QUESTION

A patient with diabetes mellitus for the past 5 years presents with vomiting and abdominal pain. She is non-compliant with medication and appears dehydrated. Investigations revealed a blood sugar value of 500 mg/dL and the presence of ketone bodies. What is the next best step in management?

A Intravenous fluids with S.C. long-acting insulin

B Intravenous fluids with S.C. regular insulin

C Intravenous insulin I.V. long-acting insulin

D **Intravenous fluids with I.V. regular insulin** CORRECT

OK RIGHT ANSWER

D. Intravenous fluids with I.V. regular insulin

WHY THIS IS RIGHT

This presentation is diabetic ketoacidosis with dehydration, hyperglycemia, and ketonemia requiring emergency management.
Insulin deficiency
□
Increased lipolysis
□
Free fatty acids released
□
Ketone body formation
□
Metabolic acidosis

At the same time:
Hyperglycemia
□
Osmotic diuresis
□
Severe dehydration

The priority is rapid isotonic IV fluids followed by continuous IV regular insulin to stop ketogenesis. Subcutaneous insulin is unreliable in shock due to poor perfusion.
Long-acting insulin has no role in acute DKA control.

A. IV fluids + SC long-acting insulin
• Long-acting insulin is too slow
• Not used in emergencies

B. IV fluids + SC regular insulin
• Subcutaneous route is unpredictable in shock/dehydration
• IV route is preferred in DKA

C. IV insulin long-acting
• Long-acting insulin is never given IV

Not suitable for acute correction

QUESTION

In Type 1 Diabetes Mellitus, what is the feature of stage 3 beta cell destruction

A Autoimmune +ve Normoglycemic presymptomatic

B **Autoimmune +ve dysglycemic symptomatic** CORRECT

C Autoimmune -ve Normoglycemic presymptomatic

D Autoimmune -ve dysglycemic presymptomatic

OK RIGHT ANSWER

B. Autoimmune +ve dysglycemic symptomatic

WHY THIS IS RIGHT

The source quiz contains the placeholder “$19” here and does not include a written explanation for this item.

QUESTION

Which receptor helps in improvement of insulin resistance in Type-2 Diabetes Mellitus with regular exercise and physical activity?

A GLUT1

B **GLUT4** CORRECT

C GLUT2

D GLUT3

OK RIGHT ANSWER

B. GLUT4

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1a” here and does not include a written explanation for this item.

QUESTION

When blood glucose levels are increased to 2-3 times their normal value and then maintained at that level for a consistent period, which of the following statements best describes the pattern of insulin secretion?

A Rise and fall rapidly

B Gradually rise and fall

C Remain elevated

D **1st it rises then remains elevated** CORRECT

RIGHT ANSWER

OK

D. 1st it rises then remains elevated

WHY THIS IS RIGHT

Phase	Timing	Source	Pattern
Phase 1	Minutes	Stored insulin	Rapid spike
Phase 2	Hours	Newly synthesized insulin	Sustained plateau

Sustained hyperglycemia produces biphasic insulin secretion: an initial rapid first phase from release of preformed insulin, followed by a prolonged second phase due to new insulin synthesis. When glucose remains high, secretion does not fall back to baseline but stays elevated. This classic two-phase response is a hallmark of normal β-cell physiology.
Loss of first phase is an early feature of type 2 diabetes.

Condition	Phase 1	Phase 2
Normal	Present	Present
Type 2 DM	Lost early	Reduced

QUESTION

A 40-year-old male presents with increased thirst and increased frequency and volume of urination. He has no other symptoms. His random blood sugar was 205 mg/dl, what appropriate investigations should be done?

i) HbA1c
ii) Urine ACR
iii) Echocardiography
iv) Fundoscopy
v) ECG
vi) Serum protein electrophoresis

A i, ii, iii, v

B i, ii, iii, vi

C i, ii, iii, v

D **i, ii, iv, v** CORRECT

RIGHT ANSWER

OK **D. i, ii, iv, v**

WHY THIS IS RIGHT

Investigation	Purpose	Why Important
HbA1c □	Long-term glucose control	Reflects 3-month average
Urine ACR □	Nephropathy screening	Detects microalbuminuria
Fundoscopy □	Retinopathy screening	Early eye damage
ECG □	Cardiovascular risk	Detect silent ischemia

Random glucose 205 mg/dL with polyuria and polydipsia suggests diabetes mellitus, so HbA1c confirms chronic hyperglycemia. Urine ACR screens for early diabetic nephropathy, while fundoscopy detects diabetic retinopathy at baseline. ECG is recommended because diabetes is a coronary risk equivalent and silent ischemia is common.

Echocardiography and serum protein electrophoresis are not routine initial evaluations in uncomplicated new diabetes.

QUESTION

A 27-year-old female patient presents with weight loss, anxiety, tachycardia, and restlessness. The resting pulse was 110 beats per minute. Which of the following tests should be done next to evaluate her condition further?

A Total T3

B Thyroid scan

C Free T4

D **TSH** CORRECT

OK RIGHT ANSWER

D. TSH

WHY THIS IS RIGHT

TSH is the best initial screening test for suspected hyperthyroidism because it is the most sensitive indicator of thyroid function.

Test	Role	When Used
TSH □	Initial screening	First test
Free T4	Confirm severity	After TSH
Total T3	Sometimes used	Less preferred
Thyroid scan	Etiology	Later step

In thyrotoxicosis, TSH is characteristically suppressed due to negative feedback from elevated thyroid hormones. Free T4 or T3 are measured only after an abnormal TSH to confirm and classify severity. Thyroid scan is reserved for etiologic evaluation, not first-line screening.

QUESTION

A patient with a history of diabetes mellitus presents with heart failure characterized by a reduced ejection fraction and low urine output. Which of the following anti-diabetic medications has been recently approved for the treatment of heart failure with reduced ejection fraction, and may also improve diuresis?

A **Empagliflozin** CORRECT

B Linagliptin

C Rosiglitazone

D Insulin degludec

OK RIGHT ANSWER

A. Empagliflozin

WHY THIS IS RIGHT

Correct answer is A because empagliflozin (SGLT2 inhibitor) is approved for heart failure with reduced ejection fraction and improves outcomes independent of diabetes control.
Empagliflozin blocks SGLT2 in proximal tubule
□
↓ Glucose reabsorption
□
↑ Glucose excretion
□
Osmotic diuresis
□
↓ Plasma volume
□
↓ Preload & afterload
□
Improves heart failure
It promotes osmotic diuresis and natriuresis, helping relieve congestion and improve cardiac function.
DPP-4 inhibitors, thiazolidinediones, and insulin do not provide this heart failure benefit.

QUESTION

A 10-year-old child presented with abdominal pain and vomiting. Lab values are given below. What is the next best step of management?

Lab Values:
RBS - 450 mg/dL
pH - 7.0
HCO3 - 4
Na - 145
K - 4.8 meq/L

A 10 ml/kg NS over 20 min (10% deficit correction) followed by insulin infusion with 20 mmol/L KCl with bicarbonate

B 20 ml/kg 1/2 NS over 20 min (7% deficit correction) followed by insulin infusion and KCl 40 meq/L

C **20 ml/kg NS over 20 min with (10% deficit correction) followed by insulin infusion and KCl 40 meq/L** CORRECT

D 20 ml/kg RL over 20 min with (10% deficit correction) followed by insulin infusion and KCl 40 meq/L

RIGHT ANSWER

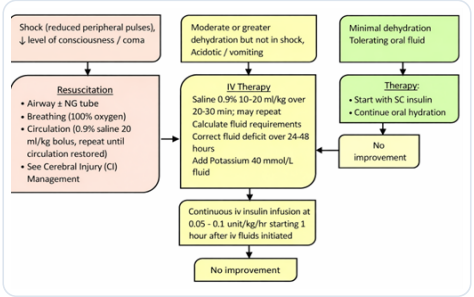
OK **C. 20 ml/kg NS over 20 min with (10% deficit correction) followed by insulin infusion and KCl 40 meq/L**

WHY THIS IS RIGHT

Correct answer is C according to ISPAD guidelines, as the labs show severe diabetic ketoacidosis (glucose 450, pH 7.0, HCO□ 4). First step is rapid isotonic fluid resuscitation with 20 ml/kg normal saline to restore perfusion.
After initial fluids, insulin infusion is started, 0.1 units/kg/hour with potassium replacement because total body potassium is depleted despite normal serum K.
Effects:
• Stops ketogenesis
• Reduces glucose
• Stops lipolysis

Bicarbonate is not routinely given unless pH < 6.9. Half-normal saline and RL are not preferred for initial resuscitation in pediatric DKA.
RL not preferred initially.
Reasons:
• Lactate -> metabolized to bicarbonate
• May interfere with ketone monitoring

NS is standard guideline recommendation



QUESTION

Insulin independent absorption occurs in all except:

A **Adipose** CORRECT

B RBC

C Pancreas

D Brain

RIGHT ANSWER

OK

A. Adipose

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1b” here and does not include a written explanation for this item.

QUESTION

Which is not considered in the diagnosis of Metabolic syndrome:

A **Hip Circumference** CORRECT

B HDL

C Glucose

D Hypertension

RIGHT ANSWER

OK **A. Hip Circumference**

WHY THIS IS RIGHT

The criteria for diagnosing metabolic syndrome usually include a combination of factors such as central obesity (usually measured by waist circumference), elevated blood pressure, elevated fasting glucose, high triglycerides, and low high-density lipoprotein (HDL) cholesterol.

Metabolic syndrome: NCEP-ATP III
1\. Central obesity:
\>102 cm (India-90cm) in men
\>88 cm (India-80cm) in women
2\. Elevated triglycerides: \>150 mg/dL
3\. HDL
< 40 mg/dL in men
< 50 mg/dL in women.
4\. Blood pressure: \>130/85 mm Hg
5\. Fasting glucose: \>100 mg/dL

Metabolic syndrome increases risk of:
• Type 2 diabetes
• Cardiovascular disease
• Stroke

Management Focus on:
Lifestyle
• weight reduction
• exercise
• diet control

Pharmacological
• statins (lipids)
• antihypertensives
• metformin (insulin resistance)

QUESTION

A 30-year-old patient with heart failure and poorly controlled type 2 diabetes (HbA1c of 7.8%) present with progressive dyspnea. Which is the preferred antidiabetic drug?

A **Dapagliflozin** CORRECT

B Glimepiride

C Metformin

D Sitagliptin

RIGHT ANSWER

OK **A. Dapagliflozin**

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1c” here and does not include a written explanation for this item.

QUESTION

Which of the following drugs used in the management of diabetes is associated with unexplained diarrhea?

A Pioglitazone

B Sitagliptin

C Glimepiride

D **Metformin** CORRECT

RIGHT ANSWER

OK

D. Metformin

WHY THIS IS RIGHT

Correct answer is D because metformin commonly causes gastrointestinal adverse effects including diarrhea due to increased intestinal glucose utilization and altered gut motility.

Mechanism	Effect
↑ intestinal motility	faster transit
↓ glucose absorption	osmotic effect
↑ bile salt malabsorption	irritation
Altered gut microbiota	fermentation changes

This side effect is dose related and often improves with gradual dose escalation or extended-release formulations. Metformin is a biguanide that primarily acts on the liver. Key actions:
1□□ ↓ Hepatic gluconeogenesis
2□□ ↑ Peripheral insulin sensitivity
3□□ ↓ Intestinal glucose absorption

Pioglitazone causes edema/weight gain. Sitagliptin is generally well tolerated. Glimepiride mainly causes hypoglycemia.

QUESTION

Which of the following drugs used in the management of type 2 diabetes is associated with an increased risk of urinary tract infections?

A **Dapagliflozin** CORRECT

B Pioglitazone

C Sitagliptin

D Metformin

OK RIGHT ANSWER

A. Dapagliflozin

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1d” here and does not include a written explanation for this item.

QUESTION

All of the following are criteria for diagnosis of DKA except:

A S. glucose > 250 mg/dL

B Arterial pH < 7.3

C Anion gap > 12

D **S. Potassium < 3.5 mEq/L** CORRECT

RIGHT ANSWER

OK **D. S. Potassium < 3.5 mEq/L**

WHY THIS IS RIGHT

Correct answer is D because diagnostic criteria for DKA include hyperglycemia (>250 mg/dL), metabolic acidosis (pH < 7.3), and increased anion gap metabolic acidosis.

Insulin deficiency
□
↑ lipolysis
□
↑ free fatty acids
□
Liver -> ketone bodies
□
Metabolic acidosis (high anion gap)

Serum potassium is often normal or elevated initially due to extracellular shift despite total body potassium depletion. Hypokalemia is a complication during treatment, not a diagnostic criterion.

QUESTION

In uncontrolled diabetes mellitus, which of the following cell types exhibit decreased glucose uptake as a result of insulin deficiency?

A RBC

B **Skeletal Muscle** CORRECT

C Liver

D Brain

OK RIGHT ANSWER

B. Skeletal Muscle

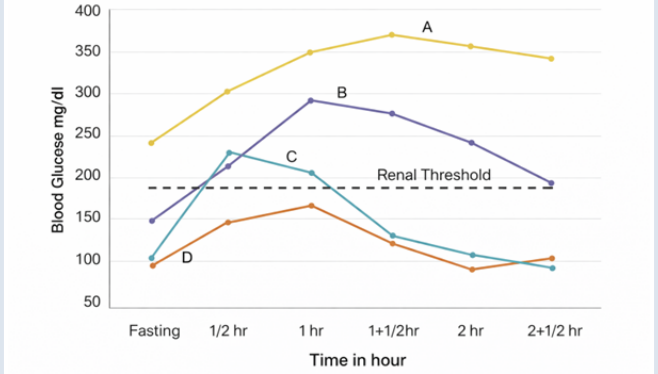
WHY THIS IS RIGHT

Correct answer is B because skeletal muscle requires insulin-dependent GLUT-4 transporters for glucose uptake, so insulin deficiency markedly reduces glucose entry into these cells.
RBCs and brain use insulin-independent transporters (GLUT-1/GLUT-3). Liver uptake via GLUT-2 is also insulin independent.

Transporter	Location	Function
GLUT-1	Brain, kidney, placenta, erythrocytes	Basal uptake of glucose, high affinity
GLUT-2	Liver, pancreatic cell, small intestine	Fed state
GLUT-3	Brain, kidney, placenta	Glucose uptake, maximum affinity for glucose
GLUT-4	Heart and skeletal muscle, adipose tissue	Insulin-stimulated glucose uptake
GLUT-5	Small intestine, sperms	Fructose

QUESTION

Oral glucose tolerance test with blood glucose levels is plotted against time. Which among the following is indicative of diabetes mellitus?



A A,B

B **A,B,C** CORRECT

C A only

D A, B, C, D

OK RIGHT ANSWER

B. A,B,C

WHY THIS IS RIGHT

The OGTT evaluates how efficiently the body handles a glucose load.
Procedure:

1. Patient fasts 8-10 hours
2. Measure fasting plasma glucose
3. Give 75 g oral glucose
4. Measure glucose at intervals
• 30 min
• 1 hr
• 2 hr (most important)

Correct answer is B because curves A, B, C remain above the renal threshold for a prolonged period and show delayed return toward baseline, indicating impaired glucose tolerance consistent with diabetes.
Persistent hyperglycemia beyond 2 hours is the key diagnostic feature of diabetes on OGTT.

Value	Diagnosis
<140	Normal
140-199	Prediabetes
≥200	Diabetes

Curve D shows a normal rise and fall with return to baseline, representing normal glucose handling.

QUESTION

A 22-year-old woman presents to the emergency department with fatigue, abdominal pain, and confusion. Laboratory results reveal the following:
Glucose (serum) 720 mg/dL
Sodium 132 meq/l
Potassium 5.2meq/l
Bicarbonate 10meq/l
Arterial pH 7.25
Creatinine 1.5mg/dl
Blood urea nitrogen 35mg/dl
Urinalysis: Positive for urine and ketones
Which of the following electrolyte findings would most likely be seen in this patient?

A **Extracellular potassium concentration increase, Intracellular potassium concentration decrease** CORRECT

B Extracellular potassium concentration increase, Intracellular potassium concentration increase

C Extracellular potassium concentration decrease, Intracellular potassium concentration increase

D Extracellular potassium concentration decrease, Intracellular potassium concentration decrease

RIGHT ANSWER

OK **A. Extracellular potassium concentration increase, Intracellular potassium concentration decrease**

WHY THIS IS RIGHT

In diabetic ketoacidosis (DKA), total body potassium is depleted, but serum (extracellular) potassium is often elevated.

Mechanism:
• Insulin deficiency prevents potassium from entering cells.
• Metabolic acidosis causes H□ to move into cells and K□ to shift out.
• This leads to increased extracellular potassium despite decreased intracellular potassium and total body potassium depletion due to osmotic diuresis.

QUESTION

64-year-old woman is brought to the emergency department due to confusion and lethargy. The patient was asymptomatic when her husband left for work in the morning, but when he arrived home, he found her in bed weak and disoriented. The patient's medical conditions include type 2 diabetes mellitus and hypertension, for which she takes multiple medications. Laboratory testing shows an elevated serum C-peptide level. If her current condition is due to an antidiabetic drug, which of the following is the most likely culprit agent?

A Acarbose

B Canagliflozin

C **Glyburide** CORRECT

D Long-acting insulin

OK RIGHT ANSWER

C. Glyburide

WHY THIS IS RIGHT

Sulfonylureas (e.g., glyburide) treat type 2 diabetes by stimulating pancreatic β-cells to release insulin through closure of ATP-sensitive K□ channels. This leads to:
• Increased endogenous insulin secretion
• Elevated C-peptide levels (because insulin is produced by the pancreas)

Excess insulin release can cause severe hypoglycemia, especially in elderly patients or with long-acting sulfonylureas like glyburide.

Why other options are incorrect:

A. Acarbose: Does not increase insulin secretion -> hypoglycemia uncommon.
B. Canagliflozin: Lowers glucose by urinary glucose excretion, not insulin release.
D. Long-acting insulin: Exogenous insulin lacks C-peptide, so C-peptide would be low.

QUESTION

A 40-year-old male presents with increased thirst and increased frequency and volume of urination. His random blood sugar was 205 mg/dl, what appropriate investigations should be done?

i) HbA1c
ii) Urine ACR
iii) Echocardiography
iv) Fundoscopy
v) ECG
vi) Serum protein electrophoresis

A i, ii, iii, v

B i, ii, iii, vi

C i, ii, iii, v

D **i, ii, iv, v** CORRECT

RIGHT ANSWER

OK **D. i, ii, iv, v**

WHY THIS IS RIGHT

In a patient with suspected or newly diagnosed diabetes mellitus, evaluation should include confirmation of glycemic status and screening for early diabetic complications.

Appropriate initial investigations include:
• HbA1c -> assesses long-term glycemic control (average glucose over \~3 months).
• Urine albumin-creatinine ratio (ACR) -> screens for diabetic nephropathy (microalbuminuria).
• Fundoscopy -> detects diabetic retinopathy.
• ECG -> evaluates cardiovascular disease, which is common in diabetics.

Tests like echocardiography and serum protein electrophoresis are not routine initial investigations for newly diagnosed diabetes unless specific clinical indications are present.

QUESTION

A 52-year-old female with diabetes mellitus has experienced a decrease in her HbA1c levels from 7.6 to 6.7 after starting a new oral medication. Laboratory tests show that she has high levels of GIP and GLP-1 and low levels of glucagon. Which of the following oral drugs was most likely administered to this patient?

A Voglibose

B **Alogliptin** CORRECT

C Pramlintide

D Liraglutide

OK RIGHT ANSWER

B. Alogliptin

WHY THIS IS RIGHT

DPP-4 inhibitors improve glycemic control by increasing endogenous incretin hormones (GLP-1 and GIP). These hormones enhance glucose-dependent insulin secretion and suppress glucagon release, leading to a reduction in blood glucose and HbA1c.

Alogliptin is a DPP-4 inhibitor that prevents the degradation of GLP-1 and GIP, resulting in elevated levels of these incretins and decreased glucagon secretion. This mechanism explains the laboratory findings of high GLP-1/GIP levels with low glucagon.

Why not other options:
A. Voglibose: α-glucosidase inhibitor; delays carbohydrate absorption. Does not increase GLP-1 or GIP.
C. Pramlintide: Amylin analogue; suppresses glucagon but does not raise GLP-1/GIP and is injectable, not oral.
D. Liraglutide: GLP-1 receptor agonist; mimics GLP-1 but does not increase endogenous GLP-1 or GIP levels and is injectable.

QUESTION

A 60-year-old woman is diagnosed with type II diabetes mellitus. She has a history of chronic kidney disease. Which of the following statement is correct about the management of this patient?

A Metformin is contraindicated in all stages of CKD

B Pioglitazone is avoided as it is exclusively metabolized by kidneys

C **Glipizide can be used in renal disease** CORRECT

D Linagliptin can be used only after dose reduction

RIGHT ANSWER

OK

C. Glipizide can be used in renal disease

WHY THIS IS RIGHT

In patients with type 2 diabetes mellitus with chronic kidney disease (CKD), the choice of antidiabetic drugs depends on their renal clearance and risk of hypoglycemia.
• Glipizide is a short-acting sulfonylurea that is primarily metabolized in the liver and produces inactive metabolites, making it safer in patients with renal impairment compared with other sulfonylureas (e.g., glyburide). Therefore, it can be used in CKD.

Key related facts:
• Metformin is not contraindicated in all CKD stages; it is avoided mainly when eGFR <30 mL/min due to risk of lactic acidosis.
• Pioglitazone is metabolized in the liver, not kidneys, so renal metabolism is not the reason for avoiding it.
• Linagliptin is excreted via the bile and does not require dose adjustment in CKD.

QUESTION

A 15-year-old girl is brought to the emergency department due to confusion, abdominal pain, and vomiting for the past two days. Her parents report that she has been excessively thirsty and urinating frequently over the last few weeks. On examination, her vital signs are as follows: temperature 37.2°C (99°F), pulse 110/min, blood pressure 90/60 mm Hg, and respiratory rate 28/min. Laboratory investigations reveal the following:
• Glucose 320 mg/dL
• Sodium 132 meq/l
• Potassium 5.2 meq/l
• Bicarbonate 12 meq/l
• Arterial pH 7.25
• Urinalysis: Positive for urine and ketones
Which of the following factors most likely contributed to the development of this patient's condition?

A Excessive caloric intake

B Genetic polymorphisms in HLA genes

C Pancreatic beta-cell amyloid deposition

D **Islet leukocytic infiltration** CORRECT

OK RIGHT ANSWER

D. Islet leukocytic infiltration

WHY THIS IS RIGHT

Type 1 diabetes mellitus results from autoimmune destruction of pancreatic β-cells by T-lymphocytes, leading to absolute insulin deficiency. The characteristic pathological finding is lymphocytic infiltration of the pancreatic islets (insulitis).

Loss of insulin causes:
• Decreased glucose uptake
• Increased lipolysis and ketone body production
• High anion gap metabolic acidosis, producing diabetic ketoacidosis (DKA).

Clinical features of DKA include:
• Polyuria and polydipsia
• Dehydration and hypotension
• Abdominal pain and vomiting
• Tachypnea (Kussmaul breathing)
• Altered mental status

QUESTION

A patient with type 2 diabetes on metformin and an add-on drug presents with mild elevation of liver transaminases on routine monitoring. The drug is stopped and liver enzymes normalize. Which add-on drug was most likely responsible?

A Sitagliptin

B **Vildagliptin** CORRECT

C Empagliflozin

D Repaglinide

RIGHT ANSWER

OK

B. Vildagliptin

WHY THIS IS RIGHT

Vildagliptin, a DPP-4 inhibitor used as add-on therapy with metformin in type 2 diabetes, is associated with reversible elevation of liver transaminases (hepatotoxicity). Therefore, periodic monitoring of liver function tests (LFTs) is recommended, and the drug should be discontinued if significant enzyme elevation occurs, after which levels usually normalize.

Why not others (brief):
• Sitagliptin: DPP-4 inhibitor but minimal hepatic toxicity; no routine LFT monitoring needed.
• Empagliflozin: SGLT2 inhibitor; main effects glycosuria, weight loss, genital infections, not hepatotoxicity.
• Repaglinide: Meglitinide causing hypoglycemia and weight gain, not typical transaminase elevation.

QUESTION

A pregnant female presents with amenorrhea and galactorrhea. MRI shows pituitary adenoma. What is the best management of this patient?

