Beat the Boards

30 USMLE Step 1 Question Walkthroughs

> Never regard study as a duty,
> but as the enviable opportunity to learn.
>
> (Albert Einstein)

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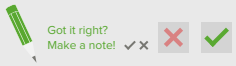
About this eBook

> I don’t love studying. I hate studying.
> I like learning. Learning is beautiful.
> *(Natalie Portman)*

USMLE questions are insanely difficult. To successfully answer them and pass the exam, you will need to engage in multi-step thinking. This book will enable you to go through the questions in a highly focused way by not only providing you with subject-specific preparation but also by helping you to acquire a systematic way of thinking. This way you will keep a cool head even with unknown, difficult questions.

With these questions you will...

- ...practice different types of USMLE questions.
- ...learn important tricks on tackling the stems of USMLE questions.
- ...get an overview of your current state of knowledge in the various USMLE topics.



Stay on top of things and save your time by noting which questions you have already answered.

You don‘t know the answer right away? We provide tips for each question.

Regardless of whether you have not understood something perfectly or you want to deepen your knowledge, we have provided you with additional sources for each question.

You can do this! We believe in you!

About Mohammad Hajighasemi-Ossareh, MD, MBA

After graduating Summa Cum Laude with a Bachelor of Science degree in Biology, Dr. Ossareh obtained his Doctorate of Medicine (M.D.) degree at the University of California, Irvine in 2016. Throughout his years of academic and clinical training, Dr. Ossareh has created and continues to operate the original YouTube Channel for pre-med and medical students with over 86,000 subscribers. Given Dr. Ossareh‘s years of experience in medical education, you will benefit from his practical knowledge base and receive priceless pearls of wisdom.

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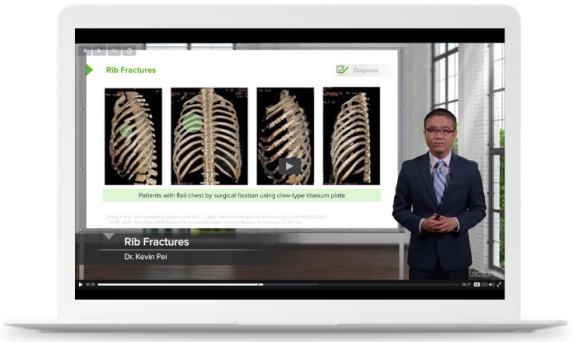

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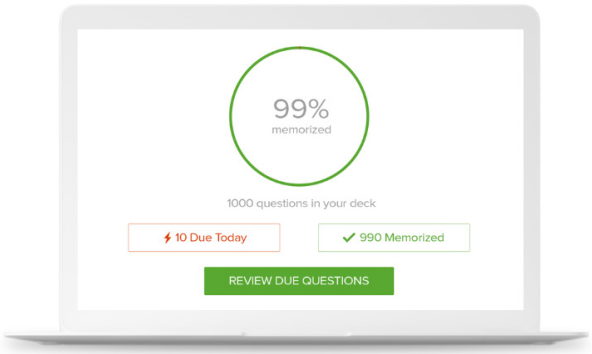

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Content of Questions

GIVE US YOUR FEEDBACK! – Help us improve your learning experience!

Got it right?
Make a note!

Question Review

A previously well 25-year-old woman was brought to the emergency department by her boyfriend because of progressive blurred vision. Examination of the eyes reveals loss of horizontal gaze, intact convergence, and nystagmus. A clinical diagnosis of multiple sclerosis is made and the patient is started on a course of corticosteroids.

What is the most likely etiology for her eye examination findings?

A Loss of reticular formations
B Loss of frontal eye fields
C Loss of bilateral MLF
D Loss of cranial nerves III
E Loss of cranial nerves VI

Need a Hint?

Steps

Step 1: Determine the type of eye disorder the patient is experiencing

Step 2: Determine the anatomical structure affected in this eye disorder

Characteristics

- Neurology question
- 2-step
- Stem is required

WATCH LECTURER VIDEO

Multiple Sclerosis
http://lectur.io/mutiplesclerosis

Walkthrough

Step 1: Determine the type of eye disorder the patient is experiencing

- Abnormal eye findings are **blurred vision, loss of horizontal gaze** and **nystagmus.**
- Blurred vision combined with loss of horizontal gaze suggests **misalignment of the eyes** in certain positions.
- Intact **convergence – adduction of both eyes towards the nose** – confirms both medial rectus muscles are well-innervated and not paralyzed (no CN III palsy).
- ➔ Combination of intact convergence and loss of horizontal gaze suggests the disorder is related to **conjugate movement of the eyes, with internuclear ophthalmoplegia** as the most likely diagnosis.

Step 2: Determine the anatomical structure affected in this eye disorder

- Conjugate eye movement requires simultaneous contraction of oculomotor muscles in both eyes that are innervated by the **oculomotor nerve** (CN III) and the **abducens nerve** (CN VI).
- Transmission of integrated information from upstream gaze centers to these nerves occurs via the white matter tract **medial longitudinal fasciculus** on each side of the brainstem, which is the structure affected in internuclear ophthalmoplegia.
- ➔ As multiple sclerosis (MS) causes generalized demyelination of white matter tracts, internuclear ophthalmoplegia is **usually bilateral in MS patients.**

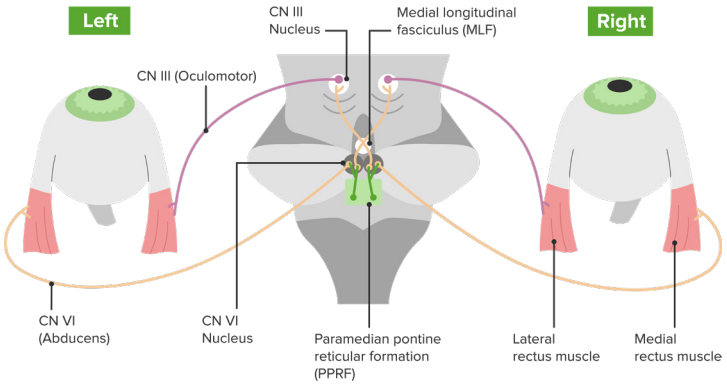


Image: *Innervation of oculomotor muscles with medial longitudinal fasciculus on each side © by Lecturio*

High-yield Facts

Multiple sclerosis:

- Causes demyelination of white matter tracts in the brain and spinal cord.
- Signs and symptoms of MS are wide-ranging, including double vision, muscle weakness, trouble with sensation, trouble with coordination, and mental disorders.
- MS usually presents either as a relapsing form with isolated attacks or as a progressive form.
- Typically diagnosed in young patients with a higher prevalence in women.
- The cause of MS is unknown and there is no cure, though symptomatic and disease-modifying treatment has proven effective.

Internuclear ophthalmoplegia:

- A disorder of horizontal conjugate lateral gaze in which the affected eye or eyes cannot adduct.
- Caused by a lesion to the medial longitudinal fasciculus (MLF) in the brainstem that transmits integrated information from upstream gaze centers to oculomotor nerves.
- In MS patients, the lesion to the MLF tends to be bilateral.

Medial longitudinal fasciculus:

- A white matter tract on each side of the brainstem that coordinates abduction of one eye with adduction of the other eye to produce conjugate horizontal gaze.
- Connects the ipsilateral nucleus of the CN VI (abduction), the contralateral nucleus of CN III (adduction) and the ipsilateral paramedian pontine reticular formation.
- Lesions result in impaired adduction in horizontal lateral gaze with convergence remaining intact.

The correct answer is: Loss of bilateral MLF!

Got it right? Make a note! ✓ ✗

Question Review

A 17-year-old girl suddenly grabs her chest and collapses to the ground while playing volleyball at school. The teacher rushes to evaluate the situation and finds that the girl has no pulse and is not breathing. He starts chest compressions. An automated external defibrillator (AED) is brought to the scene within 3 minutes and a shock is delivered. The girl regains consciousness and a regular sinus rhythm. She is rushed to the Emergency Department. Her blood pressure is 122/77 mm Hg and her pulse is 65/min and regular. An EKG shows a shortened PR interval, a wide QRS complex, a delta wave, and an inverted T wave.

Which of the following is the most likely pathology in the conduction system of this patient's heart?

A. Automatic discharge of irregular impulses in the atria
B. Impulse generation by tissue in atrioventricular node
C. Wandering atrial pacemaker
D. Accessory pathway from atria to ventricles
E. Blockage in conduction pathway

Need a Hint?

Steps

Step 1: Determine the underlying characteristics of the patient's presentation

Step 2: Determine the type of electrical conduction abnormality

Characteristics

- Cardiovascular question
- 2-step
- Stem is required

WATCH LECTURER VIDEO

Tachycardia
http://lectur.io/tachycardia

Walkthrough

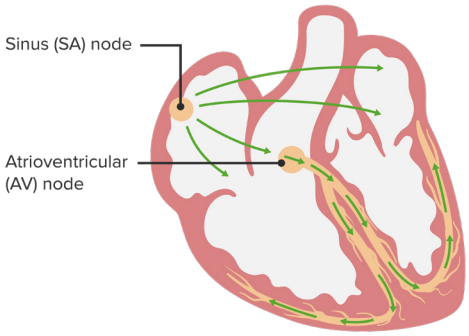
Step 1: Determine the underlying characteristics of the patient's presentation

- **Young** and otherwise **healthy patient**
- Sudden loss of consciousness **with immediate clinical recovery**
- Abnormal EKG beyond recovery: **Short PR interval, wide QRS and delta waves**
- **Acute reversible arrhythmic event** with electrical conduction abnormality

Step 2: Determine the type of electrical conduction abnormality

- **No abnormal P waves** on EKG findings
- Abnormality likely involves communication **between** atria and ventricles.
- Clinical presentation and EKG findings – in particular delta waves – make **Wolff-Parkinson-White syndrome (WPW)** most likely conduction abnormality.
- ➜ In WPW, **accessory pathway** between atria and ventricles causes excessive firing.

Normal Electrical Pathways



Some type of abnormality between the conduction times, in which the atria and ventricles are involved

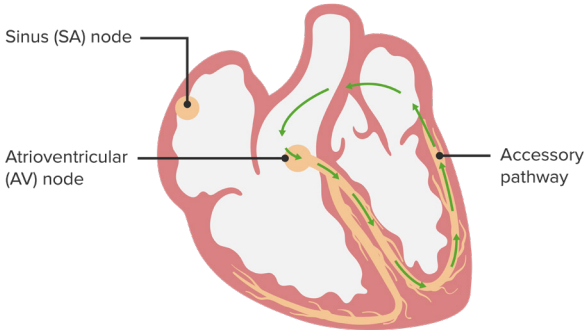


Image: Abnormal electrical pathway in Wolff-Parkinson-White syndrome © by Lecturio

High-yield Facts

Wolff-Parkinson-White syndrome:

- **Electrical conduction abnormality** of the heart
- **Accessory pathway** between the atria and the ventricles **bypassing AV node**
- Conduction through accessory pathway can create **electrical circuit.**
- **Supraventricular tachycardia** and associated symptoms
- **Delta waves** on EKG are pathognomonic. *(See image)*
- Most people with accessory pathway **never become symptomatic.**
- But **WPW is one of most common causes** of tachycardia in young people.

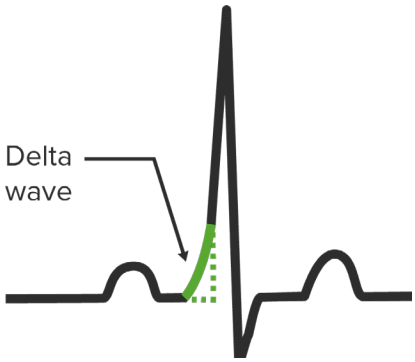


Image: *EKG tracing with delta waves (green highlight) pathognomonic of Wolff-Parkinson-White syndrome © by Lecturio*

The correct answer is: Accessory pathway from atria to ventricles!

Got it right?
Make a note! ✓ ✗

Question Review

A 40-year-old male with a past medical history of major depression presents to the clinic interested in joining a research study on depression-related sleep disturbances. He has had 2 episodes of major depression within the last 2 years, occurring once during the summer and then during the winter of the other year. He has been non-compliant with medication and has a strong desire to treat his condition with non-pharmacological methods. He would like to be enrolled into this study that utilizes polysomnography to record sleep-wave patterns.

Which of the following findings is likely associated with this patient's psychiatric condition?

A. Increased total REM sleep
B. Increased slow wave sleep
C. Late morning awakenings
D. Increased REM sleep latency
E. Associated with a seasonal pattern

Need a Hint?

Steps

Step 1: Determine the relationship between the patient's underlying psychiatric condition and sleep

Step 2: Determine the sleep phase that is most likely associated with MDD sleep disturbances

Characteristics

- Behavioral science – psychiatry
- 2-step
- Stem is required

WATCH LECTURER VIDEO

Sleep-Wake Disorders – Consciousness (PSY)
http://lectur.io/sleepwakedisorders

Walkthrough

Step 1: Determine the relationship between the patient's underlying psychiatric condition and sleep

- Patient has **major depressive disorder (MDD).**
- Typically associated with **excess sleep** (hypersomnia) or **lack of sleep** (insomnia)
- **Medications for MDD** are known for causing sleep disturbances.
- Differential includes **sleep disturbances related to MDD** and to **MMD medication.**
- Non-compliance with MDD medication narrows differential to **sleep disturbances related to MDD.**

Step 2: Determine the sleep phase that is most likely associated with MDD sleep disturbances

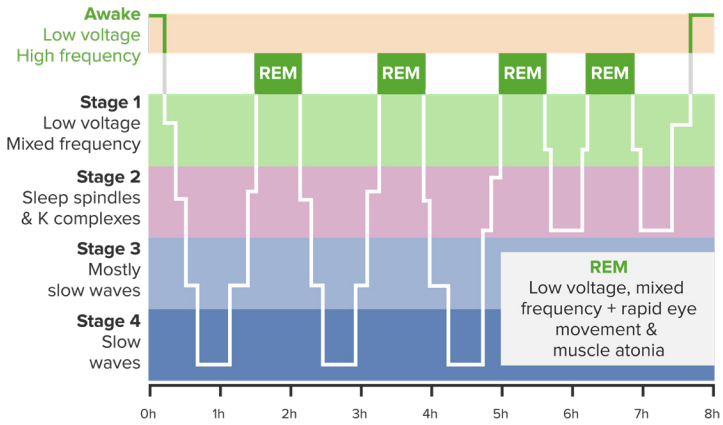


Image: *Somnogram showing normal five stages of sleep (four stages of NREM, and REM) © by Lecturio*

High-yield Facts

Major Depressive Disorder (MDD):

- Psychiatric condition characterized by **recurrent depressive episodes** lasting at last 2 weeks each.
- Patient experiences either **severe depressive mood** or **anhedonia.**
- At least one of the two symptoms **depressive mood** or **anhedonia** must be present for diagnosis – diagnostic criteria laid out in the DSM-IV-TR.
- MDD affects over 200 million people globally.
- **Twice as common** in women than in men.
- MDD-associated sleep disturbances:
  - Very early awakenings with the inability to fall back asleep (insomnia)
  - Excessive sleep (hypersomnia)
- Treatment of MDD is a combination of **psychotherapy and medication.**

Phases of sleep:

- Sleep is divided **into 5 phases:**
  - Non-rapid eye movement (NREM) phases 1–4
    NREM1: light sleep
    NREM2: light sleep
    NREM3: deep sleep
    NREM4: deep sleep
  - **REM:** Brain waves mimic activity during the waking state with eyes closed but moving rapidly from side-to-side
- Phases progress cyclically from NREM1 through REM then begin again with NREM1.
- Each cycle lasts 90–120 mins.

The correct answer is: Increased total REM sleep!

Got it right?
Make a note! ✓ ✗

Question Review

A 16-year-old high school cheerleader is brought by her mother to the Emergency Room after falling on her back during a stunt. She strongly believes that the accident happened because the team couldn't catch her "enormous and bloated" body. Lately, she has been in a lot of stress as dance regionals are soon and wants to lose 6.8 kg (15 lb). She is also experiencing intermittent palpitations and dizziness, even during rest. Physical examination reveals a slender girl with bilateral swelling on her cheeks and abrasions on the dorsum of her right hand. When her mother left the room, she admitted to taking furosemide that she found in the medicine cabinet. Her vitals are the following: temperature is 36.2°C (97.2°F), blood pressure is 90/60 mm Hg, pulse rate is 50/min, respiratory rate is 12/min, height is 162 cm (5 ft 4 in), and weight is 40.9 kg (90 lb). MRI of the thoracic spine shows a vertebral compression fracture. She is refusing to eat anything but ice.

What is the most appropriate next step in management of this patient's symptoms?

A. Admit and start parenteral nutrition
B. Stabilize the fracture with a brace and discharge patient
C. Switch furosemide to hydrochlorothiazide
D. Start bisphosphonates
E. Refer to psychiatrist for outpatient cognitive behavioral therapy

Need a Hint?

