving respiratory efficiency.

Increased Lung Compliance:

By reducing surface tension, surfactant allows the lungs to remain more elastic and compliant, enabling easier expansion during inhalation. This compliance reduces the effort needed for breathing and is crucial for proper ventilation.

Stabilization of Alveolar Size:

Surfactant helps maintain uniform alveolar size and prevents smaller alveoli from collapsing into larger ones. This stabilization is crucial to maintain consistent gas exchange throughout the lung tissue.

Prevention of Alveolar Fluid Accumulation:

Surfactant reduces the tendency of fluid to move into the alveolar spaces from the surrounding capillaries. This keeps the alveoli dry, which is vital for efficient gas exchange.

Role in Host Defense:

Surfactant also plays a role in the innate immune system. It helps trap and clear pathogens from the lungs and modulates the immune response to limit inflammation, reducing the risk of lung injury.

(1.5) Describe the histological features of type II alveolar cells and explain their role in surfactant production. How does the structure of these cells facilitate their function?

Type II alveolar cells, also known as pneumocytes, are cuboidal epithelial cells found within the alveolar walls of the lungs. These cells are responsible for the synthesis and secretion of pulmonary surfactant. Type II cells contain unique organelles called lamellar bodies, which store surfactant components

before they are secreted into the alveolar space. The lamellar bodies facilitate surfactant recycling and help maintain alveolar stability by continuously producing and replenishing surfactant. The abundant mitochondria and endoplasmic reticulum in these cells support their extensive lipid and protein synthesis, vital for surfactant production.

(1.6) Explain how the lung maturity of the fetus can be gauged by determining the ratio of DPCC to sphingomyelin.

Lung maturity of the fetus can be gauged by determining the ratio of DPPC to sphingomyelin, usually written as the L (for lecithin)/S ratio, in amniotic fluid. A ratio of two or above is evidence of maturity, because it reflects the major shift from sphingomyelin to DPPC synthesis that occurs in the pneumocytes at about 32 weeks of gestation.

(1.7) Discuss the different types of ventilatory support available in the hospital for patients with impaired ventilation.

Non-invasive Support:

(1) Nasal CPAP: Delivers continuous positive airway pressure to keep alveoli open.

(2) High-Flow Nasal Cannula: Provides warmed and humidified oxygen.

Invasive Ventilation:

- Mechanical Ventilation: For severe cases, endotracheal intubation is performed to deliver controlled breaths.

High-Frequency Oscillatory Ventilation (HFOV): Used in cases of refractory RDS to provide small, rapid breaths at high frequencies.

Hypoxia

(1) Different situations or disease conditions can lead to hypercapnic hypoventilation (elevated arterial CO_2). It can occur in:
   a) Patients with obstructive lung disease
   b) Patients with neuromuscular disorders affecting respiration
   c) Patients with chest wall deformities
   d) Patients with central respiratory depression

   (1.1) State one example for each of the situations / disease conditions (a–d) stated above. (10 marks)

   (1.2) Explain the mechanism of development of hypercapnic hypoventilation in each of the situations / disease conditions (a–d) stated above. (40 marks)

   (1.3) Explain whether ventilatory support (e.g., non-invasive ventilation or mechanical ventilation) is useful or not useful in each of the situations / disease conditions (a–d) stated above. (30 marks)

   (1.4) State the expected type of derangement of pH in the above salutations (20 marks)

(1)

(1.1) Examples (10 marks)

a) Obstructive lung disease → COPD (2.5)
b) Neuromuscular disorder → Guillain-Barré syndrome (2.5)
c) Chest wall deformity → Kyphoscoliosis (2.5)
d) Central respiratory depression → Opioid overdose (2.5)

(1.2) Mechanism of Hypercapnic Hypoventilation (40 marks)

a) Obstructive lung disease (10)

Chronic airway obstruction → increased airway resistance → reduced alveolar ventilation → CO_2 retention → hypercapnia.

b) Neuromuscular disorder (10)

Weak respiratory muscles → decreased tidal volume → hypoventilation → CO_2 retention → respiratory acidosis.

c) Chest wall deformity (10)

Reduced chest wall compliance → impaired thoracic expansion → decreased alveolar ventilation → CO_2 retention → hypercapnia.

d) Central respiratory depression (10)

CNS depression (opioids, sedatives, brainstem lesions) → reduced respiratory drive → hypoventilation → CO_2 retention → respiratory acidosis.

(1.3) Usefulness of Ventilatory Support (30 marks)

a) Obstructive lung disease → Useful: Non-invasive ventilation (NIV) assists alveolar ventilation during exacerbations. (7.5)
b) Neuromuscular disorder → Useful: Mechanical or non-invasive ventilation compensates for weak muscles. (7.5)
c) Chest wall deformity → Useful: NIV improves alveolar ventilation and gas exchange. (7.5)
d) Central respiratory depression → Useful if reversible (e.g., drug-induced); may require urgent mechanical ventilation. (7.5)

(1.4) Expected pH Derangement (20 marks)

a) Obstructive lung disease → Respiratory acidosis (acute or chronic) due to CO_2 retention. (5)
b) Neuromuscular disorder → Respiratory acidosis due to hypoventilation. (5)
c) Chest wall deformity → Respiratory acidosis due to reduced alveolar ventilation. (5)
d) Central respiratory depression → Respiratory acidosis due to decreased respiratory drive. (5)

(2)

(2.1) Describe the role of peripheral and central chemoreceptors in the regulation of respiration. (40 marks)
(2.2) Explain how non-chemical influences such as lung reflexes, higher brain centers, and proprioceptors contribute to the regulation of respiration. (30 marks)
(2.3) State the changes in ventilation that would occur in the following situations and explain the mechanisms:
a) Exercise
b) High altitude exposure
c) Sleep

(30 marks)

(2)

(2.1) Role of Chemoreceptors (40 marks)

- Central Chemoreceptors (20 marks)
- Located on ventrolateral surface of medulla. (2)
- Sensitive mainly to H^+ concentration in brain ECF/CSF. (4)
- CO_2 diffuses across blood-brain barrier → forms carbonic acid → dissociates → $\uparrow H^+$ → stimulates receptors. (6)
- ↑ Ventilation when $PaCO_2$ rises (powerful short-term regulator). (6)
- Key point: Not directly sensitive to arterial hypoxemia. (2)
- Peripheral Chemoreceptors (20 marks)
- Located in carotid bodies & aortic bodies. (4)
- Respond mainly to $\downarrow PaO_2$ (< 60 mmHg), also $\uparrow PaCO_2$ and $\uparrow H^+$. (6)
- Signal via glossopharyngeal (carotid) & vagus (aortic) nerves to medullary respiratory centers. (4)
- ↑ Ventilation rapidly in hypoxemia, metabolic acidosis, or hypercapnia. (6)

(2.2) Role of Non-Chemical Influences (30 marks)

- Lung Reflexes (10 marks)
- Hering–Breuer inflation reflex: stretch receptors → vagus → inhibit inspiration (protects from overinflation). (5)
- Irritant receptors: respond to smoke/dust → cough, bronchoconstriction, ↑ventilation. (5)
- Higher Brain Centers (10 marks)
- Voluntary control from cortex: breath-holding, hyperventilation (limited by chemical drive). (5)

- Hypothalamus/limbic system: emotions, stress, temperature → alter breathing pattern. (5)
- Proprioceptors & Other Inputs (10 marks)
- Muscle/joint proprioceptors: ↑ventilation at exercise onset (feedforward mechanism). (5)
- Pain & temperature receptors: ↑ventilation. (5)

(2.3) Ventilation in Physiological Situations (30 marks)

a) Exercise (10 marks)

Immediate ↑ventilation due to proprioceptors & motor cortex signals. (4)

Later sustained ↑ due to ↑CO_2, ↑temperature, ↑K^+, catecholamines. (4)

Overall results in ↑ tidal volume & frequency, $PaCO_2$ remains near normal. (2)

b) High Altitude (10 marks)

↓Barometric pressure → ↓PaO_2 → hypoxemia → carotid body stimulation → hyperventilation. (5)

Respiratory alkalosis develops, compensated by renal HCO_3^- excretion. (5)

c) Sleep (10 marks)

↓Metabolic rate → ↓ventilation (esp. during non-REM sleep). (3)

Chemoreceptor sensitivity ↓ (blunted response to hypoxia/hypercapnia). (4)

Irregular breathing patterns during REM sleep. (3)

Digestion of Fat

(1) A 34-year-old male presents to the clinic with complaints of frequent bloating, greasy stools, and unexplained weight loss over the past three months. He mentions that he recently began following a high-fat diet as part of a fitness program. On physical examination, his BMI is 18.5 kg/m², and his skin appears dry. Basic lab investigations reveal low levels of serum triglycerides and fat-soluble vitamins (A, D, E, and K). He is also found to have high values for Oral Glucose Tolerance Test (OGTT). Stool analysis confirms steatorrhea, and further imaging indicates pancreatic insufficiency.
The patient is diagnosed with malabsorption secondary to chronic pancreatitis.

(1.1) Describe the role of bile salts in fat digestion. How are they recycled in the enterohepatic circulation?

(1.2) Explain how pancreatic lipase and colipase contribute to fat breakdown in the duodenum.

(1.3) What are the physiological mechanisms underlying steatorrhea in pancreatic insufficiency?

(1.4) What is the reason for high OGTT in this patient?

(1.1) Describe the role of bile salts in fat digestion. How are they recycled in the enterohepatic circulation?

Bile salts are essential for efficient digestion and absorption of dietary fats, especially long-chain triglycerides.

Their main functions are:

1. Emulsification of fats
   - Bile salts are amphipathic molecules (hydrophilic + hydrophobic ends).
   - They reduce surface tension and break large fat globules into small emulsified droplets, greatly increasing the surface area available for pancreatic lipase.
2. Micelle formation
   - Products of fat digestion (free fatty acids, monoglycerides, cholesterol, fat-soluble vitamins A, D, E, K) are poorly water-soluble.
   - Bile salts form mixed micelles that:
     - Solubilize these lipids
     - Transport them across the unstirred water layer to the intestinal brush border
   - Without bile salts, lipid absorption is severely impaired → steatorrhea.

- About 95% of bile salts are reabsorbed in the terminal ileum by active Na^+-dependent transport.
- Reabsorbed bile salts enter the portal circulation and return to the liver.
- Hepatocytes extract bile salts efficiently and re-secrete them into bile.
- This recycling process occurs 6–10 times per day, conserving bile salt pool.

Physiological importance:

- Maintains an adequate bile salt concentration
- Prevents excessive hepatic synthesis of bile salts

- Ensures continued fat digestion and absorption

(1.2) Explain how pancreatic lipase and colipase contribute to fat breakdown in the duodenum.

Pancreatic lipase

- The principal enzyme for triglyceride digestion.
- Acts at the oil–water interface of emulsified fat droplets.
- Hydrolyzes triglycerides at the 1 and 3 positions, producing:
  - 2 free fatty acids
  - 1 monoglyceride

However, bile salts can inhibit lipase activity by displacing it from the fat droplet surface.

Colipase

- Secreted by the pancreas as procolipase, activated by trypsin.
- Colipase binds to both:
  - Pancreatic lipase
  - The lipid droplet surface
- This anchoring effect:

Prevents bile salts from displacing lipase

Restores and enhances lipase activity

Together, pancreatic lipase + colipase ensure efficient triglyceride digestion in the duodenum.

(1.3) What are the physiological mechanisms underlying steatorrhea in pancreatic insufficiency?

In chronic pancreatitis, there is progressive destruction of exocrine pancreatic tissue, leading to:

Key mechanisms of steatorrhea

1. Reduced pancreatic lipase secretion
   - Lipase deficiency is the earliest and most critical defect.
   - Fat digestion is far more sensitive to enzyme loss than protein or carbohydrate digestion.
2. Impaired triglyceride hydrolysis
   - Triglycerides remain largely undigested.
   - Cannot form absorbable micelles effectively.
3. Failure of micelle formation
   - Even if bile salts are present, absence of fatty acid and monoglyceride generation limits micelle formation.
4. Malabsorption of fat-soluble vitamins
   - Leads to deficiencies of vitamins A, D, E, and K, explaining:
     - Dry skin (vitamin A deficiency)
     - Other systemic features
5. Excess fat in stool
   - Undigested fats remain in the intestinal lumen
   - Produces bulky, pale, greasy, foul-smelling stools → steatorrhea

(1.4) What is the reason for high OGTT in this patient?

The elevated OGTT reflects endocrine pancreatic dysfunction.

Physiological basis

- Chronic pancreatitis damages both exocrine and endocrine components of the pancreas.
- Progressive destruction of β-cells in the islets of Langerhans leads to:
    - Reduced insulin secretion
    - Impaired glucose uptake by tissues

Consequences

- Post-prandial hyperglycemia
- Delayed glucose clearance during OGTT
- Development of pancreatogenic diabetes (Type 3c diabetes)

This explains:

- High OGTT values
- Weight loss
- Coexistence of malabsorption and glucose intolerance

Respiratory Failure

(1) A 72-year-old man presents to the emergency department with severe shortness of breath and cyanosis. He has a history of chronic heart failure. On examination, he has a respiratory rate of 28 breaths per minute, oxygen saturation of 82% on room air, and bilateral crackles on auscultation. Arterial blood gas analysis shows PaO_2 of 55 mmHg and $PaCO_2$ of 35 mmHg.

(1.1) Identify the type of respiratory failure in this patient and explain the underlying pathophysiology.

(1.2) Discuss the most likely causes and outline the initial management.

(1)

(1.1) Identify the type of respiratory failure and explain the pathophysiology

- Type I Respiratory Failure (Hypoxemic Respiratory Failure)
- Underlying Pathophysiology (related to chronic heart failure)
  - In left-sided congestive heart failure, there is:
  - ↑ Pulmonary venous pressure
  - → Pulmonary capillary hydrostatic pressure rises
  - → Fluid moves into interstitial space and alveoli (pulmonary edema)
  - This leads to:
    i. Impaired diffusion of oxygen
      - Alveoli filled with fluid → ↓ surface area for gas exchange
      - O_2 diffuses poorly across fluid-filled alveoli
      - CO_2 diffuses 20× faster than O_2 → remains normal or low

    ii. Ventilation–perfusion (V/Q) mismatch
      - Some alveoli are perfused but not ventilated properly
      - → Shunt-like effect → refractory hypoxemia

    iii. Tachypnea causes low/normal $PaCO_2$
      - Patient hyperventilates in response to hypoxemia
      - → blows off CO_2
      - → $PaCO_2$ stays normal or slightly low

(1.2) Most likely causes & initial management

- Most likely causes (in this patient)
  - The scenario strongly suggests acute decompensated left-sided heart failure causing pulmonary edema.
- Likely triggers include:
  - Myocardial ischemia / acute coronary syndrome
  - Poor medication compliance (e.g., missed diuretics)
  - Arrhythmias (atrial fibrillation, tachyarrhythmias)
  - Infections (pneumonia → increased metabolic demand)
  - Uncontrolled hypertension
  - Renal failure → fluid overload
- Initial Management (Emergency Department)

  i. Oxygen therapy
  - Start high-flow oxygen (target SpO_2 92–96%)
  - If not improving → non-invasive ventilation (NIV)
    - CPAP improves oxygenation by:
      - ↑ Functional residual capacity

- ↓ Work of breathing
- ↓ Preload/afterload (helpful in CHF)

ii. Diuretics
- IV furosemide → reduces pulmonary congestion
- Mobilizes fluid → improves oxygenation

.

iii. Vasodilators (if BP allows)
- IV nitrates (e.g., GTN)
  - ↓ Preload
  - ↓ Pulmonary congestion
  - ↓ Afterload if hypertensive

iv. Treat precipitating factors
- Rate/rhythm control if arrhythmia
- Manage myocardial ischemia
- Treat infection if suspected
- Control BP if hypertensive crisis

v. Consider assisted ventilation
- If the patient deteriorates or cannot maintain oxygenation:
  - BiPAP/CPAP
  - If severe distress or exhaustion → intubation and mechanical ventilation

(2) A 52-year-old woman presents to the emergency department with increasing shortness of breath and difficulty speaking over the past 12 hours. She has a history of generalized myasthenia gravis (MG), for which she is on pyridostigmine and prednisone. On examination, she appears fatigued, with ptosis, dysarthria, and reduced strength in her proximal limb muscles. Her respiratory rate is 28 breaths per minute, and she is using accessory muscles of respiration.

Arterial blood gas (ABG) analysis shows the following:

pH: 7.31
$PaCO_2$: 58 mmHg
PaO_2: 62 mmHg
HCO_3^-: 28 mmol/L

(2.1) What is the most likely cause of her current respiratory failure?

(2.2) What clinical and laboratory assessments should be performed to confirm the diagnosis and assess disease severity?

(2.3) What immediate management strategies should be implemented to stabilize her condition?

(2.4) What role does non-invasive or invasive ventilation play in managing respiratory failure associated with myasthenia gravis?

(2)

(2.1) Most likely cause of her respiratory failure
- Type II (hypercapnic) respiratory failure due to respiratory muscle fatigue from a Myasthenic Crisis

(2.2) Clinical and laboratory assessments (to confirm diagnosis & severity)
- Bedside Clinical Assessments

i. Vital capacity (VC)
- Most important measure of impending respiratory failure.
- VC < 15–20 mL/kg is a red flag requiring ventilatory support.
- VC decreases with respiratory muscle weakness.

ii. Negative Inspiratory Force (NIF)
- NIF worse than –20 to –30 cm H_2O = significant weakness.
- Reflects reduced muscle force, consistent with muscle fatigue

iii. Observation of work of breathing
- Accessory muscle use
- Paradoxical breathing
- Fatigue with speaking (already present)

iv. Bulbar assessment
- Speech, gag reflex, cough strength → indicates risk of aspiration.

- Laboratory / Diagnostic Confirmation

i. ABG (already done)
- Hypercapnia confirms hypoventilation → consistent with neuromuscular failure

ii. Repetitive nerve stimulation / single-fiber EMG
- Shows characteristic MG decrement at neuromuscular junction.

iii. Serum anti-AChR antibodies
- Not needed acutely but confirms disease.

iv. Chest imaging
- To rule out pneumonia/aspiration which can precipitate crisis.

(2.3) Immediate management to stabilize her condition

i. Airway & Breathing Support
- Administer oxygen
- Prepare for ventilatory support (see section 2.4)

ii. Stop or reduce pyridostigmine temporarily
- It increases secretions and can worsen respiratory distress during crisis.

iii. Start definitive therapy

Either:

a. IV immunoglobulin (IVIG) or
b. Plasmapheresis

iv. Continue steroids (prednisone)
- But understand that steroids can transiently worsen weakness; monitor closely.

v. Identify and treat triggers
- Common triggers: infection, stress, surgery, medication changes, antibiotics like aminoglycosides.

(2.4) Role of non-invasive and invasive ventilation in MG respiratory failure

- Non-invasive ventilation (NIV)
  - NIV (BiPAP) may be used early in patients who are awake, cooperative, and not severely weak, to unload fatigued respiratory muscles.
  - When respiratory muscles weaken, assisted ventilation reduces CO_2 and work of breathing
- However, NIV is NOT appropriate if:

- VC < 15 mL/kg
- Severe bulbar weakness (risk of aspiration)
- Copious secretions
- Altered consciousness

- These warrant intubation instead.
- Invasive Ventilation
  - Indications include:
    - Worsening hypercapnia
    - Rapidly falling VC
    - Weak cough, inability to clear secretions
    - Bulbar dysfunction
    - Exhaustion
  - Mechanism
    - Mechanical ventilation replaces failing respiratory muscles, restoring adequate alveolar ventilation and normalizing gas exchange.

Esophagus

(1) Discuss the functional importance of upper and lower esophageal sphincters.

- Functional Importance of the Upper and Lower Esophageal Sphincters

  The esophagus contains two physiologically important sphincters—the Upper Esophageal Sphincter (UES) and the Lower Esophageal Sphincter (LES)—which maintain a unidirectional flow of food and protect the airway and stomach. These sphincters act as zones of high resting pressure that prevent unwanted movement of air or gastric content.

- Upper Esophageal Sphincter (UES)
  - Location: Junction of pharynx and esophagus
  - Composition: Mainly striated muscle (cricopharyngeus + inferior pharyngeal constrictor)
  - Functional Importance
    i. Prevents Air Entry During Breathing
       - The UES maintains a high resting tone, preventing excess air from entering the esophagus during inspiration.
       - Without this tone, air would accumulate in the stomach (aerophagia).
    ii. Protects Airway from Esophageal Reflux
       - Acts as a barrier to prevent esophageal contents from entering the pharynx and airway.
    iii. Coordinates Swallowing
       - During swallowing, the UES relaxes momentarily to allow the bolus to pass.
       - Relaxation is tightly coordinated with pharyngeal contraction to ensure efficient propulsion and prevent aspiration
    iv. Prevents Regurgitation into the Pharynx
       - The UES closes rapidly after the bolus passes, protecting the airway from retrograde flow.
- Lower Esophageal Sphincter (LES)
  - Location: Distal esophagus at entry into stomach
  - Composition: Smooth muscle with tonic myogenic activity
  - Functional Importance
    i. Prevents Gastroesophageal Reflux
       - The LES is the primary anti-reflux barrier.
       - Its tonic contraction prevents acidic gastric contents from entering the esophagus.
    ii. Allows Passage of Food Into Stomach
       - During swallowing, the LES relaxes via vagal inhibitory fibers (receptive relaxation), synchronized with esophageal peristalsis.
       - This ensures smooth movement of the bolus into the stomach.
    iii. Works with the Diaphragm as a Dual Barrier
       - The crural diaphragm acts as an external sphincter.
       - Together with the LES, they prevent reflux, especially during:
         - coughing
         - straining
         - increased intra-abdominal pressure

iv. Maintains Intra-abdominal Esophageal Pressure
- LES prevents movement of gastric gas or fluid into the thoracic cavity, where pressure is lower.

- Clinical Correlation
  - UES dysfunction
    - Aspiration
    - Dysphagia
    - Cricopharyngeal achalasia
  - LES dysfunction
    - Low LES tone → GERD (gastroesophageal reflux disease)
    - High LES tone → Achalasia (failure of LES relaxation → dysphagia, food retention)

(2) Describe the pathophysiology of following
(2.1) Gastro Esophageal Reflux Disease (GERD)
(2.2) Barrett's Esophagus
(2.3) Achalasia

(1)

(1.1) Gastroesophageal Reflux Disease (GERD)

- Pathophysiology:

i. Decreased Lower Esophageal Sphincter (LES) Tone
- The LES normally maintains a high resting pressure to prevent gastric contents from refluxing upward.
- In GERD, LES pressure is abnormally low or undergoes excessive transient relaxations, allowing acid to enter the esophagus.
- LES acts as the primary antireflux barrier, preventing gastric acid from entering the esophagus

ii. Acid and Pepsin Injury to Esophageal Mucosa
- The esophagus lacks the protective mechanisms of the stomach (no thick mucus or bicarbonate layer).
- Refluxed gastric acid and pepsin cause:
  - mucosal inflammation (esophagitis)
  - erosions/ulcers in severe cases.

iii. Impaired Esophageal Clearance
- Poor peristalsis reduces the ability to clear acid.
- Salivary bicarbonate (which neutralizes acid) becomes less effective if clearance is slowed

iv. Hiatal Hernia Contribution
- The LES normally sits below the diaphragm, which adds an extra sphincter effect.
- In hiatal hernia, this alignment is lost → reduced LES competence → increased reflux.

v. Increased Intra-abdominal Pressure
- Obesity, pregnancy, or large meals can increase gastric pressure → pushing acid into the esophagus.

(1.2) Barrett's Esophagus

- Barrett's esophagus is a complication of chronic GERD.

i. Chronic Acid Exposure Leads to Metaplasia
   - Persistent reflux injury causes the stratified squamous epithelium of the distal esophagus to be replaced by intestinal-type columnar epithelium.
   - This is protective because columnar epithelium tolerates acid better.

ii. Metaplasia as an Adaptive Response
   - The esophagus adapts to the acid environment by changing the epithelial cell type.
   - However, this adaptation comes with increased risk.

iii. Increased Risk of Dysplasia and Adenocarcinoma
   - Columnar epithelium is more prone to:
     - genetic instability
     - dysplasia
     - progression to esophageal adenocarcinoma

- Pathophysiological Sequence
  - GERD → chronic mucosal injury → inflammation → epithelial stress → metaplasia → dysplasia → cancer (in a minority of patients).

(1.3) Achalasia

- A motility disorder caused by failure of LES relaxation and loss of esophageal peristalsis.

i. Degeneration of Myenteric (Auerbach's) Plexus Neurons
   - Especially loss of inhibitory (NO-producing) neurons.
   - Ganong explains that relaxation of the LES requires inhibitory vagal and enteric signals; loss of these → inability to relax the sphincter.

ii. LES Fails to Relax During Swallowing
   - LES pressure remains abnormally high.
   - The bolus cannot pass easily into the stomach → functional obstruction.

iii. Absence of Coordinated Peristalsis
   - Without myenteric plexus input, the esophageal body loses coordinated waves.
   - This causes:
     - retention of food
     - progressive esophageal dilation
     - dysphagia for both solids and liquids

iv. Stasis and Fermentation
   - Food retained in the esophagus undergoes fermentation → gas → foul odor.
   - Chronic stasis leads to:
     - mucosal inflammation
     - weight loss
     - risk of aspiration pneumonia

v. Increased Cancer Risk
   - Long-standing achalasia increases risk of squamous cell carcinoma.

Thyroid Gland

(1) A 28-year-old woman presents with weight loss despite good appetite, increased sweating, palpitations, heat intolerance, and irritability. On examination, she has a fine tremor, tachycardia, and a diffusely enlarged thyroid gland.
   (1.1) Describe the normal synthesis and release of thyroid hormones. (25 marks)
   (1.2) Explain how thyroid hormones are transported in blood and their mechanism of action at the cellular level. (20 marks)
   (1.3) Discuss how excess thyroid hormones lead to the clinical features observed in this patient. (35 marks)
   (1.4) Outline the main regulatory mechanisms that normally prevent such a condition. (20 marks)

(1)

(1.1) Synthesis & release (25 marks)

- Iodide trapping into follicular cells via Na^+/I^- symporter
- Which is regulated by TSH
- Transport of Iodine into colloid by Cl-/I- exchanger (pendrin)
- Oxidation of iodide to iodine by thyroid peroxidase
- Iodination of tyrosyl residues on thyroglobulin → MIT & DIT
- Coupling reactions: MIT+DIT = T3; DIT+DIT = T4
- Storage in colloid as thyroglobulin
- Endocytosis of colloid → proteolysis/lysosomal degradation → release of T3, T4 into blood

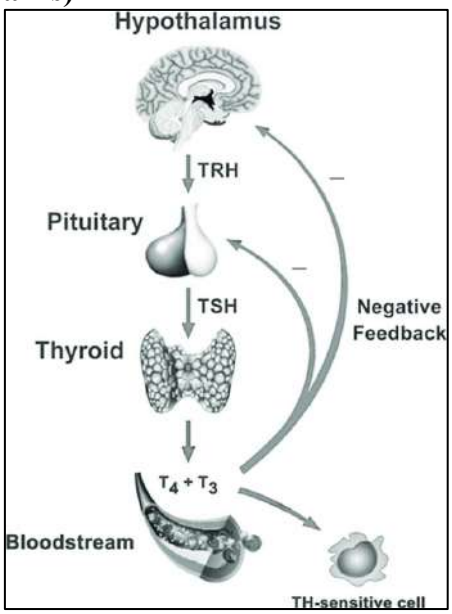
(1.2) Transport & mechanism (20 marks)

- Transport: mostly bound to plasma proteins
- Very little amount remains free
- Free form is the active form
- thyroxine-binding globulin (TBG), transthyretin, albumin
- Mechanism: T3 (more active) enters cell → binds nuclear receptor → modulates gene transcription → increases metabolic enzyme synthesis

(1.3) Effects of excess thyroid hormone (35 marks)

- ↑ Basal metabolic rate → weight loss despite appetite
- ↑ Heat production → heat intolerance, sweating
- ↑ β-adrenergic receptor expression → tachycardia, palpitations, tremor
- ↑ CNS excitability → irritability, nervousness
- Goiter: diffuse enlargement due to TSH receptor stimulation (autoimmune hyperthyroidism e.g., Graves')
- Catabolism of proteins → muscle weakness

(1.4) Regulation (20 marks)



- Hypothalamic TRH → pituitary TSH → thyroid hormones
- Negative feedback by T3, T4 on pituitary & hypothalamus
- Autoregulation: high iodine inhibits hormone synthesis (Wolff–Chaikoff effect)

(2) A 45-year-old man complains of weight gain, cold intolerance, constipation, and lethargy. On examination, he has dry coarse skin, periorbital puffiness, and bradycardia.

(2.1) Compare and contrast the metabolic effects of thyroid hormones in normal vs deficient states. (25 marks)

(2.2) Explain how the features in this patient arise from deficiency of thyroid hormones. (35 marks)

(2.3) Discuss the role of the hypothalamo-pituitary-thyroid axis in the development of hypothyroidism. (20 marks)

(2.4) Describe how iodine deficiency can result in hypothyroidism and goiter (20 marks)

(2.1) Metabolic effects (25 marks)

- Normal: ↑ BMR, ↑ O_2 consumption, ↑ carbohydrate & fat metabolism, protein turnover, thermogenesis
- Deficient: ↓ BMR, ↓ O_2 consumption, impaired carbohydrate/fat metabolism → weight gain, cold intolerance
- Reduced protein synthesis, mucopolysaccharide deposition

(2.2) Features explained (35 marks)

- Weight gain → ↓ metabolic rate
- Cold intolerance → ↓ thermogenesis
- Lethargy, mental slowness → ↓ CNS activity
- Constipation → ↓ GI motility
- Dry skin, periorbital puffiness → mucopolysaccharide accumulation (myxoedema)
- Bradycardia → ↓ β-adrenergic receptor expression

(2.3) Hypothalamo-pituitary-thyroid axis (20 marks)

- TRH (hypothalamus) → stimulates TSH (pituitary) → stimulates thyroid
- In primary hypothyroidism: thyroid fails → ↓ T3, T4, ↑ TSH due to loss of negative feedback (8)
- In secondary/tertiary hypothyroidism: pituitary/hypothalamus defect → ↓ TSH or TRH

(2.4) Iodine deficiency & goiter (20 marks)

- Iodine essential for hormone synthesis
- Deficiency → ↓ T3/T4 → loss of feedback inhibition → ↑ TSH
- TSH stimulates follicular cell hyperplasia → goiter

Adrenal Gland

(1) Write the physiological basis of following
(1.1) Dexamethasone suppression test is useful to diagnose Cushing syndrome.
(1.2) Short Synacthen test is helpful in diagnosing Addison's disease.