A Cabergoline

B **Bromocriptine** CORRECT

C Transsphenoidal surgery

D Goserelin

RIGHT ANSWER

OK

B. Bromocriptine

WHY THIS IS RIGHT

In pregnancy, the drug of choice for prolactinoma is: Bromocriptine
Because:
• Safe in pregnancy
• Extensive clinical experience
• Effectively lowers prolactin

Hypothalamus releases dopamine
□
Dopamine acts on pituitary (D2 receptors)
□
↓ Prolactin secretion

Bromocriptine is the preferred dopamine agonist in prolactinoma during pregnancy because of its long safety record for fetal exposure. It suppresses prolactin and reduces tumor size while preserving fertility.
Surgery is reserved for drug-resistant or compressive tumors.

QUESTION

A 28-year-old male presents with complaints of excessive thirst and frequent urination throughout the day. He mentions having to wake up multiple times at night to urinate. On further questioning, he reports a history of head injury one month ago. His fasting blood glucose is normal. What is the most appropriate next step in management?

A Tolvaptan

B Insulin

C **Desmopressin** CORRECT

D Hydrochlorothiazide

OK

RIGHT ANSWER

C. Desmopressin

WHY THIS IS RIGHT

The source quiz contains the placeholder “$19” here and does not include a written explanation for this item.

QUESTION

A middle-aged male patient presents with chin protrusion, excessive sweating, hormone receptor antagonist used to treat this condition?

A Octreotide

B **Pegvisomant** CORRECT

C Cabergoline

D Olcegepant

OK RIGHT ANSWER

B. Pegvisomant

WHY THIS IS RIGHT

There are three main drug classes used in acromegaly, and examiners often test their differences:

1. Somatostatin analogues -> decrease GH secretion
2. Dopamine agonists -> decrease GH (mild effect)
3. GH receptor antagonists -> block GH action

Pegvisomant belongs to the third category
The features describe acromegaly, and pegvisomant is a GH receptor antagonist that blocks peripheral GH action and lowers IGF-1. It is used when surgery or somatostatin analogs are inadequate.
Octreotide suppresses GH release but is not a receptor antagonist. Cabergoline is a dopamine agonist, and olcegepant is unrelated to GH.
A. Octreotide
• Somatostatin analogue
• ↓ GH secretion from pituitary
• Not a receptor antagonist

C. Cabergoline
• Dopamine agonist
• Mildly suppresses GH
• Mainly used in prolactinoma

D. Olcegepant
• CGRP antagonist
• Used in migraine
• No role here

QUESTION

A pituitary tumor that overproduced growth hormone was surgically removed from a patient. The resection was discovered to be insufficient. What is the patient's first-line treatment?

A Leuprolide

B Goserelin

C Nafarelin

D **Octreotide** CORRECT

RIGHT ANSWER

OK

D. Octreotide

WHY THIS IS RIGHT

Persistent acromegaly after incomplete surgery is treated first-line with somatostatin analogues.
Surgery (first-line)
□
If incomplete or residual tumor
□
Medical therapy (first-line drug = somatostatin analogue)
□
Radiotherapy (if needed)
Therefore, after incomplete surgery -> Octreotide

Octreotide suppresses GH secretion from pituitary adenoma and lowers IGF-1 levels. It is the preferred medical therapy when surgery is inadequate. GnRH analogues like leuprolide/goserelin/nafarelin have no role in GH-secreting tumors.

Situation	Best Drug
Residual acromegaly after surgery	□ Octreotide
Refractory to somatostatin analogues	Pegvisomant
Mild disease	Cabergoline

QUESTION

A female patient with infertility presents with galactorrhea. Evaluation reveals a 7mm prolactinoma. She is worried about her conception chances. What is the appropriate drug in this situation?

A **Cabergoline** CORRECT

B Bromocriptine

C Octreotide

D Pegvisomant

RIGHT ANSWER

OK

A. Cabergoline

WHY THIS IS RIGHT

Cabergoline is the preferred first-line therapy for prolactinoma because it is a long-acting dopamine agonist that effectively suppresses prolactin and shrinks tumor size.
Cabergoline = dopamine (D2) agonist
Mechanism:
Stimulates dopamine receptors in pituitary
□
Inhibits prolactin secretion
□
↓ Prolactin levels
□
Restores ovulation
□
Improves fertility
Normalization of prolactin restores ovulation and improves fertility. It is better tolerated and more effective than bromocriptine.

Feature	Cabergoline	Bromocriptine
Potency	Higher	Lower
Duration	Long acting	Short acting
Side effects	Fewer	More
Compliance	Better	Poor

Octreotide and pegvisomant act on GH, not prolactin.

QUESTION

A patient came to the medical OPD with complaints of polyuria. He has a history of undergoing total hypophysectomy. His Na+ levels are found to be 155 mEq/L, urine osmolality was 200 mOsm/L. What is the definitive management?

A DDAVP for 2 weeks and then discontinue

B **DDAVP supplementation for lifelong** CORRECT

C Upsetting of receptors so no treatment is required

D Thiazides for 2 weeks

RIGHT ANSWER

OK

B. DDAVP supplementation for lifelong

WHY THIS IS RIGHT

Total hypophysectomy causes central diabetes insipidus due to permanent ADH deficiency.
ADH normally acts on kidney collecting ducts
□
Inserts aquaporin channels
□
Water reabsorption
□
Concentrated urine

Parameter	Finding	Meaning
Serum Na□	155	Water loss (hypernatremia)
Urine osmolality	200	Dilute urine
History	Hypophysectomy	Central cause

Hypernatremia with low urine osmolality confirms inability to concentrate urine. Definitive therapy is lifelong desmopressin (DDAVP) replacement to restore water reabsorption.
Temporary therapy or thiazides are used in nephrogenic DI, not central DI.

Feature	Central DI	Nephrogenic DI
ADH	↓	Normal
Cause	Pituitary damage	Kidney resistance
Treatment	□ Desmopressin	Thiazides
Response to ADH	Yes	No

QUESTION

A 45-year-old patient presents with large, sweaty hands, macroglossia, and prominent frontal bossing. The patient also reports increasing shoe size and difficulty wearing rings that previously fit. Considering the clinical features, which of the following tests is the most appropriate initial investigation?

A GHRH levels

B **Age-matched IGF-1 levels** CORRECT

C Random GH levels

D GH levels after 2 hours of administration of 75g glucose

OK RIGHT ANSWER

B. Age-matched IGF-1 levels

WHY THIS IS RIGHT

Growth hormone (GH) is secreted in pulses
Levels fluctuate throughout the day
Random GH measurement becomes unreliable

GH acts on liver
Liver produces IGF-1
IGF-1 levels remain stable
Reflect overall GH activity
The features suggest acromegaly, and the best initial screening test is age-adjusted IGF-1 because it reflects integrated GH secretion and is persistently elevated.
Random GH fluctuates due to pulsatile release and is unreliable. Failure of GH suppression after oral glucose is confirmatory, not the first step. GHRH levels are not used for routine screening.
GH after glucose (OGTT suppression test)
This is a confirmatory test, not initial.
In normal:
Glucose
GH suppressed
In acromegaly:
GH remains high

QUESTION

Which of the following growth hormone analogue that is not used for treatment of GH deficiency but is used in the treatment of HIV-associated lipodystrophy?

A **Tesamorelin** CORRECT

B Sermorelin

C Somatropin

D Pegvisomant

OK RIGHT ANSWER

A. Tesamorelin

WHY THIS IS RIGHT

Tesamorelin is a GHRH analogue used to reduce visceral adiposity in HIV-associated lipodystrophy by stimulating endogenous GH release. It is not used for routine treatment of GH deficiency.

Drug	Type	Main Use
Somatropin	Recombinant GH	□ GH deficiency
Sermorelin	GHRH analogue	GH deficiency (stimulates GH)
Pegvisomant	GH receptor antagonist	Acromegaly
Tesamorelin □	GHRH analogue	HIV lipodystrophy

Somatropin is recombinant GH for deficiency states, while pegvisomant is a GH receptor antagonist used in acromegaly. Sermorelin is used diagnostically or for GH deficiency, not lipodystrophy.

QUESTION

A 14-year-old developed rapid growth and had long legs and large hands, and coarse facial features. Which of the following tests are used to make a confirmatory diagnosis?

A Increased GH levels

B Increased IGF1

C **No supression of GH after glucose administration** CORRECT

D No supression of IGF1 after glucose tolerance test

RIGHT ANSWER

OK

C. No supression of GH after glucose administration

WHY THIS IS RIGHT

The confirmatory test for suspected gigantism/acromegaly is failure of GH suppression after oral glucose load. Normally, hyperglycemia suppresses GH to very low levels; persistent elevation confirms autonomous GH secretion.

Test	Role	Why
IGF-1	Screening	Stable, reflects GH
OGTT (GH suppression test) □	Confirmatory	Tests feedback control
Random GH	Not useful	Fluctuates

Random GH levels fluctuate and are unreliable, while IGF-1 is a screening marker, not confirmatory. IGF-1 is not used for suppression testing.

Feature	Gigantism	Acromegaly
Age	Children	Adults
Epiphysis	Open	Closed
Growth	Height ↑	No height ↑
Features	Long limbs	Coarse features

QUESTION

In a patient with a prolactinoma, the mechanism by which prolactin causes amenorrhea is through which of the following actions?

A Downregulation of estrogen receptors

B **Decreasing GnRH** CORRECT

C Decreasing FSH

D Increasing pulse frequency of LH

RIGHT ANSWER

OK **B. Decreasing GnRH**

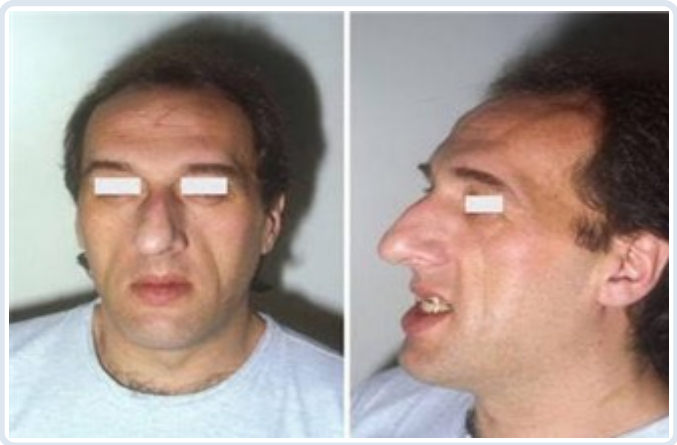
WHY THIS IS RIGHT

↑ Prolactin
□
↓ GnRH (from hypothalamus)
□
↓ LH and ↓ FSH (from pituitary)
□
↓ Ovarian estrogen production
□
Anovulation
□
Amenorrhea
Hyperprolactinemia inhibits hypothalamic GnRH secretion, suppressing the pulsatile release required for normal gonadotropin production. Reduced GnRH leads to decreased LH and FSH, causing anovulation and amenorrhea. This central inhibition is the primary mechanism in prolactinoma. Effects on estrogen receptors are secondary and not the initiating defect.

Parameter	Normal	Prolactinoma
GnRH	Normal	↓
LH/FSH	Normal	↓
Ovulation	Present	Absent
Menstruation	Regular	Absent

QUESTION

These findings are suggestive of which of the following conditions?



A Addison's disease

B Gigantism

C **Acromegaly** CORRECT

D Cushing's syndrome

RIGHT ANSWER

OK **C. Acromegaly**

WHY THIS IS RIGHT

Acromegaly results from excess growth hormone after epiphyseal closure, leading to soft tissue overgrowth and enlarged hands, feet, jaw, and facial bones. Classic features include coarse facial features, prognathism, and organomegaly. Gigantism occurs before epiphyseal closure and presents with tall stature rather than acral enlargement.

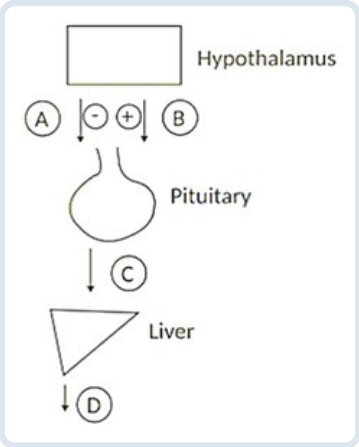
Feature	Gigantism	Acromegaly
Age	Children	Adults
Epiphysis	Open	Closed
Height	↑	No increase
Features	Long limbs	Coarse features □

System	Feature
Face	Prognathism, frontal bossing
Extremities	Large hands/feet
Tongue	Macroglossia
Skin	Thick, oily
Metabolic	Insulin resistance

Addison’s and Cushing’s do not produce bony overgrowth or acral hypertrophy.

QUESTION

Identify the hormones represented by the following:



A. A - Somatostatin B - GH C - GHRH D - IGF1

B. **A - Somatostatin B - GHRH C - GH D - IGF1** CORRECT

C. A - IGF1 B - GHRH C - GH D - Somatostatin

D. A - GHRH B - Somatostatin C - GH D - IGF1

OK

RIGHT ANSWER

B. A - Somatostatin B - GHRH C - GH D - IGF1

WHY THIS IS RIGHT

Correct answer is B because hypothalamus releases inhibitory somatostatin (A) and stimulatory GHRH (B) to regulate pituitary secretion.
The pituitary then releases growth hormone (C), which acts on the liver.
The liver produces IGF-1 (D), mediating peripheral growth effects and feedback control.

QUESTION

Many GPCR receptors act via an increase in cAMP. The steps involved in this mechanism are given below. Which of the following is the correct sequence of events in the cAMP signal transduction pathway?

1. Activation of adenylate cyclase
2. Activation of PKA
3. Conversion of ATP to cAMP
4. Phosphorylation of target proteins

A 1, 2, 3 and 4

B **1, 3, 2 and 4** CORRECT

C 1, 3, 4 and 2

D 4, 2, 1 and 3

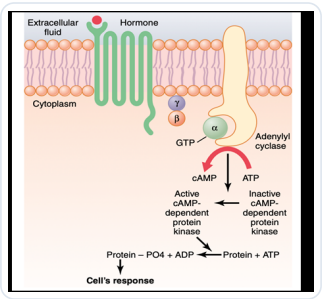
RIGHT ANSWER

OK

B. 1, 3, 2 and 4

WHY THIS IS RIGHT

Correct answer is B because GPCR activation first stimulates adenylate cyclase (1), which converts ATP to cAMP (3). cAMP then activates protein kinase A (PKA) (2).
Activated PKA phosphorylates downstream target proteins (4), producing the cellular response.



QUESTION

A 30-year-old woman, having lost her newborn, is producing breast milk, which risks developing into a breast abscess due to milk stasis and incomplete emptying. Which drug can prevent this complication?

A Mifepristone

B **Cabergoline** CORRECT

C Chlorpromazine

D Metoclopramide

RIGHT ANSWER

OK

B. Cabergoline

WHY THIS IS RIGHT

Cabergoline is a dopamine (D2) agonist that:
□ Inhibits prolactin secretion
□ Suppresses lactation

Correct answer is B because cabergoline is a dopamine agonist that suppresses prolactin secretion, thereby inhibiting lactation and preventing milk stasis.
Reducing prolactin stops continued milk production and lowers risk of engorgement and abscess.
Metoclopramide and chlorpromazine increase prolactin. Mifepristone has no role in lactation suppression.

Drug	Effect on Prolactin
Cabergoline / Bromocriptine	↓ prolactin
Antipsychotics	↑ prolactin
Metoclopramide	↑ prolactin

QUESTION

Match the Following hormones to receptors:

Column A	Column B
1. Insulin	A. Tyrosine Kinase Pathway
2. Growth Hormone	B. JAK STAT pathway
3. Acetylcholine	C. Ligand gated channel
4. Levothyroxine	D. Nuclear Receptor

A 1-C, 2-B, 3-D, 4-A

B 1-D, 2-A, 3-C, 4-B

C **1-A, 2-B, 3-C, 4-D** CORRECT

D 1-B, 2-D, 3-A, 4-C

OK

RIGHT ANSWER

C. 1-A, 2-B, 3-C, 4-D

WHY THIS IS RIGHT

1. Insulin - Tyrosine Kinase Pathway: Insulin uses a tyrosine kinase receptor. This is a cell surface receptor that, when activated, phosphorylates tyrosine residues on target proteins, triggering intracellular signaling cascades.
2. Growth Hormone - JAK-STAT Pathway: Growth hormone, along with prolactin and leptin, uses the JAK-STAT (Janus Kinase-Signal Transducer and Activator of Transcription) pathway. This is a cytokine receptor-mediated signaling mechanism.
3. Acetylcholine - Ligand-gated Channel: At the neuromuscular junction, acetylcholine operates through ligand-gated sodium channels. When acetylcholine binds, these channels open, allowing sodium ions to flow and triggering muscle contraction.
4. Levothyroxine (Thyroid Hormone) - Nuclear Receptor: Thyroid hormones act through nuclear receptors, which means they enter the cell and directly interact with DNA in the nucleus to regulate gene expression.

QUESTION

IGF-1 and IGF-2 (Insulin-like Growth Factors) are structurally most similar to which of the following molecules?

A Insulin

B **Proinsulin** CORRECT

C C peptide

D Preproinsulin

RIGHT ANSWER

OK **B. Proinsulin**

WHY THIS IS RIGHT

Stepwise synthesis

Stage	Structure
Preproinsulin	signal peptide + A + B + C
Proinsulin	A chain + B chain + C peptide
Insulin	A chain + B chain only

During processing:
• C-peptide is removed
• A and B chains remain linked by disulfide bonds

Correct answer is B because IGF-1 and IGF-2 retain structural similarity to proinsulin, including homologous A and B chains connected by a C-peptide-like domain.
Unlike insulin, IGFs are not cleaved to remove the connecting peptide, so their tertiary structure resembles intact proinsulin.
C-peptide alone and preproinsulin do not share the same functional folded structure as IGFs.

QUESTION

53-year-old man comes to the physician complaining of increased urinary frequency for the past 4 weeks. He has no burning with urination, urgency, or difficulty initiating urination. He wakes up several times at night to urinate. His mouth feels dry all the time and he drinks fluids almost every hour to alleviate his thirst. The patient's medications include hydrochlorothiazide and amlodipine for hypertension. His sister was diagnosed with diabetes mellitus at a young age. His blood pressure is 120/76 mm Hg, pulse is 85/min, and respirations are 15/min. His body mass index is 32 kg/m². Laboratory results are as follows:
Blood glucose-93 mg/dL
Serum sodium-150 mEg/L
Serum potassium-4.1 mEq/L
Bicarbonate-24 mEq/L
Blood urea nitrogen-21 mg/dL
Serum creatinine-1.1 mg/dL
Serum uric acid-10.1 mg/dL (N- 3.5-7.2 mg/dL)
Serum osmolality-314 mOsm/kg (N- 275 to 295 mOsm/kg)
Urine osmolality-124 mOsm/kg (N-500 - 800 mOsm/kg)
Which of the following is most consistent with this patient's findings?

A **Central Diabetes insipidus** — CORRECT

B Nephrogenic DI

C Primary polydipsia

D Syndrome of inappropriate antidiuretic hormone

OK RIGHT ANSWER

A. Central Diabetes insipidus

WHY THIS IS RIGHT

Central diabetes insipidus is caused by deficient secretion of antidiuretic hormone (ADH) from the posterior pituitary, leading to impaired water reabsorption in the collecting ducts.

Feature	Central Diabetes Insipidus	Nephrogenic Diabetes Insipidus	Primary Polydipsia	SIADH
Primary problem	↓ ADH secretion (posterior pituitary)	Kidney resistant to ADH	Excess water intake	Excess ADH secretion
Serum sodium	↑ (hypernatremia)	↑ (hypernatremia)	↓ or normal	↓ (hyponatremia)
Serum osmolality	↑	↑	↓	↓
Urine osmolality	↓ (dilute urine)	↓ (dilute urine)	↓	↑ (concentrated urine)
Urine volume	↑↑ (polyuria)	↑↑ (polyuria)	↑	↓
Response to desmopressin	Urine osmolality ↑	No response	Minimal change	Not used
Common causes	Head trauma, pituitary surgery, tumors	Lithium, hypercalcemia, CKD	Psychiatric disorders, psychogenic polydipsia	CNS disease, lung disease, drugs

QUESTION

Which of the following pairs of drugs and its indications is matched incorrectly?

A **Bromocriptine - Treatment of precocious puberty** CORRECT

B Octreotide - Treatment of diarrhea associated with vasoactive intestinal peptide tumors.

C Desmopressin - Treatment of diabetes insipidus

D Leuprolide - Treatment of infertility

RIGHT ANSWER

OK **A. Bromocriptine - Treatment of precocious puberty**

WHY THIS IS RIGHT

Bromocriptine is a dopamine (D2) agonist that inhibits prolactin secretion from the anterior pituitary. It is primarily used in the treatment of hyperprolactinemia, prolactin-secreting pituitary adenomas (prolactinomas), acromegaly, and Parkinson disease. It is not used for precocious puberty, making this pair incorrect.

Correct drug-indication pairs:
• Octreotide -> used for secretory diarrhea in VIPoma and also in acromegaly.
• Desmopressin -> used for central diabetes insipidus and nocturnal enuresis.
• Leuprolide -> a GnRH agonist used in infertility (controlled ovarian stimulation), prostate cancer, endometriosis, and precocious puberty.

QUESTION

Two days after vaginal delivery of a healthy newborn at term, a 32-year-old woman, gravida 2, para 2, is unable to breastfeed. Her labor was complicated by antepartum hemorrhage and she received two units of packed red blood cells. Her pulse is 99/min and blood pressure is 90/55 mm Hg. Further evaluation of this patient is most likely to show which of the following sets of serum findings?

A ACTH low, Aldosterone low, Cortisol low

B **ACTH low, Aldosterone high, Cortisol low** CORRECT

C ACTH high, Aldosterone normal, Cortisol low

D ACTH low, Aldosterone normal, Cortisol low

OK RIGHT ANSWER

B. ACTH low, Aldosterone high, Cortisol low

WHY THIS IS RIGHT

Severe postpartum hemorrhage can cause ischemic necrosis of the anterior pituitary, leading to Sheehan syndrome. This results in hypopituitarism, commonly presenting with failure to lactate due to prolactin deficiency, hypotension, and fatigue after delivery.
Hormonal pattern in Sheehan syndrome:
• ↓ ACTH -> leads to ↓ cortisol (secondary adrenal insufficiency)
• Aldosterone remains increased because it is mainly regulated by the renin-angiotensin-aldosterone system, not by ACTH.

Typical findings:
• ↓ ACTH
• ↓ Cortisol
• ↑ Aldosterone

QUESTION

A 50-year-old man comes to the OPD following transsphenoidal resection of a growth hormone secreting pituitary adenoma. Three months ago, the patient was diagnosed with acromegaly. MRI of the pituitary showed a 14 mm sellar mass pressing on the optic chiasm and extending into the right cavernous sinus. The surgeon was able to only partially resect the pituitary mass because of the extension into the right cavernous sinus. Medical therapy for acromegaly with octreotide is planned. Which of the following changes are likely to occur following octreotide treatment in this patient?

A **GH low, IGF-1 low, adenoma size low** — CORRECT

B GH low, IGF-1 low, adenoma size unchanged

C GH low, IGF-1 unchanged, adenoma size low

D GH unchanged, IGF-1 low, adenoma size unchanged

OK RIGHT ANSWER

A. GH low, IGF-1 low, adenoma size low

WHY THIS IS RIGHT

Octreotide, a somatostatin analogue, is used for medical treatment of acromegaly, especially when pituitary adenoma cannot be completely resected.
Mechanism and effects:
• Inhibits growth hormone (GH) secretion from pituitary somatotroph cells
• Leads to decreased hepatic production of IGF-1
• Has an antiproliferative effect on the pituitary adenoma, often causing tumor shrinkage

Result after treatment:
• ↓ GH
• ↓ IGF-1
• ↓ Adenoma size

QUESTION

A 6-year-old girl is brought to the OPD for evaluation of short stature. Her parents have noticed that she bears little resemblance to her 2 older siblings. Other findings include low-set ears, a high arched palate, a webbed neck, and cubitus valgus. Chromosomal analysis reveals a 45, XO karyotype. The patient is started on a medication to improve growth and normalize her height. Which of the following intracellular pathways is stimulated by the medication used in this patient?

A Binding of activated receptors to DNA to modify transcription.