Steps

Step 1: Determine the diagnosis based on the signs and symptoms

Step 2: Determine the severity of the disease stage for next step in management

Characteristics

- Behavioral science – psychiatry question
- 2-step
- Stem is required

Walkthrough

Step 1: Determine the diagnosis based on the signs and symptoms

- Patient presents with **combination of psychological and physical symptoms.**
- Psychological disorder is suggested by the patient's **distorted perception of her weight** and **self-medication with diuretics** to lose weight.
- Physical signs and symptoms suggest **severe malnutrition** with differential diagnosis including various GI disorders.

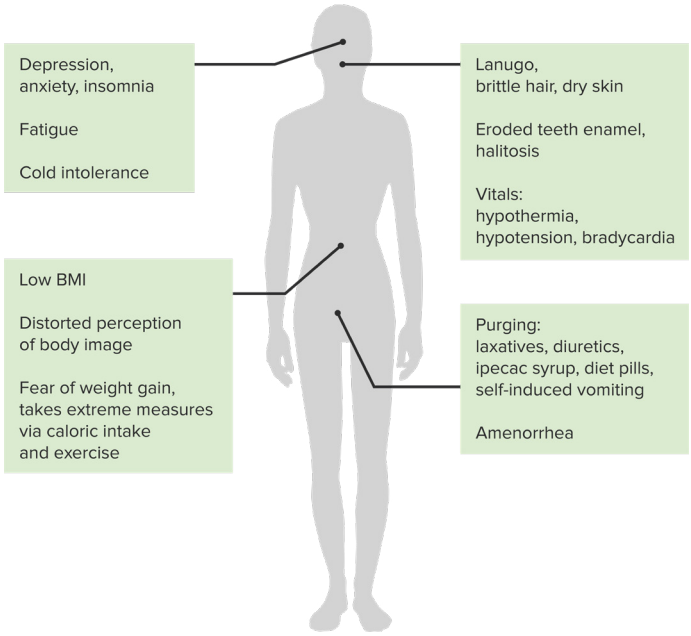
➜ Combination of psychological and physical suggests **anorexia nervosa.**

Step 2: Determine the severity of the disease stage for next step in management

- Patient is in **unstable state** with hypothermia, hypotension, and bradycardia – high risk of severe complications, in particular **heart-related due to electrolyte imbalances.**

➜ Patient needs to be **admitted and receive parenteral nutrition.**

Signs and symptoms of anorexia nervosa



High-yield Facts

Anorexia nervosa:

- **Eating disorder** characterized by excessive desire to be thin or to lose weight
- Patients generally **restrict their food intake,** may **exercise excessively,** use **laxatives,** and self-induce **vomiting** to increase weight loss.
- Complications include serious **heart conditions** and **kidney failure** due to chronic malnutrition.
- **Highest mortality rate** of any psychological disorder with cause of death being **suicide** (most common) or related to medical complications of disease
- Treatment is varied and ranges from **hospitalization** with refeeding, to **medication** and **psychotherapy.**

The correct answer is: Admit and start parenteral nutrition!

Got it right? Make a note! ✓ ✗

Question Review

A 22-year-old woman presents to the gynecologist for evaluation of amenorrhea and dyspareunia. The patient states that she recently got married and has been worried about getting pregnant. The patient states that she has never had a period and that sex has always been painful. On examination, the patient is Tanner stage V with no obvious developmental abnormalities. The vaginal exam is limited secondary to patient discomfort. The vaginal canal is hypoplastic with no visualization of the cervix

What is the most likely cause of this patient's symptoms?

A Hyperprolactinemia
B Exposure to DES in utero
C Turner syndrome
D PCOS
E Mullerian agenesis

Need a Hint?

Steps

Step 1: Determine the characteristics of the patient's clinical presentation

Step 2: Determine the most likely etiology of the clinical presentation based on answers provided

Characteristics

- Embryology question
- 2-step
- Stem is required

Walkthrough

Step 1: Determine the characteristics of the patient's clinical presentation

- Patient shows **normal external sexual development.**
- Patient shows **abnormal internal sexual functioning.**

Step 2: Determine the most likely etiology of the clinical presentation based on answers provided

- **Generalized abnormal sexual differentiation** as seen in chromosomal disorders such as Turner syndrome **unlikely**
- Relation of all signs and symptoms to **one anatomical location of the body**
- **Hormonal disorders** (hyperprolactinemia with excess prolactin and PCOS with excess androgens) **unlikely**
- ➔ Based on the above, most **likely answer is Mullerian agenesis.**

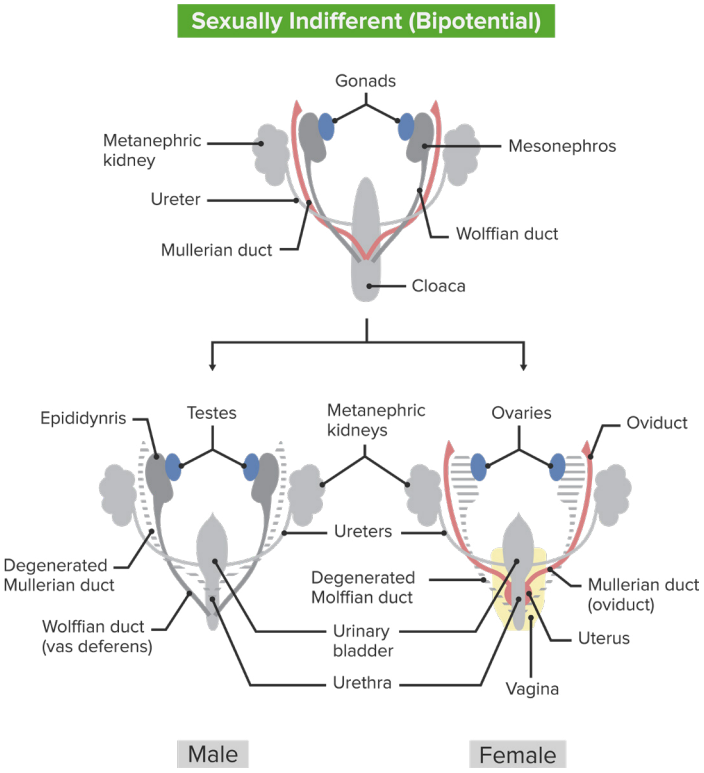


Image: *Development of male and female reproductive organs respectively from bipotential tissue © by Lecturio*

High-yield Facts

Differentiation of gonads into male vs. female:

- Type of gonads that develop (gonadal sex) is determined by the **sex chromosome complex** (XX or XY)
- Internal genitalia consist of two accessory ducts: **mesonephric ducts** and paramesonephric or **mullerian ducts**
- Mesonephric/Wolffian system is the precursor to the **male genitalia** and the paramesonephric / Mullerian to the **female reproductive system**

Mullerian agenesis:

- **Uterine abnormality** caused by genetic defects
- Results in the complete lack of **mullerian ducts,** causing lack of development of the uterus
- **Ovaries and vagina** develop from different embryological structures than uterus
- Hormone levels will be **normal** and patient will reach full pubertal development due to the **presence of the ovaries**
- **Patient will not experience periods** due to dysfunctional uterus

The correct answer is: Mullerian agenesis!

Got it right? Make a note! ✓ ✗

Question Review

A 1-month-old baby is brought to the emergency department because he had a coughing spell while feeding and turned blue. The mother says that the blue color went away when she picked the baby up and brought his knees to his chest. The physician orders a chest X-ray which shows a boot-shaped heart and he tells the mother that the baby has a condition that is caused by an anterosuperior displacement of the infundibular septum.

What are the 4 features of the baby's cardiac condition?

A. Pulmonary stenosis, left ventricular hypertrophy, ventricular septal defect, overriding aorta
B. Pulmonary regurgitation, left ventricular hypertrophy, ventricular septal defect, overriding aorta
C. Pulmonary regurgitation, right ventricular hypertrophy, atrial septal defect, overriding aorta
D. Pulmonary stenosis, right ventricular hypertrophy, atrial septal defect, overriding pulmonary artery
E. Pulmonary stenosis, right ventricular hypertrophy, ventricular septal defect, overriding aorta

Need a Hint?

Steps

Step 1: Determine the baby's diagnosis

Step 2: Determine the defects in Tetralogy of Fallot based on the answer choices

Characteristics

- Embryology – Cardiovascular question
- 2-step
- Stem is required

WATCH LECTURER VIDEO

Tetralogy of Fallot – Blood Vessel and Heart Abnormalities
http://lectur.io/tetralogyoffallo

Walkthrough

Step 1: Determine the baby's diagnosis

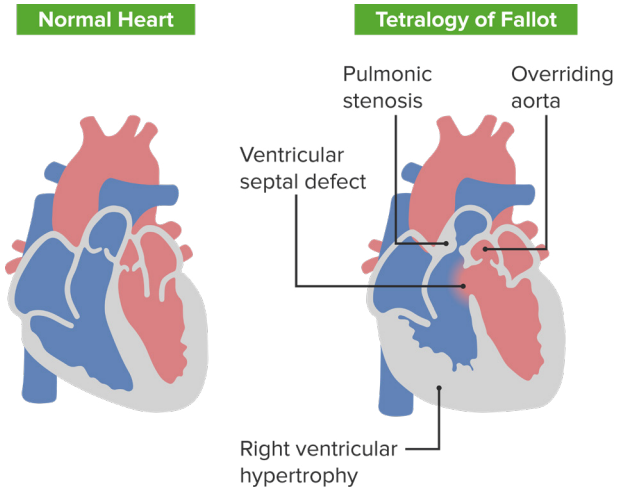
- Baby is suffering from **cyanosis** (turning blue).
- Cyanosis results from tissue **not receiving enough oxygen.**
- Differential diagnosis for **cyanotic heart defects in newborns:**
  - Tetralogy of Fallot (ToF)
  - Transposition of the great arteries
  - Pulmonary atresia

➔ Boot-shaped heart on x-ray and mention of 4 features in vignette, **Tetralogy of Fallot** is the most likely diagnosis.

Step 2: Determine the defects in Tetralogy of Fallot based on the answer choices

- Cyanosis with cardiac etiology like Tetralogy of Fallot results from the **mixing of deoxygenated** (right heart) and **oxygenated blood** (left heart).
- Mixing requires a **septal defect** and occurs down the **pressure gradient,** so in ToF, **right heart must be under higher pressure then left heart.**
- Right heart is normally low pressure, so **1 feature** of Tetralogy of Fallot must **increase right heart pressure** (pulmonary stenosis).

➔ Increased pressure results in hypertrophy (right heart) and **septal defect must be at site of significant pressure difference** (ventricles).



High-yield Facts

Cyanotic congenital heart diseases:

- Cyanotic congenital heart diseases involves **right-to-left shunts.**
- Deoxygenated blood does not pass through the lungs but **mixes directly with oxygenated blood.**
- Diseases generally **involve structural defects** to the heart but may **also** be caused by **resistance-increasing defects to the lungs.**
- Beyond cyanosis, signs and symptoms include **nail clubbing, tachycardia, tachypnea,** and **inadequate feeding.**
- **Delay in reaching developmental milestones** may also be present.

Tetralogy of Fallot:

- Cyanotic congenital heart disease with 4 features **pulmonary stenosis, right ventricular hypertrophy, overriding aorta,** and **ventricular septal defect**
- Results in **right-to-left shunt** between the ventricles
- Affected babies may experience so-called **"tet spells".**
- Diagnosis is typically performed by **echocardiography.**
- X-ray can also be used and may reveal a **boot-shaped heart** (due to right ventricular hypertrophy) in some patients.
- Treatment is **surgical correction of the defects,** generally in the first year of life.

The correct answer is: Pulmonary stenosis, right ventricular hypertrophy, ventricular septal defect, overriding aorta!

Got it right? Make a note! ✓ ✕

Question Review

Which of the following statements concerning Positive Predictive Value (PPV) is correct?

A. Positive predictive value is the proportion of tests that are true positives; if disease prevalence is low, then PPV will be low

B. Positive predictive value is the proportion of tests that are true positives; if disease prevalence is high, then PPV will be low

C. Positive predictive value is the proportion of tests that are true positives; disease prevalence has no effect on PPV

D. Positive predictive value is the proportion of tests that are false positives; if disease prevalence is low, then PPV will be low

E. Positive predictive value is the proportion of tests that are false positives; if disease prevalence is high, then PPV will be low

Need a Hint?

Steps

Step 1: Determine the formula for positive predictive value (PPV)

Step 2: Determine the influence of prevalence on PPV

Characteristics

- Embryology – Cardiovascular question
- 2-step
- Stem is required

WATCH LECTURER VIDEO

False Positives and False Negatives – Screening Tests
http://lectur.io/screeningtests

Walkthrough

Step 1: Determine the formula for positive predictive value (PPV)

- PPV – proportion of **truly positive tests** of a diagnostic test
- Formula for PPV – **number of true positives/the number of all positive tests (true positives + false positives)**

$$\frac{\text{Number of true positives}}{\text{True positives + false positives}} \leftarrow \text{Number of all positive tests}$$

Step 2: Determine the influence of prevalence on PPV

- Prevalence – **proportion of a population found to be affected by a condition**
- **Changing PPV with prevalence** (seen in image)
- **High prevalence,** increasing number of true positives and **high PPV**
- **Low prevalence,** decreasing number of true positives and **low PPV**

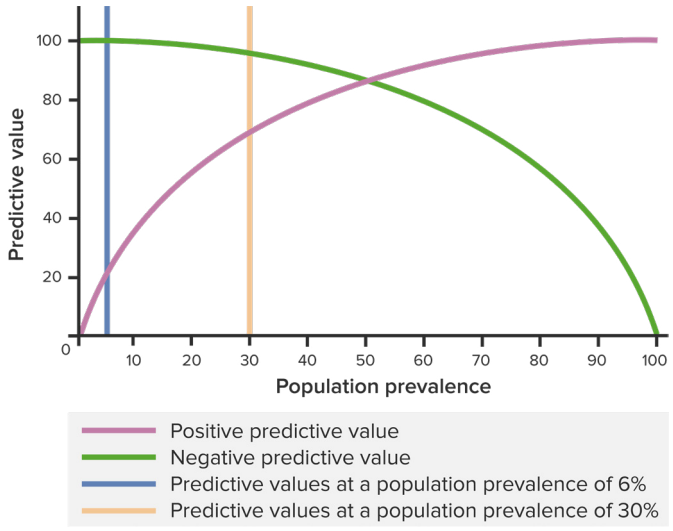


Image: *Impact of disease prevalence on a test's positive predictive value © by Lecturio*

High-yield Facts

PPV and negative predictive value (NPV):

- PPV – **Proportion of true positives** of a diagnostic test
- Reflects **how likely a positive result is a true finding**
- PPV calculated as **true positives / all positive tests** (true positives + false positives)
- Negative predictive value (NPV): proportion of truly negative tests and reflection of **likeliness of a negative result to be a true finding**

Prevalence:

- Prevalence is the **proportion of a population found to be affected by a condition** at a specific time point or during a given time period.
- Prevalence – calculated by the **number of people with condition/the total number of people in given population**
- No **confusion with incidence** – number of NEW cases per population at risk in a given time period
- Predictive value (PPV or NPV) – **influenced by prevalence**
- **PPV increase due to prevalence increase**
- **NPV decrease due to prevalence increase**

The correct answer is:
Positive predictive value is the proportion of tests that are true positives; if disease prevalence is low, then PPV will be low!

Got it right? Make a note! ✓ ✗

Question Review

A 15-year-old girl presents to the emergency department via EMS after a motor vehicle accident. The patient is in critical condition and is hemodynamically unstable. It becomes apparent that the patient may require a blood transfusion, and the parents are approached for consent. They are Jehovah's Witness and deny the blood transfusion, saying it is against their beliefs. However, the patient insists that she wants the transfusion if it will save her life. Despite the patient's wishes, the parents remain steadfast in their refusal to allow the transfusion.

Which of the following is the most appropriate course of action?

A. Give the patient the blood transfusion
B. Do not give blood transfusion due to parents' refusal
C. Give intravenous fluids to attempt to stabilize the patient
D. Consult the hospital ethics committee
E. Obtain a court order to give blood products

Need a Hint?

Steps

Step 1: Determine whether a blood transfusion is life-saving for this patient

Step 2: Determine the rights of the 15-year-old girl to decide her treatment

Characteristics

- Ethics question
- 2-step
- Stem is required

Walkthrough

Step 1: Determine whether a blood transfusion is life-saving for this patient

- Patient has lost significant amount of blood.
- Intravenous fluids may be used initially in hemodynamically unstable patients but **blood products will be required** following significant blood loss.

➜ Blood transfusion is a **life-saving treatment** in this patient.

Step 2: Determine the rights of the 15-year-old girl to decide her treatment

- **Interest of the child outweighs the religious rights** of the parents.
- As the blood transfusion is a **life-saving treatment** in this situation, it does not require the consent of the parents.

➜ In the case of a **life-threatening condition, minors have the right to overrule** the decisions of their parents with respect to their medical treatment.

High-yield Facts

Blood transfusion:

- In the case of significant blood loss, **replacement through blood transfusion** is indicated.
- If the patient is hemodynamically unstable, a blood transfusion may be considered **as life-saving treatment.**
- Initially, patients **may be treated with intravenous fluids** short-term whilst blood products are obtained from the transfusion service.