(1)

(1.1) Physiological Basis of the Dexamethasone Suppression Test (DST) in Diagnosing Cushing Syndrome

- Normal Physiology (from HPA Axis Regulation)
  - Cortisol is controlled by:
    Hypothalamus (CRH) → Pituitary (ACTH) → Adrenal cortex (cortisol)
  - Cortisol exerts negative feedback on:
    - Hypothalamus → ↓ CRH
    - Pituitary → ↓ ACTH
  - Cortisol acts as a potent inhibitor of both ACTH and CRH release through negative feedback mechanisms
- How the Dexamethasone Test Works
  - Dexamethasone is a synthetic glucocorticoid that:
    - Strongly suppresses ACTH secretion
    - Does not interfere with cortisol assays (so measured cortisol represents adrenal secretion only)
  - In normal individuals
    - Low-dose dexamethasone → ACTH suppression → decreased cortisol
      Because the HPA axis is intact and responsive to feedback.
  - In Cushing syndrome
    - Cortisol does not suppress normally due to one of the following:
      i. Pituitary adenoma (Cushing disease)
        - ACTH secretion becomes partially autonomous.
        - Negative feedback is reduced, not absent.
        - Therefore:
          - Low-dose dexamethasone: no suppression
          - High-dose dexamethasone: partial suppression (because pituitary cells still retain some feedback responsiveness)

      ii. Adrenal tumor
        - Produces excess cortisol independently of ACTH.
        - Dex suppresses ACTH, but the adrenal tumor keeps secreting cortisol anyway.
        - No suppression at any dose.

      iii. Ectopic ACTH production
        - Ectopic ACTH (e.g., small-cell lung cancer) is completely unresponsive to feedback.
        - No suppression even with high-dose dexamethasone.
- Therefore:

- DST is diagnostic because it tests the integrity of cortisol negative-feedback control.
  Failure to suppress indicates autonomous cortisol or ACTH production, the hallmark of Cushing syndrome.

(1.2) Short Synacthen test is helpful in diagnosing Addison's disease.

- Normal Physiology
  - ACTH stimulates the zona fasciculata of the adrenal cortex to produce cortisol
  - ACTH acts via cAMP and steroidogenic enzyme activation.
  - A healthy adrenal gland responds promptly with raised cortisol levels.
- How the Synacthen Test Works
  - Synacthen = synthetic ACTH (1–24)
  - Given IV or IM
  - Normal adrenal cortex → significant rise in cortisol (peak ~30–60 minutes)
- Primary Adrenal Insufficiency (Addison's disease)
  - The adrenal cortex is damaged or destroyed (autoimmune, TB, etc.).
  - It cannot respond to ACTH.
  - Therefore:
    - Baseline cortisol is low
    - ACTH stimulation → minimal or no increase in cortisol
  - This lack of cortisol response indicates failure of the adrenal gland itself
- Secondary or Tertiary Adrenal Insufficiency
  - Pituitary (↓ACTH) or hypothalamus (↓CRH) failure.
  - Chronic low ACTH → adrenal cortex atrophy.
  - Early disease → partial response
  - Late disease → poor response (similar to Addison's)
- Therefore:
  - The Synacthen test is diagnostic because it assesses the adrenal gland's ability to generate cortisol in response to ACTH.
    Failure to increase cortisol confirms primary adrenal insufficiency (Addison's) or long-standing secondary adrenal insufficiency.

Renin Angiotensin Aldosterone System

(1) Give the physiological basis for oliguria in a patient who suffered a severe hemorrhage focusing on renin angiotensin system.

- Haemorrhage leads to a hypovolemic shock
- Due to decreased blood volume
- Blood supply to aortic and carotid bodies decreases
- sympathetic discharge increased
- increases release of catecholamine
- internal baroreceptor mechanism
- act on juxtaglomerular cells to increase renin secretion
- renin converts angiotensinogen to angiotensin I
- it is converted to angiotensin II by angiotensin converting enzyme
- angiotensin II causes constriction of both afferent and efferent arterioles
- but the efferent are constricted more
- It also causes contraction of mesangial cells
- this decreases glomerular filtration rate
- angiotensin II also increases the release of ADH from posterior pituitary
- it acts on V2 receptors
- of the principal cells of cortical collecting ducts
- and increases the synthesis and insertion of aquaporin channels
- so, water reabsorption increases
- both these decrease urine output oliguria

(2) Write short notes on function and regulation of renin

- Conversion of angiotensinogen to angiotensin 1
- It activates renin-angiotensin mechanism
- To regulate ECF volume
- Regulation of secretion
- Secreted from Juxtaglomerular cells
- Of juxtaglomerular apparatus.
- Increased by: reduced pressure n afferent arterioles.
- Via intrarenal baroreceptor mechanism.
- Decreased delivery of Na+Cl- to distal convoluted tubules.
- Detected by macula densa cells.
- Increased sympathetic discharge
- Via renal nerves.
- Circulating catecholamines
- Adrenalin
- Noradrenalin
- Dopamine
- Prostaglandin
- Directly on juxtaglomerular cells
- Decreased by angiotensin 2
- This is feedback inhibition.
- ANP *ADH

Endocrine Pancreas & Diabetes

(1) A 45-year-old male presents to the clinic with complaints of excessive thirst, frequent urination, and unintentional weight loss over the past three months. Laboratory investigations reveal fasting blood glucose levels of 280 mg/dL and glycated hemoglobin (HbA1c) of 10%. A further hormonal workup shows elevated cortisol levels with a lack of suppression following a low-dose dexamethasone test.

(1.1) Discuss the physiological role of the endocrine pancreas in glucose metabolism.

(1.2) Explain how its dysfunction leads to the clinical manifestations observed in this patient.

(1.3) Elaborate on the interaction between adrenal hormones and glucose homeostasis, highlighting the potential role of hypercortisolism in this case.

(1.1) Discuss the physiological role of the endocrine pancreas in glucose metabolism.

- The endocrine pancreas, through insulin and glucagon secretion, maintains glucose homeostasis.
- Insulin, secreted by beta cells
- promotes glucose uptake by muscles
- Liver
- And adipose tissue
- Increases lipogenesis,
- Prevents glycogenolysis and gluconeogenesis
- And prevents protein catabolism
- while glucagon, secreted by alpha cells
- stimulates glycogenolysis and gluconeogenesis.
- As well as lipolysis
- Insulin and glucagon are reciprocally secreted
- Insulin is secreted when blood glucose is increased.

(1.2) Explain how its dysfunction leads to the clinical manifestations observed in this patient.

- diabetes mellitus is a metabolic disorder
- characterized by chronic hyperglycemia
- due to defects in insulin secretion and / or
- insulin action
- In type 1 DM there is insulin deficiency due to beta cell destructin
- In type 2 DM, there is insulin resistance due to a progressive insulin secretory defect
- Patients with DM has hyperglycemia, polyuria, polydipsia, and weight loss
- due to deranged glucose homeostasis
- increased lipolysis and
- increased muscle protein breakdown.

(1.3) Elaborate on the interaction between adrenal hormones and glucose homeostasis, highlighting the potential role of hypercortisolism in this case.

- The adrenal cortex secretes cortisol,
- a glucocorticoid
- whose main effect is to increase concentration of blood glucose
- at the expense of fat and proteins
- that enhances gluconeogenesis,
- reduces glucose uptake by peripheral tissues except brain ,
- and promotes protein degradation in all cells except in liver cells
- Chronic hypercortisolism
- as seen in Cushing's syndrome
- exacerbates hyperglycemia, leading to secondary diabetes.
- In this patient, both diabetes and hypercortisolism may be contributing to his symptoms, requiring further differentiation and management.

Calcium & Phosphate Metabolism

(1) A 55-year-old postmenopausal woman presents with generalized bone pain, muscle weakness, and multiple fractures over the past year. Laboratory tests reveal hypocalcemia, elevated serum parathyroid hormone (PTH), and low serum phosphate levels. A bone density scan shows osteoporosis with significant cortical bone thinning.

(1.1) Explain the physiological mechanisms regulating calcium and phosphate homeostasis.

(1.2) Discuss how the observed laboratory abnormalities contribute to the patient's clinical presentation.

(1.1) Explain the physiological mechanisms regulating calcium and phosphate homeostasis.

- Calcium and phosphate homeostasis is regulated by parathyroid hormone (PTH)
- vitamin D
- and calcitonin
- PTH increases serum calcium by stimulating bone resorption
- increasing renal calcium reabsorption
- and enhancing intestinal calcium absorption via activation of vitamin D
- Simultaneously, it promotes phosphate excretion by the kidneys.

(1.2) Discuss how the observed laboratory abnormalities contribute to the patient's clinical presentation.

- In this patient, elevated PTH with hypocalcaemia and low phosphate suggests secondary hyperparathyroidism,
- likely due to vitamin D deficiency
- Inadequate vitamin D impairs intestinal calcium absorption
- leading to compensatory PTH elevation
- Chronic PTH stimulation results in excessive bone resorption
- Osteoporosis
- increased fracture risk
- Treatment includes calcium and vitamin D supplementation to restore mineral balance and prevent further bone loss.

PHYSIOLOGY CAT-2 MOCK PAPERS & ANSWERS

2022/2023 (1st Batch)
CAT-2 Mock Exam

CAT-2 (BATCH 1) MOCK MCQS

MULTIPLE CHOICE QUESTIONS

Time: 1 hour

This paper consists of 10 True or False type questions and 10 Single Best Answer type questions.

(1) During inspiration,
- (A) Intra pleural pressure is lowest at mid inspiration.
- (B) Intra pulmonary pressure is lowest at end expiration.
- (C) Intra esophageal pressure is lowest at mid inspiration.
- (D) Rate of air flow is greatest at end inspiration.
- (E) Lung volume/intra pleural pressure relationship is the same as in expiration.

(2) Pulmonary surfactant increases,
- (A) Surface tension of the fluid lining alveolar walls.
- (B) Lung compliance.
- (C) In its effectiveness as the lungs are inflated.
- (D) In amount when pulmonary blood flow is interrupted.
- (E) In amount in fetal lungs during the last month of the pregnancy.

(3) Consequences of hypoxia at high altitudes include,
- (A) Tachycardia
- (B) Respiratory alkalosis
- (C) Noticeable cyanosis
- (D) Raised arterial blood pressure
- (E) Nausea and vomiting

(4) Carotid bodies,
- (A) Are stretch receptors in the walls of the internal carotid arteries.
- (B) Have a blood flow per unit volume similar to that in the brain.
- (C) Are influenced more by the blood pO_2 than by its oxygen content.
- (D) Generate more afferent impulses when blood H+ ion concentration rises.
- (E) And the aortic bodies are mainly responsible for the increased ventilation in hypoxia.

(5) Swallowing,
- (A) Involves a reflex activity.
- (B) Is solely under the control of the medulla.
- (C) Is associated with elevation of the larynx.
- (D) Demonstrates that not all the skeletal muscles are under voluntary control.
- (E) Affects hearing.

(6) Defecation,
- (A) Is coordinated by reflex centers in the sacral cord.
- (B) Is a reflex whose afferent limb carries impulses from stretch receptors in the colon.

(C) Is a reflex whose efferent limb travels mainly in sympathetic autonomic nerve.
(D) Is more likely to be initiated just after a meal than just before it.
(E) Can be voluntarily inhibited and facilitated.

(7) Absorption of dietary fats,
(A) Mainly occurs in the terminal ileum.
(B) Depends on the complete hydrolysis of fats to fatty acids and glycerol.
(C) Is decreased when there is reduced absorption of vitamin A, D and K.
(D) Takes place only in the lymphatics.
(E) Is dependent on the presence of calcium ions.

(8) Regarding the mechanism of hormone action,
(A) Thyroid hormone signals its message via a plasma membrane receptor.
(B) Steroid hormones attach to a specific plasma membrane receptor.
(C) Insulin signals its message via second messenger cAMP.
(D) Cortisol combines with a specific protein receptor on an intracellular organelle.
(E) Steroid hormones and peptide hormones have similar modes of action at the cellular level.

(9) Antidiuretic hormone,
(A) Is a pentapeptide.
(B) Release is triggered by a sudden loss of 50ml of blood.
(C) Pressure receptors are located in the aortic arch and carotid sinus.
(D) Has a vasoconstrictor effect even at physiological level.
(E) Stimulates the release of TSH at physiological levels.

(10) Concentration of ionized calcium in the plasma is,
(A) The main regulator of PTH secretion.
(B) Less than the free ionized calcium concentration in cells.
(C) About 50% of the total plasma calcium concentration.
(D) Reduced when plasma pH rises.
(E) Reduced when the plasma protein level rises.

Single Best Answer Questions

(1) Absent mucus transport can lead to chronic sinusitis and recurrent lung infection. Which one of the following causes absent mucus transports?
(A) Absence of mucus-secreting cells
(B) Bronchial obstruction
(C) Bronchospasm
(D) Defective ciliary motility
(E) Mucosal oedema

(2) Gastric secretions help in performing many different activities. Which one of the following is mainly affected by the absence of gastric secretions?
(A) Iron absorption
(B) Protein digestion
(C) Fat digestion

(D) Vitamin B12 absorption
(E) Protein absorption

(3) The colon is extremely efficient at conserving intestinal water. Which one of the following helps to conserve water following a low-salt diet other than osmosis?
(A) Increased expression of aquaporin-1 channels
(B) Antidiuretic hormone
(C) Aldosterone-induced expression of ENaC
(D) Hyponatremia-induced reduced colonic motility
(E) High alkalinity of chyme

(4) Patients with Cushing syndrome develop muscle weakness. What is the most contributing factor for it?
(A) K^+ depletion
(B) Reduced muscle blood flow
(C) Steroid-induced hyperglycemia
(D) Fat redistribution
(E) Na^+ retention

(5) Pancreatic secretion is influenced by neural and hormonal factors. Which of the following increases HCO_3^- rich pancreatic secretions?
(A) Acidic chyme in duodenum
(B) Fatty acids
(C) Food in the mouth
(D) Gastric distension
(E) Products of protein digestion

(6) Congenital defects in the enzymes involved in production of adreno-cortical hormones lead to congenital adrenal hyperplasia. Patients with this disease could develop virilization features. These patients are given exogenous corticosteroids. This prevents further development of virilization also. Which of the following action of corticosteroid minimizes virilization?
(A) Direct action on cytochrome P450 enzymes
(B) Glucocorticoid action
(C) Improvement in 21 β-hydroxylase level
(D) Inhibition of ACTH secretion
(E) Mineralocorticoid action

(7) In the upright posture, compared to the base of the lung, the apex of the lung has poor ventilation. However, the apex of the lung has a higher ventilation/perfusion ratio (V/Q ratio) than the base. What is the best physiological explanation for the apex to have higher V/Q ratio?
(A) Effect of the accessory muscles of inspiration is more prominent on the apical regions.
(B) Intrapleural pressure is more negative in the apex.
(C) Compliance in the apex of the lung is less compared to the base.
(D) Reduction of perfusion from base to apex is more prominent than that of ventilation.
(E) Veins in the apex of the lungs are subjected to negative pressure.

(8) Insulin secretory capacity of the pancreas is better assessed using the measurements of serum C peptide level than measuring serum insulin level. Why is it better to measure C peptide level than insulin level in plasma for assessing the secretory capacity?

(A) Both are secreted by the pancreas.
(B) C peptide and insulin are secreted in equimolar concentration.
(C) C peptide is being removed from the plasma slower than insulin.
(D) C peptide is not affected by anti-insulin antibodies.
(E) Even when the pancreas is damaged C peptide continues to be secreted.

(9) A patient on thyroxine treatment shows clinical features suggestive of hyperthyroidism. State the investigation finding that favor the diagnosis of hyperthyroidism due to exogenous thyroxin.

(A) Elevated iodine uptake by the gland
(B) Elevated T4
(C) Elevated thyroid binding globulin
(D) Elevated thyroid stimulating immunoglobulin (TSI)
(E) Elevated TSH

(10) An anesthetized male is breathing with no assistance. He is then artificially ventilated for 10 min at his normal tidal volume but at twice his normal frequency. He is ventilated with a gas mixture of 60% O_2 and 40% N_2. The artificial ventilation is stopped and he fails to breathe for several minutes. This apneic episode is due to which of the following?

(A) High arterial PO_2 suppressing the activity of the peripheral chemoreceptors
(B) Decrease in arterial pH suppressing the activity of the peripheral chemoreceptors
(C) Low arterial PCO_2 suppressing the activity of the medullary chemoreceptors
(D) High arterial PCO_2 suppressing the activity of the medullary chemoreceptors
(E) Low arterial PCO_2 suppressing the activity of the peripheral chemoreceptors

CAT-2 (BATCH 1) MOCK MCQ ANSWERS

(1)

(A) **T** – During inspiration, intrapleural pressure becomes more negative as the chest expands.

It reaches its most negative value around mid-inspiration, when the inspiratory muscles generate maximal expansion before elastic recoil begins to dominate.

(B) **F** – At end-expiration, alveolar pressure = 0 cm H_2O (equal to atmospheric pressure).

The lowest alveolar pressure occurs early in inspiration, when expanding thorax creates negative alveolar pressure to draw air in.

(C) **T** – Esophageal pressure reflects intrapleural pressure.

As intrapleural pressure becomes maximally negative mid-inspiration, esophageal pressure also becomes most negative.

(D) **F** – Airflow = pressure gradient between atmosphere and alveoli.

Maximum airflow occurs early in inspiration, when:

- alveolar pressure is most negative
- pressure gradient is greatest

By end-inspiration, alveolar pressure returns to 0, so airflow falls to zero.

(E) **F** – The pressure-volume relationship differs between inspiration and expiration → called hysteresis.

At the same intrapleural pressure, lung volume is greater during expiration than inspiration due to:

- surfactant behavior
- alveolar recruitment characteristics

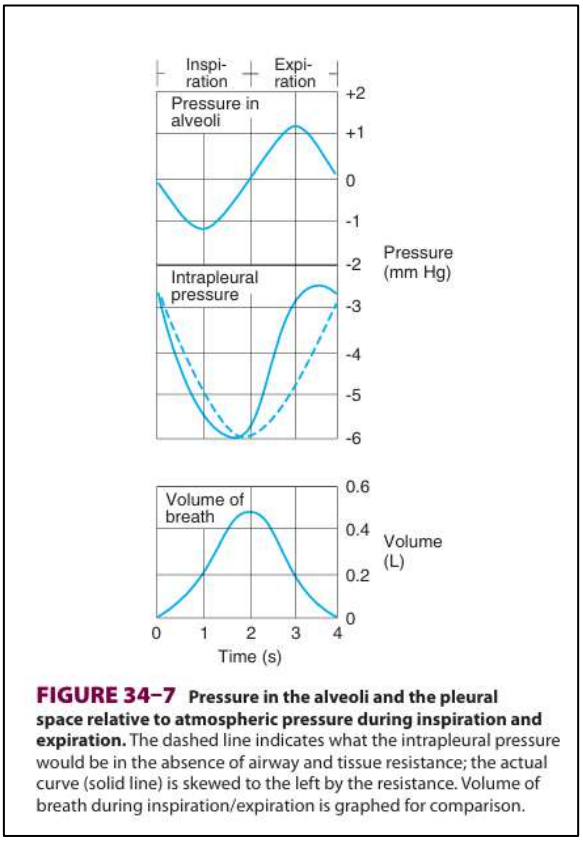


FIGURE 34–7 Pressure in the alveoli and the pleural space relative to atmospheric pressure during inspiration and expiration. The dashed line indicates what the intrapleural pressure would be in the absence of airway and tissue resistance; the actual curve (solid line) is skewed to the left by the resistance. Volume of breath during inspiration/expiration is graphed for comparison.

Ganong's Review of Medical Physiology 24th Edition p. 628

(2)

(A) **F** – Surfactant decreases surface tension.
This prevents alveolar collapse (atelectasis) and reduces the work of breathing.

(B) **T** – By lowering surface tension, surfactant increases compliance, meaning the lungs expand more easily for a given pressure.

(C) **F** – Surfactant is more effective at lower lung volumes.
When alveoli are small, surfactant molecules are more concentrated → surface tension is reduced more strongly.
As lungs inflate and alveoli enlarge, surfactant becomes less concentrated and less effective.

(D) **F** – Surfactant production decreases with reduced pulmonary blood flow because type II pneumocytes receive fewer nutrients and less hormonal stimulation.
Ischemia → impaired surfactant synthesis.

(E) T – Surfactant production increases markedly after 34–36 weeks gestation.
This is essential for preventing neonatal respiratory distress syndrome at birth.

(3)

(A) **T** – Hypoxia stimulates chemoreceptors → increased sympathetic activity.
This causes increased heart rate to improve oxygen delivery.

(B) **T** – Low atmospheric O_2 → hypoxemia → hyperventilation (primary response).
Hyperventilation lowers $PaCO_2$ → respiratory alkalosis.
Kidneys later compensate by excreting bicarbonate.

(C) **F** – At high altitude, arterial oxygen saturation decreases but hemoglobin concentration rises gradually (polycythemia).
Early on, cyanosis is not prominent, especially because low $PaCO_2$ causes vasodilation and increased blood flow to skin.
Cyanosis may occur only in chronic mountain sickness, not typical acute altitude exposure.

(D) **F** – Acute altitude hypoxia causes:
- Pulmonary vasoconstriction → ↑ pulmonary arterial pressure
- NOT ↑ systemic arterial pressure

Systemic BP may remain normal or slightly elevated due to sympathetic drive, but not markedly raised.

(E) **T** – Part of acute mountain sickness (AMS) due to:
- Hypoxia-induced cerebral edema
- Increased intracranial pressure

Symptoms: headache, nausea, vomiting, dizziness.

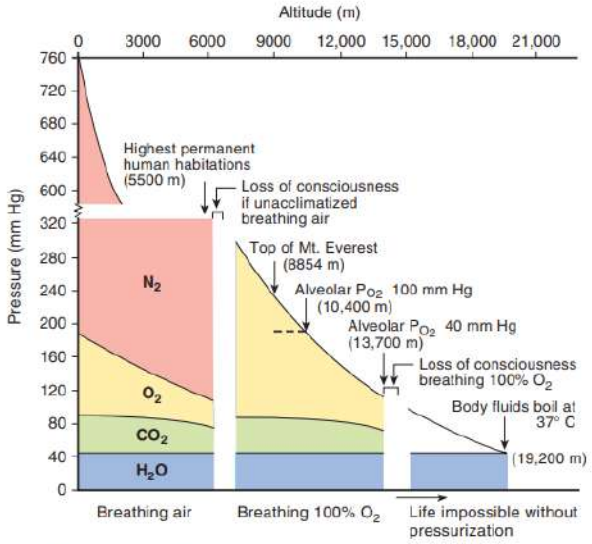


FIGURE 35–10 Composition of alveolar air in individuals breathing air (0–6100 m) and 100% O_2 (6100–13,700 m). The minimal alveolar P_{O_2} that an unacclimatized subject can tolerate without loss of consciousness is about 35–40 mm Hg. Note that with increasing altitude, the alveolar P_{CO_2} drops because of the hyperventilation due to hypoxic stimulation of the carotid and aortic chemoreceptors. The fall in barometric pressure with increasing altitude is not linear, because air is compressible.

Ganong's Review of Medical Physiology 24th Edition p. 650

(4)

(A) **F** – Carotid bodies are chemoreceptors, not stretch receptors.
They detect PaO_2, $PaCO_2$, and H^+, not stretch or pressure.
(Stretch receptors are in lungs & carotid sinus baroreceptors—not carotid bodies.)

(B) **F** – Carotid bodies have one of the highest blood flows per unit mass in the body—far higher than the brain.
This ensures they monitor arterial blood before it equilibrates with tissues.

(C) **T** – Carotid bodies respond mainly to arterial PO_2, not O_2 content.
They do not respond to anemia or CO poisoning unless PO_2 falls.

(D) **T** – Increased H^+ (acidosis) → stimulates carotid bodies → ↑ ventilation.
They are very sensitive to H^+ and PCO_2 because CO_2 rapidly diffuses into glomus cells.

(E) **F** – Carotid bodies are the main sensors for hypoxia-induced hyperventilation.
Aortic bodies also respond, but play a lesser role.

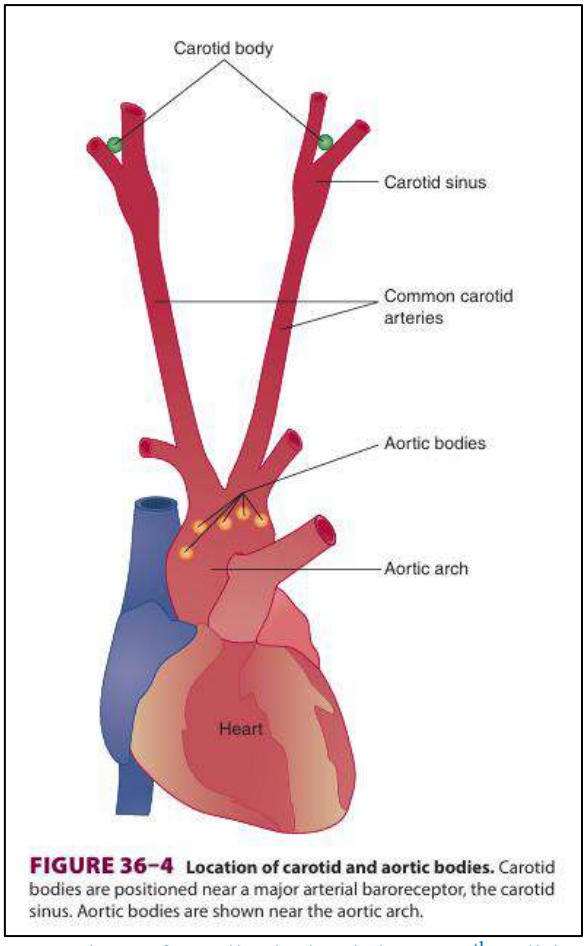


FIGURE 36–4 **Location of carotid and aortic bodies.** Carotid bodies are positioned near a major arterial baroreceptor, the carotid sinus. Aortic bodies are shown near the aortic arch.

Ganong's Review of Medical Physiology 24th Edition p. 660

(5)

(A) **T** – Swallowing begins voluntarily, but the pharyngeal and esophageal phases are reflex.
Once the bolus enters the oropharynx, the swallowing center in the medulla takes over and produces a coordinated reflex sequence.

(B) **F** – The medulla contains the main swallowing center, but the pons also participates.
Additionally, the cerebral cortex initiates the voluntary phase of swallowing.
Therefore, control is not solely medullary.

(C) **T** – During swallowing:
- Larynx elevates
- Epiglottis closes the airway

This prevents aspiration and directs the bolus into the esophagus.

(D) **T** – Swallowing uses skeletal muscles (pharyngeal constrictors, tongue, upper esophagus).
Although they are skeletal muscles, their activity during the swallowing reflex is involuntary, governed by the brainstem.
This is a classic example of reflexive control of striated muscle.

(E) **F** – Swallowing does not physiologically affect hearing.
Although swallowing can pop the Eustachian tube (equalizing pressure), hearing function itself is not altered by swallowing.

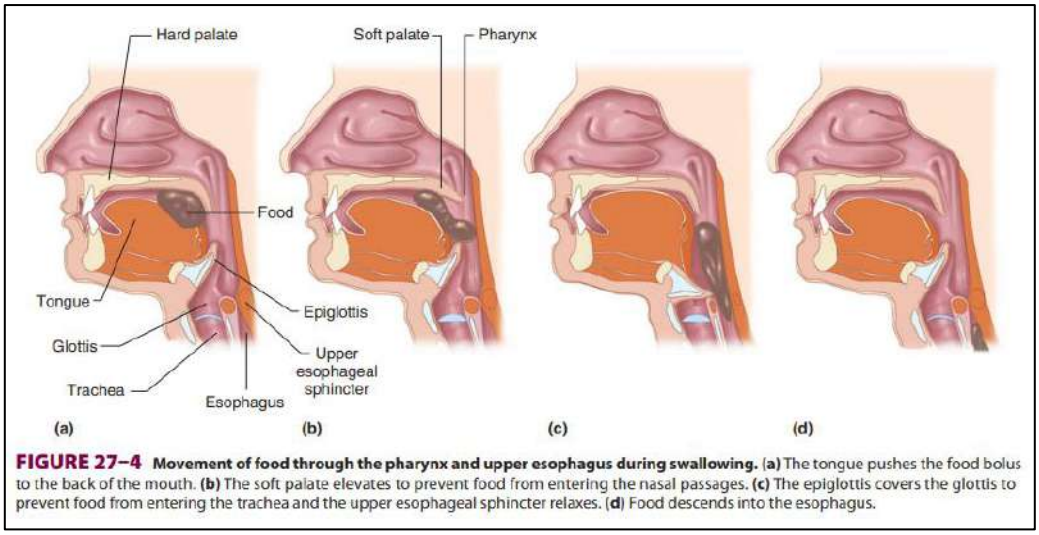


FIGURE 27–4 **Movement of food through the pharynx and upper esophagus during swallowing.** **(a)** The tongue pushes the food bolus to the back of the mouth. **(b)** The soft palate elevates to prevent food from entering the nasal passages. **(c)** The epiglottis covers the glottis to prevent food from entering the trachea and the upper esophageal sphincter relaxes. **(d)** Food descends into the esophagus.

Ganong's Review of Medical Physiology 24th Edition p. 500

(6)

(A) **T** – The acral spinal cord (S2–S4) contains the defecation reflex center.
Parasympathetic outflow from S2–S4 mediates colon contraction and internal anal sphincter relaxation.

(B) **F** – Stretch receptors activated during defecation are primarily in the rectum, not the colon.
Rectal distension → afferent signals → sacral spinal cord → initiates reflex.