B Cyclic AMP-Protein kinase A pathway

C Diacyglycerol-Protein kinase C pathway

D **JAK-STAT pathway** CORRECT

OK RIGHT ANSWER

D. JAK-STAT pathway

WHY THIS IS RIGHT

Turner syndrome (45,XO) commonly presents with short stature, webbed neck, low-set ears, high-arched palate, and cubitus valgus. Treatment for short stature is recombinant growth hormone (GH).

Growth hormone acts via the JAK-STAT signaling pathway:
• GH binds to cytokine receptor-type GH receptors
• Activates Janus kinase (JAK)
• JAK phosphorylates STAT proteins
• STATs enter the nucleus and regulate gene transcription, promoting growth (mainly via IGF-1).

QUESTION

A 32-year-old woman comes to the physician because of a 3-month history of irregular menses, milky discharge from her nipples, fatigue, and weight gain. Menses occur at irregular 25-40-day intervals and last 1-2 days with minimal flow. 5 months ago, she was started on clozapine for treatment of schizophrenia. She has hypothyroidism but has not been taking levothyroxine over the past 6 months. Visual field examination show no abnormalities. Her serum thyroid-stimulating hormone is 17.0 U/mL and serum prolactin is 85 ng/mL. Which of the following is the most likely explanation for the nipple discharge in this patient?

A **Hypothyroidism** CORRECT

B Prolactinoma

C Ectopic prolactin production

D Adverse effect of medication

RIGHT ANSWER

OK **A. Hypothyroidism**

WHY THIS IS RIGHT

Untreated primary hypothyroidism can cause hyperprolactinemia and galactorrhea due to increased thyrotropin-releasing hormone (TRH) from the hypothalamus.
• In primary hypothyroidism, decreased thyroid hormone leads to increased TRH secretion.
• TRH stimulates both TSH and prolactin release from the anterior pituitary.
• Elevated prolactin suppresses GnRH, resulting in menstrual irregularities, oligomenorrhea, and galactorrhea.

Why other options are incorrect:

B. Prolactinoma: Usually causes very high prolactin (>200 ng/mL) and often visual field defects. This patient has moderate elevation (85 ng/mL) and normal visual fields.
C. Ectopic prolactin production: Extremely rare cause of hyperprolactinemia.
D. Adverse effect of medication: Antipsychotics can increase prolactin, but clozapine has minimal prolactin-elevating effect.

QUESTION

A 28-year-old woman comes to the OPD due to a 4-month history of amenorrhea. She has also had a whitish nipple discharge from both breasts. The patient has taken several pregnancy tests at home that have been negative. She has also had increased fatigue, depressed mood, and weight gain over this time. The patient has had no headaches or vision changes. On physical examination, there is thinning of the outer third of the eyebrows. The thyroid is enlarged and nontender to palpation. The skin appears dry. Her thyroxine (T4) levels are low, TSH is elevated, and antithyroid peroxidase antibodies are positive. Prolactin levels are 30 ng/ml. Which of the following is the most likely mechanism causing this patient's elevated prolactin level?

A Activation of lactotrophs by antithyroid peroxidase antibodies

B Binding of dopamine receptors by antithyroid peroxidase antibodies

C Inhibition of dopamine release by TSH

D **Stimulation of lactotrophs by thyrotropin-releasing hormone (TRH)** CORRECT

RIGHT ANSWER

OK **D. Stimulation of lactotrophs by thyrotropin-releasing hormone (TRH)**

WHY THIS IS RIGHT

Primary hypothyroidism (e.g., autoimmune thyroiditis), low T4 leads to increased hypothalamic thyrotropin-releasing hormone (TRH) to stimulate the pituitary.

TRH has two actions on the anterior pituitary:
• ↑ TSH secretion
• ↑ Prolactin secretion by stimulating lactotrophs

This results in mild hyperprolactinemia, which can cause:
• Amenorrhea
• Galactorrhea

QUESTION

Which of the following hormone-receptor interactions acts via the IP□-DAG second messenger system?

A **Angiotensin II on vascular smooth muscle and vasopressin acting via V1 receptors on vascular smooth muscle** CORRECT

B Angiotensin II on vascular smooth muscle and vasopressin acting via V2 receptors on epithelial cells

C Angiotensin II acting on epithelial cells and vasopressin acting via V1 receptors on vascular smooth muscle

D Angiotensin II acting on epithelial cells and vasopressin acting via V2 receptors on epithelial cells

RIGHT ANSWER

OK **A. Angiotensin II on vascular smooth muscle and vasopressin acting via V1 receptors on vascular smooth muscle**

WHY THIS IS RIGHT

Hormones that act through Gq protein-coupled receptors activate phospholipase C (PLC), which generates the second messengers IP□ (inositol triphosphate) and DAG (diacylglycerol).
• IP□ -> releases Ca^{2}□ from the endoplasmic reticulum
• DAG -> activates protein kinase C

Key hormones using the IP□-DAG pathway:
• Angiotensin II (AT□ receptors) on vascular smooth muscle -> vasoconstriction
• Vasopressin (ADH) acting on V1 receptors on vascular smooth muscle -> vasoconstriction

Important contrast:
• V2 receptors of vasopressin act via Gs -> cAMP, causing water reabsorption in renal collecting ducts.

QUESTION

SIADH is not observed in which of the following conditions?

A Acute attack of porphyria

B Oat cell carcinoma of lung

C **Primary pulmonary emphysema** CORRECT

D Multiple sclerosis

RIGHT ANSWER

OK

C. Primary pulmonary emphysema

WHY THIS IS RIGHT

Syndrome of Inappropriate Antidiuretic Hormone secretion (SIADH) is characterized by excess ADH release, leading to water retention, dilutional hyponatremia, decreased serum osmolality, and concentrated urine.

Common causes of SIADH include:
• Malignancies: especially small cell (oat cell) carcinoma of lung producing ectopic ADH
• Neurological disorders: e.g., multiple sclerosis, head injury, meningitis
• Certain metabolic disorders: e.g., acute intermittent porphyria
• Drugs and pulmonary diseases (pneumonia, tuberculosis)

Primary pulmonary emphysema is not typically associated with SIADH, making it the incorrect option.

QUESTION

A 60-year-old man presents with progressive increase in the size of hands and feet, coarsening of facial features, deepening of voice, and tingling sensations in both hands suggestive of carpal tunnel syndrome. On evaluation, his serum IGF-1 level is elevated.
Which of the following findings is not expected in this patient?

A **Hypoglycemic attacks** CORRECT

B Galactorrhea

C Bitemporal hemianopia

D Colonic polyps

OK RIGHT ANSWER

A. Hypoglycemic attacks

WHY THIS IS RIGHT

Acromegaly results from excess growth hormone (GH), usually due to a pituitary somatotroph adenoma, leading to elevated IGF-1 levels. GH has anti-insulin effects, causing insulin resistance and hyperglycemia, so hypoglycemic attacks are not expected.

Common associated findings in acromegaly:
• Galactorrhea -> due to co-secretion of prolactin or pituitary stalk compression
• Bitemporal hemianopia -> compression of the optic chiasm by pituitary macroadenoma
• Colonic polyps and increased colorectal cancer risk
• Carpal tunnel syndrome from soft-tissue overgrowth

QUESTION

A 34-year-old man with adult-onset growth hormone deficiency complains of fatigue, increased fat mass, and reduced exercise capacity. He has poor adherence to daily subcutaneous injections prescribed previously. His endocrinologist switches him to a preparation that allows once-weekly administration by prolonging systemic exposure through reversible binding to circulating proteins. Which of the following drugs is most likely prescribed?

A Somatrem

B Mecasermin

C Pegvisomant

D **Somapacitan** CORRECT

OK

RIGHT ANSWER

D. Somapacitan

WHY THIS IS RIGHT

Somapacitan is a long-acting growth hormone (GH) analog used for adult growth hormone deficiency. It is designed for once-weekly subcutaneous administration because it reversibly binds to circulating albumin, which prolongs its systemic half-life and maintains sustained GH activity.

Why not the others (brief):
• Somatrem: Recombinant GH requiring daily injections.
• Mecasermin: Recombinant IGF-1, used mainly in severe primary IGF-1 deficiency, not GH replacement.
• Pegvisomant: Growth hormone receptor antagonist used to treat acromegaly, not GH deficiency.

QUESTION

A 29-year-old woman with a pituitary microadenoma has been receiving cabergoline 1 mg per week. She presents at 12 weeks of gestation. What is the most appropriate management?

A. Advise termination of pregnancy as cabergoline was continued during the first trimester

B. Follow up with serum prolactin levels monthly

C. Perform MRI of the sella to look for tumor enlargement

D. **Reassure the patient and evaluate further only if symptoms such as headache or visual disturbance develop** CORRECT

RIGHT ANSWER

OK **D. Reassure the patient and evaluate further only if symptoms such as headache or visual disturbance develop**

WHY THIS IS RIGHT

Prolactin-secreting pituitary microadenomas (microprolactinomas) are commonly treated with dopamine agonists such as cabergoline or bromocriptine, which suppress prolactin secretion and shrink the tumor.

Management during pregnancy:
• Once pregnancy is confirmed, dopamine agonists are usually discontinued in patients with microadenomas.
• The risk of tumor enlargement during pregnancy is very low (\~2-3%) for microadenomas.
• Routine MRI or prolactin monitoring is not recommended, because prolactin levels naturally rise during pregnancy and do not correlate with tumor size.

Recommended approach
• Reassure the patient
• Clinical monitoring only
• Investigate further only if symptoms of tumor expansion appear, such as:

o Headache
o Visual field defects

QUESTION

A 70-year-old man with a long-standing medical history is on multiple medications. He has developed excessive bilateral breast tissue enlargement. Which of the following drugs is most likely responsible for this condition?

A Tamoxifen

B Terbinafine

C Amiodarone

D **Goserelin** CORRECT

RIGHT ANSWER

OK

D. Goserelin

WHY THIS IS RIGHT

Gynecomastia (benign enlargement of male breast tissue) occurs due to an imbalance between estrogen and androgen activity, often caused by medications that reduce androgen levels or increase estrogenic effects.

Goserelin is a GnRH agonist used in conditions such as prostate cancer. With continuous administration, it suppresses LH and FSH secretion, leading to decreased testosterone production. The resulting relative increase in estrogen effect can cause gynecomastia and breast tenderness.

Why not other options:
• Tamoxifen - a selective estrogen receptor modulator (SERM) that is actually used to treat gynecomastia.
• Terbinafine - antifungal; gynecomastia is not a typical adverse effect.
• Amiodarone - mainly causes thyroid dysfunction, pulmonary fibrosis, hepatotoxicity, and corneal deposits, not gynecomastia.

QUESTION

An elderly male patient presents with a cough and weight loss for the last 3 months. Biopsy of the lung mass revealed tumor cells that are positive for chromogranin and synaptophysin. Which of the following paraneoplastic syndromes will most likely occur in this patient?

A Hypercalcemia

B Cushing syndrome

C Gynaecomastia

D **SIADH** CORRECT

OK

RIGHT ANSWER

D. SIADH

WHY THIS IS RIGHT

The source quiz contains the placeholder “$19” here and does not include a written explanation for this item.

QUESTION

A 30-year-old patient presents with hypertension and hypokalemic metabolic alkalosis. CT scan shows a mass of 1.5 cm over the kidney with elevated ARR. What is the next best step for the management of this patient?

A Spironolactone on life-long basis

B **Adrenalectomy** CORRECT

C ACTH stimulation test

D Dexamethasone suppression test

RIGHT ANSWER

OK **B. Adrenalectomy**

WHY THIS IS RIGHT

Once primary hyperaldosteronism is confirmed + localized adenoma, the treatment is: Surgical removal (adrenalectomy)
Aldosterone acts on kidney:

□
↑ Na□ reabsorption
□
↑ Water retention
□
Hypertension
□
↑ K□ excretion
□
Hypokalemia
□
↑ H□ excretion
□
Metabolic alkalosis

Hypertension with hypokalemic metabolic alkalosis and elevated aldosterone-renin ratio indicates primary hyperaldosteronism. A unilateral adrenal mass consistent with an aldosterone-producing adenoma is treated definitively with adrenalectomy. Surgery corrects hypertension and hypokalemia.
Lifelong spironolactone is reserved for bilateral adrenal hyperplasia or non-surgical candidates.

QUESTION

What is the likely diagnosis of a middle-aged patient presented with episodic headache, palpitations and increased 24-hour urinary levels of metanephrines?

A Neuroblastoma

B Carcinoid tumor

C Brain tumor

D **Pheochromocytoma** CORRECT

OK RIGHT ANSWER

D. Pheochromocytoma

WHY THIS IS RIGHT

Symptoms suggest: Intermittent sympathetic overactivity
Classic triad:
• Headache
• Palpitations
• Sweating

Lab clue:
• ↑ Metanephrines (very specific)

Diagnosis: Pheochromocytoma
Episodic headache, palpitations, and elevated urinary metanephrines are classic for pheochromocytoma. Excess catecholamine secretion produces paroxysmal sympathetic surges and hypertension. Urinary metanephrines are sensitive markers of catecholamine excess.
The other tumors do not produce this characteristic biochemical pattern.

Neuroblastoma
• Occurs in children
• Not typical in adults

Carcinoid tumor
• Causes flushing and diarrhea
• Due to serotonin secretion, not catecholamines

Brain tumor
• May cause headache
• Does not explain palpitations or elevated metanephrines

QUESTION

Which drug is used in the preoperative management of pheochromocytoma?

A **Phenoxybenzamine** CORRECT

B Propranolol

C Metoprolol

D Labetalol

OK RIGHT ANSWER

A. Phenoxybenzamine

WHY THIS IS RIGHT

First step is irreversible alpha-blockade with phenoxybenzamine to prevent hypertensive crisis.

Alpha blockers are given first-

Action	Result
Blocks α1 receptors	Vasodilation
Blocks α2 receptors	Reduced catecholamine effect
Prevents hypertensive crisis	During tumor manipulation

If a beta blocker is given before alpha blockade: β2 vasodilation is blocked, α-mediated vasoconstriction becomes unopposed which results in Severe hypertensive crisis
Beta-blockers are added only after adequate alpha blockade.

QUESTION

A 34-year-old patient presents with lethargy and tiredness. On examination, he has hyperpigmentation around the lips. His BP is 90/60 mm Hg and heart rate is 110/min. Labs revealed a Na□ of 125 mEq/L and a K□ of 5.5 mEq/L. What is the most appropriate treatment?

A ACTH

B **Hydrocortisone and fludrocortisone** CORRECT

C Dexamethasone

D NaCl infusion

RIGHT ANSWER

OK

B. Hydrocortisone and fludrocortisone

WHY THIS IS RIGHT

Finding	Meaning
Hyperpigmentation	↑ ACTH -> primary cause
Hypotension	cortisol deficiency
Hyponatremia	aldosterone deficiency
Hyperkalemia	aldosterone deficiency

Findings suggest primary adrenal insufficiency with cortisol and aldosterone deficiency causing hypotension, hyponatremia, hyperkalemia, and hyperpigmentation. Treatment requires glucocorticoid plus mineralocorticoid replacement.
GLUCOCORTICOID:
Hydrocortisone -> 15-25 mg/day (divided doses)
• 10 mg morning + 5 mg afternoon (± 5 mg evening)

MINERALOCORTICOID:
Fludrocortisone -> 0.05-0.2 mg/day (usually 0.1 mg once daily)

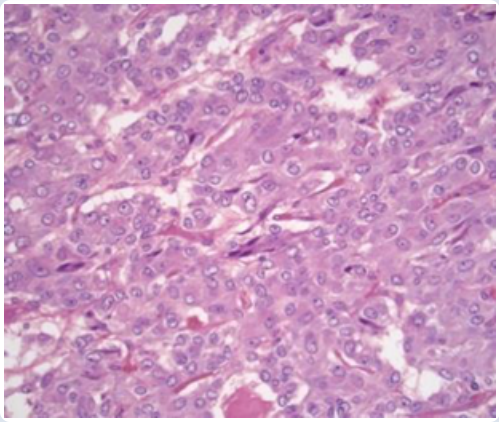
GOALS:
• Maintain BP, electrolytes (Na↑, K↓)
• Avoid hypotension & hyperkalemia

STRESS DOSE (VERY IMPORTANT):
• Increase hydrocortisone 2-3× during illness/surgery

ACUTE CRISIS:
• IV Hydrocortisone 100 mg bolus -> then 200 mg/day

QUESTION

A 55-year-old male presented with headache, palpitations, and hypertension. On evaluation, his urinary VMA and catecholamines were elevated. Imaging revealed an adrenal mass, and the patient underwent surgery and resection. Histology of the mass was shown. Which of the following is true regarding the condition?



A Mostly in children

B Mostly malignant

C **Can be seen in MEN 2A** CORRECT

D Mostly bilateral

OK RIGHT ANSWER

C. Can be seen in MEN 2A

WHY THIS IS RIGHT

Elevated urinary VMA with adrenal mass indicates pheochromocytoma, a catecholamine-secreting tumor.
Tumor of chromaffin cells (adrenal medulla)

Excess catecholamine secretion

• Vasoconstriction -> hypertension
• Cardiac stimulation -> palpitations

Episodic symptoms

Pheochromocytoma is strongly associated with:

Syndrome	Components
MEN 2A	Medullary thyroid carcinoma + pheochromocytoma + parathyroid hyperplasia
MEN 2B	Medullary thyroid carcinoma + pheochromocytoma + mucosal neuromas

Hence option C is correct
It is classically associated with MEN 2A due to RET mutation along with medullary thyroid carcinoma and hyperparathyroidism. Most pheochromocytomas are benign, unilateral, and occur in adults. Bilaterality is mainly seen in familial syndromes, not the majority.

QUESTION

A 21-year-old patient presented with weakness and fatigue. His BP was 80/60 mm Hg and his reports showed a serum Na+ of 127 mEq/L and serum K+ of 5.2 mEq/L. On examination, the patient had hyperpigmentation in the knuckles, arms, and legs. What is the diagnosis?

A Conns syndrome

B **Primary adrenal insufficiency** CORRECT

C B12 deficiency

D SIADH

OK RIGHT ANSWER

B. Primary adrenal insufficiency

WHY THIS IS RIGHT

Hypotension with hyponatremia, hyperkalemia, and diffuse hyperpigmentation is classic for primary adrenal insufficiency. Loss of aldosterone causes salt wasting and hyperkalemia, while cortisol deficiency raises ACTH, leading to pigmentation. This electrolyte pattern excludes SIADH, which has no hyperkalemia. Conn syndrome causes hypertension and hypokalemia, the opposite profile.

Condition	Na□	K□	BP	Pigmentation
Addison's	↓	↑	↓	□ Present
Conn's syndrome	↑	↓	↑	Absent
SIADH	↓	Normal	Normal	Absent
B12 deficiency	Normal	Normal	Normal	May have pigmentation but no electrolyte changes

- Hyperpigmentation = primary adrenal failure □
- Hyponatremia + hyperkalemia = aldosterone deficiency
- Hypotension = cortisol deficiency
- Most common cause = autoimmune

QUESTION

Which of the following is the correct statement about a patient with a retroperitoneal mass, episodic hypertension, palpitations, and increased urinary VMA levels?

A It is mostly in children

B It is usually malignant

C It is mostly bilateral

D **It can be associated with MEN 2A** CORRECT

OK RIGHT ANSWER

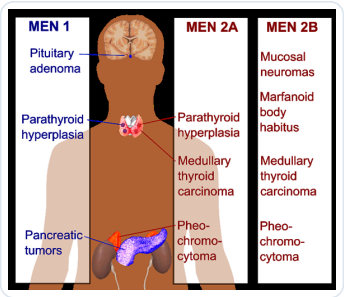
D. It can be associated with MEN 2A

WHY THIS IS RIGHT

Syndrome	Components
MEN 2A □	Medullary thyroid carcinoma + pheochromocytoma + parathyroid hyperplasia
MEN 2B	Medullary thyroid carcinoma + pheochromocytoma + mucosal neuromas

The presentation suggests pheochromocytoma, a catecholamine-secreting tumor classically causing episodic hypertension and elevated urinary VMA. Pheochromocytoma is associated with MEN 2A due to RET mutation along with medullary thyroid carcinoma and hyperparathyroidism.

Most tumors are benign, unilateral, and occur in adults. Bilateral tumors are mainly seen in familial syndromes.



QUESTION

A patient with hypertensive episodes, with an MRI abdomen showing a suprarenal mass, is planned for surgery. A 24-hour urine shows elevated metanephrine levels. What drug will be given preoperatively and also intraoperatively?

A Nicardipine

B Esmolol

C Clonidine

D **Phenoxybenzamine** CORRECT

RIGHT ANSWER

OK

D. Phenoxybenzamine

WHY THIS IS RIGHT

Phenoxybenzamine is a nonselective irreversible α-blocker used preoperatively in pheochromocytoma to prevent catecholamine-induced hypertensive crises.
Phenoxybenzamine = irreversible α-blocker
Blocks α□ and α□ receptors
Causes vasodilation

Preoperative
• Classically Phenoxybenzamine (α-blocker) -> given days before surgery

Intraoperative BP control
• Need short-acting, titratable drug
• Nicardipine (IV CCB) is preferred:
• Rapid onset
• Easily adjustable
• Controls hypertensive surges during tumor manipulation

Question asks: “preoperative AND intraoperative”
• Among options, Phenoxybenzamine seems most appropriate due to its traditional use-case in pheochromocytoma.

QUESTION

A 20-year-old patient presented with salt craving, skin pigmentation, hyponatremia, hyperkalemia, and metabolic acidosis. What is the likely diagnosis?

A Cushing syndrome

B **Addison's disease** CORRECT

C Pheochromocytoma

D Conn syndrome

RIGHT ANSWER

OK **B. Addison's disease**

WHY THIS IS RIGHT

Finding	What it indicates
Salt craving	Aldosterone deficiency
Hyperpigmentation	↑ ACTH -> primary cause
Hyponatremia	↓ aldosterone
Hyperkalemia	↓ aldosterone
Metabolic acidosis	↓ H□ excretion

Primary adrenal insufficiency causes deficiency of both cortisol and aldosterone, leading to salt wasting, hyponatremia, hyperkalemia, and metabolic acidosis. Elevated ACTH produces characteristic skin hyperpigmentation and salt craving reflects mineralocorticoid loss. This constellation is classic for Addison’s disease. Cushing and Conn syndromes cause hypertension and hypokalemia, not salt loss.

Condition	Na□	K□	BP	Pigmentation
Addison’s	↓	↑	↓	□ Present
Cushing	↑	↓	↑	Absent
Conn syndrome	↑	↓	↑	Absent
Pheochromocytoma	Normal	Normal	↑ (episodic)	Absent

QUESTION

Increase in which of the following hormone is responsible for Hyperpigmentation in Addison's disease?

A **ACTH** CORRECT

B Aldosterone

C ADH

D Cortisol

OK RIGHT ANSWER

A. ACTH

WHY THIS IS RIGHT

↓ Cortisol (adrenal failure)
□
Loss of negative feedback
□
↑ ACTH secretion (from pituitary)
□
ACTH derived from POMC (pro-opiomelanocortin)
□
POMC also produces MSH (melanocyte-stimulating hormone)
□
↑ Melanin production
□
Hyperpigmentation
In Addison disease, low cortisol removes negative feedback on the pituitary, causing marked ACTH elevation. ACTH is derived from POMC, which also produces MSH, stimulating melanocytes and causing diffuse hyperpigmentation.

Component	Derived From	Function
ACTH	POMC	Stimulates adrenal cortex
MSH	POMC	Stimulates melanocytes
Cortisol	Adrenal cortex	Negative feedback

Aldosterone and ADH do not affect skin pigmentation. Cortisol is decreased, not increased, in primary adrenal insufficiency.

QUESTION

A woman previously treated for Cushing disease underwent bilateral adrenalectomy 2 years ago. She now presents with progressive hyperpigmentation, headaches, and new-onset visual field defects. MRI shows an enlarging pituitary mass with markedly elevated ACTH levels. What is the most likely diagnosis?

- A **Nelson syndrome** — CORRECT
- B Sheehan syndrome
- C Conn's syndrome
- D Addison's disease

OK

RIGHT ANSWER

A. Nelson syndrome

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1a” here and does not include a written explanation for this item.

QUESTION

A patient was brought to the emergency department in an unconscious state. The patient is dehydrated and the blood pressure of the patient is 90/60 mm Hg. Laboratory investigations revealed the serum potassium level of 6.0 mEq/L, sodium level of 129 mEq/L, and blood glucose level of 40 mg/dL. The patient has a history of rheumatoid arthritis (RA) for which he was prescribed medication by a local physician; however, the patient abruptly discontinued the medication a few weeks ago. Based on the clinical presentation and laboratory findings, which of the following drugs should be administered as a life-saving measure to treat this patient?