Consent:

- **Jehovah's witnesses do not accept blood transfusions** based on religious grounds.
- Although parental consent is generally required for medical treatment, in the case of life-threatening situations, **the health of the minor takes priority** over any beliefs of the parents.
- Minors have capacity to make **medical decisions for themselves in life-threatening situations.**

The correct answer is: Give the patient the blood transfusion!

24-year-old (Male) with Ectopic Gland Tissue

WALK THROUGH VIDEO

Got it right? Make a note! ✓ ✗

Question Review

A 24-year-old man undergoes surgery due to a painless swelling at the base of the neck. During the operation, a superficial, 1.0 cm x 0.8 cm mass surrounded by a well-defined fibrous capsule is excised and sent for histological evaluation which reveals this tissue to be ectopic gland tissue. The image below is from the excised mass.

Which of the following glands does this tissue most likely belong to?

A Thyroid gland
B Parotid gland
C Sublingual gland
D Adrenal gland
E Submandibular gland

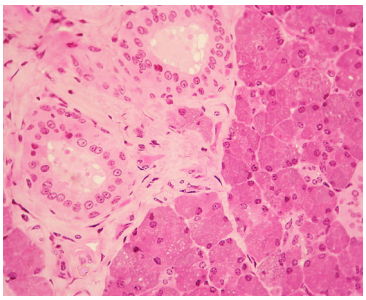


Image: *By Wbensmith – Increased contrast; cropped; CC BY 3.0, https://commons.wikimedia.org/w/index.php?curid=2845814*

Need a Hint?

Steps

Step 1: Determine the type of tissue

Step 2: Determine from which gland the tissue originates

Characteristics

- Histology question
- 2-step
- Stem is required

WATCH LECTURER VIDEO

Parotid Gland
http://lectur.io/parotidgland

Walkthrough

Step 1: Determine the type of tissue

- As seen in image beside, histology slide shows **secretory cells** arranged in acini, and ducts.
- **Endocrine glands do not have ducts,** as they secrete directly into bloodstream.
- ➜ Tissue is from **exocrine gland,** not endocrine gland.

Step 2: Determine from which gland the tissue originates

- As seen in image below, acini show **numerous basophilic granules,** typical of serous secretory cells.
- **Mucous cells** are absent.
- Looking at answer choices, only exocrine gland with solely serous cells is the **parotid.**
- **Sublingual gland consists of a mixture** of serous and mucous cells.
- Both **thyroid and adrenal glands are endocrine** and show very different histology (thyroid shows follicles with a single layer of the cuboid or low columnar epithelium, adrenal glands show cortex with 3 zones – glomerulosa, fasciculata, and reticularis and medulla).
- ➜ In conclusion, histology slide shows **ectopic tissue from the parotid gland.**

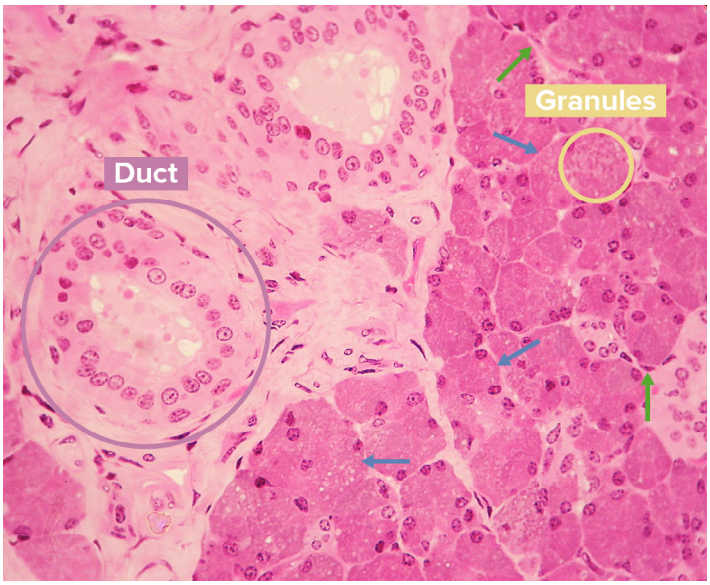


Image: *By Wbensmith – Increased contrast; cropped; Labels and Circles, CC BY 3.0, https://commons.wikimedia.org/w/index.php?curid=2845814*

High-yield Facts

Histology of the parotid gland:

- Parotid glands are the **largest salivary glands** in humans.
- They secrete through the **parotid duct** into the mouth.
- On histological exam, parotid glands show a **capsule made of dense connective tissue, striated ducts** lined with simple columnar epithelium, and intercalated ducts lined with cuboidal epithelium.
- Parotid glands also shows **abundance of fat** *(not seen in the image)* that increases with age.
- Parotid glands **only contain serous acini** while the other salivary glands (sublingual, submandibular) contain both serous and mucinous acini.

Histology of the sublingual glands:

- Sublingual glands are the **smallest major salivary glands** in humans.
- They are the only salivary glands that are **not encapsulated.**
- On histological exam, sublingual glands **stain much less than** parotid glands due to the relatively **small uptake of stain by their abundant mucinous acini.**

The correct answer is: Parotid gland!

Got it right? Make a note! ✓ ✗

Question Review

A patient is infected with a pathogen and produces many antibodies to many antigens associated with that pathogen via Th-cell activated B-cells. This takes place in the germinal center of lymphoid tissues. If the same patient is later re-infected with the same pathogen, the immune system will respond with a much stronger response, producing antibodies with greater specificity that pathogen in a shorter amount of time.

What is the term for this process that allows the B-cells to produce antibodies specific to that antigen?

A. Affinity maturation
B. Avidity
C. T-cell positive selection
D. Somatic hypermutation
E. T-cell negative selection

Need a Hint?

Steps

Step 1: Determine what immunological step is being described by “process“

Step 2: Determine what this process is called

Characteristics

- Immunology question
- 2-step
- Stem is required

WATCH LECTURER VIDEO

Germinal Center – Lymphocyte Activation
http://lectur.io/germinalcenter

Walkthrough

Step 1: Determine what immunological step is being described by "process"

- Immunological response in **case of re-infection**
- Production of antibodies by B-cells – **more specific at re-infection** than at first exposure
- Increase of **antibody specificity** after first antigen exposure

Step 2: Determine what this process is called

- Increase of antibody specificity at various stages during B-cell maturation
- After (mono)clonal expansion (image), undergoing somatic hypermutation by B-cells leading to trigger of gene recombination and mutations to increase affinity for given antigen
- Creating **multiple clones of B-cells** by somatic hypermutation
- Next step: **selection of highest-affinity B-cells** (vs. apoptosis of low-affinity ones)
- Somatic hypermutation and selection combined – **affinity maturation**
- Progress of selected B-cells to become **plasma cells for antibody production** or **memory B-cells** to further accelerate immune reaction at re-infection

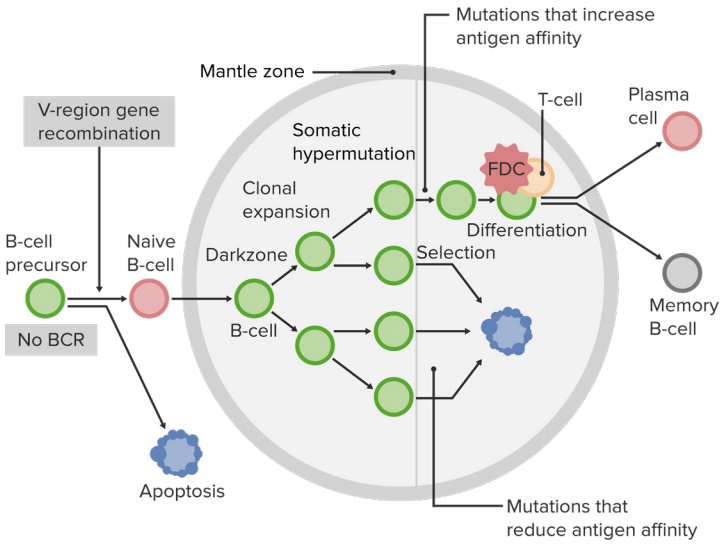


Image: *Maturation of B-cells in a germinal center © by Lecturio*

High-yield Facts

Affinity maturation:

- Production of antibodies with **highest single-site binding strength to certain antigens**
- Occurs **in germinal center** of lymph nodes
- Comprises **somatic hypermutation** and **selection of highest-affinity antibodies**

Somatic hypermutation:

- Genetic recombination and mutations that create a **wide variation of antibodies** when the organism first encounters the foreign antigen
- Provides the range of **B-cells/antibodies for selection**
- **Part of overall process** of affinity maturation

Avidity:

- Referring to **overall binding strength across multiple affinities** (binding sites)
- Generally used when describing **antibody-antigen interactions** across multiple binding sites (e.g. IgM with low affinity, but high avidity due to its 10 low-affinity binding sites)

The correct answer is: Affinity maturation!

WALK THROUGH VIDEO

Got it right? Make a note! ✓ ✗

Question Review

A 3-year-old toddler was rushed to the Emergency Department after consuming peanut butter crackers at daycare. The daycare staff report that the patient has a severe allergy to peanut butter and he was offered the crackers by mistake. The patient is in acute distress. His blood pressure is 60/40 mm Hg and heart rate is 110/min. There is audible inspiratory stridor and the respiratory rate is 27/min. Upon examination his chest is covered in a maculopapular rash. Intubation is attempted and failed due to extensive laryngeal edema. The decision for cricothyrotomy is made.

Which of the following is the most likely mechanism of this pathology?

A. C5a production
B. Release of IL-4
C. Deposition of antigen-antibody complexes
D. IL-2 secretion
E. C3b interaction

Need a Hint?

Steps

Step 1: Determine the child's diagnosis

Step 2: Determine the immunological mechanism of the reaction

Characteristics

- Immunology question
- 2-step
- Stem is required

WATCH LECTURER VIDEO

Hypersensitivity: Types
http://lectur.io/hypersensitivitytypes

Walkthrough

Step 1: Determine the Child's Diagnosis

- History is in keeping with a **severe allergy to peanuts**
- Rapid onset of hypotension, respiratory distress, laryngeal edema, and a rash is highly suggestive of **anaphylaxis**

➜ Patient has a **type I hypersensitivity** reaction to peanuts

Step 2: Determine the Immunological Mechanism of the Reaction

- Type I hypersensitivity reaction arises due the **recognition of peanut allergens as foreign antigens**
- As seen in image, Immunoglobulin E (IgE) binds to the peanut antigen(s) and activates FcεRI receptors on mast cells and basophils, which degranulate and release **vasoactive inflammatory mediators such as histamine**
- **Interleukin-4** plays a critical role in allergic disorders including food hypersensitivity and its release is **involved in antibody production**

Coombs and Gell´s Classification

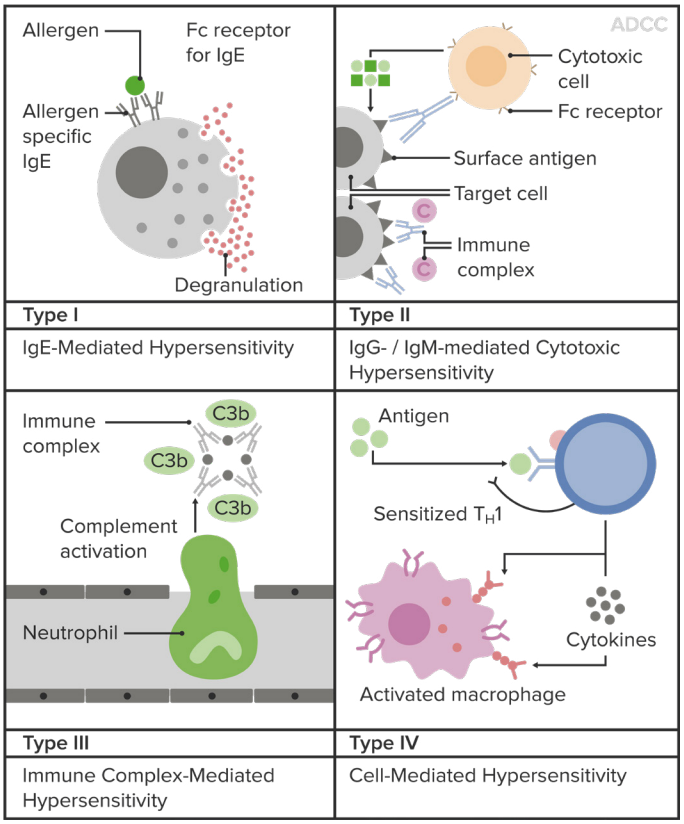


Image: *The 4 types of hypersensitivity reactions © by Lecturio*

High-yield Facts

Types of Hypersensitivity Reactions:

- **There are 4 types of hypersensitivity reactions:**
  - Type I (anaphylaxis): Acute onset triggered by **surface IgE-mediated** widespread histamine degranulation
  - Type II: Cytotoxic hypersensitivity generally mediated by IgG and/or IgM that react to **surface antigens and active the complement system** – typical example is ABO blood incompatibility
  - Type III: Deposition of **circulating immune complexes in tissue** – typical examples is rheumatoid arthritis
  - Type IV: Delayed reaction that can take up to several days, the **only hypersensitivity type** that is not mediated by antibodies but by T-cells

Mechanism of Type 1 Hypersensitivity:

- First exposure to antigen does not provoke reaction but **sensitization**
- During sensitization, IgE antibodies specific to antigen are produced and bind to **IgE-receptors on mast cells**
- Upon second exposure, antigen binds to IgE antibodies
- This causes massive degranulation of mast cells and **release of histamine**
- Interleukin-4 (IL-4) plays a key role in **initial antibody production**, including the differentiation of B cells into plasma cells to produce antibodies

The correct answer is: Release of IL-4!

Got it right?
Make a note! ✓ ✗

Question Review

A 2-year-old boy is brought to the pediatrician's office by his mother. She tells the doctor that her son's appetite has grown dramatically over the past year. He never seems to get full and eats non-stop, all day. He has gained over 4.5 kg (10 lb) in the last 3 months and he is now above the 95th percentile for weight. He attends a special education program at school due to mild mental disability. As an infant, the boy had feeding difficulties, weak muscle tone, and developmental delay. Today, his physical exam is notable for a short, obese child with a narrow forehead and almond-shaped eyes.

Which of the following genetic abnormalities is the most likely to have caused this condition?

A Paternal disomy
B Maternal chromosomal deletion
C Paternal chromosomal deletion
D Autosomal trisomy
E Sex chromosomes aneuploidy

Need a Hint?

Steps

Step 1: Determine the child's diagnosis

Step 2: Determine the genetic mechanism behind this disorder

Characteristics

- Medical genetics question
- 2-step
- Stem is required

WATCH LECTURER VIDEO

Prader-Willi Syndrome
http://lectur.io/praderwillisyndrome

Walkthrough

Step 1: Determine the child's diagnosis

- Clinical presentation shows **hypotonia**, generalized **developmental delay,** and **hyperphagia** increasing with age
- Boy also shows dysmorphic facial features, notably a **narrow forehead** and almond-shaped eyes

➜ These signs are typical of **Prader-Willi-Syndrome** (PWS)

Step 2: Determine the genetic mechanism behind this disorder

- PWS results from a dysfunction of **"genomic imprinting"** *(see image).*
- In genomic imprinting, certain alleles **physiologically silenced in parent-specific manner**
- Affected regions in PWS are **SNRPN and NDN of 15q13** *(see image).*

➜ Maternal alleles silenced in these regions and in PWS, **paternal alleles cannot be expressed** (e.g. due to deletion).

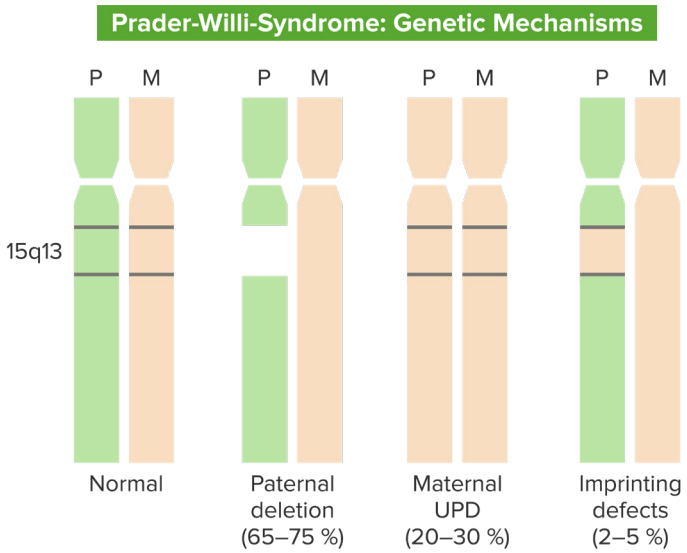


Image: *Genetic mechanisms in Prader-Willi-Syndrome © by Lecturio*

High-yield Facts

Genomic imprinting:

- Physiological process that **affects less than 1 % of all genes**
- **Silencing of certain alleles in parent-specific manner,** leaving only other parent's alleles to be expressed
- Main disorders related to genomic imprinting:
  - **PWS** (imprinting with paternal deletion)
  - **Angelman syndrome** (imprinting with maternal deletion)

Prader-Willi-Syndrome (PWS):

- Rare genetic disorder
- Typically presents during infancy with **hypotonia, feeding difficulties, and developmental delay**
- Physical abnormalities include **narrow forehead, almond-shaped eyes, hypogonadism and small hands and feet.**
- Affected regions in PWS are **SNRPN** and NDN of **15q13.**
- Cause is genomic **imprinting of maternal alleles and dysfunction of paternal alleles** (e.g. deletion).