(C) **F** – The efferent limb is parasympathetic (pelvic nerves, S2–S4).
Parasympathetic activity causes:

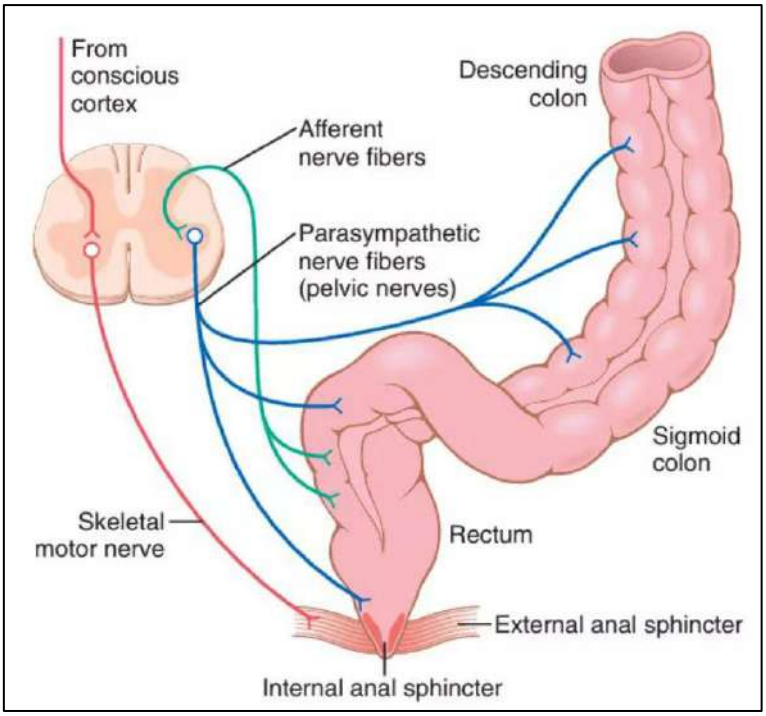
- ↑ rectal contraction
- Relaxation of the internal anal sphincter

Sympathetic nerves inhibit defecation (they keep the internal sphincter contracted).

(D) **T** – The gastrocolic reflex (mediated by vagus & gastrin) increases colon motility after eating.
This pushes fecal material into the rectum, triggering defecation reflex.
Therefore, defecation is more likely after a meal.

(E) **T** – External anal sphincter (striated muscle) allows voluntary control.
Defecation can be inhibited by contracting this sphincter.
It can be facilitated by:

- Relaxing the sphincter
- Increasing abdominal pressure (Valsalva maneuver)



http://medizzy.com/feed/40906638

(7)

(A) **F** – Fat absorption occurs mostly in the jejunum.
By the time chyme reaches the terminal ileum, most dietary fat has already been absorbed.

(B) **F** – Complete hydrolysis is not required.
Dietary triglycerides are broken down into:
- monoglycerides
- free fatty acids
- some diglycerides

These form micelles and are absorbed by enterocytes.
Inside cells, triglycerides are re-esterified, so full breakdown is not necessary.

(C) **F** – Fat malabsorption leads to deficiency of fat-soluble vitamins A, D, E, K.
Vitamin deficiency does not impair fat absorption.
Vitamins depend on fat absorption, not the other way around.

(D) **F** – Long-chain fatty acids are absorbed as chylomicrons → lymphatics (lacteals).
However, short- and medium-chain fatty acids are absorbed directly into the portal blood.
So, absorption is not exclusively lymphatic.

(E) **F** – Calcium is not required for normal fat absorption.
Fat absorption depends on:
- bile salts
- pancreatic lipase
- micelle formation
- enterocyte transport mechanisms

Calcium plays no direct physiological role here.

(8)

(A) **F** – Thyroid hormones (T_3, T_4) act through intracellular nuclear receptors.
They bind to thyroid hormone receptors (TRs) on DNA and regulate gene transcription.
They do not use membrane receptors.

(B) **F** – Steroid hormones (cortisol, aldosterone, estrogen, testosterone) are lipid soluble.
They diffuse through the membrane and bind to intracellular receptors, located in the:
- cytoplasm (e.g., glucocorticoids), or
- nucleus (e.g., estrogen).

(C) **F** – Insulin acts through a tyrosine kinase receptor, not cAMP.
The receptor autophosphorylates, activates IRS proteins, and triggers signaling cascades (PI3K, MAPK).
cAMP is used by glucagon, ADH (V_2), TSH, ACTH, etc., not insulin.

(D) **F** – Cortisol binds to a cytoplasmic receptor, not an organelle.
The hormone–receptor complex then translocates to the nucleus to regulate gene expression.

(E) **F** – Their mechanisms are fundamentally different:

Hormone Type	Receptor	Signaling
Steroid & thyroid hormones	Intracellular (cytoplasmic/nuclear)	Regulate gene transcription
Peptide & protein hormones	Cell membrane receptors	Use second messengers, kinase cascades

(9)

(A) **F** – ADH is a nonapeptide (9 amino acids).
It is synthesized in the supraoptic and paraventricular nuclei of the hypothalamus.

(B) **F** – A small blood loss (50 mL) does not trigger ADH release.
ADH release requires:
- ≥ 10% decrease in blood volume, or
- ↑ plasma osmolality (1% change triggers release)

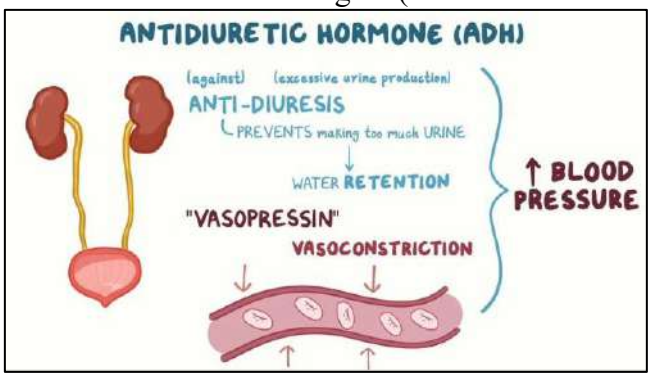
Baroreceptor-mediated ADH secretion requires significant hypovolemia, not small losses.

(C) **T** – These baroreceptors detect changes in blood pressure.
Decreased stretch → ↑ ADH secretion by reducing inhibitory signals sent to the hypothalamus.

(D) **F** – At physiological concentrations, ADH mainly acts on V_2 receptors in the kidneys → water reabsorption.
Vasoconstriction requires high (supraphysiologic) levels, acting on V_1 receptors.
So, vasoconstriction is not seen normally.

(E) **F** – ADH has no physiological role in stimulating TSH.
TSH secretion is controlled by:
- TRH (stimulatory)
- Somatostatin and dopamine (inhibitory)

ADH influences ACTH to some degree (co-release with CRH), but not TSH.



https://www.osmosis.org/video/Antidiuretic_hormone

(10)

(A) **T** – Ionized (free) Ca^{2+}, not total calcium, is the primary regulator of parathyroid hormone (PTH) release.
Even small decreases in ionized Ca^{2+} strongly stimulate PTH secretion via the calcium-sensing receptor (CaSR) on parathyroid cells.

(B) **F** – Extracellular plasma) ionized $Ca^{2+} \approx 1.1–1.3$ mmol/L.
Intracellular free $Ca^{2+} \approx 10^{-7}$ mol/L (0.0001 mmol/L) — thousands of times lower.
Cells keep intracellular Ca^{2+} extremely low for signaling purposes.
So, plasma ionized Ca^{2+} is much higher than intracellular free Ca^{2+}.

(C) **T** – Total plasma calcium $\approx 2.2 - 2.6$ mmol/L.
Ionized (free) $Ca^{2+} \approx 1.1–1.3$ mmol/L = roughly 50%.
The rest:

- 40% protein-bound (mostly albumin)
- 10% complexed (citrate, phosphate)

(D) **T** – Alkalosis → more negative charges on albumin → increased binding of calcium → ↓ free ionized Ca^{2+}.
This is why hyperventilation can cause tingling and tetany (low ionized Ca^{2+} despite normal total Ca).

(E) **F** – Increased plasma proteins → more protein-bound Ca^{2+},
but ionized Ca^{2+} remains normal due to tight homeostatic control.
Total Ca increases, but ionized Ca does not fall.

TABLE 21–1 Distribution (mg/dL) of calcium in normal human plasma.

Total diffusible		**5.36**
Ionized (Ca^{2+})	4.72	
Complexed to HCO_3^-, citrate, etc	0.64	
Total nondiffusible (protein-bound)		**4.64**
Bound to albumin	3.68	
Bound to globulin	0.96	
Total plasma calcium		**10.00**

Ganong's Review of Medical Physiology 24th Edition p. 378

Single Best Answer Questions

(1) Answer D

Effective mucus transport in the respiratory tract requires:

i. Normal mucus production
ii. Functional ciliated epithelial cells (coordinated ciliary beating)
iii. A patent airway

If cilia cannot beat properly, mucus stagnates → chronic sinusitis + recurrent lung infections.

This is the classic mechanism in:

- Primary ciliary dyskinesia (PCD)
- Kartagener syndrome
- Post-viral epithelial damage
- Smoking-induced ciliary dysfunction

Why the other options are incorrect

A. Absence of mucus-secreting cells

Would reduce mucus but NOT cause mucus stasis.

In fact, low mucus would not create the thick secretions seen in recurrent infections.

B. Bronchial obstruction

Can impair clearance but does not eliminate mucus transport globally.

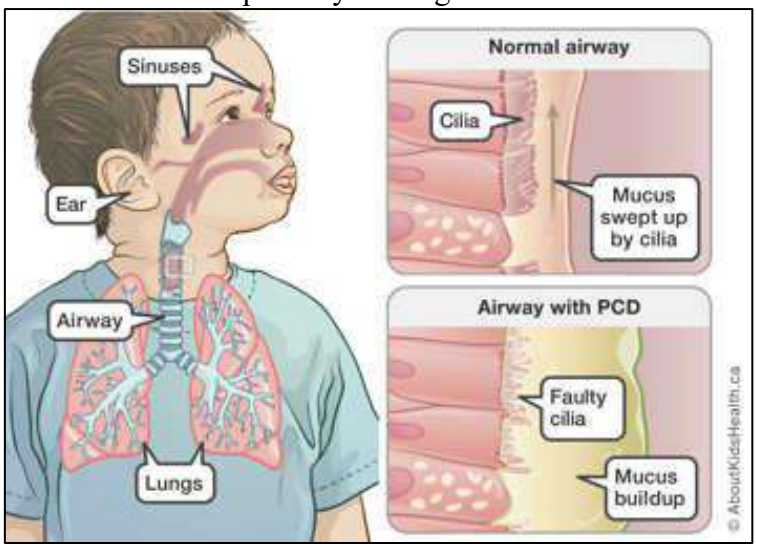
This causes localized problems, not absent mucus transport.

C. Bronchospasm

Causes airway narrowing (e.g., asthma) but cilia still function.

E. Mucosal edema

Slows airflow but does not stop ciliary beating.



https://www.aboutkidshealth.ca/primary-ciliary-dyskinesia-pcd

(2) Answer D

Gastric secretions contain intrinsic factor (IF), secreted by parietal cells, which is essential for Vitamin B_{12} absorption in the ileum.

Without gastric secretions (e.g., in pernicious anemia, total gastrectomy, atrophic gastritis):

- Intrinsic factor is absent
- Vitamin B_{12} cannot be absorbed

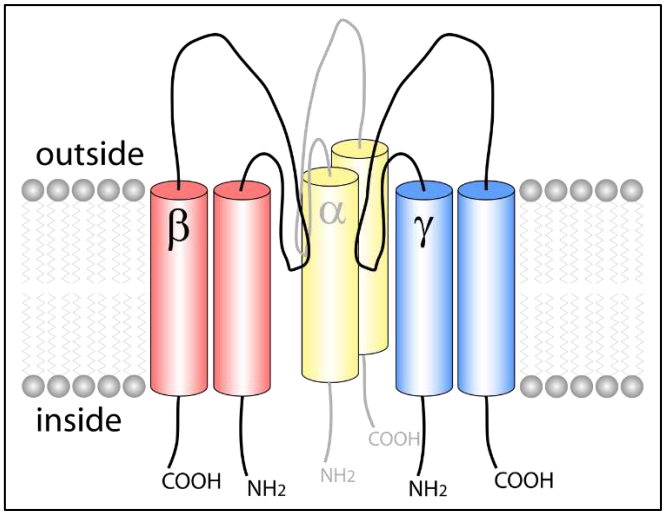
This leads to megaloblastic anemia and neurological deficits
This is the most severely impaired function when gastric secretions are absent.
A. Iron absorption
Gastric acid helps convert $Fe^{3+} \rightarrow Fe^{2+}$ (absorbable form),
but even without gastric secretion, iron absorption still occurs, although less efficiently.
Not the main affected.
B. Protein digestion
Pepsin requires gastric acid, but pancreatic proteases (trypsin, chymotrypsin) can compensate.
Protein digestion still largely occurs in the small intestine.
C. Fat digestion
Gastric secretions play a minimal role.
Fat digestion mainly depends on bile salts and pancreatic lipase.
E. Protein absorption
Occurs in the small intestine, independent of gastric secretions.

(3) Answer C

When dietary salt intake is low, the body increases aldosterone secretion.
Aldosterone acts on the colon, just as it does on the distal nephron, to:

- Upregulate ENaC (epithelial sodium channels)
- Increase Na^+ absorption
- Create an osmotic gradient
- Promote water reabsorption

This mechanism enhances water conservation in the colon independently of simple osmosis.
Colon's aldosterone-sensitive sodium transport is a key mechanism for water conservation, especially during low-salt states.
Why the other options are incorrect
A. Increased expression of aquaporin-1 channels
AQP-1 is found in proximal kidney tubules and endothelium, not the colon.
The colon primarily uses AQP-3 and AQP-4.
B. Antidiuretic hormone (ADH)
ADH affects renal collecting ducts, not the colon.
It does not regulate colonic water absorption.
D. Hyponatremia-induced reduced colonic motility
Hyponatremia does not physiologically slow colonic transit.
Motility changes are not a water-conservation mechanism.
E. High alkalinity of chyme
pH changes do not drive water conservation in the colon.



Epthelial sodium channel (ENaC) structure
https://en.wikipedia.org/wiki/Epithelial_sodium_channel

(4) Answer A

Muscle weakness in Cushing syndrome is mainly due to the catabolic effects of excess cortisol, but among the options provided, the most significant contributing factor is:

Cortisol → mineralocorticoid effect → increased K^+ excretion → hypokalemia → muscle weakness

Why hypokalemia causes muscle weakness:

- Low extracellular K^+ hyperpolarizes skeletal muscle membranes
- Makes depolarization more difficult
- Leads to muscle fatigue, cramps, and proximal muscle weakness

Excess glucocorticoids enhance renal K^+ excretion, contributing to neuromuscular dysfunction.

Why the other options are incorrect

B. Reduced muscle blood flow

Cortisol causes mild vasoconstriction, but this is not the primary cause of muscle weakness.

C. Steroid-induced hyperglycemia

Causes metabolic issues, but muscle weakness is mostly due to protein catabolism and hypokalemia, not high glucose.

D. Fat redistribution

Leads to central obesity, moon face, buffalo hump—NOT muscle weakness.

E. Na^+ retention

Leads to edema and hypertension, but does not directly cause skeletal muscle weakness.

(5) Answer A

The most powerful stimulus for HCO_3^--rich pancreatic secretion is acid entering the duodenum.

Mechanism

- Acidic chyme (low pH) stimulates S cells in the duodenum.
- S cells release secretin.
- Secretin strongly stimulates the ductal cells of the pancreas to secrete:
  - HCO_3^- (bicarbonate)

- Large volume, watery secretion
- Purpose: neutralize gastric acid and protect the duodenal mucosa.

Thus, A is clearly correct.
Why the other options are incorrect
B. Fatty acids
Stimulate CCK, which increases enzyme-rich secretion, not HCO_3^-.
C. Food in the mouth
Causes mild pancreatic stimulation via vagal pathways, mostly enzyme secretion, not high bicarbonate.
D. Gastric distension
Activates vagovagal reflexes → mostly increases enzyme-rich secretion.
Weak stimulus for HCO_3^-.
E. Products of protein digestion
Stimulate CCK, again primarily increasing enzyme secretion.

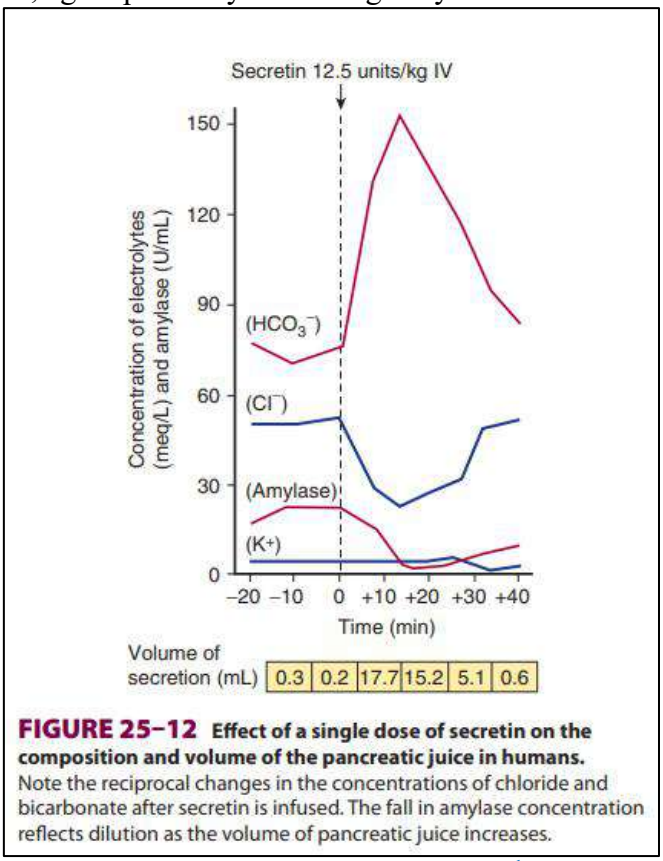


FIGURE 25–12 Effect of a single dose of secretin on the composition and volume of the pancreatic juice in humans. Note the reciprocal changes in the concentrations of chloride and bicarbonate after secretin is infused. The fall in amylase concentration reflects dilution as the volume of pancreatic juice increases.

Ganong's Review of Medical Physiology 24th Edition p. 464

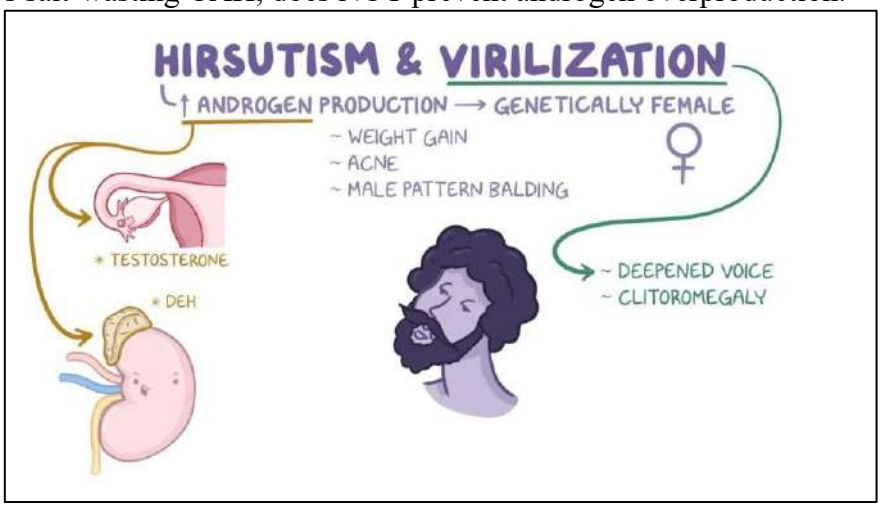
(6) Answer D

Most cases of CAH (especially 21-hydroxylase deficiency) cause:

- ↓ Cortisol
- ↑ ACTH (loss of negative feedback)
- ↑ Adrenal androgen production → virilization

Giving exogenous glucocorticoids restores the negative feedback → ↓ ACTH → ↓ adrenal androgen synthesis.
This is the primary mechanism by which virilization is prevented.
Why other options are incorrect
A. Direct action on cytochrome P450 enzymes
Glucocorticoids do NOT directly modify steroidogenic enzymes.

B. Glucocorticoid action
Replacement is important but not what stops virilization. The effect is indirect via ACTH suppression.
C. Improvement in 21-β hydroxylase level
Enzyme deficiency is genetic; steroids do NOT fix the enzyme.
E. Mineralocorticoid action
Helps salt-wasting CAH, does NOT prevent androgen overproduction.



https://www.osmosis.org/video/Virilization:_Clinical_practice

(7) Answer D
In upright posture:

- Ventilation increases from apex → base (due to better compliance at base)
- Perfusion increases MUCH more steeply from apex → base because of gravity

So, at the apex:

- Ventilation = low
- Perfusion = very low

Because perfusion falls more dramatically than ventilation, the apex has a high V/Q ratio.
Why others are incorrect:
A. Accessory muscles
Irrelevant; normal quiet breathing.
B. More negative intrapleural pressure in apex
True, but this makes alveoli overdistended and less compliant, not high V/Q directly.
C. Lesser compliance in apex
True, but this explains lower ventilation, not high V/Q.
E. Veins in apex under negative pressure
Not the V/Q determining factor.

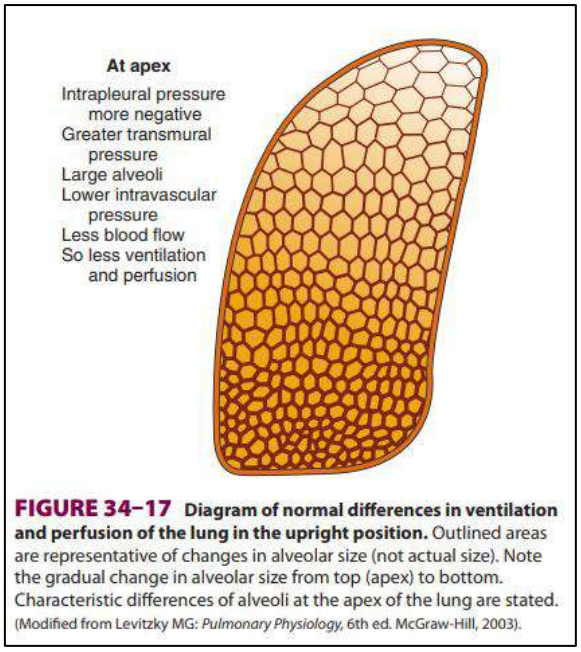


FIGURE 34–17 Diagram of normal differences in ventilation and perfusion of the lung in the upright position. Outlined areas are representative of changes in alveolar size (not actual size). Note the gradual change in alveolar size from top (apex) to bottom. Characteristic differences of alveoli at the apex of the lung are stated. (Modified from Levitzky MG: *Pulmonary Physiology*, 6th ed. McGraw-Hill, 2003).

Ganong's Review of Medical Physiology 24th Edition p. 636

(8) Answer D

Key reasons C-peptide is preferred:

- Insulin from injections interferes with lab measurement.
- Anti-insulin antibodies (common in diabetics) bind insulin, altering plasma insulin levels.
- C-peptide is not present in exogenous insulin, and antibodies do not bind it.

Therefore:
C-peptide reliably reflects endogenous β-cell insulin secretion.
Why other options are incorrect:
A. Both are secreted by the pancreas
True but not the reason it is better.
B. Secreted in equimolar concentrations
True but insufficient explanation.
C. C peptide cleared slower
True, but still not the best reason.
E. C peptide continues to be secreted even when pancreas is damaged
False; if β -cells fail, both stops.

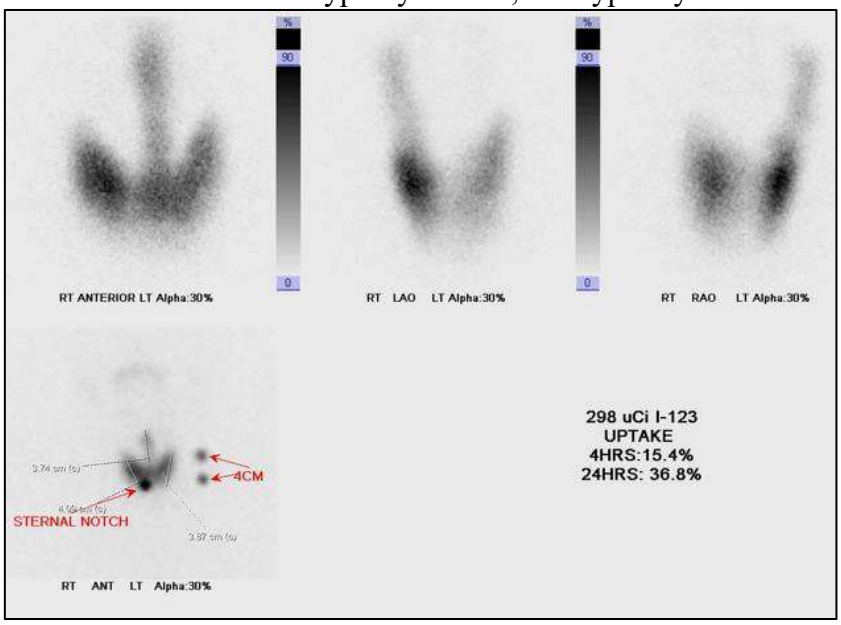
(9) Answer B

A patient who is taking exogenous thyroxine (T_4) and develops clinical features of hyperthyroidism (palpitations, weight loss, heat intolerance, tremor) will show a distinct pattern of investigation results that differentiates exogenous thyrotoxicosis from endogenous causes (Graves', toxic multinodular goiter, thyroiditis).
Exogenous thyroxine leads to high circulating T_4, which in turn suppresses pituitary TSH through the normal negative feedback mechanism. Because the thyroid gland is not overactive, all endogenous markers of thyroid stimulation (like iodine uptake and TSI) will be low or normal, not elevated.
Therefore, the key investigation finding in exogenous thyrotoxicosis is simply:

High serum T_4 with suppressed TSH and low radioiodine uptake.
This is reflected in option B.
Why other options are incorrect
A. Elevated iodine uptake by the gland
Radioiodine uptake is low in exogenous thyroxine use because the thyroid gland is not producing excess hormone.
High uptake is seen in Graves' disease or toxic nodules, where the gland actively synthesizes thyroid hormones.
C. Elevated thyroid-binding globulin (TBG)
TBG levels do not increase with exogenous T_4 administration.
TBG rises in situations like:

- Pregnancy
- Estrogen therapy
- Oral contraceptives
- Hepatitis

It is not a marker of exogenous hyperthyroidism.
D. Elevated thyroid stimulating immunoglobulin (TSI)
TSI is elevated in Graves' disease, the most common form of endogenous hyperthyroidism.
In exogenous thyroxine use:
TSI is normal
Radioiodine uptake is low, not high
Thus, TSI elevation does not fit exogenous hyperthyroidism.
E. Elevated TSH
Exogenous thyroxine suppresses TSH via negative feedback on the hypothalamus and pituitary.
Elevated TSH would indicate hypothyroidism, not hyperthyroidism.



There is increased thyroid uptake at 4- and 24-hour period on I-123 scan diffusely, consistent with Grave's disease.
Also seen is a prominent pyramidal lobe showing the increased radioactive iodine uptake.
https://radiopaedia.org/cases/pyramidal-lobe#image-51647868

(10) Answer C

This patient is hyperventilated (twice normal frequency, normal tidal volume).
Hyperventilation → excessive CO_2 washout → ↓ $PaCO_2$ (hypocapnia).
Carbon dioxide is the main driver of ventilation under anesthesia and quiet breathing because:

- Central (medullary) chemoreceptors respond strongly to ↑ CO_2 (via ↑ H^+ in CSF).
- When $PaCO_2$ falls too low, medullary chemoreceptor activity decreases sharply.

After mechanical hyperventilation:

- $PaCO_2$ is very low
- Medullary chemoreceptors are under-stimulated
- The respiratory drive becomes temporarily "switched off"
- High O_2 further reduces peripheral chemoreceptor input

Therefore, until CO_2 rises again naturally, the patient may not initiate spontaneous breaths, causing an apneic pause.
Why other options are incorrect
A. High arterial PO_2 suppressing peripheral chemoreceptors
High O_2 does suppress peripheral chemoreceptors, but this alone is not sufficient to cause apnea unless $PaCO_2$ is also low.
The dominant mechanism here is hypocapnia, not hyperoxia.
B. Decrease in arterial pH suppressing peripheral chemoreceptors
Hyperventilation causes respiratory alkalosis, not decreased pH.
D. High arterial PCO_2 suppressing medullary chemoreceptors
High CO_2 stimulates ventilation, not suppresses it.
E. Low arterial PCO_2 suppressing peripheral chemoreceptors
Peripheral chemoreceptors mainly respond to PO_2, less to CO_2.
The medullary chemoreceptors are the main CO_2 sensors controlling ventilatory drive.

CAT-2 (BATCH 1) MOCK ESSAY

(1) A 30-year-old male was admitted to the hospital following a road traffic accident. He complained of chest pain during breathing. On examination he was cyanosed, tachypneic and a part of the chest wall is moving inwards during inspiration and moving outwards during expiration.

(1.1) Explain the physiological basis for the abnormal chest wall movements seen during breathing in this patient (30 marks)

(1.2) Define cyanosis and explain the mechanism of development of cyanosis in this patient (20 marks)

(1.3) Explain the mechanism for the development of tachypnoea in this patient (30 marks)

(1.4) Explain the possible findings in arterial blood gas analysis report of this patient (20 marks)

(2) A 30-year-old woman presented to the outpatient clinic with complaints of weight loss despite increased appetite, excessive sweating, palpitations, and frequent bowel movements for the past three months. She also reports excessive thirst, frequent urination, and episodes of fatigue. She has no significant medical history, but her mother has a history of autoimmune thyroid disease.

Physical Examination: Heart rate: 110 bpm Blood pressure: 140/85 mmHg Diffuse, non-tender goiter, Proptosis and lid lag, bilateral Fine tremor of the hands.