A **Hydrocortisone** CORRECT

B Sodium bicarbonate

C Insulin

D Hypertonic saline

OK RIGHT ANSWER

A. Hydrocortisone

WHY THIS IS RIGHT

Correct answer is A because abrupt withdrawal of chronic steroids in an RA patient can precipitate acute adrenal crisis with hypotension, hypoglycemia, hyponatremia, and hyperkalemia.
Cortisol deficiency-

Effect	Result
↓ gluconeogenesis	Hypoglycemia
↓ vascular tone	Hypotension
↓ stress response	Shock

This reflects cortisol and aldosterone deficiency causing shock and electrolyte imbalance.
Immediate life-saving treatment is IV hydrocortisone with fluids, which corrects both glucocorticoid and mineralocorticoid deficiency.
Hydrocortisone provides:
Glucocorticoid effect (cortisol replacement)
Mineralocorticoid effect (aldosterone-like action)
□ Corrects:
• hypotension
• hypoglycemia
• electrolyte imbalance

Immediate steps:
1□□ IV Hydrocortisone
2□□ IV fluids (normal saline + dextrose)
3□□ Correct electrolytes

QUESTION

A 45-year-old man presented with weight gain, proximal muscle weakness, purple striae, and easy bruising. What is the likely underlying condition?

A **Hypercortisolism** CORRECT

B Hyperthyroidism

C Hyperparathyroidism

D Hyperaldosteronism

OK RIGHT ANSWER

A. Hypercortisolism

WHY THIS IS RIGHT

Correct answer is A because weight gain, proximal muscle weakness, purple striae, and easy bruising are classic features of Cushing syndrome due to excess cortisol.
Hypercortisolism causes protein catabolism, skin thinning, and central fat redistribution.

Mechanism	Clinical Effect
↑ Protein breakdown	Muscle weakness
↓ Collagen synthesis	Thin skin -> easy bruising
Fat redistribution	Central obesity
Skin thinning	Purple striae

Causes of Hypercortisolism

Type	Cause
Exogenous	Steroid therapy (most common)
ACTH-dependent	Pituitary adenoma (Cushing disease)
	Ectopic ACTH (small cell lung cancer)
ACTH-independent	Adrenal adenoma/carcinoma

Thyroid, parathyroid, or aldosterone excess do not produce this characteristic constellation of findings.

QUESTION

Which of the following is not a stimulus for catecholamine synthesis?

A Smoking

B Standing posture

C **Hyperglycemia** CORRECT

D Exercise

RIGHT ANSWER

OK

C. Hyperglycemia

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1b” here and does not include a written explanation for this item.

QUESTION

Which of the following is considered the initial recommended test for diagnosing pheochromocytoma?

A **24 hr urinary total metanephrines** CORRECT

B MIBG scan

C Plasma catecholamines

D MRI of the whole abdomen

RIGHT ANSWER

OK

A. 24 hr urinary total metanephrines

WHY THIS IS RIGHT

Pheochromocytoma is a catecholamine-secreting tumor of chromaffin cells.
Most arise from: Adrenal medulla
Extra-adrenal tumors are called: Paragangliomas
Correct answer is A because the preferred initial screening test for pheochromocytoma is measurement of metanephrines, which reflect continuous catecholamine metabolism and have high sensitivity.
24-hour urinary fractionated metanephrines are recommended for initial biochemical diagnosis.
Imaging (MRI/MIBG) is done only after biochemical confirmation. Plasma catecholamines fluctuate and are less reliable for screening.
Pheochromocytoma can occur in several genetic syndromes.

Syndrome	Gene
MEN 2A / MEN 2B	RET mutation
Von Hippel-Lindau disease	VHL gene
Neurofibromatosis type 1	NF1 gene

QUESTION

Which of the following is not seen in Conn's syndrome?

A Hypertension

B **Hypernatremia** CORRECT

C Hypokalemia

D Metabolic Alkalosis

RIGHT ANSWER

OK

B. Hypernatremia

WHY THIS IS RIGHT

Conn’s syndrome = Primary hyperaldosteronism
Cause: Excess aldosterone secretion from adrenal cortex independent of RAAS regulation.
Correct answer is B because Conn’s syndrome (primary hyperaldosteronism) causes sodium retention with volume expansion, but serum sodium is usually near normal due to escape mechanisms. Even though sodium is retained: The body activates escape mechanisms:
Aldosterone Escape
• Pressure natriuresis
• ANP release
• Increased GFR

These prevent significant sodium accumulation in plasma.
Therefore:

Sodium effect
Total body sodium ↑
Serum sodium ≈ normal

Hence hypernatremia is uncommon.
It classically presents with hypertension, hypokalemia, and metabolic alkalosis from increased potassium and hydrogen ion loss.
Thus, hypernatremia is not a typical finding.

QUESTION

A 38-year-old female patient presents with buffalo hump, moon facies, and abdominal striae. She is a known case of SLE on steroids. Which of the following is the next best investigation that should be done in her?

A **Serum Cortisol with ACTH levels** — CORRECT

B High-dose dexamethasone suppression test

C Low-dose dexamethasone followed by high-dose dexamethasone suppression test

D ACTH levels followed by the high-dose dexamethasone suppression test

RIGHT ANSWER

OK

A. Serum Cortisol with ACTH levels

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1c” here and does not include a written explanation for this item.

QUESTION

A 55-year-old female with a prolonged history of systemic lupus erythematosus, treated with multiple medications is being evaluated on autopsy. Records from the patient's most recent office visit show an elevated blood pressure and examination findings of facial plethora, truncal obesity, and skin ecchymoses. The patient's adrenal glands is most likely to reveal which of the following findings?

A Adrenocortical adenoma

B **Bilateral cortical atrophy** CORRECT

C Diffuse cortical hyperplasia

D Nodular cortical hyperplasia

OK RIGHT ANSWER

B. Bilateral cortical atrophy

WHY THIS IS RIGHT

Chronic exogenous glucocorticoid therapy (commonly used to treat autoimmune diseases like SLE) causes iatrogenic Cushing syndrome.

Mechanism:
• Exogenous steroids suppress hypothalamic CRH and pituitary ACTH via negative feedback.
• ↓ ACTH leads to reduced stimulation of the adrenal cortex.
• Over time, this causes bilateral adrenal cortical atrophy, particularly of the zona fasciculata and zona reticularis.

QUESTION

60-year-old woman comes to the OPD due to difficulty climbing stairs and dyspnea on exertion over the last 6 weeks. Blood pressure is 160/90 mm Hg and pulse is 78/min. Skin examination shows scattered ecchymoses. Laboratory results show mild hyperglycemia and elevated 24-hour urinary free cortisol. Serum cortisol level is at the upper limit of normal and is not suppressed following administration of low-dose dexamethasone. Serum ACTH level is elevated. Chest x-ray reveals a right lower lobe lung mass. Which of the following changes are most likely to occur after administration of high-dose dexamethasone in this patient?

A Increase

B Decrease

C **No change in cortisol** CORRECT

D Could be any of the above

RIGHT ANSWER

OK

C. No change in cortisol

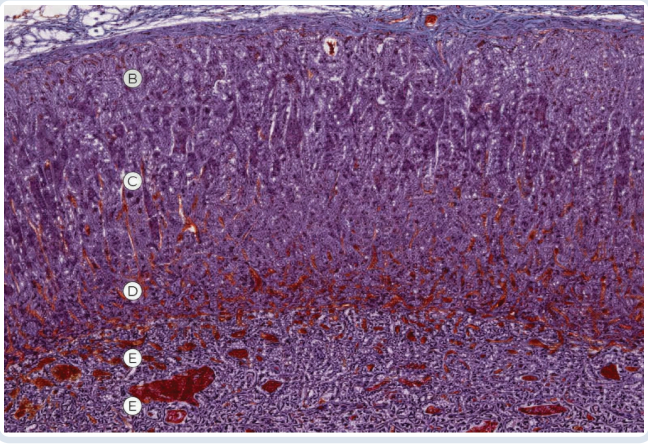
WHY THIS IS RIGHT

Ectopic ACTH syndrome occurs when a non-pituitary tumor (commonly small cell lung carcinoma) secretes ACTH, causing ACTH-dependent Cushing syndrome.

Key diagnostic principle (dexamethasone suppression test):
• Pituitary Cushing disease: Cortisol suppresses with high-dose dexamethasone.
• Ectopic ACTH production: Cortisol does NOT suppress with either low- or high-dose dexamethasone because ACTH secretion from the tumor is not regulated by normal hypothalamic-pituitary feedback.

QUESTION

A 42-year-old man comes to the physician for a follow-up examination. His blood pressure was 146/91 mm Hg at his appointment 1 month ago; subsequent home blood pressure measurements have ranged from 135/83 mm Hg to 156/96 mm Hg. His blood pressure today is 141/85 mm Hg. Pharmacotherapy with lisinopril is initiated. Administration of this drug is most likely to result in decreased activity of which of the following sections of a normal adrenal gland?



A A

B **B** CORRECT

C C

D D

OK RIGHT ANSWER

B. B

WHY THIS IS RIGHT

ACE inhibitors (e.g., lisinopril) decrease angiotensin II, leading to reduced aldosterone secretion from the zona glomerulosa of the adrenal cortex.

Adrenal gland layers (outer -> inner):
• A. Zona glomerulosa -> Aldosterone (regulated by RAAS)
• B. Zona fasciculata -> Cortisol (regulated by ACTH)
• C. Zona reticularis -> Androgens
• D. Medulla -> Catecholamines

Thus, ACE inhibition reduces angiotensin II stimulation of the zona glomerulosa, decreasing its activity.

QUESTION

A 31-year-old woman comes to the AIIMS OPD due to a 6.8-kg weight gain over the last few months. The patient also has experienced weakness and cannot lift weights that she was able to lift before the onset of her symptoms. On examination, blood pressure is 160/100 mm Hg and pulse is 88/min and regular. Neurologic examination shows proximal muscle weakness. Dark terminal hair is present on the lower abdomen. Fasting laboratory results are as follows:
Sodium-142 mEq/L
Potassium-3.6 mEq/L
Chloride-98 mEq/L
Bicarbonate-28 mEq/L
Calcium-9.2 mg/dL
TSH-2.2 mIU/L (N-0.5 to 5.0 mIU/L)
Which of the following is the most appropriate next step in evaluating this patient's condition?

A Early-morning cortisol level

B **Overnight low-dose dexamethasone suppression test** CORRECT

C Serum ACTH level

D Cosyntropin test

OK RIGHT ANSWER

B. Overnight low-dose dexamethasone suppression test

WHY THIS IS RIGHT

This patient has features suggestive of Cushing syndrome (weight gain, hypertension, proximal muscle weakness, hirsutism).

Diagnostic Algorithm:

1. Screen for hypercortisolism
-> Overnight low-dose DST / 24-hr urinary cortisol / late-night salivary cortisol

2. If positive -> measure ACTH
• Low ACTH -> adrenal tumor
• High ACTH -> pituitary (Cushing disease) or ectopic ACTH

3. Then imaging (pituitary MRI, adrenal CT, etc.)

Why Other Options Are Incorrect:
• A. Early-morning cortisol level: Not useful because cortisol normally peaks in the morning.
• C. Serum ACTH level: Done after confirming hypercortisolism, to determine the source.
• D. Cosyntropin test: Used to diagnose Addison diseases, not Cushing syndrome.

QUESTION

A 37-year-old woman comes to the OPD due to a two-month history of abdominal pain, salt craving, and weight loss. She also has dizziness on standing. Her mother and sister have hypothyroidism. Blood pressure is 100/70 mm Hg supine and 80/50 mm Hg standing. Laboratory results are as follows. Which of the following changes are expected in this patient?

A. Serum Na Low, Serum K low, urine Na low, urine K high

B. **Serum Na Low, Serum K high, urine Na high, urine K low** — CORRECT

C. Serum Na Low, Serum K low, urine Na normal, urine K high

D. Serum Na normal, Serum K low, urine Na low, urine K high

OK

RIGHT ANSWER

B. Serum Na Low, Serum K high, urine Na high, urine K low

WHY THIS IS RIGHT

This patient has primary adrenal insufficiency (Addison disease) due to adrenal cortex destruction, leading to aldosterone deficiency.

Effect of low aldosterone on kidney:
• ↓ Na□ reabsorption in distal nephron -> ↑ urinary sodium loss -> hyponatremia
• ↓ K□ excretion -> hyperkalemia
• Therefore, urine Na□ increases and urine K□ decreases.

QUESTION

A 40-year-old woman presents with a 3-month history of fatigue, weight loss, salt craving, and dizziness upon standing. She has no significant medical history and takes no medications. Her mother was recently diagnosed with hypothyroidism. On examination, her blood pressure is 100/70 mm Hg supine and 80/50 mm Hg standing. Laboratory results reveal the following:
Sodium 129 meq/l
Potassium 5.9 meq/l
Chloride 100 meq/l
Bicarbonate 21 meq/l
Creatinine 1.4 mg/dl
BUN 38mg/dl
Which of the following in circulating hormone levels are expected in this patient?

A **Cortisol low, Aldosterone low, ADH high, Norepinephrine high** CORRECT

B Cortisol normal, Aldosterone low, ADH normal, Norepinephrine normal

C Cortisol low, Aldosterone low, ADH normal, Norepinephrine normal

D Cortisol high, Aldosterone low, ADH high, Norepinephrine high

OK RIGHT ANSWER

A. Cortisol low, Aldosterone low, ADH high, Norepinephrine high

WHY THIS IS RIGHT

This patient has features of primary adrenal insufficiency (Addison disease).
• Fatigue, weight loss, salt craving
• Orthostatic hypotension
• Hyponatremia (Na□ 129 mEq/L)
• Hyperkalemia (K□ 5.9 mEq/L)
• Family history of autoimmune disease -> suggests autoimmune adrenal destruction

Hormonal pattern in primary adrenal insufficiency:
• ↓ Cortisol -> adrenal cortex failure
• ↓ Aldosterone -> Na□ loss, K□ retention, hypotension
• ↑ ADH -> due to hypovolemia and cortisol deficiency
• ↑ Norepinephrine -> compensatory sympathetic activation to maintain blood pressure

QUESTION

A 24-year-old man with congenital adrenal hyperplasia (21-hydroxylase deficiency) presents with bilateral testicular enlargement and infertility. Ultrasound shows bilateral testicular masses near the mediastinum testis, consistent with testicular adrenal rest tumors (TART). What is the most appropriate first-line treatment?

A Bilateral orchiectomy

B Radiotherapy

C **High-dose glucocorticoid therapy** CORRECT

D Chemotherapy

RIGHT ANSWER

OK

C. High-dose glucocorticoid therapy

WHY THIS IS RIGHT

In congenital adrenal hyperplasia (CAH) due to 21-hydroxylase deficiency, chronic elevation of ACTH stimulates ectopic adrenal tissue in the testes, leading to testicular adrenal rest tumors (TART). These tumors commonly occur near the mediastinum testis and can cause testicular enlargement, infertility, and obstruction of seminiferous tubules.

First-line treatment: High-dose glucocorticoid therapy

Why not other options:

A. Bilateral orchiectomy
• TART are benign and ACTH-dependent.
• Usually regress with glucocorticoids, so surgery is unnecessary.

B. Radiotherapy
• Tumors are not malignant and are hormone-driven, so radiation is not useful.

D. Chemotherapy
• Used for malignancy; TART is benign adrenal rest tissue.

QUESTION

Which of the following statements is not correct regarding primary adrenal insufficiency (Addison's disease)?

A Increased serum creatinine

B **Orthostatic hypertension is a common clinical feature** CORRECT

C Hyperkalemia is commonly present

D Skin hyperpigmentation is seen due to elevated ACTH levels

RIGHT ANSWER

OK **B. Orthostatic hypertension is a common clinical feature**

WHY THIS IS RIGHT

primary adrenal insufficiency (Addison disease) occurs due to destruction or dysfunction of the adrenal cortex, leading to deficiency of cortisol and aldosterone.

Because of volume depletion and low blood pressure, patients commonly develop orthostatic hypotension, not orthostatic hypertension.

Typical findings in Addison disease:
• Hypotension / orthostatic hypotension
• Hyperkalemia
• Hyponatremia
• Hyperpigmentation of skin and mucosa
• Possible mild increase in serum creatinine due to dehydration

QUESTION

A 50-year-old woman presents with persistent hypertension, progressive weight gain, and proximal muscle weakness. Laboratory evaluation reveals elevated serum cortisol levels with suppressed plasma ACTH. Which of the following is the next best investigation in the workup of this patient?

A Low-dose dexamethasone suppression test

B High-dose dexamethasone suppression test

C **CT scan of the abdomen** CORRECT

D MRI pituitary

OK RIGHT ANSWER

C. CT scan of the abdomen

WHY THIS IS RIGHT

Interpretation:
• High cortisol + suppressed ACTH -> ACTH-independent Cushing syndrome
• This usually indicates an adrenal source (e.g., adrenal adenoma, adrenal carcinoma, or adrenal hyperplasia).

Next step -> Imaging of the adrenal glands, typically with a CT scan of the abdomen, to identify an adrenal tumor.

Diagnostic approach (high-yield):
1. Confirm hypercortisolism (e.g., low-dose dexamethasone suppression test).
2. Measure ACTH.
3. If ACTH low -> adrenal cause -> CT abdomen.
4. If ACTH high/normal -> ACTH-dependent -> MRI pituitary or tests for ectopic ACTH.

QUESTION

A 54-year-old man with a history of chronic duodenal ulcer disease presents with new-onset muscle weakness and pedal edema. On examination, his blood pressure is elevated. Laboratory investigations reveal hypokalemia and metabolic alkalosis. He reports taking a drug Carbenoxolone for ulcer healing.
Which of the following best explains the adverse effects produced by this drug?

A Direct mineralocorticoid receptor agonism

B **Inhibition of 11β-hydroxysteroid dehydrogenase type 2** CORRECT

C Increased aldosterone secretion from adrenal cortex

D Inhibition of prostaglandin synthesis

OK RIGHT ANSWER

B. Inhibition of 11β-hydroxysteroid dehydrogenase type 2

WHY THIS IS RIGHT

Carbenoxolone can cause apparent mineralocorticoid excess by inhibiting the enzyme 11β-hydroxysteroid dehydrogenase type 2 (11β-HSD2) in the kidney.

Mechanism:
• Normally, 11β-HSD2 converts cortisol -> cortisone in renal tubular cells.
• Cortisone cannot activate mineralocorticoid receptors, protecting them from cortisol.
• Inhibition of 11β-HSD2 -> cortisol accumulates and stimulates mineralocorticoid receptors.

QUESTION

All of the following statements about NMDA receptors are true except:

A They are permeable to Ca^{2+}

B They are blocked by Mg^{2+} at resting potential

C **They mediate fast excitatory synaptic transmission** CORRECT

D They require depolarization for activation

RIGHT ANSWER

OK

C. They mediate fast excitatory synaptic transmission

WHY THIS IS RIGHT

NMDA receptors are glutamate-gated ion channels involved in synaptic plasticity, learning, and memory. They have unique properties compared with other excitatory receptors.

Key features:
• Permeable to Ca^{2+}, Na^{+}, and K^{+}
• Blocked by Mg^{2+} at resting membrane potential
• Require both glutamate binding and postsynaptic depolarization to remove the Mg^{2+} block
• Produce slow excitatory postsynaptic responses

QUESTION

Cortisone is administered into a 20 yr old woman for treatment of autoimmune disorder. Which of the following is most likely to occur ?

A Increase cortisol secretion

B **Increase insulin secretion** CORRECT

C Increase ACTH secretion

D Increase muscle mass

OK RIGHT ANSWER

B. Increase insulin secretion

WHY THIS IS RIGHT

Administration of exogenous glucocorticoids (e.g., cortisone) increases blood glucose levels by stimulating hepatic gluconeogenesis, decreasing peripheral glucose uptake, and promoting protein and fat catabolism. The resulting hyperglycemia stimulates pancreatic β-cells to increase insulin secretion.

Why not other options:

A. Increase cortisol secretion
• Exogenous cortisone suppresses the hypothalamic-pituitary-adrenal (HPA) axis.
• Negative feedback -> ↓ CRH and ↓ ACTH -> ↓ endogenous cortisol production.

C. Increase ACTH secretion
• Glucocorticoids exert negative feedback on the pituitary.
• Therefore, ACTH decreases, not increases.

D. Increase muscle mass
• Glucocorticoids cause protein catabolism.
• Leads to muscle wasting, not increased muscle mass.

QUESTION

A 35-year-old woman presents to the clinic with fatigue, hair loss, and constipation. She has a history of type 1 diabetes mellitus, well controlled on insulin, and celiac disease, which she manages with a gluten-free diet. On physical examination, she has dry skin but no other obvious abnormalities. On laboratory evaluation, her thyroid function test is abnormal.
What is the most likely underlying syndrome?

A Autoimmune polyendocrine syndrome type 1 (APS-1)

B **Autoimmune polyendocrine syndrome type 2 (APS-2)** CORRECT

C Immune dysregulation, polyendocrinopathy, enteropathy, X-linked disease

D Polyneuropathy, organomegaly, endocrinopathy, M-protein, and skin changes

OK RIGHT ANSWER

B. Autoimmune polyendocrine syndrome type 2 (APS-2)

WHY THIS IS RIGHT

Autoimmune Polyendocrine Syndrome Type 2 (APS-2) is an adult-onset autoimmune disorder characterized by the coexistence of autoimmune adrenal insufficiency (Addison disease) with autoimmune thyroid disease and/or type 1 diabetes mellitus. It typically occurs in young to middle-aged women and may be associated with other autoimmune conditions such as celiac disease, vitiligo, and pernicious anemia.

Why not other options:

A. APS-1
• Childhood onset
• Triad: mucocutaneous candidiasis + hypoparathyroidism + Addison disease
• Patient is adult (35 years) with none of these features.

C. IPEX syndrome
• X-linked FOXP3 mutation
• Occurs in male infants with severe autoimmune enteropathy.
• Patient is an adult female.

D. POEMS syndrome
• Plasma cell disorder.
• Features: polyneuropathy, organomegaly, monoclonal protein, skin changes.

QUESTION

All are true in conn syndrome except?

A High aldosterone

B Metabolic alkalosis

C **High plasma renin** CORRECT

D Diastolic HTN without edema

RIGHT ANSWER

OK

C. High plasma renin

WHY THIS IS RIGHT

Conn syndrome (primary hyperaldosteronism) is caused by excess aldosterone secretion, usually from an adrenal adenoma or bilateral adrenal hyperplasia.
Excess aldosterone -> increased Na□ reabsorption and K□/H□ excretion in the distal nephron.

Characteristic features
• High aldosterone levels
• Suppressed plasma renin activity (due to negative feedback from volume expansion)
• Hypokalemia
• Metabolic alkalosis (due to increased H□ loss)
• Diastolic hypertension without edema (because of aldosterone escape mechanism)

QUESTION

Cushing syndrome is not a feature of

A Adrenal carcinoma

B Oat cell carcinoma of lung

C **Medulloblastoma** CORRECT

D Pituitary adenoma

OK RIGHT ANSWER

C. Medulloblastoma

WHY THIS IS RIGHT

Cushing syndrome results from excess glucocorticoids (cortisol) and may occur due to ACTH-dependent or ACTH-independent causes.
ACTH-dependent causes:
• Pituitary adenoma -> called Cushing disease
• Ectopic ACTH production, commonly from small cell (oat cell) carcinoma of the lung

ACTH-independent causes:
• Adrenal tumors (e.g., adrenal carcinoma or adenoma) that produce excess cortisol.

Medulloblastoma is a malignant cerebellar tumor of childhood and does not produce ACTH or cortisol, so it is not associated with Cushing syndrome.

QUESTION

A middle aged patient presenting with change in her voice, anemia, recent hair loss and cold intolerance has been diagnosed with Hashimoto's thyroiditis. Which of the following antibody is commonly seen in such patients?

A TSIg

B **Anti thyroid peroxidase (TPO) antibody** CORRECT

C Anti thyroid binding globulin

D Anti thyroid receptor antibody

RIGHT ANSWER

OK **B. Anti thyroid peroxidase (TPO) antibody**

WHY THIS IS RIGHT

Hashimoto thyroiditis is an autoimmune destruction of the thyroid gland leading to hypothyroidism.