The correct answer is: Paternal chromosomal deletion!

Got it right? Make a note! ✓ ✗

Question Review

A 24-year-old sexually active man complains of painless warts on his penis. He is worried that he might have transmitted them to his girlfriend. Biopsy shows cells with perinuclear cytoplasmic vacuolization and nuclear enlargement. The doctor treats the patient by chemically ablating the warts with a 1% acetic acid solution. The patient encourages his girlfriend to get tested too, as he is worried that she will be at increased risk of developing a malignancy if she has contracted the condition.

Which cancer is the patient worried about?

A. Kaposi sarcoma
B. Burkitt lymphoma
C. Hairy cell leukemia
D. Hepatocellular carcinoma
E. Cervical carcinoma

Need a Hint?

Steps

Step 1: Determine the diagnosis and cause of the warts

Step 2: Determine which cancers are associated with HPV infection

Characteristics

- Microbiology question
- 2-step
- Stem is required

Walkthrough

Step 1: Determine the diagnosis and cause of the warts

- Patient has **genital warts** (condylomata accuminata).
- Genital warts are caused by **human papillomavirus (HPV)** *(see image).*
- HPV is the most **common sexually transmitted infection.**

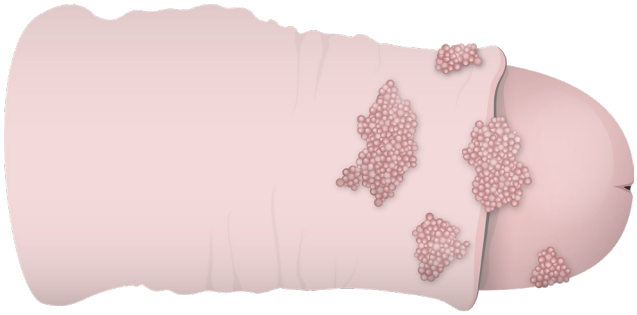


Image: *Genital warts (condylomata accuminata) © by Lecturio*

Step 2: Determine which cancers are associated with HPV infection

- HPV infection **increases the risk** of **cervical cancer** and cancers of the **vulva, vagina, penis, or anus.** *(see image)*
- Kaposi sarcoma is associated with **human herpes virus 8** (HHV8) and HIV, also known as Kaposi sarcoma-associated herpes virus (KSHV).
- Burkitt lymphoma is associated with **Epstein-Barr virus** (EBV).
- Hairy cell leukemia is associated with **human T-lymphotropic virus type 1** (HTLV-1).
- Hepatocellular carcinoma is associated with **hepatitis B and C viruses.**

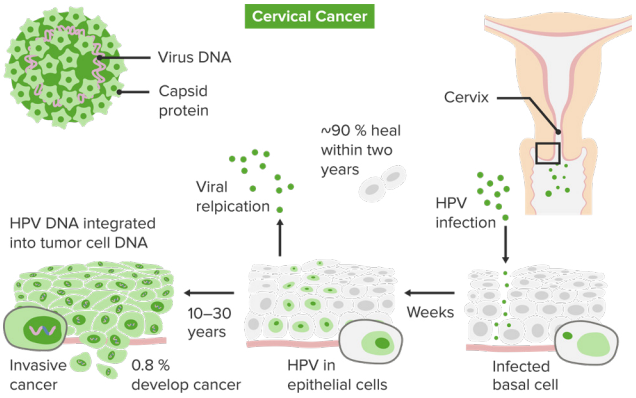


Image: *Progression of HPV infection to cervical cancer © by Lecturio*

High-yield Facts

Condylomata accuminata (genital warts):

- Usually appear as a **small bump or group of bumps** in the genital area and may be associated with **pain, discomfort** and **itching.**
- Lesions can be small, large, flat, raised or shaped like a cauliflower.
- Genital warts can affect both men and women.
- Almost all cases of genital warts are **caused by HPV** (HPV types 6 and 11).

HPV:

- HPV is the most **common sexually transmitted infection**, with 79 million Americans, most in their late teens and early 20s, infected with HPV.
- HPV is transmitted through intimate **skin-to-skin contact**, generally through vaginal or anal sex and can be transmitted by a person even if they are asymptomatic.
- HPV infects the basal layer of the skin and mucous membranes, with viral replication occurring following a latent period, leading to hyperkeratosis and wart formation.
- There are more than **70 types of HPV**, with **HPV serotypes 6 and 11** related to approximately 90 % of condylomata accuminata.
- These two subtypes have relatively **low neoplastic potential compared with other pre-neoplastic types such as HPV serotypes 16 and 18.**

The correct answer is: Cervical carcinoma!

Got it right? Make a note! ✓ ✗

Question Review

A 22-year-old student presents to the college health clinic with a 1-week history of fever, sore throat, nausea, and fatigue. He could hardly get out of bed this morning. There are no pets at home. He admits to having recent unprotected sex. His vital signs include a temperature of 101.0°F (38.3°C), a pulse of 72/min, a blood pressure of 118/63 mm Hg, and a respiratory rate of 15/min. On physical examination, he has bilateral posterior cervical lymphadenopathy, exudates over the palatine tonsil walls with soft palate petechiae, an erythematous macular rash on the trunk and arms, and mild hepatosplenomegaly.

What is the most likely diagnosis?

A. Rubella
B. Acute HIV infection
C. Toxoplasma infection
D. Infectious mononucleosis
E. Streptococcal pharyngitis

Need a Hint?

Steps

Step 1: Determine which signs and symptoms are generic to all infections vs. specific

Step 2: Determine which infectious agent causes the signs and symptoms seen in this patient

Characteristics

- Microbiology question
- 2-step
- Stem is required

Walkthrough

Step 1: Determine which signs and symptoms are generic to all infections vs. specific

- Patient has generic signs and symptoms of infection/ inflammation such as **fever, sore throat, fatigue**
- Bilateral **cervical lymphadenopathy, palatine tonsil exudates, soft palate petechiae, erythematous rash** on trunk and arms, and **hepatosplenomegaly** are to specific to certain infectious agents *(see image)*

Step 2: Determine which infectious agent causes signs & symptoms seen in this patient

- History of unprotected sex increases risk of HIV infection, but this generally does **not present with splenomegaly**, instead, skin lesions and diarrhea are more common

➜ **Given this patient's age and presentation, infection** with Epstein-Barr virus seems most likely diagnosis

➜ Epstein-Barr virus causes **infectious mononucleosis** or "kissing disease"

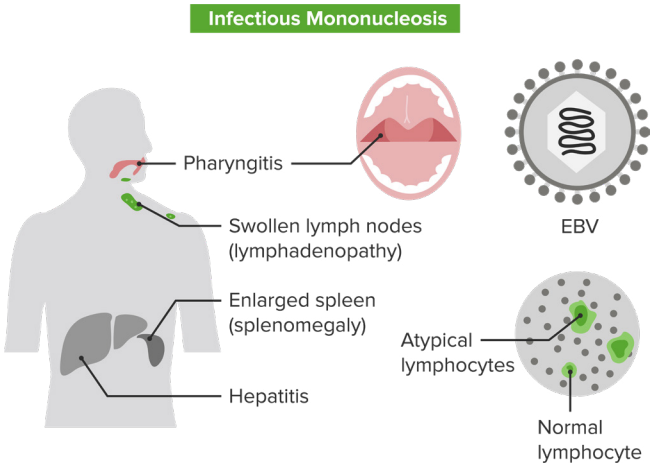


Image: *Clinical presentation of infectious mononucleosis © by Lecturio*

High-yield Facts

Epstein-Barr virus:

- Type of human herpes virus found **in saliva and genital secretions**
- Causes **infectious mononucleosis** (glandular fever)
- Also associated with **certain cancers, e.g. Burkitt's lymphoma**

Infectious mononucleosis:

- Also called **glandular fever**
- Caused by infection with **Epstein-Barr virus**
- More commonly **affects teenagers and young adults**
- Often remains asymptomatic
- Typical signs and symptoms – **fever, sore throat, erythematous spots in mouth, cervical lymphadenopathy, hepatosplenomegaly** (incl. jaundice more rarely)

The correct answer is: Infectious mononucleosis!

Got it right?
Make a note! ✓ ✗

Question Review

A 49-year-old woman is brought in to the Emergency Department by ambulance. She developed crushing chest pain and palpitations. Past medical history is significant for hypertension, hyperlipidemia, and obesity. She takes chlorthalidone, lisinopril, atorvastatin, metformin, and an oral contraceptive every day. She works as a lawyer and her job is stressful. She drinks wine with dinner every night and smokes 10 cigarettes a day. Emergency personnel stabilized her and administered oxygen while on the way to the hospital. Upon arrival, Today, his blood pressure is 120/80 mm Hg, heart rate is 120/min, respiratory rate is 22/min, and temperature is 37.7°C (99.9°F). On physical exam, she is an obese woman in acute distress. She is diaphoretic and has difficulty catching her breath. A bedside KG is performed which reveals ST elevation in leads II, III, and aVF.

Which of the following is the most probable diagnosis?

A. Inferior wall myocardial infarction
B. Lateral wall myocardial infarction
C. Posterior wall myocardial infarction
D. Anteroseptal myocardial infarction
E. Right ventricular myocardial infarction

Need a Hint?

Steps

Step 1: Determine whether acute myocardial infarction is a likely diagnosis

Step 2: Determine which part of the heart has sustained an ischemic injury

Characteristics

- Pathology – cardiovascular question
- 2-step
- Stem is required

WATCH LECTURER VIDEO

STEMI and NSTEMI: Special Inferior Wall Myocardial Infarction
http://lectur.io/stemiandnstemi

Walkthrough

Step 1: Determine whether Acute Myocardial Infarction is a Likely Diagnosis

- Patient has **risk factors** for ischemic heart disease **(e.g., obesity, diabetes).**
- **History is typical** for an acute myocardial infarction.
- **ST elevation and T wave inversion** are typical EKG findings in acute myocardial infarction.

Step 2: Determine which Part of the Heart has Sustained an Ischemic Injury

- EKG leads II, III and aVF 'view' the **inferior surface of the heart**
- ST elevation and T wave inversion in leads **II, III and aVF is a typical EKG finding** in infarction of the inferior wall of the heart

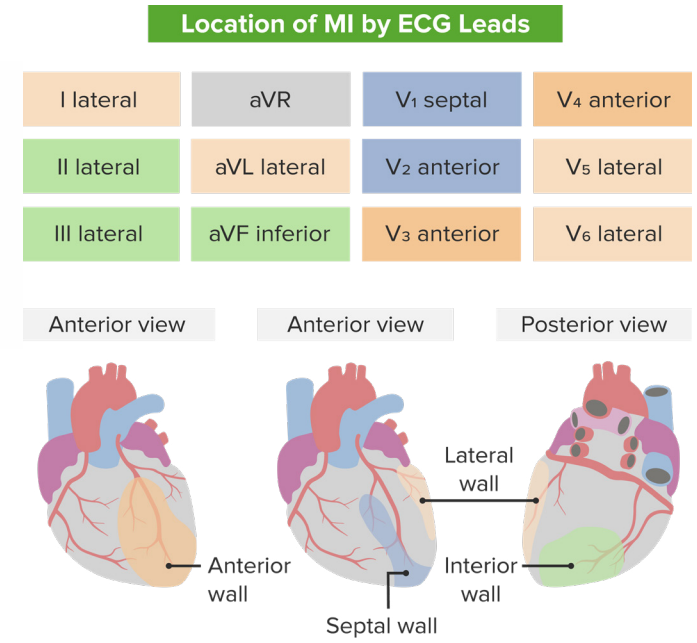


Image: *Location of myocardial infarction through EKG lead anomalies © by Lecturio*

High-yield Facts

Acute myocardial infarction:

- Typical symptoms include **severe crushing central chest pain** with radiation down the **left arm** or into the **jaw.**
- Pain is often accompanied by **sweating, nausea,** and **shortness of breath.**
- Risk factors include **smoking, obesity, hyperlipidemia, hypertension,** and **diabetes.**
- Generally associated with **typical EKG changes of ST elevation and T wave inversion (STEMI)** accompanied by elevation of cardiac enzymes in the blood

Localization of an acute myocardial:

- EKG leads in which **ST elevation and/or T wave inversion** are observed, allow localization of an acute myocardial infarction.
- **V1 to V2 are septals, V3-V4 'view' the anterior** surface,**V5, V6, I and aVL, 'view' the lateral** surface, and **II, III and aVF 'view' the inferior** surface of the heart.
- ST elevation and T wave inversion in leads **II, III and aVF are typical of an acute inferior myocardial infarction.**

The correct answer is: Inferior wall myocardial infarction (MI)!

Got it right? Make a note! ✓ ✗

Question Review

A 43-year-old man presents to his primary care provider. He is concerned about general weakness with decreased concentration over the past several months. Additionally, he has been constipated and has lost about 9.1 kg (20 lbs) unintentionally. Past medical symptoms are noncontributory. He works as a bank manager and occasionally drinks alcohol but does not smoke tobacco. Today, his blood pressure is 145/90 mm Hg, heart rate is 60/min, respiratory rate is 19/min, temperature 36.6°C (97.9°F). On the physical exam, the patient looks confused and fatigued and there is pain on palpation of the arms and legs.

A lab exam is performed which finds:

Calcium	14.5 mg/dL
Phosphate	2.2 mg/dL
PTH	18 pg/mL

PTHrP	4 pmol/L
Calcitriol	46 pg/mL
T3	120 ng/mL
T4	10.2 mcg/dL

Normal value: < 2 pmol/L
Normal value: 25–65 pg/mL

Reference values can be found at the end of the book

Taking into account the clinical and laboratory findings, what is the most likely cause of his hypercalcemia?

A Chronic kidney disease
B Hyperparathyroidism
C Hypervitaminosis D
D Malignancy
E Thyrotoxicosis

Need a Hint?

Steps

Step 1: Identify additional key lab findings beyond hypercalcemia

Step 2: Determine most likely cause of clinical and lab findings

Characteristics

- Pathology – endocrine question
- 2-step
- Stem is required

WATCH LECTURER VIDEO

Diagnosis of Hypercalcemia and Knowledge Check
http://lectur.io/diagnosisofhypercalcemia

Walkthrough

Step 1: Identify additional key lab findings beyond hypercalcemia

- Notable in patient's lab findings – **low phosphate** (reference range 3.4–4.5 mg/dL) and **elevated PTHrP** (PTH-related protein) (normal < 2.5 pmol/L)
- Other lab findings – **normal**

Step 2: Determine most likely cause of clinical and lab findings

- General focus of differential diagnosis of hypercalcemia: **primary hyperparathyroidism/hypercalcemia of malignancy**
- **Low to normal PTH levels** with hypercalcemia suggests malignancy
- **PTHrP** (PTH-related protein) – secreted by various malignant tumors and is an important **biomarker** for hypercalcemia of malignancy – is elevated in this patient.

➜ Malignancy is consistent with patient's clinical presentation of **weight loss** and **pain upon palpation** (bone pain caused by PTHrP-induced bone resorption).

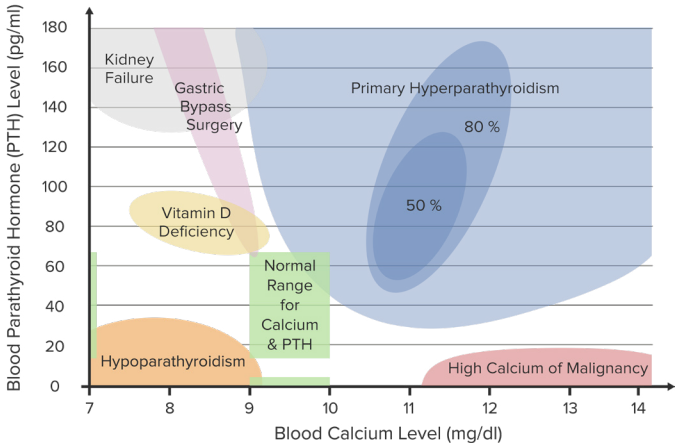


Image: *Relationship between calcium and parathyroid blood levels in different pathologies: High calcium and low to normal parathyroid hormone indicates malignancy © by Lecturio*

High-yield Facts

Hypercalcemia of malignancy:

- Relatively **common in cancer patients:** affecting up to 40 % of them
- **PTHrP-induced osteolysis accounting for majority:** Mechanisms are varied.
- Most common cancers associated with hypercalcemia – **breast, lung** and **multiple myeloma**

PTH-rP (parathyroid hormone-related protein):

- Main **biomarker** of hypercalcemia of malignancy
- Also has **physiological function** of regulating bone development
- High levels induce **abnormal bone resorption,** leading to hypercalcemia.

The correct answer is: Malignancy!