Laboratory Investigations: TSH: <0.005 (0.40- 4.5) mIU/L, Free T4: 24.5 (9.0-19) pmol/L, FT3: 14.0 (2.6—6.0) pmol/L, Thyroid receptor antibody (TRAb): 25.4 (<1), FBS 150mg/dl, HbA1c-8.2%

(2.1) What is the overall diagnosis of this patient? (15 marks)

(2.2) Name two clinical and two biochemical reasons for your answer in 2.1(20 marks)

(2.3) Briefly explain the physiological basis for having tachycardia, lid lag, proptosis and diffuse non tender goiter (45 marks)

(2.4) How do you explain for having high Fasting Blood Sugar of this patient? (20 marks)

CAT-2 (BATCH 1) MOCK ESSAY ANSWERS

(1)

(1.1) Explain the physiological basis for the abnormal chest wall movements seen during breathing in this patient (30 marks)

- This condition is called flail chest (2 marks).
- Usually results from blunt trauma associated with multiple rib fractures,
- and is defined as three or more ribs fractured in two or more places. (2 marks)
- paradoxical motion of a chest wall segment will clinically confirm the diagnosis aided by chest pain, cyanosis and tachypnea. (2 marks)
- Inspiration is an active process. (2 marks)
- The contraction of the inspiratory muscles increases intrathoracic volume. (2 marks)
- The intrapleural pressure at the base of the lungs, which is normally Negative (2 marks) /about –2.5 mm Hg (relative to atmospheric)
- at the start of inspiration, decreases (2 marks)/ to about –6 mm Hg.
- The lungs are pulled into a more expanded position. (2 marks)
- The pressure in the airway becomes slightly negative, and air flows into the lungs. (2 marks)
- Same time the flail segment will sucked in by the negative intrathoracic pressure because the flail segment behaves as a separate segment because of lost its attachment to muscles and ribs. (4 marks)
- at the end of inspiration, the lung recoil begins to pull the chest back to the expiratory position. (2 marks)
- where the intrathoracic pressure increases. (2 marks)
- The flail segment will move outwards due to that while the remaining part of the chest wall come inwards (4 marks)

(1.2) Define cyanosis and explain the mechanism of development of cyanosis in this patient (20 marks)

- Cyanosis is Dusky bluish color discoloration of the tissues. (3 marks)
- When the deoxygenated hemoglobin concentration is more than 5g/dl (3 marks)
- During inspiration, the negative intrathoracic pressure causes the flail segment to move inward (paradoxical movement), reducing lung expansion on the affected side. (3 marks)
- During expiration, the flail segment moves outward, further impairing normal ventilation causing poor oxygenation. (3 marks)
- Ultimately leading to Ventilation-Perfusion Mismatch (3 marks)
- leading to hypoxemia. (3 marks)
- As oxygen saturation drops, the hemoglobin in red blood cells carries less oxygen, leading to desaturated (deoxygenated) hemoglobin in the bloodstream. (2 marks)

(1.3) Explain the mechanism for the development of tachypnoea in this patient (30 marks)

- Decreased Ventilation causes Decreased partial pressure of oxygen, increased CO_2 in blood and reduced pH/ increased H^+ (6 marks)

- Stimulation of Peripheral Chemoreceptors in carotid and aortic bodies mainly by Decreased oxygen. Reduced pH and H^+ have a mild effect on peripheral chemoreceptors. (3 marks)
- When partial pressure of CO_2 in blood is high, CO_2 will diffuse in to CSF and form H^+. They stimulate central chemoreceptors which in turn stimulate respiratory center in brain stem. (3 marks)
- Afferents from peripheral chemoreceptors travers via Vagus nerve and glossopharyngeal nerve stimulate the respiratory center. (3 marks)
- Respiratory center consists of group of neurons namely,
- Medulla - Pre Botzinger-complex - pacemaker cells - initiate Spontaneous rhythmic respiration (3 marks)
- Above respiration is modified by Pneumotaxic and apneustic centers in Pons. (3 marks)
- In pons - pneumotaxic center which controls the switch off of the inspiratory signal and start expiration. Early switch off will increase the respiratory rate. (6 marks)
- In addition, dorsal and ventral groups of respiratory neurons in medulla regulate the respiration too. (3 marks)

(1.4) Explain the possible findings in arterial blood gas analysis report of this patient (20 marks)

- Features of Type 1 respiratory failure - PO2 reduced – due to reduced ventilation (2 marks)
- Type 2 respiratory failure - PO_2 reduced & PCO2 increased (Due to CO_2 will not get exhaled) (3 marks)

Respiratory acidosis

- Compensated - pH 7.35- 7.45, High PCO_2 and increased bicarbonate. (5 marks)
- partially compensated - pH < 7.35, High PCO_2 and increased bicarbonate. (5 marks)
- uncompensated - pH < 7.35, High PCO_2, Normal Bicarbonate. (5 marks)

(2)

(2.1) What is the overall diagnosis of this patient? (15 marks)

- Graves' Disease with Diabetes mellitus. (15 marks)

(2.2) Name two clinical and two biochemical reasons for your answer in 2.1 (20 marks)

Clinical Reasons:

- Diffuse, non-tender goiter – Common in Graves' disease due to thyroid-stimulating antibodies causing gland enlargement. (5 marks)
- Proptosis and lid lag – Classic signs of thyroid eye disease (orbitopathy), seen in Graves' disease. (5 marks)

Biochemical Reasons:

- Suppressed TSH (<0.005 mIU/L) (5 marks)
- Elevated TRAb (25.4, normal <1) – Confirms Graves' disease (5 marks)

(2.3) Briefly explain the physiological basis for having tachycardia, lid lag, proptosis and diffuse non tender goiter (45 marks)

Tachycardia

- Thyroid hormones (T3 and T4) increase the number of β-adrenergic receptors
- and affinity to them in the heart, (3 marks)
- leading to increased heart rate and (3 marks)
- increased contractility by increasing proportion of alpha myosin heavy chains. (3 marks)
- Has both ionotropic (3 marks)
- and chronotropic action. (3 marks)

Lid Lag & Proptosis

- Lid Lag: Increased sympathetic stimulation (3 marks)
- causes excessive contraction of the levator palpebrae superioris muscle, (3 marks)
- leading to difficulty in moving the upper eyelid downward. (3 marks)
- Proptosis Graves' disease triggers immune-mediated inflammation of orbital fibroblasts, (3 marks)
- causing swelling of extraocular muscles and pushing the eyeball forward. (3 marks)

Diffuse, Non-Tender Goiter

- TRAb stimulates the thyroid gland excessively, (3 marks)
- leading to hypertrophy and hyperplasia of thyroid follicular cells, (3 marks)
- causing an enlarged thyroid. (3 marks)
- Since this is not due to infection or inflammation, it remains non-tender. (3 marks)

(2.4) How do you explain for having high Fasting Blood Sugar of this patient? (20 marks)

Actions of Thyroid hormones in carbohydrate metabolism.

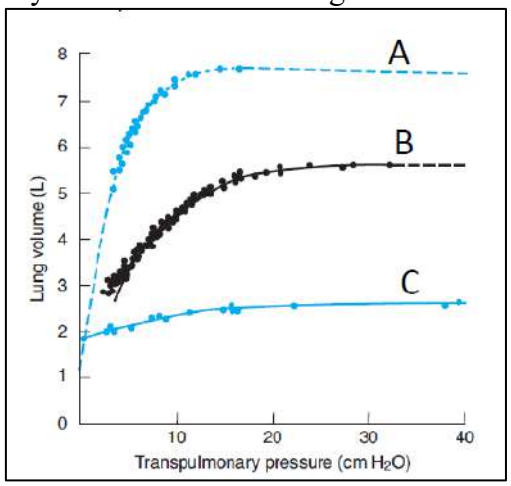
- Excess thyroid hormones (T3 and T4) increase glucose production in the liver (4 marks)
- Increased rate of carbohydrate absorption in the gut.
- This contributes to postprandial hyperglycemia and elevated fasting blood sugar levels. (4 marks)
- Permissive action with catecholamines leads to Hepatic Glycogen Depletion by potentiating the effects of catecholamines. (4 marks)
- Increased breakdown of Insulin.
- Actions of Insulin will be absent increasing blood glucose levels. such as – Increasing glucose uptake by peripheral tissue, and preventing glycogenolysis and gluconeogenesis. (4 marks)
- Possible Autoimmune Diabetes Mellitus.
- The presence of autoimmune thyroid disease (Graves' disease) and a family history of autoimmune disorders raise suspicion for type 1 diabetes or LADA. (4 marks)

CAT-2 (BATCH 1) MOCK OSPE

(1)

(1.1) Name the equipment
(1.2) List two measurements that could be measured with this equipment

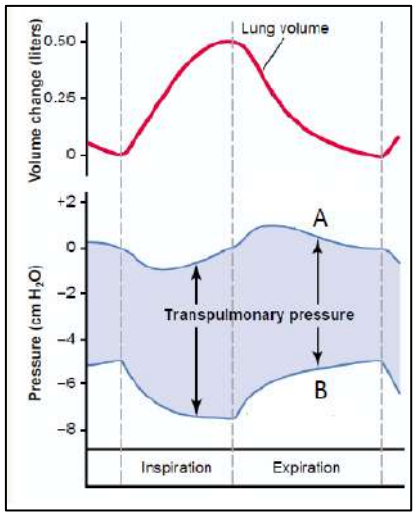
(2) Given above are static expiratory pressure volume curves of lungs in 3 individuals A, B, C. B is a normal healthy adult.
(2.1) Identify the conditions of A & C
(2.2) What is the parameter given by the gradient of the graph?
(2.3) State what happens to the parameter in a
A. Tension Pneumothorax patient
B. Premature baby born in 27th week of gestation



(3)

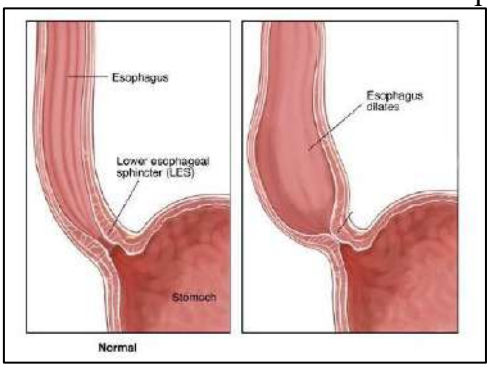
(3.1) What is the instrument given above?
(3.2) What is the measurement taken from this instrument?
(3.3) State what happens to the above-mentioned measurement in the following conditions.
A. Asthma
B. Lung fibrosis

(4)



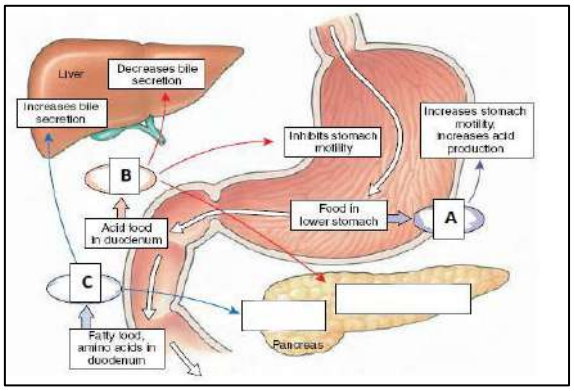
(4.1) What are given by A & B?
(4.2) What is meant by transpulmonary pressure?
(4.3) At the end of expiration, what happens to the transpulmonary pressure and Transthoracic pressure?

(5) Lower esophageal sphincter (LES) is a physiological sphincter located at the lower end of the esophagus.
(5.1) Write 3 component of which maintain the tone of the LES
(5.2) Following picture shows a motor disorder of the esophagus.



A. What is the diagnosis?
B. State the cause for this condition.

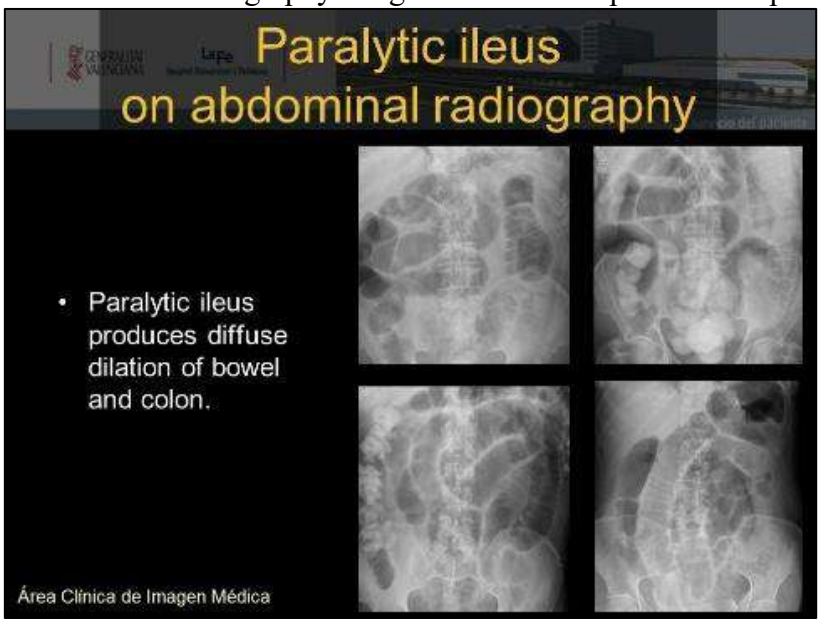
(6)



The above diagram shows the regulation of certain hormones of the GI tract.
(6.1) Name the hormones A, B, C

(6.2) State the action of B & C on the pancreas
(6.3) Which of the above hormones have
C. Positive feedback
D. Negative feedback

(7) These are abdominal radiography images taken from a patient with paralytic ileus.



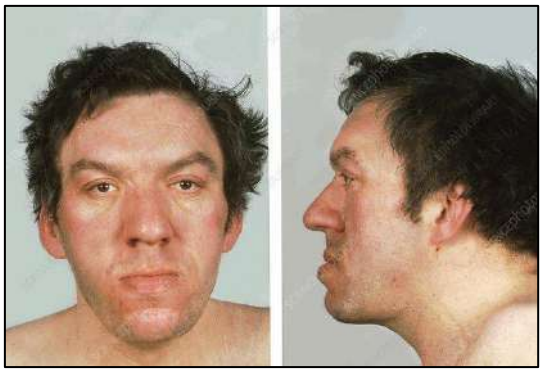
(7.1) List two symptoms this patient might present with
(7.2) Name one common etiology to develop this condition
(7.3) List two complications of paralytic ileus

(8) Demonstrate how you would instruct a patient to get ready for an OGTT (Live Station)

(9)

(9.1) What's the physical sign demonstrated in the image?
(9.2) What is the underlying endocrine condition?
(9.3) Name three etiological factors that cause the condition you mentioned.

(10)



(10.1) Name two physical signs you observe in this patient.
(10.2) What is the clinical diagnosis?
(10.3) List two biochemical tests to diagnose this condition

CAT-2 (BATCH 1) MOCK OSPE ANSWERS

(1)

(1.1) Spirometer

(1.2) Tidal volume, inspiratory reserve volume, expiratory reserve volume, forced vital capacity, forced expiratory volume in 1 second (any 2)

(2)

(2.1) A – Emphysema
C – Lung fibrosis

(2.2) Lung compliance

(2.3)

A. Reduced
B. Reduced

(3)

(3.1) Peak expiratory flow meter

(3.2) Peak expiratory flow rate

(3.3)

C. Reduced
D. Normal/slightly reduced

(4)

(4.1) A – Intrapulmonary pressure
B – Intrapleural pressure

(4.2) The difference between the alveolar pressure and intrapleural pressure

(4.3) Transpulmonary pressure – becomes positive (+5 cmH_2O)
Transthoracic pressure – becomes negative (-5 cmH_2O)

(5)

(5.1) More prominent esophageal smooth muscle at the junction with the stomach (intrinsic sphincter)
Fibers of the crural portion of the diaphragm surrounding the esophagus (extrinsic sphincter)
Oblique/sling fibers of the stomach wall (creates a flap valve)

(5.2)

E. Achalasia cardia
F. The myenteric plexus of the esophagus is deficient at the LES in this condition and the release of NO and VIP is defective.

(6)

(6.1) A – Gastrin
B – Secretin
C – CCK

(6.2) Secretin increases the HCO_3^- secretion by the duct cells of the pancreas
CCK stimulates the pancreas to release enzymes like amylase, lipase, trypsin etc. in pancreatic juice, augments the action of secretin to produce an alkaline pancreatic secretion

(6.3)

G. Gastrin – When stomach pH rises (less acidic), gastrin release can be promoted to increase HCl secretion.
H. Gastrin – When stomach pH drops below ~2 (high acidity), gastrin secretion is inhibited.
Secretin – When duodenal pH rises toward neutrality, secretin release decreases.
CCK – When bile and pancreatic enzymes effectively digest nutrients, CCK release diminishes.

(7)

(7.1) Vomiting, abdominal distension, constipation (any 2)
(7.2) Fractures of spine and pelvis, retroperitoneal hemorrhage, peritonitis, hypokalemia, drugs (ganglion blockers, anticholinergic agents), abdominal surgery, immobilization (any 1)
(7.3) Dehydration, electrolyte imbalances, bowel ischemia, bowel perforation, sepsis (any 2)

(8) "For the OGTT, please continue your normal diet for 3 days, including plenty of carbohydrates.
The night before the test, fast for 8–12 hours—you can drink only water.
On the morning of the test, come to the lab without eating breakfast.
We'll take your fasting blood sample, give you a sweet glucose drink, and then take more blood after 2 hours.
Please stay seated, don't eat or smoke during the test, and you can go back to normal activities afterward."

(9)

(9.1) Purple striae
(9.2) Cushing syndrome
(9.3) Corticosteroid drug use, pituitary tumors, adrenal gland tumors, ectopic ACTH producing tumors (any 3)

(10)

(10.1) Protrusion of jaw, protrusion of brow, intradental separation, large tongue (any 2)
(10.2) Acromegaly
(10.3) Plasma GH levels, serum IGF-1 levels, OGTT, serum prolactin (any 2)

2023/2024 (2nd Batch) CAT-2 Mock Exam

CAT-2 (BATCH 2) MOCK MCQS

MULTIPLE CHOICE QUESTIONS

Time: 1 hour

This paper consists of 10 True or False type questions and 10 Single Best Answer type questions.

(1) Diabetes Insipidus causes fall in,
- (A) Extracellular but not intracellular blood volume.
- (B) Extracellular osmolality.
- (C) Intracellular osmolality.
- (D) Osmolality of urine.
- (E) Water reabsorption from PCT.

(2) 1,25 dihydrocolicalciferol,
- (A) Synthesis is inhibited by PTH.
- (B) Induces formation of calcium binding proteins.
- (C) Increases absorption of Ca from GIT.
- (D) Causes mineralization of newly formed osteoids.
- (E) Deficiency can cause rickets in children.

(3) A 39-year-old man with type 1 diabetes mellitus is brought to the emergency room. An insulin treatment expected to be decreased his,
- (A) Blood glucose concentration
- (B) Blood K+ concentration
- (C) Urinary glucose concentration
- (D) Blood Ph
- (E) Breathing rate

(4) Glucocorticoids,
- (A) Mobilize fatty acids from adipose tissue.
- (B) Decrease protein synthesis in the liver.
- (C) Decrease peripheral glucose utilization.
- (D) Suppress the immune system.
- (E) Stimulate gluconeogenesis.

(5) T/F regarding gastric juice?
- (A) Vagal stimulation increases gastric juice secretion.
- (B) Secretion is associated with decreased pH in venous blood which is draining stomach.
- (C) Essential for vitamin B12 absorption.
- (D) Secretion is increased by hormone secretion.
- (E) Increases in Zollinger Ellison syndrome

(6) T/F regarding the physiology of gastrointestinal system?
- (A) The chemoreceptor trigger zone is responsible for vomiting during motion sickness.
- (B) Defecation reflex starts with the distension of the rectum.

(C) Irritation of gastric intestinal mucosa is a stimulation for vomiting.
(D) Saliva is hypotonic.
(E) Sleep inhibits the secretion of saliva.

(7) Saliva,
(A) Different salivary gland secretions have similar composition.
(B) Does not have blood group agglutinogens.
(C) Contains enzymes essential for digestion of carbohydrates.
(D) Increase secretion when sympathetic nerve supply is stimulated.
(E) pH is 5-6.

(8) T/F regarding the mechanism of inspiration?
(A) Alveolar Pressure is positive relative to the atmosphere.
(B) External intercostal muscles elevate the ribs.
(C) Intrathoracic volume is decreased in inspiration.
(D) Diaphragm contract and increase its convexity towards thoracic cavity.
(E) During inspiration elastic recoil of lung increases.

(9) T/F regarding carotid bodies,
(A) They are stretch receptors in the walls of the internal carotid artery.
(B) Carotid body is located in the bifurcation of the common carotid artery.
(C) When carotid body senses hypoxia it increases heart rate.
(D) They generate more afferent impulses when blood H^+ ion concentration rises.
(E) They are responsible for the increased ventilation in hypoxia.

(10) T/F regarding hypoxia?
(A) Hypoxia due to anemia presents with normal PaO_2.
(B) CO poisoning causes significant cyanosis.
(C) In heart failure, hypoxia stimulates the central chemo receptors.
(D) In ischaemic hypoxia, PO_2 and Hb levels are normal.
(E) Due to hypoxia, cellular oxidative enzyme levels increase.

Single Best Answer Questions

(1) A 28-year-old woman develops excessive bleeding during childbirth. Later, she presents with failure of lactation, fatigue, loss of pubic hair, and features of hypopituitarism. Laboratory investigations reveal reduced levels of GH, LH, FSH, ACTH, prolactin, ADH, and oxytocin. What is the most likely diagnosis?
(A) Pituitary tumor
(B) Sheehan's syndrome
(C) Acromegaly
(D) Cushing's syndrome
(E) GH deficiency

(2) 30-year-old woman complains of cold intolerance, weight gain and tiredness. What is the appropriate investigation?
(A) Radioactive iodine uptake
(B) Circulatory TSH concentration

(C) Thyroxine (T4)
(D) Tri iodo thyroxin (T3)
(E) Free T

(3) A 30-year-old man is undergoing an investigation of hypertension. He complains about episodic headaches, palpitations and sweating. VMA elevation was shown in urine test. What is the possible diagnosis?

(A) Thyrotoxicosis
(B) Acromegaly
(C) Cushing syndrome
(D) Primary hyperaldosteronism
(E) Pheochromocytoma

(4) A 70-year-old woman is admitted with SOB and cough. She is a known COPD patient. On examination she has a normal respiratory rate. O_2 saturation of 88% was observed. The most likely step you would take out initially on this patient is,

(A) Peak flow assessment
(B) ABG analysis
(C) Obtain sputum for a culture
(D) Urine dipstick
(E) Artificial ventilation

(5) A 49-year-old heavy smoker visits the medical clinic complaining of dry cough and generalized body swelling during last month of period. His lung function shows FEV1/FVC ratio 50 and chest X-ray shows hyperinflated lungs. Lung function did not reverse with the treatment of beta 2 agonists. What is the most likely diagnosis?

(A) Asthma
(B) Emphysema
(C) Lung fibrosis
(D) Pneumonia
(E) Pleural effusion

(6) Which is the organ in which you do not find air in a healthy person?

(A) Bile duct
(B) Rectum
(C) Small intestine
(D) Transverse colon
(E) Stomach

(7) A 25-year-old man presented with recurrent right upper quadrant pain suggestive of biliary pain. There was no clear evidence of liver cell damage or gallstone. Ultrasound examination after intra venous infusion of CCK indicate dilation of common bile duct. Further IV infusion of the secretin caused pancreatic duct dilation. These findings are most consistent with,

(A) A defect in the secretions of bile by hepatocytes
(B) A defect in secreting of bile by hepatocytes
(C) Spasm of sphincter of Oddi

(D) A Defect in the processing of bile by cholangiocytes
(E) A Defect in the processing pancreatic secretion by pancreatic epithelium cells

(8) A 35-year-old man with a history of chronic cough and weight loss undergoes a chest X-ray, which shows cavitary lesions localized to the upper lobes of the lungs. From a physiological perspective, which of the following best explains why *Mycobacterium tuberculosis* has a predilection for the lung apices?

(A) Higher pulmonary perfusion in apices than bases
(B) Greater lymphatic drainage at the apices
(C) Reduced alveolar ventilation at the apices compared to bases
(D) Higher alveolar oxygen tension in the apices compared to bases
(E) Reduced elastic recoil of alveoli at the apices

(9) A 32-year-old man with moderate persistent asthma is started on a combination of inhaled corticosteroids and a long-acting β_2-agonist (LABA). Which of the following best explains the rationale for this combination in long-term asthma control?

(A) LABA prevents airway inflammation while corticosteroids act as bronchodilators
(B) Corticosteroids reduce airway inflammation, while LABA provides sustained bronchodilation to counter reversible obstruction
(C) LABA and corticosteroids both act by increasing cyclic AMP in airway smooth muscle
(D) Corticosteroids prevent airway remodeling, while LABA prevents mucus secretion
(E) LABA is short-acting, so corticosteroids prolong its bronchodilator effect

(10) A 30-year-old professional diver is brought to the emergency department after rapidly ascending from a deep-sea dive. Soon after surfacing, he developed confusion, dizziness, joint pain, and difficulty in concentration. Which of the following best explains the underlying pathophysiology of his condition?

(A) Decreased oxygen carrying capacity due to carboxyhemoglobin formation
(B) Inhibition of cytochrome oxidase by dissolved nitrogen in tissues
(C) Excess dissolved nitrogen altering excitability of neuronal membranes
(D) Formation of nitrogen gas bubbles in blood and tissues during rapid decompression, leading to vascular obstruction and tissue ischemia
(E) Increased pulmonary compliance leading to alveolar hypoventilation

CAT-2 (BATCH 2) MOCK MCQ ANSWERS

(1) Due to an ADH deficiency or failure of kidneys to respond to ADH water reabsorption from collecting ducts is reduced.

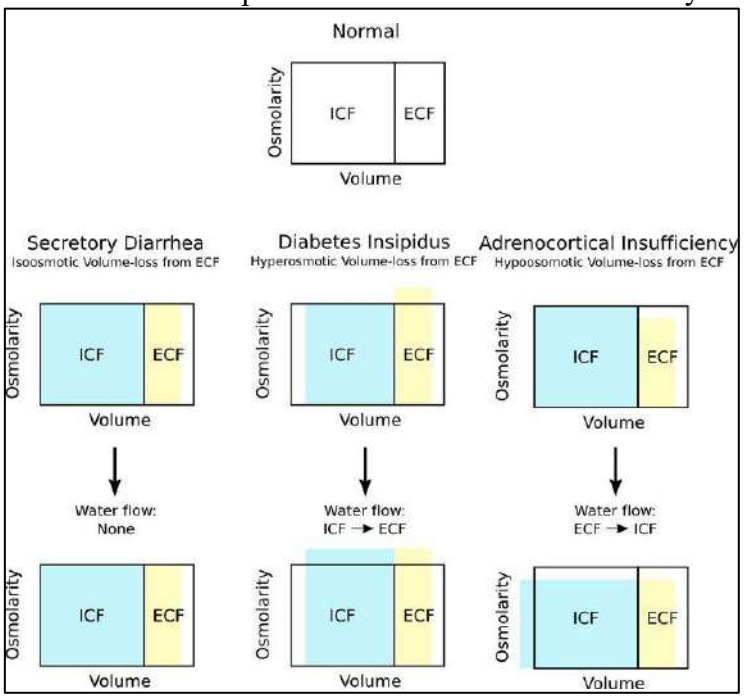
(A) **F** – ECF volume and ICF volume are decreased.

(B) **F** – Extracellular osmolality ↑

(C) **F** – Intracellular osmolality ↑

(D) **T** – Urine volume is decreased → hypotonic to plasma→ osmolality ↓

(E) **F** – Water reabsorption from PCT is not controlled by ADH.



https://www.pinterest.com/pin/614882155344104903/

(2)

(A) **F** – PTH increases the formation of 1,25 dihydrodeoxycholecalceferol in kidneys.

(B) **T** – Increases calbindin

(C) **T** – By increasing the formation of calbindin proteins, Ca absorption from the GIT is increased

(D) **T**

(E) **T** – Vitamin D deficiency causes defective calcification of bone matrix which can cause rickets in children

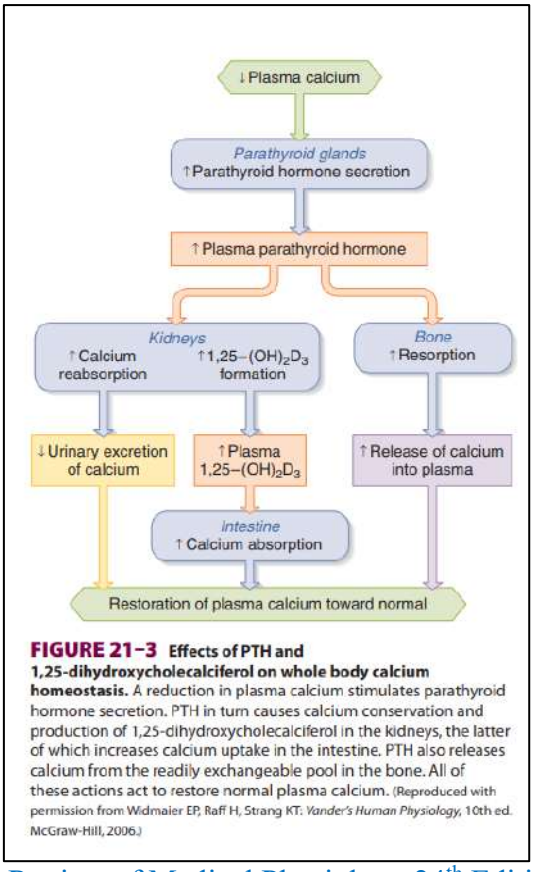


FIGURE 21–3 **Effects of PTH and 1,25-dihydroxycholecalciferol on whole body calcium homeostasis.** A reduction in plasma calcium stimulates parathyroid hormone secretion. PTH in turn causes calcium conservation and production of 1,25-dihydroxycholecalciferol in the kidneys, the latter of which increases calcium uptake in the intestine. PTH also releases calcium from the readily exchangeable pool in the bone. All of these actions act to restore normal plasma calcium. (Reproduced with permission from Widmaier EP, Raff H, Strang KT: *Vander's Human Physiology*, 10th ed. McGraw-Hill, 2006.)

Ganong's Review of Medical Physiology 24th Edition p. 381

(3)

(A) **T**
(B) **T** – Insulin causes transport of K^+ into the cells
(C) **T**
(D) **F** – ketoacidosis is a hallmark of type 1 diabetes → pH is reduced. Insulin treatment will reduce FA metabolism → ↓ formation of ketone bodies→ pH is increased
(E) **T** – Ketoacidosis may cause hyperventilation (Kussmaul breathing)

TABLE 24–2 Effects of insulin on various tissues.