It is characterized by high titers of anti-thyroid peroxidase (TPO) antibodies.
Anti-TPO antibodies mediate thyroid follicular cell damage via immune mechanisms.

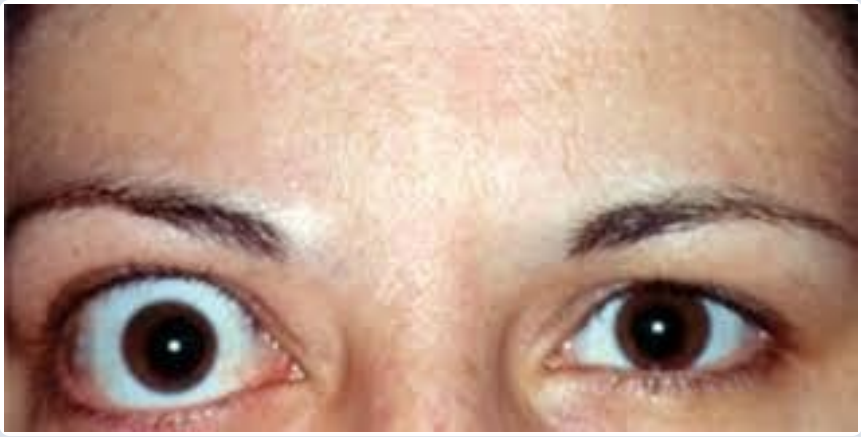
Thyroid peroxidase (TPO) function:
• Oxidation of iodide
• Iodination of tyrosine
• Coupling -> T3 & T4 formation

Anti-TPO antibodies:
• Attack TPO enzyme
• Impair hormone synthesis
• Cause gland destruction

 Hallmark of Hashimoto thyroiditis
TSIg and anti-TSH receptor antibodies are associated with Graves' disease, not Hashimoto thyroiditis.

QUESTION

A 23-year-old medical student with a 2-month history of palpitations, sweating, and restlessness. He also complains of a sweaty palm. His appearance is as shown in the picture. What is the next step in evaluation?



A Thyroid receptor antibody

B Radioactive Iodine Uptake

C **Ultrasensitive TSH** CORRECT

D Anti-thyroid peroxidase antibody

RIGHT ANSWER

OK

C. Ultrasensitive TSH

WHY THIS IS RIGHT

Ultrasensitive TSH is the best initial test in suspected hyperthyroidism because it is the most sensitive marker of thyroid axis status. Thyrotoxicosis typically shows suppressed TSH before T3/T4 become markedly elevated. Antibody tests and radioactive uptake are used later to determine etiology. Screening always begins with TSH.
A. Thyroid receptor antibody
• Used to confirm Graves disease

B. Radioactive iodine uptake (RAIU)
• Used to differentiate causes

D. Anti-TPO antibody
• Seen in Hashimoto thyroiditis

QUESTION

Which of the following is false in patients with thyroid eye disease?

A It can lead to vision loss

B **Response to vision symptoms is adjuvant to thyrotoxicosis response** CORRECT

C NOSPECS classification is used

D 10% of euthyroid patients can have eye involvement.

RIGHT ANSWER

OK **B. Response to vision symptoms is adjuvant to thyrotoxicosis response**

WHY THIS IS RIGHT

The source quiz contains the placeholder “$19” here and does not include a written explanation for this item.

QUESTION

A young woman presents with weight loss, tachycardia, heat intolerance, and tremors. She has exophthalmos on examination. What is the diagnosis?

A Parkinson's disease

B **Graves' disease** CORRECT

C Addison's disease

D Alzheimer's disease

OK RIGHT ANSWER

B. Graves' disease

WHY THIS IS RIGHT

Graves’ disease is an autoimmune hyperthyroid state characterized by weight loss, tachycardia, heat intolerance, and tremors due to excess thyroid hormone.
Autoantibodies (TSH receptor antibodies)
□
Stimulate TSH receptors
□
↑ T3 and T4 production
□
Hyperthyroidism
Exophthalmos is a hallmark feature caused by autoimmune orbital fibroblast activation and glycosaminoglycan deposition.
• Most common cause of hyperthyroidism = Graves’ disease
• Pathognomonic sign = exophthalmos □
• Caused by TSH receptor antibodies
• TSH ↓, T3/T4 ↑

This eye involvement is specific for Graves among thyrotoxic states. The other options do not produce thyrotoxicosis with ophthalmopathy.

QUESTION

A woman at 8 weeks of gestation is diagnosed with hyperthyroidism. Which of the following is the most appropriate treatment option?

A Methimazole

B Carbimazole

C **Propylthiouracil** CORRECT

D Radioactive iodine

RIGHT ANSWER

OK **C. Propylthiouracil**

WHY THIS IS RIGHT

Propylthiouracil is preferred in the first trimester because methimazole/carbimazole are teratogenic and linked to embryopathy.

Trimester	Drug of Choice	Reason
1st trimester	□ PTU	Less teratogenic
2nd & 3rd trimester	Methimazole	Less hepatotoxic

PTU also reduces peripheral T4->T3 conversion, helping rapid control of thyrotoxicosis. After the first trimester, therapy is often switched to methimazole due to PTU hepatotoxicity risk.

Drug	Mechanism
PTU	↓ T3/T4 synthesis + ↓ T4 -> T3 conversion
Methimazole	↓ T3/T4 synthesis only

Methimazole causes teratogenic effects:

Defect	Description
Aplasia cutis	Absence of scalp skin
Choanal atresia	Nasal blockage
Esophageal atresia	Feeding difficulty

Radioactive iodine is absolutely contraindicated in pregnancy.

QUESTION

Identify the transporter of iodine from follicular cells to follicular cavity within the thyroid gland:

A Na+ I- Symporter

B **Pendrin** CORRECT

C Calmodulin

D Claudin

RIGHT ANSWER

OK **B. Pendrin**

WHY THIS IS RIGHT

Iodide from blood
□ *(Basolateral membrane)*
Na□/I□ symporter (NIS)
□
Iodide enters follicular cell
□ *(Apical membrane)*
Pendrin □
□
Iodide moves into colloid
□
Thyroid hormone synthesis begins

Transporter	Location	Function
NIS (Na□/I□ symporter)	Basolateral membrane	Blood -> cell
Pendrin □	Apical membrane	Cell -> colloid
TPO (enzyme)	Colloid	Organification

Pendrin transports iodide from thyroid follicular cells across the apical membrane into the follicular lumen for organification. The Na□/I□ symporter works on the basolateral membrane to bring iodide into the cell from blood. Defective pendrin impairs iodide efflux into colloid. This apical transport step is essential for thyroid hormone synthesis.

QUESTION

In the management of a thyroid storm, a critical and life-threatening condition due to an excess of thyroid hormone, a variety of medications are used to control the symptoms and the overactive thyroid. Which of the following drugs is NOT typically used in the treatment of thyroid storm?

A Propranolol

B Lugol's Iodine

C Acetaminophen

D **Aspirin** CORRECT

OK RIGHT ANSWER

D. Aspirin

WHY THIS IS RIGHT

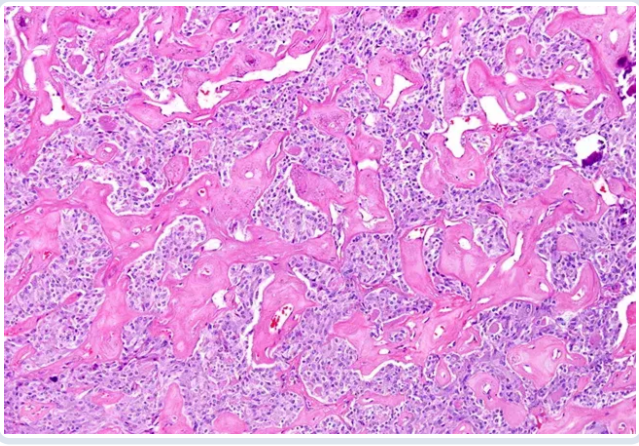
Thyroid storm = extreme thyrotoxicosis -> life-threatening emergency
Management aims to:
• Block hormone synthesis
• Block hormone release
• Control symptoms
• Reduce peripheral effects

Aspirin is avoided in thyroid storm because salicylates displace T3/T4 from thyroid-binding globulin, increasing free hormone levels and worsening thyrotoxicosis.
Beta-blocker (Propranolol)
□
Antithyroid drug (PTU / Methimazole)
□
Iodine (Lugol's iodine)
□
Steroids
□
Supportive care (fluids, cooling, paracetamol)
Propranolol controls adrenergic symptoms and reduces peripheral T4->T3 conversion, while Lugol's iodine blocks hormone release.

Drug	Role in Thyroid Storm
Propranolol □	Controls tachycardia + ↓ T4->T3
PTU	↓ synthesis + ↓ T4->T3
Methimazole	↓ synthesis
Lugol's iodine □	↓ hormone release
Steroids	↓ T4->T3 + adrenal support
Paracetamol (Acetaminophen)	Fever control

QUESTION

A patient presents with neck swelling, compressing the trachea and esophagus. Histopathological assessment shows cell nests and pink extracellular amyloid stroma. What is the cell of origin of this tumor?



A **Parafollicular C cells** — CORRECT

B Askanazy cells

C Hurthle cells

D Follicular cells

RIGHT ANSWER

OK **A. Parafollicular C cells**

WHY THIS IS RIGHT

This is a classic description of: Medullary Thyroid Carcinoma (MTC)
Hallmark clues:
• Amyloid stroma
• Neuroendocrine tumor pattern (cell nests)
• Thyroid mass causing compression

Parafollicular C cells
• Derived from neural crest
• Located between thyroid follicles
• Secrete: Calcitonin

The amyloid represents calcitonin-derived deposits produced by malignant parafollicular C cells.
Thus, the tumor originates from neural crest-derived C cells, not follicular or Hurthle cells.

QUESTION

A woman presents with signs of hyperthyroidism. Which feature is not typically associated with her condition?

A **Reduced protein breakdown** CORRECT

B Increased heart rate

C Decreased peripheral vascular resistance

D Increased stroke volume

RIGHT ANSWER

OK **A. Reduced protein breakdown**

WHY THIS IS RIGHT

Hyperthyroidism increases the basal metabolic rate and produces a hypercatabolic state.

Key physiologic effects include:
• Increased protein breakdown (proteolysis) -> muscle wasting and weakness
• Increased heart rate and stroke volume -> high cardiac output
• Decreased peripheral vascular resistance due to vasodilation

QUESTION

Regarding thyroid storm, which statement is false?

A **Mortality is 10% despite treatment** — CORRECT

B It may follow non-thyroid surgeries

C Symptoms can include diarrhea, vomiting, jaundice, and confusion

D High-dose propranolol can reduce T4 to T3 conversion

RIGHT ANSWER

OK **A. Mortality is 10% despite treatment**

WHY THIS IS RIGHT

Thyroid storm is a life-threatening exacerbation of thyrotoxicosis characterized by severe hypermetabolic state and multisystem involvement.
• High fever, tachycardia, agitation, confusion
• Gastrointestinal symptoms: nausea, vomiting, diarrhea, jaundice
• Can be precipitated by stressors such as infection, trauma, or surgery (including non-thyroid surgery).

Management principles:
• β-blockers (high-dose propranolol) to control adrenergic symptoms and reduce peripheral conversion of T4 -> T3
• Antithyroid drugs (PTU or methimazole)
• Iodine and glucocorticoids

Mortality remains high (\~20-30% even with treatment), therefore the statement “mortality is 10% despite treatment” is false.

QUESTION

51-year-old woman comes to the OPD due to progressively worsening fatigue, weight gain, and constipation for the past 6 months. Physical examination shows mild, diffuse enlargement of the thyroid gland. Biopsy of this patient's thyroid is most likely to show which of the following findings?

A **https://corebtr-assets-production.s3.ap-south-1.amazonaws.com/questions_image/option/01KN3PNJ9403PXE0JHTXWHVAVV.jpg** CORRECT

B https://corebtr-assets-production.s3.ap-south-1.amazonaws.com/questions_image/option/01KN3PNJE3Z4JFCMMSQ71A74F2.jpg

C https://corebtr-assets-production.s3.ap-south-1.amazonaws.com/questions_image/option/01KN3PNJKSQPB8YN8VBJFBM7D2.jpg

D https://corebtr-assets-production.s3.ap-south-1.amazonaws.com/questions_image/option/01KN3PNJRXHAD4AZG6E4SNXQJM.jpg

RIGHT ANSWER

OK **A. https://corebtr-assets-production.s3.ap-south-1.amazonaws.com/questions_image/option/01KN3PNJ9403PXE0JHTXWHVAVV.jpg**

WHY THIS IS RIGHT

Hashimoto thyroiditis is the most common cause of hypothyroidism in adults and typically presents with fatigue, weight gain, constipation, and diffuse thyroid enlargement.

Characteristic histopathology:
• Dense lymphocytic infiltration of the thyroid
• Germinal center formation
• Destruction of thyroid follicles
• Hurthle (oxyphilic) cell metaplasia

Option	Diagnosis	Key Histological Feature
A	Hashimoto thyroiditis	Dense lymphocytic infiltration with germinal centers and follicular destruction
B	Graves disease	Tall columnar epithelium with papillary infoldings and scalloped colloid
C	Subacute (De Quervain) thyroiditis	Granulomatous inflammation with multinucleated giant cells
D	Papillary thyroid carcinoma	Papillary structures with nuclear changes (Orphan Annie nuclei, nuclear grooves, psammoma bodies)

QUESTION

A 29-year-old woman comes to the OPD due to hair loss. The patient has fatigue and decreased libido, and her menstrual cycles have not returned since her delivery 8 months ago. The thyroid is nontender. Laboratory results show decreased serum TSH and free thyroxine (T4) levels. Which of the following is the most likely diagnosis in this patient?

A Chronic autoimmune thyroiditis

B Graves disease

C Postpartum thyroiditis

D **Secondary hypothyroidism** CORRECT

OK RIGHT ANSWER

D. Secondary hypothyroidism

WHY THIS IS RIGHT

Secondary hypothyroidism occurs due to pituitary or hypothalamic dysfunction, resulting in decreased TSH secretion and low thyroid hormone levels (T4). Failure of menstrual cycles to return after delivery, along with fatigue and low libido, suggests postpartum pituitary dysfunction (e.g., Sheehan syndrome) causing secondary hypothyroidism. Key diagnostic pattern:
• Low TSH
• Low T4

This contrasts with primary hypothyroidism, where TSH is elevated.

Why not other options:
A. Chronic autoimmune thyroiditis (Hashimoto)
• Primary hypothyroidism -> ↑ TSH, ↓ T4
• Patient has ↓ TSH and ↓ T4, so not primary.

B. Graves disease
• Causes hyperthyroidism -> ↓ TSH, ↑ T4
• Patient has low T4, not high.

C. Postpartum thyroiditis
• Usually, transient hyperthyroid phase -> hypothyroid phase
• Hypothyroid phase shows ↑ TSH, ↓ T4.

QUESTION

A 45-year-old woman is admitted to the intensive care unit due to severe sepsis caused by a ruptured abdominal abscess. She has been receiving high-dose intravenous corticosteroids for hypotension management over the past three days. On examination, she is alert but appears critically ill. Laboratory results are as follows:
• TSH: 1.2 µU/mL (normal range: 0.4-4.0 µU/mL)
• Free T4: 1.1 ng/dL (normal range: 0.8-1.8 ng/dL)
• Free T3: 0.6 pg/mL (normal range: 2.0-4.4 pg/mL)
Which of the following is the most likely explanation for these findings?

A Chronic lymphocytic thyroiditis

B Drug-induced hypothyroidism

C Subclinical hypothyroidism

D **Euthyroid sick syndrome** CORRECT

RIGHT ANSWER

OK **D. Euthyroid sick syndrome**

WHY THIS IS RIGHT

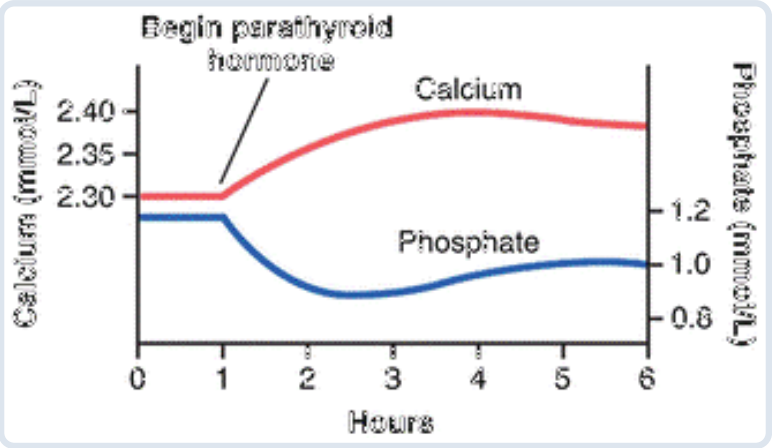
Euthyroid sick syndrome (non-thyroidal illness syndrome) occurs in severe systemic illness such as sepsis, trauma, or critical illness. Thyroid gland function is normal, but peripheral thyroid hormone metabolism is altered. Typical laboratory pattern:
• ↓ Free T3 (most common finding)
• Normal or ↓ T4
• Normal or slightly ↓ TSH

Mechanism:
• Severe illness and stress hormones (e.g., cytokines, glucocorticoids)
• ↓ peripheral conversion of T4 -> T3 due to reduced 5′-deiodinase activity

Condition	TSH	T4	T3	Key Feature
Chronic lymphocytic thyroiditis (Hashimoto)	↑	↓	↓	Autoimmune destruction of thyroid → primary hypothyroidism
Drug-induced hypothyroidism	↑	↓	↓	Caused by drugs (e.g., amiodarone, lithium)
Subclinical hypothyroidism	↑	Normal	Normal	Early thyroid failure; mild/asymptomatic
Euthyroid sick syndrome	Normal or ↓	Normal or ↓	↓	Seen in severe illness (e.g., sepsis); ↓ T4→T3 conversion

QUESTION

The adjoining graph shows the effect of PTH on blood calcium and phosphate in an experimental animal. How does PTH increase plasma calcium?



A By increasing osteoclast activity

B By increasing osteoblastic activity

C By decreasing renal excretion

D **By increasing osteoclastic activity and decreasing renal excretion of calcium** CORRECT

RIGHT ANSWER

OK **D. By increasing osteoclastic activity and decreasing renal excretion of calcium**

WHY THIS IS RIGHT

The source quiz contains the placeholder “$19” here and does not include a written explanation for this item.

QUESTION

PTH increases Calcium in blood. This is due to:

A Rapid release of calcium by osteocytes

B Rapid release of calcium by osteoclasts

C **Rapid release of calcium from bones and increased calcium reabsorption in kidneys** CORRECT

D Increase calcium reabsorption in kidneys

RIGHT ANSWER

OK

C. Rapid release of calcium from bones and increased calcium reabsorption in kidneys

WHY THIS IS RIGHT

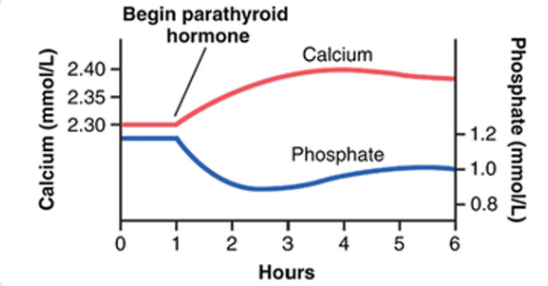
Parathyroid hormone (PTH) primarily acts to increase the concentration of calcium in the blood. It achieves this through several mechanisms:

1. Stimulation of osteoclasts to resorb bone, leading to the release of calcium from the bones.
2. Increased absorption of calcium in the intestines.
3. Stimulation of the kidneys to reabsorb more calcium, reducing its excretion in the urine.

a) Rapid release by osteocytes
PTH mainly works via osteoblast -> osteoclast activation

b) Rapid release by osteoclasts
Does not include kidney contribution

d) Increase calcium reabsorption in kidneys
Kidney alone cannot significantly raise Ca without bone contribution



QUESTION

Which among the following drugs decreases the serum parathyroid hormone levels?

A Risedronate

B **Risedronate** CORRECT

C Lithium

D Diuretics

RIGHT ANSWER

OK **B. Risedronate**

WHY THIS IS RIGHT

Correct answer is B because calcitriol (active vitamin D) increases intestinal calcium absorption and raises serum calcium, which suppresses parathyroid hormone via negative feedback.

Key regulators of PTH:

Factor	Effect on PTH
Low Ca^{2}□	↑ PTH
High Ca^{2}□	↓ PTH
Calcitriol	↓ PTH
Phosphate	↑ PTH

Calcitriol acts on:

Organ	Effect
Intestine	↑ Ca absorption
Kidney	↑ Ca reabsorption
Bone	↑ remodeling

Risedronate reduces bone resorption but does not directly suppress PTH. Lithium can increase PTH and cause hyperparathyroidism. Diuretics have variable effects and are not primary PTH suppressors.

QUESTION

Which of the following is an adverse effect associated with the long-term use of intermittent teriparatide for the treatment of osteoporosis?

A Osteonecrosis of the jaw bone

B **Osteosarcoma** CORRECT

C Subtrochanteric fracture

D Loss of bone mass

OK RIGHT ANSWER

B. Osteosarcoma

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1a” here and does not include a written explanation for this item.

QUESTION

A 32-year-old woman presents with recurrent renal calculi, proximal muscle weakness, and low mood. Laboratory evaluation shows elevated serum PTH with normal serum calcium. Primary hyperparathyroidism is suspected. Which of the following is the investigation of choice for preoperative localization of the abnormal gland?

A Neck ultrasound

B MRI neck

C **Tc-99m sestamibi scan** CORRECT

D CT scan neck

OK RIGHT ANSWER

C. Tc-99m sestamibi scan

WHY THIS IS RIGHT

• Recurrent renal stones + proximal weakness + depression -> classic hyperparathyroid symptom cluster
• ↑ PTH with normal Ca -> early / normocalcemic primary hyperparathyroidism

Why Tc-99m Sestamibi is IOC
• Mechanism: Radiotracer (Tc-99m sestamibi) is taken up by mitochondria-rich parathyroid adenoma cells
• Shows persistent uptake in abnormal parathyroid tissue (vs thyroid washout)
• High sensitivity for detecting single adenoma (most common cause)

Step 4: Why not other options?
• A) Neck ultrasound
• Operator dependent
• Misses ectopic glands
• B) MRI neck
• Used only if sestamibi inconclusive
• D) CT neck
• 4D-CT used in difficult/recurrent cases, not first-line

QUESTION

Which of the following is not typically associated with Autoimmune Polyendocrine Syndrome Type 1 (APS-1)?

A Hypoparathyroidism is common

B **Type 1 Diabetes Mellitus association is high** CORRECT

C Association with mucocutaneous candidiasis is high

D Early onset in infancy

OK RIGHT ANSWER

B. Type 1 Diabetes Mellitus association is high

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1b” here and does not include a written explanation for this item.

QUESTION

A 35-year-old woman comes to the OPD due to moderate pain in her lower back, hips, and knees for the past several weeks. She has no associated morning stiffness. The patient has end-stage renal disease due to diabetic nephropathy and has been on Hemodialysis for the past 2 years. Her other medical problems include type 1 diabetes mellitus diagnosed 20 years ago and sarcoidosis diagnosed 8 years ago. Physical examination shows no obvious bone or joint deformity, and she has full range of motion in all joints. Laboratory results are as follows:
Sodium-136 mEq/L
Potassium-3.9 mEq/L
Bicarbonate-23 mEq/L
Creatinine-3.1 mg/dL
Calcium-7.9 mg/dL (N-8.5 to 10.5 mg/dl)
Phosphorus-6.1 mg/dL (N-2.8 to 4.5 mg/dL)
Albumin-4.1 g/dL (N-3.4 to 5.4 g/dL)
Which of the following is the most likely histopathological abnormality underlying this patient's current symptoms?