WALK THROUGH VIDEO

Got it right? Make a note! ✓ ✗

Question Review

A 33-year-old female presents to the clinic for evaluation of yellowish discoloration of her skin and eyes. She has had this problem for 6 months but states that it's getting worse over the past few weeks. She also complains of repeated bouts of bloody diarrhea and abdominal pain that have been very embarrassing for her. On examination, her blood pressure is 110/60 mm Hg, pulse is 90/min, respirations are 19/min, and temperature is 36.6°C (97.8°F). Her abdomen is lax with no tenderness or rebound tenderness. Rectal examination shows blood and mucus in the rectal vault. Her sclera looks yellow along with the face and upper body.

Laboratory results are obtained and show:

Serum sodium	140 mEq/L
Serum potassium	3.8 mEq/L

Alanine amino**t**ransferase (ALT)	250 U/L
Aspartate amino**t**ransferase (AST)	170 U/L
Alkaline **p**hosphatase (ALP)	120 U/L

Reference values can be found at the end of the book

Which of the following antibodies would you expect to find in this patient?

A. Anti-mitochondrial antibody
B. Anti-endomysial IgA
C. Perinuclear anti-neutrophil cytoplasmic antibodies (p-ANCA)
D. Anti-cyclic citrullinated peptide (anti-CCP)
E. Anti-double stranded DNA (anti-dsDNA)

Need a Hint?

Steps

Step 1: Determine the diagnosis

Step 2: Determine the antibody associated with the diagnosis

Characteristics

- Pathology – gastrointestinal question
- 2-step
- Stem is required

WATCH LECTURER VIDEO

Crohn's Disease
http://lectur.io/ischemicheartcrohns

Walkthrough

Step 1: Determine the diagnosis

- **Bloody diarrhea** and **abdominal pain** combined with **jaundice**
- Suggestive of **generalized gastrointestinal disorder**
- Differential includes **inflammatory bowel disease** (IBD: Crohn's and ulcerative colitis), **malignancy,** and **GI infection.**
- **Malignancy unlikely** given the absence of related symptoms and patient's age
- **Infection also unlikely** given the absence of fever and long duration of symptoms
- Crohn's and ulcerative colitis (UC) have **distinctive features.**
- Crohn's can affect **entire GI tract** and commonly **skips segments**.
- Often presents with **extraintestinal symptoms** such as lesions to the skin and mouth
- UC affects the **large bowel and does not skip segments.**
- UC seems most likely diagnosis and is supported by jaundice, as UC patients often suffer from **primary sclerosing cholangitis (PSC).**

Step 2: Determine the antibody associated with the diagnosis

- Most patients with UC-associated primary sclerosing cholangitis test positive for **P-ANCA antibodies.**
- The other answer choices are associated with pathologies unlikely in this patient.

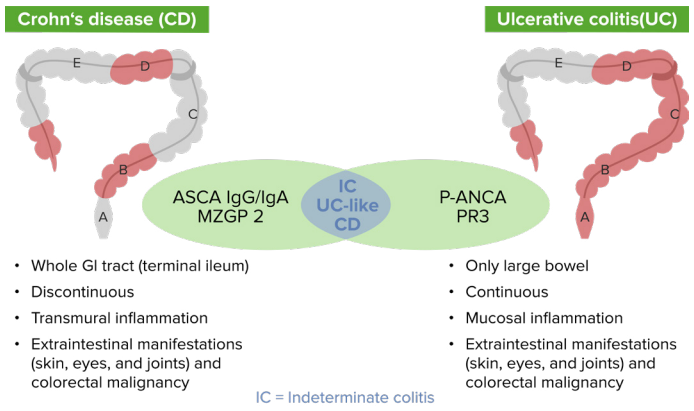


Image: *Comparison of inflammatory bowel diseases Crohn's and ulcerative colitis. © by Lecturio*

High-yield Facts

Inflammatory bowel disease (IBD):

- Group of inflammatory disorders affecting GI tract: **ulcerative colitis** and **Crohn's disease**
- Ulcerative colitis affects **continuous segments** of the GI system and is most common in the **rectum and descending colon.**
- Crohn's disease generally affects **intermittent segments of GI** system.
- Crohn's often diagnosed based on **findings in the mouth**
- Common IBD symptoms: **Bloody diarrhea, abdominal bloating,** and **pain**
- Treatment may be **surgical** or medical with **immunosuppression**

The correct answer is: Perinuclear anti-neutrophil cytoplasmic antibodies (p-ANCA)!

Got it right?
Make a note! ✓ ✕

Question Review

A 6-year-old boy is brought to a pediatric clinic by his mother with complaints of fever, malaise, and cough for the past 2 days. He frequently complains of a sore throat and has difficulty eating solid foods. The mother mentions that initially, the fever was low-grade and intermittent in nature but later became high-grade and continuous. The boy was born at 39 weeks gestation via spontaneous vaginal delivery. He is up to date on all vaccines and is meeting all developmental milestones. Past medical history is noncontributory. He takes a multivitamin every day. The mother reports that he does well in school and is helpful around the house. Today, his blood pressure is 110/65 mm Hg, heart rate 110/min, respiratory rate 32/min and, blood pressure of 110/65 mm Hg, and temperature of 38.3°C (101.0°F). On physical exam, the boy appears uncomfortable and has difficulty breathing. His heart is mildly tachycardic with a regular rhythm and his lungs are clear to auscultation bilaterally. Oropharyngeal examination shows his palatine tonsils are covered with pus and there is erythema of the surrounding mucosa.

Which of the following mediators is responsible for this patient's abnormal temperature?

A Leukotriene D4
B Thromboxane A2
C Prostaglandin E2
D Prostaglandin I2
E Prostaglandin F2

Need a Hint?

Steps

Step 1: Determine type of abnormal temperature

Step 2: Determine which chemical mediator produces fever

Characteristics

- Pathology – general question
- 2-step
- Stem is required

WATCH LECTURER VIDEO

Body Temperature Evaluation
http://lectur.io/bodytemperature

Walkthrough

Step 1: Determine type of abnormal temperature

- Normal body temperature is **36.5–37.5 °C** (97.7–99.5 °F)
- Differential diagnosis for abnormal temperature is **hypothermia** (< 36.5 °C (97.7 °F)) and **hyperthermia** (fever, > 37.5 °C (99.5 °F))
- Temperature recordings show that the **boy has a fever**, compatible with his mother's statements and the boy's clinical presentation is the possibility of infection (tonsillitis)

Step 2: Determine which chemical mediator produces fever

- Fever is caused by **alteration of the hypothalamic thermoregulatory center's "set point"**
- Substances that induce fever are referred to as **"pyrogens"**, both exogenous and endogenous
- Endogenous pyrogens include **IL-1, TNF-alpha, IL-6 and prostaglandin E2 (PGE-2)**, the latter in particular in acute inflammation
- Of the answer choices provided, **PGE-2 is the mediator inducing fever**

➜ PGE-2 arises from **arachidonic acid**

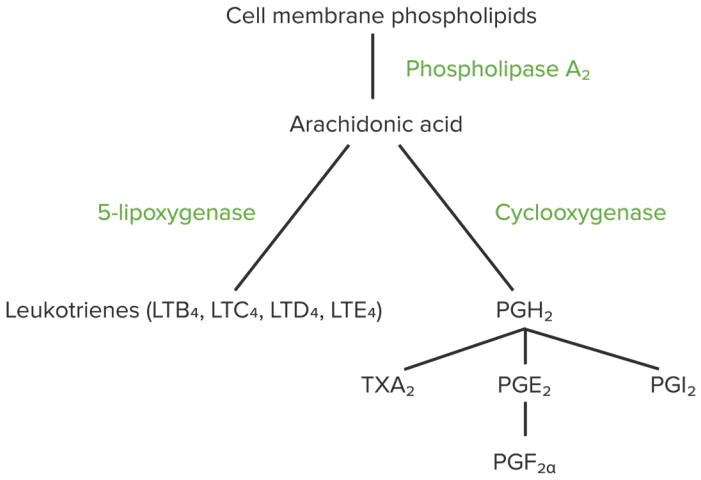


Image: *Derivatives of arachidonic acid © by Lecturio*

High-yield Facts

Fever:

- Caused by fever-inducing substances from outside or inside the body **("pyrogens")**
- Pyrogens change **hypothalamic regulatory set point**
- Change of set point induces **vasoconstriction** to reduce heat loss and **shivering** to increase heat production
- Underlying causes are **manifold** and generally related to **infection or inflammation**

The correct answer is: Prostaglandin E2!

WALK THROUGH VIDEO

Got it right? Make a note! ✓ ✗

Question Review

A 40-year-old woman presents to the emergency department with severe left upper quadrant pain (duration 3 hours, stabbing quality, 10/10 on the pain scale). Past medical history is significant for sickle cell anemia. Physical examination is significant for severe tenderness to palpation in the left upper quadrant. Significant splenomegaly is also noted. The patient is admitted to the hospital for close observation and placed on DVT prophylaxis as part of routine protocol. Laboratory findings drawn some time after admission demonstrate a normal PT and elevated PTT.

Which of the following factors is most directly affected by her DVT prophylaxis?

A VII
B XIIa
C VIIa
D X
E XII

Need a Hint?

Steps

Step 1: Determine which DVT prophylaxis the patient is receiving

Step 2: Determine which clotting factors are most affected by heparin

Characteristics

- Pathology – hematology question
- 2-step
- Stem is required

WATCH LECTURER VIDEO

Heparin vs. Warfarin
http://lectur.io/heparinvswarfarin

Walkthrough

Step 1: Determine which DVT prophylaxis the patient is receiving

- **Heparin** is routinely used for DVT prophylaxis in the hospital
- Heparin has **rapid onset of action**
- Can be **discontinued on discharge** without longer term effects

Step 2: Determine which clotting factors are most affected by heparin

- Heparin binds to enzyme inhibitor antithrombin III
- This in turn **inactivates thrombin and activated factor X** (factor Xa), interfering with the **common pathway** *(see image)*
- Normal prothrombin time **(PT) is consistent** with functioning tissue factor (extrinsic) coagulation pathway
- **Prolongation of PTT** is observed with inhibition of the **contact activation (intrinsic) and common pathway**

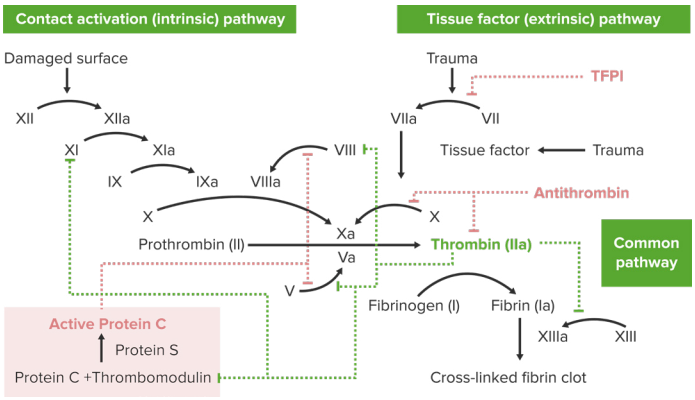


Image: *Intrinsic and extrinsic pathways of coagulation. © by Lecturio*

High-yield Facts

Heparin:

- **Fast onset of action** and a **short half-life**
- Can be easily **discontinued in hospital with no post-discharge effects**
- Interferes with common pathway of coagulation by binding to antithrombin III which in **turn inactivates thrombin and activated factor X**
- **Heparin prolongs PTT** which is a measure of the functioning of the contact activation (intrinsic) and common pathway
- Heparin has **no effect on the PT** which is a measure of the functioning of the tissue factor (extrinsic) pathway

The correct answer is: X!

Got it right?
Make a note! ✓ ✗

Question Review

A 3-year-old boy presents to the office with his mother. She states that her son seems weak and unwilling to walk. He only learned how to walk recently after a very notable delay. The boy was born at 39 weeks gestation via spontaneous vaginal delivery. He is up to date on all vaccines and is meeting all verbal and social milestones but he has a great deal of trouble with gross and fine motor skills. Past medical history is noncontributory. He takes a multivitamin every day. The mother states that some boys on her side of the family have had similar symptoms and worries that her son might have the same condition. Today, his blood pressure is 110/65 mm Hg, heart rate is 90/min, respiratory rate is 22/min, and his temperature is 37.0°C (98.6°F). On physical exam, the body appears well developed and pleasant. He sits and listens and follows direction. His heart has a regular rate and rhythm and his lungs are clear to auscultation bilaterally. He struggles to get up to a standing position after sitting on the floor. A genetic study is performed that reveals a significant deletion in the gene that codes for dystrophin.

Which of the following is the most likely diagnosis?

A. Duchenne muscular dystrophy
B. Becker muscular dystrophy
C. Limb-girdle muscular dystrophy
D. Myotonic muscular dystrophy
E. Emery-Dreifuss muscular dystrophy

Need a Hint?

Steps

Step 1: Determine whether the symptoms suggest an inherited muscular dystrophy

Step 2: Determine which type of muscular dystrophy the child might have

Characteristics

- Pathology – musculoskeletal question
- 2-step
- Stem is required

WATCH LECTURER VIDEO

Duchenne vs. Becker Muscular Dystrophy
http://lectur.io/duchennebeckermusculardyst

Walkthrough

Step 1: Determine whether the symptoms suggest an inherited muscular dystrophy

- Muscular dystrophy is characterized by **progressive weakness** and **loss of muscle mass.**
- Symptoms of the most common types of muscular dystrophy begin in childhood, **mostly in boys.**
- Majority of cases have a **family history** but in some cases there is no family history due to a spontaneous gene mutation.

Step 2: Determine which type of muscular dystrophy the child might have

- Presentation consistent with **Duchenne muscular dystrophy (DMD).**
- DMD has **onset in early childhood**, with **boys most commonly affected.**
- DMD is caused by the **absence of functional dystrophin.**
- Becker muscular dystrophy is unlikely as symptoms **generally begin in teens.**
- Limb-girdle muscular dystrophy is unlikely as **hip and shoulder muscles** are usually affected first.
- Myotonic muscular dystrophy is unlikely as **facial and neck muscles** are usually affected first.
- Emery-Dreifuss muscular dystrophy is unlikely as presentation is **generally in teenage years.**

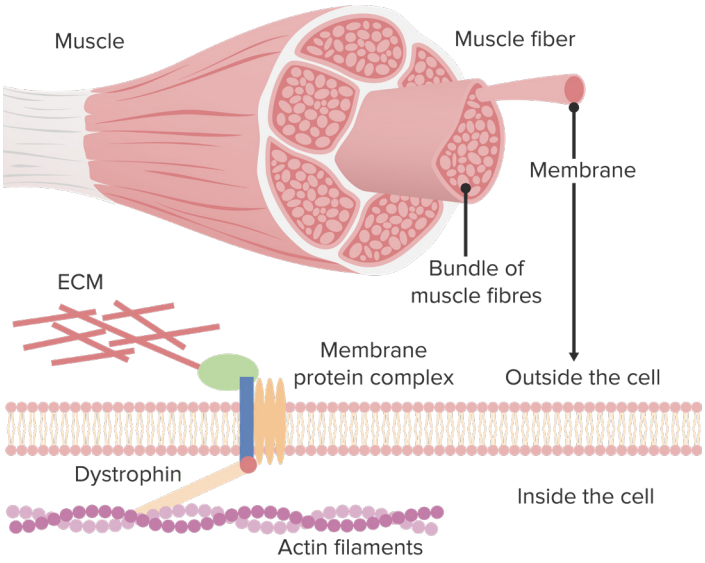


Image: *Pathophysiology of Duchenne's muscular dystrophy © by Lecturio*

High-yield Facts

Muscular dystrophy:

- Group of diseases characterized by **progressive weakness** and **loss of muscle mass**
- Caused by **gene mutations** that interfere with production of key proteins for muscle health
- Several different types of muscular dystrophy, with **most common form (Duchenne)** affecting around one in 3,500 boys
- Some variants of muscular dystrophy present in **childhood**, with others presenting in **adolescence or later.**
- Majority of the cases have family history but can also result from **spontaneous gene mutation.**

Duchenne muscular dystrophy:

- Caused by absence of **dystrophin**, a protein that is important for muscle health
- Symptoms generally begin **between the ages 3 and 5.**
- Characterized by **frequent falls, difficulty running, jumping** and **climbing stairs**, waddling gait, walking on toes, and large calf muscles
- Disease has **X-linked recessive inheritance pattern**, primarily affecting boys, with girls as carriers (although in rare cases girls may be affected).
- No known cure and **death generally occurs in the teens due to involvement of heart and respiratory muscles.**

The correct answer is: Duchenne muscular dystrophy!

Got it right? Make a note! ✓ ✗

Question Review

A 42-year-old woman presents with facial asymmetry. She says yesterday she noticed that her face appeared to be deviated to the right. She denies any trauma or recent travel. Her past medical history is noncontributory. Her vitals are a blood pressure of 110/78 mm Hg, temperature of 36.5°C (97.8°F), pulse of 78/min, and respiratory rate of 11/min. On physical examination, there is drooping of the left side of the face. The left nasolabial fold is absent, and she is unable to close her left eye or wrinkle the left side of her forehead. When the patient is asked, the resulting asymmetry is shown in the given photograph (see image below). The remainder of the neurologic exam is normal. A noncontrast CT scan of the head is unremarkable.