Adipose tissue
Increased glucose entry
Increased fatty acid synthesis
Increased glycerol phosphate synthesis
Increased triglyceride deposition
Activation of lipoprotein lipase
Inhibition of hormone-sensitive lipase
Increased K^+ uptake
Muscle
Increased glucose entry
Increased glycogen synthesis
Increased amino acid uptake
Increased protein synthesis in ribosomes
Decreased protein catabolism
Decreased release of gluconeogenic amino acids
Increased ketone uptake
Increased K^+ uptake
Liver
Decreased ketogenesis
Increased protein synthesis
Increased lipid synthesis
Decreased glucose output due to decreased gluconeogenesis, increased glycogen synthesis, and increased glycolysis
General
Increased cell growth

Ganong's Review of Medical Physiology 24th Edition p. 434

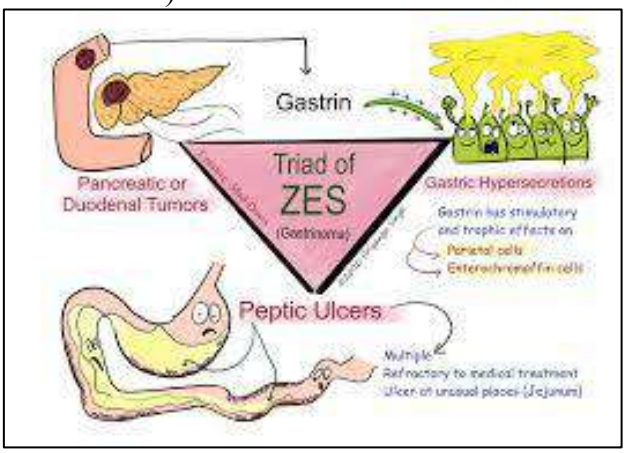
(4)

(A) **T** – enhance lipolysis and increase FFA
(B) **F** – protein synthesis in liver is increased while catabolism increased in muscles
(C) **T**
(D) **T** – anti-inflammatory action
(E) **T**

(5)

(A) **T** – parasympathetic stimulation by vagus nerve stimulates all cells of the gastric gland (ECL cells, chief cells, parietal cells stimulated by ACh from vagus nerve, and vagal outflow will release GRP and stimulate G cells to release Gastrin, D cells are not stimulated) to secrete gastric juice.
(B) **F** – the secretion of protons into the lumen by parietal cells is accompanied by the release of equivalent numbers of HCO_3^- ions into the bloodstream, which is later used to neutralize gastric acidity. This raise in pH after a meal, first in the portal blood then later in the systemic blood, is called postprandial alkaline tide
(C) **T** – Intrinsic factor secreted by parietal cells is needed to absorb vitamin B12 in the terminal ileum.
(D) **T** – Secretion of gastric juice by parietal cells, chief cells and ECL cells is stimulated by gastrin hormone.

(E) **T** – Zollinger Ellison syndrome is characterized by a triad of gastric acid hypersecretion, peptic ulcers and gastrinoma (pancreatic or duodenal tumors).



https://creativemeddoses.com/topics-list/zollinger-ellison-syndrome-zes/

(6)

(A) **T** – But the brain stem vomiting center motion is mainly responsible for motion vertigo. But Lesions in the area postrema chemoreceptor trigger zone have a little effect on the vomiting response to gastrointestinal irritation or motion sickness.

(B) **T** – Distention of the rectum with feces initiates reflex contractions of its musculature and the desire to defecate. In humans, the sympathetic nerve supply to the internal (involuntary) anal sphincter is excitatory, whereas the parasympathetic supply is inhibitory. This sphincter relaxes when the rectum is distended. The urge to defecate first occurs when rectal pressure increases to about 18 mm Hg. When this pressure reaches 55 mm Hg, the external as well as the internal sphincter relaxes and there is reflex expulsion of the contents of the rectum. This is why reflex evacuation of the rectum can occur even in the setting of spinal injury.

(C) **T** – The Impulses are relayed from the mucosa to the medulla via sympathetic nerves and vagi to mainly nucleus tractus solitarius and to chemoreceptor trigger zone.

(D) **T** – The composition of the saliva modified as it flows from acini out in to ducts. Na + and Cl- are excreted and K+ and bicarbonate are added. Because the ducts are relatively impermeable to water, the loss of NaCl makes the saliva hypotonic.

(E) **T** – sleep inhibits the secretion of saliva. Saliva production decreases significantly during sleep compared to when you are awake. This is because the salivary glands are less active at night due to reduced stimulation from chewing, speaking, or sensory input, as well as changes in nervous system activity.

During sleep, the parasympathetic nervous system predominantly regulates bodily functions. Although the parasympathetic system generally promotes salivation, the lack of stimuli and changes in bodily priorities during sleep result in reduced saliva secretion.

(7)

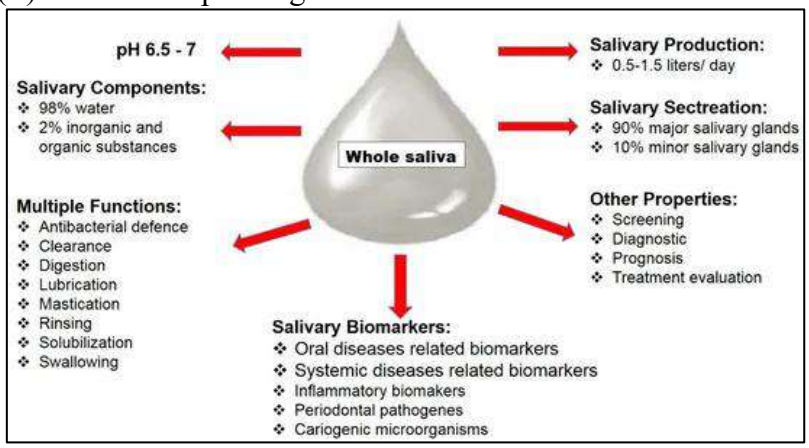
(A) **F** – Parotid gland is a serous gland: secretes non viscous saliva containing water, electrolytes & enzymes. Submandibular and sublingual glands are mixed glands: secrete viscous saliva rich in mucin

(B) **F** – Saliva contains blood group antigens/Agglutinogens (only ABO, not Rh)

(C) **T** – Saliva contains salivary alpha amylase (optimal pH 6.7 - action stops in stomach) for chemical digestion of starch to maltose.

(D) **T** – Sympathetic input slightly modifies the composition of saliva by increasing the proteinaceous content but has little influence on the volume. Both sympathetic and parasympathetic systems stimulate salivation, but the effect is stronger in parasympathetic.

(E) **F** – Normal pH range of saliva is 6.7-7



https://www.mdpi.com/2075-4418/7/1/7

(8)

(A) **F** – Alveolar pressure is negative relative to the atmospheric pressure.

(B) **T** – Being one of the accessory respiratory muscles, the external intercostals elevate ribs during forced inspiration. This increases the transverse and anteroposterior diameter of the lungs, which in turn decreases the intrapleural pressure.

(C) **F** – Intra thoracic volume increase during inspiration.

(D) **F** – Diaphragm contracts but it decreases its convexity

(E) **F** – Relaxation of the inspiratory muscles allows the increased alveolar elastic recoil to decrease the volume of the alveoli, increasing alveolar pressure above atmospheric pressure. It increases in the expiration

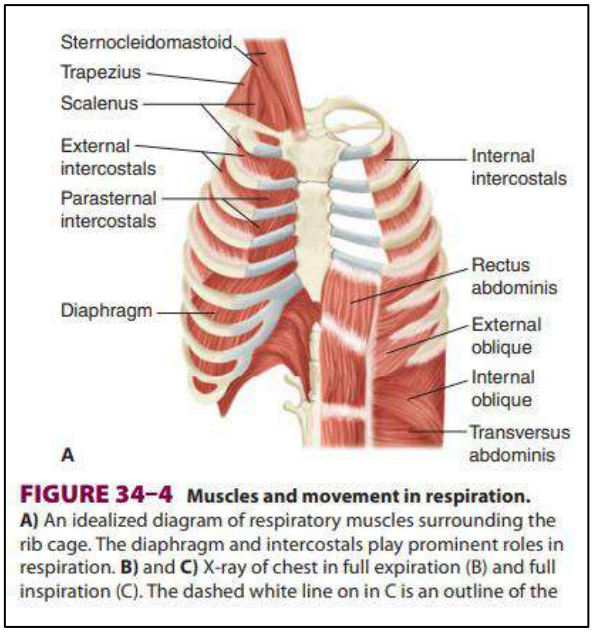


FIGURE 34–4 **Muscles and movement in respiration.** **A)** An idealized diagram of respiratory muscles surrounding the rib cage. The diaphragm and intercostals play prominent roles in respiration. **B)** and **C)** X-ray of chest in full expiration (B) and full inspiration (C). The dashed white line on in C is an outline of the

Ganong's Review of Medical Physiology 24th Edition p. 626

(9)

(A) **F** – Carotid bodies are chemoreceptors. Not stretch receptors. (Baroreceptors)
(B) **T** – Carotid bodies are located in the bifurcation of the common carotid artery.
(C) **T** – When carotid bodies senses hypoxia it increases heart rate, blood pressure, and ventilation rate.
(D) **T** – When blood pH drops, carotid bodies stimulate and generate more afferent impulses.
(E) **T** – In hypoxic condition carotid bodies are responsible for compensatory hyperventilation.

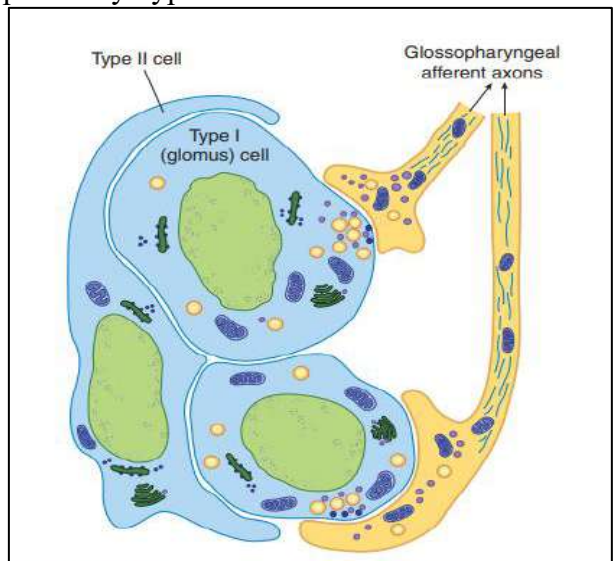


FIGURE 36–5 **Organization of the carotid body.** Type I (glomus) cells contain catecholamines. When exposed to hypoxia, they release their catecholamines, which stimulate the cuplike endings of the carotid sinus nerve fibers in the glossopharyngeal nerve. The glia-like type II cells surround the type I cells and probably have a sustentacular function.

Ganong's Review of Medical Physiology 24th Edition p. 660

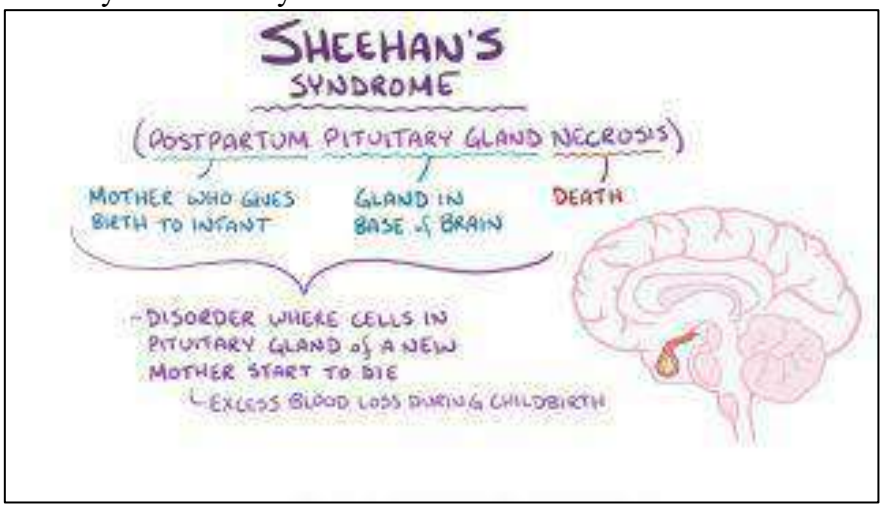
(10)

(A) **T** – True. In anemic hypoxia, arterial PO_2 is normal but amount of Hb to carry oxygen is reduced. So, due to anemia hypoxia can be occurred.
(B) **F** – Cyanosis is taken place because of the venous to arterial shunts. In ASD, large amount of deoxygenated venous blood bypass the lung and dilute the oxygenated blood in right to left shunt. This causes cyanosis.
(C) **F**
(D) **T** – In ischemic hypoxia, Hb level and Po2 level is normal but, the blood flow to a tissue is low.
(E) **T**

Single Best Answer Questions

(1) Answer B

B. Sheehan's syndrome – Correct. Severe postpartum hemorrhage causes ischemic necrosis of the anterior pituitary (due to hypoperfusion of the enlarged gland during pregnancy). This results in hypopituitarism: ↓ GH, LH, FSH, ACTH, PRL, and in some cases posterior pituitary involvement → ↓ ADH and oxytocin. Classic presentation includes failure of lactation (low prolactin) and amenorrhea.
Why other options are incorrect:
A. Pituitary tumor – Can cause hypopituitarism by compression, but not specifically related to postpartum hemorrhage.
C. Acromegaly – Due to GH-secreting pituitary adenoma, leads to excess GH, not deficiency.
D. Cushing's syndrome – Caused by excess cortisol, usually due to ACTH-producing tumor or adrenal pathology, not associated with childbirth hemorrhage.
E. GH deficiency – Only explains lack of growth hormone, but here multiple pituitary hormones (panhypopituitarism) are affected.
Thus, the clinical scenario of childbirth hemorrhage + hypopituitarism is classically Sheehan's syndrome.



https://www.osmosis.org/learn/Sheehan_syndrome

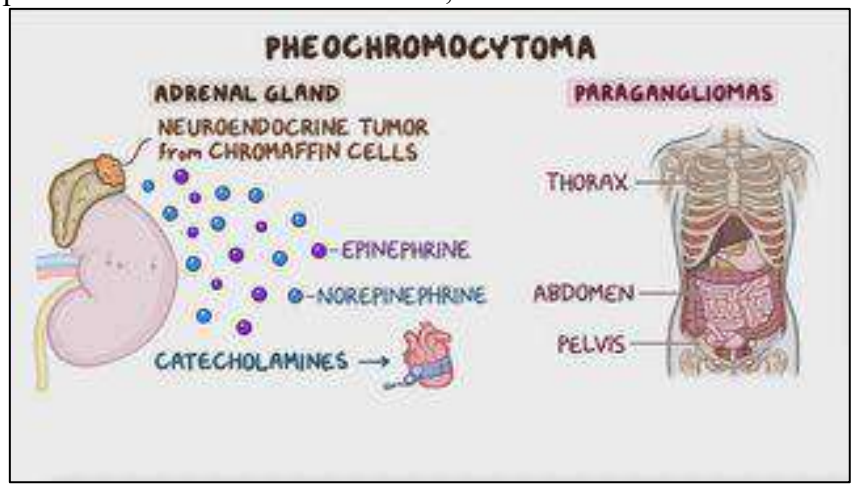
(2) Answer B

Explanation

TSH measurement is the single most sensitive and specific screening test for primary hypothyroidism.
In primary hypothyroidism (thyroid gland failure), circulating TSH is elevated due to loss of negative feedback from low T4 and T3.
Free T4 is also important, but TSH rises much earlier than a fall in T4, making TSH the best initial test.
Serum T3 is less reliable in hypothyroidism because conversion of T4 to T3 may maintain near-normal T3 levels until late stages.
Radioactive iodine uptake is used primarily in the evaluation of hyperthyroidism to assess gland activity, not in suspected hypothyroidism.
Total T4 may be influenced by variations in thyroxine-binding globulin (TBG), so free T4 or TSH is preferred.

(3) Answer E

Explanation
Pheochromocytoma is a catecholamine-secreting tumor of chromaffin cells, usually in the adrenal medulla.
Classic features include paroxysmal hypertension, episodic headaches, palpitations, diaphoresis, and anxiety. These symptoms are due to episodic release of epinephrine and norepinephrine.
Catecholamines are metabolized in the liver and other tissues to metanephrines and ultimately to vanillylmandelic acid (VMA), which is excreted in the urine.
Elevated urinary VMA is a key biochemical marker for pheochromocytoma.
Why other options are incorrect:
A. Thyrotoxicosis: Causes tachycardia, weight loss, tremors, heat intolerance, but not elevated urinary VMA.
B. Acromegaly: Due to excess GH, presents with acral enlargement, prognathism, diabetes mellitus—not catecholamine excess.
C. Cushing's syndrome: Caused by chronic glucocorticoid excess (endogenous or exogenous). Symptoms: central obesity, striae, muscle weakness—not episodic catecholamine surges.
D. Primary hyperaldosteronism: Characterized by hypertension with hypokalemia and metabolic alkalosis, but not associated with elevated VMA.



https://www.osmosis.org/learn/Pheochromocytoma:_Clinical_sciences

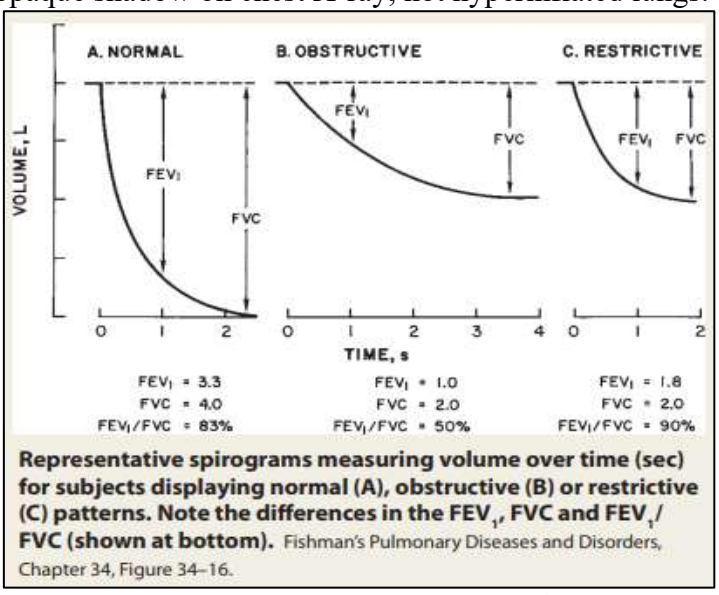
(4) Answer B

Explanation

In COPD, chronic hypoxemia and hypercapnia are common. An O_2 saturation of 88% indicates significant hypoxemia.
ABG analysis is the first step to determine the exact degree of hypoxemia (PaO_2), hypercapnia ($PaCO_2$), and acid-base status (respiratory acidosis or compensation).
Peak flow assessment is mainly useful in asthma exacerbations, not in acute hypoxemia with COPD.
Sputum culture is important if infection is suspected, but it is not the immediate first step in assessing severity of hypoxemia.
Urine dipstick is unrelated to the presenting complaint.
Artificial ventilation is not initiated immediately without first assessing the ABG values and clinical status.

(5) Answer B

Explanation
Emphysema is a chronic obstructive pulmonary disease characterized by irreversible destruction of alveolar walls and loss of elastic tissue, leading to airway collapse during expiration.
In smokers, protease–antiprotease imbalance (e.g., excess elastase activity and reduced α_1-antitrypsin protection) contributes to alveolar destruction.
Clinical features include progressive dyspnea, chronic cough, hyperinflated chest (barrel-shaped thorax), reduced FEV_1/FVC (<70%), and poor bronchodilator reversibility.
Asthma usually shows reversible airflow limitation after bronchodilator therapy, which was absent in this case.
Lung fibrosis is a restrictive lung disease (low total lung capacity, normal or high FEV_1/FVC ratio), not obstructive.
Pneumonia presents with fever, productive cough, and consolidation on chest X-ray, not hyperinflation.
Pleural effusion shows stony dullness on percussion, reduced breath sounds, and an opaque shadow on chest X-ray, not hyperinflated lungs.



Representative spirograms measuring volume over time (sec) for subjects displaying normal (A), obstructive (B) or restrictive (C) patterns. Note the differences in the FEV_1, FVC and FEV_1/FVC (shown at bottom). Fishman's Pulmonary Diseases and Disorders, Chapter 34, Figure 34–16.

Ganong's Review of Medical Physiology 24th Edition p. 630

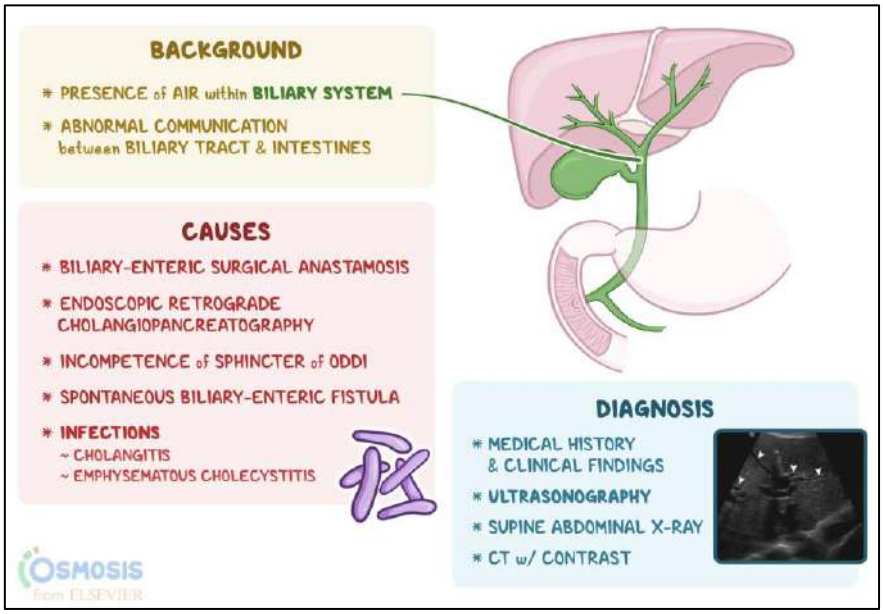
(6) Answer A

Explanation

Air in the stomach, small intestine, colon, and rectum is a normal finding because swallowed air and bacterial fermentation generate intraluminal gas.

Air in the biliary tree (pneumobilia) is abnormal and usually occurs when there is an abnormal communication between the intestine and biliary tract (e.g., gallstone ileus, biliary-enteric fistula, or after surgical instrumentation).

Hence, among the options listed, bile duct is the only structure where air is abnormal.



https://www.osmosis.org/answers/pneumobilia

(7) Answer C

Explanation

Cholecystokinin (CCK) normally stimulates contraction of the gallbladder and relaxation of the sphincter of Oddi, allowing bile to flow into the duodenum.

Secretin primarily stimulates the secretion of bicarbonate-rich fluid from the pancreatic ductal cells and also promotes pancreatic ductal flow.

In this patient, both the common bile duct and pancreatic duct dilated after administration of CCK and secretin, indicating that outflow from both ducts was obstructed at their common junction (ampulla of Vater).

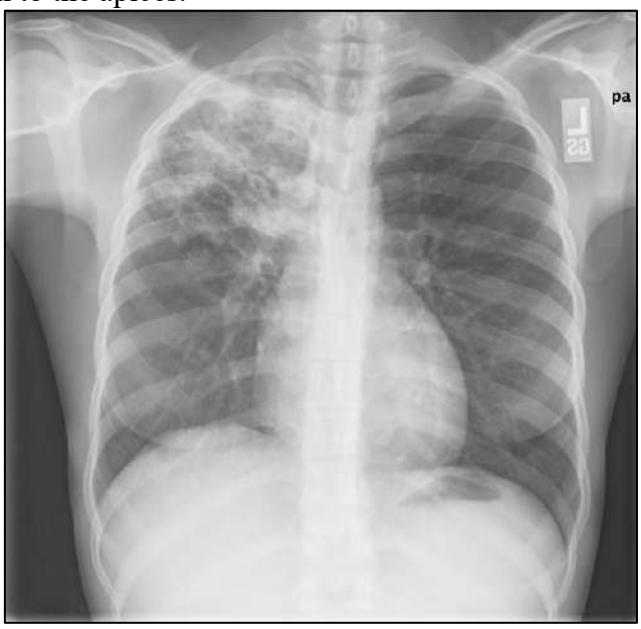
This is most consistent with functional obstruction due to spasm of the sphincter of Oddi, rather than a defect in bile formation or pancreatic secretion.

Hepatocyte or cholangiocyte defects cause impaired bile secretion or conjugation but would not explain the simultaneous ductal dilatation in response to CCK and secretin.

(8) Answer D

According to the regional differences in ventilation–perfusion (V/Q) ratios described in Ganong's Physiology, the lung apex has relatively low perfusion due to gravity-dependent distribution of pulmonary blood flow, while ventilation is also reduced but to a lesser degree. As a result, the V/Q ratio is high at the apices, leading to higher alveolar PO_2 compared to the bases. Mycobacterium tuberculosis is an obligate aerobe, and thus preferentially

proliferates in regions with higher oxygen tension — explaining its localization to the apices.



Multifocal patchy opacities in the right upper lobe with thickening and upward shift of the minor fissure.
https://radiopaedia.org/cases/pulmonary-tuberculosis-29

(9) Answer B

Explanation:

Asthma is characterized by chronic inflammation, mucus hypersecretion, and reversible airway obstruction due to bronchoconstriction. Corticosteroids suppress inflammatory pathways, reducing edema and mucus production, while LABAs maintain prolonged relaxation of bronchial smooth muscle by β_2-receptor–mediated cAMP accumulation. Together, they target both major components of asthma pathophysiology — inflammation and bronchoconstriction — thereby providing superior long-term control compared to either therapy alone (Ganong's Physiology).

(10) Answer D

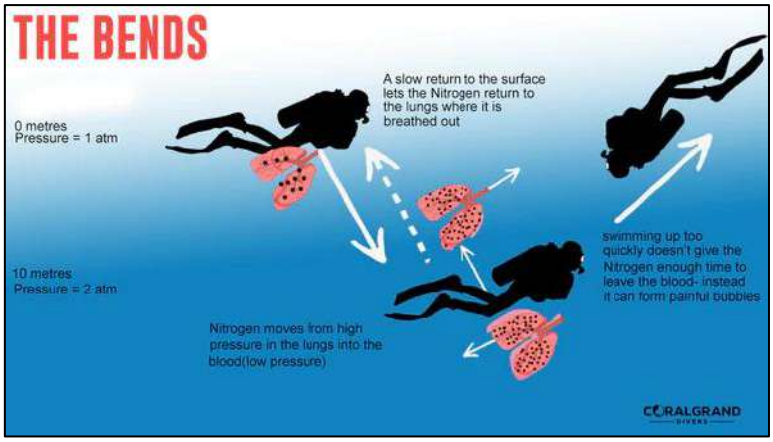
During deep-sea diving, high ambient pressure causes excess nitrogen to dissolve in blood and tissues.

On rapid ascent, the sudden fall in pressure causes nitrogen to come out of solution, forming bubbles in the circulation and tissues.

These bubbles mechanically obstruct vessels and trigger inflammatory responses → causing the bends" (joint pain), neurological symptoms, and cardiopulmonary manifestations.

This is called decompression sickness, distinct from nitrogen narcosis (which is due to dissolved nitrogen acting like an anesthetic at high pressure).

Options A, B, C, and E are not consistent with the pathology of rapid ascent.



https://coralgranddivers.com/blogs/coral-blog/can-i-fly-after-scuba-diving?srsltid=AfmBOorXHFMeBhl3rJEWboM3AsNZfI_c-NFxySrg1SlvodCiDQ7oDqM1

CAT-2 (BATCH 2) MOCK ESSAY

(1) A 46-year-old man with a history of Chronic Liver Cell Disease is presenting abdominal distension.
The following signs are seen on examination - icterus, flapping tremors, ascites and gynecomastia.
(1.1) List three proteins that are produced by the liver. (15 marks)
(1.2) List three other functions of the liver. (15 marks)
(1.3) Explain the physiological basis of developing ascites in this patient. (60 marks)
(1.4) List two other signs that could arise in this patient. (10 marks)

(2) Write short notes on following
(2.1) Physiology of neonatal respiratory distress syndrome (20 marks)
(2.2) Effects of High-altitude respiration and chronic mountain sickness. (40 marks)
(2.3) Effects of voluntary hyperventilation (40 marks)

CAT-2 (BATCH 2) MOCK ESSAY ANSWERS

(1)

(1.1) List three proteins produced by the liver:

- Albumin – maintains plasma oncotic pressure and transports various substances.
- Clotting factors – e.g., fibrinogen, prothrombin (factors II, VII, IX, X).
- Transport proteins – e.g., transferrin (iron transport), ceruloplasmin (copper transport).

(1.2) List three other functions of the liver:

- Metabolism of carbohydrates, fats, and proteins – e.g., glycogen storage, gluconeogenesis, urea formation.
- Detoxification and excretion – e.g., ammonia detoxification, drug metabolism, bile formation.
- Storage of vitamins and minerals – e.g., vitamin A, D, B12, and iron/copper.

(1.3) Explain the physiological basis of developing ascites in this patient:

- Ascites is the accumulation of fluid in the peritoneal cavity.
- Physiological basis of developing ascites in this patient:
  - Altered Starling forces:
    - Hydrostatic pressure: Fibrosis and cirrhosis increase portal venous pressure, raising the hydrostatic pressure in splanchnic capillaries.
    - Oncotic pressure: Impaired hepatic synthesis of albumin reduces plasma oncotic pressure.
    - Net effect: According to Starling's law, the balance between hydrostatic and oncotic pressures favors fluid movement from capillaries into the peritoneal cavity, leading to ascites.
  - Renin-Angiotensin-Aldosterone System (RAAS) activation:
    - Reduced effective circulating blood volume due to splanchnic vasodilation (from nitric oxide and other vasodilators in cirrhosis) and edema (most of volume in tissues) is sensed as hypovolemia by the kidneys.
    - Renin release from the juxtaglomerular cells → angiotensin II formation → systemic vasoconstriction.
    - Aldosterone secretion from the adrenal cortex → sodium and water retention in the kidneys.
    - ADH (vasopressin) release → water reabsorption from renal collecting ducts.
    - Combined effect: increased intravascular volume, but due to continued portal hypertension and hypoalbuminemia, the fluid preferentially accumulates in the peritoneal cavity rather than correcting effective circulating volume, exacerbating ascites.
    - Stimulate hypothalamus and increase thirst.
    - Constrict glomerular vessels (Efferent arteriole> afferent)
    - Mesangial cell contraction
    - Directly increase absorption of Na+ in PCT

- Ascites in cirrhosis results from the interaction of elevated hydrostatic pressure, reduced plasma oncotic pressure, and renal sodium/water retention via RAAS and ADH, all acting together to shift fluid into the peritoneal space.