A Autoimmune parathyroid destruction

B Granulomatous infiltration of parathyroid

C PTH adenoma

D **PTH hyperplasia** CORRECT

OK RIGHT ANSWER

D. PTH hyperplasia

WHY THIS IS RIGHT

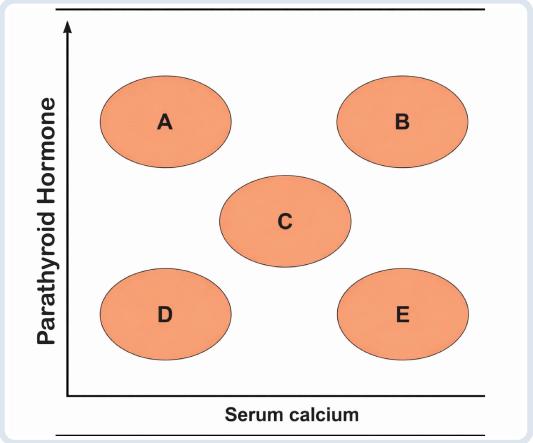
Chronic kidney disease (CKD) leads to secondary hyperparathyroidism due to disturbances in calcium-phosphate metabolism.

Mechanism:
• ↓ Renal phosphate excretion -> Hyperphosphatemia
• ↓ Renal 1α-hydroxylase activity -> ↓ calcitriol (active vitamin D)
• ↓ Intestinal calcium absorption -> Hypocalcemia

These changes chronically stimulate the parathyroid glands, causing diffuse parathyroid hyperplasia and increased parathyroid hormone (PTH) secretion.

QUESTION

A 64-year-old man comes to the OPD with fatigue. He has hypertension and poorly controlled diabetes complicated by nephropathy and peripheral neuropathy. His renal function has declined steadily over the last few years. On the graph below, area "C" shows the normal relationship between serum concentrations of free calcium and parathyroid hormone. Which of the following areas most likely represents this patient's current metabolic state?



A. A — CORRECT

B. B

C. C

D. E

RIGHT ANSWER

OK

A. A

WHY THIS IS RIGHT

Chronic kidney disease (CKD) commonly causes secondary hyperparathyroidism due to:
• Decreased phosphate excretion -> hyperphosphatemia
• Reduced renal 1α-hydroxylase activity -> ↓ calcitriol (active vitamin D)
• Decreased intestinal calcium absorption

These changes lead to hypocalcemia, which stimulates the parathyroid glands to produce increased parathyroid hormone (PTH).

QUESTION

A 68-year-old woman comes to the OPD due to a burning sensation in her chest and throat for the past 2 weeks. Associated symptoms include trouble swallowing. Medical history is significant for osteoporosis, and she has no known drug allergies. It is determined that the patient's current symptoms are caused by one of her medications, which is discontinued. Her symptoms subsequently resolve. The medication responsible for this patient's presentation is also associated with which of the following side effects?

A Hypercalcemia

B **Osteonecrosis of the jaw** CORRECT

C Venous thromboembolism

D Endometrial hyperplasia

OK RIGHT ANSWER

B. Osteonecrosis of the jaw

WHY THIS IS RIGHT

This patient likely developed pill-induced esophagitis due to an oral bisphosphonate (commonly used for osteoporosis).

Mechanism:

Bisphosphonates (e.g., alendronate) inhibit osteoclast-mediated bone resorption, increasing bone mineral density. However, they can irritate the esophageal mucosa, causing burning chest pain, dysphagia, and esophagitis if taken improperly.

Important adverse effects of bisphosphonates:
• Esophagitis and esophageal ulceration
• Osteonecrosis of the jaw (especially after dental procedures)
• Atypical femoral fractures with long-term use

QUESTION

Which of the following laboratory findings is characteristic of pseudohypoparathyroidism?

A **Low urinary cyclic AMP (cAMP) level after parathyroid hormone (PTH) administration** CORRECT

B Normal urinary cyclic AMP (cAMP) level after PTH administration

C Elevated serum calcium level

D Elevated urinary cyclic AMP (cAMP) level after PTH administration

OK RIGHT ANSWER

A. Low urinary cyclic AMP (cAMP) level after parathyroid hormone (PTH) administration

WHY THIS IS RIGHT

Pseudohypoparathyroidism is a disorder characterized by end-organ resistance to parathyroid hormone (PTH) despite normal or elevated PTH levels.
• Caused by a defect in the Gsα protein signaling pathway.
• The kidney does not respond to PTH, so the cAMP second messenger is not generated properly.

Typical laboratory findings
• Low serum calcium
• High serum phosphate
• Elevated PTH levels
• Low urinary cyclic AMP (cAMP) after administration of exogenous PTH

QUESTION

A patient with diabetes mellitus for the past 5 years presents with vomiting and abdominal pain. She is non-compliant with medication and appears dehydrated. Investigations revealed a blood sugar value of 500 mg/dL and the presence of ketone bodies. What is the next best step in management?

A Intravenous fluids with S.C. long-acting insulin

B Intravenous fluids with S.C. regular insulin

C Intravenous insulin I.V. long-acting insulin

D **Intravenous fluids with I.V. regular insulin** CORRECT

OK RIGHT ANSWER

D. Intravenous fluids with I.V. regular insulin

WHY THIS IS RIGHT

This presentation is diabetic ketoacidosis with dehydration, hyperglycemia, and ketonemia requiring emergency management.
Insulin deficiency
□
Increased lipolysis
□
Free fatty acids released
□
Ketone body formation
□
Metabolic acidosis

At the same time:
Hyperglycemia
□
Osmotic diuresis
□
Severe dehydration

The priority is rapid isotonic IV fluids followed by continuous IV regular insulin to stop ketogenesis. Subcutaneous insulin is unreliable in shock due to poor perfusion.
Long-acting insulin has no role in acute DKA control.

A. IV fluids + SC long-acting insulin
• Long-acting insulin is too slow
• Not used in emergencies

B. IV fluids + SC regular insulin
• Subcutaneous route is unpredictable in shock/dehydration
• IV route is preferred in DKA

C. IV insulin long-acting
• Long-acting insulin is never given IV

Not suitable for acute correction

QUESTION

In Type 1 Diabetes Mellitus, what is the feature of stage 3 beta cell destruction

A Autoimmune +ve Normoglycemic presymptomatic

B **Autoimmune +ve dysglycemic symptomatic** CORRECT

C Autoimmune -ve Normoglycemic presymptomatic

D Autoimmune -ve dysglycemic presymptomatic

OK RIGHT ANSWER

B. Autoimmune +ve dysglycemic symptomatic

WHY THIS IS RIGHT

The source quiz contains the placeholder “$19” here and does not include a written explanation for this item.

QUESTION

Which receptor helps in improvement of insulin resistance in Type-2 Diabetes Mellitus with regular exercise and physical activity?

A GLUT1

B **GLUT4** CORRECT

C GLUT2

D GLUT3

OK RIGHT ANSWER

B. GLUT4

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1a” here and does not include a written explanation for this item.

QUESTION

When blood glucose levels are increased to 2-3 times their normal value and then maintained at that level for a consistent period, which of the following statements best describes the pattern of insulin secretion?

A Rise and fall rapidly

B Gradually rise and fall

C Remain elevated

D **1st it rises then remains elevated** CORRECT

RIGHT ANSWER

OK

D. 1st it rises then remains elevated

WHY THIS IS RIGHT

Phase	Timing	Source	Pattern
Phase 1	Minutes	Stored insulin	Rapid spike
Phase 2	Hours	Newly synthesized insulin	Sustained plateau

Sustained hyperglycemia produces biphasic insulin secretion: an initial rapid first phase from release of preformed insulin, followed by a prolonged second phase due to new insulin synthesis. When glucose remains high, secretion does not fall back to baseline but stays elevated. This classic two-phase response is a hallmark of normal β-cell physiology.
Loss of first phase is an early feature of type 2 diabetes.

Condition	Phase 1	Phase 2
Normal	Present	Present
Type 2 DM	Lost early	Reduced

QUESTION

A 40-year-old male presents with increased thirst and increased frequency and volume of urination. He has no other symptoms. His random blood sugar was 205 mg/dl, what appropriate investigations should be done?

i) HbA1c
ii) Urine ACR
iii) Echocardiography
iv) Fundoscopy
v) ECG
vi) Serum protein electrophoresis

A i, ii, iii, v

B i, ii, iii, vi

C i, ii, iii, v

D **i, ii, iv, v** CORRECT

RIGHT ANSWER

OK

D. i, ii, iv, v

WHY THIS IS RIGHT

Investigation	Purpose	Why Important
HbA1c □	Long-term glucose control	Reflects 3-month average
Urine ACR □	Nephropathy screening	Detects microalbuminuria
Fundoscopy □	Retinopathy screening	Early eye damage
ECG □	Cardiovascular risk	Detect silent ischemia

Random glucose 205 mg/dL with polyuria and polydipsia suggests diabetes mellitus, so HbA1c confirms chronic hyperglycemia. Urine ACR screens for early diabetic nephropathy, while fundoscopy detects diabetic retinopathy at baseline. ECG is recommended because diabetes is a coronary risk equivalent and silent ischemia is common.

Echocardiography and serum protein electrophoresis are not routine initial evaluations in uncomplicated new diabetes.

QUESTION

A 27-year-old female patient presents with weight loss, anxiety, tachycardia, and restlessness. The resting pulse was 110 beats per minute. Which of the following tests should be done next to evaluate her condition further?

A Total T3

B Thyroid scan

C Free T4

D **TSH** CORRECT

RIGHT ANSWER

OK

D. TSH

WHY THIS IS RIGHT

TSH is the best initial screening test for suspected hyperthyroidism because it is the most sensitive indicator of thyroid function.

Test	Role	When Used
TSH □	Initial screening	First test
Free T4	Confirm severity	After TSH
Total T3	Sometimes used	Less preferred
Thyroid scan	Etiology	Later step

In thyrotoxicosis, TSH is characteristically suppressed due to negative feedback from elevated thyroid hormones. Free T4 or T3 are measured only after an abnormal TSH to confirm and classify severity. Thyroid scan is reserved for etiologic evaluation, not first-line screening.

QUESTION

A patient with a history of diabetes mellitus presents with heart failure characterized by a reduced ejection fraction and low urine output. Which of the following anti-diabetic medications has been recently approved for the treatment of heart failure with reduced ejection fraction, and may also improve diuresis?

A **Empagliflozin** CORRECT

B Linagliptin

C Rosiglitazone

D Insulin degludec

OK RIGHT ANSWER

A. Empagliflozin

WHY THIS IS RIGHT

Correct answer is A because empagliflozin (SGLT2 inhibitor) is approved for heart failure with reduced ejection fraction and improves outcomes independent of diabetes control.
Empagliflozin blocks SGLT2 in proximal tubule
□
↓ Glucose reabsorption
□
↑ Glucose excretion
□
Osmotic diuresis
□
↓ Plasma volume
□
↓ Preload & afterload
□
Improves heart failure
It promotes osmotic diuresis and natriuresis, helping relieve congestion and improve cardiac function.
DPP-4 inhibitors, thiazolidinediones, and insulin do not provide this heart failure benefit.

QUESTION

A 10-year-old child presented with abdominal pain and vomiting. Lab values are given below. What is the next best step of management?

Lab Values:
RBS - 450 mg/dL
pH - 7.0
HCO3 - 4
Na - 145
K - 4.8 meq/L

A 10 ml/kg NS over 20 min (10% deficit correction) followed by insulin infusion with 20 mmol/L KCl with bicarbonate

B 20 ml/kg 1/2 NS over 20 min (7% deficit correction) followed by insulin infusion and KCl 40 meq/L

C **20 ml/kg NS over 20 min with (10% deficit correction) followed by insulin infusion and KCl 40 meq/L** CORRECT

D 20 ml/kg RL over 20 min with (10% deficit correction) followed by insulin infusion and KCl 40 meq/L

RIGHT ANSWER

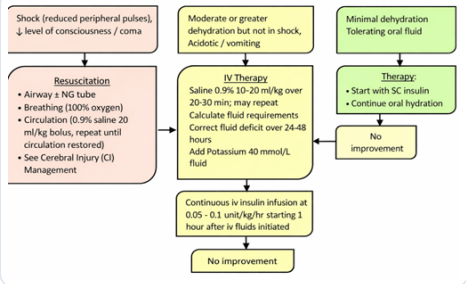
OK **C. 20 ml/kg NS over 20 min with (10% deficit correction) followed by insulin infusion and KCl 40 meq/L**

WHY THIS IS RIGHT

Correct answer is C according to ISPAD guidelines, as the labs show severe diabetic ketoacidosis (glucose 450, pH 7.0, HCO□ 4). First step is rapid isotonic fluid resuscitation with 20 ml/kg normal saline to restore perfusion.
After initial fluids, insulin infusion is started, 0.1 units/kg/hour with potassium replacement because total body potassium is depleted despite normal serum K.
Effects:
• Stops ketogenesis
• Reduces glucose
• Stops lipolysis

Bicarbonate is not routinely given unless pH < 6.9. Half-normal saline and RL are not preferred for initial resuscitation in pediatric DKA.
RL not preferred initially.
Reasons:
• Lactate -> metabolized to bicarbonate
• May interfere with ketone monitoring

NS is standard guideline recommendation



QUESTION

Insulin independent absorption occurs in all except:

A **Adipose** CORRECT

B RBC

C Pancreas

D Brain

RIGHT ANSWER

OK

A. Adipose

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1b” here and does not include a written explanation for this item.

QUESTION

Which is not considered in the diagnosis of Metabolic syndrome:

A **Hip Circumference** CORRECT

B HDL

C Glucose

D Hypertension

RIGHT ANSWER

OK **A. Hip Circumference**

WHY THIS IS RIGHT

The criteria for diagnosing metabolic syndrome usually include a combination of factors such as central obesity (usually measured by waist circumference), elevated blood pressure, elevated fasting glucose, high triglycerides, and low high-density lipoprotein (HDL) cholesterol.

Metabolic syndrome: NCEP-ATP III
1\. Central obesity:
\>102 cm (India-90cm) in men
\>88 cm (India-80cm) in women
2\. Elevated triglycerides: \>150 mg/dL
3\. HDL
< 40 mg/dL in men
< 50 mg/dL in women.
4\. Blood pressure: \>130/85 mm Hg
5\. Fasting glucose: \>100 mg/dL

Metabolic syndrome increases risk of:
• Type 2 diabetes
• Cardiovascular disease
• Stroke

Management Focus on:
Lifestyle
• weight reduction
• exercise
• diet control

Pharmacological
• statins (lipids)
• antihypertensives
• metformin (insulin resistance)

QUESTION

A 30-year-old patient with heart failure and poorly controlled type 2 diabetes (HbA1c of 7.8%) present with progressive dyspnea. Which is the preferred antidiabetic drug?

A **Dapagliflozin** CORRECT

B Glimepiride

C Metformin

D Sitagliptin

RIGHT ANSWER

OK **A. Dapagliflozin**

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1c” here and does not include a written explanation for this item.

QUESTION

Which of the following drugs used in the management of diabetes is associated with unexplained diarrhea?

A Pioglitazone

B Sitagliptin

C Glimepiride

D **Metformin** CORRECT

RIGHT ANSWER

OK

D. Metformin

WHY THIS IS RIGHT

Correct answer is D because metformin commonly causes gastrointestinal adverse effects including diarrhea due to increased intestinal glucose utilization and altered gut motility.

Mechanism	Effect
↑ intestinal motility	faster transit
↓ glucose absorption	osmotic effect
↑ bile salt malabsorption	irritation
Altered gut microbiota	fermentation changes

This side effect is dose related and often improves with gradual dose escalation or extended-release formulations. Metformin is a biguanide that primarily acts on the liver. Key actions:
1□□ ↓ Hepatic gluconeogenesis
2□□ ↑ Peripheral insulin sensitivity
3□□ ↓ Intestinal glucose absorption

Pioglitazone causes edema/weight gain. Sitagliptin is generally well tolerated. Glimepiride mainly causes hypoglycemia.

QUESTION

Which of the following drugs used in the management of type 2 diabetes is associated with an increased risk of urinary tract infections?

A **Dapagliflozin** CORRECT

B Pioglitazone

C Sitagliptin

D Metformin

OK RIGHT ANSWER

A. Dapagliflozin

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1d” here and does not include a written explanation for this item.

QUESTION

All of the following are criteria for diagnosis of DKA except:

A S. glucose > 250 mg/dL

B Arterial pH < 7.3

C Anion gap > 12

D **S. Potassium < 3.5 mEq/L** CORRECT

RIGHT ANSWER

OK

D. S. Potassium < 3.5 mEq/L

WHY THIS IS RIGHT

Correct answer is D because diagnostic criteria for DKA include hyperglycemia (>250 mg/dL), metabolic acidosis (pH < 7.3), and increased anion gap metabolic acidosis.

Insulin deficiency
□
↑ lipolysis
□
↑ free fatty acids
□
Liver -> ketone bodies
□
Metabolic acidosis (high anion gap)

Serum potassium is often normal or elevated initially due to extracellular shift despite total body potassium depletion. Hypokalemia is a complication during treatment, not a diagnostic criterion.

QUESTION

In uncontrolled diabetes mellitus, which of the following cell types exhibit decreased glucose uptake as a result of insulin deficiency?

A RBC

B **Skeletal Muscle** CORRECT

C Liver

D Brain

RIGHT ANSWER

OK **B. Skeletal Muscle**

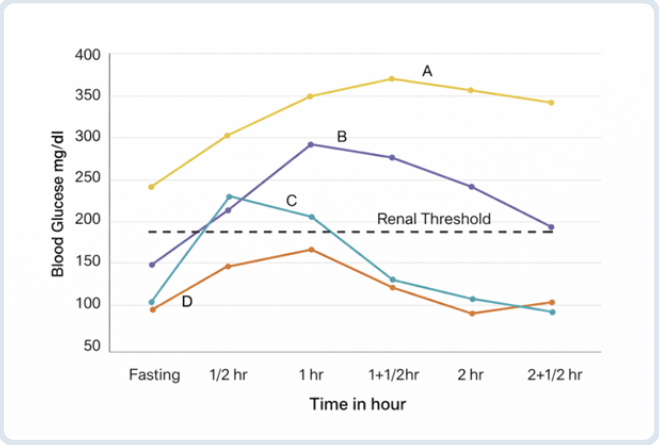
WHY THIS IS RIGHT

Correct answer is B because skeletal muscle requires insulin-dependent GLUT-4 transporters for glucose uptake, so insulin deficiency markedly reduces glucose entry into these cells.
RBCs and brain use insulin-independent transporters (GLUT-1/GLUT-3). Liver uptake via GLUT-2 is also insulin independent.

Transporter	Location	Function
GLUT-1	Brain, kidney, placenta, erythrocytes	Basal uptake of glucose, high affinity
GLUT-2	Liver, pancreatic cell, small intestine	Fed state
GLUT-3	Brain, kidney, placenta	Glucose uptake, maximum affinity for glucose
GLUT-4	Heart and skeletal muscle, adipose tissue	Insulin-stimulated glucose uptake
GLUT-5	Small intestine, sperms	Fructose

QUESTION

Oral glucose tolerance test with blood glucose levels is plotted against time. Which among the following is indicative of diabetes mellitus?



A A,B

B **A,B,C** CORRECT

C A only

D A, B, C, D

OK RIGHT ANSWER

B. A,B,C

WHY THIS IS RIGHT

The OGTT evaluates how efficiently the body handles a glucose load.
Procedure:

1. Patient fasts 8-10 hours
2. Measure fasting plasma glucose
3. Give 75 g oral glucose
4. Measure glucose at intervals
• 30 min
• 1 hr
• 2 hr (most important)

Correct answer is B because curves A, B, C remain above the renal threshold for a prolonged period and show delayed return toward baseline, indicating impaired glucose tolerance consistent with diabetes.
Persistent hyperglycemia beyond 2 hours is the key diagnostic feature of diabetes on OGTT.

Value	Diagnosis
<140	Normal
140-199	Prediabetes
≥200	Diabetes

Curve D shows a normal rise and fall with return to baseline, representing normal glucose handling.

QUESTION

A pregnant female presents with amenorrhea and galactorrhea. MRI shows pituitary adenoma. What is the best management of this patient?

A Cabergoline

B **Bromocriptine** CORRECT

C Transsphenoidal surgery

D Goserelin

OK RIGHT ANSWER

B. Bromocriptine

WHY THIS IS RIGHT

In pregnancy, the drug of choice for prolactinoma is: Bromocriptine
Because:
• Safe in pregnancy
• Extensive clinical experience
• Effectively lowers prolactin

Hypothalamus releases dopamine
□
Dopamine acts on pituitary (D2 receptors)
□
↓ Prolactin secretion

Bromocriptine is the preferred dopamine agonist in prolactinoma during pregnancy because of its long safety record for fetal exposure. It suppresses prolactin and reduces tumor size while preserving fertility.
Surgery is reserved for drug-resistant or compressive tumors.

QUESTION

A 28-year-old male presents with complaints of excessive thirst and frequent urination throughout the day. He mentions having to wake up multiple times at night to urinate. On further questioning, he reports a history of head injury one month ago. His fasting blood glucose is normal. What is the most appropriate next step in management?

A Tolvaptan

B Insulin

C **Desmopressin** CORRECT

D Hydrochlorothiazide

OK RIGHT ANSWER

C. Desmopressin

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1e” here and does not include a written explanation for this item.

QUESTION

A middle-aged male patient presents with chin protrusion, excessive sweating, hormone receptor antagonist used to treat this condition?

A Octreotide

B **Pegvisomant** CORRECT

C Cabergoline

D Olcegepant

OK RIGHT ANSWER

B. Pegvisomant

WHY THIS IS RIGHT

There are three main drug classes used in acromegaly, and examiners often test their differences:

1. Somatostatin analogues -> decrease GH secretion
2. Dopamine agonists -> decrease GH (mild effect)
3. GH receptor antagonists -> block GH action

Pegvisomant belongs to the third category
The features describe acromegaly, and pegvisomant is a GH receptor antagonist that blocks peripheral GH action and lowers IGF-1. It is used when surgery or somatostatin analogs are inadequate.
Octreotide suppresses GH release but is not a receptor antagonist. Cabergoline is a dopamine agonist, and olcegepant is unrelated to GH.
A. Octreotide
• Somatostatin analogue
• ↓ GH secretion from pituitary
• Not a receptor antagonist

C. Cabergoline
• Dopamine agonist
• Mildly suppresses GH
• Mainly used in prolactinoma

D. Olcegepant
• CGRP antagonist
• Used in migraine
• No role here

QUESTION

A pituitary tumor that overproduced growth hormone was surgically removed from a patient. The resection was discovered to be insufficient. What is the patient's first-line treatment?

A Leuprolide

B Goserelin

C Nafarelin

D **Octreotide** CORRECT

RIGHT ANSWER

OK

D. Octreotide

WHY THIS IS RIGHT

Persistent acromegaly after incomplete surgery is treated first-line with somatostatin analogues.
Surgery (first-line)
□
If incomplete or residual tumor
□
Medical therapy (first-line drug = somatostatin analogue)
□
Radiotherapy (if needed)
Therefore, after incomplete surgery -> Octreotide

Octreotide suppresses GH secretion from pituitary adenoma and lowers IGF-1 levels. It is the preferred medical therapy when surgery is inadequate. GnRH analogues like leuprolide/goserelin/nafarelin have no role in GH-secreting tumors.

Situation	Best Drug
Residual acromegaly after surgery	□ Octreotide
Refractory to somatostatin analogues	Pegvisomant
Mild disease	Cabergoline

QUESTION

A female patient with infertility presents with galactorrhea. Evaluation reveals a 7mm prolactinoma. She is worried about her conception chances. What is the appropriate drug in this situation?

A **Cabergoline** CORRECT

B Bromocriptine

C Octreotide

D Pegvisomant

RIGHT ANSWER

OK

A. Cabergoline

WHY THIS IS RIGHT

Cabergoline is the preferred first-line therapy for prolactinoma because it is a long-acting dopamine agonist that effectively suppresses prolactin and shrinks tumor size.
Cabergoline = dopamine (D2) agonist
Mechanism:
Stimulates dopamine receptors in pituitary
□
Inhibits prolactin secretion
□
↓ Prolactin levels
□
Restores ovulation
□
Improves fertility
Normalization of prolactin restores ovulation and improves fertility. It is better tolerated and more effective than bromocriptine.

Feature	Cabergoline	Bromocriptine
Potency	Higher	Lower
Duration	Long acting	Short acting
Side effects	Fewer	More
Compliance	Better	Poor

Octreotide and pegvisomant act on GH, not prolactin.

QUESTION

A patient came to the medical OPD with complaints of polyuria. He has a history of undergoing total hypophysectomy. His Na+ levels are found to be 155 mEq/L, urine osmolality was 200 mOsm/L. What is the definitive management?