Which of the following is the most likely cause of her presentation?

A. Varicella zoster infection
B. Lyme Disease
C. Idiopathic
D. Cerebrovascular accident
E. Malignancy

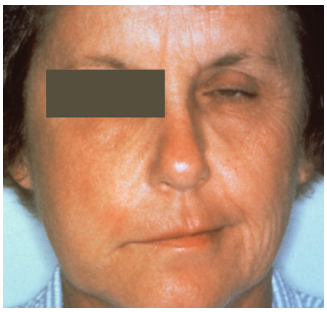


Image: *CDC, 1993, PD*
https://phil.cdc.gov/Details.aspx?pid=6633

Need a Hint?

Steps

Step 1: Determine which nerve is affected

Step 2: Determine cause of facial nerve palsy

Characteristics

- Pathology – neurology question
- 2-step
- Stem is required

WATCH LECTURER VIDEO

Cranial Nerve VII: Facial Nerve
http://lectur.io/cranialnerve

Walkthrough

Step 1: Determine which nerve is affected

- Signs suggest **facial nerve palsy.**
- Facial nerve palsy can be due to either **central lesion** (supranuclear lesion) or peripheral lesion to **facial nerve** itself.
- Supranuclear and peripheral lesions **present with different symptoms** *(see image)*
- In **one-sided** supranuclear lesions, patients **maintain the ability to move** their forehead and close their eye.
- This is due to **bilateral upper motor innervation of upper face.**
- In peripheral lesions, patients are **unable to move any part of their face on affected side**
- Patient shows **peripheral lesion.**

Step 2: Determine cause of facial nerve palsy

- Peripheral or lower motor neuron facial nerve palsy may occur following
  - Skull fracture or injury to the face
  - Head or neck tumor
  - Middle ear damage (such as following varicella zoster infection)
- Direct nerve damage due to Lyme disease
- It may also be idiopathic (without known cause)

➜ In **absence of any history or signs suggestive of an underlying cause**, the facial nerve palsy is likely to be **idiopathic**, generally referred to as **Bell's palsy**

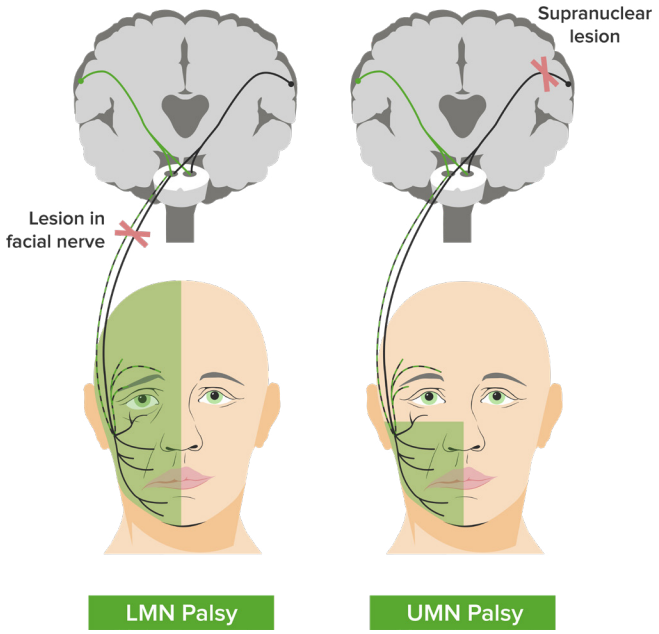


Image: *Pathophysiology and presentation of lower vs. upper motor neuron facial palsies © By: Lecturio*

High-yield Facts

Facial nerve palsy:

- Characterized by **unilateral facial weakness**
- Other symptoms include **loss of taste** and **decreased salivation** and **tear secretion.**
- Lower motor neuron facial palsy: **Patient cannot close eye or raise forehead** with all muscle weakness on the ipsilateral side.
- Upper motor neuron facial palsy: Upper face **is spared** due to the bilateral upper motor innervation.

Causes of facial nerve palsy:

- Main cause of upper motor neuron facial palsy is **cerebrovascular accident**, which results in lower facial weakness contralateral to the neurological lesion.
- Lower motor neuron facial palsy may be due to **trauma at base of the skull, head or neck tumor, middle ear damage,** or from **direct nerve damage** due to Lyme disease.
- Most **lower motor neuron facial palsies have no known cause** and are termed idiopathic (Bell's palsy).

The correct answer is: Idiopathic!

Got it right? Make a note! ✓ ✗

Question Review

A 62-year-old man goes to the emergency room for an intense lower abdominal pain associated with inability to urinate. Physical examination shows tenderness of the lower abdomen bilaterally. Rectal examination reveals an enlarged, smooth, and symmetrical prostate. The ER team fails to pass a Foley catheter through the urethra, and the urology team decides to place a suprapubic catheter to drain the urine and relieve the patient's symptoms. An ultrasound shows dilation of the collecting system in both kidneys. Laboratory test shows an elevated serum creatinine of 1.6 mg/dL for an estimated glomerular filtration rate (eGFR) of 50 ml/min/1.73 m^2. The patient visits the urology team for a follow-up visit 3 weeks after the acute event, in which he claims to have close to normal urination. However, his serum creatinine stays elevated at 1.5 mg/dL.

What renal gross findings correlate with this patient's condition?

A. Varicella zoster infection
B. Lyme Disease
C. Idiopathic
D. Cerebrovascular accident
E. Malignancy

Need a Hint?

Steps

Step 1: Determine the likely diagnosis

Step 2: Determine the likely macroscopic renal changes

Characteristics

- Pathology – renal question
- 2-step
- Stem is required

WATCH LECTURER VIDEO

Prostatitis (Prostate Infection): Types
http://lectur.io/prostatitis

Walkthrough

Step 1: Determine the likely diagnosis

- Patient is presenting with **acute urinary obstruction** secondary to benign prostatic hypertrophy (**BPH**) *(see image)*
- Presence of elevated creatinine three weeks after relief of acute obstruction is suggestive of **renal damage due to chronic urinary outflow obstruction** secondary to BPH

Step 2: Determine the likely macroscopic renal changes

- Chronic urinary outflow obstruction results in dilatation of urinary tract and renal damage (**obstructive nephropathy**)
- ➜ Obstructive nephropathy is characterized by progressive atrophy of the renal cortex, leading to **thin cortical rim** of the kidney

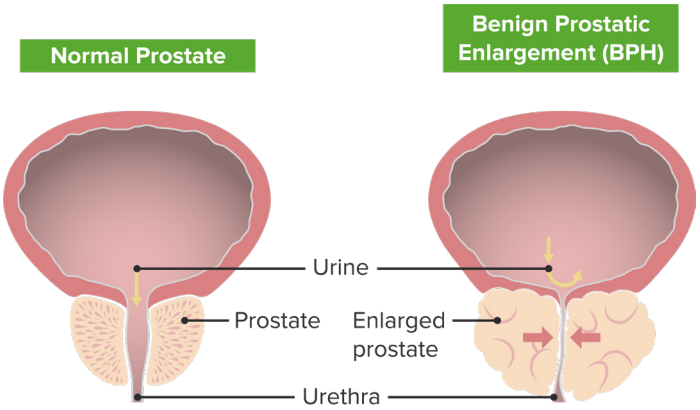


Image: *Urinary obstruction in benign prostatic hyperplasia (BPH) © by Lecturio*

High-yield Facts

Benign prostatic hypertrophy:

- **Common in men after middle age** (present in around half of men aged over 50 years)
- Commonly presents with symptoms related to **increased frequency** of urination (typically at night), **difficulty** starting urination or a weak urine stream
- BPH can present as **acute urinary obstruction** with abdominal pain and urinary retention

Obstructive nephropathy:

- **BPH can result in an obstructive nephropathy** due to chronic urinary outflow obstruction
- Obstructive nephropathy is characterized by **atrophy of the renal cortex**
- Typical macroscopic findings of obstructive nephropathy is the **thinning of cortical rim** of the kidney

The correct answer is: Thin cortical rim!

Got it right? Make a note! ✓ ✗

Question Review

A 72-year-old man presents to his primary care provider frustrated about the frequency he wakes up at night to urinate. He comments that he is even avoiding drinking liquids at night, but the symptoms have progressively worsened. Past medical history is significant for hypertension and hyperlipidemia. He takes lisinopril, atorvastatin, and a multivitamin every day. Today, his blood pressure is 120/80 mm Hg, heart rate is 90/min, respiratory rate is 17/min, and temperature is 37.0°C (98.6°F). On physical exam, he appears tired. His heart has a regular rate and rhythm and his lungs are clear to auscultation bilaterally. A bedside bladder ultrasound reveals a full bladder. Digital rectal exam reveals an enlarged and symmetrical prostate free of nodules, consistent with benign prostatic enlargement. The patient declines a prostate biopsy that would provide a definitive diagnosis and requests a less invasive treatment.

Which of the following is recommended to treat this patient's enlarged prostate?

A Tamsulosin
B Finasteride
C Prazosin
D Tadalafil
E Leuprolide

Need a Hint?

Steps

Step 1: Determine the likely diagnosis

Step 2: Determine the most appropriate treatment

Characteristics

- Pathology – reproductive question
- 2-step
- Stem is required

WATCH LECTURER VIDEO

Antiandrogen (Androgen Receptor Antagonists)
http://lectur.io/antiandrogen

Step 1: Determine the likely diagnosis

- Patient complaining of **nocturia**
- Nocturia is the common presenting symptom of **benign prostatic hyperplasia** (BPH)

➔ Absence of other symptoms makes BPH the most likely diagnosis

Step 2: Determine the most appropriate treatment

- **Finasteride:**
  - Effective in reducing prostate size in BPH
  - **Well tolerated in elderly** patients *(see image – most side effects related to sexual function due to inhibition of conversion of testosterone to dihydrotestosterone)*
- **Tamsulosin and prazosin:**
  - Effective in reducing nocturia in BPH
  - **Significant side effects** are orthostatic hypotension and syncope
  - Not suitable for the elderly
- **Tadalafil:**
  - Treatment of **erectile dysfunction**
- **Leuprolide:**
  - GnRH receptor agonist for **hormone-responsive cancers** such as prostate and breast cancers

➔ **Finasteride is the most appropriate treatment**

Mode of Action of Dutasteride and Finasteride

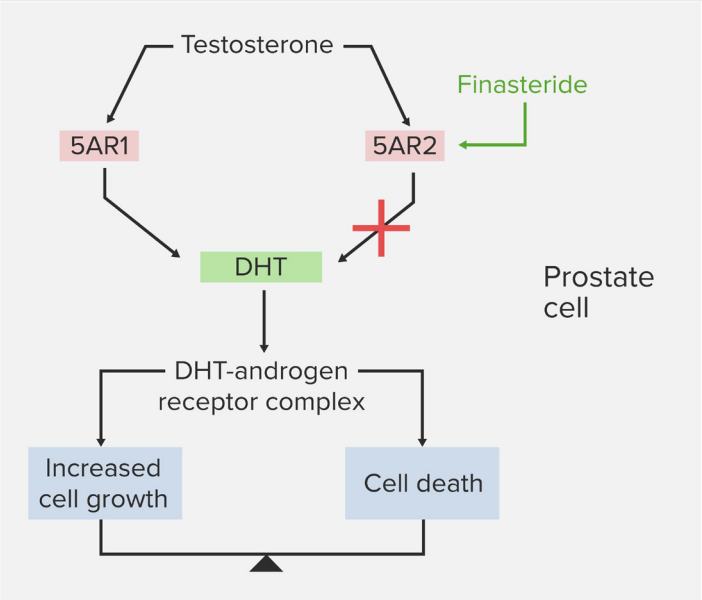


Image: *Mechanism of action of 5-alpha reductase inhibitors © by Lecturio*

Benign prostatic hypertrophy (BPH):

- Common in **men** after **middle age**
- Common urinary **symptoms:**
  - Frequent or urgent need to urinate
  - Increased need to urinate at night (nocturia)
  - Difficulty starting urination
  - Weak urine stream or stop-start stream
  - Dribbling at end of urination
  - Inability to completely empty the bladder

Treatment of BPH:

- **Alpha-1 adrenergic blockers and 5α-reductase inhibitors**
- Alpha blockers (e.g. tamsulosin and prazosin) effective but common **side effects** include orthostatic hypotension
- 5α-reductase inhibitors effective and well tolerated
- Surgery **generally reserved for patients** in whom drug treatment ineffective

The correct answer is: Finasteride!

WALK THROUGH VIDEO

Got it right? Make a note! ✓ ✗

Question Review

A 62-year-old man presents to the emergency department for evaluation of a 2-year history of increasing shortness of breath. He also has an occasional non-productive cough. The symptoms get worse with exertion. The medical history is significant for hypertension and he takes chlorthalidone. He is a smoker with a 40-pack-year smoking history. On physical examination, the patient is afebrile, the blood pressure is 125/78 mm Hg, the pulse is 90/min, and the respiratory rate is 18/min. The BMI is 31 kg/m^2. The oxygen saturation is 94 % at rest on room air. A pulmonary examination reveals decreased breath sounds bilaterally, but is otherwise normal with no wheezes or crackles. The remainder of the examination is unremarkable. A chest radiograph shows hyperinflation of both lungs with mildly increased lung markings, but no focal findings.

Based on this clinical presentation, which of the following is most likely?

A FEV1/FVC of 65 %
B Decreased total lung capacity
C Increased DLCO
D Metabolic acidosis
E FEV1 of 82 %

Need a Hint?

Steps

Step 1: Determine the likely diagnosis

Step 2: Determine the likely lung function test results

Characteristics

- Pathology – respiratory question
- 2-step
- Stem is required

WATCH LECTURER VIDEO

Spirometry – Laboratory Diagnostics
http://lectur.io/spirometry

Walkthrough

Step 1: Determine the likely diagnosis

- Characteristic symptoms and signs of **emphysema** *(see image)*
- Long history of **smoking**
- Smoking is the **leading cause of COPD**

Step 2: Determine the likely lung function test results

- COPD is characterized by **reduction in FEV1/FVC**
- Reduced FEV1 due **to comprised expiratory flow** (due to low lung recoil) with relatively normal vital capacity
- Emphysema is characterized by alveolar wall destruction, decreasing overall surface area for gas transfer resulting in **decreased diffusing capacity (DLCO)**
- Total **lung capacity increased** in emphysema due to hyper-inflation

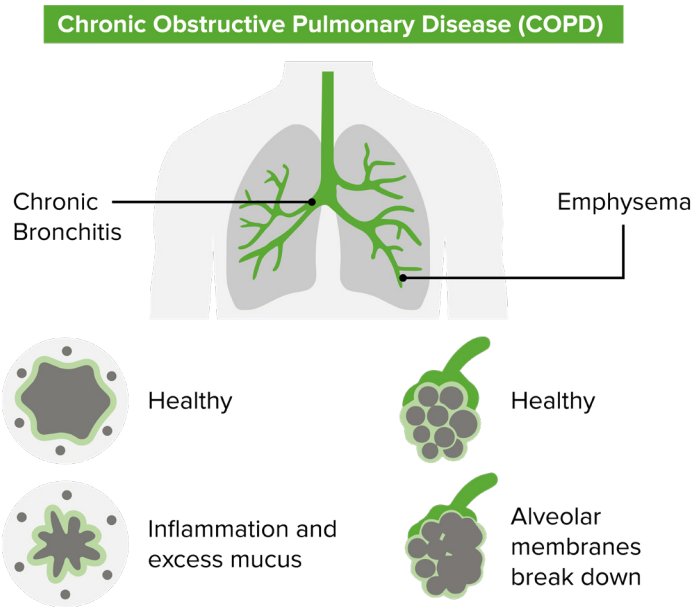


Image: *Chronic bronchitis and emphysema in COPD © by Lecturio*

High-yield Facts

COPD:

- Smoking is the **leading cause**
- Two subtypes: **chronic bronchitis** and **emphysema** *(see image)*
- Both types are characterized by **increasing shortness of breath** due to airways obstruction, with spirometry showing reduced FEV1/FVC (below 70 %)
- Chronic bronchitis is characterized by **increase in mucous production** and **cough**
- Emphysema characterized by **destruction of the alveolar walls**, with loss of elastic recoil of lungs resulting in hyperinflation and increased total lung capacity

The correct answer is: FEV1/FVC of 65 %!

Got it right? Make a note! ✓ ✗

Question Review

A 45-year-old male was shown to have a blood pressure of 142/90 mmHg at a health fair. Despite modifying his lifestyle, his blood pressure remained elevated on two separate subsequent occasions. He was prescribed an anti-hypertensive medication. After 3 weeks, the swelling of the lips shown in the accompanying photograph was observed.

What is the most likely cause of this finding?

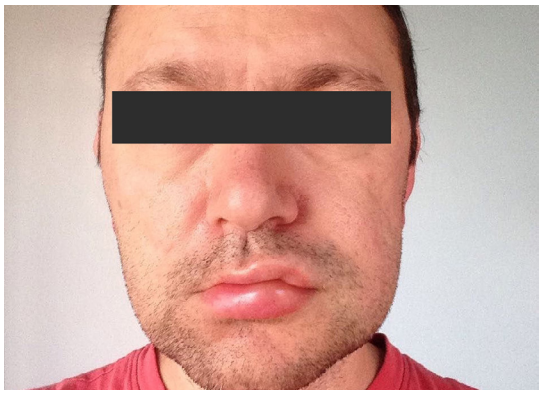


Image: *Giles Turnbull, Shout out the swollen face massive, CC BY 2.0*

A. Verapamil
B. Amlodipine
C. Lisinopril
D. Hydrochlorthiazide
E. Furosemide

Need a Hint?