(1.4) List two other signs that could arise in this patient:

- Spider angiomas (spider nevi) – due to altered estrogen metabolism by the diseased liver.
- Palmar erythema – also linked to hyperestrogenism.
- (Other possible signs: easy bruising, hepatosplenomegaly, flapping tremors, hepatic encephalopathy).

(2)

(2.1) Physiology of Neonatal Respiratory Distress Syndrome (RDS)

- Cause: Deficiency of pulmonary surfactant in premature infants.
- Surfactant Function: Reduces alveolar surface tension, preventing alveolar collapse during expiration (per Laplace's law).
- Pathophysiology:
  - Inadequate surfactant → high alveolar surface tension → alveolar collapse (atelectasis).
  - Decreased lung compliance → increased work of breathing.
  - Ventilation-perfusion mismatch → hypoxemia and CO_2 retention.

(2.2) Effects of High-altitude respiration and chronic mountain sickness.

- Immediate Effects of High Altitude:
  - ↓ Atmospheric PO_2 → ↓ alveolar PO_2 → hypoxemia.
  - ↑ Ventilation (hypoxic ventilatory response) → respiratory alkalosis.
- Acclimatization:
  - Occurs As a compensatory response to hypoxia
  - Persistent Hyperventilation and
  - ↑ 2,3-BPG in RBCs → increase P50 and shifts Hb-O2 dissociation curve to right → facilitates O_2 unloading to tissues.
- Delayed changes
  - ↑ Erythropoietin → polycythemia → ↑ O_2-carrying capacity.
  - Active transport of H+ in to CSF/ lactic acidosis in brain → ↑ ventilatory response to hypoxia.
  - Tissue changes - ↑ mitochondria, ↑ Cytochrome oxidase, ↑ Myoglobin
- Chronic Mountain Sickness:
  - Excessive polycythemia → hyperviscosity → fatigue, headaches, hypoxemia.
  - Peripheral chemoreceptors become insensitive to hypoxia.
  - Pulmonary hypertension due to hypoxic pulmonary vasoconstriction.
  - Right heart strain and potential cor pulmonale.

(2.3)

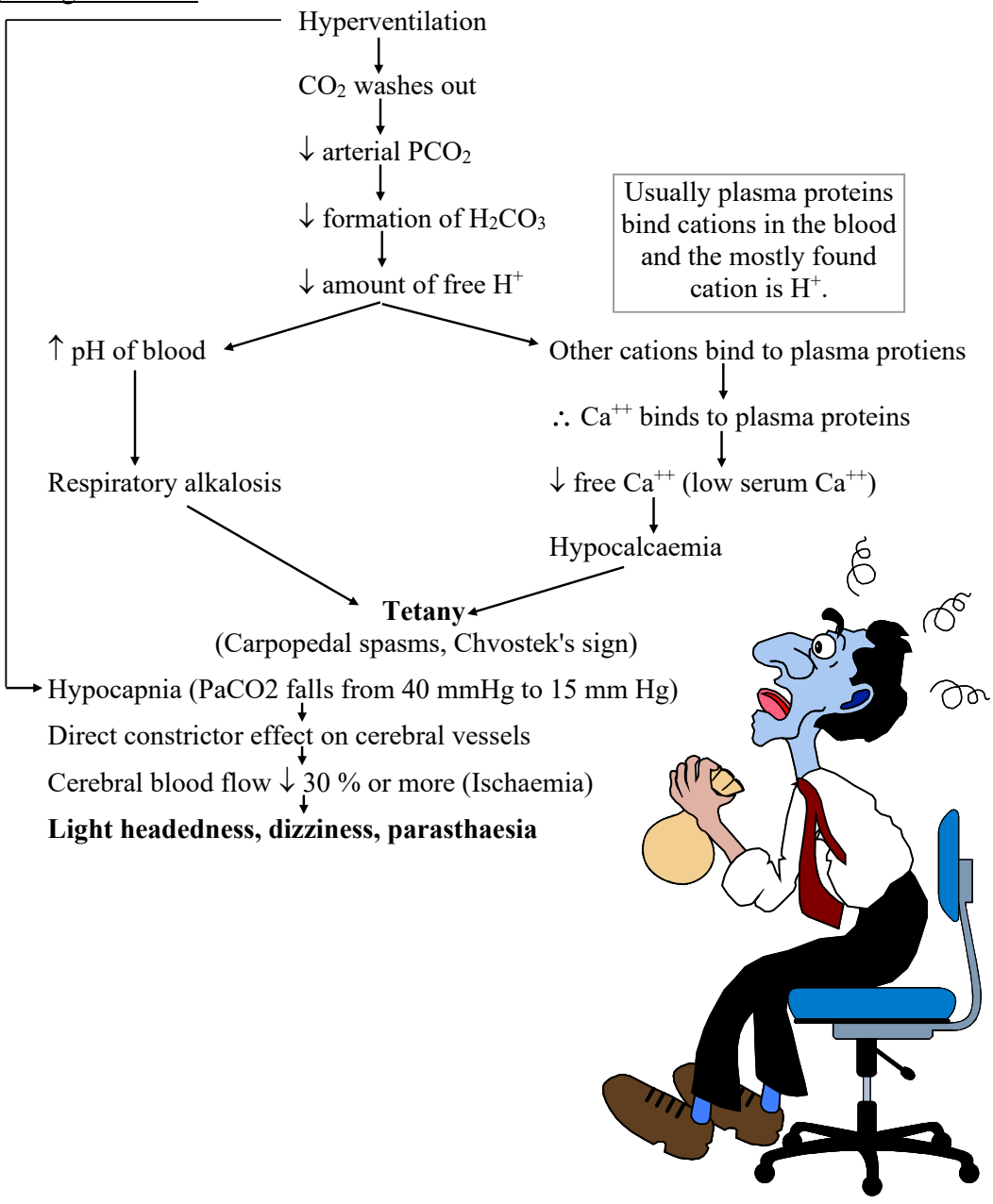
Characteristic posture of the hands (**Trousseau's sign**)

If not appearing, augment using a riva roci cuff, keeping it inflated to above the arterial pressure for some time.

Chvostek's sign - a twitching of facial muscles when lightly tapped with a patellar hammer at the site of exit of the facial nerve below and in front of the ear.

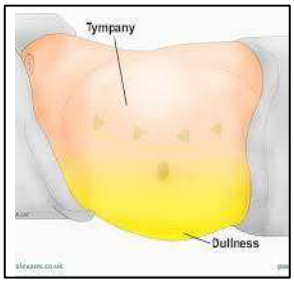
Ask about light-headedness, dizziness and parasthaesia.

Physiological Basis :



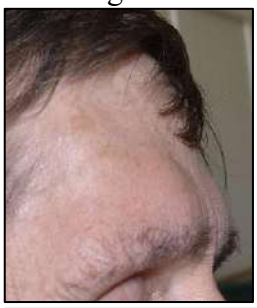
CAT-2 (BATCH 2) MOCK OSPE

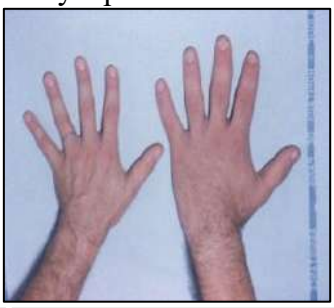
(1) A B C

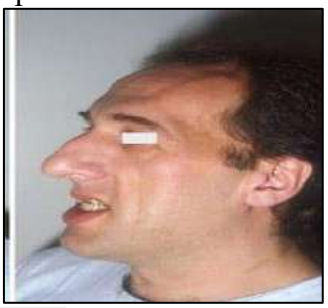


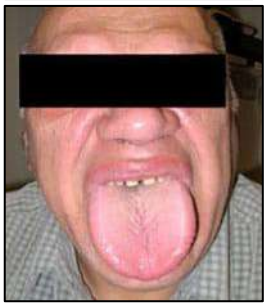
(1.1) Identify the signs A, B, C.
(1.2) Give two reasons for B.
(1.3) Give one reason for C

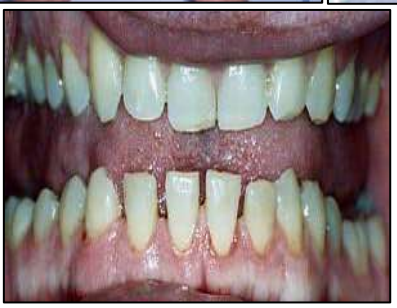
(2) These signs are shown by a patient who has a specific hormone disorder.











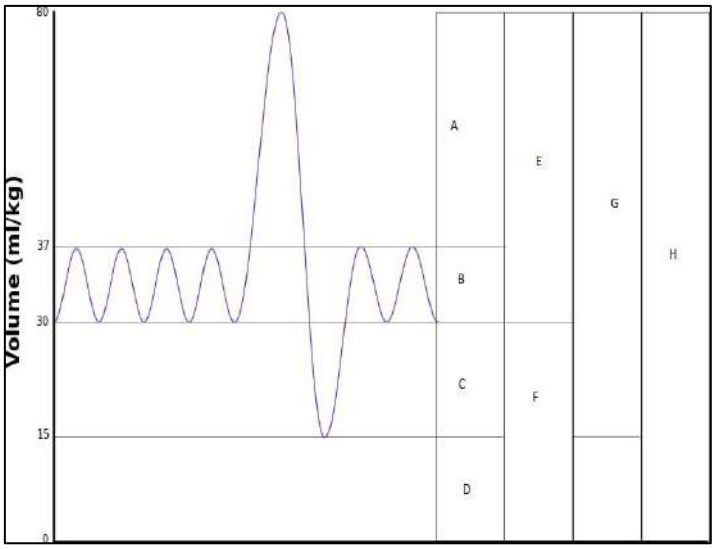
(2.1) Identify the disease condition.
(2.2) What is the hormone affected
(2.3) Identify 3 signs shown here.

(3)

Parameter	Values	
	Laboratory	Normal
Arterial blood gas		
pH	7.26	7.35-7.45
$Paco_2$, mm Hg	29	35-45
Pao_2, mm Hg	128	90-100
Bicarbonate, mEq/L	16	22-26

(3.1) Identify the acid base disorder related to the above laboratory findings.
(3.2) Give a condition which leads to above acid base disorder.

(4)



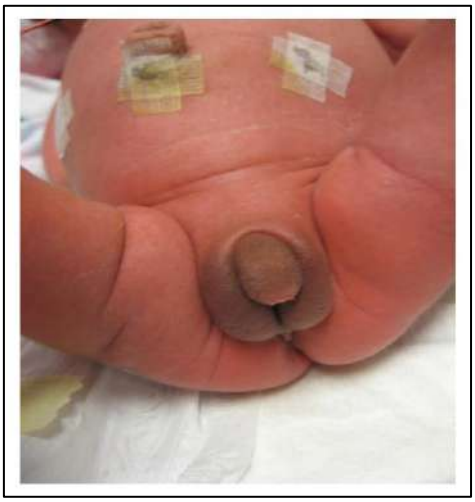
(4.1) Identify A, B, C, D

(4.2) Fill the blanks with letters

IC = …… + ……

VC = …… + ...

(5) A Female baby was born with male external genitalia. Her blood pressure is very low.

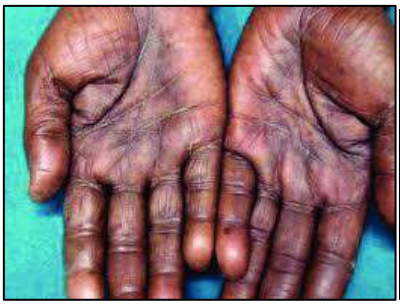


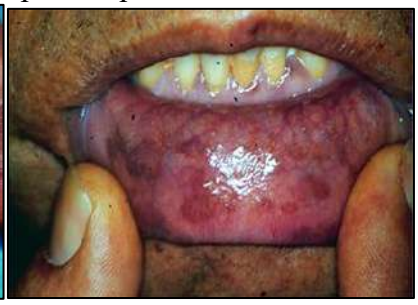
(5.1) Identify the condition.

(5.2) Name an enzyme which is deficient in this baby.

(5.3) Name two hormones which are deficient in this baby.

(6) A Patient comes to a clinic with these signs. On examination his blood pressure is very low and also he has an elevated plasma potassium level.



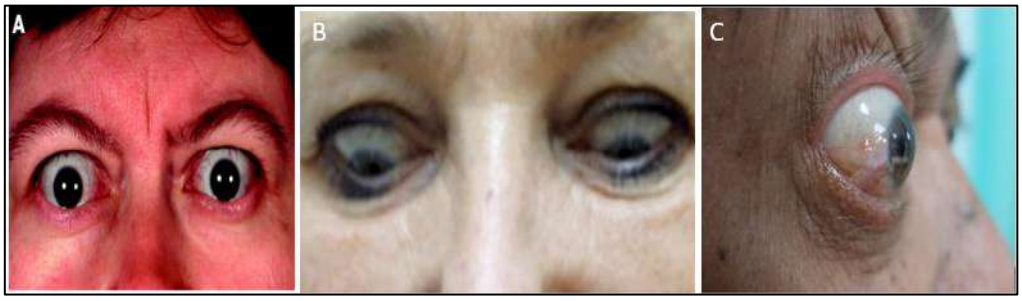


(6.1) Identify the disease condition.
(6.2) Comment on his blood pH.
(6.3) Which hormone is responsible for pigmentation on his buccal cavity and palmar crease?

(7)

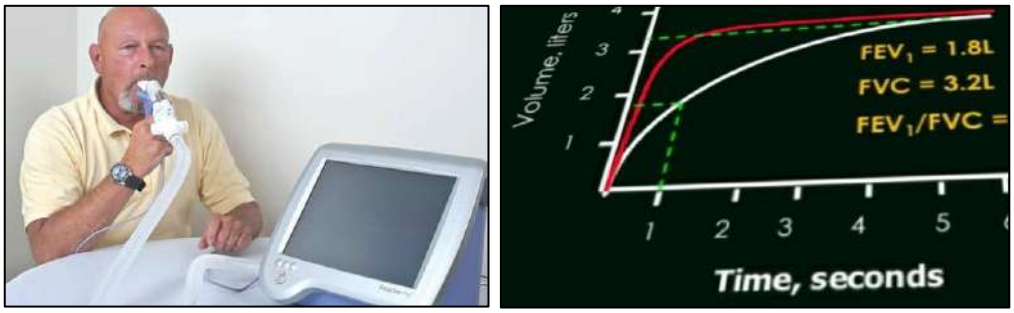
(7.1) Identify the instrument.
(7.2) What is measured by it?
(7.3) Name a condition which leads to a value more than the normal healthy value of this measurement.
(7.4) Name a condition which leads to a value less than the normal healthy value of this measurement.

(8)



(8.1) Identify the signs A, B, C
(8.2) Give one hormone which is reduced than its normal value in this patient.
(8.3) What is the reason for A and B?

(9)



(9.1) Identify the instrument

(9.2) Calculate the FEV1/FVC ratio
(9.3) Give one example for a disease which he suffers from

(10) You have to identify this instrument and advise the patient on how to use it (Live station)

CAT-2 (BATCH 2) MOCK OSPE ANSWERS

(1)
(1.1) A – Shifting dullness
B – Clubbing
C – Koilonychia
(1.2) Lung cancer, lung abscess, empyema, TB with secondary infection, infective endocarditis (any 2)
(1.3) Iron deficiency

(2)
(2.1) Acromegaly
(2.2) Growth hormone
(2.3) Prognathism
Macroglossia
Enlarged extremities
Supraorbital bulging (any 3)

(3)
(3.1) Partially compensated metabolic acidosis (pH and $[HCO_3^-]$ is low to compensate it CO_2 is washed out however pH does not return to normal so it is partially compensated)
(3.2) Lactic acidosis
Diabetic keto acidosis
Severe diarrhea
Chronic kidney disease/failure (any1)

(4)
(4.1) A - Inspiratory Reserve Volume
B - Tidal volume
C - Expiratory Reserve Volume
D - Residual volume
(4.2) IC = A + B
VC = C + E

(5)
(5.1) Congenital Adrenal Hyperplasia (deficiency of below enzymes leads to accumulation of pregnenolone and that produces more androgens which leads to masculinization ultimately resulting into ambiguous genitalia)
(5.2) B Hydroxylase
11 B Hydroxylase
(5.3) Cortisol
Aldosterone

(6)
(6.1) Addison's disease (pigmentation is a special feature for Addison's disease)
(6.2) pH is low (because of low aldosterone, H+ secretion is reduced)
(6.3) ACTH (low aldosterone and cortisol reduces the feedback inhibition of ACTH therefore more ACTH is released.

ACTH also have some action on MSH which secretes more melatonin causing pigmentation)

(7)

(7.1) Urinometer

(7.2) Specific gravity of urine

(7.3) It is increased when there are more solutes in urine

- glycosuria (Diabetes Mellitus)
- proteinuria (Nephrotic syndrome)
- SIADH

(7.4) It is reduced when there is more water in urine.

- Diabetes Insipidus (low ADH)
- Chronic kidney failure

(8)

(8.1) A – Lid Retraction
B – Lid Lag
C – Proptosis

(8.2) TSH (in this patient, elevated T3 and T4 will feed-back inhibit TSH)

(8.3) Elevated T3 has permissive action on catecholamines.
Catecholamines are responsible for muscle spasms.
So, levator palpabrae superioris will contract

(9)

(9.1) Spirometer

(9.2) 0.56

(9.3) Normal value is 0.7-0.8. Here it is reduced. So, this should be an obstructive disease

- Bronchial asthma
- Emphysema
- Chronic bronchitis (any 1)

(10) Before proceeding to the live station, you should know this order:

- Say good morning!
- Introduce yourself.
- Take concern of patient.
- Advice the patient how to use it.
- Take three readings.
- Say thank you!

• **How to Use the Peak Flow Meter**

1. Set the cursor to zero. NB Do not touch the cursor when breathing out.
2. Stand up and hold the peak flow meter horizontally in front of the mouth.
3. Take a deep breath in and close the lips firmly around the mouthpiece, making sure there is no air leak around the lips.
4. Breathe out as hard and as fast as possible.
5. Note the number indicated by the cursor.
6. Return cursor to zero and repeat this sequence twice more, thus obtaining three readings.

- *The highest or best reading of all three measurements is the peak flow at that time. The highest reading should be recorded in the patient's daily asthma diary or recorded on a peak flow chart. To ensure results of the peak flow meter are comparable, the patient is advised to use the meter in the same way each time and at the same time each day.*

PHYSIOLOGY CONTINUOUS ASSESSMENT-2

2022/2023 (1st Batch) CAT-2 Papers & Answers

CAT-2 (BATCH 1) MCQ PAPER

**Question numbers 01 to 10 are True or False type questions. Mark your answer for each
of the 5 responses (A to E)**

(1) True or false regarding the defense mechanisms of the respiratory system?
(A) Defective ciliary motility causes recurrent lung infections.
(B) Mucociliary clearance is enhanced by surfactant.
(C) Activation of pulmonary alveolar macrophages occur in chronic smoking.
(D) Mucosal immunity is provided by IgA.
(E) Filtration ability of the upper airways does not allow any particulates to enter into the lower airways.

(2) True or False regarding the breathing mechanisms?
(A) Inward movement of the chest wall occurs due to the destruction of elastic tissues in the lungs in emphysema.
(B) Forced expiration is an active process.
(C) Inspiration is a passive process.
(D) Loss of negative intrapleural pressure leads to lung collapse.
(E) Diaphragmatic breathing is more prominent in females than in males.

(3) True or false regarding lung volumes and capacities?
(A) Functional residual capacity is increased in patients with chronic obstructive pulmonary disease.
(B) Inspiratory capacity is the volume of air that is maximally inspired after maximal expiratory effort.
(C) Residual volume of a healthy adult is approximately 1.2 L.
(D) Total lung capacity can be measured using a spirometer.
(E) Vital capacity is reduced when the external intercostal muscles are paralyzed.

(4) Which of the following shift/s the oxygen-haemoglobin dissociation curve to the left?
(A) Decreased 2,3 DPG.
(B) Fever.
(C) Fetal haemoglobin.
(D) Hypercapnia.
(E) Metabolic acidosis.

(5) True or false regarding pancreatic secretion?
(A) It contains both trypsinogen and trypsin inhibitors.
(B) Pancreatic enzymes act on carbohydrates and proteins.
(C) Lack of pancreatic juice in the duodenum leads to reduction in fat content in feces.
(D) It becomes thick due to a genetic defect in Cl^- channel in duct epithelium in cystic fibrosis.
(E) Secretion of Pancreatic juice rich in bicarbonate is stimulated by cholecystokinin.

(6) True or false regarding digestion and absorption of nutrients?
(A) Calcium absorption takes place mainly in the jejunum.
(B) Some tripeptides are hydrolyzed by intracellular peptidases in small intestine.
(C) Maltase is located in small intestinal brush border.
(D) Both glucose and galactose use Na^+ dependent glucose transporter to enter the enterocytes.
(E) The basolateral transport of iron, out of the enterocytes is facilitated by transferrin.

(7) True or false regarding thyroid hormones?
(A) Most of the circulating thyroxine is bound to albumin.
(B) Most of the Triiodothyronine (T3) is produced within the thyroid gland.
(C) Triiodothyronine (T3) is the primary mediator of the physiologic effects.
(D) Thyroid hormones act by binding to receptors on the cell membrane.
(E) Thyroxine (T4) is the main end product of the thyroid gland.

(8) True or false regarding Growth Hormone (GH)?
(A) It exerts its effects mostly through somatomedins.
(B) Its excess in childhood results in acromegaly.
(C) Its excess causes diabetes mellitus.
(D) Its secretion is stimulated by high blood glucose levels.
(E) Its secretion is stimulated by somatostatin.

(9) True or false regarding glucose transporters (GLUT)?
(A) GLUT 2 is the main glucose transporter in the muscle cells.
(B) GLUT 4 is mainly located in the pancreatic beta cells.
(C) GLUT 4 transporters in the liver are stimulated by insulin.
(D) GLUTs are translocated rather than synthesized by insulin.
(E) GLUTs enhance the facilitated diffusion of glucose across cell membranes.

(10) Which of the following hormones is/are correctly matched with its type?
(A) IGF-1 (Insulin-like Growth Factor-1) – Autocrine Hormones
(B) Cortisol – Endocrine Hormone
(C) Oxytocin – Neuroendocrine Hormones
(D) Somatostatin – Paracrine Hormone
(E) Thyroxine (T4) – Paracrine Hormones

Question number 11 to 20 are Single Best Response (SBR) type. Select the best response to each question.

(11) A premature baby with low birth weight, born at 32 weeks of gestation, developed signs of respiratory distress shortly after birth, including tachypnea, nasal flaring, and intercostal retractions. Chest X-rays revealed diffuse ground-glass opacities and air bronchograms.
Which of the following best explains the pathophysiology underlying this baby's respiratory condition?
(A) Increased alveolar surface tension due to deficiency of pulmonary surfactant.
(B) Increased alveolar pressure due to defective reabsorption of fluid.
(C) Obstruction of the upper airways leading to decreased airflow.
(D) Persistent pulmonary hypertension causing right-to-left shunting.

(E) Underdeveloped alveolar architecture leading to decreased lung compliance.

(12) A 20-year-old male presented with acute attack of bronchial asthma. During his hospital stay he was treated with bronchodilators and corticosteroids.
Which of the following lung function findings would be the most useful in assessing the improvement of his clinical condition?
(A) Flow-volume loop
(B) FEV1/FVC ratio
(C) FEV1
(D) FVC
(E) PEFR

(13) A 55-year-old male with a history of diabetes and heavy smoking presented to the Accident and Emergency (A&E) department with sudden-onset excruciating pain in his left lower limb lasting for one hour. On examination, the affected limb was cold and clammy, with absent dorsalis pedis and posterior tibial pulses. The SpO2 of the affected limb was 60%. Clinically, arterial thromboembolism was diagnosed, and confirmatory investigations were planned.
What is the most likely explanation for the reduced SpO_2 in the affected limb?
(A) Anaemic hypoxia
(B) Histotoxic hypoxia
(C) Hypoxic hypoxia
(D) Mechanical hypoxia
(E) Stagnant hypoxia

(14) A 55-year-old male with a history of chronic alcohol use presented with persistent upper abdominal pain, significant weight loss, and frequent, oily stools. Laboratory tests revealed elevated blood glucose level.
Which of the following is the most likely underlying cause of this patient's condition?
(A) Bile acid deficiency.
(B) Malabsorption syndrome.
(C) Pancreatic insufficiency due to chronic inflammation.
(D) Premature activation of pancreatic enzymes.
(E) Small intestinal bacterial overgrowth.

(15) A 30-year-old male presented with progressive jaundice, dark urine, and generalized fatigue for two weeks. He also reported nausea, loss of appetite, and mild right upper quadrant discomfort. There was no history of alcohol use or recent travel. On examination, he had icteric sclera, hepatomegaly, and mild tenderness over the liver. Laboratory investigations showed: Total bilirubin - 8.5 mg/dL with direct fraction - 6.2 mg/dL (Total Bilirubin 0.2 - 1.2 mg/dL), AST - 820 U/L (10-40U/L), ALT – 1000 U/L (7-56U/L), ALP - 120 U/L (44-147U/L), Albumin - 3.5 g/dL (3.5-5.0g/L), Prothrombin time (PT) - Mildly elevated.
Which of the following best explains the pathophysiology of this condition?
(A) Biliary obstruction leading to reduced bilirubin excretion into the intestine.
(B) Excessive hemolysis leading to unconjugated hyperbilirubinemia.

(C) Increased hepatic synthesis of bilirubin due to enzyme overactivity.
(D) Increased enterohepatic circulation of bilirubin.
(E) Impaired handling of bilirubin due to hepatocellular damage.

(16) A 40-year-old male underwent gastric bypass surgery two weeks ago for morbid obesity. Shortly after resuming oral intake, he developed episodes of dizziness, sweating, palpitations, and abdominal discomfort within 30 minutes of eating. These symptoms were particularly noticed after consuming meals high in simple sugars. On examination, his vital signs were stable, but he appeared anxious during the episodes.
What is the most likely underlying mechanism of his symptoms?
(A) Chronic malabsorption leading to nutrient deficiency.
(B) Delayed gastric emptying leading to food stagnation.
(C) Insulin resistance leading to postprandial hyperglycemia.
(D) Increased gastric acid secretion leading to acid base disturbance.
(E) Rapid gastric emptying causing fluid shift into the small bowel.

(17) A 30-year-old lady underwent a surgery for primary hyperparathyroidism. On the first postoperative day, she developed muscle cramps, tingling sensations in her hands and feet, and intermittent spasms. On examination, Chvostek's sign was positive, and Trousseau's sign was elicited. Her vital signs were stable, and no other abnormalities were noted.
What is the most likely physiological explanation for her symptoms?
(A) Hypermagnesemia.
(B) Hypocalcemia.
(C) Hypokalemia
(D) Hypophosphatemia.
(E) Vitamin D deficiency.

(18) A 30-year-old lady is being evaluated for unintentional weight loss of 5 kg over 3 months, palpitations, sweating and hair loss. Examination revealed a cachectic lady, with sweaty palms, red eyes with proptosis (bulging eyes). Her thyroid gland is diffusely enlarged. TSH - 0.005 mIU/L (0.4-4.0), Free T4 - 50 pmol/L (10 -18).
What is the most likely diagnosis?
(A) Graves' disease.
(B) TSH secreting pituitary tumor.
(C) Toxic multi nodular goiter.
(D) Toxic adenoma.
(E) Thyroiditis.

(19) A 50-year-old male with type 2 diabetes was brought unconscious to the hospital following overdose of his diabetic medications. His capillary blood glucose level showed low blood glucose value of 40 mg/dL. Which of the following counter regulatory hormones has the most potent action in reversing hypoglycemia in this patient?
(A) Cortisol
(B) Epinephrine
(C) Glucagon
(D) Growth hormone

(E) Norepinephrine

(20) A 40-year-old female presented with progressive weight gain, central obesity, moon face, and proximal muscle weakness. She also reported easy bruising, thinning of the skin, and menstrual irregularities. On examination, she had facial plethora, dorsocervical fat pad ("buffalo hump"), abdominal striae, and hypertension. Her fasting blood glucose was elevated.
What is the best initial test for the diagnosis of this lady?

(A) High-dose dexamethasone suppression test (HDDST)
(B) Low-dose dexamethasone suppression test (LDDST)
(C) Overnight dexamethasone suppression test. (ODST)
(D) Serum ACTH levels
(E) 24-hour urinary free cortisol (UFC)

CAT-2 (BATCH 1) MCQ PAPER ANSWERS

(1) TTTTF

(2) FTFTF

(3) TFTFT

(4) TFTFF

(5) TTFTF

(6) TTTTF

(7) FFTFT

(8) TFTFF

(9) FFFTT

(10) TTTTF

(11) A

(12) E

(13) E

(14) C

(15) E

(16) E

(17) B

(18) A

(19) C

(20) C

CAT-2 (BATCH 1) STRUCTURED ESSAY (SEQ) PAPER

(1) A 50-year-old woman presented with increased sleepiness, lethargy, cold intolerance, and constipation. On examination, she was obese, had a hoarse voice and a pulse rate of 55 beats per minute. Investigations revealed the following: WBC - 6,000/µL (Reference range: 4,000–11,000/µL), Hemoglobin (Hb) - 11 g/dL (Reference range: 11–14 g/dL), Platelets - 230,000/µL, TSH - 35 mIU/L (Reference range: 0.4–4.0 mIU/L), Free T4 - 8pmol/L (Reference range: 10–18) pmol/L).

(1.1) What is the most likely diagnosis? (10 marks)

(1.2) List three additional clinical signs you would like to assess during the clinical examination (15 marks).