A DDAVP for 2 weeks and then discontinue

B **DDAVP supplementation for lifelong** CORRECT

C Upsetting of receptors so no treatment is required

D Thiazides for 2 weeks

RIGHT ANSWER

OK **B. DDAVP supplementation for lifelong**

WHY THIS IS RIGHT

Total hypophysectomy causes central diabetes insipidus due to permanent ADH deficiency.
ADH normally acts on kidney collecting ducts
□
Inserts aquaporin channels
□
Water reabsorption
□
Concentrated urine

Parameter	Finding	Meaning
Serum Na□	155	Water loss (hypernatremia)
Urine osmolality	200	Dilute urine
History	Hypophysectomy	Central cause

Hypernatremia with low urine osmolality confirms inability to concentrate urine. Definitive therapy is lifelong desmopressin (DDAVP) replacement to restore water reabsorption.
Temporary therapy or thiazides are used in nephrogenic DI, not central DI.

Feature	Central DI	Nephrogenic DI
ADH	↓	Normal
Cause	Pituitary damage	Kidney resistance
Treatment	□ Desmopressin	Thiazides
Response to ADH	Yes	No

QUESTION

A 45-year-old patient presents with large, sweaty hands, macroglossia, and prominent frontal bossing. The patient also reports increasing shoe size and difficulty wearing rings that previously fit. Considering the clinical features, which of the following tests is the most appropriate initial investigation?

A GHRH levels

B **Age-matched IGF-1 levels** CORRECT

C Random GH levels

D GH levels after 2 hours of administration of 75g glucose

OK RIGHT ANSWER

B. Age-matched IGF-1 levels

WHY THIS IS RIGHT

Growth hormone (GH) is secreted in pulses
Levels fluctuate throughout the day
Random GH measurement becomes unreliable

GH acts on liver
Liver produces IGF-1
IGF-1 levels remain stable
Reflect overall GH activity
The features suggest acromegaly, and the best initial screening test is age-adjusted IGF-1 because it reflects integrated GH secretion and is persistently elevated.
Random GH fluctuates due to pulsatile release and is unreliable. Failure of GH suppression after oral glucose is confirmatory, not the first step. GHRH levels are not used for routine screening.
GH after glucose (OGTT suppression test)
This is a confirmatory test, not initial.
In normal:
Glucose
GH suppressed
In acromegaly:
GH remains high

QUESTION

Which of the following growth hormone analogue that is not used for treatment of GH deficiency but is used in the treatment of HIV-associated lipodystrophy?

A **Tesamorelin** CORRECT

B Sermorelin

C Somatropin

D Pegvisomant

OK RIGHT ANSWER

A. Tesamorelin

WHY THIS IS RIGHT

Tesamorelin is a GHRH analogue used to reduce visceral adiposity in HIV-associated lipodystrophy by stimulating endogenous GH release. It is not used for routine treatment of GH deficiency.

Drug	Type	Main Use
Somatropin	Recombinant GH	□ GH deficiency
Sermorelin	GHRH analogue	GH deficiency (stimulates GH)
Pegvisomant	GH receptor antagonist	Acromegaly
Tesamorelin □	GHRH analogue	HIV lipodystrophy

Somatropin is recombinant GH for deficiency states, while pegvisomant is a GH receptor antagonist used in acromegaly. Sermorelin is used diagnostically or for GH deficiency, not lipodystrophy.

QUESTION

A 14-year-old developed rapid growth and had long legs and large hands, and coarse facial features. Which of the following tests are used to make a confirmatory diagnosis?

A Increased GH levels

B Increased IGF1

C **No supression of GH after glucose administration** CORRECT

D No supression of IGF1 after glucose tolerance test

RIGHT ANSWER

OK

C. No supression of GH after glucose administration

WHY THIS IS RIGHT

The confirmatory test for suspected gigantism/acromegaly is failure of GH suppression after oral glucose load. Normally, hyperglycemia suppresses GH to very low levels; persistent elevation confirms autonomous GH secretion.

Test	Role	Why
IGF-1	Screening	Stable, reflects GH
OGTT (GH suppression test) □	Confirmatory	Tests feedback control
Random GH	Not useful	Fluctuates

Random GH levels fluctuate and are unreliable, while IGF-1 is a screening marker, not confirmatory. IGF-1 is not used for suppression testing.

Feature	Gigantism	Acromegaly
Age	Children	Adults
Epiphysis	Open	Closed
Growth	Height ↑	No height ↑
Features	Long limbs	Coarse features

QUESTION

In a patient with a prolactinoma, the mechanism by which prolactin causes amenorrhea is through which of the following actions?

A Downregulation of estrogen receptors

B **Decreasing GnRH** CORRECT

C Decreasing FSH

D Increasing pulse frequency of LH

OK RIGHT ANSWER

B. Decreasing GnRH

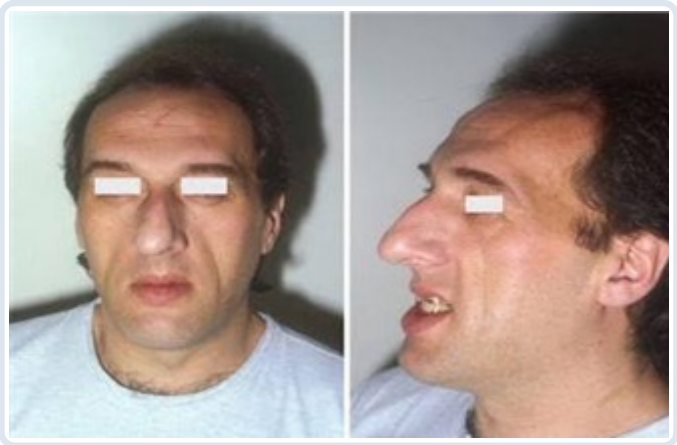
WHY THIS IS RIGHT

↑ Prolactin
□
↓ GnRH (from hypothalamus)
□
↓ LH and ↓ FSH (from pituitary)
□
↓ Ovarian estrogen production
□
Anovulation
□
Amenorrhea
Hyperprolactinemia inhibits hypothalamic GnRH secretion, suppressing the pulsatile release required for normal gonadotropin production. Reduced GnRH leads to decreased LH and FSH, causing anovulation and amenorrhea. This central inhibition is the primary mechanism in prolactinoma. Effects on estrogen receptors are secondary and not the initiating defect.

Parameter	Normal	Prolactinoma
GnRH	Normal	↓
LH/FSH	Normal	↓
Ovulation	Present	Absent
Menstruation	Regular	Absent

QUESTION

These findings are suggestive of which of the following conditions?



A Addison's disease

B Gigantism

C **Acromegaly** CORRECT

D Cushing's syndrome

RIGHT ANSWER

OK **C. Acromegaly**

WHY THIS IS RIGHT

Acromegaly results from excess growth hormone after epiphyseal closure, leading to soft tissue overgrowth and enlarged hands, feet, jaw, and facial bones. Classic features include coarse facial features, prognathism, and organomegaly. Gigantism occurs before epiphyseal closure and presents with tall stature rather than acral enlargement.

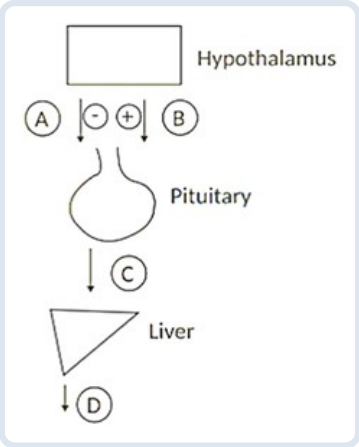
Feature	Gigantism	Acromegaly
Age	Children	Adults
Epiphysis	Open	Closed
Height	↑	No increase
Features	Long limbs	Coarse features □

System	Feature
Face	Prognathism, frontal bossing
Extremities	Large hands/feet
Tongue	Macroglossia
Skin	Thick, oily
Metabolic	Insulin resistance

Addison’s and Cushing’s do not produce bony overgrowth or acral hypertrophy.

QUESTION

Identify the hormones represented by the following:



A A - Somatostatin B - GH C - GHRH D - IGF1

B **A - Somatostatin B - GHRH C - GH D - IGF1** CORRECT

C A - IGF1 B - GHRH C - GH D - Somatostatin

D A - GHRH B - Somatostatin C - GH D - IGF1

OK RIGHT ANSWER

B. A - Somatostatin B - GHRH C - GH D - IGF1

WHY THIS IS RIGHT

Correct answer is B because hypothalamus releases inhibitory somatostatin (A) and stimulatory GHRH (B) to regulate pituitary secretion.
The pituitary then releases growth hormone (C), which acts on the liver.
The liver produces IGF-1 (D), mediating peripheral growth effects and feedback control.

QUESTION

Many GPCR receptors act via an increase in cAMP. The steps involved in this mechanism are given below. Which of the following is the correct sequence of events in the cAMP signal transduction pathway?

1. Activation of adenylate cyclase
2. Activation of PKA
3. Conversion of ATP to cAMP
4. Phosphorylation of target proteins

A 1, 2, 3 and 4

B **1, 3, 2 and 4** CORRECT

C 1, 3, 4 and 2

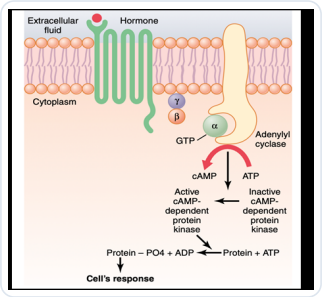
D 4, 2, 1 and 3

OK RIGHT ANSWER

B. 1, 3, 2 and 4

WHY THIS IS RIGHT

Correct answer is B because GPCR activation first stimulates adenylate cyclase (1), which converts ATP to cAMP (3). cAMP then activates protein kinase A (PKA) (2).
Activated PKA phosphorylates downstream target proteins (4), producing the cellular response.



QUESTION

A 30-year-old woman, having lost her newborn, is producing breast milk, which risks developing into a breast abscess due to milk stasis and incomplete emptying. Which drug can prevent this complication?

A Mifepristone

B **Cabergoline** CORRECT

C Chlorpromazine

D Metoclopramide

OK RIGHT ANSWER

B. Cabergoline

WHY THIS IS RIGHT

Cabergoline is a dopamine (D2) agonist that:
 Inhibits prolactin secretion
 Suppresses lactation

Correct answer is B because cabergoline is a dopamine agonist that suppresses prolactin secretion, thereby inhibiting lactation and preventing milk stasis.
Reducing prolactin stops continued milk production and lowers risk of engorgement and abscess.
Metoclopramide and chlorpromazine increase prolactin. Mifepristone has no role in lactation suppression.

Drug	Effect on Prolactin
Cabergoline / Bromocriptine	↓ prolactin
Antipsychotics	↑ prolactin
Metoclopramide	↑ prolactin

QUESTION

Match the Following hormones to receptors:

Column A	Column B
1. Insulin	A. Tyrosine Kinase Pathway
2. Growth Hormone	B. JAK STAT pathway
3. Acetylcholine	C. Ligand gated channel
4. Levothyroxine	D. Nuclear Receptor

A 1-C, 2-B, 3-D, 4-A

B 1-D, 2-A, 3-C, 4-B

C **1-A, 2-B, 3-C, 4-D** CORRECT

D 1-B, 2-D, 3-A, 4-C

OK RIGHT ANSWER

C. 1-A, 2-B, 3-C, 4-D

WHY THIS IS RIGHT

1. Insulin - Tyrosine Kinase Pathway: Insulin uses a tyrosine kinase receptor. This is a cell surface receptor that, when activated, phosphorylates tyrosine residues on target proteins, triggering intracellular signaling cascades.
2. Growth Hormone - JAK-STAT Pathway: Growth hormone, along with prolactin and leptin, uses the JAK-STAT (Janus Kinase-Signal Transducer and Activator of Transcription) pathway. This is a cytokine receptor-mediated signaling mechanism.
3. Acetylcholine - Ligand-gated Channel: At the neuromuscular junction, acetylcholine operates through ligand-gated sodium channels. When acetylcholine binds, these channels open, allowing sodium ions to flow and triggering muscle contraction.
4. Levothyroxine (Thyroid Hormone) - Nuclear Receptor: Thyroid hormones act through nuclear receptors, which means they enter the cell and directly interact with DNA in the nucleus to regulate gene expression.

QUESTION

IGF-1 and IGF-2 (Insulin-like Growth Factors) are structurally most similar to which of the following molecules?

A Insulin

B **Proinsulin** CORRECT

C C peptide

D Preproinsulin

OK RIGHT ANSWER

B. Proinsulin

WHY THIS IS RIGHT

Stepwise synthesis

Stage	Structure
Preproinsulin	signal peptide + A + B + C
Proinsulin	A chain + B chain + C peptide
Insulin	A chain + B chain only

During processing:
• C-peptide is removed
• A and B chains remain linked by disulfide bonds

Correct answer is B because IGF-1 and IGF-2 retain structural similarity to proinsulin, including homologous A and B chains connected by a C-peptide-like domain.
Unlike insulin, IGFs are not cleaved to remove the connecting peptide, so their tertiary structure resembles intact proinsulin.
C-peptide alone and preproinsulin do not share the same functional folded structure as IGFs.

QUESTION

An elderly male patient presents with a cough and weight loss for the last 3 months. Biopsy of the lung mass revealed tumor cells that are positive for chromogranin and synaptophysin. Which of the following paraneoplastic syndromes will most likely occur in this patient?

A Hypercalcemia

B Cushing syndrome

C Gynaecomastia

D **SIADH** CORRECT

OK RIGHT ANSWER

D. SIADH

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1f” here and does not include a written explanation for this item.

QUESTION

A 30-year-old patient presents with hypertension and hypokalemic metabolic alkalosis. CT scan shows a mass of 1.5 cm over the kidney with elevated ARR. What is the next best step for the management of this patient?

A Spironolactone on life-long basis

B **Adrenalectomy** CORRECT

C ACTH stimulation test

D Dexamethasone suppression test

RIGHT ANSWER

OK **B. Adrenalectomy**

WHY THIS IS RIGHT

Once primary hyperaldosteronism is confirmed + localized adenoma, the treatment is: Surgical removal (adrenalectomy)
Aldosterone acts on kidney:

□
↑ Na□ reabsorption
□
↑ Water retention
□
Hypertension
□
↑ K□ excretion
□
Hypokalemia
□
↑ H□ excretion
□
Metabolic alkalosis

Hypertension with hypokalemic metabolic alkalosis and elevated aldosterone-renin ratio indicates primary hyperaldosteronism. A unilateral adrenal mass consistent with an aldosterone-producing adenoma is treated definitively with adrenalectomy. Surgery corrects hypertension and hypokalemia.
Lifelong spironolactone is reserved for bilateral adrenal hyperplasia or non-surgical candidates.

QUESTION

What is the likely diagnosis of a middle-aged patient presented with episodic headache, palpitations and increased 24-hour urinary levels of metanephrines?

A Neuroblastoma

B Carcinoid tumor

C Brain tumor

D **Pheochromocytoma** CORRECT

RIGHT ANSWER

OK

D. Pheochromocytoma

WHY THIS IS RIGHT

Symptoms suggest: Intermittent sympathetic overactivity
Classic triad:
• Headache
• Palpitations
• Sweating

Lab clue:
• ↑ Metanephrines (very specific)

Diagnosis: Pheochromocytoma
Episodic headache, palpitations, and elevated urinary metanephrines are classic for pheochromocytoma. Excess catecholamine secretion produces paroxysmal sympathetic surges and hypertension. Urinary metanephrines are sensitive markers of catecholamine excess.
The other tumors do not produce this characteristic biochemical pattern.

Neuroblastoma
• Occurs in children
• Not typical in adults

Carcinoid tumor
• Causes flushing and diarrhea
• Due to serotonin secretion, not catecholamines

Brain tumor
• May cause headache
• Does not explain palpitations or elevated metanephrines

QUESTION

Which drug is used in the preoperative management of pheochromocytoma?

A **Phenoxybenzamine** CORRECT

B Propranolol

C Metoprolol

D Labetalol

OK RIGHT ANSWER

A. Phenoxybenzamine

WHY THIS IS RIGHT

First step is irreversible alpha-blockade with phenoxybenzamine to prevent hypertensive crisis.

Alpha blockers are given first-

Action	Result
Blocks α1 receptors	Vasodilation
Blocks α2 receptors	Reduced catecholamine effect
Prevents hypertensive crisis	During tumor manipulation

If a beta blocker is given before alpha blockade: β2 vasodilation is blocked, α-mediated vasoconstriction becomes unopposed which results in Severe hypertensive crisis
Beta-blockers are added only after adequate alpha blockade.

QUESTION

A 34-year-old patient presents with lethargy and tiredness. On examination, he has hyperpigmentation around the lips. His BP is 90/60 mm Hg and heart rate is 110/min. Labs revealed a Na□ of 125 mEq/L and a K□ of 5.5 mEq/L. What is the most appropriate treatment?

A ACTH

B **Hydrocortisone and fludrocortisone** CORRECT

C Dexamethasone

D NaCl infusion

OK RIGHT ANSWER

B. Hydrocortisone and fludrocortisone

WHY THIS IS RIGHT

Finding	Meaning
Hyperpigmentation	↑ ACTH -> primary cause
Hypotension	cortisol deficiency
Hyponatremia	aldosterone deficiency
Hyperkalemia	aldosterone deficiency

Findings suggest primary adrenal insufficiency with cortisol and aldosterone deficiency causing hypotension, hyponatremia, hyperkalemia, and hyperpigmentation. Treatment requires glucocorticoid plus mineralocorticoid replacement.
GLUCOCORTICOID:
Hydrocortisone -> 15-25 mg/day (divided doses)
• 10 mg morning + 5 mg afternoon (± 5 mg evening)

MINERALOCORTICOID:
Fludrocortisone -> 0.05-0.2 mg/day (usually 0.1 mg once daily)

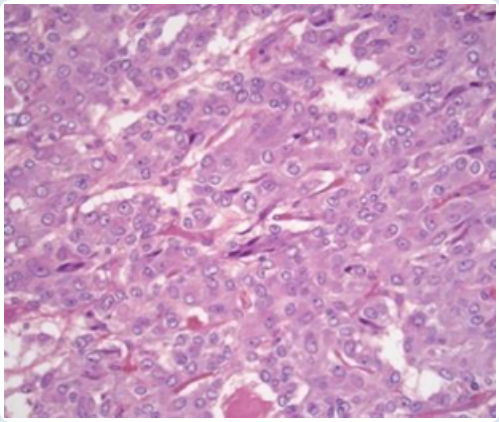
GOALS:
• Maintain BP, electrolytes (Na↑, K↓)
• Avoid hypotension & hyperkalemia

STRESS DOSE (VERY IMPORTANT):
• Increase hydrocortisone 2-3× during illness/surgery

ACUTE CRISIS:
• IV Hydrocortisone 100 mg bolus -> then 200 mg/day

QUESTION

A 55-year-old male presented with headache, palpitations, and hypertension. On evaluation, his urinary VMA and catecholamines were elevated. Imaging revealed an adrenal mass, and the patient underwent surgery and resection. Histology of the mass was shown. Which of the following is true regarding the condition?



A Mostly in children

B Mostly malignant

C **Can be seen in MEN 2A** CORRECT

D Mostly bilateral

OK RIGHT ANSWER

C. Can be seen in MEN 2A

WHY THIS IS RIGHT

Elevated urinary VMA with adrenal mass indicates pheochromocytoma, a catecholamine-secreting tumor.
Tumor of chromaffin cells (adrenal medulla)

Excess catecholamine secretion

• Vasoconstriction -> hypertension
• Cardiac stimulation -> palpitations

Episodic symptoms

Pheochromocytoma is strongly associated with:

Syndrome	Components
MEN 2A	Medullary thyroid carcinoma + pheochromocytoma + parathyroid hyperplasia
MEN 2B	Medullary thyroid carcinoma + pheochromocytoma + mucosal neuromas

Hence option C is correct
It is classically associated with MEN 2A due to RET mutation along with medullary thyroid carcinoma and hyperparathyroidism. Most pheochromocytomas are benign, unilateral, and occur in adults. Bilaterality is mainly seen in familial syndromes, not the majority.

QUESTION

A 21-year-old patient presented with weakness and fatigue. His BP was 80/60 mm Hg and his reports showed a serum Na+ of 127 mEq/L and serum K+ of 5.2 mEq/L. On examination, the patient had hyperpigmentation in the knuckles, arms, and legs. What is the diagnosis?

A Conns syndrome

B **Primary adrenal insufficiency** CORRECT

C B12 deficiency

D SIADH

OK RIGHT ANSWER

B. Primary adrenal insufficiency

WHY THIS IS RIGHT

Hypotension with hyponatremia, hyperkalemia, and diffuse hyperpigmentation is classic for primary adrenal insufficiency. Loss of aldosterone causes salt wasting and hyperkalemia, while cortisol deficiency raises ACTH, leading to pigmentation. This electrolyte pattern excludes SIADH, which has no hyperkalemia. Conn syndrome causes hypertension and hypokalemia, the opposite profile.

Condition	Na□	K□	BP	Pigmentation
Addison's	↓	↑	↓	□ Present
Conn's syndrome	↑	↓	↑	Absent
SIADH	↓	Normal	Normal	Absent
B12 deficiency	Normal	Normal	Normal	May have pigmentation but no electrolyte changes

• Hyperpigmentation = primary adrenal failure □
• Hyponatremia + hyperkalemia = aldosterone deficiency
• Hypotension = cortisol deficiency
• Most common cause = autoimmune

QUESTION

Which of the following is the correct statement about a patient with a retroperitoneal mass, episodic hypertension, palpitations, and increased urinary VMA levels?

A It is mostly in children

B It is usually malignant

C It is mostly bilateral

D **It can be associated with MEN 2A** CORRECT

RIGHT ANSWER

OK

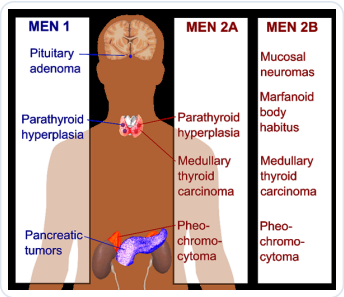
D. It can be associated with MEN 2A

WHY THIS IS RIGHT

Syndrome	Components
MEN 2A □	Medullary thyroid carcinoma + pheochromocytoma + parathyroid hyperplasia
MEN 2B	Medullary thyroid carcinoma + pheochromocytoma + mucosal neuromas

The presentation suggests pheochromocytoma, a catecholamine-secreting tumor classically causing episodic hypertension and elevated urinary VMA. Pheochromocytoma is associated with MEN 2A due to RET mutation along with medullary thyroid carcinoma and hyperparathyroidism.

Most tumors are benign, unilateral, and occur in adults. Bilateral tumors are mainly seen in familial syndromes.



QUESTION

A patient with hypertensive episodes, with an MRI abdomen showing a suprarenal mass, is planned for surgery. A 24-hour urine shows elevated metanephrine levels. What drug will be given preoperatively and also intraoperatively?

A Nicardipine

B Esmolol

C Clonidine

D **Phenoxybenzamine** CORRECT

RIGHT ANSWER

OK

D. Phenoxybenzamine

WHY THIS IS RIGHT

Phenoxybenzamine is a nonselective irreversible α-blocker used preoperatively in pheochromocytoma to prevent catecholamine-induced hypertensive crises.
Phenoxybenzamine = irreversible α-blocker
Blocks α□ and α□ receptors
Causes vasodilation

Preoperative
• Classically Phenoxybenzamine (α-blocker) -> given days before surgery

Intraoperative BP control
• Need short-acting, titratable drug
• Nicardipine (IV CCB) is preferred:
• Rapid onset
• Easily adjustable
• Controls hypertensive surges during tumor manipulation

Question asks: “preoperative AND intraoperative”
• Among options, Phenoxybenzamine seems most appropriate due to its traditional use-case in pheochromocytoma.

QUESTION

A 20-year-old patient presented with salt craving, skin pigmentation, hyponatremia, hyperkalemia, and metabolic acidosis. What is the likely diagnosis?

A Cushing syndrome

B **Addison's disease** CORRECT

C Pheochromocytoma

D Conn syndrome

OK RIGHT ANSWER

B. Addison's disease

WHY THIS IS RIGHT

Finding	What it indicates
Salt craving	Aldosterone deficiency
Hyperpigmentation	↑ ACTH -> primary cause
Hyponatremia	↓ aldosterone
Hyperkalemia	↓ aldosterone
Metabolic acidosis	↓ H□ excretion

Primary adrenal insufficiency causes deficiency of both cortisol and aldosterone, leading to salt wasting, hyponatremia, hyperkalemia, and metabolic acidosis. Elevated ACTH produces characteristic skin hyperpigmentation and salt craving reflects mineralocorticoid loss. This constellation is classic for Addison’s disease. Cushing and Conn syndromes cause hypertension and hypokalemia, not salt loss.