Steps

Step 1: Determine the diagnosis

Step 2: Determine the cause of the patient's angioneurotic edema

Characteristics

- Pharmacology question
- 2-step
- Stem is required

WATCH LECTURER VIDEO

Angioedema
http://lectur.io/angioedema

Walkthrough

Step 1: Determine the diagnosis

- **Swollen, edematous,** and **red lips**
- Typical of angioneurotic edema
- **Angioneurotic edema** is characterized by red swelling usually near eyes and lips
- Can also occur on hands, feet, and inside of throat

Step 2: Determine the cause of the patient's angioneurotic edema

- Angioneurotic edema can be hereditary **or acquired** (e.g. medication-related).
- Hereditary is due to inherited C1 esterase inhibitor deficiency.
- **Nothing in patient history** suggests hereditary angioneurotic edema.
- Patient is receiving **antihypertensive treatment.**
- Commonly prescribed antihypertensive agents include:
  - Thiazide diuretics
  - Angiotensin-converting enzyme (ACE inhibitors)
  - Angiotensin-receptor blockers (ARBs)
  - Calcium channel blockers (CCBs)
  - Direct renin inhibitors
- ➔ **ACE inhibitors** (e.g. Lisinopril) **well recognized to cause an angioneurotic edema** due to increased levels of potent vasodilator **bradykinin** *(see image)*
- Of the other antihypertensive agents listed, CCBs can cause edema, but peripherally.

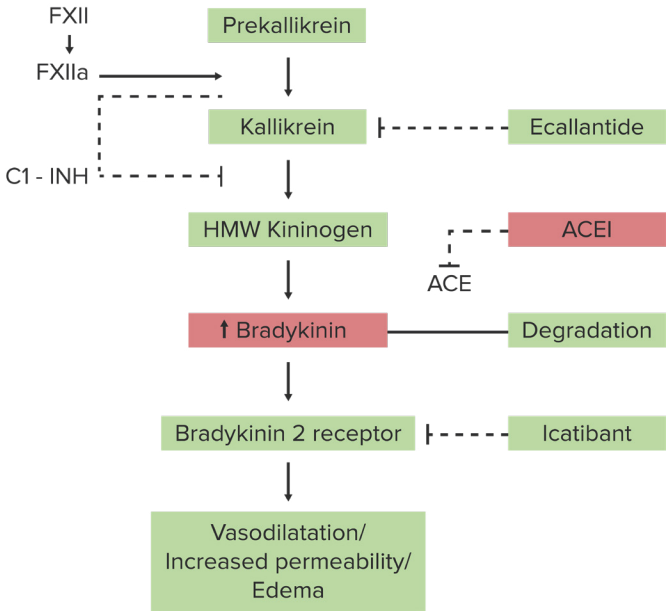


Image: *Mechanism of angioedema induced by ACE inhibitors © By: Lecturio*

High-yield Facts

Angioneurotic edema and angiotensin-converting enzyme (ACE) inhibitors:

- ACE inhibitors leading cause of **drug-induced angioedema**
- Most common presentation:
  - Swelling of lips, tongue, or face
  - Itching generally absent
- Signs and symptoms range from mild and clinically insignificant to severe and life-threatening.
- Symptoms usually occur **within first 3 months of starting treatment.**
- Can occur shortly **after the first dose up to 2 years** after beginning ACE inhibitors

The correct answer is: Lisinopril!

Got it right? Make a note! ✓ ✗

Question Review

The parents of a 16-year-old boy with type 1 diabetes mellitus present requesting information about the drug, exenatide, an injectable drug that only needs to be administered once a week. The patient's blood glucose levels have been difficult to control on his current insulin regimen due to poor compliance, and he has had difficulty putting on weight despite eating copiously. The patient is afebrile and vital signs are within normal limits. His BMI is 19 kg/m^2.

Which of the following best describes why the patient should not be switched to exenatide?

A. Insulin production by the pancreas is insufficient for exenatide to function
B. Exenatide suppresses glucagon secretion which increases the risk of hypoglycemia
C. Suppression of appetite makes it even harder for him to gain weight
D. Exenatide is contraindicated in children below 18 years
E. Gastric emptying is inhibited by exenatide

Need a Hint?

Steps

Step 1: Determine the reason for hyperglycemia in type 1 diabetes

Step 2: Determine the mechanism of action of exenatide and its suitability for type 1 diabetes

Characteristics

- Pharmacology question
- 2-step
- Stem is required

WATCH LECTURER VIDEO

DPP-4 Inhibitors and GLP-1 Analogues – Diabetes Medications
http://lectur.io/dpp4inhibitorsandglp1

Walkthrough

Step 1: Determine the reason for hyperglycemia in type 1 diabetes

- Pancreas produces **little or no insulin**
- **Cause is destruction of beta cells** in islets of Langerhans

Step 2: Determine the mechanism of action of exenatide and its suitability for type 1 diabetes

- Exenatide is glucagon-like peptide-1 receptor agonist **(GLP-1 receptor agonist)** *(see image)*
- Exenatide **increases insulin secretion** from pancreas in response to meals
- Exenatide is **indicated in type 2 diabetes**
- Exenatide is **not suitable for type 1 diabetes** as the few remaining beta cells in type 1 diabetes already maximally stimulated

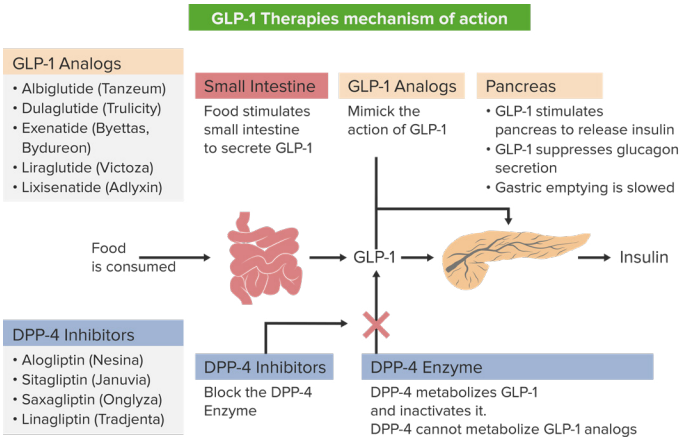


Image: *Mechanism of action of different GLP-1 therapies incl. exenatide, a GLP-1 analog*
© by Lecturio

High-yield Facts

Type 1 diabetes:

- Pancreas produces **little or no insulin**
- **Cause is the destruction of beta cells**
- Likely due to **autoimmune mechanism,** with some **genetic predisposition**
- **Treatment** of choice in type 1 diabetes is **insulin** as the few remaining beta cells already functioning at maximum capacity

GLP-1 receptor agonists:

- Part of group of **incretin mimetics**
- **Increase insulin secretion** from pancreas
- **Decrease glucagon secretion** by the liver in response to meals
- Indicated for type 2 diabetes
- Not suitable for type 1 diabetes as remaining islet cells are already maximally stimulated
- Exenatide is GLP-1 agonist

The correct answer is: Insulin production by the pancreas is insufficient for exenatide to function!

Got it right? Make a note! ✓ ✗

Question Review

A 24-year-old woman is brought to the ER by her co-workers after they found her unconscious at her cubicle when they returned from lunch. They tell you that she has diabetes but do not know anything more about her condition. Her vitals show a pulse of 110/min, respiratory rate of 24/min, a temperature of 36.7°C (98.0°F) and a blood pressure of 90/60 mm Hg. On physical examination, the patient is breathing heavily and gives irrelevant responses to questions. Skin and mucous membranes appear dry. Examination of the abdomen reveals mild diffuse tenderness to palpation.

Deep tendon reflexes in the extremities are 1+ bilaterally. Laboratory findings are significant for the following:

Finger stick glucose: 630 mg/dL

Arterial blood gas analysis:	
pH	7.1
Po_2	90 mm Hg
Pco_2	33 mm Hg
HCO_3	8 mEq/L

Serum:	
Sodium	135 mEq/L
Potassium	3.1 mEq/L
Chloride	136 mEq/L
Blood urea nitrogen	20 mg/dL
Serum creatinine	1.2 mg/dL

Urine examination shows:	
Glucose	Positive
Ketones	Positive
Leucocytes	Negative
Nitrite	Negative
RBC	Negative
Casts	Negative

Reference values can be found at the end of the book

The patient is immediately started on a bolus of IV 0.9 % NaCl. Which of the following is the next best step in the management of this patient?

A. Infuse $NaHCO_3$ slowly
B. Switch fluids to 0.45 % NaCl
C. Start IV insulin infusion
D. Replace potassium intravenously
E. Start IV 5 % dextrose

Need a Hint?

Steps

Step 1: Determine the likely diagnosis

Step 2: Determine the most urgent action

Characteristics

- Pharmacology question
- 2-step
- Stem is required

WATCH LECTURER VIDEO

Diabetic Ketoacidosis (DKA) – Diabetes Complications
http://lectur.io/diabeticketoacidosis

Walkthrough

Step 1: Determine the likely diagnosis

- Patient has **diabetic ketoacidosis** due to uncontrolled hyperglycemia *(see image)*
- **Low potassium**, typical in diabetic ketoacidosis
- Due to **potassium depletion** associated with increased diuresis
- Reduced PH but is not below 7

Step 2: Determine the most urgent action

- **Hypokalemia** (low potassium) can be life-threatening
- Hypokalemia **exacerbated by insulin treatment** as glucose entry into cells will draw potassium in with it
- ➔ **Replace potassium** via intravenous infusion
- Bicarbonate indicated in **severe acidosis (pH below 7)** – not applicable in this patient

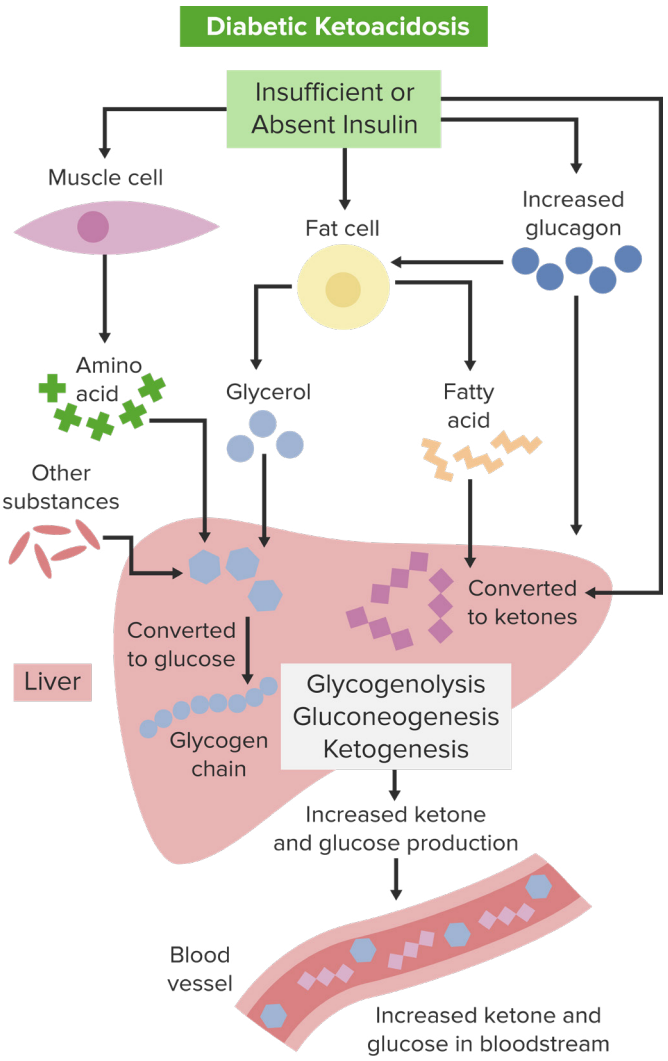


Image: *Pathophysiology of diabetic ketoacidosis © by Lecturio*

High-yield Facts

Diabetic ketoacidosis:

- **Life-threatening** complication of diabetes
- Patients with diabetic ketoacidosis require **rehydration** and **IV insulin**
- Hypokalemia must be corrected with IV potassium **before administration of IV insulin**
- If not, hypokalemia will be exacerbated by insulin therapy as **glucose entering the cells will draw in potassium with it**
- Use of IV bicarbonate in diabetic ketoacidosis should generally only **be considered in severe acidosis (pH < 7.0)**

The correct answer is: Replace potassium intravenously!

Got it right? Make a note! ✓ ✗

Question Review

An 85-year-old man presents with reappearance of his Parkinson's disease (PD) symptoms over the last few months. He says he has been treated with various drugs over the last 20 years, but currently his symptoms worsen as he nears the time for his next dose of medication. His movements have been slower lately and it's difficult to initiate voluntary movements. Past medical history is significant for hypertension, diagnosed 10 years ago and well-managed on medication. His current medications are levodopa/carbidopa, rasagiline, aspirin, and captopril. Vitals are a pulse of 70/min, a respiratory rate of 15/min, a blood pressure of 130/76 mm Hg, and a temperature of 36.7°C (98.1°F). Physical examination reveals the expected 'pill-rolling' resting tremor, which is alleviated by movement. Increased tone of arm muscles and resistance to passive movement at the joints is noted. When asked to walk across the room, he has difficulty taking the first step and has a stooped posture and takes short, shuffling, rapid steps.

Laboratory test show:

Serum glucose (fasting)	97 mg/dL	Cholesterol (total)	190 mg/dL
Sodium	141 mEq/L	HDL-cholesterol	42 mg/dL
Potassium	4.0 mEq/L	LDL-cholesterol	70 mg/dL
Chloride	100 mEq/L	Triglycerides	184 mg/dL

Reference values can be found at the end of the book

The patient is started on a drug that increases the efficacy of his current anti-PD medications. Which of the following is the drug that was most likely added to this patient's current regimen?

A Benztropine
B Selegiline
C Atorvastatin
D Entacapone
E Bromocriptine

Need a Hint?

Steps

Step 1: Determine the likely diagnosis

Step 2: Determine which drug would increase the efficacy of his levodopa/carbidopa

Characteristics

- Pharmacology question
- 2-step
- Stem is required

WATCH LECTURER VIDEO

MAO and COMT Inhibitors – Treatment of Movement Disorders
http://lectur.io/maoandcomtinhibitors

Walkthrough

Step 1: Determine the likely diagnosis

- **'End-of-dose'** deterioration or **'wearing off'**
- **Worsening of Parkinsonian symptoms** a few hours before next dose of levodopa/carbidopa
- Seen after **long-term use of levodopa/carbidopa** in advanced Parkinson's disease
- Blood results unremarkable

Step 2: Determine which drug would increase the efficacy of his levodopa/carbidopa

- **Inhibitors of catechol-O-methyltransferase (COMT)** increase efficacy of levodopa/carbidopa
- Mechanism is inhibition of breakdown of catecholamine neurotransmitters
- ➔ Entacapone is selective, reversible COMT inhibitor
- Patient already receiving a **monoamine oxidase-B inhibitor** (rasagiline)

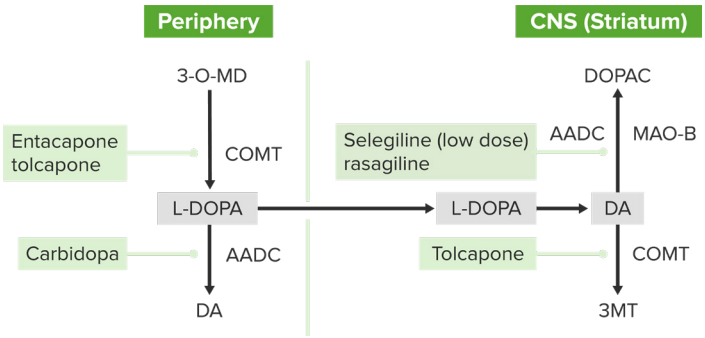


Image: *Pathophysiology of diabetic ketoacidosis © by Lecturio*

High-yield Facts

End-of-dose deterioration:

- Levodopa is the **most effective symptomatic treatment** of Parkinson's disease
- Levodopa **always given with peripheral decarboxylase inhibitor** (carbidopa or benserazide) to reduce conversion to dopamine
- Progressive loss of neurons with advanced disease causes **shortened duration** of each **levodopa dose**
- Return of symptoms some hours before the next dose, so-called **'end-of-dose deterioration'** or **'wearing off'**
- End-of-dose deterioration can be improved by the increasing frequency of levodopa doses or by addition of **COMT inhibitor** (such as **entacapone**)
- **COMT inhibitors** reduce breakdown of catecholamine neurotransmitters

The correct answer is: Entacapone!