(1.3) Explain how the thyroid gland is regulated via the hypothalamic-pituitary-thyroid axis in a normal person. (50 marks)

(1.4) Describe the physiological basis of the elevated TSH levels in this patient (25 marks)

(2) Explain the physiological basis of the following:

(2.1) The increase in respiratory rate during moderate physical exercise. (40 marks)

(2.2) Reduced peak expiratory flow rate (PEFR) in an acute attack of bronchial asthma. (35 marks)

(2.3) The administration of salbutamol in patients with bronchial asthma. (25 marks)

CAT-2 (BATCH 1) SEQ PAPER ANSWERS

(1)

(1.1) What is the most likely diagnosis? (10 marks)

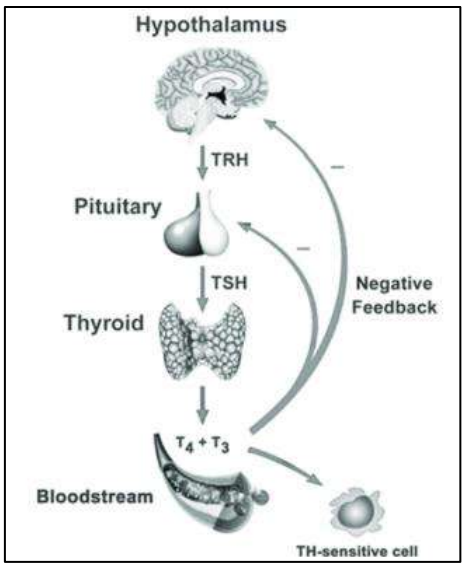
Primary Hypothyroidism (10 marks)

(1.2) List three additional clinical signs you would like to assess during the clinical examination (15 marks).

- Dry skin
- Slow relaxing ankle jerk/ reflexes
- Goiter
- Hair thinning or hair loss including the outer third of the eye brows.
- Brille nails
- Puffy face and swelling

(Any of three signs each answer carries 5 marks)

(1.3) Explain how the thyroid gland is regulated via the hypothalamic-pituitary-thyroid axis in a normal person. (50 marks)



1.The hypothalamus secretes thyrotropin-releasing hormone (TRH) in response to low thyroid hormone levels or increased metabolic demand.

2.TRH travels through the hypothalamic-pituitary portal system to the anterior pituitary gland.

3.TRH stimulates the thyrotropic cells in the anterior pituitary gland to release thyroid-stimulating hormone (TSH) into the bloodstream in a pulsatile manner.

4.TSH acts on thyroid follicular cells via its receptors, which are typical G-protein-coupled transmembrane receptors that activate adenylyl cyclase.

5.TSH stimulates the synthesis and secretion of thyroxine (T_4) and triiodothyronine (T_3) in the thyroid gland.

6.One-third of circulating T_4 is converted to T_3, the biologically active form, in peripheral tissues through the action of peripheral deiodinases

7.When circulating T_3 and T_4 levels rise, they inhibit TRH secretion from the hypothalamus and reduce TSH release from the pituitary gland.

8.This negative feedback mechanism prevents excessive thyroid hormone production, maintaining homeostasis.

9.When circulating T_3 and T_4 levels fall, they stimulate TRH secretion from the hypothalamus and increase TSH release from the pituitary gland.
10.Thus, thyroid gland function is regulated via the hypothalamic-pituitary-thyroid (HPT) axis.
(10 marks for the diagram, 4 marks for each point: 4 × 10 = 40 marks.)

(1.4) Describe the physiological basis of the elevated TSH levels in this patient (25 marks)

1.In primary hypothyroidism, the thyroid gland fails to produce sufficient thyroid hormones—thyroxine (T_4) and triiodothyronine (T3).
2. When T_4 and T_3 levels drop in, the feedback inhibition on hypothalamus and pituitary is reduced.
3. The hypothalamus responds to low T_4 and T_3 by increasing the release of thyrotropin-releasing hormone (TRH), which stimulates the anterior pituitary to
release more TSH.
4. In response to the increased TRH stimulation and the lack of sufficient T_4 and T_3, thyrotrophic cells in the anterior pituitary increase TSH synthesis and secretion in an attempt to stimulate the failing thyroid gland to produce more hormones.
5. Since the thyroid gland is unable to respond adequately due to primary dysfunction, the elevated TSH fails to normalize T_4 and T_3 levels, leading to persistently high TSH concentrations in the blood.
(5 marks for each point 5 × 5 = 25 marks)

(2)

(2.1) The increase in respiratory rate during moderate physical exercise. (40 marks)

1. During moderate exercise, the respiratory rate increases to enhance oxygen delivery and carbon dioxide removal, thereby meeting the body's higher metabolic demands. This increase is regulated by both neural (motor cortex) and chemical (chemoreceptor) mechanisms.
2. The immediate response primarily occurs via neural control, while the sustained increase in respiratory rate is maintained through chemical changes.
3.Ventilation increase abruptly with the onset of exercise presumably due to psychic stimuli and afferent impulses from proprioceptors in muscles, tendons and joints.
4.The motor cortex sends simultaneous signals to both the respiratory centers in the medulla and the muscles involved in movement, leading to an anticipatory increase in respiratory rate.
5. Proprioceptors in muscles, tendons, and joints detect movement and send signals via mechanoreceptors to the brainstem, further stimulating the respiratory centers.
6.With moderate exercise the increase is due mostly to an increase in the depth of respiration. This is accompanied by an increase in the respiratory rate when the exercise is more strenuous.
7. During exercise, increased metabolic activity leads to higher production of carbon dioxide (CO_2), which lowers blood pH. Chemoreceptors detect these changes and stimulate ventilation.

8. Although arterial oxygen saturation remains relatively stable in moderate exercise, a slight decrease in tissue oxygen levels (due to increased consumption) can stimulate peripheral chemoreceptors.
9. The production of lactic acid during exercise lowers blood pH, further activating chemoreceptors to enhance ventilation.
10. Increased body temperature during exercise directly stimulates the respiratory centers, contributing to an increased respiratory rate.
11. Exercise increase the plasma K+ level and this increase may stimulate the peripheral chemoreceptors.
12. 12. After exercise, ventilation remains elevated for some time to repay the oxygen debt, clear accumulated CO_2, and restore acid-base balance.

(4 marks for the diagram, 3 marks for each point: 3 × 12 = 36 marks.)

(2.2) Reduced peak expiratory flow rate (PEFR) in an acute attack of bronchial asthma. (35 marks)

1. During an asthma attack, there is airway narrowing (bronchoconstriction), inflammation, and increased mucus production.
2.These factors greatly increase resistance to airflow.
3.PEFR (Peak Expiratory Flow Rate) reflects how quickly air can be expelled from the lungs in one second of forceful exhalation.
4. When the airways are narrowed and obstructed, the speed of air exiting the lungs is significantly reduced, resulting in a lower PEFR.
5. Therefore, a reduced PEFR is commonly observed during an acute attack of bronchial asthma.

(7 marks for each point 5 × 7 = 35 marks.)

(2.3) The administration of salbutamol in patients with bronchial asthma. (25 marks)

1. Salbutamol is a selective β2-adrenergic receptor agonist.
2. It binds to β2-adrenergic receptors on bronchial smooth muscle cells, causing relaxation (bronchodilation) via stimulatory G-protein and increased cAMP.
3. In an acute asthma attack, excessive bronchoconstriction—along with inflammation and mucus production—narrows the airways, making breathing difficult.
4. By relaxing bronchial smooth muscle, salbutamol significantly reduces airflow resistance, allowing easier breathing.
5. Therefore, salbutamol is used as a rescue therapy in patients with bronchial asthma.

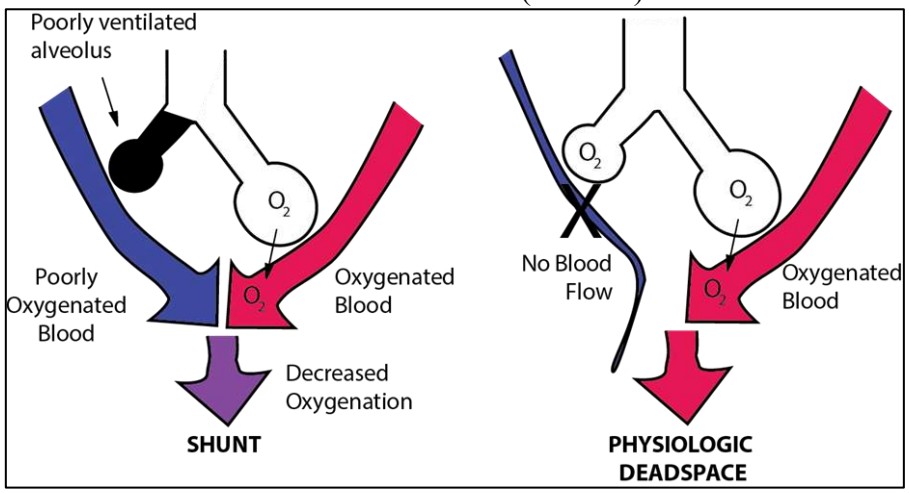
(5 marks for each point 5 × 5 = 25 marks)

CAT-2 (BATCH 1) OSPE QUESTIONS

(1)

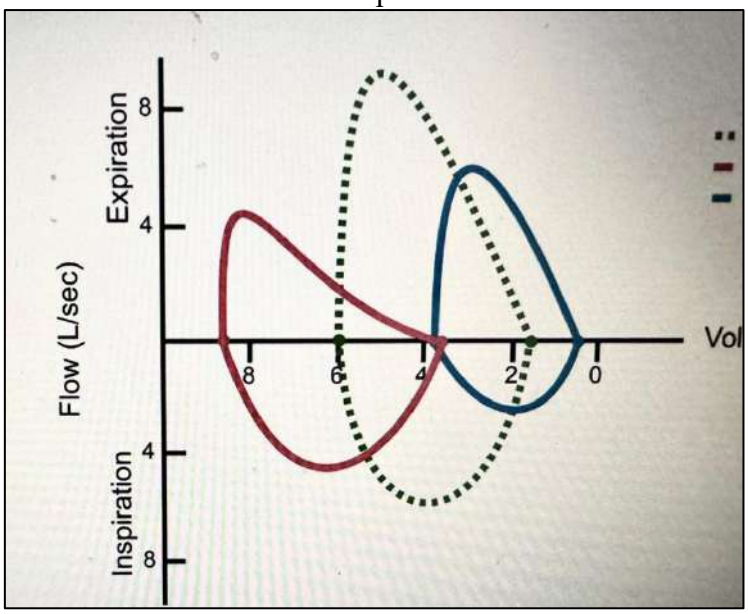
(1.1) Name A and B. (6 marks)

(1.2) Give one clinical condition for A and B. (4 marks)



A B

(2) These are flow volume curves of three patients.

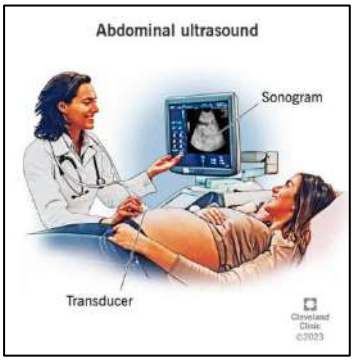


(2.1) Name A, B and C curves. (6 marks)

(2.2) Give one clinical condition for A and C curves. (4 marks)

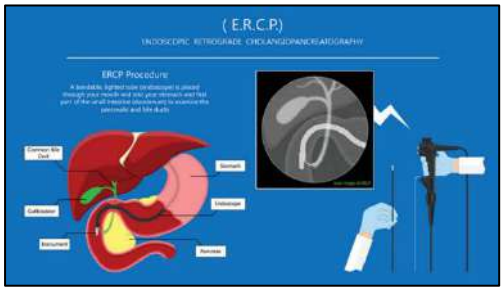
(3) Select the most appropriate gastrointestinal investigation for each clinical indication.

A

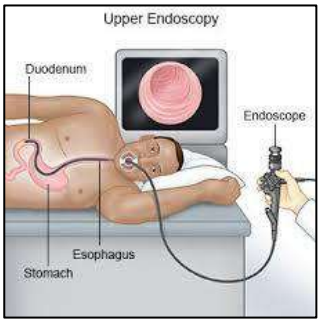


B

C



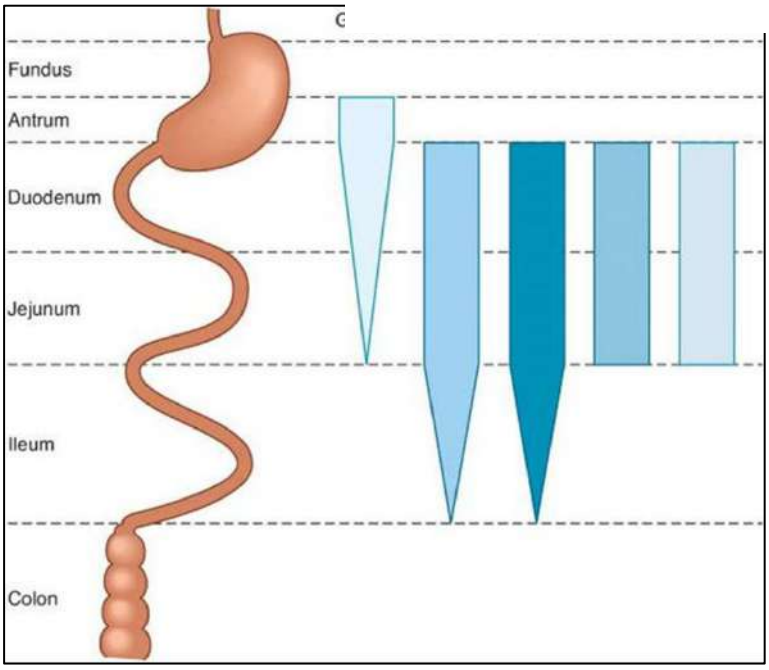
D



(3.1) For bile or pancreatic duct obstruction (2.5 marks)
(3.2) For esophageal, gastric, and duodenal evaluation. (2.5 marks)
(3.3) For small bowel abnormalities. (2.5 marks)
(3.4) For gallstones, liver pathology, or ascites (2.5 marks)

(4) This picture illustrates five gastrointestinal hormones along the length of the GI tract.
Name A to E (10 marks)

A B C D E



(5) A 45-year–old male with type 1 diabetes mellitus was admitted with severe fatigue and weakness and shortness of breath for 3 days.

ABG- PH 7.25(7.35-7.45)	serum Electrolytes
PCo2- 24 mmHg (35-45)	Na+ 135mmol/L (135-145)
PO2 -98 mmHg	K+ 5.2 mmol/L (4.5-5.5
HCO3- 12mmol/L (22-26)	Cl- 95 mmol/L
Blood glucose 450mg/dl	

(5.1) What is the acid base abnormality of this patient? (2 marks)
(5.2) Calculate the anion gap (4 marks)
(5.3) Give two possible conditions for these metabolic abnormalities (2 marks)

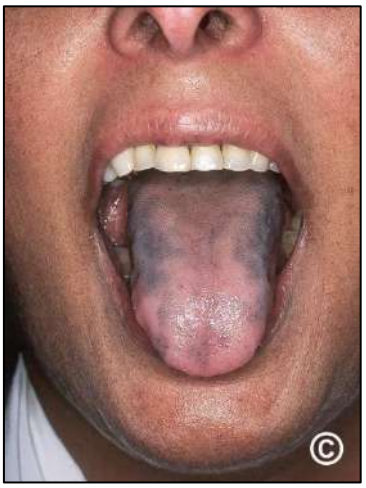
(6)

(6.1) Name two physical signs you observe in this patient. (4 marks)
(6.2) What is the most likely diagnosis? (6 marks)

(7) 74-year-old male was admitted with a history of episodic fainting attacks. This ECG was taken at ETU.

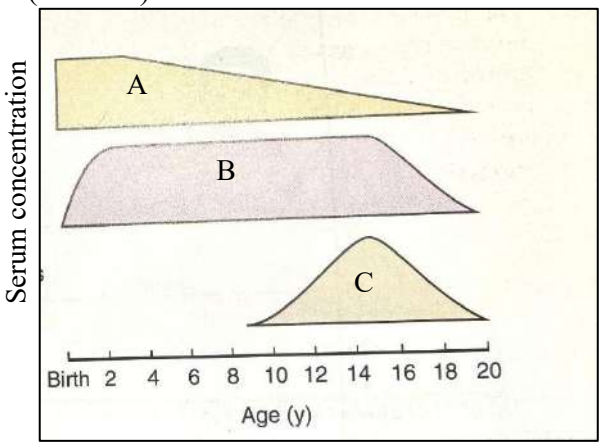
(7.1) What is the ECG diagnosis? (4 marks)
(7.2) What is the pulse rate? (3 marks)
(7.3) What would you expect to observe in the patient's JVP (Jugular venous Pressure). (3 marks)

(8) This patient has been experiencing progressive fatigue, weight loss and generalized weakness for the past three months. On examination, he has low blood pressure (90/60mmHg).



(8.1) What is the physical sign shown? (4 marks)
(8.2) Name two biochemical abnormalities seen in this patient? (4 marks)
(8.3) What is the confirmatory test for the diagnosis of this patient? (2 marks)

(9) This diagram shows relative importance of three hormones in human growth at various ages.
(9.1) Name hormones A, B and C. (6 marks)
(9.2) Which one/ones, is/are primarily responsible for the growth spurt of puberty? (4 marks)



(10) (Live station)
Demonstrate how to correctly measure Peak Expiratory Flow (PEF) using this device. (PEF meter is provided)

CAT-2 (BATCH 1) OSPE ANSWERS

(1)
(1.1) A – shunt / V/Q (ventilation/perfusion) mismatch – low V/Q (3 marks)
B – physiological dead space / High V/Q (3 marks)
(1.2) A – pneumonia/pulmonary oedema/atelectasis/mucous plugging/pulmonary A-V fistula (any 1 of these – 2 marks)
B – cardiogenic shock/emphysema/pulmonary embolus (any 1 of these – 2 marks)

(2)
(2.1) A – obstructive lung disease pattern (2 marks)
B – normal flow volume loop (2 marks)
C – restrictive lung disease pattern (2 marks)
(2.2) A – COPD, bronchial asthma, bronchiectasis (any 1 of these – 2 marks)
C – pulmonary fibrosis, interstitial lung disease, kyphoscoliosis (any 1 of these – 2 marks)

(3)
(3.1) ERCP (C) (2.5 marks)
(3.2) UGIE (D) (2.5 marks)
(3.3) BMFT (B) (2.5 marks)
(3.4) Abdominal ultrasound (A) (2.5 marks)

(4) A – Gastrin
B – CCK
C – secretin
D – GIP
E – motilin (2 marks for each answer)

(5)
(5.1) Metabolic acidosis/Metabolic acidosis with respiratory compensation (2 marks)
Anion gap = $(Na^+ + K^+) – (Cl^- + HCO_3^-)$ (2 marks)
= (135.0 - 5.2) – (95.0 + 12.0) (1 mark)
= 33.2 (1 mark) (total 4 marks)
(5.2) Diabetic ketoacidosis, lactic acidosis (2 marks)

(6)
(6.1) Exophthalmos/ptosis/prominent bulging of the eyes (2 marks)
Goiter (2 marks)
(6.2) Graves' disease (6 marks)

(7)
(7.1) Complete heart block (4 marks)
(7.2) 43 bpm (3 marks)
(7.3) Canon wave (3 marks)

(8)
(8.1) Hyperpigmentation of the buccal mucosa/tongue (4 marks)

(8.2) Hyponatremia/hyperkalemia/hypoglycemia
(8.3) Deficiency of glucocorticoids/mineralocorticoids (2 × 2 marks)
(8.4) Short synacthen test/short corticosteroid test/short ACTH stimulant test (2 marks)

(9)
(9.1) A – thyroid hormones/thyroxine (2 marks)
B – Growth hormone (2 marks)
C – Androgen and estrogen (sex hormones) (2 marks)
(9.2) B and C (2 × 2 = 4 marks)

2023/2024 (2nd Batch) CAT-2 Papers & Answers

CAT-2 (BATCH 2) MCQ PAPER

Question numbers 01 to 10 are True or False type questions. Mark your answer for each
of the 5 responses (A to E)

(1) True or False regarding the mechanism of breathing?
(A) Compliance of the lungs and chest wall is measured as the slope of the pressure-volume curve of the total respiratory system.
(B) Destruction of elastic tissue in the lungs causes the chest wall to spring outward.
(C) During forced expiration, the intrapleural pressure becomes more negative.
(D) If the chest wall is penetrated and opened to the outside environment, the lungs can collapse.
(E) Transmural pressure (transpulmonary pressure) helps to keep the lungs inflated.

(2) Which of the following conditions is/are correctly matched with the corresponding ventilation-perfusion (V/Q) ratio?
(A) Asthma - Low
(B) Collapsed alveoli - High
(C) Dead space - Low
(D) Pulmonary embolism - High
(E) Shunt – High

(3) True or False regarding oxygen and carbon dioxide transport in human blood?
(A) About 20-30% of carbon dioxide is transported as carbamino-hemoglobin bound to globin chains of Hb.
(B) Oxygen is predominantly carried bound to hemoglobin.
(C) The Bohr effect enhances O_2 unloading in tissues due to increased CO_2 and H^+ concentration.
(D) The majority of carbon dioxide is transported as bicarbonate ions (HCO_3^-) in plasma.
(E) The R state of hemoglobin increases unloading of oxygen at tissue level.

(4) True or False regarding the functions of surfactant?
(A) It increases the surface tension of alveoli.
(B) It helps in immune defense of the lungs.
(C) It helps to prevent pulmonary oedema.
(D) It reduces the uniform inflation of alveoli.
(E) It increases functional residual capacity (FRC).

(5) True or False regarding digestion and absorption?
(A) Absorption of undigested proteins occurs more in adults than in infants.
(B) Iron overload can cause hemosiderin to build up in the body tissues.
(C) Lactose intolerance is caused by a deficiency of a brush border enzyme.
(D) Villus atrophy leading to malabsorption is seen in gluten sensitive enteropathy.

(E) Vitamin B12 absorption requires intrinsic factor produced by the stomach.

(6) True or False regarding the vomiting reflex?

(A) Parasympathetic activity through the vagus nerve causes salivation and reverse peristalsis.
(B) The glottis closes during vomiting to prevent food from entering the airway.
(C) The lower esophageal sphincter (LES) and esophagus relax before gastric contents are ejected.
(D) The vomiting center is located in the medulla oblongata.
(E) Vomiting induced by stimulation of vestibular apparatus is mediated via chemoreceptor trigger zone.

(7) True or False regarding the factors that increase the gastric motility include?

(A) Acetylcholine
(B) Cholecystokinin
(C) Gastrin
(D) GIP
(E) Secretin

(8) True or False regarding thyroid hormone synthesis?

(A) Iodide is actively transported into thyroid follicular cells by the sodium-iodide symporter.
(B) MIT and DIT combine to form T_3 and T_4 within thyroglobulin.
(C) Oxidation of iodide and iodination of tyrosine residues occur inside the thyroid follicular cells.
(D) Thyroglobulin is synthesized by the parafollicular (C) cells of the thyroid gland.
(E) Thyroid peroxidase enzyme is essential for hormone synthesis.

(9) True or False regarding hormonal effects on calcium metabolism?

(A) Estrogen inhibits osteoblast function.
(B) Glucocorticoids increase bone resorption.
(C) Growth hormone decreases intestinal calcium absorption
(D) Hyperthyroidism increases the risk of osteoporosis and fractures.
(E) Insulin enhances calcium and phosphate uptake into bone

(10) True or False regarding hypoglycemia?

(A) Autonomic symptoms are the first symptom to appear in hypoglycemia.
(B) CNS glucose utilization is largely dependent on Insulin.
(C) Fasting hypoglycemia is a characteristic feature of insulinoma.
(D) It stimulates the sympathetic nervous system via hypothalamus.
(E) When blood glucose drops below 30 mg/dL, counter regulatory hormones are activated.

Question no. 11 to 20 are Single Best Answer (SBA) type. Select the best response to each question.

(11) A 45-year-old man presents for evaluation of chronic cough and exertional breathlessness. His spirometry shows a markedly reduced FEV_1 and a reduced FEV_1/FVC ratio.

Which of the following feature is most consistent with the underlying lung condition?

(A) Decreased residual volume (RV).
(B) Increased diffusion capacity for carbon monoxide (DLCO).
(C) Increased elastic recoil of the lungs.
(D) Increased total lung capacity (TLC) due to air trapping.
(E) Reduced Functional Vital Capacity (FVC).

(12) Ventilation is increased with moderate to severe exercise.

The main stimulus for the increase in ventilation in this situation is:

(A) Hypercapnia
(B) Hypoxia
(C) Increased temperature
(D) Lactic acid
(E) Proprioceptors

(13) The pulmonary blood flow is regulated by both passive and active mechanisms. The regional blood flow is regulated in order to maintain optimum V/Q ratio.

Which of the following mechanism is mainly responsible for regulating regional pulmonary blood flow to maintain optimum V/Q ratio?

(A) Acetylcholine induced vasoconstriction.
(B) Autoregulation by myogenic theory.
(C) Hypoxic pulmonary vasoconstriction.
(D) Sympathetic innervation.
(E) Vasodilatation by histamine.

(14) A 45-year-old man with a long history of chronic pancreatitis presents with bulky, pale, foul-smelling stools that float in water. His blood glucose level is slightly elevated.

Which of the following mechanism best explains the cause of his steatorrhoea?

(A) Damage to intestinal villi reducing absorptive surface area.
(B) Decreased pancreatic enzyme secretion causing defective fat digestion.
(C) Rapid intestinal transit time limiting nutrient absorption.
(D) Reduced bicarbonate secretion into intestine.
(E) Reduced secretion of bile salts leading to impaired micelle formation.

(15) Hypersecretion of gastric acid can be controlled by different treatment methods.

What is the most effective treatment to reduce acid secretion?

(A) Antacids
(B) H2 receptor blockers
(C) Partial gastrectomy
(D) Prostaglandin analogs
(E) Proton pump blockers

(16) A 60-year-old woman presents with infrequent bowel movements, hard stools, and straining for the past 3 months. She has a history of

hypothyroidism and is on long-term calcium supplements. Physical examination and colonoscopy are normal.
Which of the following is the most likely physiological explanation for her constipation?
(A) Decreased colonic motility due to excess sympathetic stimulation.
(B) Decreased colonic smooth muscle tone due to hypothyroidism.
(C) Decreased intestinal secretion of water and electrolytes.
(D) Decreased sensitivity of rectal stretch receptors.
(E) Hypercalcemia.

(17) A 45-year-old man presents with progressive difficulty in swallowing both solids and liquids, regurgitation of undigested food, and nocturnal cough. There is no weight loss or vomiting of blood. Achalasia of the cardia was suspected.
Which of the following investigations is the best test to diagnose this condition?
(A) A. Barium swallow
(B) Chest X-ray
(C) CT scan of the thorax
(D) Esophageal manometry
(E) Upper gastrointestinal endoscopy

(18) A 32-year-old woman presents with central obesity, thin limbs, muscle weakness, and purple striae on her abdomen. Her blood pressure is elevated, and laboratory investigations reveal hyperglycemia and increased plasma cortisol levels.
Which of the following physiological effect of cortisol best explains her hyperglycemia?
(A) Decreased lipolysis in adipose tissue.
(B) Increased glucose uptake by skeletal muscle.
(C) Inhibition of glycogen storage.
(D) Promotion of gluconeogenesis.
(E) Stimulation of glycogen breakdown in the liver

(19) A 40-year-old woman presents with palpitations, anxiety, and weight loss. Examination shows a diffuse goiter and exophthalmos. Laboratory findings revealed low TSH, high T_3, and T_4.
Which of the following best explain the cause of her thyrotoxicosis?
(A) Autoantibodies stimulating TSH receptors on thyroid follicular cells.
(B) Ingestion of excess thyroxine tablets.
(C) Mutation causing defective TSH receptors.
(D) Thyroid peroxidase enzyme deficiency.
(E) TSH-secreting pituitary adenoma.

(20) A 20-year-old man presents with polyuria, polydipsia, and weight loss. Laboratory results show fasting blood glucose of 500 mg/dL, and urine positive for ketones.
Which of the following best explain the cause of ketone body formation in this patient?
(A) Decreased glucagon secretion.

(B) Decreased lipolysis due to low insulin levels.
(C) Increased fatty acid oxidation.
(D) Increased glucose uptake by adipose tissue.
(E) Increased synthesis of triglycerides in the liver.