Condition	Na□	K□	BP	Pigmentation
Addison’s	↓	↑	↓	□ Present
Cushing	↑	↓	↑	Absent
Conn syndrome	↑	↓	↑	Absent
Pheochromocytoma	Normal	Normal	↑ (episodic)	Absent

QUESTION

Increase in which of the following hormone is responsible for Hyperpigmentation in Addison's disease?

A ACTH — CORRECT

B Aldosterone

C ADH

D Cortisol

OK RIGHT ANSWER

A. ACTH

WHY THIS IS RIGHT

↓ Cortisol (adrenal failure)
□
Loss of negative feedback
□
↑ ACTH secretion (from pituitary)
□
ACTH derived from POMC (pro-opiomelanocortin)
□
POMC also produces MSH (melanocyte-stimulating hormone)
□
↑ Melanin production
□
Hyperpigmentation
In Addison disease, low cortisol removes negative feedback on the pituitary, causing marked ACTH elevation. ACTH is derived from POMC, which also produces MSH, stimulating melanocytes and causing diffuse hyperpigmentation.

Component	Derived From	Function
ACTH	POMC	Stimulates adrenal cortex
MSH	POMC	Stimulates melanocytes
Cortisol	Adrenal cortex	Negative feedback

Aldosterone and ADH do not affect skin pigmentation. Cortisol is decreased, not increased, in primary adrenal insufficiency.

QUESTION

A woman previously treated for Cushing disease underwent bilateral adrenalectomy 2 years ago. She now presents with progressive hyperpigmentation, headaches, and new-onset visual field defects. MRI shows an enlarging pituitary mass with markedly elevated ACTH levels. What is the most likely diagnosis?

A **Nelson syndrome** CORRECT

B Sheehan syndrome

C Conn's syndrome

D Addison's disease

OK RIGHT ANSWER

A. Nelson syndrome

WHY THIS IS RIGHT

The source quiz contains the placeholder “$20” here and does not include a written explanation for this item.

QUESTION

A patient was brought to the emergency department in an unconscious state. The patient is dehydrated and the blood pressure of the patient is 90/60 mm Hg. Laboratory investigations revealed the serum potassium level of 6.0 mEq/L, sodium level of 129 mEq/L, and blood glucose level of 40 mg/dL. The patient has a history of rheumatoid arthritis (RA) for which he was prescribed medication by a local physician; however, the patient abruptly discontinued the medication a few weeks ago. Based on the clinical presentation and laboratory findings, which of the following drugs should be administered as a life-saving measure to treat this patient?

A **Hydrocortisone** CORRECT

B Sodium bicarbonate

C Insulin

D Hypertonic saline

OK RIGHT ANSWER

A. Hydrocortisone

WHY THIS IS RIGHT

Correct answer is A because abrupt withdrawal of chronic steroids in an RA patient can precipitate acute adrenal crisis with hypotension, hypoglycemia, hyponatremia, and hyperkalemia.
Cortisol deficiency-

Effect	Result
↓ gluconeogenesis	Hypoglycemia
↓ vascular tone	Hypotension
↓ stress response	Shock

This reflects cortisol and aldosterone deficiency causing shock and electrolyte imbalance.
Immediate life-saving treatment is IV hydrocortisone with fluids, which corrects both glucocorticoid and mineralocorticoid deficiency.
Hydrocortisone provides:
Glucocorticoid effect (cortisol replacement)
Mineralocorticoid effect (aldosterone-like action)
□ Corrects:
• hypotension
• hypoglycemia
• electrolyte imbalance

Immediate steps:
1□□ IV Hydrocortisone
2□□ IV fluids (normal saline + dextrose)
3□□ Correct electrolytes

QUESTION

A 45-year-old man presented with weight gain, proximal muscle weakness, purple striae, and easy bruising. What is the likely underlying condition?

A **Hypercortisolism** CORRECT

B Hyperthyroidism

C Hyperparathyroidism

D Hyperaldosteronism

OK RIGHT ANSWER

A. Hypercortisolism

WHY THIS IS RIGHT

Correct answer is A because weight gain, proximal muscle weakness, purple striae, and easy bruising are classic features of Cushing syndrome due to excess cortisol.
Hypercortisolism causes protein catabolism, skin thinning, and central fat redistribution.

Mechanism	Clinical Effect
↑ Protein breakdown	Muscle weakness
↓ Collagen synthesis	Thin skin -> easy bruising
Fat redistribution	Central obesity
Skin thinning	Purple striae

Causes of Hypercortisolism

Type	Cause
Exogenous	Steroid therapy (most common)
ACTH-dependent	Pituitary adenoma (Cushing disease)
	Ectopic ACTH (small cell lung cancer)
ACTH-independent	Adrenal adenoma/carcinoma

Thyroid, parathyroid, or aldosterone excess do not produce this characteristic constellation of findings.

QUESTION

Which of the following is not a stimulus for catecholamine synthesis?

A Smoking

B Standing posture

C **Hyperglycemia** CORRECT

D Exercise

RIGHT ANSWER

OK

C. Hyperglycemia

WHY THIS IS RIGHT

The source quiz contains the placeholder “$21” here and does not include a written explanation for this item.

QUESTION

Which of the following is considered the initial recommended test for diagnosing pheochromocytoma?

A **24 hr urinary total metanephrines** CORRECT

B MIBG scan

C Plasma catecholamines

D MRI of the whole abdomen

RIGHT ANSWER

OK **A. 24 hr urinary total metanephrines**

WHY THIS IS RIGHT

Pheochromocytoma is a catecholamine-secreting tumor of chromaffin cells.
Most arise from: Adrenal medulla
Extra-adrenal tumors are called: Paragangliomas
Correct answer is A because the preferred initial screening test for pheochromocytoma is measurement of metanephrines, which reflect continuous catecholamine metabolism and have high sensitivity.
24-hour urinary fractionated metanephrines are recommended for initial biochemical diagnosis.
Imaging (MRI/MIBG) is done only after biochemical confirmation. Plasma catecholamines fluctuate and are less reliable for screening.
Pheochromocytoma can occur in several genetic syndromes.

Syndrome	Gene
MEN 2A / MEN 2B	RET mutation
Von Hippel-Lindau disease	VHL gene
Neurofibromatosis type 1	NF1 gene

QUESTION

Which of the following is not seen in Conn's syndrome?

A Hypertension

B **Hypernatremia** CORRECT

C Hypokalemia

D Metabolic Alkalosis

OK RIGHT ANSWER

B. Hypernatremia

WHY THIS IS RIGHT

Conn’s syndrome = Primary hyperaldosteronism
Cause: Excess aldosterone secretion from adrenal cortex independent of RAAS regulation.
Correct answer is B because Conn’s syndrome (primary hyperaldosteronism) causes sodium retention with volume expansion, but serum sodium is usually near normal due to escape mechanisms. Even though sodium is retained: The body activates escape mechanisms:
Aldosterone Escape
• Pressure natriuresis
• ANP release
• Increased GFR

These prevent significant sodium accumulation in plasma.
Therefore:

Sodium effect
Total body sodium ↑
Serum sodium ≈ normal

Hence hypernatremia is uncommon.
It classically presents with hypertension, hypokalemia, and metabolic alkalosis from increased potassium and hydrogen ion loss.
Thus, hypernatremia is not a typical finding.

QUESTION

A 38-year-old female patient presents with buffalo hump, moon facies, and abdominal striae. She is a known case of SLE on steroids. Which of the following is the next best investigation that should be done in her?

A **Serum Cortisol with ACTH levels** CORRECT

B High-dose dexamethasone suppression test

C Low-dose dexamethasone followed by high-dose dexamethasone suppression test

D ACTH levels followed by the high-dose dexamethasone suppression test

RIGHT ANSWER

OK

A. Serum Cortisol with ACTH levels

WHY THIS IS RIGHT

The source quiz contains the placeholder “$22” here and does not include a written explanation for this item.

QUESTION

A middle aged patient presenting with change in her voice, anemia, recent hair loss and cold intolerance has been diagnosed with Hashimoto's thyroiditis. Which of the following antibody is commonly seen in such patients?

A TSIg

B **Anti thyroid peroxidase (TPO) antibody** CORRECT

C Anti thyroid binding globulin

D Anti thyroid receptor antibody

RIGHT ANSWER

OK **B. Anti thyroid peroxidase (TPO) antibody**

WHY THIS IS RIGHT

Hashimoto thyroiditis is an autoimmune destruction of the thyroid gland leading to hypothyroidism.

It is characterized by high titers of anti-thyroid peroxidase (TPO) antibodies.
Anti-TPO antibodies mediate thyroid follicular cell damage via immune mechanisms.

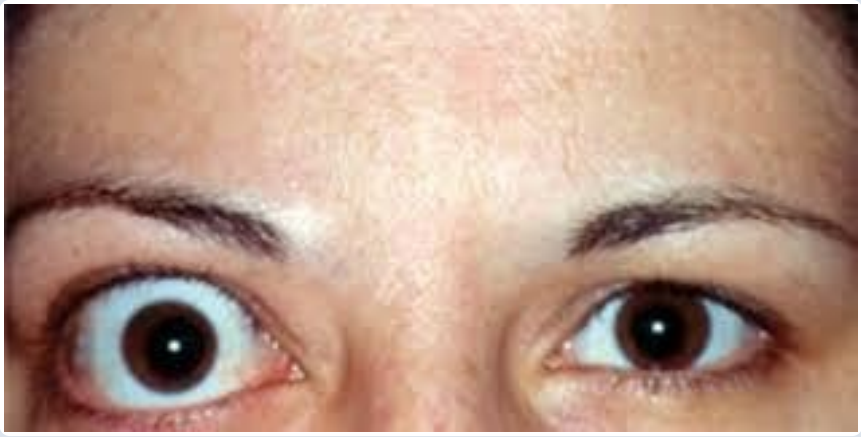
Thyroid peroxidase (TPO) function:
• Oxidation of iodide
• Iodination of tyrosine
• Coupling -> T3 & T4 formation

Anti-TPO antibodies:
• Attack TPO enzyme
• Impair hormone synthesis
• Cause gland destruction

 Hallmark of Hashimoto thyroiditis
TSIg and anti-TSH receptor antibodies are associated with Graves' disease, not Hashimoto thyroiditis.

QUESTION

A 23-year-old medical student with a 2-month history of palpitations, sweating, and restlessness. He also complains of a sweaty palm. His appearance is as shown in the picture. What is the next step in evaluation?



A Thyroid receptor antibody

B Radioactive Iodine Uptake

C **Ultrasensitive TSH** CORRECT

D Anti-thyroid peroxidase antibody

RIGHT ANSWER

OK **C. Ultrasensitive TSH**

WHY THIS IS RIGHT

Ultrasensitive TSH is the best initial test in suspected hyperthyroidism because it is the most sensitive marker of thyroid axis status. Thyrotoxicosis typically shows suppressed TSH before T3/T4 become markedly elevated. Antibody tests and radioactive uptake are used later to determine etiology. Screening always begins with TSH.
A. Thyroid receptor antibody
• Used to confirm Graves disease

B. Radioactive iodine uptake (RAIU)
• Used to differentiate causes

D. Anti-TPO antibody
• Seen in Hashimoto thyroiditis

QUESTION

Which of the following is false in patients with thyroid eye disease?

A. It can lead to vision loss

B. **Response to vision symptoms is adjuvant to thyrotoxicosis response** — CORRECT

C. NOSPECS classification is used

D. 10% of euthyroid patients can have eye involvement.

RIGHT ANSWER

OK

B. Response to vision symptoms is adjuvant to thyrotoxicosis response

WHY THIS IS RIGHT

The source quiz contains the placeholder “$19” here and does not include a written explanation for this item.

QUESTION

A young woman presents with weight loss, tachycardia, heat intolerance, and tremors. She has exophthalmos on examination. What is the diagnosis?

A Parkinson's disease

B **Graves' disease** CORRECT

C Addison's disease

D Alzheimer's disease

OK RIGHT ANSWER

B. Graves' disease

WHY THIS IS RIGHT

Graves’ disease is an autoimmune hyperthyroid state characterized by weight loss, tachycardia, heat intolerance, and tremors due to excess thyroid hormone.
Autoantibodies (TSH receptor antibodies)
□
Stimulate TSH receptors
□
↑ T3 and T4 production
□
Hyperthyroidism
Exophthalmos is a hallmark feature caused by autoimmune orbital fibroblast activation and glycosaminoglycan deposition.
• Most common cause of hyperthyroidism = Graves’ disease
• Pathognomonic sign = exophthalmos □
• Caused by TSH receptor antibodies
• TSH ↓, T3/T4 ↑

This eye involvement is specific for Graves among thyrotoxic states. The other options do not produce thyrotoxicosis with ophthalmopathy.

QUESTION

A woman at 8 weeks of gestation is diagnosed with hyperthyroidism. Which of the following is the most appropriate treatment option?

A Methimazole

B Carbimazole

C **Propylthiouracil** CORRECT

D Radioactive iodine

RIGHT ANSWER

OK **C. Propylthiouracil**

WHY THIS IS RIGHT

Propylthiouracil is preferred in the first trimester because methimazole/carbimazole are teratogenic and linked to embryopathy.

Trimester	Drug of Choice	Reason
1st trimester	□ PTU	Less teratogenic
2nd & 3rd trimester	Methimazole	Less hepatotoxic

PTU also reduces peripheral T4->T3 conversion, helping rapid control of thyrotoxicosis. After the first trimester, therapy is often switched to methimazole due to PTU hepatotoxicity risk.

Drug	Mechanism
PTU	↓ T3/T4 synthesis + ↓ T4 -> T3 conversion
Methimazole	↓ T3/T4 synthesis only

Methimazole causes teratogenic effects:

Defect	Description
Aplasia cutis	Absence of scalp skin
Choanal atresia	Nasal blockage
Esophageal atresia	Feeding difficulty

Radioactive iodine is absolutely contraindicated in pregnancy.

QUESTION

Identify the transporter of iodine from follicular cells to follicular cavity within the thyroid gland:

A Na+ I- Symporter

B **Pendrin** CORRECT

C Calmodulin

D Claudin

OK RIGHT ANSWER

B. Pendrin

WHY THIS IS RIGHT

Iodide from blood
□ *(Basolateral membrane)*
Na□/I□ symporter (NIS)
□
Iodide enters follicular cell
□ *(Apical membrane)*
Pendrin □
□
Iodide moves into colloid
□
Thyroid hormone synthesis begins

Transporter	Location	Function
NIS (Na□/I□ symporter)	Basolateral membrane	Blood -> cell
Pendrin □	Apical membrane	Cell -> colloid
TPO (enzyme)	Colloid	Organification

Pendrin transports iodide from thyroid follicular cells across the apical membrane into the follicular lumen for organification. The Na□/I□ symporter works on the basolateral membrane to bring iodide into the cell from blood. Defective pendrin impairs iodide efflux into colloid. This apical transport step is essential for thyroid hormone synthesis.

QUESTION

In the management of a thyroid storm, a critical and life-threatening condition due to an excess of thyroid hormone, a variety of medications are used to control the symptoms and the overactive thyroid. Which of the following drugs is NOT typically used in the treatment of thyroid storm?

A Propranolol

B Lugol's Iodine

C Acetaminophen

D **Aspirin** CORRECT

OK RIGHT ANSWER

D. Aspirin

WHY THIS IS RIGHT

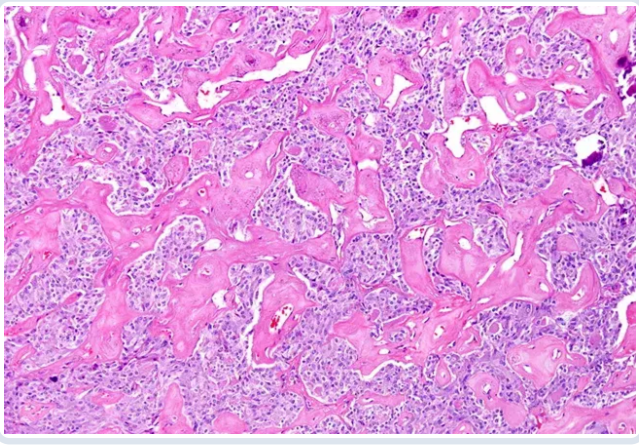
Thyroid storm = extreme thyrotoxicosis -> life-threatening emergency
Management aims to:
• Block hormone synthesis
• Block hormone release
• Control symptoms
• Reduce peripheral effects

Aspirin is avoided in thyroid storm because salicylates displace T3/T4 from thyroid-binding globulin, increasing free hormone levels and worsening thyrotoxicosis.
Beta-blocker (Propranolol)
□
Antithyroid drug (PTU / Methimazole)
□
Iodine (Lugol's iodine)
□
Steroids
□
Supportive care (fluids, cooling, paracetamol)
Propranolol controls adrenergic symptoms and reduces peripheral T4->T3 conversion, while Lugol's iodine blocks hormone release.

Drug	Role in Thyroid Storm
Propranolol □	Controls tachycardia + ↓ T4->T3
PTU	↓ synthesis + ↓ T4->T3
Methimazole	↓ synthesis
Lugol's iodine □	↓ hormone release
Steroids	↓ T4->T3 + adrenal support
Paracetamol (Acetaminophen)	Fever control

QUESTION

A patient presents with neck swelling, compressing the trachea and esophagus. Histopathological assessment shows cell nests and pink extracellular amyloid stroma. What is the cell of origin of this tumor?



A **Parafollicular C cells** CORRECT

B Askanazy cells

C Hurthle cells

D Follicular cells

RIGHT ANSWER

OK **A. Parafollicular C cells**

WHY THIS IS RIGHT

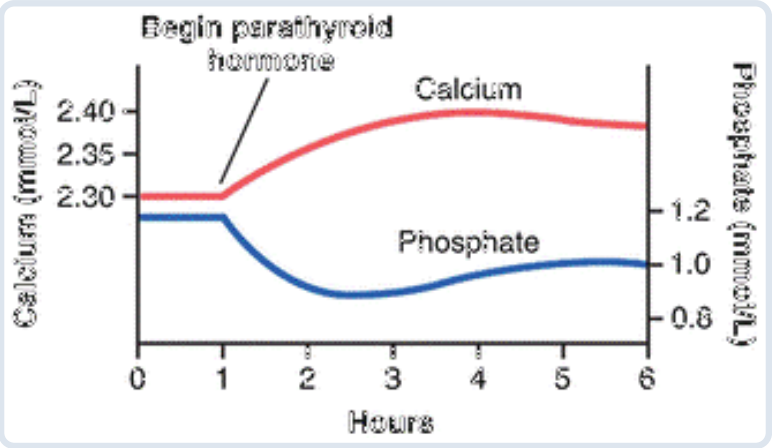
This is a classic description of: Medullary Thyroid Carcinoma (MTC)
Hallmark clues:
• Amyloid stroma
• Neuroendocrine tumor pattern (cell nests)
• Thyroid mass causing compression

Parafollicular C cells
• Derived from neural crest
• Located between thyroid follicles
• Secrete: Calcitonin

The amyloid represents calcitonin-derived deposits produced by malignant parafollicular C cells.
Thus, the tumor originates from neural crest-derived C cells, not follicular or Hurthle cells.

QUESTION

The adjoining graph shows the effect of PTH on blood calcium and phosphate in an experimental animal. How does PTH increase plasma calcium?



A By increasing osteoclast activity

B By increasing osteoblastic activity

C By decreasing renal excretion

D **By increasing osteoclastic activity and decreasing renal excretion of calcium** CORRECT

OK RIGHT ANSWER

D. By increasing osteoclastic activity and decreasing renal excretion of calcium

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1a” here and does not include a written explanation for this item.

QUESTION

PTH increases Calcium in blood. This is due to:

A Rapid release of calcium by osteocytes

B Rapid release of calcium by osteoclasts

C **Rapid release of calcium from bones and increased calcium reabsorption in kidneys** CORRECT

D Increase calcium reabsorption in kidneys

RIGHT ANSWER

OK **C. Rapid release of calcium from bones and increased calcium reabsorption in kidneys**

WHY THIS IS RIGHT

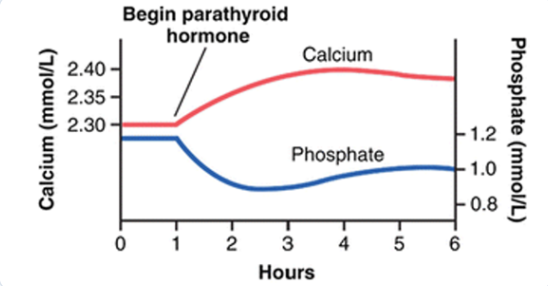
Parathyroid hormone (PTH) primarily acts to increase the concentration of calcium in the blood. It achieves this through several mechanisms:

1. Stimulation of osteoclasts to resorb bone, leading to the release of calcium from the bones.
2. Increased absorption of calcium in the intestines.
3. Stimulation of the kidneys to reabsorb more calcium, reducing its excretion in the urine.

a) Rapid release by osteocytes
PTH mainly works via osteoblast -> osteoclast activation

b) Rapid release by osteoclasts
Does not include kidney contribution

d) Increase calcium reabsorption in kidneys
Kidney alone cannot significantly raise Ca without bone contribution



QUESTION

Which among the following drugs decreases the serum parathyroid hormone levels?

A Risedronate

B **Risedronate** CORRECT

C Lithium

D Diuretics

RIGHT ANSWER

OK **B. Risedronate**

WHY THIS IS RIGHT

Correct answer is B because calcitriol (active vitamin D) increases intestinal calcium absorption and raises serum calcium, which suppresses parathyroid hormone via negative feedback.

Key regulators of PTH:

Factor	Effect on PTH
Low Ca^{2}□	↑ PTH
High Ca^{2}□	↓ PTH
Calcitriol	↓ PTH
Phosphate	↑ PTH

Calcitriol acts on:

Organ	Effect
Intestine	↑ Ca absorption
Kidney	↑ Ca reabsorption
Bone	↑ remodeling

Risedronate reduces bone resorption but does not directly suppress PTH. Lithium can increase PTH and cause hyperparathyroidism. Diuretics have variable effects and are not primary PTH suppressors.

QUESTION

Which of the following is an adverse effect associated with the long-term use of intermittent teriparatide for the treatment of osteoporosis?

A Osteonecrosis of the jaw bone

B **Osteosarcoma** CORRECT

C Subtrochanteric fracture

D Loss of bone mass

RIGHT ANSWER

OK **B. Osteosarcoma**

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1b” here and does not include a written explanation for this item.

QUESTION

A 32-year-old woman presents with recurrent renal calculi, proximal muscle weakness, and low mood. Laboratory evaluation shows elevated serum PTH with normal serum calcium. Primary hyperparathyroidism is suspected. Which of the following is the investigation of choice for preoperative localization of the abnormal gland?

A Neck ultrasound

B MRI neck

C **Tc-99m sestamibi scan** CORRECT

D CT scan neck

OK RIGHT ANSWER

C. Tc-99m sestamibi scan

WHY THIS IS RIGHT

• Recurrent renal stones + proximal weakness + depression -> classic hyperparathyroid symptom cluster
• ↑ PTH with normal Ca -> early / normocalcemic primary hyperparathyroidism

Why Tc-99m Sestamibi is IOC
• Mechanism: Radiotracer (Tc-99m sestamibi) is taken up by mitochondria-rich parathyroid adenoma cells
• Shows persistent uptake in abnormal parathyroid tissue (vs thyroid washout)
• High sensitivity for detecting single adenoma (most common cause)

Step 4: Why not other options?
• A) Neck ultrasound
• Operator dependent
• Misses ectopic glands
• B) MRI neck
• Used only if sestamibi inconclusive
• D) CT neck
• 4D-CT used in difficult/recurrent cases, not first-line

QUESTION

Which of the following is not typically associated with Autoimmune Polyendocrine Syndrome Type 1 (APS-1)?

A Hypoparathyroidism is common

B **Type 1 Diabetes Mellitus association is high** CORRECT

C Association with mucocutaneous candidiasis is high

D Early onset in infancy

OK RIGHT ANSWER

B. Type 1 Diabetes Mellitus association is high

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1c” here and does not include a written explanation for this item.