Got it right?
Make a note! ✓ ✗

Question Review

A 15-year-old boy and his mother were referred to a pulmonology clinic. She is concerned that her son is having some breathing difficulty for the past few months. It is worse with exercise. The family is especially concerned because the boys older brother has cystic fibrosis. Past medical history is noncontributory. Today, his blood pressure is 119/80 mm Hg, heart rate is 90/min, respiratory rate is 17/min, and temperature is 37°C (98.6°F). On physical exam, he appears well-developed and well-nourished. His heart has a regular rate and rhythm and his lungs are clear to auscultation bilaterally. During the exam, he is brought into a special room to test his breathing. A clamp is placed on his nose and he is asked to take in as much air as he can and then forcefully expire all the air into a spirometer.

The volume of expired air represents which of the following?

A. Expiratory reserve volume
B. Functional residual capacity
C. Tidal volume
D. Total lung capacity
E. Vital capacity

Need a Hint?

Steps

Step 1: Consider which parameters are measured by spirometry

Step 2: Consider which of the above parameters represents the volume of forced expiration after deep inspiration

Characteristics

- Physiology question
- 2-step
- Stem is required

Walkthrough

Step 1: Consider which parameters are measured by spirometry

- **Volumes of air** inspired and expired by the lungs
- **FEV1, vital capacity, tidal volume, and inspiratory and expiratory reserve volumes** all measured by spirometry *(see image)*

Step 2: Consider which of the above parameters represents the volume of forced expiration after deep inspiration

- **Maximum volume** of air **forcibly expired after deepest possible inspiration** represents **vital capacity**
- ➜ Vital capacity is sum of **inspiratory reserve volume, tidal volume, and expiratory reserve volume.**
- Total lung capacity is vital capacity plus residual volume.

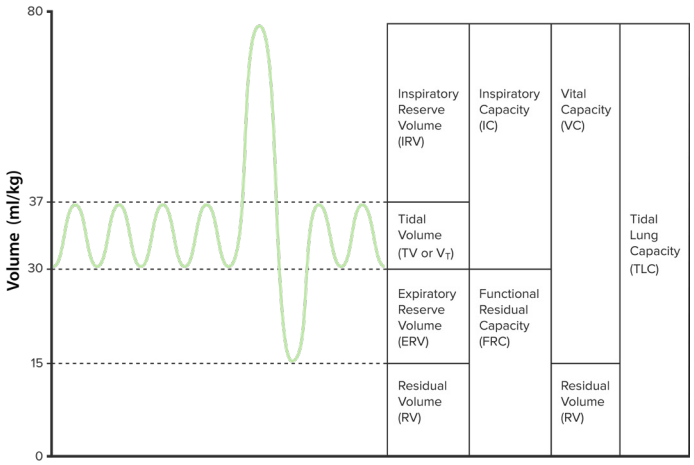


Image: *Respiratory volumes measured in spirometry © by Lecturio*

High-yield Facts

Spirometry:

- Spirometry measures the **volume of air inspired and expired by the lungs.**
- Spirometry can measure **FEV1, vital capacity, tidal volume, inspiratory,** and expiratory **reserve volumes.**
- Spirometry **cannot measure residual volume.**
- Total lung capacity is **vital capacity plus residual volume.**
- **Total lung capacity** cannot be measured by spirometry (residual volume unknown).

The correct answer is: Vital capacity!

Got it right? Make a note! ✓ ✗

Question Review

A 28-year-old woman presents to a physician with repeated muscle cramps for the last 2 weeks. She mentions that she commonly has these in the lower limbs and back. She also has a constant tingling sensation around her mouth. On physical examination, her vital signs are stable. The Trousseau sign and Chvostek sign are present and deep tendon reflexes are exaggerated. A comprehensive blood test reveals the following:

Na^+	140 mEq/L
K^+	4.5 mEq/L
Chloride	100 mEq/L

Bicarbonate	24 mEq/L
Creatinine	0.9 mEq/L
Ca^{2+}	7.0 mEq/L

Reference values can be found at the end of the book

Which of the following electrophysiologic mechanisms best explains the clinical features of the woman?

A Stimulation of GABA (γ-aminobutyric acid) receptors
B Increased firing threshold for action potential
C Reduction of afterhyperpolarization
D Inhibition of Na^+ and Ca^{2+} currents through cyclic nucleotide-gated (CNG) channels
E Inhibition of sodium current through sodium leak channels (NALCN)

Need a Hint?

Steps

Step 1: Determine the likely cause of her symptoms

Step 2: Determine the mechanisms by which hypocalcemia increases neuronal excitability

Characteristics

- Physiology question
- 2-step
- Stem is required

WATCH LECTURER VIDEO

Acute Hypocalcemia
http://lectur.io/acutehypocalcemia

Walkthrough

Step 1: Determine the likely cause of her symptoms

- Patient has marked **hypocalcemia** (normal range 2.2 to 2.7 mmol/L)
- Hypocalcemia leads to **increased neuronal excitability**
- **Trousseau sign** is a reliable clinical sign of latent tetany in patients with hypocalcemia, and is often accompanied by exaggerated reflexes

Step 2: Determine the mechanisms by which hypocalcemia increases neuronal excitability

- Hypocalcemia increases neuronal excitability through a number of mechanisms such as:
  - Increasing the resting membrane potential and **decreasing the firing threshold**
  - **Inhibiting GABA receptors**
  - **Reducing afterhyperpolarization**
  - **Increasing currents** through cyclic nucleotide-gated (**CNG**) and sodium leak channels (**NALCN**)

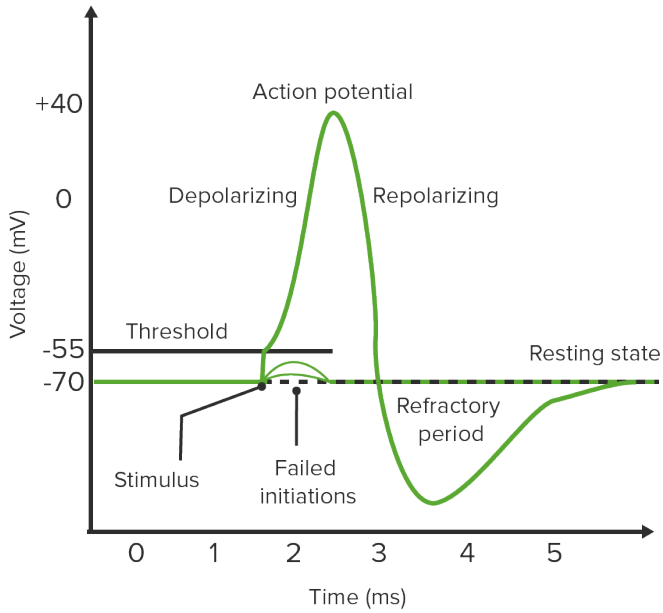


Image: *Electrical potential in neuronal firing © by Lecturio*

High-yield Facts

Hypocalcemia and neuronal excitability:

- Hypocalcemia can cause **increased neuronal excitability**
- This may present as **muscle stiffness** and cramps or **generalized tetany**
- **Trousseau sign** is a reliable clinical sign of latent tetany
- Hypocalcemia increases neuronal excitability through a number of mechanisms

The correct answer is: Reduction of afterhyperpolarization!

Blood

Blood, Plasma, Serum	Reference Range	SI Reference
* Alanine aminotransferase (ALT)	8–20 U/L	8–20 U/L
Amylase, serum	25–125 U/L	25–125 U/L
* Aspartate aminotransferase (AST)	8–20 U/L	8–20 U/L
Bilirubin, serum (adult) Total // Direct	0.1–1.0 mg/dL // 0.0–0.3 mg/dL	2–17 µmol/L // 0–5 µmol/L
* Calcium, serum (Ca^{2+})	8.4–10.2 mg/dL	2.1–2.8 mmol/L
* Cholesterol, serum	Rec: < 200 mg/dL	Rec: < 5.2 mmol/L
Cortisol, serum	0800 h: 5–23 µg/dL // 1600 h: 3–15 µg/dL	0800 h: 138–635 nmol/L // 1600 h: 82–413 nmol/L
	2000 h: ≤ 50% of 0800 h	2000 h: Fraction of 0800 h: ≤ 0.50
Creatine kinase, serum	Male: 25–90 U/L	Male: 25–90 U/L
	Female: 10–70 U/L	Female: 10–70 U/L
* Creatinine, serum	0.6–1.2 mg/dL	53–106 µmol/L

Electrolytes, serum	Reference Range	SI Reference
Sodium (Na^+)	136–145 mEq/L	136–145 mmol/L
Chloride (Cl^-)	95–105 mEq/L	95–105 mmol/L
* Potassium (K^+)	3.5–5.0 mEq/L	3.5–5.0 mmol/L
Bicarbonate (HCO_3^-)	22–28 mEq/L	22–28 mmol/L
Magnesium (Mg^{2+})	1.5–2.0 mEq/L	0.75–1.0 mmol/L

Estriol, total, serum (in pregnancy)	Reference Range	SI Reference
24–28 wks // 32–36 wks	30–170 ng/mL // 60–280 ng/mL	104–590 // 208–970 nmol/L
28–32 wks // 36–40 wks	40–220 ng/mL // 80–350 ng/mL	140–760 // 280–1210 nmol/L
Ferritin, serum	Male: 15–200 ng/mL	Male: 15–200 µg/L
	Female: 12–150 ng/mL	Female: 12–150 µg/L
Follicle-stimulating hormone, serum/plasma	Male: 4–25 mIU/mL	Male: 4–25 U/L
	Female: premenopause 4–30 mIU/mL	Female: premenopause 4–30 U/L
	midcycle peak 10–90 mIU/mL	midcycle peak 10–90 U/L
	postmenopause 40–250 mIU/mL	postmenopause 40–250 U/L

Gases, arterial blood (room air)	Reference Range	SI Reference
pH	7.35–7.45	$[H^+]$ 36–44 nmol/L
Pco_2	33–45 mm Hg	4.4–5.9 kPa
Po_2	75–105 mm Hg	10.0–14.0 kPa
* Glucose, serum	Fasting: 70–110 mg/dL	Fasting: 3.8–6.1 mmol/L
	2-h postprandial: < 120 mg/dL	2-h postprandial: < 6.6 mmol/L
Growth hormone - arginine stimulation	Fasting: < 5 ng/mL	Fasting: < 5 µg/L
	provocative stimuli: > 7 ng/mL	provocative stimuli: > 7 µg/L

Blood

Immunoglobulins, serum	Reference Range	SI Reference
IgA	76–390 mg/dL	0.76–3.90 g/L
IgE	0–380 IU/mL	0–380 kIU/L
IgG	650–1500 mg/dL	6.5–15 g/L
IgM	40–345 mg/dL	0.4–3.45 g/L
Iron	50–170 µg/dL	9–30 µmol/L
Lactate dehydrogenase, serum	45–90 U/L	45–90 U/L
Luteinizing hormone, serum/plasma	Male: 6–23 mIU/mL	Male: 6–23 U/L
	Female: follicular phase 5–30 mIU/mL	Female: follicular phase 5–30 U/L
	midcycle 75–150 mIU/mL	midcycle 75–150 U/L
	postmenopause 30–200 mIU/mL	postmenopause 30–200 U/L
Osmolality, serum	275–295 mOsmol/kg H_2O	275–295 mOsmol/kg H_2O
Parathyroid hormone, serum, N-terminal	230–630 pg/mL	230–630 ng/L
* Phosphatase (alkaline), serum (p-NPP at 30°C)	20–70 U/L	20–70 U/L
* Phosphorus (inorganic), serum	3.0–4.5 mg/dL	1.0–1.5 mmol/L
Prolactin, serum (hPRL)	< 20 ng/mL	< 20 µg/L

* Proteins, serum	Reference Range	SI Reference
Total (recumbent)	6.0–7.8 g/dL	60–78 g/L
Albumin	3.5–5.5 g/dL	35–55 g/L
Globulin	2.3–3.5 g/dL	23–35 g/L
Thyroid-stimulating hormone, serum or plasma	0.5–5.0 µU/mL	0.5–5.0 mU/L
Thyroidal iodine (^{123}I) uptake	8 %–30 % of administered dose/24 h	0.08–0.30/24 h
Thyroxine (T_4), serum	5–12 µg/dL	64–155 nmol/L
Triglycerides, serum	35–160 mg/dL	0.4–1.81 mmol/L
Triiodothyronine (T_3), serum (RIA)	115–190 ng/dL	1.8–2.9 nmol/L
Triiodothyronine (T_3) resin uptake	25 %–35 %	0.25–0.35
* Urea nitrogen, serum	7–18 mg/dL	1.2–3.0 mmol/L
* Uric acid, serum	3.0–8.2 mg/dL	0.18–0.48 mmol/L

* Included in the Biochemical Profile (SMA-12)

Hematologic

Hematologic	Reference Range	SI Reference
Bleeding time (template)	2–7 minutes	2–7 minutes
Erythrocyte count	Male: 4.3–5.9 million/mm3	Male: 4.3–5.9 x 1012/L
	Female: 3.5–5.5 million/mm3	Female: 3.5–5.5 x 1012/L
Erythrocyte sedimentation rate (Westergren)	Male: 0–15 mm/h	Male: 0–15 mm/h
	Female: 0–20 mm/h	Female: 0–20 mm/h
Hematocrit	Male: 41 %–53 %	Male: 0.41–0.53
	Female: 36 %–46 %	Female: 0.36–0.46
Hemoglobin A_{1c}	≤ 6 %	≤ 0.06 %
Hemoglobin, blood	Male: 13.5–17.5 g/dL	Male: 2.09–2.71 mmol/L
	Female: 12.0–16.0 g/dL	Female: 1.86–2.48 mmol/L
Hemoglobin, plasma	1–4 mg/dL	0.16–0.62 mmol/L

Leukocyte count and differential	Reference Range	SI Reference
Leukocyte count	4500–11,000/mm^3	4.5–11.0 x 109/L
Segmented neutrophils	54 %–62 %	0.54–0.62
Bands	3 %–5 %	0.03–0.05
Eosinophils	1 %–3 %	0.01–0.03
Basophils	0 %–0.75 %	0–0.0075
Lymphocytes	25 %–33 %	0.25–0.33
Monocytes	3 %–7 %	0.03–0.07
Mean corpuscular hemoglobin	25.4–34.6 pg/cell	0.39–0.54 fmol/cell
Mean corpuscular hemoglobin concentration	31 %–36 % Hb/cell	4.81–5.58 mmol Hb/L
Mean corpuscular volume	80–100 μm^3	80–100 fL
Partial thromboplastin time (activated)	25–40 seconds	25–40 seconds
Platelet count	150,000–400,000/mm^3	150–400 x 10^9/L
Prothrombin time	11–15 seconds	11–15 seconds
Reticulocyte count	0.5 %–1.5 % of red cells	0.005–0.015
Thrombin time	< 2 seconds deviation from control	< 2 seconds deviation from control

Volume	Reference Range	SI Reference
Plasma	Male: 25–43 mL/kg	Male: 0.025–0.043 L/kg
	Female: 28–45 mL/kg	Female: 0.028–0.045 L/kg
Red cell	Male: 20–36 mL/kg	Male: 0.020–0.036 L/kg
	Female: 19–31 mL/kg	Female: 0.019–0.031 L/kg

Cerebrospinal

Cerebrospinal Fluid	Reference Range	SI Reference
Cell count	0–5/mm^3	0–5 x 10^6/L
Chloride	118–132 mEq/L	118–132 mmol/L
Gamma globulin	3 %–12 % total proteins	0.03–0.12
Glucose	40–70 mg/dL	2.2–3.9 mmol/L
Pressure	70–180 mm H_2O	70–180 mm H_2O
Proteins, total	< 40 mg/dL	< 0.40 g/L

Sweat, Urine, BMI

Sweat	Reference Range	SI Reference
Chloride	0–35 mmol/L	0–35 mmol/L

Urine	Reference Range	SI Reference
Calcium	100–300 mg/24 h	2.5–7.5 mmol/24 h
Chloride	Varies with intake	Varies with intake
Creatinine clearance	Male: 97–137 mL/min	Male: 97–137 mL/min
	Female: 88–128 mL/min	Female: 88–128 mL/min

Estriol, total (in pregnancy)	Reference Range	SI Reference
30 wks	6–18 mg/24 h	21–62 µmol/24 h
35 wks	9–28 mg/24 h	31–97 µmol/24 h
40 wks	13–42 mg/24 h	45–146 µmol/24 h
17-Hydroxycorticosteroids	Male: 3.0–10.0 mg/24 h	Male: 8.2–27.6 µmol/24 h
	Female: 2.0–8.0 mg/24 h	Female: 5.5–22.0 µmol/24 h
17-Ketosteroids, total	Male: 8–20 mg/24 h	Male: 28–70 µmol/24 h
	Female: 6–15 mg/24 h	Female: 21–52 µmol/24 h
Osmolality	50–1400 mOsmol/kg H_2O	50–1400 mOsmol/kg H_2O
Oxalate	8–40 µg/mL	90–445 µmol/L
Potassium	Varies with diet	Varies with diet
Proteins, total	< 150 mg/24 h	< 0.15 g/24 h
Sodium	Varies with diet	Varies with diet
Uric acid	Varies with diet	Varies with diet

Body Mass Index (BMI)	Adult: 19–25 kg/m^2