CAT-2 (BATCH 2) MCQ PAPER ANSWERS

(1) TTFTT

(2) TFFTF

(3) TTTTF

(4) FTTFT

(5) FTTTT

(6) TTTTF

(7) TFTFF

(8) TTFFT

(9) FTFTT

(10) TFTTF

(11) D

(12) D

(13) C

(14) B

(15) E

(16) B

(17) D

(18) D

(19) A

(20) C

CAT-2 (BATCH 2) STRUCTURED ESSAY PAPER

(1) A 52-year-old man presents with yellowish discoloration of eyes, pale-colored stools, dark urine, and generalized pruritus for the past two weeks.
Laboratory investigations reveal markedly elevated conjugated (direct) bilirubin.
- (1.1) Define jaundice (10 marks)
- (1.2) List three possible causes of jaundice in this patient (15 marks)
- (1.3) With the help of a labeled diagram, explain the physiological basis of the following observations:
  - (1.3.1) Pale-colored stool (20 marks)
  - (1.3.2) Dark urine (15 marks)
  - (1.3.3) Generalized pruritus. (10 marks)
- (1.4) Few weeks later, the patient develops gross ascites and bleeding from puncture sites.
  - (1.4.1) Name two possible causes for the development of ascites in this patient. (10 marks)
  - (1.4.2) Name two possible causes for the bleeding tendency in this patient. (10 marks)
- (1.5) Mention two radiological investigations that would be helpful for further evaluation of this patient. (10 marks)

(2) A 45-year-old woman was admitted to a medical ward with complaints of tingling sensations around her mouth and fingertips, along with intermittent muscle cramps in her hands and feet. Laboratory investigations revealed a serum calcium level of 6.5 mg/dL (normal: 8.5–10.5 mg/dL)
- (2.1) List two possible causes for this biochemical abnormality. (10 marks)
- (2.2) Name two bedside examinations you would perform to confirm your diagnosis. (20 marks)
- (2.3) With the help of a diagram, explain the homeostatic mechanisms involved in restoring plasma calcium towards normal in this patient. (50 marks)
- (2.4) List two ECG abnormalities that occur in this patient. (20 marks)

CAT-2 (BATCH 2) SEQ PAPER ANSWERS

(1)

(1.1) Define jaundice. (10 marks)

Yellowish discoloration of the skin, sclerae, and mucous membranes caused by elevated
serum bilirubin, usually when it is exceeds 2–3 mg/dL

Marking scheme (10 marks)
Yellow discoloration of skin/sclera – 3 marks
Due to hyperbilirubinaemia – 4 marks
Mention of threshold level (>2–3 mg/dL) – 3 mark

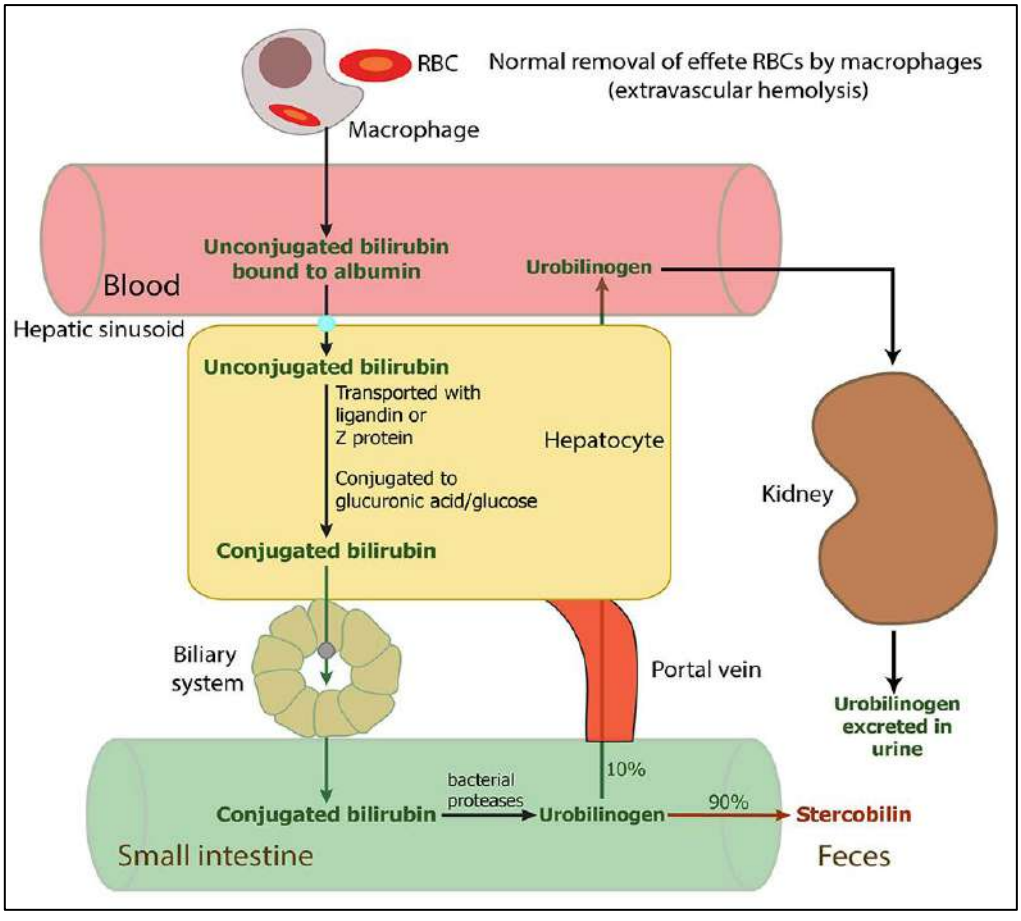
(1.2) List three possible causes of jaundice in this patient. (15 marks)

Any three of the following:
1. Obstruction due to gallstones (choledocholithiasis)
2. Carcinoma of the head of the pancreas causing biliary obstruction
3. Cholangiocarcinoma (bile duct tumour)
4. Primary sclerosing cholangitis
5. Stricture of the common bile duct (post-inflammatory/post-surgical)

Marking scheme (15 marks)
Each correct cause – 5 marks × 3

(1.3) Explain the physiological basis of the following observations.

Bilirubin metabolism



(1.3.1) Pale-colored stools (20 marks)

Bilirubin is conjugated in the liver and excreted into bile.
In obstructive jaundice, bile flow into the intestine is blocked.
Therefore, conjugated bilirubin does not reach the intestine.
Intestinal bacteria cannot convert bilirubin into urobilinogen.
Absence of urobilinogen leads to absence of stercobilin
Hence stools become pale or clay-coloured

Marking scheme.
Clear diagram and normal bilirubin metabolism – 5 marks
Obstruction preventing bile entry – 5 marks
Absence of urobilinogen formation – 5 marks
Absence of stercobilin – 5 marks

(1.3.2) Dark urine (10 marks)

In obstructive jaundice, conjugated bilirubin accumulates in blood.
Conjugated bilirubin is water soluble.
It is filtered by the kidneys and excreted in urine.
Presence of conjugated bilirubin gives urine a dark brown.

Marking scheme
Raised conjugated bilirubin – 4 marks
Water solubility – 4 marks
Renal excretion – 4 marks
Explanation of dark colour – 3 marks

(1.3.3) Generalized pruritus (10 marks)

Obstruction to bile flow leads to retention of bile acids and bile salts in blood.
These substances deposit in the skin.
They stimulate cutaneous nerve endings, producing intense itching.

Marking scheme (10 marks)
Bile acid retention – 4 marks
Skin deposition – 3 marks
Nerve stimulation causing itch – 3 marks

(1.4)

(1.4.1) Two possible causes for ascites. (10 marks)

Portal hypertension due to chronic liver disease
Hypoalbuminaemia causing reduced plasma oncotic pressure

Marking scheme (10 marks)
Each correct cause – 5 marks × 2

(1.4.2) Two possible causes for bleeding tendency. (10 marks)

Reduced synthesis of clotting factors (II, VII, IX, X) due to liver dysfunction
Vitamin K deficiency due to impaired bile-mediated fat absorption

Marking scheme (10 marks)
Each correct cause – 5 marks × 2

(1.5) Mention two radiological investigations helpful for further evaluation. (10 marks)

Ultrasound scan of abdomen
CT scan / MRCP of hepatobiliary system

Marking scheme (10 marks)
Each correct investigation – 5 marks × 2

(2)

(2.1) List two possible causes for this biochemical abnormality. (10 marks)

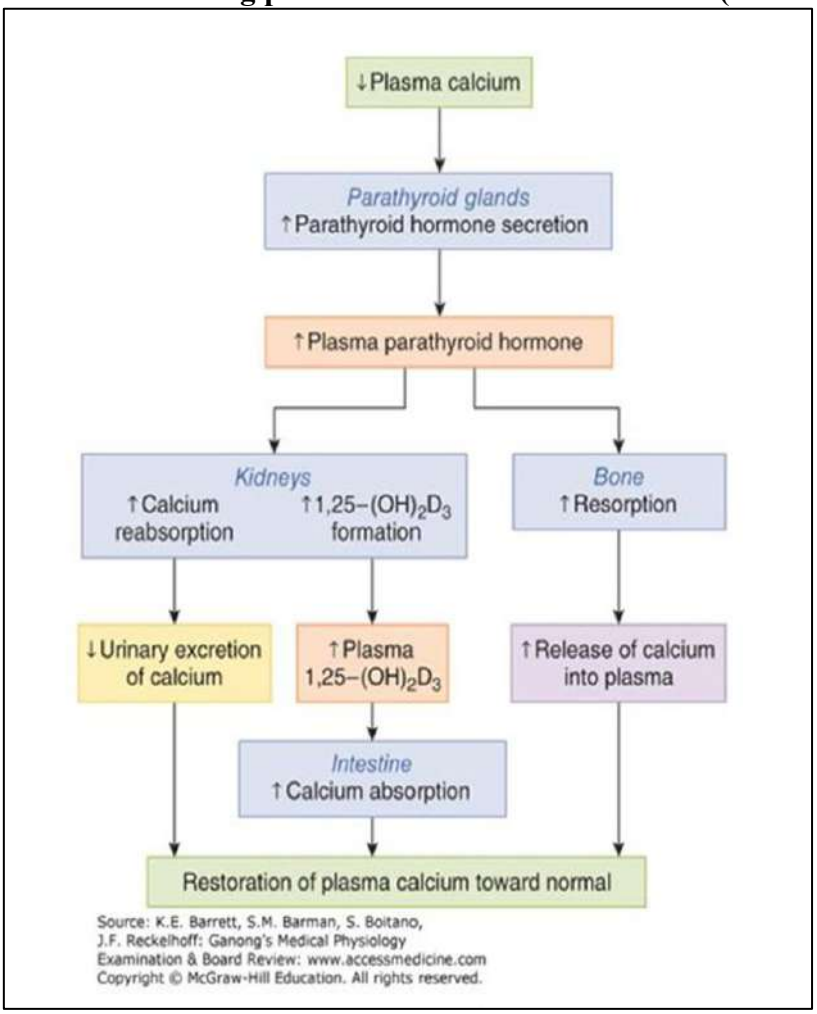
1.Hypoparathyroidism (e.g., post-thyroid/parathyroid surgery, autoimmune).
2.Vitamin D deficiency / impaired vitamin D activation (e.g., malabsorption, chronic kidney disease).
(Other acceptable causes: chronic kidney disease, acute pancreatitis, massive transfusion with citrate, hypomagnesaemia

Marking scheme (10 marks)
Any two correct causes: 5 marks each = 10 marks

(2.2) Name two bedside examinations you would perform to confirm your diagnosis. (20 marks)

Marking scheme (20 marks)
Trousseau's sign: 10 marks
Chvostek's sign: 10 marks

(2.3) With the help of a diagram, explain the homeostatic mechanisms involved in restoring plasma calcium towards normal. (50 marks)

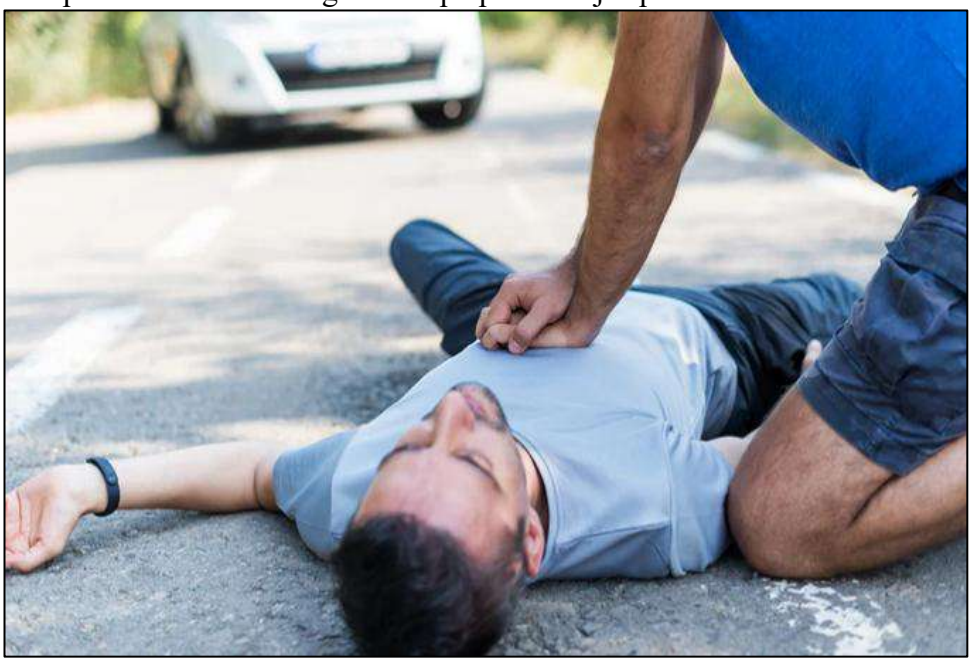


↓ Plasma ionized Ca^{2+}
CaSR (calcium-sensing receptors) in parathyroid chief cells detect low Ca^{2+}
→ ↑ Parathyroid hormone (PTH) secretion
PTH acts on target organs
A. Bone
PTH → activates osteoclasts
↑ Bone resorption → ↑ release of Ca^{2+} (and phosphate) to blood
B. Kidney
↑ Ca^{2+} reabsorption in distal tubule → reduce urinary excretion of calcium
↓ Phosphate reabsorption in proximal tubule → phosphaturia
→ helps raise free Ca^{2+} (less Ca–phosphate complexing)
↑ 1α-hydroxylase activity → converts 25(OH)D to $1,25(OH)_2D$ (calcitriol)
C. Intestine (via calcitriol)
Calcitriol → ↑ intestinal absorption of Ca^{2+} (and phosphate)
Net effect:
↑ Plasma Ca^{2+} back toward normal (negative feedback reduces PTH once Ca^{2+} normalizes)
Marking scheme (50 marks)
1.well-labeled diagram/flowchart 10 marks
2.Identify low ionized Plasma Ca^{2+} by CaSR/parathyroid): 5 marks
3.Hormonal response: ↑ PTH secretion: 5 marks
4.Bone actions (osteoclast activation ↑ Ca^{2+} release): 5 marks
5.Renal actions (↑ distal Ca^{2+} reabsorption): 5 marks
6.Renal phosphate effect (↓ proximal phosphate reabsorption → phosphaturia → ↑ free Ca^{2+}): 5 marks
7.Vitamin D activation (↑ 1α-hydroxylase → ↑ calcitriol): 5 marks
8.Intestinal effect via calcitriol (↑ Ca^{2+} absorption): 5 marks
9.Negative feedback (restoration of Ca^{2+} suppresses PTH): 5 marks
Total = 50 marks

(2.4) List two ECG abnormalities that can occur in this patient (20 marks)
1.Prolonged QT interval (mainly due to ST-segment prolongation).
2.Ventricular arrhythmias (e.g., torsade's de pointes, ventricular ectopic).
Marking scheme (20 marks)
Any two correct ECG abnormalities: 10 marks each = 20 marks

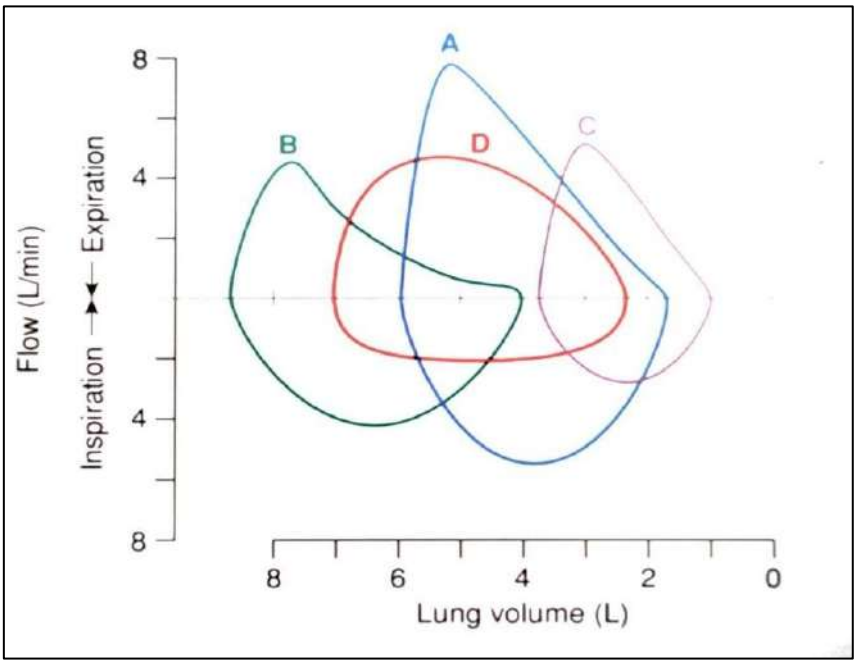
CAT-2 (BATCH 2) OSPE QUESTIONS

(1) You find a man lying on the road who is unresponsive. On quick assessment, he has no spontaneous breathing and no palpable major pulses.



(1.1) List three initial steps you would take immediately. (03 Marks)
(1.2) State two steps that you would follow when giving chest compressions according to Basic Life Support (BLS) guidelines. (04 Marks)
(1.3) State the compression-to-ventilation ratio for single rescuer Cardio Pulmonary Resuscitation (CPR) in this person. (03 Marks)

(2)



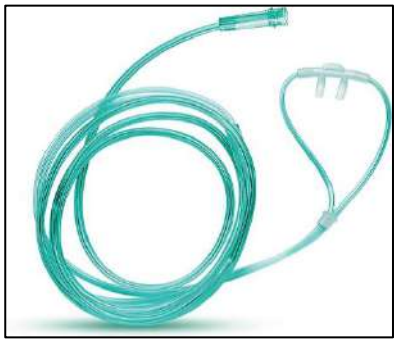
(2.1) Identify A.B, C and D. (8 marks)
(2.2) State one condition that could give rise to each of the loop B and C. (2 marks)

(3) A

B

C

D



(3.1) Identify A, B, C and D (4 marks)

(3.2) State which device out of devices A, B, C and D would be the best device to use in each of the following clinical conditions/situations. (6 marks)

(3.2.1) Low flow oxygen delivery for stable patients

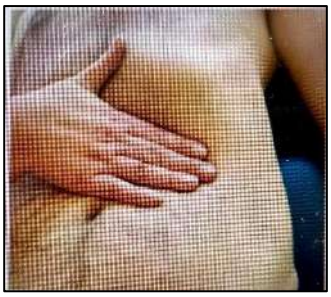
(3.2.2) Moderate hypoxemia

(3.2.3) COPD

(3.2.4) Severe hypoxemia

(4) A

B



C

(4.1) Identify the intra-abdominal organs being examined in figures A, B and C (3 marks)

(4.2) State one clinical finding you would observe for each enlarged organ in A, B and C during your palpation. (3 marks)

(4.3) Mention two additional steps you would perform to differentiate the enlarged organs identified in B and C. (4 marks)

(5) From investigations A, B, C and D, select the most appropriate investigation for each clinical indication.

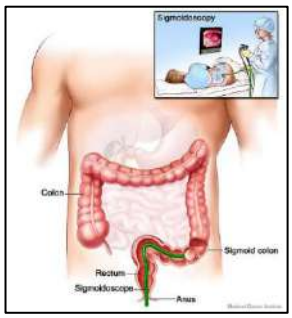
(5.1) Peptic ulcer disease (2.5 marks)

(5.2) Ascites (2.5 marks)

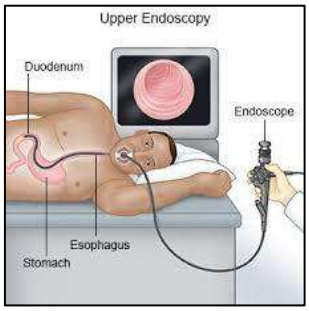
(5.3) Small bowel disease (2.5 marks)

(5.4) Left side colonic neoplasm (2.5 marks)

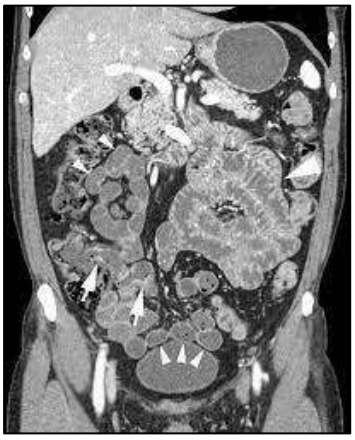
A



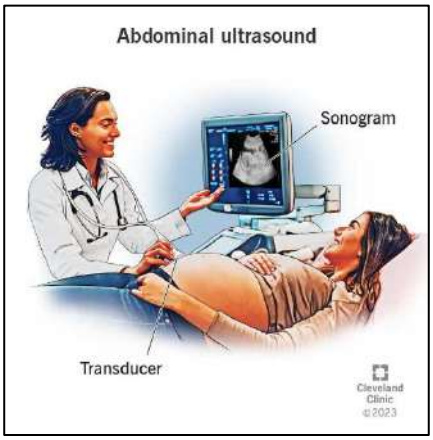
B



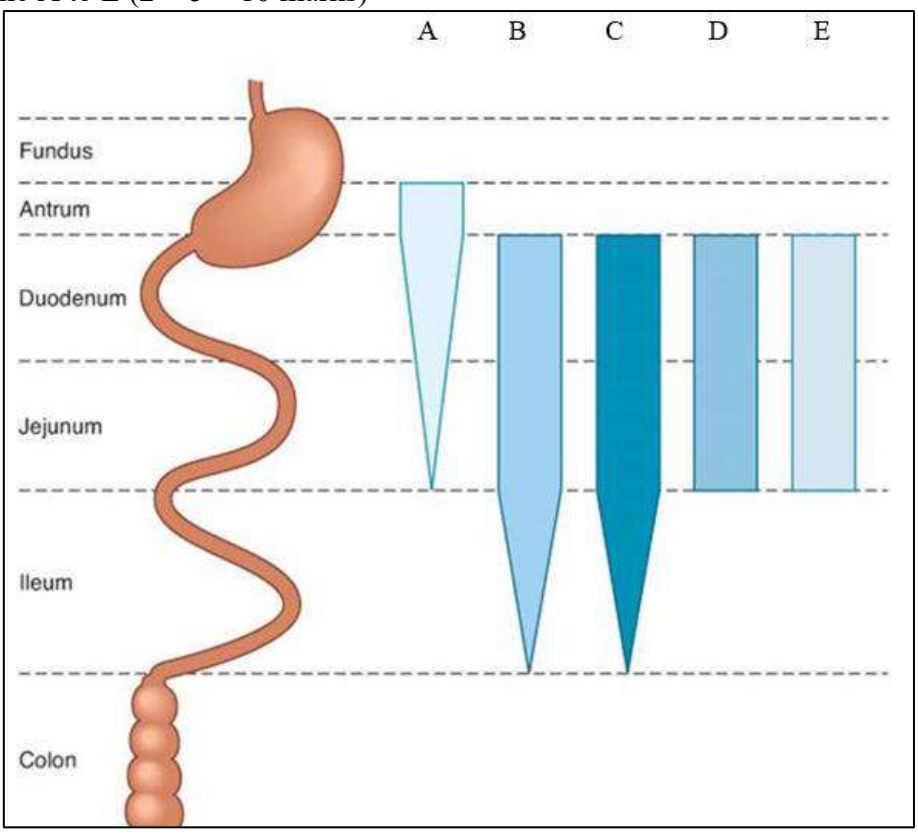
C



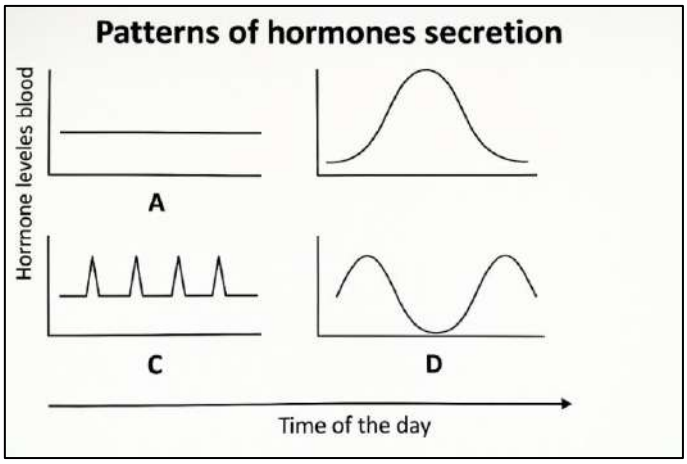
D



(6) This picture illustrates five gastrointestinal hormones along the length of the GI tract.
Name A to E (2 × 5 = 10 marks)

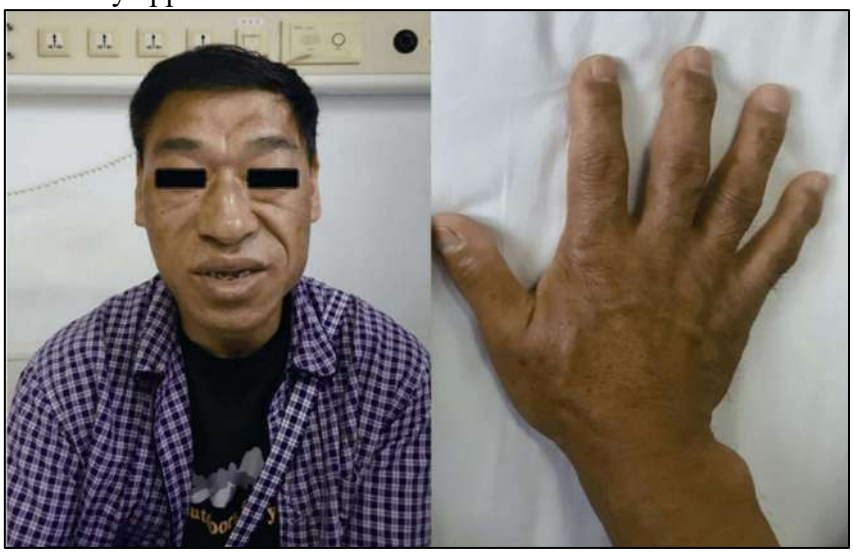


(7)



(7.1) Identify the pattern of hormone secretion shown in each graph. (6 marks)
(7.2) Name one hormone that follows each pattern. (4 marks)

(8) This patient complains of headache and excessive sweating along with progressive changes in body appearance



(8.1) State **two** abnormalities seen in this patient. (04 Marks)
(8.2) What is the name given for this condition? (02 Marks)
(8.3) What is the hormone responsible for this condition? (04 Marks)

(9) A 35-year-old woman presents with weight gain, facial puffiness, and easy bruising.
Her baseline serum cortisol level measured at 9 a.m. is very high.
To evaluate the cause for high cortisol level in the blood, she was given a drug at 11pm, and the serum cortisol level was re-measured the next morning at 9 am.
(9.1) What is the name of the test performed? (3 marks)
(9.2) What drug was given at 11pm? (3 marks)
(9.3) What is the physiological basis of this test? (4 marks)

(10) Live station
Demonstrate how to correctly measure Peak Expiratory Flow Rate (PEFR) using this device. (Peak flow meter is provided)

CAT-2 (BATCH 2) OSPE ANSWERS

(1)

(1.1) Ensure safety of scene.
Check responsiveness (shake and shout).
Call for help/start CPR. (1 × 3 = 3 marks)

(1.2) Hand position: Heel of one hand on center of chest (lower half of sternum), other hand on top.
Depth: At least 5 cm (2 inches).
Rate: 100–120 compressions per minute.
Allow complete chest recoil after each compression. (4 marks)

(1.3) Compression-to-ventilation ratio: 30:2. (3 marks)

(2)

(2.1) A normal flow -volume curve
B. obstructive lung disease
C. Restrictive lung disease
D Fixed upper airway obstruction (2 × 4 =8 marks)

(2.2) B-Asthma/COPD/chronic bronchitis, emphysema
C-pulmonary fibrosis/kyphoscoliosis/obesity/interstitial lung disease
(1 × 2 = 2 marks)

(3)

(3.1) A — non-rebreather mask (Reservoir mask)
B – Simple face mask
C – Venturi mask
D) Nasal cannula (Nasal prongs) (1 × 4 = 4 marks)

(3.2) a) Nasal cannula (D)
b) Simple face mask (B)
c) Venturi mask (C)
d) Non-rebreather mask (A) (1.5 × 4 = 6 marks)

(4)

(4.1) A Liver B – Spleen C – Kidney (1 × 3 = 3 marks)

(4.2) An Enlarged Liver-- Smooth, firm edge palpable below the right costal margin on inspiration.
B – Enlarged Spleen (Splenomegaly)-- A firm mass with a notched anterior border palpable moving inferomedial from left to right.
C – Enlarged Kidney (Renal mass) --A bimanually palpable mass that is ballotable but does not move with respiration. (1 × 3 = 3marks)

(4.3) To differentiate Spleen (B) from Kidney (C):
1. Check for movement with respiration
Spleen moves downwards on inspiration; kidney shows minimal movement.
2.Look for a splenic notch
Anterior notch suggests spleen; kidneys never have a palpable notch.
3.Attempt balloting
Kidney is ballotable; spleen is NOT.
4.Percussion over the mass
Spleen causes dullness (because it enlarges anteriorly).

Kidney often retains resonant band on percussion due to overlying transverse colon.
(Any two of the above steps are acceptable.) (2 × 2 = 4)

(5)
(5.1) B
(5.2) D
(5.3) C
(5.4) A (2.5 × 4 = 10 marks)

(6) A Gastrin
B CCK
C Secretin
D GIP
E Motilin (2 × 5 = 10 marks)

(7)
(7.1) A – Continuous/constant secretion
B – Circadian/diurnal secretion
C – Pulsatile secretion
D – Cyclical secretion (1 × 4 = 4 marks)
(7.2) A – Continuous/constant secretion – Thyroxin(T4)/Aldosterone/prolactin
B – Circadian/diurnal secretion – Cortisol/ACTH/Testosterone
C – Pulsatile secretion – GnRH/GH
D – Cyclical secretion – Estrogen/progesterone (1.5 × 4 = 4 marks)

(8)
(8.1) Coarse facial features with prognathism (enlarged jaw, prominent facial bones)
Enlarged, broad hands with thickened fingers (spade-like hands)
(2 × 2 = 4 marks)
(8.2) Acromegaly. (2 marks)
(8.3) Growth Hormone (4 marks)

(9)
(9.1) Overnight dexamethasone suppression test (ODST) (3 marks)
(9.2) Dexamethasone (3 marks)
(9.3) Dexamethasone is a potent synthetic glucocorticoid that provides negative feedback to the hypothalamus and anterior pituitary, suppressing ACTH secretion. In normal individuals, this suppression leads to reduced cortisol production by 9 a.m. In Cushing's syndrome, cortisol secretion becomes autonomous or ACTH-dependent and resistant to feedback, so the morning cortisol fails to suppress. (4 marks)

MESSAGE OF THE DEAN

As the Dean of the Uva Wellassa Medical Faculty, it is with deep pride and sincere enthusiasm that I introduce to you "Mastering Physiology–Part II: A Comprehensive Guide to MCQs, Essays, and OSPEs." This publication marks an important milestone in our continued dedication to improving the educational experience of students.

In the constantly advancing field of medicine, a strong understanding of Physiology remains fundamental to future achievements. This guide has been carefully developed to provide you with an extensive resource that not only addresses a broad spectrum of question formats including MCQs, essays, and OSPEs but also delivers detailed answers and explanations to strengthen learning.

The purpose of this book is to provide you with essential tools required to perform well in examinations and to enhance your understanding of anatomical concepts. We are confident that this guide will function as a valuable companion in your academic journey, providing clarity, confidence, and a solid framework for studies.

I convey my sincere appreciation to faculty members and contributors who have committed their knowledge and effort to bring this resource to fruition. It is our wish that you will regard this guide as a source of guidance and motivation as you progress through medical education.

Wishing you continued success in your studies and future medical profession career.

Sincerely,

Senior Professor Muditha Vidanapathirana,
MBBS (Col), DLM, MD, MA (SJP), FFFLM (UK)
Dean,
Faculty of Medicine,
Uva Wellassa University of Sri Lanka

Rs. 1500/-

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